National Institute on Alcohol Abuse and Alcoholism

Alcohol Consumption and Problems in the General Population

Findings From the 1992 National Longitudinal Alcohol Epidemiologic Survey

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
National Institutes of Health

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June 2002

National Institutes of Health

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Acknowledgment

The National Institute on Alcohol Abuse and Alcoholism gratefully acknowledges the Census Bureau Field Representatives who collected data for the National Longitudinal Alcohol Epidemiologic Survey (NLAES) in 1992 and those who made the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) in 2001–2002 the landmark study it is today.

Through their dedication and generosity, the Census Bureau Field Representatives and NLAES and NESARC participants have greatly contributed to reducing the pain and suffering of Americans and their families who have struggled with alcohol, drug, and mental health problems. Their contribution to advancing science in these areas is immeasurable.

The NLAES and NESARC also could not have succeeded without the 86,000 Americans who participated in the surveys. Their cooperation and willingness to sacrifice their time to serve the public health are admirable and laudable.

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Foreword

The purpose of this publication is to consolidate in a single volume more than 40 journal articles based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES) of the adult population of the United States. It is intended as a companion to *Drinking in the United States: Main Findings From the 1992 National Longitudinal Alcohol Epidemiologic Survey*, a compilation of NLAES data tables published by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) in 1998 (NIH Publication No. 99–3519).

The articles gathered in this volume (all authored or co-authored by staff of NIAAA's Division of Biometry and Epidemiology) originally appeared in more than a dozen scientific journals over the last 8 years. They address topics including alcohol consumption patterns, alcohol dependence and abuse, medical consequences of alcohol consumption, family history of alcohol problems, alcohol and depression, alcohol and smoking, and treatment for alcohol dependence. Yet scientists, educators, and students seeking to understand the distribution of alcohol consumption and problems in the United States and wanting to identify studies based on this large and comprehensive national survey are hard-pressed to do so. A search of online bibliographic databases using the search terms "NLAES" or "National Longitudinal Alcohol Epidemiologic Survey" yields only a fraction of the articles actually published. Searches using other, more general, terms yield only some of them. Thus, in the absence of this compilation of NLAES articles, the full range of scientific knowledge and insight gained (to date) from analysis of the NLAES data might be known only to a small number of scientists and to persons already interested in the epidemiology of alcohol consumption and problems who also are skilled in bibliographic research.

We hope this compilation will help realize the potential of the NLAES for guiding the professional education of physicians, epidemiologists, and other scientists; informing the deliberations of public health officials and policymakers; and influencing the future of epidemiologic research.

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In 1992, the National Institute on Alcohol Abuse and Alcoholism (NIAAA) conducted the National Longitudinal Alcohol Epidemiologic Survey (NLAES), the most ambitious and comprehensive survey of its type ever performed. The NLAES included extensive questions about alcohol consumption. In addition to items designed to provide psychiatric classifications of alcohol- and drug-use disorders and major depression, the survey included items to capture information on a family history of alcoholism, alcohol and drug treatment, health conditions, and basic demographic characteristics. Direct face-to-face interviews were held with 42,862 respondents, 18 years of age and older, and living in the contiguous United States and the District of Columbia. The household response rate was 91.9 percent and the person response rate was 97.4 percent.

The major purposes of the NLAES were to:

- determine the prevalence of various drinking levels, alcohol use disorders, and their associated disabilities;
- identify risk factors associated with the initiation and/or maintenance of various drinking levels, alcohol use disorders, and their associated disabilities;
- determine the economic impact of alcohol and drug use disorders on impaired productivity in the workplace;
- examine the relationships between alcohol use disorders and their associated disabilities, including drug use disorders, major depression and physical disorders;
- estimate the size, characteristics, and time trends of populations of special concern, including people who abuse alcohol and other drugs and people in the general population who are otherwise impaired by alcohol and drug abuse;
- provide more complete recording and tabulation of the consequences of alcohol and drug abuse;
- develop and implement a comprehensive data capability for alcohol and drug abuse and dependence;
- obtain information on alcohol and drug treatment utilization among people in the general population who are not represented in periodic surveys of treatment facilities or populations in treatment;
- provide information concerning access and barriers to alcohol-related treatment services, particularly among low-income groups, women, young adults, and minorities; and
- determine the number of people in the population who are in need of, but not currently receiving, treatment for alcohol and/or drug abuse and dependence and their associated disabilities.

NLAES Design

The NLAES featured a complex multistage design. Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probability proportional to size. Approximately 200 PSUs were included in the 1992 NLAES sample, 52 of which were self-representing—that is, selected with certainty. Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically. Oversampling of the Black (i.e., African American) population was accomplished at this stage of sample selection. The decision to oversample the Black population was based on the fact that higher rates of alcohol-related disease (i.e., liver cirrhosis) are observed in that particular group.

Segments next were divided into clusters of approximately four to eight housing units, and all occupied housing units were included in the NLAES. Within each household, 1 randomly selected respondent, 18 years of age or older, was chosen to participate in the survey. Oversampling of young adults, 18–29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavy drinking population subgroup. This subgroup of young adults was sampled at a ratio of 2.25 to 1.00.

Because of the complex survey design of the NLAES, variance estimation procedures that assume a simple random sample could not be employed. Research has shown that clustering and stratification of the NLAES sample may result in standard errors much

larger than those obtained with a simple random sample of equal size. To take into account the NLAES sample design, all standard errors were generated with software programs that use appropriate statistical techniques to adjust for sample design characteristics.

Compilation of NLAES Information

In publishing this collection of articles based on the NLAES, NIAAA has two key objectives. The first is to provide a comprehensive compilation of research articles prepared by staff within NIAAA's Division of Biometry and Epidemiology. This monograph serves as a companion volume to the NLAES data compendium, *Drinking in the United States: Main Findings From the 1992 National Longitudinal Alcohol Epidemiologic Survey*, published in 1998. The second objective is to disseminate these basic findings as widely as possible, with a view toward stimulating ideas for future studies that, ultimately, will be useful in better understanding alcohol use, abuse and dependence and their associated physical and psychiatric disorders.

SUMMARY OF FINDINGS

This monograph is divided into 11 main chapters, each of which details an important aspect of the NLAES survey. The following sections briefly summarize the results of each chapter.

Chapter 1. Alcohol Consumption Patterns

General population surveys such as the NLAES are an invaluable source of data on alcohol consumption. Unlike sales data, which estimate only the total volume of alcohol consumed, survey data on alcohol consumption provide estimates of drinking status (e.g., abstainers, current drinkers, former drinkers) and drinking patterns (e.g., overall frequency of drinking, usual and largest quantities consumed, and frequency of hazardous drinking). Also, because survey data are linked with other individual-level data, they describe variations in consumption among different groups of drinkers and can be used to study the association between alcohol intake and different types of alcohol-related outcomes.

Subgroup Variation in U.S. Drinking Patterns: Results of the 1992 National Longitudinal Alcohol Epidemiologic Study—provides an overview of U.S. drinking patterns within various sociodemographic subgroups of the adult population. Those results indicate that 44.4 percent of adults 18 years of age and over were classified by the NLAES as current drinkers, that is, as having consumed at least 12 alcoholic drinks in the year preceding interview. An additional 21.6 percent were classified as former drinkers, and 34.0 percent were classified as lifetime abstainers who had never consumed 12 or more drinks in a single year. The prevalence of current drinking decreased with age but increased with education and income, was higher for men than women, was reduced among Black and Hispanic adults, and varied according to marital status and place of residence. Among current drinkers, heavy drinking tended to be most common below age 30, among men, and among people with relatively low levels of education and income.

Beyond Black, White and Hispanic: Race, Ethnic Origin and Drinking Patterns in the United States—examines variations in consumption by race and ethnic origin in greater detail. The findings suggest that cultural forces exert a strong influence on drinking behavior and that those forces may persist even after many generations of presumed acculturation. This paper reports that among Whites, those of Hispanic and Native American origin were the least likely to drink but consumed the most alcohol on days when they did drink. Whites of Southern and Eastern European descent tended to have more moderate drinking patterns and to consume more wine than those of Northern and Central European origin. Among both Blacks and Hispanic Whites, those of Caribbean ancestry demonstrated the most moderate drinking patterns. Within the category of "other" race (e.g., neither Black nor White) people of Asian origin (with the exception of those from Japan) were the most moderate drinkers. Although some of the Black/White differences disappeared after adjusting for marital status, income, and education, most of these differences by ethnic origin remained significant even after accounting for those factors.

Temporal Drinking Patterns and Variation in Social Consequences—describes a unique feature of the NLAES—the inclusion of questions regarding days of the week and times of day when drinking took place. Among regular drinkers who did not restrict their drinking to a few special occasions, 60.3 percent typically did not drink until 6:00 p.m. or later. Less than one-tenth reported usually drinking before 3:00 p.m., and only 1.2 percent drank before 11:00 a.m. Late-night drinking (between midnight and 6:00 a.m.) was reported by 7.7 percent of drinkers overall and was most common among young, unmarried, and heavy-volume drinkers. Among late-night drinkers, the rates of interpersonal problems and hazardous alcohol use were tripled; the rates of job, school, and legal prob-

lems were even more sharply increased. After adjusting for other individual characteristics, late-night drinking remained a significant predictor of all outcomes except legal problems, and interactions between the timing of drinking and other factors suggested that the importance of timing may be affected by where and with whom drinking takes place.

Drinking Patterns Among Individuals With and Without DSM–IV Alcohol Use Disorders—documents differences in 11 measures of past-year alcohol consumption among 3 groups of drinkers. The groups included (1) those who did not meet the DSM–IV criteria for either past-year alcohol abuse or dependence, (2) those with abuse only, and (3) those with dependence (with or without abuse). For all measures of quantity and frequency of drinking, including frequency of heavy drinking, intoxication, daily drinking, and drinking on weekdays and in the morning, the values for abusers were approximately midway between those for people without any disorder and those with dependence. Of the 11 measures that were examined, frequency of intoxication showed the strongest association with the probability of having an alcohol use disorder. Variations in consumption by age, gender, and race/ethnicity were similar for people with and without alcohol disorders, suggesting that the cultural, physiological, and normative factors that influence alcohol consumption exert an influence even in the presence of abuse and dependence and should be considered when designing approaches to treatment.

Chapter 2. Alcohol Dependence and Abuse

In addition to items on alcohol consumption, the NLAES was the first nationwide household survey to include a fully structured diagnostic interview for alcohol use disorders based on the most recent criteria from the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM–IV) (American Psychiatric Association, 1994). These items allowed for classification of respondents as meeting criteria for alcohol dependence or alcohol abuse in the year immediately before the interview and/or at any time before that year.

Prevalence of DSM–IV Alcohol Abuse and Dependence: United States, 1992—was the first paper to publish national one-year prevalence estimates of DSM–IV alcohol dependence, alcohol abuse, or both, for the total adult U.S. population. The prevalence estimate for combined alcohol abuse and dependence was 7.4 percent (representing 13.76 million people). Prevalence was greater among males (11.0 percent) than females (4.1 percent) and among nonBlacks (7.7 percent) than among Blacks (5.3 percent). Prevalence for males was 22.1, 10.7, 5.6, and 1.2 percent for ages 18 to 29, 30–44, 45–64, and 65 and older, respectively; prevalence for females was 9.9, 4.0, 1.5, and 0.3 percent for ages 18 to 29, 30–44, 45–64, and 65 and older, respectively.

Prevalence and Correlates of Alcohol Use and DSM–IV Alcohol Dependence in the United States: Results of the National Longitudinal Alcohol Epidemiologic Survey—presents updated estimates of the prevalence, and examines the correlates, of alcohol use and DSM–IV alcohol dependence. The prevalence of lifetime alcohol use was 66.0 percent, with 44.4 percent of the respondents reporting regular alcohol use during the past 12 months. Lifetime and 12-month prevalence of alcohol dependence were estimated at 13.3 percent and 4.4 percent, respectively. Men were significantly more likely than women to use alcohol, and alcohol use and dependence were much more common among cohorts born after Prohibition and after World War II. Members of the youngest cohorts, between the ages of 18 and 34 years at the time of the interview, were more likely to use drugs, to become dependent, and to persist in dependence compared to the older cohorts. In addition, the conditional probability of dependence among users was greatest in Cohort 1 (born between 1968 and 1974) after early adolescence, compared to Cohort 2 (born between 1958 and 1967), despite the finding that the probability of lifetime use was lower in Cohort 1 compared to Cohort 2. The sociodemographic covariates of first-time use, onset of dependence, and persistence of dependence varied as a function of the stage of progression. Implications of these findings are discussed in terms of changes over time in drinking patterns, dependence liability, and vulnerability among recent alcohol users.

Gender Differences in the Risk of Alcohol Dependence: United States, 1992—reveals that 23.7 percent of male lifetime drinkers and 15.4 percent of female lifetime drinkers met DSM–IV criteria for lifetime alcohol dependence (i.e., dependence during the year preceding the interview or in any 12-month period prior to that year). The median interval from first drink to onset of dependence was 3.6 years for men and 3.0 years for women. After using survival techniques to adjust for potential gender differences in exposure to risk of developing alcohol dependence, the cumulative conditional probability of having experienced onset of dependence was 35.1 percent for men and 24.6 percent for women. The conditional proba-

bility of onset of dependence was equal for men and women in the first year after initiation of drinking, about 30 percent higher for men in the period 1 to 4 years after the first drink, and about 45 percent higher for men thereafter. After using proportional-hazards models to adjust for the effects of age cohort, race and ethnicity, family history of alcoholism, and age at first drink, these period-specific risk ratios remained virtually unchanged. When a measure of average daily ethanol intake during periods of heaviest consumption was included, it rendered most of the gender differences statistically insignificant. Results revealed a slightly elevated risk of dependence in women within the first year after initiation of drinking among the heaviest drinkers. The excess risk of dependence in men was mostly limited to those with average daily intakes of less than one ounce of ethanol. These results suggest that different frequencies of binge drinking might help to account for gender differences and that men's and women's relative risks of developing alcohol dependence may vary as a function of lifecycle state, with men's greatest risk occurring during the college/young adult years.

Age at Onset of Alcohol Use and Its Association With DSM–IV Alcohol Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey—focuses on data from 27,616 current and former drinkers in the NLAES to examine the relationship between age at first-time use of alcohol and the prevalence of lifetime DSM–IV alcohol abuse and alcohol dependence among all U.S. adults 18 years of age and over and within subgroups defined by sex and race. The rates of lifetime dependence declined from more than 40 percent among people who started drinking at age 14 or younger to roughly 10 percent among those who started drinking at age 20 or older. The rates of lifetime abuse declined from just over 11 percent among those who initiated use of alcohol at age 16 or younger to approximately 4 percent among those whose onset of use was at age 20 or older. After using multivariate logistic regression models to adjust for potential confounders, the odds of dependence decreased by 14 percent with each increasing year of age at onset of use, and the odds of abuse decreased by 8 percent.

Comorbidity Between DSM–IV Alcohol and Drug Use Disorders: Results From the National Longitudinal Alcohol Epidemiologic Survey—examines the comorbidity (or simultaneous occurrence) of DSM–IV alcohol and drug use disorders (abuse only, dependence only, and combined abuse/dependence). The study reviews the following drugs: any drug, prescription drug, sedative, tranquilizer, amphetamine, cannabis (marijuana), cocaine, and hallucinogens. The prevalence of any drug use disorder with a history of an alcohol use disorder was 23.1 percent, compared with 2.3 percent among respondents who did not have an alcohol use disorder. Virtually all odds ratios for specific drug groups were significantly greater than 1.0, demonstrating that the comorbidity of alcohol and drug use disorders is pervasive in the general population.

Alcohol and Drug Use, Abuse, and Dependence Among Welfare Recipients—presents national estimates of heavy drinking, drug use, and alcohol and drug abuse and/or dependence among recipients of selected welfare programs. Small percentages of AFDC, WIC, food stamp, SSI, and Medicaid recipients were heavy drinkers (6.4 to 13.8 percent), used drugs (3.8 to 9.8 percent), or abused or were dependent on alcohol (4.3 to 8.2 percent) or other drugs (1.3 to 3.6 percent). These rates among welfare recipients were similar to NLAES national estimates for heavy drinking (14.5 percent), any drug use (5.0 percent), alcohol abuse and/or dependence (7.4 percent) and drug abuse and/or dependence (1.5 percent). Those rates also were comparable to rates of heavy drinking (14.8 percent), drug use (5.1 percent), alcohol abuse and/or dependence (7.5 percent), and drug abuse and/or dependence (1.5 percent) among the subpopulation of the United States not receiving welfare benefits. Although a minority of welfare recipients were shown to have alcohol or drug problems, substance abuse prevention and treatment services are needed among high-risk sub-groups of this population.

Chapter 3. Medical Consequences of Alcohol Consumption

The NLAES included questions about 22 specific medical conditions. Respondents were asked if they ever had the condition, if they had the condition in the last 12 months, how old they were when they first had the condition, and if the condition had been diagnosed or confirmed by a physician. These items provide a strong basis for looking at alcohol's effect on health.

Medical Consequences of Alcohol Consumption—United States, 1992—examines the relationship of various health problems to drinking level. Respondents were classified as lifetime abstainers (i.e., those who never had 12 or more drinks in any one year), former drinkers (i.e., those who had 12 or more drinks in at least one year but not in the last 12 months), light drinkers (i.e., those who had an average of less than 3

drinks per week), moderate drinkers (i.e., those who had 3 to 14 drinks per week), moderately heavy drinkers (i.e., those who had more than 2 but less than 5 drinks per day), or very heavy drinkers (i.e., those who averaged 5 or more drinks per day). Results showed that lifetime abstainers and very heavy drinkers had a significantly elevated prevalence of coronary heart disease compared to light and moderate drinkers. Very heavy drinkers also had greater risk than other groups for diseases of the liver or pancreas and certain types of cancer.

Alcoholic Beverage Preference and Risks of Alcohol-Related Medical Consequences: A Preliminary Report From the National Longitudinal Alcohol Epidemiologic Survey—examines morbidity associated with different types of alcoholic beverage (e.g., beer, wine, distilled spirits). Drinking levels in this study were defined as in the study mentioned above with the following differences: light-to-moderate drinkers consumed an average of up to 1.0 ounces of ethanol (or 2 standard drinks) per day for men and 0.5 ounces per day for women; heavy drinkers drank more than 1.0 but no more than 2.5 ounces per day for men and more than 0.5 but no more than 1.67 ounces per day for women; respondents with average consumption levels that were greater than heavy were classified as very heavy drinkers. Beverage preference was assigned to respondents with more than 85 percent of total ethanol intake from one specific type of alcohol beverage. The findings showed that the rates of various physical disorders tended to be lower among beer and wine drinkers than among abstainers or distilled-spirits drinkers.

Chapter 4. Family History of Alcohol Problems

Both genetic and environmental exposure to alcoholic relatives appears to increase the risk that a person will become dependent on alcohol at some time in his or her life. Family, twin, and adoption studies have consistently documented familial aggregation of alcohol problems, and the role of family history appears to be particularly strong in early onset alcoholism. The NLAES classification of alcohol use disorders in survey respondents, coupled with respondent reports of alcohol problems in 18 different types of first- and second-degree blood relatives, have been used to study the role of family history of alcoholism in several ways.

Estimates of U.S. Children Exposed to Alcohol Abuse and Dependence in the Family—links data on alcohol use disorders in NLAES respondents with information on other household members to yield estimates of the number of children in the United States who are exposed to alcohol abuse and dependence in the family. This study found that 9.7 million children 17 years of age and younger (roughly 15 percent of all children in this age range) were living in households containing one or more adults with alcohol abuse or dependence. In most cases (70.4 percent), the adult with alcohol problems was a parent; in other cases, abuse and/or dependence was present in siblings, other relatives living in the household, or unrelated adult household members. In addition, 28.0 million children (43 percent) lived in households where at least one adult had at some point in his or her life abused or been dependent on alcohol, although not necessarily during the child's lifetime. Thus the true estimate of the number of children exposed to alcohol problems in the home is somewhere between 9.7 and 28.0 million.

The Impact of a Family History of Alcoholism on the Relationship Between Age at Onset of Alcohol Use and DSM–IV Alcohol Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey—examines the impact of familial alcoholism on the relationship between age at onset of drinking and the risk of subsequently developing alcohol dependence. These results found that both age at onset of alcohol use and family history of alcoholism were strongly and independently associated with the risk of dependence. People who began drinking before age 14 were almost 4 times more likely to become alcoholic than those who began drinking at age 21 or older. This finding held true for people with and without a family history of alcoholism. Similarly, people with a family history of alcoholism were approximately twice as likely to become dependent themselves, regardless of the age at which they started drinking. This finding suggests that early drinking is not merely a manifestation of an underlying familial propensity to alcohol problems, but rather a separate and potentially modifiable risk factor for alcoholism.

The Link Between Family History and Early Onset Alcoholism: Earlier Initiation of Drinking or More Rapid Development of Dependence?—investigates the especially strong link between a family history of alcoholism and early—as opposed to late—onset alcoholism. The paper probes whether this link reflects the influence of earlier initiation of drinking, or a more rapid progression from drinking to dependence among people with positive family histories. The study found that having a high proportion of alcoholic relatives more strongly increased the risk of initiating drinking at an early age than it did at later ages. People with alcoholism in 25 percent of their relatives were 2.2 times more likely to start drinking before age 15 than were

people without alcoholic relatives, but their risk was only 1.3 times as great for starting to drink at ages 18 and older. After accounting for age at initiation of drinking, the direct effect of familial alcoholism on the risk of developing dependence increased over time. That is, people with a positive family history of alcoholism had a greater excess risk of developing dependence 5 or more years after the onset of drinking than they did within the first 5 years of drinking. Thus the association between familial alcoholism and early onset alcohol dependence primarily appears to reflect initiation of drinking at earlier ages. This suggests that not only genetic but also environmental factors, such as availability of liquor and poor supervision of youth, may help to explain the association between family history and early onset alcoholism.

Chapter 5. Alcohol and Depression

The NLAES included questions designed to facilitate assessment of DSM–IV major depression. Diagnoses of major depression required the presence of at least five depressive symptoms (necessarily including depressed mood or loss of pleasure and interest) nearly every day for most of the day for at least the same 2-week period. In contrast to the earlier DSM–III–R definition of major depression, social and/or occupational dysfunction also must have been present during the disturbance, and episodes of DSM–IV major depression exclusively resulting from bereavement and physical illness were excluded.

Comorbidity Between DSM–IV Alcohol Use Disorders and Major Depression: Results of a National Survey—explores detailed patterns of comorbidity between DSM–IV alcohol use disorders and major depression using a representative sample of the United States population. Comorbidity rates and associations between DSM–IV alcohol use disorders and major depression were expressed as odds ratios with confidence intervals adjusted for the complex design characteristics of the NLAES. Comorbidity analyses were presented according to sex, ethnicity, and age for past-year, prior-to-past-year, and lifetime diagnoses. Virtually all odds ratios were significantly greater that 1.0, demonstrating that comorbidity of alcohol use disorders and major depression is pervasive in the general population. The magnitude of the association remained stable across the three time frames examined, but diagnostic and subgroup variations in comorbidity were noted. The association between alcohol dependence and major depression was greater than the association between alcohol abuse and major depression. Further, the association between alcohol abuse and major depression was consistently greater for women and Blacks, compared to their male and nonBlack counterparts.

The Relationship Between DSM–IV Alcohol Use Disorders and DSM–IV Major Depression: Examination of the Primary-Secondary Distinction in a General Population Sample—describes the primary-secondary distinction with respect to DSM–IV alcohol use disorders and major depression. Respondents included primary (onset of first episode of depression preceded onset of alcohol use disorder), secondary (onset of first episode of alcohol use disorder preceded onset of depression), and concurrent (first episodes of depression and alcohol use disorder occurred at same age) depressives; those with major depression only; and those with alcohol use disorders only. The respondents were compared on a large number of variables, including sociodemographic, alcohol-related, depression-related, and drug-related. One of the most significant findings from this study was that primary depressives had more serious episodes of major depression than any other group. Respondents in the primary depression group reported a greater number of episodes of depression, a greater number of depressive symptoms, and were significantly more likely than the other depressive groups to report suicidal thinking, ideation, or attempts during their worst period of depression.

Gender Differences in DSM–IV Alcohol Use Disorders and Major Depression as Distributed in the General Population: Clinical Implications—examines gender differences within and between five groups of subjects drawn from a large representative sample of the U.S. population and classified as having either major depression only; alcohol use disorder only; or primary, secondary, or concurrent depression. This study sought to determine if these diagnostic profiles were (1) consistent with those drawn on clinical samples and (2) suggestive of potential clinical implications. Respondents who met DSM–IV criteria for classification into these five mutually exclusive groups were compared within and between groups by gender on the characteristics of each disorder. The results were consistent with those of other studies: (1) gender distributions of alcohol use disorder and depressive disorder remained nearly mirror opposites, and (2) the severity of comorbid disorders was greater than the severity of either condition when it occurred alone. Findings of particular interest were that the synergistic effects of an alcohol and a depressive condition operate equally for both men and women with concurrent depression. This points to the necessity of attending carefully to gender biases when dealing with comorbid conditions, lest alcoholism in the presence of depression not be taken seriously enough in

women and vice versa in men. Additionally, women with primary depression are at high risk for suicide and thus may require special attention in the evaluative phase of treatment.

Family History of Alcoholism and Gender: Their Combined Effects on DSM–IV Alcohol Dependence and Major Depression—uses multiple logistic regression models to assess the extent of familial alcoholism; to examine its association with the odds of DSM–IV lifetime alcohol dependence, major depression, and their comorbid occurrence; and to determine gender differences in the magnitude of this association. After adjusting for potential confounders, family history saturation (i.e., the proportion of alcoholic first- and second-degree relatives) was associated with increased odds of dependence only, depression only, and all primary-secondary concurrent combinations. The estimated effects were greatest for comorbid dependence and depression, next highest for dependence only, and lowest for depression only. Differences in odds ratios among these groups increased with the degree of family history saturation but were statistically significant at all levels of saturation. The effects of family history were greater for men than women for primary depression, but only at the higher levels of saturation. Among people with lifetime major depression, family history of alcoholism had a positive independent association with the conditional odds of having experienced comorbid alcohol dependence. It had a weaker but significant association with the odds of comorbid depression conditional upon having experienced dependence, and this association was stronger among men than among women. For most outcomes, family history effects were stronger for paternal male and maternal female relatives than for paternal female and maternal male relatives.

Familial Aggregation of DSM–IV Alcohol Use Disorders: Examination of the Primary-Secondary Distinction in a General Population Sample—uses NLAES data to explore the familial aggregation of alcoholism in subgroups of men and women classified with respect to the primary-secondary distinction as it relates to DSM–IV major depression and alcohol use disorders. The risk of alcoholism among first- and second-degree relatives was examined among the following comorbid subgroups: (1) those with primary major depression and secondary alcohol use disorder; (2) those with secondary major depression and primary alcohol use disorder; and (3) those in which the onset of alcohol use disorder and major depression were concurrent. For comparison, there were two groups of respondents with major depression only and normal control subjects. The study focused on whether the results of familial aggregation studies supporting the independent transmission of alcoholism and major depression could be confirmed. Results showed significantly greater aggregate rates of alcoholism among first- and second-degree relatives of men and women with major depression only, compared with normal control subjects. This finding suggests that alcoholism and major depression may be alternate manifestations of the same underlying disorder. The discrepancy between clinical research findings and this general population study provides support for the hypothesis that alcoholism and depression are similar disorders and establishes the relevance of the primary-secondary distinction in studies of familial aggregation.

Chapter 6. Alcohol and Smoking

Studies of clinical samples, adolescents, college students, and the general adult population have all confirmed a positive association between drinking and smoking. Drinkers are more likely than nondrinkers to be smokers and vice versa, and using either of these substances increases the risk of starting and continuing to use the other. Among the factors that may help to explain this association are cross-tolerance (using either substance increases tolerance to the other), shared genetic and environmental influences, and the ability of each substance to counteract some of the adverse effects of the other. Also, ethanol may affect nicotine metabolism, and disinhibition from alcohol use may counteract restraints against smoking. Data from the NLAES have been used to study the relationship between drinking and smoking from two different perspectives—considering smoking as a risk factor for developing alcohol use disorders and considering drinking as a risk factor for continued smoking.

Drinking as a Risk Factor for Sustained Smoking—examines the prevalence and characteristics of alcohol use disorders as a function of age at onset of smoking. These results found that the prevalence of lifetime alcohol dependence increased from 7.8 percent among lifetime nonsmokers to 12.4 percent in people who started smoking at age 17 or older, 18.9 percent of those who initiated smoking at ages 14 to 16 and 28.6 percent of those who started smoking at age 13 or younger. Similar linear patterns linked age at onset of smoking with the risk of lifetime alcohol abuse, the duration and severity of abuse and dependence episodes, and history of treatment for alcohol use disorders. In addition, the proportion of alcoholic relatives and volume of alcohol consumption in the past year and during the period of heaviest drinking were greater for

smokers than nonsmokers and were greatest among those who started smoking at the youngest ages.

Age at Smoking Onset and Its Association With Alcohol Consumption and DSM–IV Alcohol Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey—examines smoking as a function of drinking status, volume, and pattern, considering both lifetime and past-year time frames. The study found that past-year and former drinkers were twice as likely as lifetime abstainers (or nondrinkers) to have ever smoked, 59.9 percent and 63.3 percent, versus 28.2 percent. In addition, the prevalence of lifetime smoking rose from 49.1 percent among those who were light drinkers during their period of heaviest drinking to 72.9 percent for those who were heavy drinkers. More than two-thirds of people with lifetime alcohol abuse or dependence were lifetime smokers, compared to less than half of those without an alcohol use disorder. Heavy drinkers and people with alcohol abuse or dependence also were the more likely to smoke on a daily basis, to smoke more cigarettes on days when they did smoke, and were the least likely to have stopped smoking. The patterns were similar when past-year rather than lifetime measures were examined. In addition to smoking cessation being negatively associated with volume of alcohol consumption and alcohol use disorders in the past year, the likelihood of stopping smoking also decreased as the number of heavy drinking (i.e., five or more drinks) days increased. These findings—which link smoking cessation with volume of alcohol intake, frequency of heavy drinking, and alcohol use disorders—support virtually all of the existing proposed mechanisms for the association of smoking and alcohol.

Chapter 7. Drug Dependence and Abuse

The NLAES included questions that allowed for the assessment of DSM–IV drug use disorders. With appropriate algorithms, these items could classify respondents as meeting criteria for DSM–IV past-year and prior-to-past-year drug abuse or dependence for the following eight categories of drugs: sedatives, tranquilizers, painkillers, stimulants, marijuana, cocaine or crack, heroin, and methadone. These drug diagnoses enabled the analyses of prevalence of drug use disorders and studies of the comorbidity of alcohol use disorders, drug use disorders, and major depression.

Prevalence and Correlates of Drug Use and DSM–IV Drug Dependence in the United States: Results of the National Longitudinal Alcohol Epidemiologic Survey—presents updated estimates of the prevalence and examines the correlates of drug use and dependence in a representative sample of the U.S. population. The prevalence of lifetime drug use was 15.6 percent, with 4.9 of the respondents reporting drug use during the past 12 months. Lifetime and 12-month prevalence of drug dependence were estimated at 2.9 percent and 0.8 percent, respectively. Men were significantly more likely than women to use drugs, and drug use and dependence were much more common among cohorts born after World War II. The data indicated that in younger cohorts the rates of dependence among women were quickly approaching the rates among men. Members of the youngest cohort—ages 18 to 24 at the time of the interview—were more likely to use drugs, to become dependent, and to persist in dependence, than were the older cohorts, including Cohort 2, who experienced adolescence at the height of widespread introduction of illicit drugs among youth. The demographic correlates of first use, onset of dependence, and persistence of dependence varied as a function of the stage of progression.

Age of Onset of Drug Use and Its Association With DSM–IV Drug Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey—studies the relationship between early onset of drug use and the development of lifetime DSM–IV drug abuse and dependence using a representative sample of the U.S. population. Prevalences of lifetime drug abuse and dependence were estimated for each year of age of onset of drug use from age 13 and younger, to age 21 and older for the overall sample of drug users by race and gender. Linear logistic analyses were conducted to assess the relationship between age of drug use onset and lifetime drug use disorders controlling for important covariates. Early onset drug use was a significant predictor of the development of drug abuse over the life course. Early onset drug use also was a significant predictor of the subsequent development of lifetime alcohol dependence among males, females, and nonBlacks, but not among Blacks. After adjusting for important model covariates, the likelihood of lifetime drug abuse and dependence among the total sample of lifetime drug users was reduced by 4 percent and 5 percent (respectively) for each year that drug use onset was delayed.

The Relationship Between Cannabis Use and DSM–IV Cannabis Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey—examines the risk of DSM–IV cannabis (marijuana) abuse and dependence at different cannabis-use

levels in a representative sample of the United States general population. The prevalence of past-year alcohol dependence among those with DSM–IV cannabis abuse or dependence was 62.9 percent, compared to 26.2 percent among people who were negative for cannabis abuse or dependence. The association between cannabis use and cannabis abuse and dependence was determined by logistic regression analyses. Risk of cannabis abuse and dependence was found to increase with the frequency of smoking occasions and to decrease slightly with age. More severe comorbidity was associated with dependence rather than with abuse, suggesting that cannabis might be used to self-medicate major depression. Among women, but not among men, the strength of the association between cannabis use and abuse increased as a function of the number of marijuana cigarettes smoked.

Parallels to Early Onset Alcohol Use in the Relationship of Early Onset Smoking With Drug Use and DSM–IV Drug and Depressive Disorders: Findings From the National Longitudinal Epidemiologic Survey—examines whether (1) early onset of regular tobacco use is as predictive of drug use and depressive disorders as it is of alcohol use disorders; and (2) a biological commonality (as measured by a family history of alcoholism and both early onset and severity of disease) among all three disorders can be evidenced in a large nationally representative sample. Prevalences of lifetime drug use, abuse and dependence, and major depressive disorders, as well as indices of their severity, were compared among smoking groups defined by age at onset of regular tobacco use and among nonsmokers. Linear logistic regression analyses, controlling for important covariates, including a family history positive for alcoholism, were conducted to assess the relationship between age at smoking onset and drug use, abuse and dependence, as well as depressive disorders. The results supported each of the above hypotheses. Moreover, the results suggest that smoking may play an equally insidious, if not a more important, role as does drinking in the use and development of dependence on illicit substances and depression.

Chapter 8. Other Alcohol-Related Problems

The impact of alcohol use extends beyond alcohol use disorders, morbidity, and mortality to include a variety of social consequences, including psychological, interpersonal, job, and legal problems. Driving while under the influence of alcohol is among the most serious of these problems, and alcohol has been cited as a factor in 4 out of 10 fatal crashes in the United States. In addition, alcohol is increasingly being studied as a risk factor for violence, whether the violence is turned outward in the form of violent crime, fighting, or partner abuse or inward in the form of suicidal thoughts and attempts. Several analyses have used data from the NLAES to explore the association of alcohol use with impaired driving, suicide attempt and ideation, and alcohol-related fighting.

Alternative Definitions of High Risk for Impaired Driving: The Overlap of High Volume, Frequent Heavy Drinking and Alcohol Dependence—uses impaired driving as a means for comparing three high-risk groups of drinkers. These included high-volume drinkers (i.e., those with an average daily intake of one or more ounces of ethanol), frequent heavy drinkers (i.e., those who drank five or more drinks, one or more times a week), and dependent drinkers. Surprisingly, only a moderate overlap was found among these three groups. Of those drinkers who fell into any of the high-risk groups, only 14.2 percent were in all three groups. High-risk drinkers taken as a whole (i.e., those in one or more of the high-risk groups) comprised only 26.7 percent of all current drinkers. Yet those drinkers consumed 71.7 percent of the total amount of ethanol consumption reported by all current drinkers, accounted for 88.3 percent of all heavy-drinking days, and accounted for 79.5 percent of all impaired driving incidents. The small group of people who met all three definitions of risk drinking, while comprising only 3.8 percent of current drinkers, accounted for 21.3 percent of all ethanol intake, 30.6 percent of all heavy drinking days, and 36.4 percent of all impaired driving incidents.

Suicidal Ideation Among the United States Drinking Population: Results From the National Longitudinal Alcohol Epidemiologic Survey—examines past-year suicidal ideation (i.e., thinking a lot about suicide or wanting to die) as a function of alcohol intake, age at first drink, frequency of intoxication, alcohol dependence, family history of dependence, drug use, drug use disorders, and treatment for alcohol and/or drug problems, and depression. Major depression, bereavement, and sociodemographic risk factors also were considered, and separate analyses were conducted for men and women. Past-year major depression was the most significant predictor of suicidal ideation for men and women. Although comorbid alcohol dependence did not add to the increased risk in people with de-

pression, it did increase the odds of suicidal ideation by factors of 2.2 and 1.6, respectively, among men and women without major depression. Both family history of alcoholism and having had an alcohol use disorder prior to the past year increased the risk of suicidal ideation among men but not women; however, drug use and drug use disorders were significant risk factors only for women. The proportion of drinking occasions resulting in intoxication, age at first drink, and history of treatment for alcohol and/or drugs all were positively associated with suicidal ideation in bivariate analyses, but all failed to retain their statistical significance in multivariate analyses.

Alcohol, Drugs, Fighting and Suicide Attempt/Ideation—examines an expanded suicide outcome that comprises both suicide attempt and suicide ideation. The paper also looks at alcohol- and/or drug-related fighting. Like the study mentioned above ("Suicidal Ideation Among the United States Drinking Population: Results From the National Longitudinal Alcohol Epidemiologic Survey"), this study assessed the associations between these outcomes and alcohol and drug use. Unlike the other study, it did not account for a history of major depression or alcohol or drug use disorders. It did, however, examine the effects of individual drugs and of multiple versus single drug use. When drug use was ignored, both the average daily volume of ethanol intake and the proportion of drinking occasions resulting in intoxication were positively related to both the risk of fighting and of suicide attempt or ideation. But after accounting for drug use, the relationship between volume of intake and suicide attempt or ideation fell short of statistical significance. In addition, the increased risk of fighting associated with frequent intoxication was nonsignificant for people who also used marijuana but who did not use any other (i.e., multiple) drugs. The drugs associated with an increased risk of fighting were cocaine and/or stimulants and multiple drugs. Use of marijuana increased the risk of fighting among women but not among men. For the outcome of suicide attempt or ideation, the use of marijuana, sedatives and/or tranquilizers, cocaine and/or stimulants, and multiple drugs all acted as positive risk factors, with no difference in effect found between men and women.

Chapter 9. Treatment and Alcohol Dependence

More than half a million people receive treatment for alcoholism in the United States each day. Although data from clinical samples provide valuable information on the treatment population and correlates of treatment outcome, they cannot be used to predict entry into treatment or to identify factors that may prevent at-risk people from seeking or obtaining help with drinking problems. Survey data from the general population are uniquely well-suited to these tasks; in addition, they provide the sole means for comparing the natural history of alcohol use disorders among people who do and those who do not receive treatment. Providing information on treatment has been one of the most valuable applications for the NLAES data.

Toward an Alcohol Treatment Model: A Comparison of Treated and Untreated Respondents With DSM–IV Alcohol Use Disorders in the General Population—examines entry into alcohol treatment in association with three types of factors: predisposing factors that reflect an underlying propensity to seek treatment, enabling factors that reflect individual and community level resources facilitating the decision to seek treatment, and illness-severity factors that directly affect the need for treatment. The results revealed a complex set of interactions between illness severity and past treatment history, low education, and unemployment. For example, the positive effect of illness severity as an impetus for obtaining treatment was reduced by past treatment and less than a high school education and was increased by past-year employment. These findings suggest the need to examine individual determinants of alcohol treatment within the larger context of organizational and sociopolitical factors.

Correlates of Past-Year Status Among Treated and Untreated Persons With Former Alcohol Dependence: United States, 1992—examines past-year status among treated and untreated people who met the criteria for alcohol dependence at some point prior to the past year. Disregarding treatment status, 27.8 percent of these former alcoholics were classified with alcohol abuse or dependence in the past year; 22.3 percent abstained from drinking; and 49.9 percent reported drinking without meeting the criteria for abuse or dependence. Compared with people who did not receive treatment for their alcohol problems, those who received treatment were more likely to be abstinent (28.8 percent versus 16.4 percent) or to still have an alcohol use disorder (33.2 percent versus 25.8 percent). In multivariate analyses, treatment status modified the relationships between past-year status and many of its correlates, including sociodemographic factors and severity, age at onset, and rapidity of onset of the original disorder. These results suggest that the

findings from treatment studies may not apply to alcoholics who do not seek treatment.

Gender Differences in the Probability of Alcohol Treatment—compares the probability of obtaining treatment for alcohol problems for men and women. Overall, 23.0 percent of the men with lifetime alcohol abuse and/or dependence reported receiving treatment, compared to 15.1 percent of women with a lifetime alcohol use disorder. However, women who obtained treatment did so more rapidly than men, seeking treatment an average of 2.1 years after the onset of the disorder versus 5.0 years for men. A multivariate analysis that controlled for severity and age at onset, presence of comorbid disorders, and treatment for drug use disorders indicated that the greater probability of receiving treatment for alcohol problems found in men decreased with the severity of the disorder. Whereas men meeting the minimum standards for abuse or dependence were 75 percent and 67 percent more likely to have received treatment than women with comparable levels of severity, the effect of gender became nonsignificant among those with more than 15 positive symptoms of abuse or dependence.

Symptoms and Characteristics of Individuals With Different Types of Recovery From DSM–IV Alcohol Dependence—compares three groups of lifetime drinkers: 1) former alcoholics who achieved remission through abstinence, 2) former alcoholics who achieved remission despite continued drinking, and 3) people who never met the criteria for alcohol dependence. Members of the second group lay between members of the first and third groups in terms of most indicators of alcohol use and problems. Both groups of former alcoholics were equally likely to have experienced withdrawal, were drinking more or for longer than intended, and had developed symptoms of tolerance. Members of the first group, however, were far more likely to have experienced continued use despite physical or psychological consequences, to spend a lot of time drinking, and to give up activities in favor of drinking. The earlier onsets of heavy drinking and dependence in the second group, coupled with a reduced likelihood that they had received treatment, supports the existence of a developmentally limited type of alcoholism that is subject to remission without treatment in early adulthood.

The Influence of Comorbid Major Depression and Substance Use Disorders on Alcohol and Drug Treatment: Results of a National Survey—looks at treatment status as a possible explanation for why the associations among alcohol use disorders, drug use disorders, and major depression are generally smaller in magnitude in general population surveys than in studies based on clinical samples. Among people with alcohol use disorders, the prevalence of alcohol treatment increased from 7.8 percent in those without a comorbid drug use disorder or major depression and 14.9 percent in those with only a comorbid drug use disorder, to 20.6 percent of those with only comorbid major depression, and 35.3 percent of those with both a comorbid drug use disorder and major depression. Similarly, the presence of comorbid alcohol use disorders or major depression tended to increase the probability of drug treatment among those with drug use disorders. The increased tendency for people with multiple disorders to seek treatment suggests that measures of comorbidity based on clinical samples will overstate the true level of association in the general population.

Barriers to Alcoholism Treatment: Reasons for Not Seeking Treatment in a General Population Sample—examines barriers to alcohol treatment with an emphasis on differences in perceived barriers according to gender, race, and age. Of the 18.2 percent of NLAES respondents who were classified with lifetime alcohol abuse or dependence, only a small fraction (i.e., 12.7 percent of these) reported that they had perceived a need for treatment but failed to seek it. The reasons most commonly cited were (1) thinking they should be able to handle the problem themselves (28.9 percent), (2) not thinking the problem was serious enough (23.4 percent), (3) thinking that the problem would get better by itself (20.1 percent), (4) desire to keep drinking (12.6 percent), (5) fear of inability to pay (11.3 percent), and (6) embarrassment (11.2 percent). Compared to men, women were significantly more likely to cite (1) not knowing where to go for help, (2) inability to arrange for child care, or (3) feeling that the drinking was a symptom of another problem, as reasons for not seeking treatment. Reasons more commonly cited by Black than nonBlack respondents included (1) inability to arrange child care, (2) too long a wait, and (3) not wanting to go. Younger respondents were the most likely to cite of lack of time and fear of job loss as reasons for not seeking treatment.

Variations in the Prevalence of Alcohol Use Disorders and Treatment by Insurance Status—examines alcohol use disorders and treatment as a function of insurance status. The prevalence of past-year alcohol abuse and dependence varied from a high of 12.6 percent among those with no health insurance to a low of 1.2 percent among Medicare recipients. The prevalence of alcohol use disorders among people with private health

insurance was relatively low (6.7 percent). However, this form of insurance coverage was so widespread that nearly two-thirds of all people in need of alcohol treatment were privately insured. Of adults with past-year alcohol use disorders, the proportion who received alcohol treatment was highest for those with military insurance (20.3 percent); intermediate for those with no insurance (14.7 percent), Medicaid (14.9 percent) or Medicare (13.1 percent); and lowest for those with private insurance (7.7 percent). The discrepancy in treatment access by type of insurance coverage was greatest for those receiving inpatient treatment; there was little difference in the utilization of 12-step programs, which typically are free. These results demonstrate that access to alcohol treatment should be more equitably distributed across the public and private sectors.

Chapter 10. Guidelines for Public Health

The United States and other countries have developed guidelines for moderate or low-risk drinking that attempt to specify consumption levels at which alcohol poses little or no risk, or at which the possible benefits of alcohol consumption outweigh risks associated with excessive consumption. The NLAES provides considerable detail on amounts and patterns of alcohol consumption, including beverage-specific information on quantity, frequency, and size of drinks. The NLAES data also provide information on respondents' usual consumption and consumption during periods of heavier-than-usual drinking as well as items that asked about specific drinking patterns, such as the frequency of drinking five or more drinks per day and drinking enough to feel drunk. The detail of these consumption items, combined with various outcome items, provide a rich base of data for exploring the impact of various moderate-drinking guidelines on overall health.

Reducing Alcohol Use Disorders via Decreased Consumption: A Comparison of Population and High-Risk Strategies—compares three alternative scenarios for effecting a 25 percent reduction in U.S. alcohol consumption in terms of their respective impacts on the prevalence of alcohol abuse and dependence. The three approaches were (1) an overall 25 percent reduction in the volume of ethanol intake for all current drinkers, (2) an equivalent absolute reduction taken only among drinkers whose current intake ever exceeds moderate drinking guidelines, and (3) an equivalent reduction taken only among drinkers whose current intake usually exceeds moderate drinking guidelines. The per-occasion cut-off point for moderate consumption was set at the intake level demonstrated to produce psychomotor impairment, and was based on each person's total body water level. The impact of reducing consumption on the prevalence of alcohol use disorders was estimated using a logistic regression model that adjusted for sociodemographic characteristics, family history of alcoholism, and age at first drink, and which took into account interactions between the consumption and other variables. Taking an overall 25 percent reduction in intake resulted in the same decrease in the prevalence of abuse and dependence (21.7 percent) as was achieved by taking an equal volume of reduction among only those drinkers whose consumption usually exceeded the moderate drinking cut-off point. Restricting the reduction in consumption to those drinkers whose consumption ever exceeded this cut-off point resulted in a slightly greater reduction in alcohol use disorders (24.6 percent).

U.S. Low-Risk Drinking Guidelines: An Examination of Four Alternatives—compares four sets of U.S. low-risk drinking guidelines (two interpretations of the U.S. Dietary Guidelines and two variations of the NIAAA physicians' guidelines) in terms of adherence and whether they predicted five different alcohol-related outcomes. Using data from 17,542 adults who drank 12 or more drinks in the past year, sensitivity, specificity, overall accuracy, positive and negative predictive values, and odds ratios of the various drinking guidelines (having exceeded them with different degrees of frequency) were assessed as predictors of alcohol dependence, impaired driving, liver disease, peptic ulcer, and hypertension. The proportion of past-year regular drinkers exceeding the four sets of guidelines varied. For example, 20.9 percent had an average intake that exceeded the weekly limits; 21.0–42.7 percent exceeded the daily guidelines at least once a week; and 69.2–94.2 percent "ever exceeded" the daily limits in the year preceding the interview. Sensitivity and odds ratios were highest for "ever exceeding" the Dietary Guidelines daily limits, intermediate for "ever exceeding" the two variations based on the NIAAA physicians' guidelines, and lowest for exceeding the Dietary Guidelines interpreted as weekly limits. The opposite pattern was observed for specificity and overall predictive accuracy. When "frequently exceeding" the daily limits was considered, their sensitivity declined but the specificity and positive predictive value increased. If sensitivity and specificity are deemed equally important, the NIAAA physicians' guidelines incorporating both daily and weekly limits appear to be the most effective in balancing these dimensions in the prediction of a variety of alcohol-related outcomes.

Chapter 11. Measurement and Methods

In addition to generating descriptive statistics and testing hypotheses, the NLAES data have been used to examine the effects of different measurement approaches, to test the psychometric properties of various survey items, and to explore the nosological structure of various criteria for alcohol use disorders. These types of methodological analyses are critical to establishing the reliability and validity of measures of alcohol use and alcohol problems and to understanding the impact that assumptions underlying their construction may have on the interpretation of survey results.

Volume of Ethanol Consumption: Effects of Different Approaches to Measurement—uses data from the NLAES in conjunction with alcohol data from the 1988 National Health Interview Survey (NHIS) to compare estimates of average daily ethanol consumption; selected percentiles of the volume distribution; and the proportion of drinkers exceeding a volume-based cut-off point for moderate drinking, as derived from eight different approaches to measuring alcohol consumption. The approaches differed in terms of overall versus beverage-specific questions, length of reference period, use of standard versus respondent-specified drink sizes, and inclusion of measures of atypical heavy drinking. The estimated volume was highly sensitive to the number and type of questions asked and ranged from an average daily volume of 0.43 to 0.72 ounces. In addition, changes in formulation that resulted in small differences in mean volume often resulted in far larger increases in the proportion of drinkers exceeding a given cut-off point or in the association between consumption and alcohol use disorders.

Theoretical and Observed Subtypes of DSM–IV Alcohol Abuse and Dependence in a General Population Sample—quantifies the degree of heterogeneity of the DSM–IV alcohol abuse and dependence categories by comparing the number of theoretical subtypes of each category with those actually observed in the NLAES. Only 11 (47.8 percent) of the 23 theoretically predicted subtypes of abuse were represented in the data, with three subtypes accounting for approximately 90 percent of all past-year cases of abuse. Hazardous use was the single most prevalent subtype of abuse. Only 53 (53.5 percent) of the 99 theoretically predicted subtypes of dependence were observed, with six subtypes accounting for almost 70 percent of all past-year cases. All of the six subtypes contained one or more criteria for physiological dependence and one or more criteria for impaired control, suggesting they are potentially important for defining features of dependence. Although these results indicate that the diagnostic categories for both abuse and dependence were relatively homogeneous, heterogeneity was increased among men, Whites, and younger people.

The Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS): Reliability of Alcohol and Drug Modules in a General Population Sample—describes the reliability of the alcohol and drug modules of the NLAES survey instrument (i.e., AUDADIS), as estimated by a test-retest design carried out in a general population sample. After correcting for chance agreement in the interview and re-interview, the AUDADIS showed good-to-excellent reliability (i.e., kappa >.70) for past-year measures of alcohol consumption; use of amphetamines, cannabis, cocaine, and heroin; alcohol abuse and dependence; and drug abuse and dependence (any drug, cannabis and cocaine). For most alcohol and drug use disorders, an order effect was observed, with a slight decrease in prevalence in the re-interview. Although the reliability of alcohol and drug use disorder classifications generally is higher in clinical samples than in the general population (because of more borderline cases in the latter), the level of reliability obtained for the AUDADIS matched or exceeded those reported elsewhere for patient samples.

DSM–IV, DSM–III–R, and ICD–10 Alcohol and Drug Abuse/Harmful Use and Dependence, United States, 1992: A Nosological Comparison—uses the NLAES data to examine concordance in the classification of alcohol and drug dependence and abuse as based on the DSM–IV, DSM–III–R, and ICD–10 diagnostic criteria for these disorders. For most substances, the prevalence of dependence was highest when estimated by the DSM–III–R criteria. For abuse, the highest prevalence estimates derived from the DSM–IV criteria. For harmful use, the ICD–10 classification resulted in considerably lower prevalence estimates than for either DSM–IV or DSM–III–R abuse. There was excellent agreement across classification systems in the diagnosis of dependence, both for alcohol and for drugs. Cross-system comparisons between DSM–III–R and DSM–IV dependence were good to excellent, but concordance of either of the abuse diagnoses with ICD–10 harmful use was consistently poor. These results confirm the success of international efforts to integrate the DSM and ICD classifications of dependence but suggest that the conceptualization of harmful use needs more theoretical and empirical examination.

The Validity of DSM–IV Alcohol Abuse: Drunk Drivers Versus All Others—describes how almost half of the NLAES respondents who met the DSM–IV criteria for alcohol abuse were classified as abusers on the basis of only

one symptom, driving after drinking too much. To test the differential validity of this subgroup of abuse, three groups were compared with relation to their drinking patterns, family history of alcoholism, perceived need for treatment and comorbid conditions. Those three groups included (1) abusers who reported only drunk driving, (2) abusers who reported other symptoms (alone or in addition to drunk driving), and (3) those with no alcohol diagnosis. Whereas the second and third groups showed significant differences for all measures that were compared, the first and third groups differed in terms of only about half of the measures. The first group of abusers differed from the second in terms of being less likely to have comorbid depression, to use drugs, and to report blackouts, and by reporting fewer occasions of drinking to intoxication. This research suggests the need for more work in resolving difficulties with the DSM-IV alcohol abuse category.

SUMMARY

As evidenced by this compilation of findings, the NLAES offers valuable insight into the drinking practices and alcohol and drug use disorders and their associated disabilities of the U.S. population. As shown in the studies reported here, NLAES has provided a strong foundation for the design of future surveys in alcohol epidemiology. The next generation of epidemiological surveys will benefit greatly from the high standards for measurement and statistical and survey

CHAPTER 1
ALCOHOL CONSUMPTION PATTERNS

Subgroup Variation in U.S. Drinking Patterns: Results of the 1992 National Longitudinal Alcohol Epidemiologic Study

Deborah A. Dawson, Bridget F. Grant, S. Patricia Chou, and Roger P. Pickering

Data from the 1992 National Longitudinal Alcohol Epidemiologic Study (NLAES) revealed that 44% of U.S. adults 18 years of age and older were current drinkers who had consumed at least 12 drinks in the year preceding the interview. Twenty-two percent were former drinkers, and 34% were lifetime abstainers. These figures represent an 8% decrease in the prevalence of current drinking relative to 1988. The proportion of current drinkers decreased with age, was higher for men than women, increased with education and income, was lower than average among Black and Hispanic adults, was highest among never-married adults and lowest among those who were widowed, was lower in the South than in other regions, and was lower in rural than urban areas.

The probabilities of ever having consumed five or more (5+) drinks or having been intoxicated in the past year revealed similar patterns to those already noted, but the probabilities of heavy drinking or intoxication on a weekly or more frequent basis showed no variation by race or ethnicity. Average daily consumption of more than 1 ounce of ethanol differed from the preceding measure of heavy drinking in its variation across population subgroups, declining less sharply with age and exhibiting a U-shaped pattern with respect to income. Examination of the prevalence of heavy drinking among current drinkers rather than within the total population revealed several differences, the most striking reversal being that the probability of heavy drinking decreased with education and income. Multiple logistic regression models predicting the various drinking outcomes indicated that most of the differentials held true after adjusting for intercorrelation among the sociodemographic variables.

Understanding how alcohol consumption patterns vary among different subgroups of the U.S. population is essential to interpreting sociodemographic differentials in the prevalence of alcohol-related problems. Because studies of risk factors for alcohol problems often adjust for level of consumption (Chou, 1994; Dawson & Archer, 1993; Grant & Harford, 1989, 1990, Gruenewald, 1991; Harford, Grant, & Hasin, 1991), an understanding of how consumption varies within the population is necessary to estimate the indirect effects (via consumption level) of sociodemographic characteristics on outcomes such as physical morbidity and alcohol abuse and dependence. Differentials in consumption also are useful in targeting prevention and treatment efforts toward those individuals at highest risk or most in need of services. Finally, comparison of subgroup differences over time may help to illustrate the dynamics of how drinking patterns change, by distinguishing those subpopulations in which the changes first occur or in which countertrends are apparent.

With these ends in mind, differentials in consumption have been studied and compared using data from a variety of population-based samples. Trend studies based on the national alcohol studies conducted in 1964, 1979, 1984, and 1990 (Hilton, 1991b; Midanik & Clark, 1994) and on the 1983 and 1988 National Health Interview Surveys (Williams & DeBakey, 1992) revealed that although differentials may have increased or decreased over time, certain patterns were observed during all of those periods. These include greater rates of abstention among women, non-Whites, and the elderly; rates of current drinking that increase directly with income and education; and higher rates of heavy drinking among men. The factors associated with heavy drinking vary according to whether its prevalence is estimated within the general population or only among current drinkers, suggesting that disaggregating the probability of heavy drinking into the base probability of being a drinker at all and the conditional probability of going on to become a

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heavy drinker would result in different risk factors for the two sets of probabilities.

This article has three purposes. First, it presents the most recent sociodemographic differentials in the distribution of drinking status and in the prevalence of various commonly used indicators of heavy drinking. Second, it contrasts the risk factors for drinking per se and for heavy drinking, by presenting the prevalence of heavy drinking among current drinkers as well as within the general population. Third, it tests for the independent effects of each of the sociodemographic characteristics on the drinking outcomes by means of multiple logistic regression models that adjust for their intercorrelation.

METHODS

Data sources

The data described in this article were collected in the 1992 National Longitudinal Alcohol Epidemiologic Study (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism with field work conducted by the U.S. Bureau of the Census. The NLAES sample consisted of 42,862 U.S. adults 18 years of age and over, selected at random from a nationally representative sample of households. The household and sample person response rates for the NLAES were 92% and 97%, respectively. The complex, multistage sample design of the NLAES featured oversampling of both the Black population and young adults between the ages of 18 and 29 (Grant, Peterson, Dawson, & Chou, 1994). In order to account for the impact of the sample design on variance estimation, all of the standard errors and confidence intervals presented in this article were generated using SUDAAN (Research Triangle Institute, 1992), a software package that accommodates complex sample designs.

The NLAES interview was conducted in respondents' homes by experienced Census Bureau interviewers, lasted approximately 1 hour, and collected information concerning alcohol consumption and problems, drug use and problems, periods of low mood, utilization of alcohol and drug treatment, alcohol-related physical morbidity, family history of alcoholism, and sociodemographic background characteristics. Proxies were not permitted. Data were not verified by means of collaterals or physical testing, but a test-retest study of the NLAES consumption measures in a general population sample (Grant, Harford, Dawson, Chou, & Pickering, 1995) indicated that reliabilities generally exceeded .70 and often exceeded .90. A validation study conducted in a clinical sample is now being evaluated.

Consumption measures

For this analysis, current drinkers were defined as persons who had consumed at least 12 alcoholic drinks in the year preceding the interview. Former drinkers were defined as persons who had consumed at least 12 drinks in a 12-month period at some point in their lives, but not during the year immediately preceding the interview. Lifetime abstainers were defined as persons who had never consumed at least 12 drinks in a 1-year period. These definitions were used in 1983 and 1988 national alcohol surveys conducted in conjunction with the National Health Interview Survey because they ease the burden on interviewers and respondents by skipping very light drinkers past inappropriate and annoying questions on detailed aspects of alcohol consumption and alcohol-related problems. The NLAES sample contained 18,352 current drinkers, on whom many of the estimates in this article are based, as well as 9,264 former drinkers and 15,246 lifetime abstainers.

Frequencies of drinking 5+ drinks and of intoxication were based on the following questions:

- During the last 12 months, about how often did you have five or more drinks of any type of alcohol in a single day?

- About how often would you say you USUALLY drank enough to feel drunk during the last 12 months? By drunk, I mean times when your speech was slurred, you felt unsteady on your feet, or you had blurred vision.

Average daily ethanol intake, which forms the basis for one of the measures of heavy drinking, was based on a series of questions. For the 1-year period immediately preceding the interview, the NLAES obtained separate measures of alcohol intake for beer, wine, and liquor. Within each of these beverage types, the patterns of usual and heaviest consumption during the past year were distinguished. The measures obtained for each type of beverage included frequency of drinking (converted to number of drinking days per year), typical number of drinks consumed per drinking day, and typical size of drink (ounces of beer, wine, or liquor). Ounces of beverage were converted to ounces of ethanol using the following conversion factors: .045 for beer, .121 for wine, and .409 for liquor (DISCUS,

1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams, Clem, & Dufour, 1993).

The annual volume of ethanol for each beverage type was calculated as follows: oz. $ethanol_{beverage}$ = (total drinking days – heavy drinking days per $year_{beverage}$ x number of drinks per usual drinking $day_{beverage}$ x oz. of beverage in typical drink consumed on usual drinking $day_{beverage}$ x ethanol conversion $factor_{beverage}$) + (heavy drinking days per $year_{beverage}$ x number of drinks per heavy drinking $day_{beverage}$ x oz. of beverage in typical drink consumed on heavy drinking $day_{beverage}$ x ethanol conversion $factor_{beverage}$). These volumes were then summed over the three beverage types, and the resulting volume was divided by 365. Missing data on alcohol consumption were imputed using modal values for specific items. Data were not imputed for individuals who had missing values for more than three of the nine components of total ethanol intake (frequency, quantity, and drink size for each of the three types of beverage). This level of nonresponse was observed in 2% of all current drinkers and was highest (4%) in the Black subpopulation.

RESULTS

The NLAES estimated that 44% of adults 18 years of age and older were current drinkers who had consumed at least 12 drinks in the preceding year (Table 1). Twenty-two percent were former drinkers, and 34% were lifetime abstainers. The distribution of adults by drinking status showed considerable variation among population subgroups. With advancing age, the proportion of current drinkers declined and the proportion of former drinkers increased. After age 30, the proportion of lifetime abstainers increased directly with age. Based on their self-reports, men were more likely than women to be current drinkers, whereas women were more likely to be lifetime abstainers. Below age 45, there were proportionately more female than male former drinkers; at later ages, the opposite was true. The proportions of current drinkers were higher for non-Blacks than Blacks and for non-Hispanics than Hispanics, were lowest among the widowed and highest among the never married, increased with education and income, were lower in the South than in other regions, and were higher in urban than rural areas. The patterns with respect to lifetime abstention were just the opposite of these, except that persons living in central cities were about as likely to be abstainers as those living in rural areas. The percentages of former drinkers did not vary strongly or consistently according to most of the sociodemographic characteristics. Exceptions were ethnic origin and marital status, with former drinkers underrepresented among Hispanics and the never married. The proportion of former drinkers increased steadily with age among men, but not among women.

Because of the different definitions used in the NLAES and the series of national alcohol surveys conducted in 1964, 1979, 1984, and 1990, the distribution of drinking status cannot be compared directly; however, the patterns of the socioeconomic differentials in the NLAES data match those that have been noted in the earlier surveys (Hilton, 1991a, 1991b; Midanik & Clark, 1994; Williams & DeBakey, 1992). A more direct comparison can be made with data from the 1988 National Health Interview Alcohol Supplement, which used definitions of drinking status similar to those used in the NLAES. Data from the NHIS indicated that in 1988, 52% of adults were current drinkers, 18% were former drinkers, and 30% were lifetime abstainers (Dawson & Archer, 1992). Thus, between 1988 and 1992, the proportion of current drinkers appears to have decreased as a function of both more individuals having stopped drinking and more young adults postponing initiation of drinking. This finding is consistent with results of the 1990 national alcohol survey, which found a 5% decrease between 1984 and 1990 in the proportion of current drinkers, defined as persons who had *any* alcoholic drinks in the preceding year (Midanik & Clark, 1994). The comparison of the NLAES and the NHIS data indicated that the reduction in the prevalence of current drinking was of equal magnitude for men and women, as did the comparison of the 1984 and 1990 survey results.

Table 2 shows the percentages of U.S. adults who met various definitions of heavy drinking during the preceding year: ever having consumed 5+ drinks on a single day (24.6% of all adults), having consumed 5+ drinks at least once a week (5.5%), ever having been intoxicated (20.4%), having been intoxicated at least once a week (1.7%), and having consumed an average of more than 1 ounce ethanol (i.e., more than two drinks) per day (8.7%). For the two measures based on the frequency of having had 5+ drinks in the past year, the prevalences decreased sharply with age. Men were over twice as likely as women to report having drunk 5+ drinks at least once and more than four times as likely to report having done so on a weekly or more frequent basis. Although non-Blacks were more likely than Blacks to report having ever consumed 5+ drinks, there was no significant variation in this measure by Hispanic origin. Having consumed 5+ drinks weekly did not vary by either race or Hispanic origin.

Table 1. Percentage Distribution of Adults 18 Years of Age and Over by Drinking Status, According to Selected Sociodemographic Characteristics: United States, 1992

	Current Drinkers	Former Drinkers	Lifetime Abstainers
Both sexes			
All ages	44.4 (0.4)	21.6 (0.3)	34.0 (0.4)
Age 18–29	53.4 (0.7)	14.6 (0.4)	32.0 (0.6)
Age 30–44	50.2 (0.5)	23.1 (0.5)	26.7 (0.5)
Age 45–64	40.5 (0.7)	25.6 (0.6)	33.9 (0.7)
Age 65+	24.5 (0.6)	23.3 (0.6)	52.2 (0.8)
Male			
All ages	55.8 (0.5)	22.5 (0.4)	21.7 (0.4)
Age 18–29	64.2 (0.9)	11.5 (0.5)	24.3 (0.8)
Age 30–44	60.7 (0.8)	22.1 (0.6)	17.2 (0.6)
Age 45–64	51.0 (1.0)	29.3 (0.8)	19.7 (0.8)
Age 65+	36.4 (1.1)	31.8 (1.1)	31.8 (1.1)
Female			
All ages	33.9 (0.5)	20.8 (0.4)	45.3 (0.6)
Age 18–29	42.6 (0.9)	17.7 (0.6)	39.7 (0.9)
Age 30–44	39.8 (0.7)	24.1 (0.6)	36.1 (0.7)
Age 45–64	30.7 (0.8)	22.0 (0.7)	47.2 (0.9)
Age 65+	16.1 (0.6)	17.3 (0.6)	66.6 (0.9)
Race			
Black	32.5 (0.8)	18.8 (0.6)	48.7 (0.9)
Nonblack	45.9 (0.8)	22.0 (0.3)	32.1 (0.5)
Ethnicity			
Hispanic	38.4 (1.2)	14.9 (0.8)	46.7 (1.2)
Non-Hispanic	44.9 (0.4)	22.3 (0.3)	32.8 (0.4)
Marital status			
Currently married	44.0 (0.5)	24.3 (0.4)	31.6 (0.5)
Divorced or separated	47.2 (0.8)	23.5 (0.7)	29.3 (0.8)
Widowed	19.6 (0.7)	19.4 (0.7)	61.0 (0.9)
Never married	53.0 (0.6)	12.7 (0.4)	34.3 (0.6)
Education			
Less than 12 years	27.9 (0.7)	22.6 (0.6)	49.5 (0.7)
12 years	40.7 (0.6)	23.1 (0.5)	36.2 (0.6)
13–15 years	50.4 (0.7)	21.4 (0.5)	28.2 (0.5)
16+ years	57.3 (0.6)	19.1 (0.5)	23.6 (0.5)
Income			
Less than $11,400	33.6 (0.9)	19.6 (0.5)	46.8 (0.8)
$11,400–21,599	36.3 (0.7)	22.8 (0.5)	40.9 (0.7)
$21,600–44,999	44.9 (0.6)	23.9 (0.5)	31.2 (0.6)
$45,000 or more	56.9 (0.6)	19.5 (0.4)	23.6 (0.5)
Region			
Northeast	47.0 (0.7)	19.4 (0.5)	33.6 (0.8)
Midwest	47.9 (1.0)	23.7 (0.8)	28.4 (1.0)
South	38.6 (0.7)	21.1 (0.5)	40.3 (0.6)
West	47.4 (0.8)	22.3 (0.5)	30.3 (0.9)
Urbanicity			
Urban, central city	44.5 (0.6)	20.0 (0.4)	35.5 (0.6)
Other urban	47.2 (0.6)	21.8 (0.4)	31.0 (0.5)
Rural	39.6 (0.7)	23.1 (0.6)	37.3 (0.9)

Note. Figures in parentheses are standard errors of percentages.

Table 2. Percentage of Adults 18 Years of Age and Over with Various Indicators of Heavy Drinking During Past Year, According to Selected Sociodemographic Characteristics: United States, 1992

	Ever Drank 5+ Drinks	Drank 5+ Drinks Weekly	Ever Intoxicated	Intoxicated Weekly	Average Daily Ethanol Intake of 1+ Ounces
Both sexes					
All ages	24.6 (0.3)	5.5 (0.2)	20.4 (0.3)	1.7 (0.1)	8.7 (0.2)
Age 18–29	41.3 (0.7)	9.7 (0.4)	39.1 (0.7)	4.0 (0.3)	11.2 (0.4)
Age 30–44	28.1 (0.5)	5.4 (0.2)	23.2 (0.4)	1.3 (0.1)	8.4 (0.3)
Age 45–64	15.8 (0.5)	3.9 (0.2)	9.7 (0.4)	0.8 (0.1)	8.9 (0.3)
Age 65+	5.1 (0.3)	1.4 (0.1)	2.5 (0.2)	0.3 (0.1)	5.2 (0.3)
Male					
All ages	35.6 (0.5)	9.1 (0.3)	27.6 (0.5)	2.7 (0.1)	13.7 (0.3)
Age 18–29	54.0 (1.0)	15.3 (0.7)	48.9 (0.9)	6.1 (0.4)	17.1 (0.7)
Age 30–44	40.3 (0.7)	8.7 (0.4)	30.7 (0.7)	2.0 (0.2)	13.1 (0.5)
Age 45–64	24.7 (0.8)	6.8 (0.5)	14.2 (0.6)	1.4 (0.2)	13.5 (0.6)
Age 65+	9.8 (0.6)	2.6 (0.3)	4.7 (0.4)	0.6 (0.2)	9.1 (0.6)
Female					
All ages	14.4 (0.3)	2.1 (0.1)	13.8 (0.3)	0.8 (0.1)	4.1 (0.2)
Age 18–29	28.8 (0.8)	4.1 (0.3)	29.5 (0.8)	1.9 (0.3)	5.3 (0.4)
Age 30–44	16.1 (0.5)	2.2 (0.2)	15.9 (0.5)	0.7 (0.1)	3.8 (0.2)
Age 45–64	7.6 (0.4)	1.2 (0.2)	5.5 (0.4)	0.3 (0.1)	4.5 (0.3)
Age 65+	1.8 (0.2)	0.5 (0.1)	0.9 (0.1)	0.1 (<0.1)	2.4 (0.2)
Race					
Black	17.2 (0.7)	5.5 (0.4)	13.1 (0.7)	1.8 (0.2)	7.7 (0.5)
Nonblack	25.5 (0.4)	5.4 (0.2)	21.3 (0.4)	1.7 (0.1)	8.8 (0.2)
Ethnicity					
Hispanic	23.9 (1.0)	5.5 (0.6)	18.2 (0.9)	1.6 (0.3)	7.4 (0.6)
Non-Hispanic	24.6 (0.4)	5.4 (0.2)	20.6 (0.3)	1.7 (0.1)	8.8 (0.2)
Marital status					
Currently married	21.6 (0.4)	3.9 (0.2)	17.0 (0.3)	1.0 (0.1)	7.7 (0.2)
Divorced or separated	27.1 (0.7)	7.9 (0.5)	22.5 (0.7)	2.6 (0.3)	11.3 (0.5)
Widowed	5.2 (0.4)	1.4 (0.2)	2.8 (0.3)	0.1 (0.1)	3.2 (0.3)
Never married	39.8 (0.9)	10.4 (0.5)	36.5 (0.8)	4.0 (0.3)	12.4 (0.5)
Education					
Less than 12 years	17.7 (0.6)	6.4 (0.4)	12.6 (0.5)	2.1 (0.2)	7.6 (0.4)
12 years	24.5 (0.5)	6.1 (0.3)	19.4 (0.4)	1.9 (0.1)	8.7 (0.3)
13–15 years	29.5 (0.7)	5.9 (0.3)	26.2 (0.7)	2.0 (0.2)	9.4 (0.4)
16+ years	25.3 (0.6)	3.3 (0.2)	22.1 (0.6)	0.8 (0.1)	8.9 (0.4)
Income					
Less than $11,400	22.6 (0.9)	7.2 (0.5)	19.3 (0.9)	3.1 (0.3)	9.1 (0.5)
$11,400–21,599	21.4 (0.6)	5.3 (0.3)	17.6 (0.5)	1.7 (0.2)	7.9 (0.3)
$21,600–44,999	25.2 (0.5)	5.3 (0.2)	21.0 (0.4)	1.5 (0.1)	8.2 (0.3)
$45,000 or more	27.5 (0.5)	4.6 (0.3)	22.6 (0.5)	1.1 (0.1)	9.6 (0.3)
Region					
Northeast	24.1 (0.7)	5.1 (0.3)	18.8 (0.6)	1.4 (0.2)	8.6 (0.4)
Midwest	28.2 (0.8)	6.5 (0.3)	23.4 (0.8)	2.1 (0.2)	8.7 (0.3)
South	21.7 (0.6)	5.0 (0.3)	17.9 (0.5)	1.7 (0.2)	8.1 (0.4)
West	25.5 (0.7)	5.3 (0.3)	22.7 (0.6)	1.6 (0.2)	9.8 (0.4)
Urbanicity					
Urban, central city	25.8 (0.5)	6.3 (0.3)	21.8 (0.5)	2.0 (0.2)	9.6 (0.3)
Other urban	24.5 (0.5)	5.0 (0.2)	20.6 (0.4)	1.5 (0.1)	8.5 (0.3)
Rural	23.1 (0.6)	5.3 (0.3)	18.5 (0.5)	1.6 (0.2)	7.9 (0.3)

Note. Figures in parentheses are standard errors of percentages.

Table 3. Percentage of Current Drinkers With Various Indicators of Heavy Drinking During Past Year, According to Selected Sociodemographic Characteristics: United States, 1992

	Ever Drank 5+ Drinks	Drank 5+ Drinks Weekly	Ever Intoxicated	Intoxicated Weekly	Average Daily Ethanol Intake of 1+ Ounces
Both sexes					
All ages	55.6 (0.6)	12.3 (0.3)	46.1 (0.5)	3.8 (0.2)	19.8 (0.4)
Age 18–29	77.6 (0.7)	18.2 (0.7)	73.4 (0.8)	7.5 (0.5)	21.1 (0.7)
Age 30–44	56.2 (0.7)	10.8 (0.5)	46.4 (0.7)	2.7 (0.3)	16.9 (0.5)
Age 45–64	39.3 (0.9)	9.8 (0.6)	24.1 (0.8)	2.0 (0.2)	22.1 (0.8)
Age 65+	21.0 (1.1)	5.7 (0.6)	10.2 (0.8)	1.2 (0.3)	21.4 (1.0)
Male					
All ages	64.1 (0.6)	16.4 (0.5)	49.6 (0.7)	4.9 (0.3)	24.8 (0.5)
Age 18–29	84.2 (0.7)	23.9 (1.0)	76.2 (0.9)	9.6 (0.7)	26.8 (1.1)
Age 30–44	66.6 (0.9)	14.4 (0.7)	50.6 (0.9)	3.3 (0.4)	21.8 (0.8)
Age 45–64	48.6 (1.3)	13.5 (0.9)	27.9 (1.1)	2.7 (0.4)	26.8 (1.1)
Age 65+	26.9 (1.6)	7.3 (0.8)	13.0 (1.2)	1.6 (0.4)	25.1 (1.5)
Female					
All ages	42.7 (0.7)	6.2 (0.3)	40.9 (0.7)	2.3 (0.2)	12.2 (0.4)
Age 18–29	67.6 (1.1)	9.7 (0.8)	69.2 (1.1)	4.5 (0.6)	12.5 (0.8)
Age 30–44	40.6 (1.0)	5.4 (0.5)	40.1 (1.0)	1.7 (0.3)	9.7 (0.6)
Age 45–64	25.0 (1.1)	4.0 (0.5)	18.0 (1.1)	0.9 (0.2)	14.9 (1.0)
Age 65+	11.5 (1.3)	3.3 (0.7)	5.6 (0.8)	0.5 (0.3)	15.3 (1.3)
Race					
Black	53.2 (1.5)	17.1 (1.2)	40.6 (1.6)	5.6 (0.7)	24.5 (1.3)
Nonblack	55.8 (0.5)	11.9 (0.3)	46.6 (0.6)	3.7 (0.2)	19.4 (0.4)
Ethnicity					
Hispanic	62.5 (0.5)	14.4 (1.4)	47.3 (1.9)	4.1 (0.8)	19.7 (1.5)
Non-Hispanic	55.0 (0.5)	12.2 (0.3)	46.0 (0.6)	3.8 (0.2)	19.8 (0.4)
Marital status					
Currently married	49.3 (0.6)	9.0 (0.3)	38.8 (0.6)	2.3 (0.2)	17.7 (0.4)
Divorced or separated	57.6 (1.1)	16.9 (1.0)	47.8 (1.2)	5.5 (0.6)	24.4 (1.1)
Widowed	26.6 (1.8)	7.4 (0.9)	14.2 (1.3)	0.7 (0.3)	16.8 (1.3)
Never married	75.3 (0.8)	19.7 (0.8)	69.1 (0.9)	7.6 (0.6)	23.7 (0.8)
Education					
Less than 12 years	63.9 (1.2)	23.0 (1.2)	45.6 (1.3)	7.6 (0.7)	27.7 (1.2)
12 years	60.6 (0.8)	15.2 (0.6)	48.0 (0.8)	4.6 (0.3)	21.6 (0.7)
13–15 years	58.6 (1.0)	11.7 (0.6)	52.0 (1.1)	4.0 (0.4)	18.9 (0.6)
16+ years	44.2 (0.8)	5.7 (0.4)	38.7 (0.8)	1.3 (0.2)	15.7 (0.6)
Income					
Less than $11,400	67.6 (1.3)	21.4 (1.1)	57.9 (1.5)	9.2 (0.8)	27.5 (1.1)
$11,400–21,599	59.3 (1.0)	14.8 (0.7)	48.7 (1.0)	4.7 (0.4)	22.1 (0.8)
$21,600–44,999	56.4 (0.8)	11.9 (0.5)	46.8 (0.8)	3.3 (0.3)	18.5 (0.6)
$45,000 or more	48.5 (0.7)	8.0 (0.4)	39.8 (0.7)	1.9 (0.2)	16.9 (0.6)
Region					
Northeast	51.6 (1.1)	10.9 (0.7)	40.1 (1.0)	3.0 (0.4)	18.5 (0.7)
Midwest	59.1 (1.0)	13.7 (0.6)	49.1 (1.2)	4.3 (0.4)	18.2 (0.7)
South	56.6 (1.0)	13.1 (0.6)	46.5 (1.2)	4.3 (0.4)	21.2 (0.8)
West	54.1 (0.9)	11.2 (0.6)	48.2 (1.0)	3.4 (0.4)	21.0 (0.7)
Urbanicity					
Urban, central city	58.3 (0.8)	14.2 (0.6)	49.1 (0.9)	4.6 (0.4)	21.9 (0.6)
Other urban	52.2 (0.8)	10.6 (0.4)	43.8 (0.8)	3.3 (0.3)	18.3 (0.5)
Rural	58.7 (1.0)	13.4 (0.7)	46.9 (1.0)	4.0 (0.4)	20.0 (0.7)

Note. Figures in parentheses are standard errors of percentages.

Regardless of frequency, self-reported consumption of 5+ drinks was highest for the never married and lowest for widowed individuals. The proportion of individuals who reported having ever drunk 5+ drinks increased up through the level of 13 to 15 years of school, then declined among college graduates, whereas it increased steadily according to income. In contrast, the proportions of persons who reportedly drank 5+ drinks at least weekly decreased with both education and income. With respect to geographic indicators, the prevalence of binge drinking was most common in the Midwest.

For the two measures based on frequency of intoxication, the sociodemographic differentials in prevalence mirrored those for the measures based on drinking 5+ drinks, with two exceptions. The gender differential was narrower with respect to intoxication, reflecting women's greater propensity to become intoxicated at equivalent levels of ethanol intake as a result of their lower total body water (Goist & Sutker, 1985), and the educational differential with respect to ever having been intoxicated was stronger than that for ever having consumed 5+ drinks.

The fifth measure of heavy drinking, average daily intake of more than 1 ounce of ethanol, showed patterns of prevalence that were somewhat different from the other four measures. This is not surprising, because it is reflective of overall frequency of drinking and typical level of consumption as well as of frequency of binge drinking. The main ways in which the sociodemographic patterns for this indicator differed from the others were a smaller decline in prevalence with age, a prevalence among the divorced or separated that was nearly as high as that for the never married, a U-shaped pattern with regard to income, and the highest prevalence in the West rather than the Midwest.

As with the distribution of drinking status, the sociodemographic differentials in these indicators of heavy drinking were similar to those that have been reported for earlier studies (Hilton, 1991a, 1991b; Midanik & Clark, 1994; Williams & DeBakey, 1992), but compar-

Table 4. Adjusted Odds Ratios and 95% Confidence Intervals Reflecting Associations Between Selected Sociodemographic Characteristics and Assorted Drinking Outcomes: United States, 1992.

	Current Drinker[a]	Ever Drank 5+ Drinks[b]	Drank 5+ Drinks Weekly[b]	Ever Intoxicated[b]	Intoxicated Weekly[b]	Average Daily Ethanol Intake of 1+ Ounces[b]
Age group						
Age 30–44	0.78 (0.74–0.83)	0.45 (0.41–0.50)	0.77 (0.66–0.89)	0.38 (0.34–0.41)	0.50 (0.39–0.65)	n.s. (0.83–1.08)
Age 45–64	0.56 (0.51–0.61)	0.21 (0.18–0.24)	0.68 (0.57–0.82)	0.14 (0.12–0.16)	0.36 (0.26–0.51)	1.38 (1.18–1.60)
Age 65+	0.35 (0.32–0.39)	0.07 (0.06–0.08)	0.30 (0.23–0.38)	0.05 (0.04–0.06)	0.20 (0.14–0.28)	1.21 (1.02–1.45)
Gender						
Male	2.39 (2.27–2.53)	3.00 (2.77–3.25)	3.15 (2.77–3.58)	1.64 (1.51–1.78)	2.36 (1.86–2.98)	2.38 (2.15–2.63)
Race						
Black	0.58 (0.53–0.63)	0.54 (0.46–0.63)	n.s. (0.77–1.15)	0.51 (0.44–0.60)	n.s. (0.70–1.15)	n.s. (0.88–1.23)
Ethnicity						
Hispanic	0.73 (0.65–0.82)	n.s. (0.74–1.08)	n.s. (0.64–1.03)	0.71 (0.58–0.85)	n.s. (0.45–1.09)	0.76 (0.62–0.93)
Marital status						
Widowed	0.88 (0.79–0.98)	n.s. (0.88–1.36)	n.s. (0.88–1.73)	n.s. (0.72–1.15)	0.43 (0.21–0.91)	n.s. (0.68–1.07)
Divorced or separated	1.57 (1.45–1.70)	1.43 (1.27–1.59)	1.90 (1.61–2.24)	1.45 (1.30–1.63)	2.14 (1.60–2.88)	1.46 (1.27–1.66)
Never married	1.17 (1.09–1.27)	1.58 (1.41–1.76)	1.80 (1.56–2.09)	1.52 (1.36–1.70)	1.85 (1.41–2.43)	1.43 (1.26–1.62)
Education						
12 years	1.37 (1.26–1.48)	0.79 (0.68–0.92)	0.64 (0.54–0.76)	n.s. (0.85–1.14)	0.65 (0.49–0.87)	0.84 (0.72–0.98)
13–15 years	1.74 (1.59–1.90)	0.62 (0.53–0.73)	0.43 (0.36–0.52)	n.s. (0.84–1.14)	0.46 (0.34–0.61)	0.70 (0.60–0.82)
16+ years	2.05 (1.87–2.25)	0.38 (0.32–0.44)	0.23 (0.19–0.28)	0.69 (0.60–0.80)	0.22 (0.15–0.31)	0.57 (0.49–0.67)
Income						
$11,400–21,599	n.s. (0.99–1.17)	0.82 (0.71–0.95)	0.75 (0.63–0.89)	0.85 (0.74–0.97)	0.60 (0.45–0.79)	0.77 (0.67–0.89)
$21,600–44,999	1.27 (1.18–1.37)	0.78 (0.68–0.89)	0.68 (0.57–0.80)	0.82 (0.72–0.93)	0.51 (0.39–0.66)	0.66 (0.57–0.75)
$45,000 or more	1.76 (1.62–1.92)	0.69 (0.59–0.79)	0.58 (0.48–0.71)	0.72 (0.62–0.82)	0.40 (0.28–0.56)	0.65 (0.56–0.76)
Region						
Midwest	n.s. (0.97–1.18)	1.32 (1.17–1.50)	n.s. (1.00–1.43)	1.43 (1.27–1.62)	n.s. (0.94–1.78)	n.s. (0.83–1.08)
South	0.76 (0.71–0.83)	n.s. (0.98–1.26)	n.s. (0.87–1.26)	1.23 (1.09–1.40)	n.s. (0.86–1.64)	n.s. (0.97–1.28)
West	n.s. (0.85–1.02)	n.s. (0.95–1.21)	n.s. (0.81–1.18)	1.43 (1.27–1.62)	n.s. (0.74–1.48)	1.17 (1.02–1.33)
Urbanicity						
Other urban	n.s. (0.94–1.07)	0.84 (0.76–0.92)	0.84 (0.73–0.96)	0.87 (0.79–0.96)	n.s. (0.68–1.08)	0.86 (0.78–0.96)
Rural	0.81 (0.75–0.87)	n.s. (0.91–1.14)	n.s. (0.78–1.08)	n.s. (0.86–1.08)	n.s. (0.68–1.18)	0.90 (0.81–0.99)

Note. Figures in parentheses are 95% confidence intervals for odds ratios. Reference categories for odds ratios are as follows: Age group = age 18–29; gender = female; race = non-Black; ethnicity = non-Hispanic; marital status = married; education = 0–11 years; income = < $11,400; region = Northeast; urbanicity = central city.

[a]Odds ratios among adults 18 years of age and over. [b]Odds ratios among current drinkers.

isons over time in the prevalence of the indicators were confounded by measurement differences. For example, the NLAES estimate of 8.6% of the adult population that consumed an average of 1 or more ounces of ethanol per day is slightly higher than the estimate based on the 1988 NHIS—15.2% of current drinkers (Dawson & Archer, 1992), corresponding to 7.8% of the total population. This reflects the fact that the NLAES calculation of average intake incorporated both usual and atypical heavy drinking patterns, whereas the NHIS calculation was based solely on usual consumption.

To some extent, the patterns in Table 2 were affected by the distribution of current drinkers in the population, because only current drinkers were at risk of heavy drinking during the preceding year. Some interesting differences in patterns emerged when the prevalence of these indicators of heavy drinking was estimated among current drinkers rather than for the total population (Table 3). The age and gender differentials were reduced, the prevalences of weekly intoxication and consumption of 5+ drinks were shown to be higher for Black than non-Black drinkers, and all of the heavy drinking indicators decreased directly in prevalence with education and income. The prevalence of drinking an average of more than 1 ounce of ethanol per day fell to its lowest level at ages 30 to 44 and was essentially invariant at other ages.

In examining Tables 1 through 3, one might question whether some of the apparent differentials are based on spurious correlations that exist among the sociodemographic variables. Multiple logistic regression models were estimated for each drinking outcome in order to test whether these associations held true after adjusting for the effects of the other sociodemographic variables. Table 4 shows the odds ratios generated by these models. After adjustment for other model covariates, widowhood was not associated with any of the heavy drinking outcomes except for weekly intoxication, and its association with the odds of being a current drinker was of marginal significance. The apparent excess prevalence among Blacks of weekly consumption of 5+ drinks and weekly intoxication was not statistically significant, although their decreased odds of ever having drunk 5+ drinks or having been intoxicated was maintained. The odds of ever having been intoxicated were lower for college graduates than all others, but did not vary among those with less education. Geographic region and urbanicity had inconsistent and often insignificant relationships with most of the drinking variables. Otherwise, most of the adjusted sociodemographic differentials were similar in direction to those already noted in the earlier tables, except that the adjustment process increased the magnitude of the gender differential relative to those for the other sociodemographic variables.

DISCUSSION

Some of the correlates of self-reported alcohol consumption behaved in a consistent manner no matter what drinking outcome was considered. For example, both the probability of being a current drinker and all of the indicators of heavy drinking reportedly were higher for men than women and higher for never married, separated, and divorced individuals than for persons who were married. Other factors, such as education, income, and age, differed in their effect on the various outcomes. The probability of being a current drinker was positively associated with increasing education and income, whereas the probability of drinking heavily—conditional on already being a current drinker—was inversely related to both education and income. Although age was negatively associated with both the probability of being a current drinker and with the conditional probabilities of the binge drinking indicators, the heavy-drinking indicator based on average daily ethanol intake was more prevalent among persons aged 45 and older than among younger drinkers. These differences suggest that the norms governing the acceptability of various drinking styles vary among population subgroups, as argued by others (e.g., Johnson, Armor, Polich, & Stambul, 1977; Knupfer, 1984; Makela, 1978).

The issue of conditional versus unconditional probabilities takes on importance with respect to the targeting of prevention and treatment measures. Prevention campaigns aimed at reaching the largest numbers of heavy drinkers within the general population (e.g., mass media messages and advertising campaigns) should consider the unconditional prevalences of heavy drinking indicators coupled with the underlying population sizes of the various subgroups to estimate the number of heavy drinkers in various target audiences. Alternatively, moderate drinking messages posted in liquor stores would do better to consider the conditional probabilities of heavy drinking, because those messages will be seen by subgroups of current drinkers rather than of the general population. Likewise, assessment of treatment modalities would be more affected by the conditional than the unconditional probabilities, because only current drinkers are at risk of requiring treatment. Of particular interest in this regard are the differences in conditional prevalences among the various indicators of heavy drinking. They suggest that

treatment for problems associated with binge drinking should be aimed at a different subgroup of drinkers than treatment for problems associated with daily consumption of three or four drinks.

This study presented estimates of alcohol consumption based on retrospective self-reported data collected in a cross-sectional survey of a sample of U.S. households. Certain limitations are inherent in data such as these. For example, the data were subject to recall bias, age and cohort effects could not be distinguished, and persons living outside households (e.g., migrant, homeless, and institutionalized populations) were not represented. Moreover, the analysis was restricted to evaluation of individual associations between a single sociodemographic characteristic and a single drinking outcome. Much could be learned by examining interactions among these associations. For example, studies by Herd (1990) and Corbett, Mora, and Ames (1991) indicate that differentials by gender, income, and education may not be the same within minority populations as within the general population. Data from a sample of adults residing in New York state revealed that gender differences within the general population were almost nil at the upper levels of family income (Barnes, Welte, & Dintcheff, 1991). Thus, studying effect interaction within the context of an analysis with a specific exposure variable is strongly recommended to enhance the incomplete understanding that can be derived from analysis of bivariate relationships or main effects models.

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Beyond Black, White and Hispanic: Race, Ethnic Origin and Drinking Patterns in the United States

Deborah A. Dawson

This study used data on 42,862 U.S. adults, including 18,352 past-year drinkers, to describe differentials by race and national origin in U.S. drinking patterns. Age–sex standardized estimates were presented within 21 categories of ethnic origin for whites and within five categories each for individuals of black and other races. Of the three racial groups, whites were the most likely to drink, but blacks had the highest volume of intake and frequency of heavy drinking. Differences by ethnic origin within racial categories were as marked as differentials between races. Compared to whites of European origin, those of Hispanic and Native American origin were less likely to drink but consumed more alcohol on days when they drank. Whites of Southern and Eastern European origin drank proportionately more wine and demonstrated more moderate drinking patterns (lower intake per drinking day and/or less frequent heavy drinking,) than those of Northern or Central European origin. Hispanics of Caribbean origin were less prone to heavy drinking than other white Hispanics; similarly, blacks from the English-speaking Caribbean showed more moderate drinking patterns than other blacks. Individuals of Asian origin, in particular those of non-Japanese origin, had the most moderate drinking patterns within the category of other race. Although the black/white differentials in volume of intake and frequency of heavy drinking disappeared after adjusting for marital status, education and income, most of the differences by ethnic origin retained their statistical significance if not their original magnitudes. These findings indicate that cultural forces exert a strong effect on drinking behavior. Differences among European whites with respect to prevalence of drinking, beverage preference and frequency of heavy drinking suggest that the association between ethnic origin and drinking behavior may persist even after many generations of presumed acculturation.

The use of the categories black, white and Hispanic has become increasingly common, if not standard, in describing ethnic differentials in large-scale national studies of U.S. drinking patterns. Incorporating information on race as well as national origin, these categories describe the three largest ethnic groups in the United States, and black and Hispanic often are the only nonwhite ethnic categories for which survey sample sizes permit reliable estimates. In addition, recent surveys have found significant differences in drinking behavior among these groups (see, for example, Midanik and Clark; 1994; Caetano and Kaskutas, 1995; Dawson, in press; Dawson et al., 1995; Johnson et al., 1998; Substance Abuse Mental Health Services Administration, 1998). Data consistently indicate that whites are more likely to drink than blacks. Some studies indicate that whites also are more likely than Hispanics to be drinkers, especially among women. This differential is most evident when the definition of a drinker and/or the reference period used result in infrequent drinkers being excluded from the comparison. In addition, some studies have found higher rates of heavy drinking and drinks consumed per occasion among those blacks and Hispanics who do drink, at least among those who drink fairly regularly. Ignoring the implications of these differences when planning programs for the prevention and treatment of alcohol problems could have serious public health ramifications because of the size of the black and Hispanic minority populations.

Despite the public health benefits of collecting and disseminating information for the country's three largest ethnic groups, the focus on blacks, whites and Hispanics has been criticized on several counts. First, it places an apparent emphasis on race and origin as determining factors in and of themselves, ignoring the effects that differential socioeconomic status, gender roles, educational levels and so forth may have on ethnic variation in alcohol use and problems (Heath, 1990–1991; Blanc, 1993; Collins, 1996). Second, it ignores

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other minority populations such as the rapidly growing category of Asian Americans, either omitting them altogether or lumping them into an "other" category. (Although the U.S. government now splits "other" races into three categories, American Indian/Alaska native, Asian, and native Hawaiian/ Pacific islander, there are rarely enough cases to analyze these groups separately.) Third, it ignores heterogeneity *within* these categories, the 'other' category included.

Heterogeneity within ethnic groups has been demonstrated in particular among Hispanics (Gordon, 1985, 1989; Caetano, 1988), with research findings generally indicating the highest levels of heavy drinking and alcohol problems among Mexican Americans and the lowest levels among Cuban Americans, with Hispanics of other Caribbean origins, e.g., Puerto Rican and Dominican, lying between these extremes. Other studies have indicated differences in drinking patterns among individuals in the residual 'other' ethnic group, such as a higher rate of heavy drinking among those of Japanese descent than among those of Chinese, Filipino or Korean descent (Klatsky et al., 1983; Chi et al., 1989) and a higher prevalence of alcohol consumption and alcohol abuse among native Hawaiians than among Hawaiians of Filipino or Chinese origin (Ahern, 1985; Murakami, 1985). While some of these subgroup comparisons have been based on large numbers of cases (e.g., the Kaiser-Permanente patients studied by Klatsky et al., 1983), the samples from which they were drawn are not nationally representative and thus may not be generalizable to the United States as a whole.

The early national alcohol surveys detailed many differences in drinking prevalence and pattern among whites of European origin. The first of these surveys (Cahalan et al., 1969) found that the prevalence of drinking was highest among individuals whose national identity was Italian, followed by Russian, Polish or Baltic. Those whose national identity was English, Scotch or Scotch Irish were the least likely to be drinkers. Among drinkers, heavy drinking was most common among persons of Irish, Italian, Russian, Polish and Baltic identity. Using data on different aspects of heavy drinking collected in the third national survey, Cahalan and Room (1974) noted that the ratio of very heavy drinking (12+ drinks at least once a month) to steady fairly heavy drinking (5+ drinks at least once a week) was lowest among Germans, Italians and Eastern Europeans. Differentials such as these, which provided the basis for a rich ethnocultural literature in recent decades, e.g., the studies of Polish American, Italian American and Irish American drinking patterns by Freund (1985), Simboli (1985) and Stivers (1983), have been largely ignored in more recent analyses of ethnic differentials, and there are few recent quantitative comparisons of whites of different European origins. Equally sparse are quantitative data comparing ethnic subgroups of blacks (e.g., African Americans vs. blacks of West Indian/Caribbean descent), despite a large literature describing black drinking patterns and their sociocultural context (Gaines, 1985; Harper and Saifnoorian, 1991; Herd and Grube, 1996).

The purpose of this study is to fill some of these gaps by providing descriptive data on U.S. drinking patterns within categories defined by both race and the finest possible breakdown of ethnic origin. Using nationally representative data for U.S. adults 18 years of age and over, it describes the prevalence of past-year drinking, volume of ethanol intake, beverage preference, quantity and frequency of consumption, and frequency of heavy drinking. Data are presented within 21 categories of ethnic origin for whites and five categories each for members of black and other races and are standardized by age and sex to remove the confounding effects of these two correlates of drinking behavior. Within 12 slightly broader categories of ethnic origin, multivariate models further adjust for the effects of marital status, education and income.

METHOD

Study sample

This analysis is based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and which gathered information on alcohol consumption and alcohol problems from a nationally representative household sample of 42,862 U.S. adults 18 years of age and over (Grant et al., 1994). The household and sample person response rates were 92% and 97%, respectively. Estimates of the prevalence of past-year drinking are based on the full sample; estimates of volume and pattern of alcohol consumption are based on the 18,352 individuals identified as past-year drinkers, i.e., who consumed at least 12 alcoholic drinks in the year preceding the interview.

Measures

The NLAES household-screening respondent was asked the race and ethnic origin of all household members. Respondents were shown cards containing pre-

coded responses from which selections were made. The response options for race ("What is the race of each person in this household?") were white; black; American Indian, Eskimo or Aleut; Asian or Pacific islander; and other. The latter three were combined into the "other" race category used in this analysis. Response options for ethnic origin ("What is the origin or descent of each person in this household?") included 58 different countries or regions, along with "other" (see Appendix A, which also indicates the total sample size and number of past-year drinkers in each category). Individuals in the "other" origin category—primarily those who cited their origin as "American" or who were unable to choose a single ethnic origin—and in categories too small to analyze and too diverse to combine were included in the racial totals but otherwise excluded from presentation.

Because ethnic origin was asked independently of race, some categories of origin were reported within multiple race groups. For example, in addition to whites of Greek origin, a fairly large number of blacks reported being of Greek descent, possibly reflecting trade routes between ancient Egypt, Rome and Greece (Bernal, 1987; Burstein, 1995). There were many respondents of Hispanic origin within each of the three race categories. Presumably, Hispanics of "other" race included both persons of indigenous descent and those who mistakenly believed that Hispanic was a racial designation and thus reported "other." Similarly, there were large numbers of persons of native American descent in both the white and "other" race categories.

Data on alcohol consumption were collected directly from the survey respondent, with no proxies allowed. Prevalence of drinking was defined as having consumed at least 12 drinks in the year preceding the interview and was ascertained through a series of screening questions. Past-year drinkers were asked the frequency of having consumed 5+ drinks, using categories whose midpoints were converted to days per year (e.g., 3–4 days per week = 3.5 x 52 = 182 days). In addition, in separate sets of questions for beer, wine and distilled spirits, respondents were asked the overall frequency of consumption, usual quantity of drinks consumed and their typical size, largest quantity of drinks consumed and their typical size, and frequency of consuming the largest quantity. From these data, the following measures were obtained:

1. The overall frequency of drinking any alcohol was estimated as the mean of: a) the sum of the beverage-specific frequencies, not allowed to exceed 365, and b) the largest individual frequency. For example, if a respondent reported drinking beer 3–4 days a week (182 days per year), wine once a month (12 days per year) and distilled spirits 1–2 days a week (78 days), then the frequency would be set to ([182 + 12 + 78] + 182)/2 = 227 days per year.
2. For each type of beverage, a measure of annual volume of ethanol intake was constructed that took into account both usual and heaviest consumption of that beverage: [(overall frequency minus frequency of drinking largest quantity) x usual quantity x typical size x ethanol conversion factor] + [frequency of drinking heaviest quantity x largest quantity x typical size x ethanol conversion factor]. Ethanol conversion factors, i.e., the proportion of ethanol content per ounce of beverage, were assumed to be 0.045 for beer, 0.121 for wine and 0.409 for spirits (DISCUS, 1985; Kling, 1989; Turner, 1990; Modern Brewery Age, 1992; Williams et al., 1993). The beverage-specific annual volumes, as calculated above, were summed across beverages to obtain the total annual volume of intake, which was divided by 365 to obtain average daily ethanol intake.
3. Average intake per drinking day was estimated by dividing the annual volume of intake (see above) by the number of drinking days per year. Thus, this measure also reflects both typical and heavy consumption and is not simply the usual or most typical quantity of drinks consumed.
4. The proportions of ethanol intake in the form of beer, wine and distilled spirits were estimated by dividing the beverage-specific volumes by the overall volume of ethanol intake.

In a test-retest survey conducted in a representative community sample (Grant et al., 1995), average daily ethanol intake had a reliability coefficient of 0.73, in the excellent or near excellent range. The reliability of the frequency of drinking 5 + drinks was not estimated, but overall frequency of drinking had a reliability coefficient of 0.76.

Statistical analysis

The analysis of the detailed breakdown by ethnic origin is based on pairwise comparisons of consumption: a) among the three racial groups of white, black and other, and b) between each racial group total and its component ethnic origin categories. The significance levels for the between-group racial comparisons are cited in the text; those for the within-racial-group differences by ethnic origin are indicated in the tables. All data were standardized for sex and age (using age groups 18–29, 30–49, 50+) in order to reduce any

confounding that might result from different demographic compositions of the various racial and ethnic groups. In this process, the age–sex-specific means for the consumption measures of each individual ethnic or racial group were reweighted using the age-sex breakdown of a standard population—either the total U.S. adult population (for estimating the prevalence of past-year drinking) or all past-year drinkers (for all other measures). Subsequent multivariate analyses of the broader ethnic origin categories used multivariate logistic or linear regression models to control for age, sex, marital status (married vs. other), education (high school graduate and college graduate, with less than high school as the referent), and family income.

The standardized estimates, regression parameters and their respective variances were estimated by the SUDAAN software package (Shah et al., 1996), which uses Taylor series linearization to adjust variance estimates for complex sample design characteristics such as clustering and stratification. Because of the multiple comparisons made in this analysis, two levels of statistical significance are indicated on the tables, $p < 0.05$ and $p < 0.001$. Although differences meeting either of these tests of significance are cited in the text, the former may be considered of marginal significance or merely suggestive of differences.

RESULTS

Table 1 examines three aspects of alcohol consumption—the overall prevalence of past-year drinking (i.e., of having consumed at least 12 drinks in the year pre-

Table 1. Prevalence of Drinking, Mean Daily Volume of Ethanol Intake and Distribution of Ethanol Intake by Beverage Type, by Detailed Categories of Race and Origin: Standardized by Age and Sex

	Prevalence of Past-year Drinking	*Mean Daily Ethanol Intake (ounces)*	*Total Ethanol Intake (%) Consumed In:*		
			Beer	*Wine*	*Spirits*
White[a]	47.4 (0.5)	0.70 (0.01)	52.8 (0.4)	26.3 (0.3)	20.9 (0.3)
Northern European	49.1 (0.6)*	0.73 (0.03)	52.5 (0.6)	25.8 (0.5)	21.7 (0.4)
English, Scottish, Welsh	46.3 (0.9)	0.70 (0.04)	50.2 (0.9)	27.9 (0.7)	21.9 (0.7)
Irish	52.3 (1.0)**	0.80 (0.07)	54.6 (0.9)	24.6 (0.8)	20.8 (0.7)
French, Swiss	51.6 (1.6)*	0.66 (0.06)	52.8 (1.6)	25.2 (1.3)	22.0 (1.2)
Belgian, Dutch	43.8 (2.3)	0.78 (0.10)	53.6 (2.3)	22.9 (1.9)	23.6 (2.0)
Scandinavian	51.2 (1.7)	0.67 (0.05)	54.0 (1.7)	23.7 (1.3)	22.3 (1.2)
Central European	52.4 (0.9)**	0.66 (0.02)	53.5 (0.6)	25.1 (0.6)	21.4 (0.5)
German	51.5 (1.0)**	0.66 (0.02)	54.6 (0.7)	24.0 (0.6)	21.4 (0.6)
Austrian	50.1 (1.0)*	0.68 (0.11)	40.1 (3.7)**	40.8 (4.2)**	19.1 (2.9)
Polish, Hungarian, Czechoslovakian, Yugoslavian	56.3 (1.6)**	0.64 (0.04)	50.8 (1.3)	27.7 (1.1)	21.5 (1.0)
Southern European	50.3 (1.3)*	0.66 (0.04)	44.1 (1.3)**	37.4 (1.2)**	18.5 (0.9)*
Italian	50.7 (1.4)*	0.65 (0.04)	44.1 (1.4)**	37.0 (1.3)**	18.9 (1.0)
Greek	45.0 (4.8)*	0.57 (0.07)	37.3 (4.1)**	42.7 (4.7)**	19.9 (3.3)
Spanish, Portugese	50.8 (3.4)	0.76 (0.10)	47.0 (3.3)	38.5 (3.1)**	14.5 (1.9)**
Eastern European	48.4 (2.6)	0.63 (0.09)	38.4 (2.1)**	39.5 (1.9)**	22.0 (1.8)
Middle Eastern	37.9 (4.6)	0.51 (0.14)	41.6 (4.5)*	36.5 (4.0)*	21.9 (3.0)
Native American	37.4 (2.2)**	0.92 (0.09)*	63.8 (2.9)**	13.8 (1.8)**	22.4 (2.3)
Hispanic	34.9 (1.4)**	0.65 (0.05)	61.5 (1.7)**	23.1 (1.4)*	15.3 (1.1)**
Mexican, Chicano, etc.	36.0 (1.9)**	0.76 (0.08)	66.3 (2.0)**	21.4 (1.6)*	12.3 (1.3)**
Central/South American	35.7 (2.9)**	0.54 (0.06)*	53.5 (4.2)	30.0 (4.0)	16.5 (2.4)
Caribbean	32.8 (2.9)**	0.40 (0.06)**	51.3 (4.6)	23.8 (3.4)	25.0 (3.2)
Black[a]	31.1 (0.8)	0.93 (0.06)	56.0 (1.2)	18.0 (0.8)	26.0 (1.0)
African American	31.8 (0.9)	0.98 (0.06)	55.9 (1.3)	17.1 (0.9)	27.0 (1.1)
African	26.4 (3.9)	0.88 (0.15)	59.0 (7.0)	19.3 (4.7)	21.7 (6.8)
Hispanic	30.4 (4.6)	0.87 (0.31)	45.2 (3.4)*	36.3 (4.6)**	18.5 (4.9)
Non-Hispanic Caribbean	25.7 (4.0)	0.29 (0.05)**	54.4 (4.6)	28.9 (3.5)*	16.7 (3.0)**
Greek	25.1 (5.1)	0.59 (0.12)*	60.2 (9.3)	26.4 (7.4)	13.4 (3.6)*
Other[a]	27.1 (1.4)	0.65 (0.06)	59.6 (2.1)	22.4 (2.0)	18.1 (1.6)
Japanese	29.7 (4.6)	0.76 (0.21)	43.0 (4.6)**	41.4 (4.5)**	15.6 (2.7)
Pacific Islander	32.4 (4.0)	0.71 (0.11)	50.7 (5.4)	33.0 (4.5)*	16.3 (2.7)
Native American	38.2 (3.8)*	0.92 (0.18)	73.6 (5.1)*	13.7 (3.6)*	12.7 (3.1)
Hispanic	34.4 (3.0)*	0.73 (0.11)	63.7 (3.3)	15.4 (3.3)	20.9 (3.5)
Non-Japanese Asian	17.1 (1.7)**	0.34 (0.07)**	53.8 (3.4)	26.9 (3.7)	19.3 (3.5)

Notes. Figures in parentheses are standard errors of estimates.
[a]Includes individuals with missing or other ethnic origins.
*Estimate is marginally different ($p < 0.05$) from that for racial group as a whole.
**Estimate is significantly different ($p < 0.001$) from that for racial group as a whole.

ceding interview), volume of consumption as indicated by average daily ethanol intake, and beverage preference. In terms of racial differences, the prevalence of past-year drinking was highest for individuals of white race ($p < 0.001$) and was marginally higher for those of black than other races ($p < 0.05$). Average daily volume of consumption was greater for blacks than for either whites or individuals of other races ($p < 0.001$). Although all three racial groups consumed the majority of their total ethanol intake in the form of beer, beer preference was marginally less predominant among whites than among blacks and persons of other races ($p < 0.05$).

Among whites, individuals of European origin demonstrated a prevalence of past-year drinking that exceeded the racial total; whereas the prevalence of past-year drinking was below the racial average for individuals of native American and Hispanic origin. The

Table 2. Frequency of Drinking, Intake per Drinking Day and Frequency of Heavy Drinking, by Detailed Categories of Race and Origin: Standardized by Age and Sex

	Mean Frequency of Drinking	*Mean Ethanol Intake per Drinking Day*	*Mean Frequency of Drinking 5+ Drinks*
White[a]	110.1 (1.0)	2.19 (0.02)	21.6 (0.6)
Northern European	112.4 (1.8)	2.19 (0.04)	22.0 (1.0)
English, Scottish, Welsh	113.6 (2.7)	2.08 (0.05)*	18.5 (1.3)*
Irish	111.7 (2.8)	2.36 (0.09)	25.5 (1.7)*
French, Swiss	105.2 (4.7)	2.10 (0.08)	21.2 (2.5)
Belgian, Dutch	117.9 (6.9)	2.26 (0.15)	29.8 (6.1)
Scandinavian	111.7 (5.0)	2.15 (0.08)	22.0 (2.4)
Central European	107.7 (2.0)	2.12 (0.03)*	21.8 (1.2)
German	109.1 (2.2)	2.13 (0.04)	22.8 (1.5)
Austrian	117.0 (12.5)	1.98 (0.17)	13.8 (4.7)**
Polish, Hungarian, Czechoslovakian, Yugoslavian	101.6 (3.8)*	2.07 (0.05)*	18.6 (2.0)
Southern European	112.0 (4.2)	2.09 (0.05)	15.4 (1.7)**
Italian	109.4 (4.6)	2.09 (0.06)	15.7 (1.9)*
Greek	111.2 (11.3)	2.12 (0.28)	6.3 (2.3)**
Spanish, Portugese	129.2 (12.0)	2.01 (0.12)	17.7 (4.5)
Eastern European	107.4 (7.1)	1.84 (0.09)**	18.7 (4.0)
Middle Eastern	82.6 (10.8)	1.92 (0.17)	10.4 (5.2)
Native American	124.4 (9.0)	2.73 (0.20)*	33.8 (5.4)*
Hispanic	90.9 (5.1)*	2.51 (0.08)**	23.3 (3.7)
Mexican, Chicano, etc.	99.4 (7.5)	2.66 (0.10)**	29.5 (5.8)
Central/South American	92.9 (11.9)	2.30 (0.16)	15.9 (3.0)
Caribbean	59.0 (5.2)**	2.31 (0.20)	10.6 (2.1)**
Black[a]	115.5 (3.2)	2.33 (0.07)	30.6 (2.2)
African American	117.9 (3.4)	2.40 (0.08)	32.7 (2.5)
African	132.7 (15.4)	2.11 (0.25)	26.1 (11.1)
Hispanic	113.7 (11.7)	2.42 (0.39)	9.7 (3.9)**
Non-Hispanic Caribbean	59.9 (9.7)**	1.50 (0.14)**	4.7 (1.3)**
Greek	94.2 (11.2)	2.03 (0.36)	22.4 (11.5)
Other[a]	91.6 (6.3)	2.44 (0.14)	23.1 (3.4)
Japanese	129.8 (20.1)	1.80 (0.18)**	17.3 (8.0)
Pacific Islander	99.3 (10.9)	2.39 (0.17)	32.7 (14.9)
Native American	85.9 (11.2)	3.39 (0.35)*	36.5 (12.2)
Hispanic	101.1 (12.3)	3.08 (0.35)	31.9 (6.6)
Non-Japanese Asian	68.1 (6.7)**	1.49 (0.09)**	3.8 (1.4)**

Notes. Figures in parentheses are standard errors of estimates.
[a]Includes individuals with missing or other ethnic origins.
*Estimate is marginally different ($p < 0.05$) from that for racial group as a whole.
**Estimate is significantly different ($p < 0.001$) from that for racial group as a whole.

only differences among whites in terms of volume of intake were that Hispanics of Caribbean and South/Central American origin consumed less alcohol than average and whites of native American origin consumed more alcohol than average. Whites exhibited more variation in terms of beverage preference. Individuals of Austrian, Southern European, Eastern European and Middle Eastern origin all reported a relatively high wine intake and relatively low beer intake. Those of native American and Hispanic origin were the opposite, with an above-average preference for beer and a below-average preference for wine. In addition, individuals of both Hispanic and Southern European origin reported a lower than average relative intake of distilled spirits.

Among blacks, there were no statistically significant differences by origin in the prevalence of past-year drinking. Lower than average volumes of intake were reported by black drinkers of Greek and especially of non-Hispanic Caribbean origin. Among the latter, the volume of consumption was less than one third that of all blacks combined. Blacks of both Hispanic and non-Hispanic Caribbean origin revealed a stronger preference for wine than other blacks. Hispanic blacks consumed a below average proportion of intake in the form of beer, and blacks of both non-Hispanic Caribbean and Greek origin consumed less than average in the form of distilled spirits.

The prevalence of drinking among individuals of other races was above the racial group total for those of native American and Hispanic origin and below the group total for those of non-Japanese Asian descent. The latter also reported a lower than average volume of consumption among those that did drink, less than half that of Japanese, Pacific islanders, and native Americans and Hispanics of other race. In terms of beverage preference, individuals of Japanese origin reported a relatively low intake of beer and high intake of wine, whereas those of native American origin did just the opposite. Pacific islanders also showed a stronger than average preference for wine.

Table 2 presents data on three drinking pattern measures — overall frequency, intake per drinking day, and frequency of heavy drinking. The mean overall frequency of drinking did not differ for whites and blacks, but was lower for members of other races ($p < 0.05$ relative to whites and $p < 0.001$ relative to blacks). There were not significant differences by race in the average volume of ethanol consumed per drinking day. The mean frequency of drinking 5+ drinks was higher for blacks than whites ($p < 0.001$), with individuals of other races not significantly different from either.

Among whites, Hispanics of Caribbean descent reported drinking less than two thirds as often as all whites combined. As a result, the mean frequency for all Hispanics was slightly below the group average, as was the frequency for individuals of Polish/Hungarian/Czechoslovakian/Yugoslavian origin. A number of European origins were associated with lower than average volumes of intake per drinking day, with the greatest reduction in volume among those of Eastern European descent. Whites of native American and Mexican/Chicano origin reported considerably higher volumes of intake per drinking day than all whites. Whites of Irish and native American origin were the only groups to report an above-average frequency of heavy drinking (i.e., of drinking 5 + drinks). Groups with lower than average frequencies of heavy drinking included those of English/Scottish/Welsh, Austrian, Southern European and Caribbean Hispanic origin.

The importance of Caribbean origin was apparent among blacks, as well. Blacks from the non-Hispanic Caribbean reported a lower overall frequency of drinking, lower volume of intake per drinking day and less frequent heavy drinking than did all blacks. Hispanic Blacks also reported a lower than average frequency of heavy drinking, but did not differ in terms of overall frequency or quantity per drinking day.

Among individuals of other races, those of non-Japanese Asian origin fell strikingly below the racial group average in terms of all three drinking-pattern measures; that is, they drank less frequently and in lower quantities per drinking day and drank 5+ drinks less often than average. In addition, volumes of intake per drinking day were below average for individuals of Japanese origin and above average for those of native American origin; however, these groups did not differ in terms of frequency of drinking (overall or heavy).

In order to test whether the ethnic and racial differences presented in Tables 1 and 2 would survive after adjusting for the additional confounders of marital status, education and income, some of the smaller categories had to be combined. Whites of French, Swiss, Belgian, Dutch, and Scandinavian origin were combined into "other Northern European," and whites of Austrian and Polish/Hungarian/Czechoslovakian/Yugoslavian descent were combined into "other Central European." All of the categories of European origin were restricted to individuals of white race. Individuals of Hispanic origin were combined into one of two categories, either "Mexican/Chicano" or "other Hispanic," without respect to race. Similarly, individuals of native American origin were combined into a single category regardless of whether of white or

other race. Accordingly, the category of "black" includes only non-Hispanic blacks, and the category of "Asian Pacific islander" excludes both Hispanics and native American of other races.

Tables 3 and 4 present the multivariate beta parameters corresponding to the revised ethnic origin categories, using the category of English/Scottish/Welsh as the reference group against which each of these parameters is evaluated. The beta parameters from the logistic model predicting the prevalence of past-year drinking (Table 3) can be exponentiated to yield odds ratios. For example, the odds of past year drinking were only 57% times as great for blacks as for whites of English/Scottish/Welsh origin, OR = $e^{-0.56} = 0.57$. The beta parameters for all of the other measures in Tables 3 and 4 are derived from linear regression models and can be interpreted as differences in mean values between the ethnic category in question and English/Scottish/Welsh.

As indicated in Table 3 all of the comparable racial and ethnic differences in prevalence of drinking that were observed in the more detailed breakdown of Table 1 retained their significance in the multivariate analyses. With respect to average volume of intake, the excess volumes that were formerly significant for blacks and for white native Americans lost their statistical significance. (The reduced levels of intake among blacks of non-Hispanic Caribbean and Greek origin also lost their significance, but this was the result of the reconfiguration of groups and the different reference category.) The variations in beverage preference by ethnic origin retained their general pattern and level of significance, although the magnitudes of the differences, especially the beer preference of Hispanics and native Americans, were reduced. With the addition of the controls, a few additional differences in beverage preference were observed, for example, an increased beer preference and decreased wine preference among individuals of Irish and German descent ($p < 0.05$).

As shown in Table 4, the majority of differences in drinking pattern that were observed in Table 2 were maintained in the multivariate analysis. Highlighting only those results in which changes occurred that were not the result of the reconfigured categories: 1) The reduced overall frequency of drinking among Hispanics of Mexican/Chicano origin became statistically significant ($p < 0.05$). 2) The increased frequencies of drinking 5+ drinks among blacks and whites of native American origin lost their statistical significance. 3) The excess consumption per drinking day of individuals of Irish origin took on a marginal level of statistical significance ($p < 0.05$).

DISCUSSION

The presentation of simple, descriptive data on ethnic differentials is not intended to promote stereotypes of ethnic drinking patterns, nor to suggest that race and origin per se are the cause of the differentials. Rather, the intent of this paper is to document differences that may prove useful in the development of hypotheses regarding *how* ethnic differentials evolve—hypotheses that are best tested in studies that focus on limited comparisons rather than on the broad range of comparisons included in this study.

Table 3. Prevalence of Drinking, Mean Daily Volume of Ethanol Intake and Distribution of Ethanol Intake by Beverage Type, by Broad Categories of Race and Origin: Beta Parameters Adjusted for Age, Sex, Marital Status, Education and Income

	Prevalence of Past-year Drinking[a]	*Mean Daily Ethanol Intake (ounces)*[b]	*Total Ethanol Intake (%) Consumed In:*		
			Beer[b]	*Wine*[b]	*Spirits*[b]
English, Scottish, Welsh	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)
Irish	0.25 (0.05)**	0.06 (0.08)	0.04 (0.01)*	−0.03 (0.01)*	−0.01 (0.01)
Other Northern European	0.17 (0.05)*	−0.04 (0.05)	0.02 (0.01)	−0.03 (0.01)*	0.00 (0.01)
German	0.26 (0.05)**	−0.07 (0.05)	0.03 (0.01)*	−0.03 (0.01)*	−0.01 (0.01)
Other Central European	0.38 (0.07)**	−0.07 (0.06)	−0.01 (0.01)	0.01 (0.01)	−0.00 (0.01)
Southern European	0.21 (0.06)*	−0.08 (0.06)	−0.07 (0.02)**	0.10 (0.01)**	−0.03 (0.01)*
Eastern European	−0.08 (0.11)	−0.08 (0.09)	−0.10 (0.02)**	0.10 (0.02)**	0.00 (0.02)
Mexican, Chicano, etc.[c]	−0.24 (0.09)*	−0.14 (0.09)	0.08 (0.02)**	−0.02 (0.01)	−0.06 (0.01)**
Other Hispanic[c]	−0.49 (0.10)**	−0.29 (0.08)**	−0.02 (0.02)	0.02 (0.02)	0.00 (0.02)
Black	−0.56 (0.05)**	0.12 (0.08)	0.01 (0.01)	−0.04 (0.01)**	0.03 (0.01)*
Asian/Pacific Islander	−1.28 (0.10)**	−0.23 (0.08)**	0.04 (0.02)	−0.02 (0.02)	−0.02 (0.02)
Native American[c]	−0.20 (0.10)*	0.11 (0.11)	0.07 (0.03)*	−0.06 (0.02)*	−0.01 (0.02)

Notes. Figures in parentheses are standard errors of estimates.
[a]Based on a logistic regression model.
[b]Based on a linear regression model.
[c]Regardless of race.
*Estimate is marginally different ($p < 0.05$) from that for English/Scottish/Welsh.
**Estimate is significantly different ($p < 0.001$) from that for English/Scottish/Welsh.

This study found that differences by ethnic origin *within* racial categories were as marked as racial differentials in drinking behaviors. Among whites, the major distinction was between those of European descent and those of other descents. Not only Hispanics but also whites of Middle Eastern and native American origin demonstrated drinking patterns that were distinct from whites of European origin. Moreover, among whites of European descent, those from Southern and Eastern Europe differed from those from Northern and Central Europe in terms of some aspects of drinking behavior such as beverage preference and frequency of heavy drinking. Also, Hispanics of Mexican descent had far different drinking patterns than those of Caribbean descent. Likewise, blacks from the English-speaking Caribbean differed from those of other origins, and among individuals of other races, those of Asian descent were distinct from those of native American, Pacific islander or Hispanic descent. Even among Asians, those of Japanese origin showed drinking patterns that differed from those of other, non-Japanese Asian origins. These findings clearly demonstrate that cultural forces exert a strong effect on drinking behavior, with beverage preference and frequency of heavy drinking showing residual effects even after many decades of presumed acculturation.

This study found several examples of ethnic differentials transcending race. Individuals of native American origin reported exceptionally high volumes of consumption and frequencies of heavy drinking regardless of whether they were of white or other race. Similarly, the effect of Caribbean origin was about the same for both whites and blacks, in both cases corresponding to significantly lower rates of drinking, reduced volume of consumption and less frequent heavy drinking. The effect of Greek origin was generally to lower prevalence, quantity and frequency of drinking among both blacks and whites, although these differences were not consistently significant.

The excess volume of consumption, level of intake per drinking day and frequency of heavy drinking that distinguished whites and blacks disappeared after adjustment for social indicators, although blacks remained less likely to be drinkers. However, most of the differentials involving whites, Hispanics, native Americans and individuals of Asian descent retained their level of statistical significance, despite some loss of magnitude, after multivariate adjustment. The persistence of these differentials by ethnic origin suggests that drinking patterns and beverage preference are not solely a function of social class, but rather of a broader range of cultural determinants.

The results of this study also indicated interesting relationships among various measures of drinking volume and pattern. Among individuals of Mexican/Chicano origin, for example, their excess frequencies of drinking 5+ drinks lost statistical significance after adjusting for measures of social class; however, their average intakes per drinking day remained significantly increased, exceeding those for all other ethnic groups. This suggests that they consumed *more* drinks on their heavy drinking days than did members of other ethnic

Table 4. Frequency of Drinking, Intake per Drinking Day and Frequency of Heavy Drinking, by Broad Categories of Race and Origin: Beta Parameters Adjusted for Age, Sex, Marital Status, Education and Income

	Mean Frequency of Drinking[a]	*Mean Ethanol Intake per Drinking Day*[a]	*Mean Frequency of Drinking 5+ Drinks*[a]
English, Scottish, Welsh	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)
Irish	−2.68 (4.05)	0.20 (0.10)*	5.55 (1.97)*
Other Northern European	−3.68 (4.09)	−0.00 (0.07)	3.06 (2.31)
German	−5.55 (3.57)	−0.02 (0.06)	2.62 (1.86)
Other Central European	−11.75 (4.77)	−0.04 (0.07)	−1.43 (2.23)
Southern European	−2.63 (5.38)	−0.07 (0.07)	−5.02 (2.23)*
Eastern European	−10.00 (7.83)	−0.18 (0.09)*	0.60 (3.90)
Mexican, Chicano, etc.[b]	−19.44 (6.58)*	0.28 (0.12)*	−0.54 (4.55)
Other Hispanic[b]	−31.43 (6.63)**	−0.04 (0.14)	−9.63 (3.18)*
Black	2.28 (4.37)	−0.09 (0.09)	4.51 (2.73)
Asian/Pacific Islander	−24.49 (7.57)*	−0.44 (0.10)**	−4.43 (4.57)
Native American[b]	−4.24 (6.34)	0.69 (0.22)*	4.62 (4.38)

Notes. Figures in parentheses are standard errors of estimates.
[a]Based on a linear regression model.
[b]Regardless of race.
*Estimate is marginally different ($p < 0.05$) from that for English/Scottish/Welsh.
**Estimate is significantly different ($p < 0.001$) from that for English/Scottish/Welsh.

groups. At the same time, neither of these two groups had an above-average overall volume of intake, because their high intakes on days when they drank were offset by below-average frequencies of drinking. In an example of the opposite effect, individuals of Eastern European origin had a significantly reduced intake per drinking day despite no decrease in the frequency of heavy drinking, suggesting relatively low levels of intake on their heavy-drinking days. These findings mirror those reported by Cahalan and Room (1974), in their comparisons by ethnic group of very heavy versus steady fairly heavy drinking and indicate that a simple measure of frequency of drinking 5+ drinks is inadequate to distinguish types of heavy-drinking patterns that may be associated with different types of consequences.

The classic distinction between "wet cultures" (in which drinking is highly prevalent and occurs frequently but rarely leads to heavy drinking, inebriation or acute alcohol problems) and "dry" cultures (in which drinking is less prevalent and less frequent but more often is concentrated into bouts of intoxication and associated adverse consequences) was not supported by these data. For example, among the five categories of European origin with an above average prevalence of drinking, none exhibited an increased frequency of drinking. Only one (Southern European) demonstrated a reduced frequency of heavy drinking, balanced by another (Irish) in which the frequency of heavy drinking was above average. Among individuals of Asian descent, who exhibited a "dry" pattern with low prevalence and infrequent drinking, the rate of heavy drinking was low as well. This suggests that there may be drinking patterns that lie outside the implied continuum between the classic wet and dry patterns, for example, those in which a strong physiological contraindication to drinking (such as the flushing response of many individuals of Asian origin) affects the selectivity of who becomes a drinker. Alternatively, the NLAES' exclusion of infrequent drinkers may account for some of the apparent deviations from this wet/dry continuum.

This exclusion of individuals who consumed less than 12 drinks from the ranks of past-year drinkers also limits direct comparisons with other studies of representative U.S. populations. For example, whereas this study found a lower prevalence of past-year drinking among Hispanic whites than among all whites, 34.9% versus 47.4%, the 1990 National Alcohol Survey found drinking to be equally prevalent among Hispanics and whites, 66.6% and 65.9% respectively (Midanik and Clark, 1994). This discrepancy indicates not only the magnitude of the proportion of infrequent drinkers, but also that variation in this proportion across ethnic groups affects estimates of ethnic differentials in the prevalence of drinking. Additionally, because the measures of alcohol consumption presented in this study were based on past-year drinkers, the exclusion of infrequent drinkers almost certainly led to higher volumes of intake and frequencies of heavy and overall drinking among past-year drinkers than would have been obtained if the infrequent drinkers had been included in the basis for these estimates. Again, this effect would be felt most strongly among ethnic groups with high proportions of infrequent drinkers. Unfortunately, these groups cannot be identified from this study alone but only by comparison with other studies, none of which have examined so many different ethnic categories.

Because the NLAES did not collect information to assess acculturation, these data cannot be used at the individual level to address its impact on drinking patterns. However, at the aggregate level, the data clearly showed the greatest variation and deviation from majority (European-white) group drinking patterns among a) the more recent immigrant groups of Hispanics and Asians, and b) blacks, whose assimilation arguably has been slowed by a long history of racial discrimination and segregation. Among whites of European origin, there was a great deal of homogeneity with respect to quantity and frequency of drinking, although beverage preference generally continued to reflect the cultural influences of the countries of origin, as in the high proportion of wine consumed by individuals of Southern European descent. Two anomalous findings were the low consumption levels and high proportions of wine consumed by individuals of Austrian and Eastern European (primarily Russian) descent and the low proportions of wine consumed by individuals of French and Swiss (primarily French) descent—each of which contradicts the beverage preference and/or consumption level of the country of origin (Commodity Board for the Distilled Spirits Industry, 1995). The former may reflect a high proportion of Jewish individuals among Austrian and Russian immigrants, although this cannot be verified because religion was not asked of NLAES respondents. Low consumption levels and the use of wine in religious rituals have been documented among Jewish Americans in many studies that have examined ethnic and religious differentials in drinking practices (Glassner and Berg, 1985; Bales, 1991). The surprisingly low levels of wine consumed by persons of French or Swiss origin may reflect the fact that the French immigrated to the United States many decades

Appendix A. Response Options for Ethnic Origin

Response Category	Total N	Past-year Drinkers	Response Category	Total N	Past-year Drinkers
African American (Black, Negro, or Afro-American)	5483	1719	Korean	123	34
African (e.g., Egyptian, Nigerian, Algerian)	189	69	Lebanese	60	23
American Indian	853	325	Malaysian	5	2
Australian, New Zealander	23	15	Mexican	717	236
Austrian	148	71	Mexican-American	667	279
Belgian	49	21	Norwegian	596	310
Canadian	294	141	Polish	1095	564
Central American (e.g., Nicaraguan, Guatamelan)	175	50	Puerto Rican	312	95
Chicano	44	25	Russian	528	230
Chinese	230	50	Scottish	945	428
Cuban	188	59	Samoan	5	0
Czechoslovakian	332	167	South American (e.g., Brazilian, Chilean, Columbian)	208	82
Danish	181	83	Spanish (Spain), Portuguese	325	160
Dutch	754	285	Swedish	551	276
English	4613	1987	Swiss	117	54
Filipino	185	54	Taiwanese	37	9
Finnish	95	47	Turkish	13	9
French	1093	515	Vietnamese	89	16
German	6384	3169	Welsh	215	111
Greek	251	91	Yugoslavian	145	74
Guamanian	14	4	Other Asian (Thai, Laotian, Cambodian, Burmese)	83	15
Hungarian	186	96	Other Carribbean or West Indian (Spanish speaking)	91	26
Indian, Afghanistani, Pakistani	159	44	Other Carribbean or West Indian (Non-Spanish speaking)	212	60
Indonesian	4	2	Other Eastern European (Romanian, Bulgarian, Albanian)	151	74
Iranian	43	17	Other middle Eastern (Saudi Arabian, Kuwaiti, Qatari, Syrian, Omani)	41	13
Iraqi	15	3	Other Pacific islander (Okinawan)	34	11
Irish	3892	1936	Other Spanish	63	26
Israeli	51	21	Other	6675	2803
Italian	1839	897			
Japanese	314	126			
Jordanian	13	5			

earlier on average than individuals from the Southern European wine region (U.S. Bureau of the Census, 1975), and often by way of Canada, at a time when wine may have been more difficult to obtain or to produce locally and when other beverages may have been substituted and incorporated into the immigrants' drinking culture.

The measure of ethnic origin used in this analysis is by no means a proxy for the broader concept of ethnicity. Among other limitations, it fails to incorporate the effects of religion and religiosity and their effects on drinking socialization and the development of abusive drinking patterns (McCready et al., 1983; Stivers, 1983). Earlier studies of drinking practices in the United States (Cahalan and Room, 1974; Cahalan et al., 1969) found that religious differences within categories of national identity were associated with a great deal of variation in drinking behavior, with conservative Protestants the least likely to drink and to drink heavily and Roman Catholics the most likely to do so. In addition, it must be recognized that ethnic origin is not necessarily an antecedent of drinking behavior, but rather that an individual may adopt an ethnic origin (or select one among the many that make up his ancestry) that most accurately reflects his desired or actual lifestyle, including patterns of drinking. As stated earlier, the intent of this paper was to provide the broadest possible description of differentials by ethnic origin, not to *explain* those differentials nor to suggest that ethnic origin per se was an explanatory factor. Through some of the questions raised above, it is hoped that this paper will provide the basis for further exploration of how drinking patterns are culturally transmitted and how aspects of this transmission might be manipulated to encourage moderate drinking practices among drinkers of all ethnic origins.

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Drinking Patterns and Their Consequences: Report From an International Meeting

Temporal Drinking Patterns and Variation in Social Consequences

Deborah A. Dawson

Temporal drinking patterns and their associated social consequences are described for a sample of U.S. adults aged 18 years and over who drank at least 12 drinks in the preceding year and did not restrict their drinking to special occasions (n = 16,086). The earliest time of day when these current regular drinkers reported usually drinking was between 6 a.m. and 11 a.m. for 1.2%, between 11 a.m. and 3 p.m. for 7.3%, between 3 p.m. and 6 p.m. for 31.2%, and after 6 p.m. for 60.3%. Less than one-tenth (7.7%) reported any drinking (not necessarily their earliest drinking) between midnight and 6 a.m. Characteristics associated with above-average rates of both early (6 a.m.–3 p.m.) and late-night (midnight–6 a.m.) drinking included male gender, black race, low education and income and heavy quantity of ethanol intake per drinking day. Early drinking was also characteristic of the elderly and daily drinkers. Prior to adjusting for background variables and quantity and frequency of intake, early drinking was associated with a two- to nine-fold increase in the risk of alcohol-related interpersonal problems, hazardous use, job/school problems and legal problems, and late-night drinking was associated with a three- to eight-fold increase in their prevalence. After adjusting for these factors in multiple logistic regression models, early drinking was associated with a 54% increase in the odds of interpersonal problems, a 39% increase in the odds of hazardous use and a 52% increase in the odds of legal problems. The association between early drinking and job/school problems fell just short of statistical significance. After adjusting for other factors, late-night drinking retained a significant association with all of the outcomes except legal problems. The magnitude of its association was greater than that of early drinking but varied substantially (i.e. interacted) with quantity of intake, race, ethnicity and gender.

INTRODUCTION

In contemporary industrial societies, time is a fundamental element of social organization (Zerubavel, 1981). The advent of industrialization brought about the separation of home and workplace, creating a distinction between work and leisure time. This resulted in a different set of behavioral norms for each of these periods and reinforced disapproval of drinking as a daytime activity (Tyrell, 1979; Rorabaugh, 1979). "Day and night, weekday and weekend, work-time and leisure time: these mark the boundaries of ordinary separation of abstinence-time from drinking-time in a wide range of American groups and sub-cultures" (Gusfield, 1987). Stopping by the tavern on the way home from work or having a drink shortly after getting home create a passage of mood that is culturally recognized in phrases such as "happy hour" (marking the end of the working day) or "Thank God It's Friday" (marking the end of the working week). Gusfield (1987) has argued that social drinking not only marks but actually facilitates the transition from work to leisure by creating a festive mood, promoting social solidarity through the dissolution of hierarchy and creating a cover or excuse for social faux pas.

Temporal drinking patterns and norms vary widely across cultures and social classes. MacAndrew & Edgerton (1969) have pointed out that many societies tolerate occasional or ritualized bouts of drunkenness, periods of "time out" during which standards of socially acceptable behavior are relaxed to a significant degree (although remaining within culturally agreed-upon limits.) In some European countries, alcohol consumption in the workplace typifies many working-class occupations, resulting from a combination of dif-

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ficult working conditions, easy access to alcohol, lack of supervision, and social pressure to drink (Wuthrich, 1987). A study of English office workers found that three-quarters thought that the consumption of two pints of beer or a bottle of wine at lunch was acceptable (Roberts, Cyster & McEwen, 1988). In European and Latin American cultures where wine is the predominant alcoholic beverage, alcohol consumption with the midday meal is typical. In drier cultures, or cultures where alcohol is not typically consumed with meals, midday drinking is less common and would be expected to be less socially acceptable. In most cultures morning drinking is associated with relief of alcohol withdrawal symptoms (Dawson *et al.,* 1996) and thus presumably would deviate from accepted drinking behaviors. In contemporary American society, failure to distinguish the varying levels of appropriateness of drinking at different times can lead to social disapproval or stronger sanctions, as Jellinek recognized in his definition of alpha (purely psychological) alcohol dependence: drinking that "contravenes such rules as society tacitly agrees upon—such as time, occasion, locale, amount and effect of drinking—but does not lead to 'loss of control' or 'inability to abstain'..."(Jellinek, 1960).

Beyond the question of how social norms affect reactions to drinking, temporal aspects of alcohol consumption might be expected to influence outcomes in more direct ways. Normal diurnal variations in alertness could modify or at least augment the effects of ethanol in terms of producing drowsiness and reducing reactivity (Headley, 1976). A recent study in which women considered to be moderate drinkers were given various doses of ethanol at 1:00 p.m. and at 6:30 p.m. found that the afternoon doses yielded significantly greater impairment in terms of reaction times and sleepiness factors (Horne & Gibbons, 1991). Similar results have been reported among men (Roehrs *et al.,* 1992). In addition, darkness could increase the risks of accidents following late-night drinking, and detection bias resulting from more vigilant police activity during the late-night hours could also impact on legal consequences during this period. Not surprisingly, alcohol-related moving vehicle accidents represent the one social consequence of drinking that has been linked most strongly with temporal aspects of drinking. Results of Minnesota roadside surveys conducted in 1985 and 1986 found that the frequency of drinking and driving increased dramatically after midnight, with an increase in driving while intoxicated incidents (DWIs) at about the time of bar closings (Tix & Palmer, 1988). Similar data have been reported for other states and types of vehicles (Hoxie *et al.,* 1988; Shore *et al.,* 1988). However, the nature of these data preclude defining late-night drinking as an independent risk factor for alcohol-related accidents. The preponderance of late-night fatal crashes may simply reflect the fact that persons driving late at night have had more time to drink and thus may have consumed more ethanol than people driving earlier in the evening.

Few studies have investigated temporal patterns of drinking (other than day of the week—see, for example, Cahalan *et al.,* 1969; Argeriou, 1975 and Harford & Gerstel, 1981) or their social consequences. Wilks & Callan (1990) found differences by beverage type in the time of day when alcohol was consumed, but their findings were based on a small non-representative sample of Australian students. Arfken (1988), using data from a representative sample of U.S. adults, found daily peaks in consumption during the early evening hours that were positively correlated with the distribution of motor vehicle accidents. However, no attempt was made to link drinking patterns with other social outcomes and the data, collected in 1979, may no longer reflect U.S. drinking patterns. The aim of this exploratory study is to describe temporal aspects of drinking in the United States using data from a recent national population study of drinking practices and alcohol-related problems and to investigate the link between the time of day when drinking occurs and various types of social consequences. The analysis investigates subgroup variation in the timing of drinking and uses multivariate models to assess the association between two temporal aspects of alcohol consumption—early and late-night drinking—and social consequences of drinking, adjusting for the effects of both sociodemographic characteristics and the quantity and frequency of drinking.

METHODS

Sample

This analysis is based on data from the 1992 National Longitudinal Alcohol Epidemiologic Study (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and conducted by the U.S. Bureau of the Census. The NLAES data were collected in personal interviews with adults 18 years of age and over, selected at random from a nationally representative sample of U.S. households. The NLAES sample size of 42,862 reflects household and sample person response rates of 92% and 97%, respectively. This analysis was based on current drinkers—individuals who drank at least 12 alcoholic

drinks in the year preceding interview. After excluding the 8.1% of current drinkers who drank only on special occasions and from whom data on time of consumption were not obtained, the sample size for this analysis was 16,086. Statistics reported in this analysis were derived using the SUDAAN software package (Research Triangle Institute, 1995), which employs Taylor series linearization techniques to adjust variance estimates for characteristics of complex, multistage sample designs such as that used in the NLAES (Grant *et al.,* 1994).

Measures

Respondents were asked on which days of the week they usually had something to drink in a typical month during the preceding year. For each day of the week on which they reported drinking, they were asked whether they usually had something to drink during various time periods, e.g. 6 a.m.–11 a.m., 11 a.m.–3 p.m., and so forth. These data were aggregated over all days of the week to determine the earliest time of day when drinking occurred and whether drinking took place after midnight (between midnight and 6 a.m.).

Eleven social consequences of drinking (see Knupfer, 1967; Cahalan, 1970; Cahalan & Room, 1974 for the derivation of these measures) were identified from a broad list of alcohol-related problems that were designed to operationalize the definitions of alcohol abuse and dependence according to several diagnostic systems. People were coded as having experienced each consequence if they reported that it ever happened in the 12 months preceding interview.

Quantity and frequency of drinking were derived from a series of questions on the usual and heaviest amounts of beer, wine and liquor consumed in the preceding year. Frequency, quantity and size of drink for each type of beverage were used to estimate annual volume of ethanol intake, assuming ethanol conversion factors of 0.045 for beer, 0.121 for wine and 0.409 for liquor (DISCUS, 1985; Kling, 1989; Turner, 1990; Modern Brewery Age, 1992; Williams, Clem & Dufour, 1993). Number of drinking days per year was estimated by taking the average of the sum of the beverage-specific frequencies and the largest individual beverage-specific frequency, with the result not allowed to exceed 365. Ethanol intake per drinking day was estimated by dividing annual intake by this frequency.

Individuals were defined as having a positive family history of alcoholism if they reported that any of 18 different types of first- and second-degree biological relatives had ever been alcoholics or problem drinkers. An alcoholic or problem drinker was defined as "a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover."

Analysis

In the first stage of the analysis, the 11 different indicators of social consequences were categorized to increase their prevalences and limit the number of outcome variables. Results of factor analysis and examination of correlations among these items led to grouping them into four broad categories: interpersonal problems, hazardous use, job/school problems, and legal problems (see Appendix I). The second stage of the analysis consisted of estimating simple descriptive statistics (distributions by timing of drinking for all current drinkers and within population subgroups) and bivariate associations between the timing variables and the four outcome measures. In the third stage of the analysis, the temporal drinking variables were collapsed into two dichotomous dummy variables, drinking before 3 p.m. and drinking after midnight, and were entered into hierarchical multiple logistic regression models predicting the odds of each of the four outcomes.

In the first stage of the model building, each outcome was predicted solely on the basis of background variables: age, gender, race and ethnicity, marital status, education, employment status and income, residential characteristics and family history of alcoholism. Quantity and frequency of drinking were entered in the second stage, along with total body water because of its mediating effect on the BAL resulting from a given level of intake (Goist & Sutker, 1985). The two timing variables were entered in the third stage, and interactions between the timing and other variables were evaluated in the fourth stage. The model predicting job/school problems was restricted to people who had ever been employed in the year preceding interview. In it, the dummy variable for past-year employment was replaced by dummy variables for professional and blue-collar occupations. In order to improve model fit, natural log transforms were applied to the measures of quantity and frequency of drinking.

RESULTS

As shown in Table 1, 1.2% of U.S. drinkers reported usually drinking in the period between 6 a.m. and 11 a.m., 7.3% reported their earliest usual drinking as having occurred between 11 a.m. and 3 p.m., 31.2% reported their earliest usual drinking between 3 p.m. and

6 p.m., and 60.3% of all drinkers typically did not start drinking until 6 p.m. or later. Less than one-tenth of all drinkers (7.7%) reported typically drinking after midnight on any day. Within population subgroups, there was considerable variation in the timing of drinking. Older drinkers were more likely than younger drinkers to drink early in the day, but less likely to drink after midnight. The population subgroups with above-average rates of both early *and* late-night drinking were men, people of black race, people who did not graduate from college, and people with annual family incomes of less than $21,600. The unmarried were more likely than the married to be late-night drinkers, and daily drinkers were more likely to drink early in the day than were people who drank less frequently. The prevalence of both early and late-night drinking increased directly with ethanol intake per drinking day.

As indicated in Table 2, the proportions of drinkers who experienced symptoms of the four problem domains varied strongly according to temporal characteristics of drinking. Individuals who drank before 3 p.m. were two to nine times as likely to have experienced these problems as those who did not drink until 6 p.m. or later. Within the area of interpersonal problems (adverse reactions from significant others), drinking before 11 a.m. had twice as strong an effect as drinking between 11 a.m. and 3 p.m. The other problem domains were equally strongly associated with *any* early drinking. Late-night drinking was associated with a

Table 1. Temporal drinking characteristics of current drinkers 18 years of age and over, by selected sociodemographic and consumption characteristics

	No. of cases	% Distribution by earliest time of day when drank				% Who drank after midnight
		6 a.m.–11 a.m.	11 a.m.–3 p.m.	3 p.m.–6 p.m.	6 p.m.–6 a.m.	
All current drinkers[a]	16 086	1.2 (0.1)	7.3 (0.2)	31.2 (0.5)	60.3 (0.6)	7.7 (0.3)
Age (years)						
18–29	4805	1.0 (0.2)	5.5 (0.4)	17.7 (0.7)	75.8 (0.8)	16.5 (0.8)
30–54	8322	1.0 (0.1)	8.7 (0.4)	32.8 (0.6)	58.4 (0.7)	4.4 (0.3)
55 +	2959	2.2 (0.3)	9.8 (0.6)	51.9 (1.0)	36.1 (1.0)	1.5 (0.3)
Male	8749	1.7 (0.2)	9.6 (0.4)	35.0 (0.7)	53.8 (0.8)	8.5 (0.4)
Female	7337	0.5 (0.1)	4.0 (0.3)	25.4 (0.7)	70.1 (0.7)	6.5 (0.4)
Black	1676	3.0 (0.5)	9.9 (1.0)	27.2 (1.4)	59.9 (1.6)	11.0 (1.0)
Non-black	14 110	1.0 (0.1)	7.1 (0.3)	31.6 (0.5)	60.3 (0.6)	7.4 (0.4)
Hispanic	893	1.7 (0.6)	10.3 (1.4)	27.6 (1.8)	60.4 (2.1)	8.5 (1.2)
Non-Hispanic	14 991	1.2 (0.1)	7.2 (0.3)	31.4 (0.5)	60.2 (0.6)	7.6 (0.3)
Married	8550	1.0 (0.1)	7.9 (0.3)	35.6 (0.6)	55.5 (0.7)	3.7 (0.3)
Unmarried	7398	1.5 (0.2)	6.5 (0.4)	24.0 (0.6)	68.1 (0.7)	14.1 (0.7)
College graduate	4730	0.4 (0.1)	4.6 (0.3)	33.0 (0.8)	62.0 (0.9)	3.9 (0.4)
Not college graduate	11 189	1.5 (0.1)	8.5 (0.3)	30.4 (0.6)	59.5 (0.7)	9.3 (0.4)
Annual family income						
Less than $21 600	5569	2.1 (0.2)	8.4 (0.5)	27.7 (0.8)	61.8 (1.0)	11.9 (0.8)
$21 600–44 999	5215	1.0 (0.2)	8.0 (0.5)	31.6 (0.8)	59.4 (0.8)	7.1 (0.5)
$45 000 or more	5302	0.6 (0.1)	5.9 (0.4)	33.9 (0.8)	59.6 (0.8)	4.6 (0.4)
Ever employed[b]	13 380	0.9 (0.1)	6.9 (0.3)	29.5 (0.5)	62.7 (0.6)	8.3 (0.4)
Never employed[b]	2613	3.0 (0.4)	10.2 (0.6)	41.5 (1.2)	45.3 (1.2)	4.3 (0.7)
Urban	12 286	1.2 (0.1)	7.3 (0.3)	31.1 (0.5)	60.4 (0.6)	7.9 (0.4)
Rural	3796	1.2 (0.2)	7.6 (0.5)	31.4 (1.0)	59.8 (1.2)	6.9 (0.7)
Northeast	3560	0.9 (0.1)	6.3 (0.1)	30.5 (1.1)	62.3 (1.4)	9.2 (0.7)
Midwest	4413	1.1 (0.2)	6.3 (0.6)	29.3 (1.1)	63.3 (1.3)	9.5 (0.6)
South	4714	1.2 (0.2)	8.0 (0.5)	30.9 (0.9)	59.9 (1.0)	6.5 (0.7)
West	3399	1.7 (0.2)	8.8 (0.6)	34.7 (1.1)	54.8 (1.3)	5.6 (0.5)
Daily drinker	1352	5.5 (0.7)	18.1 (1.3)	66.1 (1.6)	10.3 (0.9)	8.1 (0.9)
Non-daily drinker	14 592	0.8 (0.1)	6.4 (0.3)	28.1 (0.6)	64.7 (0.6)	7.7 (0.4)
Ethanol intake per drinking day						
Less than 1.0 oz.	2896	0.4 (0.1)	4.9 (0.5)	37.6 (1.1)	57.1 (1.1)	1.9 (0.3)
1.0–2.4 oz.	8576	0.7 (0.1)	6.2 (0.3)	31.8 (0.6)	61.3 (0.7)	4.8 (0.3)
2.5 oz or more	4367	2.4 (0.3)	10.9 (0.6)	26.4 (0.9)	60.3 (1.0)	16.2 (0.9)

Figures in parentheses are standard errors of percentages. Numbers show unweighted numbers of cases, but percentages are based on weighted data.
[a]Excluding those who drank only on special occasions.
[b]During year preceding interview.

three- to eight-fold increase in the prevalences of the different types of social consequences. Interestingly, its association with job and school problems was far stronger than its association with either legal problems, which encompasses motor vehicle accidents, or hazardous use, which encompasses driving after having had too much to drink.

Table 3 presents the four hierarchical stages of the multiple logistic regression model predicting the odds of interpersonal problems. All main effects, regardless of statistical significance, are presented in each stage of the model. The fourth-stage model shows only those interaction terms whose statistical significance was at or below the $p = 0.05$ level. The sociodemographic background variables included in each stage of the model generally yielded results consistent with past research examining abuse and dependence as outcomes (Grant & Harford, 1990; Dawson & Archer, 1993); that is, the odds of interpersonal problems decreased with age, were higher for males than females, were positively associated with family history of alcoholism and were inversely related to education and income. Having been employed at some point during the year reduced the odds of interpersonal problems due to drinking. Region showed a surprisingly strong effect, with increased odds of interpersonal problems in the Midwest and West.

The addition of quantity and frequency of drinking in the second stage of the model reduced the effects of gender, education, income and family history of alcoholism and revealed an increased risk of interpersonal problems among married people. As would be expected, both quantity and frequency of drinking were positively associated with the odds of interpersonal problems. Adding the two dummy variables for timing of drinking in the third stage of the model revealed a significant negative effect of black race but did not materially affect the other model parameters. The effect of early drinking was positive and statistically significant, while the effect of late-night drinking was not significant. In stage four, the inclusion of interactions between the timing and other variables revealed a negative interaction between quantity and late-night drinking and a positive interaction between black race and late-night drinking.

Since the changes that occurred across the hierarchical stages of model building were similar for all four problem domains, only the final (fourth-stage) models are presented for the domains of hazardous use, job/school problems and legal problems (Table 4). Early drinking was associated with increased odds of all of the outcomes except job/school problems, with which its association fell just short of statistical significance (OR= $e^{0.597} = 1.82$, $p = 0.06$). Late-night drinking was associated with increased odds of all of the outcomes except legal problems. Because of the frequent interactions between the temporal and other predictor variables, interpretation of the effects of the timing variables is not straightforward. Table 5 presents the range of odds ratios over different population subgroups.

Drinking before 3 p.m. increased the odds of interpersonal problems, hazardous use and legal problems by 54%, 39% and 52%, respectively. The effect of late-night drinking consistently decreased with quantity of ethanol intake per drinking day. Its effect was two to four times as great among people who drank two drinks (1 oz ethanol) per drinking day as among those who drank eight drinks (4.0 oz ethanol). There was variation by gender and ethnicity in the odds ratios associated with late-night drinking, with stronger adverse effects (that is, larger odds ratios) among women and minorities for some of the outcomes. Thus, for example, the odds of interpersonal problems were in-

Table 2. Percentage of current drinkers experiencing selected types of social consequences of drinking during past year, by timing of drinking

	Interpersonal problems	Hazardous use	Job/school problems	Legal problems
All current drinkers[a]	7.5 (0.2)	16.6 (0.4)	0.6 (0.1)	2.1 (0.1)
Earliest time of day when started drinking				
6 a.m.–11 a.m.	30.4 (3.7)	31.7 (3.9)	2.7 (1.3)	6.6 (1.7)
11 a.m.–3 p.m.	16.0 (1.3)	27.8 (1.6)	2.3 (0.6)	4.7 (0.8)
3 p.m.–6 p.m.	8.3 (0.5)	16.4 (0.7)	0.7 (0.2)	2.0 (0.2)
6 p.m.–6 a.m.	5.5 (0.3)	15.1 (0.5)	0.3 (0.1)	1.8 (0.2)
Ever drank between midnight and 6 a.m.				
Yes	19.4 (1.3)	43.3 (1.8)	3.2 (0.7)	6.2 (0.8)
No	6.4 (0.2)	14.3 (0.4)	0.4 (0.1)	1.8 (0.1)

Figures in parentheses are standard errors of percentages.
[a]Excluding those who drank only on special occasions.

Table 3. Hierarchical logistic regression models predicting odds of having experienced interpersonal problems during past year

	Stage 1 background variables only			Stage 2 quantity/frequency variables added			Stage 3 timing variables added			Stage 4 timing interactions added		
Intercept	−1.868	0.221	<0.001	−5.852	0.433	<0.001	−5.653	0.442	<0.001	−5.755	0.444	<0.001
Main Effects												
Age	−0.040	0.003	<0.001	−0.038	0.004	<0.001	−0.039	0.004	<0.001	−0.039	0.004	<0.001
Male	0.743	0.077	<0.001	0.345	0.149	0.023	0.309	0.156	0.051	0.298	0.156	0.060
Black	0.046	0.115	0.690	−0.156	0.136	0.255	−0.292	0.139	0.039	−0.542	0.158	0.001
Hispanic	−0.273	0.151	0.075	−0.301	0.172	0.083	−0.290	0.180	0.111	−0.298	0.179	0.101
Married	−0.114	0.080	0.159	0.311	0.091	0.001	0.326	0.094	<0.001	0.329	0.094	<0.001
College graduate	−0.750	0.101	<0.001	−0.481	0.110	<0.001	−0.420	0.112	<0.001	−0.416	0.113	<0.001
Income	−0.002	0.001	0.020	−0.001	0.001	0.160	−0.001	0.001	0.226	−0.001	0.001	0.252
Employed	−0.340	0.120	0.006	−0.240	0.132	0.071	−0.210	0.138	0.131	−0.217	0.137	0.116
Urban	−0.118	0.083	0.159	−0.100	0.094	0.291	−0.084	0.098	0.398	−0.088	0.098	0.376
Midwest	0.381	0.115	0.001	0.364	0.125	0.005	0.324	0.127	0.012	0.318	0.127	0.014
South	0.226	0.117	0.057	0.129	0.123	0.296	0.146	0.124	0.240	0.142	0.124	0.254
West	0.483	0.117	<0.001	0.461	0.125	<0.001	0.445	0.128	<0.001	0.435	0.127	0.001
Family history of alcoholism	0.936	0.093	<0.001	0.761	0.099	<0.001	0.743	0.102	<0.001	0.745	0.102	<0.001
Total body water	—	—	—	−0.012	0.010	0.206	−0.012	0.010	0.221	−0.012	0.010	0.250
Ethanol intake per drinking day[a]	—	—	—	1.383	0.081	<0.001	1.311	0.083	<0.001	1.394	0.088	<0.001
Frequency of drinking[b]	—	—	—	0.715	0.048	<0.001	0.681	0.048	<0.001	0.688	0.048	<0.001
Drank before 3 p.m.	—	—	—	—	—	—	0.440	0.114	<0.001	0.433	0.114	<0.001
Drank after midnight	—	—	—	—	—	—	0.160	0.119	0.180	0.665	0.274	0.017
Interactions												
After midnight * ethanol intake per drinking day[a]	—	—	—	—	—	—	—	—	—	−0.463	0.180	0.012
After midnight * black	—	—	—	—	—	—	—	—	—	0.964	0.334	0.005

[a]Ounces of ethanol, on a log scale.
[b]Days per year, on a log scale.

Table 4. Logistic regression models predicting odds of having experienced selected social consequences of drinking during past year

	Hazardous use			Job/school problems			Legal problems		
Intercept	−4.383	0.321	<0.001	−11.024	1.727	<0.001	−5.614	0.696	<0.001
Main effects									
Age	−0.049	0.003	<0.001	−0.061	0.023	0.009	−0.043	0.007	<0.001
Male	0.498	0.109	<0.001	−0.703	0.637	0.274	0.753	0.246	0.003
Black	−0.910	0.137	<0.001	0.424	0.440	0.339	−0.275	0.267	0.306
Hispanic	−0.505	0.139	<0.001	−1.011	0.599	0.096	0.022	0.251	0.932
Married	−0.314	0.063	<0.001	0.371	0.429	0.391	−0.357	0.167	0.035
College graduate	−0.096	0.070	0.174	−0.813	0.606	0.184	−0.707	0.219	0.002
Income	−0.001	0.001	0.344	−0.009	0.007	0.219	−0.003	0.002	0.078
Employed	0.193	0.119	0.105	NA	NA	NA	0.205	0.305	0.499
Professional occupation	NA	NA	NA	0.006	0.533	0.990	NA	NA	NA
Blue collar occupation	NA	NA	NA	−0.193	0.396	0.628	NA	NA	NA
Urban	−0.046	0.072	0.519	−0.101	0.341	0.767	−0.351	0.145	0.018
Midwest	0.534	0.095	<0.001	−0.546	0.489	0.268	0.311	0.216	0.154
South	0.042	0.096	0.658	−0.155	0.394	0.696	−0.227	0.229	0.325
West	0.357	0.096	<0.001	0.197	0.380	0.606	0.349	0.205	0.092
Family history of alcoholism	0.494	0.066	<0.001	0.191	0.432	0.661	0.393	0.153	0.012
Total body water (dl.)	−0.001	0.007	0.988	0.037	0.036	0.316	0.002	0.016	0.879
Ethanol intake per drinking day[a]	1.248	0.061	<0.001	2.395	0.333	<0.001	1.193	0.122	<0.001
Frequency of drinking[b]	0.621	0.033	<0.001	0.869	0.243	<0.001	0.342	0.084	<0.001
Drank before 3 p.m.	0.332	0.100	0.001	0.597	0.315	0.063	0.417	0.198	0.039
Drank after midnight	1.288	0.199	<0.001	2.625	0.794	0.002	−0.073	0.196	0.711
Interactions									
After midnight * ethanol intake per drinking day[a]	−0.560	0.150	<0.001	−1.217	0.487	0.015	—	—	—
After midnight * male	−0.410	0.180	0.025	—	—	—	—	—	—
After midnight * Hispanic	0.960	0.418	0.023	—	—	—	—	—	—

[a]Ounces of ethanol, on a log scale.
[b]Days per year, on a log scale.

creased by a factor of 2.29 – 5.10 (depending upon average ethanol intake) among black drinkers who drank after midnight; among non-black drinkers, late-night drinking increased the odds of interpersonal problems only among those with low levels of intake and only by a factor of 1.94.

DISCUSSION

This study found that temporal aspects of drinking were associated with a wide range of social consequences. Both early and late-night drinking increased the odds of interpersonal problems. This is not surprising, since both of these drinking behaviors are uncommon among U.S. drinkers, and apparently neither falls within norms of socially acceptable drinking styles. The racial differential in the effect of late-night drinking was unexpected. Since late-night drinking is more common among black than non-black drinkers, one would expect it to be a more accepted behavior within that subpopulation. Instead, late-night drinking was associated with a greater excess risk of interpersonal problems among black drinkers. Perhaps this reflects a perception on the part of family and friends that being out after midnight, for whatever reason, entails a greater set of risks for blacks than for non-blacks, or perhaps it reflects racial differences in drinking venue or the extent to which spouses or friends are included in late-night drinking occasions.

The association between late-night drinking and hazardous use varied according to both gender and Hispanic ethnicity, with higher odds ratios for women and Hispanics than for men and non-Hispanics. If women were more likely than men to consider the risk of sexual victimization among the drinking-related activities that could have resulted in their getting hurt, this could help to explain the higher odds ratios among women. The greater odds ratios among Hispanics do not suggest any obvious explanation, but may reflect ethnic differences in terms of density, loudness, gender mix and so forth in the settings in which late-night drinking takes place.

The surprisingly strong association between late-night drinking and job/school problems may reflect the consequences of job loss rather than a cause. That is, this association may simply indicate that people who

Table 5. Odds ratios for timing of drinking with respect to selected social consequences of drinking, by average ethanol intake per drinking day

	Average ethanol intake per drinking day		
	1.0 ounces (2 drinks)	2.5 ounces (5 drinks)	4.0 ounces (8 drinks)
Odds ratios for odds of having experienced interpersonal problems			
Drank before 3 p.m.	1.54 (1.23–1.93)	1.54 (1.23–1.93)	1.54 (1.23–1.93)
Drank after midnight			
Black	5.10 (2.54–10.26)	3.34 (1.84–6.05)	2.29 (1.47–4.92)
Non-black	1.94 (1.13–3.32)	1.27* (0.95–1.69)	1.02* (0.80–1.30)
Odds ratios for odds of having experienced hazardous use			
Drank before 3 p.m.	1.39 (1.15–1.70)	1.39 (1.15–1.70)	1.39 (1.15–1.70)
Drank after midnight			
Hispanic male	6.28 (2.48–15.91)	3.76 (1.66–8.52)	2.89 (1.31–6.37)
Hispanic female	9.47 (3.85–23.30)	5.67 (2.53–12.70)	4.36 (1.98–9.61)
Non-Hispanic male	2.41 (1.59–3.65)	1.44 (1.13–1.83)	1.11* (0.88–1.40)
Non-Hispanic female	3.62 (2.45–5.35)	2.17 (1.66–2.84)	1.67 (1.24–2.25)
Odds ratios for odds of having experienced job/school problems			
Drank before 3 p.m.	1.82* (0.98–3.37)	1.82* (0.98–3.37)	1.82* (0.98–3.37)
Drank after midnight	13.81 (2.91–65.49)	4.53 (2.03–10.10)	2.56 (1.45–4.52)
Odds ratios for odds of having experienced legal problems			
Drank before 3 p.m.	1.52 (1.03–2.24)	1.52 (1.03–2.24)	1.52 (1.03–2.24)
Drank after midnight	0.93* (0.63–1.36)	0.93* (0.63–1.36)	0.93* (0.63–1.36)

[a]Ounces of ethanol, on a log scale.
[b]Days per year, on a log scale.
*Not statistically significant ($p > 0.05$).

have lost a job are more able to drink late at night because they do not have to get up early to go to work. Alternatively, late-night drinking could be associated with hangovers and lateness that could increase the odds of both problems at work or school and actual job loss. While early drinking, which would occur during office hours for most workers, would presumably be an even greater risk factor for job problems, its lack of significance relative to that of late-night drinking may simply reflect its lower prevalence. Ideally, future studies examining job/school problems should investigate early and late-night drinking in separate models that take into account usual work hours.

The lack of significant association between late-night drinking and legal problems suggests that the excess of fatal crashes after midnight is more the effect of the quantity of ethanol consumed by persons driving at that time than of any inherent risks associated with drinking during that time period. The positive association between early drinking and this outcome is consistent with a Finnish study that found that problem drinkers were more likely to be detected by random breath testing during morning traffic than at other times (Dunbar, Pentilla & Pikkarainen, 1987).

In all the models in which late-night drinking was significantly associated with outcome, its effect was reduced by an interaction with average ethanol intake per drinking occasion. This reflects the fact that most very heavy drinkers reported drinking after midnight. The estimation of regression parameters assumes that all of the predictor variables are independent (i.e. uncorrelated). The fact that late-night drinking is positively correlated with quantity would result in overestimation of their cumulative impact at higher levels of consumption if only the main effects parameters were considered. The negative interaction between these two variables corrects for this association. In addition, late-night drinking coupled with low levels of consumption suggests a pattern of drinking that does not begin until late at night. This may be reflective of a lifestyle that would have an above-average association with various types of social problems.

Indeed, it is important to recognize that all of the adverse social consequences associated with early and late-night drinking may reflect characteristics of people who drink at those times in addition to or instead of innate risks attributable to drinking during those time periods. By controlling for demographic and socioeconomic factors, this analysis adjusted for many aspects of drinker selectivity, but others, such as differential psychological characteristics and social networks, may play a role in shaping the patterns of association described in this paper.

The results of this study suggest the need for additional research into two areas. One is the persistent regional differentials in social consequences that persist even after adjusting for urbanicity, income, ethnicity and so forth. It would be interesting to consider the impact of ecological factors such as speed limits, density of retail outlets and closing times for public drinking places as possible contributors to these differentials. Secondly, the preceding speculation as to the underlying causes of the interactions between the timing and sociodemographic variables reinforces the need to consider contextual variables—e.g., where and with whom drinking takes place—as important concomitants of drinking time. Only by collecting data on both temporal and contextual aspects of drinking can the joint and independent effects of these variables be understood.

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Appendix 1. Prevalences, factor analysis scores, and problems domains for symptom items representing social consequences of drinking

Symptom item	Prevalence	Factor loading values			Problem domain
		Factor 1	Factor 2	Factor 3	
Continue to drink even though you knew it was causing you trouble with your family or friends	3.3	0.67386	−0.10249	−0.37882	Interpersonal problems
Have arguments with your spouse, boy/girlfriend, family or friends because of your drinking	6.0	0.65584	−0.18410	−0.39357	Interpersonal problems
Drift apart from a spouse, boy/girlfriend, relative or friend you cared about because of your drinking	1.2	0.62771	0.18607	−0.34064	Interpersonal problems
Drive a car, motorcycle, truck, boat or any other vehicle after having too much to drink	11.9	0.51299	−0.38211	0.19344	Hazardous use
Get into a situation while or after drinking that increased your chances of getting hurt, like swimming, using machinery or walking in a dangerous area or around heavy traffic	4.8	0.53014	−0.39860	0.08696	Hazardous use
Get into a physical fight while/right after drinking	2.6	0.52958	−0.16990	0.04352	Hazardous use
Accidentally injure yourself while under the influence of alcohol, e.g. have a bad fall or cut yourself badly, get hurt in a traffic accident, or anything like that	2.4	0.51370	−0.14442	0.16006	Hazardous use
Lose a job because of your drinking	0.2	0.44745	0.68962	0.04702	Job/school problems
Have job or school trouble because of your drinking, like missing too much work, not doing your work well, being demoted at work, or dropping out of school	0.6	0.53517	0.58810	0.03364	Job/school problems
Have a car, motorcycle, truck, boat or other accident because of your drinking	0.6	0.36257	0.06753	0.62482	Legal problems
Get arrested or held at a police station because of your drinking	1.6	0.45611	−0.00695	0.46221	Legal problems

Drinking Patterns Among Individuals With and Without DSM–IV Alcohol Use Disorders

Deborah A. Dawson

Objective: *The purpose of this study was to compare alcohol consumption patterns among individuals with and without alcohol use disorders, using a representative sample of the general population that would not exaggerate differences as a result of selection biases associated with treatment for alcohol problems.* Method: *Based on data from 18,352 past-year drinkers selected from a nationally representative sample of U.S. households, 11 measures of past-year alcohol consumption were compared for three diagnostic groups: (1) individuals who did not meet the criteria for either alcohol abuse or dependence, i.e., those without a DSM-IV alcohol use disorder (AUD); (2) those classified with abuse only; and (3) those classified with alcohol dependence, with or without abuse.* Results: *For all measures reflecting frequency and quantity of drinking, frequency of heavy drinking and intoxication, and frequency of atypical temporal drinking patterns, the values for abusers lay midway between those for individuals without an AUD and those with dependence. Individuals with alcohol use disorders drank a greater proportion of their ethanol intake in the form of beer and a lower proportion in the form of wine than did those without an AUD. Of all the consumption measures considered, frequency of intoxication showed the strongest association with the probability of having an AUD, followed by frequency of drinking 5+ drinks, prevalence of morning drinking and total volume of intake. The ratios of consumption measures for individuals with disorders relative to those without an AUD showed relatively little significant variation across demographic subgroups of the population.* Conclusions: *The findings supported the distinction between the disorders of alcohol abuse and dependence, and implicated loss of control as an important element of this distinction. They also indicated that even among individuals with alcohol use disorders, demographic differentials reflecting cultural, physiological and normative forces were maintained and should be considered in approaches to treatment.*

Although the concept of alcoholism as a disease had been introduced by the middle of the nineteenth century (see review in Keller and Doria, 1991), it was not until the publication of Jellinek's *The Disease Concept of Alcoholism* more than a century later (Jellinek, 1960) that the distinction between excessive drinking and alcoholism achieved widespread public acceptance. Jellinek identified loss of control over drinking as the critical factor distinguishing two types of alcoholics: "alcohol addicts" and "habitual symptomatic excessive drinkers." Of these two groups, Jellinek argued that only alcohol addicts had a disease, that being loss of control over drinking and not excessive consumption per se. He similarly argued that the disorder that defined habitual symptomatic excessive drinkers was not frequent intoxication but the array of psychological and social problems from which frequent intoxication provided a temporary release (Jellinek, 1991).

As the diagnostic criteria for the classification of alcohol use disorders (AUDs) have evolved over past decades (Keller and Doria, 1991; Schuckit et al., 1991), they have steadily moved toward defining these disorders in terms of problems associated with excessive drinking rather than in terms of any specific level or pattern of consumption. In the United States, this movement was typified by the shift from the DSM-II (American Psychiatric Association, 1968) definition of alcoholism as a unitary personality disorder in which two of the three categories for alcoholics were characterized by frequency of intoxication to the problem-based criteria for alcohol abuse and dependence featured in the Feighner et al. (1972) and DSM-III criteria and their successors (American Psychiatric Association, 1980, 1987, 1994). Items concerning quantity and frequency of consumption that were initially considered for inclusion in these criteria were dropped because of concerns regarding their reliability and validity and because it was found that they could be omitted without any effect on diagnosis (Guze et al., 1969).

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Although it could be argued that excessive consumption is implicit in the criteria for AUDs, there is no fixed volume of ethanol intake or frequency of intoxication that is either necessary or sufficient for a classification of alcohol dependence, alcohol abuse or harmful drinking. In an analysis that examined various thresholds of volume, frequency of heavy drinking and frequency of intoxication as screeners for DSM-IV (American Psychiatric Association, 1994) alcohol dependence (Dawson, 1994), no single consumption measure demonstrated the levels of sensitivity and specificity that have been reported for screening instruments based on counts of alcohol-related problems (see, for example, Bush et al., 1987; Martin et al., 1990; Smart et al., 1991). Studies that have attempted to quantify the association between alcohol intake and alcohol use disorders have done so primarily by treating consumption measures as risk factors for dependence and/or abuse, specific problem domains or severity scores (i.e., problem counts). A number of analyses based on nationally representative samples of the U.S. population have demonstrated significant positive associations between these outcomes and volume of ethanol intake, overall frequency of drinking and quantity consumed per drinking day, frequency of heavy drinking and temporal drinking patterns (Caetano et al., 1997; Dawson, 1996a; Dawson and Archer, 1993; Dawson et al., 1995; Grant and Harford, 1989, 1990; Harford et al., 1991).

Beyond studies that have examined consumption measures as risk factors, there is only a small body of descriptive literature comparing levels and patterns of consumption among individuals with and without alcohol use disorders. York and Welte (1994) compared a treatment sample of 273 alcoholics with 133 nonalcoholic social drinkers of the same age and sex who were nominated by the alcoholics. Although their analysis focused on gender differences within each of the two samples, their data revealed striking differences between the alcoholic and nonalcoholic groups as well. For example, the proportion of days on which drinking occurred was four to five times greater for the alcoholic men and women; average ethanol intake per drinking day (adjusted for total body water) was six to seven times higher among the alcoholics; and maximum ethanol intake per drinking day was five to six times higher. Alcoholics of both sexes consumed a greater proportion of their total ethanol intake in the form of distilled spirits than did nonalcoholics.

Another analysis comparing 260 male alcoholics in inpatient alcohol treatment programs with a community sample of 160 male controls (Connors et al., 1986) found that 51% of the inpatients had at least one arrest for public intoxication, compared with 13% of the controls who were classified as problem drinkers and 3% of the controls classified as nonproblem drinkers. The alcoholic inpatients also had higher endorsement scores for alcohol's effects in most domains than did the controls. In a similar study based on 109 inpatient alcohol treatment patients and 97 age/sex matched controls (Olenick and Chalmers, 1991), scale scores for daily quantity of alcohol were more than twice as high for male alcoholics as for male controls and more than three times as high for female alcoholics as for female controls.

In a comparison of 45 women presenting for alcohol outpatient treatment with 42 matched controls who had been interviewed as part of a household survey 3 years earlier, Hanna (1991) found that the clinical sample consumed more than four times as much ethanol per drinking occasion than did the controls and reported greater proportions of drinking occasions lasting longer than 1 hour and occurring on weekdays and at times other than with dinner or in the evening. The clinical sample also reported a greater proportion of drinking occasions that took place at home and when alone.

All of the descriptive studies cited above were based on clinical samples of alcoholics. Research has shown that treated alcoholics have far more severe symptoms than those who do not enter treatment (Dawson, 1996b; Grant, 1996a), so it is not unreasonable to suspect that their consumption levels might be higher as well. Few of the studies defined alcoholics in a manner consistent with the current definition of alcohol dependence, and none examined abusers. The use of control groups composed of medical patients could have biased the comparisons by either yielding controls whose conditions contraindicated alcohol consumption or were associated with heavy consumption. Only one of the studies used a control group that was representative of a general community population, and none employed national population samples. These limitations diminish the considerable potential value of such descriptive studies to add to our understanding of the health, social and economic consequences of alcohol use disorders, many of which are tied directly to level and pattern of alcohol intake. This analysis remedies many of the limitations of previous descriptive studies. Using a large, nationally representative sample of past-year drinkers classified in accordance with the current DSM-IV criteria for alcohol abuse and dependence, it compares various measures of alcohol consumption among three groups: (1) individuals with neither abuse nor dependence, (2) individuals with abuse only and (3) individuals with alcohol dependence, with or without abuse.

METHOD

Sample

This analysis is based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and gathered information on alcohol consumption and alcohol-related problems from a nationally representative household sample of 42,862 U.S. adults 18 years of age and over (Grant et al., 1994a). The household and sample person response rates were 92% and 97%, respectively. This analysis was restricted to the 18,352 individuals identified as past-year drinkers, i.e., those who consumed at least 12 alcoholic drinks in the year preceding interview.

Measures

To be classified with past-year alcohol abuse, an individual had to meet one or more of the four DSM-IV criteria for abuse during the year preceding interview: (1) continued use despite social or interpersonal problems caused by drinking, (2) recurrent drinking in situations in which drinking is hazardous, (3) recurrent neglect of role obligations as a result of drinking and (4) recurrent alcohol-related legal problems. To be classified with alcohol dependence, an individual had to meet three or more of the seven DSM-IV criteria for dependence during the year preceding interview: (1) tolerance; (2) withdrawal (including relief or avoidance of withdrawal); (3) persistent desire or unsuccessful attempts to cut down on or stop drinking; (4) much time spent drinking, obtaining alcohol or recovering from its effects; (5) reduction or cessation of important activities in favor of drinking; (6) impaired control over drinking; and (7) continued use despite physical or psychological problems caused by drinking. Criteria *not* associated with duration qualifiers were considered to be satisfied if an individual reported one or more positive symptoms of the criterion during the past year. Criteria *with* duration qualifiers were considered to be satisfied if a person reported two or more symptoms during the past year or one symptom that occurred at least two times during the past year. To be consistent with the syndromal definition of the withdrawal criterion, two or more positive symptoms were required in addition to satisfaction of the duration qualifier.

The NLAES included separate sets of questions regarding the past-year consumption of beer, wine and distilled spirits. The overall frequency of drinking any alcohol was estimated as the mean of the sum of the beverage-specific frequencies (not allowed to exceed 365) and the largest individual frequency. Annual volume of ethanol intake was calculated as the sum of the beverage-specific volumes: ([total frequency$_{bev}$ minus frequency of drinking heaviest quantity$_{bev}$] x usual quantity$_{bev}$ x usual size$_{bev}$ x ethanol conversion factor$_{bev}$) + (frequency of drinking heaviest quantity$_{bev}$ x heaviest quantity$_{bev}$ x size of heaviest quantity$_{bev}$ x ethanol conversion factor$_{bev}$), using ethanol conversion factors of .045 for beer, .121 for wine and .409 for spirits (Beer analysis, 1992; Distilled Spirits Council of the United States, 1985; Kling, 1989; Turner, 1990; Williams et al., 1993). Annual volume of intake was divided by 365 to obtain average ethanol intake per day and by the overall frequency of drinking to obtain average intake per drinking day. Each of these values was in turn converted to its equivalent in terms of 14 g standard drinks. Frequencies of drinking 5+ drinks and of intoxication were asked directly. Drinkers were counted as drinking every day or nearly every day if their overall frequency of drinking was 312 or more days per year—the midpoint assigned to the category of drinking "nearly every day." Morning and weekday drinking were ascertained from questions that asked about days of the week and times of day when drinking typically took place.

In a test-retest survey conducted in a representative community sample (Grant et al., 1995), the classification of past-year alcohol use disorders (no disorder, abuse or dependence) demonstrated a reliability (kappa) coefficient of 0.76, the numbers of past-year abuse and dependence symptoms had coefficients of 0.73 and 0.75, respectively, and average daily ethanol intake had a reliability coefficient of 0.73—all in the excellent or near excellent range (Fleiss, 1981). The reliabilities of the frequencies of drinking 5+ drinks and of intoxication were not estimated, but overall frequency of drinking had a reliability coefficient of 0.76.

Analysis

The analysis is based on pairwise comparisons of consumption measures among the three diagnostic groups of no AUD, abuse only, and dependence, for the total population of past-year drinkers and within subcategories defined by gender, age and race/ethnicity. In addition, the results describe two sets of ratios: (1) the ratios of the consumption measures for individuals with abuse only relative to those for individuals without an AUD, and (2) the ratios for dependent individuals relative to those without an AUD. Standard errors and tests of differences were obtained using SUDAAN (Shah et al., 1995), a software package that employs

Taylor series linearization to account for complex sample design characteristics such as clustering, stratification and multistage sampling. Standard errors of ratios (not presented because of space limitations) were calculated by means of the delta method (Stuart and Ord, 1987), in which first-order derivatives are used to estimate the variance of any function of random variables: $Var(\mu_1/\mu_2) = Var(\mu_1)/\mu_2^2 + \mu_1^2 Var(\mu_2)/\mu_2^4 - 2\mu_1 Cov(\mu_1\mu_2)/\mu_2^3$. Because of the multiple comparisons made in this analysis, differences were not cited in the text unless they achieved a significance level of $p < .001$; significance levels of $.001 < p < .05$ were termed suggestive of differences or marginally significant.

RESULTS

Comparisons across diagnostic groups for all past-year drinkers

Table 1 compares 11 consumption measures for all past-year drinkers with neither abuse nor dependence, with abuse only, and with alcohol dependence. The basic measures of drinking level (average volume of in-

Table 1. Selected measures of past-year drinking pattern for past-year drinkers 18 years of age and over, by past-year DSM-IV alcohol use disorders and gender

	Neither abuse nor dependence	Abuse only	Dependence with or without abuse
Total			
Mean intake (14 g drinks) per day	0.8 ± 0.0	2.1 ± 0.1[aa]	3.5 ± 0.2[aa,bb]
Mean number of drinking days per year	100.2 ± 1.0	142.8 ± 4.1[aa]	171.6 ± 3.7[aa,bb]
Mean intake (14 g drinks) per drinking day	3.1 ± 0.0	5.2 ± 0.1[aa]	7.0 ± 0.2[aa,bb]
Mean days per year when drank 5+ drinks	12.8 ± 0.5	48.6 ± 3.1[aa]	83.2 ± 2.9[aa,bb]
Mean days per year when intoxicated	3.1 ± 0.2	16.4 ± 1.1[aa]	38.8 ± 2.0[aa,bb]
% of total ethanol intake in beer	50.6 ± 0.5	68.4 ± 1.1[aa]	66.1 ± 0.9[aa]
% of total ethanol intake in wine	28.1 ± 0.4	13.2 ± 0.7[aa]	12.2 ± 0.6[aa]
% of total ethanol intake in distilled spirits	21.3 ± 0.3	18.4 ± 0.8[aa]	21.7 ± 0.7[b]
% who drank daily or near daily	11.7 ± 0.3	17.5 ± 1.5[aa]	25.7 ± 1.3[aa,bb]
% who drank on weekdays	27.5 ± 0.4	41.4 ± 1.7[aa]	48.2 ± 1.4[aa,b]
% who drank in the morning	0.7 ± 0.1	1.8 ± 0.5[a]	3.7 ± 0.5[aa,b]
n of cases	15,442	1,186	1,724
Men			
Mean intake (14 g drinks) per day	1.0 ± 0.0	2.4 ± 0.1[aa]	4.1 ± 0.2[aa,bb]
Mean number of drinking days per year	111.2 ± 1.5	156.4 ± 4.9[aa]	182.2 ± 4.7[aa,bb]
Mean intake (14 g drinks) per drinking day	3.3 ± 0.0	5.5 ± 0.1[aa]	7.7 ± 0.3[aa,bb]
Mean days per year when drank 5+ drinks	17.3 ± 0.7	56.6 ± 4.0[aa]	95.9 ± 3.9[aa,bb]
Mean days per year when intoxicated	3.5 ± 0.2	17.7 ± 1.5[aa]	44.3 ± 2.8[aa,bb]
% of total ethanol intake in beer	62.5 ± 0.5	73.2 ± 1.2[aa]	72.0 ± 1.0[aa]
% of total ethanol intake in wine	17.7 ± 0.4	9.0 ± 0.7[aa]	7.5 ± 0.6[aa]
% of total ethanol intake in distilled spirits	19.8 ± 0.4	17.7 ± 1.0[a]	20.5 ± 0.9[b]
% who drank daily or near daily	13.8 ± 0.5	20.9 ± 1.8[aa]	29.5 ± 1.7[aa,bb]
% who drank on weekdays	30.6 ± 0.6	44.0 ± 2.0[aa]	49.2 ± 1.8[aa]
% who drank in the morning	1.0 ± 0.1	2.1 ± 0.6	4.6 ± 0.7[aa]
n of cases	8,028	826	1,101
Women			
Mean intake (14 g drinks) per day	0.6 ± 0.0	1.3 ± 0.1[aa]	2.4 ± 0.1[aa,bb]
Mean number of drinking days per year	85.0 ± 1.4	103.7 ± 5.4[aa]	147.5 ± 5.3[aa,bb]
Mean intake (14 g drinks) per drinking day	2.8 ± 0.0	4.6 ± 0.2[aa]	5.6 ± 0.2[aa,bb]
Mean days per year when drank 5+ drinks	6.7 ± 0.4	25.8 ± 3.2[aa]	54.7 ± 3.6[aa,bb]
Mean days per year when intoxicated	2.6 ± 0.2	12.8 ± 1.1[aa]	26.3 ± 2.6[aa,bb]
% of total ethanol intake in beer	34.1 ± 0.6	54.5 ± 2.1[aa]	52.8 ± 1.6[aa]
% of total ethanol intake in wine	42.5 ± 0.5	25.2 ± 1.6[aa]	22.7 ± 1.5[aa]
% of total ethanol intake in distilled spirits	23.4 ± 0.4	20.2 ± 1.6[a]	24.5 ± 1.3[b]
% who drank daily or near daily	8.8 ± 0.4	7.8 ± 1.7	17.0 ± 1.7[aa,bb]
% who drank on weekdays	23.2 ± 0.6	34.0 ± 2.9[aa]	45.9 ± 2.4[aa,b]
% who drank in the morning	0.3 ± 0.1	0.9 ± 0.4	1.7 ± 0.5[aa]
n of cases	7,414	360	623

Note: Figures after "±" are standard errors. A standard error of 0.0 indicates a value of less than 0.05.
[aa]Significantly different from estimate for those with neither abuse nor dependence ($p < .001$).
[a]Marginally different from estimate for those with neither abuse nor dependence ($.001 < p < .05$).
[bb]Significantly different from estimate for those with abuse only ($p < .001$).
[b]Marginally different from estimate for those with abuse only ($.001 < p < .05$).

take per day and per drinking day) and pattern (frequencies of any drinking, of drinking 5+ drinks and of intoxication) were all lowest for individuals without an AUD, of intermediate value for abusers and highest for those with dependence. For each of these measures, the value for abusers was close to midway between the values for individuals without an AUD and those with dependence (e.g., frequency of intoxication rose from 3.1 days among those with no disorder to 16.4 days for abusers and 38.8 days for those with dependence).

Beer accounted for just half (50.6%) of the total ethanol intake of individuals without an AUD but accounted for two-thirds of the intake of those with abuse only (68.4%) or dependence (66.1%). Individuals with abuse or dependence drank significantly *less* of their total intake in the form of wine (13.2% and 12.2%, respectively) than those without an AUD (28.1%). Whereas the proportions of intake in the forms of beer and wine were the same for abusers and those with dependence, the data suggested that the proportion of intake consumed in the form of distilled spirits was slightly lower for abusers (18.4%) than for individuals either with dependence (21.7%, $.001 < p < .05$) or without an AUD (21.3%, $p < .001$).

Table 2. Selected measures of past-year drinking pattern for past-year drinkers 18 years of age and over, by past-year DSM-IV alcohol use disorders and age

	Neither abuse nor dependence	Abuse only	Dependence with or without abuse
Ages 18-29			
Mean intake (14 g drinks) per day	0.8 ± 0.0	1.9 ± 0.1[aa]	3.1 ± 0.2[aa,bb]
Mean number of drinking days per year	67.3 ± 1.3	126.3 ± 5.8[aa]	146.6 ± 4.4[aa]
Mean (14 g drinks) per drinking day	4.0 ± 0.1	5.6 ± 0.2[aa]	7.2 ± 0.2[aa,bb]
Mean days per year when drank 5+ drinks	13.4 ± 0.6	43.6 ± 2.6[aa]	79.0 ± 3.7[aa,bb]
Mean days per year when intoxicated	5.4 ± 0.3	20.2 ± 1.6[aa]	42.3 ± 2.9[aa,bb]
% of total ethanol intake in beer	61.3 ± 0.6	70.8 ± 1.3[aa]	69.4 ± 1.1[aa]
% of total ethanol intake in wine	22.4 ± 0.6	12.0 ± 0.8[aa]	11.2 ± 0.8[aa]
% of total ethanol intake in distilled spirits	16.3 ± 0.4	17.2 ± 1.0	19.4 ± 0.9
% who drank daily or near daily	2.7 ± 0.3	11.3 ± 2.2[aa]	16.3 ± 1.6[aa]
% who drank on weekdays	17.5 ± 0.7	36.9 ± 2.5[aa]	44.3 ± 1.9[aa]
% who drank in the morning	0.6 ± 0.2	1.3 ± 0.4	2.3 ± 0.6[a]
n of cases	3,730	611	859
Ages 30-54			
Mean intake (14 g drinks) per day	0.8 ± 0.0	2.2 ± 0.1[aa]	3.8 ± 0.4[aa,bb]
Mean number of drinking days per year	95.6 ± 1.3	155.5 ± 6.7[aa]	193.6 ± 6.1[aa,bb]
Mean (14 g drinks) per drinking day	3.0 ± 0.0	4.8 ± 0.2[aa]	6.7 ± 0.4[aa,bb]
Mean days per year when drank 5+ drinks	12.7 ± 0.6	54.3 ± 6.4[aa]	84.3 ± 4.9[aa,bb]
Mean days per year when intoxicated	2.6 ± 0.2	11.4 ± 1.6[aa]	34.4 ± 3.0[aa,bb]
% of total ethanol intake in beer	50.9 ± 0.6	66.9 ± 1.7[aa]	64.1 ± 1.6[aa]
% of total ethanol intake in wine	30.0 ± 0.5	15.1 ± 1.2[aa]	13.6 ± 1.1[aa]
% of total ethanol intake in distilled spirits	19.1 ± 0.4	17.9 ± 1.2	22.3 ± 1.3[a,b]
% who drank daily or near daily	9.9 ± 0.4	22.6 ± 2.4[aa]	33.8 ± 2.2[aa,bb]
% who drank on weekdays	25.6 ± 0.6	44.0 ± 2.7[aa]	50.9 ± 2.3[aa,b]
% who drank in the morning	0.5 ± 0.1	2.0 ± 0.9	4.4 ± 0.8[aa,b]
n of cases	8,202	521	1742
Ages 55+			
Mean intake (14 g drinks) per day	1.1 ± 0.0	3.3 ± 0.7[a]	5.6 ± 0.8[aa,b]
Mean number of drinking days per year	152.9 ± 2.6	225.0 ± 20.4[aa]	255.0 ± 12.5[aa]
Mean (14 g drinks) per drinking day	2.3 ± 0.0	4.9 ± 0.4[aa]	7.7 ± 0.5[aa,b]
Mean days per year when drank 5+ drinks	12.6 ± 1.0	56.9 ± 15.7[a]	118.4 ± 14.1[aa,b]
Mean days per year when intoxicated	1.7 ± 0.3	17.4 ± 5.7[a]	36.7 ± 6.8[aa,b]
% of total ethanol intake in beer	36.5 ± 0.9	52.3 ± 6.1[a]	48.3 ± 4.1[a]
% of total ethanol intake in wine	30.4 ± 0.7	10.6 ± 2.9[aa]	10.5 ± 2.0[aa]
% of total ethanol intake in distilled spirits	33.1 ± 0.7	37.1 ± 5.7	41.2 ± 4.1
% who drank daily or near daily	27.5 ± 0.9	46.1 ± 7.8[a]	57.9 ± 5.3[aa]
% who drank on weekdays	44.8 ± 1.0	72.7 ± 6.8[aa]	67.1 ± 4.9[aa]
% who drank in the morning	1.5 ± 0.2	6.3 ± 2.7	13.4 ± 3.8[aa]
n of cases	3,510	54	123

Note: Figures after "±" are standard errors. A standard error of 0.0 indicates a value of less than 0.05.
[aa]Significantly different from estimate for those with neither abuse nor dependence ($p < .001$).
[a]Marginally different from estimate for those with neither abuse nor dependence ($.001 < p < .05$).
[bb]Significantly different from estimate for those with abuse only ($p < .001$).
[b]Marginally different from estimate for those with abuse only ($.001 < p < .05$).

The proportion of drinkers who drank on a daily or near daily basis was twice as great for dependent drinkers (25.7%) as for those without an AUD (11.7%), with abusers again midway between the two (17.5%). Weekday drinking was suggestive of a difference between individuals in the abuse and dependence categories (41.4% and 48.2%, respectively; $.001 < p < .05$) and was less prevalent among those without an AUD (27.5%). Morning drinking was suggestive of a difference between abusers and individuals without an AUD (0.7% and 1.8%; $.001 < p < .05$) and was higher among those with dependence (3.7%).

Comparisons across diagnostic groups within gender categories

As shown in the bottom two panels of Table 1, the gender-specific patterns generally mirrored those that were observed for all current drinkers, with the following major exceptions: (1) all differences in the proportion of intake in the form of distilled spirits were of only marginal significance ($.001 < p < .05$) for both men and women, and (2) among women, the prevalence of daily/near daily drinking did not differ for abusers and those without an AUD. In addition, a few

Table 3. Selected measures of past-year drinking pattern for past-year drinkers 18 years of age and over, by past-year DSM-IV alcohol use disorders and race/ethnicity

	Neither abuse nor dependence	Abuse only	Dependence with or without abuse
Non-Hispanic white			
Mean intake (14 g drinks) per day	0.8 ± 0.0	2.0 ± 0.1[aa]	3.5 ± 0.2[aa,bb]
Mean number of drinking days per year	102.6 ± 1.2	139.9 ± 4.1[aa]	170.5 ± 4.0[aa,bb]
Mean intake (14 g drinks) per drinking day	3.0 ± 0.0	5.1 ± 0.1[aa]	7.1 ± 0.5[aa,bb]
Mean days per year when drank 5+ drinks	12.2 ± 0.5	46.1 ± 2.7[aa]	84.3 ± 3.4[aa,bb]
Mean days per year when intoxicated	2.9 ± 0.2	16.3 ± 1.1[aa]	38.9 ± 2.3[aa,bb]
% of total ethanol intake in beer	48.8 ± 0.5	68.4 ± 1.2[aa]	65.4 ± 1.1[aa]
% of total ethanol intake in wine	29.5 ± 0.4	13.4 ± 0.7[aa]	12.4 ± 0.7[aa]
% of total ethanol intake in distilled spirits	21.7 ± 0.3	18.3 ± 0.9[aa]	22.1 ± 0.9
% who drank daily or near daily	12.5 ± 0.4	16.4 ± 1.5[aa]	24.9 ± 1.4[aa,bb]
% who drank on weekdays	29.3 ± 0.5	40.2 ± 1.8[aa]	50.8 ± 1.6[aa,bb]
% who drank in the morning	0.6 ± 0.1	1.5 ± 0.4[a]	3.0 ± 0.5[aa,b]
n of cases	12,667	1,019	1,293
Non-Hispanic black			
Mean intake (14 g drinks) per day	1.1 ± 0.1	3.8 ± 0.7[aa]	4.7 ± 0.6[aa]
Mean number of drinking days per year	98.6 ± 3.1	201.7 ± 18.6[aa]	212.8 ± 9.1[aa]
Mean intake (14 g drinks) per drinking day	3.4 ± 0.1	5.9 ± 0.6[aa]	7.1 ± 0.5[aa]
Mean days per year when drank 5+ drinks	19.7 ± 1.9	63.4 ± 16.1[a]	104.5 ± 9.7[aa,b]
Mean days per year when intoxicated	4.5 ± 0.7	24.3 ± 9.3[a]	51.1 ± 6.8[aa,b]
% of total ethanol intake in beer	57.9 ± 1.3	53.9 ± 4.7	61.0 ± 2.9
% of total ethanol intake in wine	18.3 ± 0.9	16.1 ± 4.2	12.6 ± 2.0[aa]
% of total ethanol intake in distilled spirits	23.8 ± 1.1	30.0 ± 4.1	26.4 ± 2.6
% who drank daily or near daily	9.2 ± 0.9	36.3 ± 7.3[aa]	36.3± 3.7[aa]
% who drank on weekdays	21.2 ± 1.3	55.0 ± 6.9[aa]	46.2 ± 4.1[aa]
% who drank in the morning	1.6 ± 0.4	8.2 ± 4.3	9.3 ± 2.4[aa]
n of cases	1,485	68	220
Hispanic			
Mean intake (14 g drinks) per day	0.8 ± 0.1	2.6 ± 0.6[a]	2.4 ± 0.3[aa]
Mean number of drinking days per year	82.7 ± 3.9	157.5 ± 23.5[aa]	134.5 ± 12.8[aa]
Mean intake (14 g drinks) per drinking day	3.8 ± 0.1	5.6 ± 0.4[aa]	6.7 ± 0.4[aa]
Mean days per year when drank 5+ drinks	14.7 ± 1.6	73.4 ± 28.2[a]	55.6 ± 6.8[aa]
Mean days per year when intoxicated	3.3 ± 0.6	9.8 ± 2.9[a]	22.9 ± 3.6[aa,b]
% of total ethanol intake in beer	59.5 ± 1.6	80.7 ± 3.4[aa]	76.4 ± 2.8[aa]
% of total ethanol intake in wine	24.7 ± 1.4	8.7 ± 2.3[aa]	9.4 ± 1.5[aa]
% of total ethanol intake in distilled spirits	15.9± 1.0	10.6 ± 2.2[a]	14.1 ± 2.3
% who drank daily or near daily	8.4 ± 1.1	24.4 ± 8.4	20.3 ± 4.1[aa]
% who drank on weekdays	17.8 ± 1.0	45.8 ± 8.0[aa]	28.9 ± 4.4
% who drank in the morning	0.9 ± 0.4	1.7 ± 1.7	4.8 ± 2.3
n of cases	835	68	143

Note: Figures after "±" are standard errors. A standard error of 0.0 indicates a value of less than 0.05.
[aa]Significantly different from estimate for those with neither abuse nor dependence ($p < .001$).
[a]Marginally different from estimate for those with neither abuse nor dependence ($.001 < p < .05$).
[bb]Significantly different from estimate for those with abuse only ($p < .001$).
[b]Marginally different from estimate for those with abuse only ($p < .001$).

of the differences that were of marginal significance for the total population were nonsignificant within one or both of the gender groups.

Comparisons across diagnostic groups within age categories

Table 2 presents consumption data for the three diagnostic groups within three age categories: 18–29 years, 30–54 years and 55 years of age and older. Age-specific comparisons among the three diagnostic groups differed from those for the total population of current drinkers in a number of ways. Among individuals 18–29 years of age: (1) there were no differences in the proportions of total intake consumed in distilled spirits; (2) dependent individuals and abusers did not differ in terms of overall frequency of drinking and were equally likely to be daily or near daily drinkers; and (3) the prevalence of morning drinking did not differentiate abusers and those with dependence and was only marginally lower for individuals without an AUD than for those with dependence. Among individuals 30–54 years of age, the comparisons across the three diagnostic groups matched those for the total population, except that the differences across groups in the proportion of intake in the form of distilled spirits were non-significant or only marginally significant. Among individuals 55 years of age and older: (1) the proportion of ethanol intake in the form of beer was only marginally lower ($.001 < p < .05$) for individuals without an AUD relative to abusers and those with dependence, and (2) many of the pairwise differences involving abusers were of only marginal significance or not statistically significant at all because of the small number of abusers in this age group ($n = 54$). As with

Table 4. Ratios of selected measures of past-year drinking pattern among past-year drinkers 18 years of age and over with DSM-IV alcohol abuse and dependence relative to those with neither abuse nor dependence, by gender, age and race/ethnicity

				Age			Non-Hispanic		
	Total	Men	Women	18-29	30-54	55+	White	Black	Hispanic
Ratios for individuals with abuse only relative to those with neither abuse nor dependence									
Mean intake (14 g drinks) per day	2.6	2.3	2.0	2.4	2.6	3.3	2.4	3.8	3.2
Mean number of drinking days per year	1.4	1.4	1.2[a]	1.9	1.6[b]	1.5[b]	1.4	2.1[dd]	1.9
Mean intake (14 g drinks) per drinking day	1.7	1.7	1.7	1.4	1.6[b]	2.1[b]	1.7	1.8	1.5
Mean days per year when drank 5+ drinks	3.8	3.3	3.9	3.3	4.3	4.5	3.8	3.2	5.0
Mean days per year when intoxicated	5.3	5.1	4.9	3.7	4.4	10.2	5.6	5.4	3.0[d]
% of total ethanol intake in beer	1.4	1.2	1.6[aa]	1.2	1.3[bb]	1.4	1.4	0.9[dd]	1.4[ee]
% of total ethanol intake in wine	0.5	0.5	0.6	0.5	0.5	0.4	0.5	0.9	0.4[e]
% of total ethanol intake in distilled spirits	0.9	0.9	0.9	1.1	0.9	1.1	0.8	1.3[d]	0.7[e]
% who drank daily or near daily	1.5	1.5	0.9[a]	4.2	2.3	1.7[b]	1.3	4.0[d]	2.9
% who drank on weekdays	1.5	1.4	1.5	2.1	1.7	1.6[b]	1.4	2.6[dd]	2.6[d]
% who drank in the morning	2.6	2.1	3.0	2.2	4.0	4.2	2.5	5.1	1.9
Ratios for individuals with dependence (w/wo abuse) relative to those with neither abuse nor dependence									
Mean intake (14 g drinks) per day	4.2	4.0	3.5	3.8	4.6	5.7[b]	4.2	4.7	2.8[d]
Mean number of drinking days per year	1.7	1.6	1.7	2.2	2.0	1.7[bb,c]	1.7	2.2[dd]	1.6[e]
Mean intake (14 g drinks) per drinking day	2.2	2.3	2.0[a]	1.8	2.3[b]	3.3[bb]	2.4	2.2	1.7[dd]
Mean days per year when drank 5+ drinks	6.5	5.5	8.2[aa]	5.9	6.6	9.4[b]	6.9	5.3	3.8[dd]
Mean days per year when intoxicated	12.5	12.7	10.1	7.8	13.2[b]	21.6[b]	13.4	11.4	6.9[d]
% of total ethanol intake in beer	1.3	1.2	1.5[aa]	1.1	1.3[bb]	1.3	1.3	1.1[dd]	1.3[e]
% of total ethanol intake in wine	0.4	0.4	0.5	0.5	0.5	0.3	0.4	0.7[d]	0.4[e]
% of total ethanol intake in distilled spirits	1.0	1.0	1.0	1.2	1.2	1.2	1.0	1.1	0.9
% who drank daily or near daily	2.2	2.1	1.9	6.0	3.4[b]	2.1[bb,cc]	2.0	3.9[dd]	2.4
% who drank on weekdays	1.8	1.6	2.0[a]	2.5	2.0[b]	1.5[bb,c]	1.7	2.2	1.6
% who drank in the morning	5.3	4.6	5.3	3.8	8.8	8.9	5.0	5.8	5.3

[aa]Significantly different from ratio for men ($p < .001$).
[bb]Significantly different from ratio for those aged 18-29 ($p < .001$).
[cc]Significantly different from ratio for those aged 30-54 ($p < .001$).
[dd]Significantly different from ratio for non-Hispanic whites ($p < .001$).
[ee]Significantly different from ratio for non-Hispanic blacks ($p < .001$).
[a]Marginally different from ratio for men ($.001 < p < .05$).
[b]Marginally different from ratio for those aged 18-29 ($.001 < p < .05$).
[c]Marginally different from ratio for those aged 30-54 ($.001 < p < .05$).
[d]Marginally different from ratio for non-Hispanic whites ($.001 < p < .05$).
[e]Marginally different from ratio for non-Hispanic blacks ($.001 < p < .05$).

gender, many of the differences that were of marginal significance for the total population were nonsignificant within individual age groups.

Comparisons across diagnostic groups within race/ethnic categories

Table 3 compares consumption measures across the three diagnostic groups within three categories of race/ethnicity: non-Hispanic whites (hereafter referred to as whites), non-Hispanic blacks (hereafter referred to as blacks) and Hispanics. Among whites, the significant differences mirrored those for the total population, with the addition that dependent individuals had a higher prevalence of weekday drinking than did abusers. Because of their small numbers of cases, the other two racial/ethnic categories demonstrated fewer significant differences across diagnostic groups. Among blacks, the only differences that retained their level of significance were that (1) individuals with either abuse or dependence had larger volumes of intake per day and per drinking day, a higher overall frequency of drinking, and a greater prevalence of daily/near daily and weekday drinking than those without an AUD; and (2) those with dependence (but not the abusers) also had higher frequencies of heavy drinking and intoxication and a higher prevalence of morning drinking. None of the differences between abusers and dependent individuals retained their level of significance, although the higher frequencies of heavy drinking and intoxication among the latter were of marginal significance. Among Hispanics, the following differences remained significant: (1) individuals with either abuse or dependence had higher frequencies of drinking, higher volumes of intake per drinking day and proportionately more ethanol consumed in the form of beer and less in the form of wine than did those without an AUD; (2) those with abuse only reported more weekday drinking that those without an AUD; and (3) those with dependence reported a higher overall volume of intake and greater prevalence of daily or near daily drinking than those without an AUD. As with blacks, none of the differences between abusers and dependent individuals retained their level of significance, although the higher frequency of intoxication among those with dependence was marginally significant.

Comparison of consumption ratios across consumption measures

Table 4 presents two set of ratios, one comparing the consumption measures for abusers to those for individuals without an AUD and the second comparing individuals with dependence to those without an AUD. These ratios are shown for all current drinkers and within categories of gender, age and race/ethnicity. Among all current drinkers, the consumption measure whose ratios deviated most strongly from 1.00 (i.e., that indicated the strongest level of association with alcohol use disorders) was frequency of intoxication, followed by frequency of heavy drinking (5+ drinks), prevalence of morning drinking and average intake per day. Although the magnitudes of the ratios varied dramatically across consumption measures, the ratios for each individual measure showed little variation across demographic subgroups. Only 18 of the 154 pairwise comparisons (12%) revealed differences that were significant at the $p < .001$ level; another 27 (18%) were of marginal significance ($.001 < p < .05$). This is not inconsistent with the observed variation in significance level among demographic subgroups, which was largely the result of unequal subgroup sample sizes and does not preclude consistency in the *magnitudes* of the ratios.

Among the ratios for individuals with abuse only compared to those without an AUD, only the following differences were significant: First, the ratio for the proportion of total intake in the form of beer was higher for women than for men (1.6 vs 1.2), was slightly higher for those aged 30–54 than for those aged 18–29 (1.3 vs 1.2) and was lower for blacks (0.9) than for either whites or Hispanics (1.4 each). Second, the ratios for overall frequency of drinking (mean number of drinking days per year) and prevalence of weekday drinking were higher for blacks than for whites (2.1 vs 1.4 and 2.6 vs 1.4, respectively). Among the differences that were of marginal significance, some of the more striking were: (1) lower ratios among individuals aged 30 and over for overall frequency of drinking, accompanied by higher ratios for intake per drinking day, and (2) lower ratios among individuals aged 55 and over for the prevalence of daily/near daily and weekday drinking.

When individuals with dependence were compared to those without an AUD, there was more demographic variation in the ratios. The ratios for overall frequency of consumption were disproportionately low among individuals aged 55 and over and disproportionately high among blacks. The ratios for intake per drinking day were increased within the oldest age group and decreased among Hispanics. The ratio for frequency of intoxication was likewise reduced among Hispanics. For the proportion of intake in the form of beer, the ratios were increased among women and individuals aged 30–54 and decreased among blacks. Among individuals aged 55 and over, the ratios for both daily/near daily drinking and weekday drinking were reduced, and among blacks, the ratio for

daily/near daily drinking was increased. Among the numerous differences of marginal significance were lower ratios among Hispanics for average intake per day and frequency of intoxication and higher ratios at age 30 and over for frequency of intoxication.

DISCUSSION

These data from a national population sample revealed that a variety of consumption measures were lowest for individuals without an AUD, higher for those with alcohol abuse and highest for those with alcohol dependence. Regardless of measure, the values for abusers were positioned midway between those of dependent individuals and individuals without an AUD, with all between-group differences statistically significant for the total sample of past-year drinkers.

Compared to studies based on clinical samples of alcoholics and nonclinic controls, this study found smaller differences between the consumption patterns of individuals with and without alcohol use disorders. The data from the study by York and Welte (1991), for example, indicated that male alcoholics drank 6.1 times as many drinks per drinking day as did male social drinkers, and that female alcoholics drank 7.4 times as many drinks per drinking day as did female social drinkers, based on their most recent drinking patterns. Based on lifetime measures, the ratios were 3.3 and 3.9, respectively. In Hanna's study of female drinkers, this ratio was 4.3. In the present study, based on consumption patterns during the year preceding interview, the ratio for individuals with dependence relative to those without an AUD was 2.3 for men and 2.0 for women. This discrepancy illustrates the extent to which comparisons based on clinical samples (i.e., those that are likely to be selected in terms of the severity of their disorder) may overestimate differences in the consumption patterns of *all* individuals with alcohol use disorders relative to those without an AUD. The higher ratios for women than men in the York and Welte study (1991), which were not replicated in the present study, also support arguments that women may enter treatment at a more severe stage of impairment than do men (Farid and Clarke, 1992; Hasin et al., 1988)—making clinic-based comparisons even more biased for women than for men.

The findings of this study are interesting in relation to the debate regarding the dimensionality of alcohol use disorders. Despite the residual nature of the abuse category in the DSM-IV, there is much evidence that abuse and dependence represent two distinct disorders. Psychometric analyses have demonstrated that the symptoms of abuse and dependence yield two separate factors, with the more severe factor more closely linked to familial alcoholism (Muthen, 1995, 1996; Muthen et al., 1993), and Hasin and associates (1990, 1997) have demonstrated that the two disorders can be differentiated in terms of their natural histories. Grant (1996b) has argued that if alcohol abuse and dependence merely reflected different cutpoints on a single underlying continuum of problems, then abuse—purportedly the milder of the two disorders—should be the more prevalent. In fact, the opposite is true: dependence is more prevalent than abuse (Grant et al., 1994b). Although in part a reflection of the residual nature of the abuse classification, this also suggests that abuse and dependence do *not* reflect a single underlying continuum of problems, despite many similarities.

In view of the preceding, it is surprising that abusers in this study lay midway between dependent and unaffected drinkers on *all* consumption measures—both those that reflect heavy drinking episodes (e.g., frequency of heavy drinking and intoxication) and those that might be considered more reflective of chronic and/or relief drinking (e.g., overall frequency of drinking and prevalence of daily/near daily and morning drinking). This finding suggests that in terms of drinking *behavior,* abuse is a milder but not essentially different disorder than dependence, and given the distinct nature of abuse and dependence in terms of symptom arrays and natural history, this argues for the wisdom of omitting consumption from the criteria for these disorders. (This finding also may reflect the fact that two-thirds of the individuals classified with dependence additionally met the criteria for abuse. Differences between the drinking behavior of dependent individuals with and without abuse would be a fruitful topic for future research.)

Based on the mean frequencies of drinking 5+ drinks and of intoxication, this study's results suggest that abusers experienced intoxication on about one-third of their heavy drinking days, whereas those with dependence experienced intoxication on closer to half of their heavy drinking days. Contrary to Jellinek's argument that loss of control is the critical element distinguishing alcoholics from habitual excessive drinkers, these data could be interpreted as more evidence that the difference between the two disorders is merely one of degree. One factor that might help to reconcile this apparent loss of control among abusers with Jellinek's typology is drinking motivation, an aspect of consumption that is rarely queried in alcohol studies. Abusers, who are heavily composed of young men, might drink either with the intention of becoming intoxicated or

without making any attempt to prevent it, whereas individuals with dependence might become intoxicated because they are unable to stop drinking when they want or intend to do so. Again, this suggests that factors other than consumption patterns per se are more critical indicators of any essential differences that may distinguish alcohol abuse from dependence.

As indicated by the relatively small amount of significant variation in the ratios of the consumption measures across demographic subgroups of the population, this study found that consumption showed few interactions with gender, age and race/ethnicity in its association with alcohol use disorders. Put another way, abusers and individuals with alcohol dependence demonstrated most of the same demographic differentials in drinking behavior as did those without an AUD. Thus, even in the face of alcohol-related problems that may include loss of control, it would appear that the cultural, physiological and normative influences that underlie these demographic differentials continue to exert an effect on drinking patterns.

The demographic variations that were observed in the ratios expressing associations between consumption and alcohol use disorders indicated that the more normative a drinking behavior is within a population subgroup, the less strongly it is associated with the probability of alcohol abuse or dependence. For example, the consumption of a large proportion of ethanol in the form of beer was more common among individuals with abuse or dependence than among those without an AUD, presumably because beer is the least expensive source of ethanol and thus appealing to those whose primary goal is intoxication. Yet the association between beer drinking and alcohol use disorders was weaker for men than for women, reflecting the fact that beer is the overwhelming beverage of choice among men irrespective of whether or not they suffer from alcohol abuse or dependence. Similarly, the ratios expressing the associations of weekday and daily or near daily drinking with alcohol dependence decreased with age in this study, reflecting the fact that these temporal drinking patterns are increasingly prevalent with advancing age irrespective of the presence of an AUD. Variations such as these indicate why it would be difficult to use any set pattern or level of consumption as a criterion for alcohol use disorders.

This study has provided an unusually representative picture of consumption patterns among individuals with and without alcohol use disorders, but because of the amount of information presented, the analysis was restricted to comparisons of mean consumption levels. It would be useful in future studies to examine the *distribution* of consumption measures across diagnostic categories, looking, for example, at the degree of overlap among the distributions for the three categories of no AUD, abuse only and dependence. The characteristics of individuals in these overlapping areas are of considerable interest, because they might identify risk or protective factors that would help to explain why some drinkers develop alcohol use disorders when others with similar volumes or even patterns of consumption do not.

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CHAPTER 2
ALCOHOL DEPENDENCE AND ABUSE

Prevalence of DSM–IV Alcohol Abuse and Dependence: United States, 1992

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For the first time, results are presented on the prevalence of alcohol abuse and dependence in the United States in 1992, according to the most recent psychiatric classification of alcohol-related disorders from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition *(DSM–IV). More than 7 percent of adults surveyed met DSM–IV criteria for 1-year alcohol abuse, alcohol dependence, or both. Males were almost three times more likely than females to meet the criteria for alcohol abuse and/or dependence; however, the male-to-female ratio was lowest in the youngest age group among nonblack respondents, suggesting that the rates of these disorders in nonblack females may be catching up.*

This Epidemiologic Bulletin presents prevalence and population estimates of alcohol abuse and dependence in the United States for the year 1992. The definitions for these alcohol-related disorders were based on the most recent criteria from the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM–IV) (American Psychiatric Association [APA] 1994). Prevalence defines the weighted percentage of respondents classified with a DSM–IV diagnosis, and population estimate refers to the number of people in the United States receiving a DSM–IV diagnosis of alcohol abuse, alcohol dependence, or both. One-year prevalence estimates were derived from self-reports of symptoms of alcohol abuse and dependence on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES). The figures presented in this bulletin are the first estimates of DSM–IV alcohol abuse and dependence to be reported at the national level.

BACKGROUND AND PROCEDURES

Prevalence and population estimates of alcohol abuse and dependence were based on the 1992 NLAES, a nationwide household survey sponsored by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). Field work for the study was conducted by the Bureau of the Census. For the NLAES, direct face-to-face interviews were conducted with 42,862 respondents, 18 years of age and older, in the contiguous United States and the District of Columbia. The household-response rate for the NLAES was 91.9 percent, and the person-response rate was 97.4 percent.

The NLAES featured a complex multistage design (Massey et al. 1989). Primary sampling units (PSU's)[1] were stratified according to sociodemographic criteria and were selected with probability proportional to size. Approximately 2,000 PSU's were in the 1992 NLAES sample, 52 of which were self-representing—that is, selected with certainty. Within PSU's, geographically defined secondary sampling units, referred to as segments, were selected systematically for each sample. Oversampling of the black population was accomplished at this stage of sample selection. The decision to oversample the black population was based on the higher observed rates of alcohol-related disease (i.e., liver cirrhosis) in this group.

Segments then were divided into clusters of approximately four to eight housing units, and all occupied housing units were included in the NLAES. Within each household, one randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18–29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavy drinking population subgroup. This subgroup of young adults was sampled at a ratio of 2.25 percent to 1.00.

Because of the complex survey design of the NLAES, variance estimation procedures that assume a

[1] For a definition of this term and others used in this article, see glossary on p. 52.

Reprinted from Alcohol Health & Research World (*now titled* Alcohol Research & Health), *Volume 18, Number 3, pp. 243–248 1994, National Institute on Alcohol Abuse and Alcoholism.*

simple random sample cannot be employed. Research has shown that clustering and stratification of the NLAES sample may result in standard errors much larger than those that would be obtained with a simple random sample of equal size. To take into account the NLAES sample design, all standard errors of the prevalence estimates presented here were generated using SUDAAN (Research Triangle Institute 1994), a software program that uses appropriate statistical techniques to adjust for sample design characteristics.

DSM–IV CLASSIFICATION

The 1992 NLAES included an extensive list of questions designed to assess the presence of symptoms of alcohol abuse and dependence during the 12-month period preceding the interview. We developed these questions, in part, to operationalize the DSM–IV criteria for alcohol-related disorders. Although the DSM–IV classification was not published until the second quarter of 1994, all of the specific diagnostic criteria for alcohol abuse and dependence were known prior to beginning the NLAES interviews (APA 1991) and therefore were incorporated into the final survey instrument in their entirety. What was not known prior to taking the NLAES into the field was which of the diagnostic criteria would be relegated to abuse and dependence categories. However, once all relevant DSM–IV diagnostic criteria were incorporated into the NLAES, computer algorithms could be designed to represent accurately the placement of the criteria within abuse and dependence categories consistent with the finalized diagnostic criteria. Correspondence of the DSM–IV criteria with individual NLAES questions is shown in the sidebar.

According to DSM–IV, a diagnosis of alcohol abuse requires that a person exhibit a maladaptive pattern of alcohol use, leading to clinically significant impairment or distress, as demonstrated by at least one of the following: (1) continued use despite a social or interpersonal problem caused or exacerbated by the effects of drinking; (2) recurrent drinking in situations in which alcohol use is physically hazardous; (3) recurrent drinking resulting in a failure to fulfill major role obligations; or (4) recurrent alcohol-related legal problems. A diagnosis of alcohol dependence requires that a person meet at least three of seven criteria defined for dependence in any 12-month period (see sidebar).

In the *Diagnostic Statistical Manual of Mental Disorders, Third Edition, Revised* (DSM–III–R) (APA 1987), the duration criteria associated with abuse and dependence specify that some of the symptoms of the

GLOSSARY

Cluster sampling: A sampling method in which each sampling unit is a collection of persons, units, or elements of interest.

Oversampling: A sampling technique used to bolster the numbers within low-prevalence subgroups of the population in order to achieve adequate numbers suitable for statistical analysis.

Primary sampling units: Comprehensive, mutually exclusive categories, consisting of all persons, units, or elements of interest, usually identified in the first stage of a multistage sampling design. For example, *primary sampling units* can consist of geographic regions of the United States (e.g., cities) defined in terms of sociodemographic criteria.

Selected with probability: This typically refers to the selection of sampling units according to predetermined probabilities. For example, *primary sampling units* may be selected that have probabilities proportional to size.

Selected with certainty: This typically refers to the selection of sampling units with a probability of 1.0. For example, if *primary sampling units* are designated to be selected in proportion to their size, it follows that the largest of the units will be selected with certainty.

Simple random sample: A method of drawing samples such that each person, element, or unit has an equal probability of being selected.

Stratification: The classification of all persons, units, or elements of interest into comprehensive, mutually exclusive categories.

Variance estimation procedures: A technique that allows estimation of the amount of dispersion around a measure of data, such as a percentage or mean.

Weighted percentage: Percentages that have been adjusted to account for all aspects of the sample design (e.g., differential rates of selection, *oversampling).*

disorder must occur continuously during a month or repeatedly over a longer period of time. Unlike that of the DSM–III–R, the duration criteria of the DSM–IV abuse and dependence categories are associated with the individual diagnostic criteria and not the categories of abuse and dependence per se. The duration criterion for both alcohol-related disorders defines the repetitiveness with which certain diagnostic criteria must occur during a 12-month period for these criteria to be considered positive. As shown in the sidebar, the duration criteria for abuse and dependence are not associated with all diagnostic criteria and are defined by

Table 1. Prevalence and Population Estimates[1] of DSM–IV Alcohol Abuse and Dependence by Age, Sex, and Ethnicity: United States, 1992

	Alcohol Abuse Only			Alcohol Dependence Only			Alcohol Dependence With Abuse			Total Alcohol Abuse and Dependence		
Ethnicity/ Sex/Age	Prevalence (%)	S.E.	Population Estimate	Prevalence (%)	S.E.	Population Estimate	Prevalence (%)	S.E.	Population Estimate	Prevalence (%)	S.E.	Population Estimate
Nonblack Males	4.93	(0.21)	3,928	2.10	(0.14)	1,673	4.30	(0.21)	3,423	11.33	(0.34)	9,024
18–29	10.02	(0.58)	2,031	3.91	(0.34)	792	9.55	(0.55)	1,935	23.48	(0.84)	4,758
30–44	4.81	(0.30)	1,308	2.01	(0.23)	546	4.07	(0.32)	1,107	10.89	(0.47)	2,961
45–64	2.51	(0.31)	521	1.44	(0.22)	300	1.66	(0.25)	346	5.61	(0.44)	1,167
65+	0.60	(0.19)	69	0.31	(0.09)	36	0.30	(0.09)	35	1.21	(0.23)	140
Black Males	2.50	(0.43)	237	2.48	(0.39)	235	3.27	(0.42)	310	8.25	(0.72)	782
18–29	3.97	(0.92)	116	3.92	(0.99)	115	4.44	(1.01)	130	12.33	(1.70)	361
30–44	2.78	(0.78)	96	2.58	(0.65)	89	3.39	(0.71)	117	8.75	(1.21)	302
45–64	1.17	(0.58)	25	1.12	(0.35)	24	2.90	(0.72)	62	5.19	(0.97)	111
65+	0.00	(0.00)	0	0.74	(0.50)	7	0.08	(0.08)	1	0.82	(0.51)	8
Total Males	4.67	(0.19)	4,165	2.14	(0.13)	1,908	4.19	(0.19)	3,733	11.00	(0.32)	9,806
18–29	9.26	(0.52)	2,147	3.91	(0.34)	907	8.90	(0.50)	2,065	22.07	(0.77)	5,119
30–44	4.58	(0.28)	1,404	2.07	(0.21)	635	4.00	(0.29)	1,225	10.65	(0.45)	3,264
45–64	2.38	(0.28)	546	1.41	(0.20)	324	1.78	(0.24)	408	5.57	(0.41)	1,278
65+	0.55	(0.18)	69	0.34	(0.09)	43	0.29	(0.08)	36	1.18	(0.22)	148
Nonblack Females	1.62	(0.11)	1,379	1.20	(0.10)	1,019	1.43	(0.10)	1,216	4.25	(0.20)	3,614
18–29	4.29	(0.34)	851	2.60	(0.28)	515	4.10	(0.35)	814	10.99	(0.64)	2,180
30–44	1.58	(0.17)	428	1.23	(0.15)	335	1.13	(0.15)	307	3.94	(0.27)	1,070
45–64	0.42	(0.10)	93	0.68	(0.14)	149	0.35	(0.07)	75	1.45	(0.19)	317
65+	0.04	(0.03)	7	0.13	(0.06)	20	0.12	(0.07)	20	0.29	(0.09)	47
Black Females	0.71	(0.16)	84	1.30	(0.21)	153	0.87	(0.18)	102	2.88	(0.32)	339
18–29	1.24	(0.42)	43	1.70	(0.41)	59	0.38	(0.15)	13	3.32	(0.60)	115
30–44	0.98	(0.30)	40	1.18	(0.33)	48	2.02	(0.49)	83	4.18	(0.65)	171
45–64	0.02	(0.02)	0	1.70	(0.52)	45	0.20	(0.12)	5	1.92	(0.54)	50
65+	0.00	(0.00)	0	0.00	(0.00)	0	0.00	(0.00)	0	0.00	(0.00)	0
Total Females	1.51	(0.10)	1,463	1.21	(0.09)	1,172	1.36	(0.09)	1,318	4.08	(0.18)	3,953
18–29	3.83	(0.30)	894	2.46	(0.25)	574	3.55	(0.30)	827	9.84	(0.56)	2,295
30–44	1.50	(0.15)	469	1.23	(0.14)	383	1.25	(0.15)	391	3.98	(0.25)	1,243
45–64	0.38	(0.09)	93	0.79	(0.14)	194	0.33	(0.07)	81	1.50	(0.18)	368
65+	0.04	(0.03)	7	0.12	(0.05)	20	0.11	(0.06)	20	0.27	(0.09)	47
Total	3.03	(0.11)	5,628	1.66	(0.08)	3,080	2.72	(0.11)	5,052	7.41	(0.20)	13,760
18–29	6.54	(0.33)	3,041	3.18	(0.21)	1,481	6.22	(0.30)	2,893	15.94	(0.53)	7,415
30–44	3.02	(0.16)	1,873	1.64	(0.13)	1,018	2.61	(0.17)	1,615	7.27	(0.26)	4,506
45–64	1.35	(0.15)	639	1.09	(0.12)	518	1.03	(0.12)	488	3.47	(0.22)	1,645
65+	0.25	(0.08)	75	0.21	(0.05)	63	0.18	(0.05)	55	0.64	(0.10)	193

[1]All population estimates are in thousands.

NOTE: Components may not always sum to the totals displayed in the table because of rounding.

qualifiers, such as "recurrent," "often," and "persistent" desire or unsuccessful "efforts."

To satisfy the duration criterion for abuse, a respondent must have experienced two or more symptoms of an abuse criterion associated with a duration qualifier at least once during the past year, or alternatively, at least one symptom of that diagnostic criterion must have occurred at least twice during the past year. For those abuse criteria not associated with a duration qualifier, a related symptom need only have occurred once in the past year to be counted as positive toward an abuse diagnosis.

Similarly, to satisfy the duration criterion for dependence, at least one symptom of a diagnostic criterion associated with a duration qualifier must have occurred at least twice over the course of the year preceding the interview, or alternatively, two or more symptoms related to these criteria must have occurred at least once during the same time period.

The diagnosis of dependence presented in this bulletin was qualified further in an important way. Because the withdrawal criterion of alcohol dependence is defined in DSM–IV as a withdrawal syndrome (i.e., a cluster of symptoms), at least two symptoms of withdrawal, which met the duration criterion, had to occur during the past 12 months. It should be noted, however, that withdrawal is not required for a DSM–IV diagnosis of dependence. The DSM–IV diagnostic category for dependence could be specified further by evidence of physiological dependence (i.e., evidence of either tolerance or withdrawal, including drinking to relieve or avoid withdrawal) or no physiological dependence (i.e., no evidence of tolerance and withdrawal).

SUMMARY OF FINDINGS

Table 1 presents the 1-year prevalence rates, standard errors, and population estimates of DSM–IV alcohol abuse and dependence by age, sex, and ethnicity. The DSM–IV abuse and dependence groups formed by the 1992 NLAES were mutually exclusive. Respondents classified as alcohol abusers did not meet criteria for alcohol dependence; however, those who met criteria for dependence were classified as to whether they also met the criteria for alcohol abuse. Hierarchically, the DSM–IV does not allow a diagnosis of abuse in the presence of dependence, and thus all respondents classified in this bulletin as alcohol dependent with and without abuse would receive only a formal diagnosis of dependence. The purpose of disaggregating respondents classified as dependent with and without abuse merely was to provide more detail concerning the diagnostic status of respondents classified as alcohol dependent.

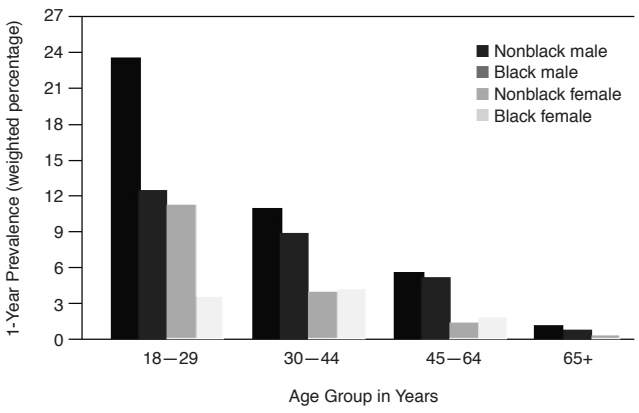
Table 2. Ratios of Prevalence of DSM–IV Alcohol Abuse and Dependence by Age and Ethnicity: United States, 1992

Ethnicity/Age (years)	Male-to-Female Ratio
Nonblack	
18–29	2.1
30–44	2.8
45–64	3.9
65+	4.2
Black	
18–29	3.7
30–44	2.1
45–64	2.7
65+	—
Total	
18–29	2.2
30–44	2.7
45–64	3.7
65+	4.4

NOTE: Male-to-female ratio is undefined for blacks because of the female rate of 0.0.

The 1-year prevalence of combined alcohol abuse and dependence in the NLAES sample was 7.41 percent, representing 13,760,000 Americans (table 1). Slightly more respondents were classified as alcohol dependent (4.38 percent) than as abusing alcohol (3.03 percent). Among those respondents meeting DSM–IV diagnostic criteria for dependence, the greatest proportion also met criteria for alcohol abuse. The predomi-

Figure 1. Prevalence of DSM-IV Alcohol Abuse and Dependence by Age, Sex, and Ethnicity: United States, 1992



nance of the dual abuse-dependence diagnosis was generally consistent for each age, sex, and ethnic subgroup of the population. The majority of respondents with alcohol dependence diagnoses also were classified with physiological dependence (4.25 percent) in contrast to no physiological dependence (0.13 percent) (data not shown).

One-year prevalence of alcohol abuse and dependence combined was much greater among males (11.00 percent) than females (4.08 percent). Prevalence also was greater among nonblacks (7.68 percent) than among blacks (5.28 percent) (data not shown). Rates for nonblack males and females exceeded the rates for their black counterparts by 27.18 percent and 32.23 percent, respectively.

Prevalence rates of alcohol abuse and dependence were higher among respondents under 45 years than among those 45 years or older, regardless of sex or

1992 National Longitudinal Alcohol Epidemiologic Survey:

DSM–IV Alcohol Abuse and Dependence Diagnostic Criteria and Associated Questionnaire Items

Diagnostic Criteria for Alcohol Abuse

Diagnostic Criterion: Continued to drink despite social or interpersonal problem caused by drinking

Questionnaire Item:

- Continue to drink even though you knew it was causing you trouble with your family or friends.

Diagnostic Criterion: Recurrent drinking in situations where alcohol use is physically hazardous*

Questionnaire Items:

- Drive a car, motorcycle, truck, boat, or other vehicle after having too much to drink.
- Get into a situation while drinking or after drinking that increased your chances of getting hurt—like swimming, using machinery, or walking in a dangerous area or around heavy traffic.

Diagnostic Criterion: Recurrent alcohol-related legal problems*

Questionnaire Item:

- Get arrested or held at a police station because of your drinking.

Diagnostic Criterion: Recurrent drinking resulting in failure to fulfill major role obligations at work, school, or home*

Questionnaire Items:

- Get drunk or have a hangover when you were supposed to be doing something important—like being at work, school, or taking care of your home or family.
- Get drunk or have a hangover when you were actually doing something important—like being at work, school, or taking care of your home or family.

Diagnostic Criteria for Alcohol Dependence[1]

Diagnostic Criterion: Tolerance[2]

Questionnaire Items:

- Find that your usual number of drinks had much less effect on you than it once did.
- Find that you had to drink much more than you once did to get the effect you wanted.

Diagnostic Criterion: Withdrawal syndrome[3] or withdrawal relief/avoidance

Questionnaire Items:

- Have any of the following experiences happened when the effects of alcohol were wearing off [Pause], several hours after drinking [Pause], or the morning after drinking? For example, did you *ever*:
  - (a) Have trouble falling asleep or staying asleep.
  - (b) Find yourself shaking when the effects of alcohol were wearing off.
  - (c) Feel depressed, irritable, or nervous.
  - (d) Feel sick to your stomach or vomit when the effects of alcohol were wearing off.
  - (e) Have a very bad headache.
  - (f) Find yourself sweating or your heart beating fast when the effects of alcohol were wearing off.
  - (g) See, feel, or hear things that were not really there.
  - (h) Have fits or seizures when the effects of alcohol were wearing off.
- Take a drink to get over any of the bad aftereffects of drinking.
- Take a drug other than aspirin, Tylenol™, or Advil™ to keep from having a hangover or to get over the bad aftereffects of drinking.
- Take a drink to keep from having a hangover or to make yourself feel better when you had one.

Diagnostic Criterion: Drinking larger amounts over a longer period of time than intended*

Questionnaire Items:

- Start drinking even though you decided not to or promised yourself you would not.
- End up drinking more than you meant to.
- Keep on drinking for a much longer period of time than you had intended to.

Diagnostic Criterion: Persistent desire or unsuccessful efforts to cut down or control drinking*

Questionnaire Items:

- Want to stop or cut down on your drinking.
- Try to stop or cut down on your drinking but found you could not do it.

Diagnostic Criterion: Important social, occupational, or recreational activities given up or reduced in favor of drinking

Questionnaire Items:

- Give up or cut down on activities that were important to you in order to drink—like work, school, or associating with friends or relatives.
- Give up or cut down on activities that you were interested in or that gave you pleasure in order to drink.

Diagnostic Criterion: Great deal of time spent in activities to obtain alcohol, to drink, or to recover from its effects

Questionnaire Items:

- Spend so much time drinking that you had little time for anything else.
- Spend a lot of time being sick or with a hangover from drinking.
- Spend a lot of time making sure that you always had alcohol available.

Diagnostic Criterion: Continued to drink despite knowledge of having a persistent or recurrent physical or psychological problem caused or exacerbated by drinking

Questionnaire Items:

- Continued to drink even though you knew it was making you feel depressed, uninterested in things, or suspicious or distrustful of other

*In order for the criterion to be positive, either: (a) two or more symptoms must have occurred at least once, or (b) one or more symptoms must have occurred at least twice during the past year.

[1]Dependence diagnoses can be specified with physiological dependence (i.e., evidence of either tolerance or withdrawal) or without physiological dependence (i.e., no evidence of either tolerance or withdrawal).

[2]Tolerance need have occurred only once during the past year for the criterion to be positive.

[3]Two or more symptoms of withdrawal must have occurred at least twice during the past year for the criterion to be positive.

ethnicity (table 1). For males, the prevalence rate in the youngest age group (18 to 29 years) was 22.07 percent. The rate decreased approximately 50 percent among 30-to-44-year-old males (10.65) and was reduced to 1.18 among those 65 years and older. For females, the highest prevalence rate also was found in the youngest age group (9.84 percent), with the rates falling steadily to 0.27 percent in females 65 years and older. Possible explanations for the decline in alcohol abuse and dependence rates with age may include faulty recall accompanying increasing age, lower survival rates among alcoholics, and various response styles. Alternately, the age gradient may reflect a true cohort effect; that is, that alcohol abuse and dependence are more prevalent among the younger generation of Americans.

Ethnic groups showed striking patterns of age-related 1-year prevalence rates of alcohol abuse and dependence (figure 1). Among the youngest males, the prevalence rate in nonblacks (23.48) was 1.9 times greater than in blacks (12.33). In the remaining age groups, the rates for nonblacks and blacks converge, with a slight predominance among nonblacks. The patterns for nonblack and black females were similar to those of males, except the black female rate exceeded the nonblack female rate among 30-to-64-year-old groups.

Although alcohol abuse and dependence were greater among males than among females, there was evidence of convergence of the rates between the sexes in the youngest age groups (table 2). The male-to-female ratios (i.e., male rate divided by the female rate) were lowest in the 18-to-29-year-old group. However, when the male-to-female ratio was examined separately for each ethnic group, it was clear that the rate converged among the youngest age groups only among nonblacks. In contrast, the male-to-female ratio was much lower among blacks in the 30-to-64-year-old groups. Thus, alcohol abuse and dependence were more prevalent in the younger age groups, particularly among nonblack females.

DISCUSSION

More than 7 percent of adults surveyed met DSM–IV criteria for 1-year alcohol abuse, alcohol dependence, or both. Males were almost three times more likely than females to meet the criteria for alcohol abuse and/or dependence. However, that the male-to-female ratio is lowest in the youngest age group among nonblacks suggests that nonblack females may be catching up. This phenomenon does not generalize to black females because the male-to-female ratios in blacks were shown to decrease as a function of age. Possible reasons for the greater discrepancy between male and female rates of alcohol abuse and dependence among younger blacks compared with younger nonblacks include differential age-related role responsibilities or differences in perceived social acceptability of drinking per se between the ethnic groups in the general population.

The overall prevalence estimates and corresponding population estimates of alcohol abuse and dependence presented here do not differ greatly from those for the years 1984 (Williams et al. 1989) or 1988 (Grant et al. 1991), even though these earlier figures were based on diagnostic criteria from the DSM–III (APA 1980) and the DSM–III–R, respectively. The prevalence of DSM–III alcohol abuse and dependence reported by the 1984 National Survey on Alcohol Use was 8.58 percent for the total sample, with an associated population estimate of 15,100,000. The corresponding DSM–III–R prevalence rate for the 1988 National Health Interview Survey was 8.63 percent, representing 15,295,000 Americans. Although these figures are nearly identical to the prevalence of DSM–IV alcohol-related disorders found in the 1992 NLAES sample, caution must be exercised in assuming the stability of these rates between 1984 and 1992. Because definitions of disorders differed among the three surveys, no conclusions can be made concerning the rates of alcohol abuse and dependence over time.

Although the purpose of this Epidemiologic Bulletin is to present the national rates of alcohol abuse and dependence according to the most recent psychiatric classification of alcohol-related disorders (i.e., the DSM–IV), provisions also were made within the NLAES to measure alcohol abuse and dependence by historic diagnostic classifications (i.e., the DSM–III and DSM–III–R). Representation of multiple definitions of alcohol-related disorders will facilitate direct comparisons between the NLAES DSM–III estimates and the DSM–III estimates of the 1984 National Survey on Alcohol Use and between the NLAES DSM–III–R estimates and the DSM–III–R estimates derived from the 1988 National Health Interview Survey. It remains to be seen if trends exist over time in alcohol abuse and dependence. Such trends will become evident once the diagnostic definitions across these surveys are equalized. To this end, a series of reports focusing on trends in alcohol-related disorders between the years 1984 and 1992 currently are being prepared by NIAAA. These reports will present, for the first time, changes in the rates for alcohol abuse and dependence over the last decade.

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Prevalence and Correlates of Alcohol Use and DSM–IV Alcohol Dependence in the United States: Results of the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant

Objective: *The purpose of this study was to present updated estimates of the prevalence of, and to examine the correlates of, alcohol use and DSM-IV alcohol dependence in a representative sample of the U.S. population.* Method: *This study was based on the National Institute on Alcohol Abuse and Alcoholism's National Longitudinal Alcohol Epidemiologic Survey (NLAES), a representative sample (*N = *42,862) of the United States population aged 18 years and older.* Results: *The prevalence of lifetime alcohol use was 66.0%, with 44.4% of the respondents reporting alcohol use during the past 12 months. Lifetime and 12-month prevalences of alcohol dependence were estimated at 13.3% and 4.4%, respectively. Men were significantly more likely than women to use alcohol, and alcohol use and dependence were much more common among cohorts born after Prohibition and after World War II. Members of the youngest cohorts, between the ages of 18 and 24 years at the time of the interview, were more likely to use drugs, to become dependent and to persist in dependence compared to the older cohorts. In addition, the conditional probability of dependence among users was greatest in Cohort 1 (born between 1968 and 1974) after early adolescence compared to Cohort 2 (born between 1958 and 1967), despite the finding that the probability of lifetime use was lower in Cohort 1 compared to Cohort 2. The sociodemographic correlates of first use, onset of dependence and persistence of dependence varied as a function of the stage of progression.* Conclusions: *Implications of these findings are discussed in terms of changes over time in drinking patterns, dependence liability and vulnerability among recent alcohol users.*

Although the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the National Institute on Drug Abuse and, more recently, the Substance Abuse and Mental Health Administration have conducted national surveys to monitor alcohol use prevalence and trends since the early 1960s, very few of these surveys have measured psychiatric syndromes that constitute alcohol dependence (Department of Health and Human Services, 1993; Johnston et al., 1992). Estimates of alcohol use alone are not informative in determining the magnitude and characteristics of that subgroup of alcohol users who are experiencing serious alcohol-related problems that require treatment or in determining the critical relationship between alcohol use and dependence.

Among those national surveys that have included provisions for the measurement of alcohol use and dependence are three NIAAA-sponsored surveys: the 1984 Seventh Survey on Alcohol Use (Williams et al., 1989), the 1988 Alcohol Supplement of the National Health Interview Survey (Grant et al., 1991) and, most recently, the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES)(Grant et al., 1994a). Prior to the fielding of the most recent NLAES by NIAAA, the National Institute on Mental Health sponsored two surveys that have reported national estimates of the prevalence of alcohol use and dependence. The first was the Epidemiologic Catchment Area (ECA) Survey (Robins et al., 1991), in which 18,571 respondents aged 18 years and older were interviewed in a series of five community-based epidemiologic studies in the early 1980s. The second was the National Comorbidity Survey (NCS), a national probability sample of 8,098 respondents, aged 15 to 54, conducted in 1991 (Kessler et al., 1994). However, both surveys had methodological problems that limited their ability to examine the relationship between alcohol use and dependence. The five community-based surveys underlying the ECA were not nationally representative of the U.S. adult population. Further, the ECA estimates are now over a decade old. The representativeness and precision of the NCS can be called into question because of its exclusion of adults 55 years and older and its rel-

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atively small sample size, respectively. Estimates of the prevalence of alcohol dependence in both surveys also used diagnostic classifications no longer in use, namely the Diagnostic and Statistical Manual of Mental Disorders–Third Edition (DSM-III) (American Psychiatric Association, 1980) used in the ECA and the DSM-III-Revised (DSM-III-R) (American Psychiatric Association, 1987) used in the NCS.

The purpose of the present article is to present updated estimates of the prevalence of, and to examine the demographic correlates of, alcohol use and dependence and their relationship in a representative sample of the U.S. population based on the NLAES (Grant et al., 1994b; Massay et al., 1989). The NLAES was designed to overcome many of the methodological problems inherent in the previous population surveys (i.e., the ECA and NCS). First, the diagnoses were based on the most current psychiatric classification, the DSM-IV (American Psychiatric Association, 1994). Second, unlike the ECA, the NLAES utilized a nationally representative sample of the United States adult population and, unlike the NCS, it included respondents aged 55 years and older. Compared to the NCS, the larger sample size of the NLAES, 42,862 respondents, enabled both detailed and precise estimation of the prevalence of alcohol use and dependence within important sociodemographic subgroups of the population. One of the major advances introduced with the NLAES was the more reliable measurement of alcohol dependence appearing on the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS) (Grant and Hasin, 1992). The AUDADIS was the first psychiatric interview of its kind to undergo a separate test-retest study in that population for which it was designed, that is, the general population.

METHOD

Sample

This study was based on data from the NLAES, a national probability survey sponsored by the NIAAA. Fieldwork for the NLAES was conducted by the United States Bureau of the Census. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous states, including the District of Columbia. The household response rate for this representative sample of the United States population was 91.9% and the sample person response rate was 97.4%.

The NLAES featured a complex multistage design. Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probability proportional to their population size. From a sampling frame of approximately 2,000 PSUs, 198 were selected for inclusion in the NLAES sample, including 52 which were self-representing—that is, selected with certainty. Because PSUs were selected proportional to their size, the largest 52 PSUs in the United States were selected into the sample with certainty (probability of 1.0) and were referred to as self-representing PSUs. Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically for sample. Oversampling of the black population was accomplished at this stage of sample selection to secure adequate numbers for analytic purposes. Within each household, one randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18 to 29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavier drinking population subgroup. This subgroup of young adults was randomly sampled at a ratio of 2.25:1.00.

Alcohol use and alcohol dependence assessment

Measures of alcohol use and dependence were derived from the AUDADIS, a fully structured diagnostic interview designed to be administered by trained interviewers that were not clinicians. In the AUDADIS, 12-month alcohol users included all respondents who had consumed 12 or more drinks during the year preceding the interview. Lifetime alcohol use was defined as drinking at least 12 drinks of alcohol in any 1 year of the respondent's life. Lifetime alcohol users, by definition, included all 12-month alcohol users in addition to former drinkers.

The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV criteria for alcohol dependence. These questions are described in detail elsewhere (Grant et al., 1994a). Although the DSM-IV was not published until 1994, the specific diagnostic criteria of interest were known prior to the conduct of NLAES (American Psychiatric Association, 1991) and were, therefore, incorporated in their entirety within the AUDADIS.

Consistent with the DSM-IV, AUDADIS diagnoses of alcohol dependence require that in any 1 year a respondent meet at least three of the following seven criteria defined for dependence: (1) tolerance; (2) withdrawal or avoidance of withdrawal; (3) persistent

desire or unsuccessful attempts to cut down or stop drinking; (4) spending much time drinking, obtaining alcohol, or recovering from its effects; (5) giving up or reducing occupational, social or recreational activities in favor of drinking; (6) impaired control over drinking; and (7) continuing to drink despite a physical or psychological problem caused or exacerbated by drinking.

The AUDADIS diagnoses of 12-month alcohol dependence satisfied both the clustering and duration criteria of the DSM-IV classification. Twelve-month diagnoses of alcohol dependence required the occurrence of at least three dependence criteria in the year preceding the interview, and the duration qualifiers associated with some dependence criteria must have been satisfied. The duration qualifiers are defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often" and "persistent" appearing in the description of the diagnostic criteria. Diagnoses that pertained to the period prior to the past year were also measured as syndromes, or the clustering of three or more dependence criteria in the past, that occurred (1) most days for at least 1 month; (2) repeatedly for a few months or longer; or (3) around the same time. Respondents classified with a lifetime diagnosis of dependence encompassed all those who had ever experienced an episode of dependence in the past 12 months and/or prior to the past year, rather than those demonstrating the required number of symptoms of these disorders over the life course.

In a separate test-retest study conducted in the general population, reliability coefficients (kappas) associated with alcohol dependence were .79 for the past year and .73 for lifetime. Kappas for age of onset and use variables used in this study ranged from .70 to .84 (Grant et al., 1995).

Statistical analyses

The analyses presented in this article included the estimation of 12-month and lifetime prevalences of alcohol use and dependence, estimates of cohort-specific curves for cumulative probabilities of use and dependence and sociodemographic correlates of use and dependence. Prevalence estimates were stratified by sex based on the importance of sex differences in alcohol use and alcohol use disorders documented in the preponderance of past research (Department of Health and Human Services, 1993; Helzer et al., 1991; Kessler et al., 1994). Because of the multiple comparisons conducted in this study, the significance level for all tests was set at $p < .001$. Because of the complex survey design of the NLAES, variance estimation procedures that assume a simple random sample are not appropriate. To take into account the NLAES sample design, all standard errors of the prevalence estimates were generated using SUDAAN (Research Triangle Institute, 1995), a software program that uses Taylor series linearization to adjust for sample design characteristics.

Cumulative age-at-onset curves for alcohol use and alcohol dependence were generated using survival analysis methods (Lee, 1980) with individuals withdrawn from the risk of dependence at the time of their last reported use of alcohol. Prevalences were estimated within five cohorts for 5-year age intervals (e.g., cumulative probabilities of use by the ages 4, 9, 14, 19 and 24 years among respondents aged 18–24 years at the time of the interview). Cumulative probabilities for ages that exceeded the current ages of some cohort members (e.g., cumulative prevalence by age 24 years in the cohort currently between 18 and 24 years) were based on the at-risk subsample of the cohort at the beginning of the 5-year interval. Since the formula used to calculate the standard errors of these cumulative probabilities assumed a simple random sample, the standard errors in these analyses were also adjusted using SUDAAN estimates of the design effects on the simple probability of the outcome variable (i.e., use or dependence).

Associations between alcohol use and alcohol dependence and sociodemographic correlates were expressed in terms of odds ratios. Odds ratios and their 95% confidence intervals were derived from separate logistic regression analyses using the SUDAAN LOGISTIC program that also adjusted for the complex sampling design of the NLAES. In each logistic regression, a single demographic variable was treated as a predictor, with alcohol use or dependence serving as the outcome variable. An odds ratio of greater than 1.0 reflected a positive association between a sociodemographic and an outcome variable and was statistically significant if its 95% confidence intervals did not encompass the value of 1.0.

RESULTS

Prevalence of lifetime and 12-month use

Table 1 presents the lifetime and 12-month prevalence of alcohol use by sex and age. The overall prevalence of lifetime alcohol use was 66.0%, with 44.4% of the respondents reporting alcohol use during the last 12 months. Men were significantly more likely than women to have used alcohol both on a lifetime (78.3% vs 54.7%, $t = 40.1$, $p < .0001$) and 12-month basis

(55.8% vs 33.9%, $t < 36.9$, $p < .0001$). There was also an inverse relationship between 12-month use and age, regardless of sex. Rates for 12-month use among the three youngest age groups significantly exceeded those of the two oldest age groups (*t* tests between 3.7 and 31.7 for pairwise comparisons, all of them significant at $p < .001$).

For lifetime use, age, years at risk and cohort were confounded, requiring the disaggregation of age and cohort effects. Figure 1 presents the age of the respondent's first drink represented as curves for the age-specific cumulative probabilities of lifetime use for each of the five birth cohorts in the NLAES. Table 2 presents the associated cumulative probabilities along with their standard errors. Cohort 1 (born between 1968 and 1974) represents the youngest NLAES cohort, followed by the older respondents in Cohort 2 (born between 1958 and 1967), Cohort 3 (born between 1948 and 1957), Cohort 4 (born between 1938 and 1947) and Cohort 5 (born between 1894 and 1937). When examining Figure 1, it will be useful to recognize that the majority of respondents born in Cohort 5 went through early (9 to 14 years), middle (15 to 19 years) and late (20 to 24 years) adolescence and early adulthood (25 to 29 years) during Prohibition. Also respondents in Cohorts 4 and 5, born before, during or shortly after World War II, would have completed high school by the mid-1960s (i.e., before the widespread introduction of illicit drugs into the youth subculture). Cohort 3 went through early adolescence in the early years of the youth drug subculture, Cohort 2 at its height and Cohort 1 after the decline in drug use over the past decade.

The shape of the age of onset curves for each cohort were remarkably similar. For each cohort, the cumulative probability of alcohol use began to rise dramatically in early adolescence, rose sharply until late adolescence at which time the cumulative probability of use stabilized. By the end of late adolescence, 42.2% of Cohort 5, 59.1% of Cohort 4, 67.1% of Cohort 3, 72.2% of Cohort 2 and 74.6% of Cohort 1 had experiences with using alcohol.

There were no statistically significant differences between Cohort 1 and Cohort 2 in the cumulative probability of use for all age categories. However, all other intercohort differences were statistically significant, indicating a sharp rise in alcohol use from Cohort 5 to Cohorts 1 and 2 (*z* tests between 3.4 and 24.1 for pairwise comparisons, all of them significant at $p < .001$). With few exceptions, sex-specific cohort differences mirrored those observed in the total sample (Table 2). However, among men, there were no significant differences in the cumulative probability of use between cohorts during late adolescence or between Cohorts 2 and 3 beginning in late adolescence. For both sexes there were also no significant intercohort differences between Cohorts 3, 4 and 5 for the period of early adolescence. In general, the cumulative probability of use for each age range was consistently greater for men than women.

Prevalence of lifetime dependence

The results in Table 3 show that 13.3% of the respondents had a lifetime history of alcohol dependence, while 20.1% of lifetime alcohol users were classified with an alcohol dependence diagnosis. Men were significantly more likely to have a history of alcohol dependence (18.6% vs 8.4%, $t = 24.7$, $p < .0001$) and were also more likely than women to be dependent in the subsample of lifetime alcohol users (23.7% vs 15.4%, $t = 15.1$, $p < .0001$). Thus, the sex differences observed in lifetime dependence were due to the increased likelihood among men to both use alcohol and become dependent compared to women.

Table 3 also shows an age-cohort effect. In the total sample, respondents in Cohorts 4 and 5 were significantly less likely to have a lifetime history of alcohol dependence than those in the three youngest cohorts (*t* tests between 3.3 and 27.5 for pairwise comparisons, all of them significant at $p < .001$). For both men and women, respondents in Cohorts 4 and 5 were significantly less likely than the younger cohorts to have a lifetime history of dependence, and were also less likely than the younger cohorts to be dependent in the subsample

Table 1. Prevalence of lifetime and 12-month alcohol use by sex and age (mean ± SE)

	Male		Female		Total	
Age	Lifetime	12-month	Lifetime	12-month	Lifetime	12-month
Total	78.3 ± 0.42	55.8 ± 0.51	54.7 ± 0.56	33.9 ± 0.47	66.0 ± 0.41	44.4 ± 0.40
18-24	70.3 ± 1.19	61.3 ± 1.26	55.8 ± 1.27	41.4 ± 1.29	63.1 ± 0.96	51.4 ± 1.01
25-34	82.7 ± 0.72	65.7 ± 0.86	65.8 ± 0.78	42.4 ± 0.76	74.2 ± 0.56	53.9 ± 0.62
35-44	82.9 ± 0.73	59.2 ± 0.95	63.0 ± 0.90	39.2 ± 0.85	72.9 ± 0.64	49.1 ± 0.67
45-54	81.6 ± 0.98	54.3 ± 1.25	56.2 ± 1.10	33.0 ± 1.05	68.7 ± 0.79	43.4 ± 0.86
55+	72.8 ± 0.82	40.9 ± 0.89	39.1 ± 0.80	20.6 ± 0.61	53.9 ± 0.69	29.5 ± 0.58

of lifetime users (t tests between 4.4 and 23.1 for pairwise comparisons, all of them significant at $p < .0001$).

A disaggregation of the age and cohort effects observed in Table 3 is shown in Figure 2 where age of onset of alcohol dependence is presented in curves for age-specific cumulative conditional probabilities of lifetime alcohol dependence among lifetime alcohol users. There was a consistent trend for each successive cohort to have much greater prevalence of dependence among users. In fact, after early adolescence, all intercohort differences were statistically significant for each age range (z tests between 4.9 and 28.3 for pairwise comparisons, all of them significant at $p < .0001$).

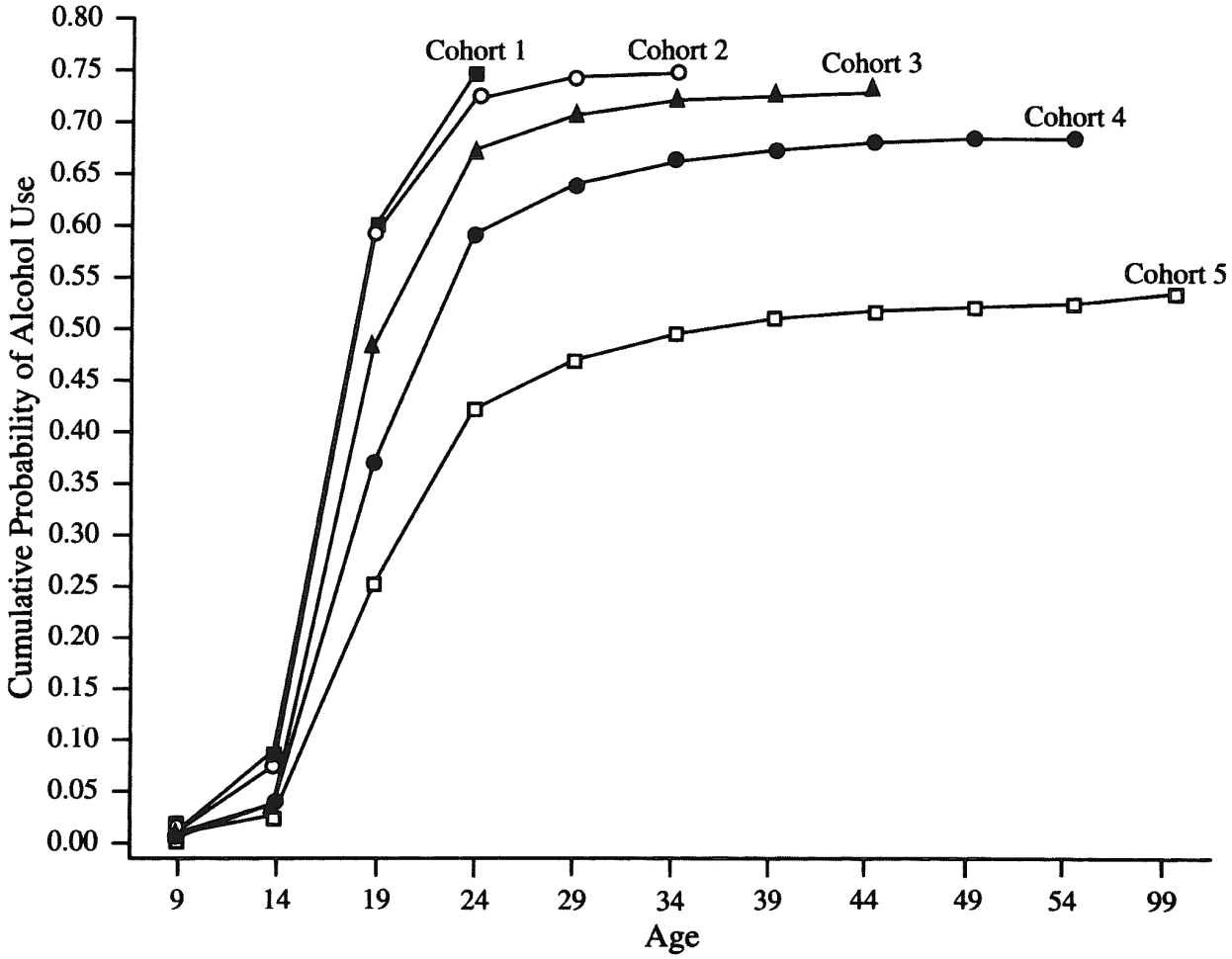
As can be seen in Table 4, only 2.6% and 7.1% of the respondents in Cohorts 5 and 4, respectively, who reported using alcohol had a history of dependence by age 24 compared to 11.9%, 19.4%, and 43.6% of Cohorts 3, 2 and 1, respectively. It is of interest that the cumulative probability of dependence among users was greater in Cohort 1 than in Cohort 2 for both middle adolescence ($z = 9.9$, $p < .0001$) and late adolescence ($z = 14.8$, $p < .0001$).

In general, the probability of alcohol dependence was greater among men than among women, and all intercohort differences noted for the total sample were found when cohort differences were examined for each sex (z tests between 3.4 and 17.8 for pairwise comparisons, all of them significant at $p < .001$).

Prevalence of 12-month dependence

Table 5 shows that 4.4% of the NLAES respondents were alcohol dependent during the 12 months preceding the interview, representing 9.9% of the 12-month users and 20.4% of the respondents with a history of alcohol dependence prior to the past year. The overall prevalence of 12-month dependence was significantly

Figure 1. Cumulative probability of alcohol use, by cohort



greater for men than women (6.3% vs 2.6%, $t = 14.4$, $p < .0001$). This was because men are more likely than women to use alcohol at some time in their lives, but are also more likely than women to become dependent once they use and more likely than women to persist in their dependence once it has developed. This observation is supported by the finding of sex differences in 12-month dependence among the subsample of 12-month users ($t = 7.2$, $p < .0001$) and among those respondents who had dependence in the past ($t = 4.5$, $p < .0001$).

The results in Table 5 also show that the prevalence of 12-month dependence was greater among 18-to-24 year olds compared to those in the four older age groups (t tests between 7.5 and 17.9 for pairwise comparisons, all of them significant at $p < .0001$). As shown in the last two columns of Table 5, this relationship results from youngest respondents not only being more likely than older respondents to be recent alcohol users, but also being more likely to become dependent once using and more likely to persist in dependence once it had developed. That is, there were significant differences in the prevalence of 12-month dependence between the youngest and older respondents in the subsample of 12-month users (t tests between 8.7 and 18.3 for all comparisons, all of them significant at $p < .0001$) and among those respondents with prior to the past year alcohol dependence (t tests between 5.7 and 11.5 for pairwise comparisons, all of them significant at $p < .0001$). An identical relationship was found between 25-to-34 year olds and the three oldest age groups (t tests between 3.6 and 17.1 for pairwise comparisons, all of them significant at $p < .0001$). The age difference in 12-month dependence observed between 18-to-24 year old respondents and the four older age groups and between 25-to-34 year olds and the three oldest age groups was preserved for men and women with only one exception. Among women, age differences between 25-to-34 year olds and the older groups were due to an increased likelihood among 25-to-34 year olds compared to the older respondents to use alcohol and to become dependent, but 25-to-34 year olds were no more likely than the older respondents to persist in dependence once it had developed.

Table 2. Cumulative probability of alcohol use by sex and cohort

	Age category (years)									
Sex/cohort	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-98
Total										
1	.088 ± .007	.600 ± .013	.746 ± .014	–	–	–	–	–	–	–
2	.073 ± .005	.592 ± .009	.722 ± .008	.742 ± .008	.748 ± .008	–	–	–	–	–
3	.037 ± .004	.483 ± .010	.671 ± .009	.707 ± .009	.719 ± .009	.726 ± .009	.729 ± .009	–	–	–
4	.037 ± .005	.370 ± .012	.591 ± .012	.634 ± .012	.662 ± .012	.673 ± .011	.680 ± .011	.682 ± .011	.684 ± .012	–
5	.026 ± .002	.249 ± .007	.422 ± .008	.470 ± .008	.496 ± .008	.509 ± .008	.517 ± .008	.521 ± .008	.525 ± .008	.536 ± .008
Male										
1	.105 ± .009	.676 ± .015	.814 ± .015	–	–	–	–	–	–	–
2	.090 ± .007	.687 ± .011	.810 ± .009	.827 ± .009	.832 ± .009	–	–	–	–	–
3	.055 ± .005	.604 ± .012	.785 ± .010	.816 ± .009	.822 ± .009	.827 ± .009	.831 ± .009	–	–	–
4	.057 ± .007	.505 ± .015	.748 ± .013	.791 ± .012	.803 ± .012	.808 ± .012	.811 ± .012	.812 ± .012	.815 ± .012	–
5	.048 ± .004	.413 ± .010	.631 ± .010	.679 ± .010	.699 ± .010	.708 ± .010	.713 ± .009	.715 ± .009	.718 ± .009	.725 ± .009
Female										
1	.071 ± .008	.523 ± .017	.676 ± .019	–	–	–	–	–	–	–
2	.056 ± .005	.500 ± .011	.635 ± .011	.659 ± .011	.667 ± .011	–	–	–	–	–
3	.020 ± .003	.366 ± .011	.561 ± .012	.600 ± .012	.618 ± .011	.629 ± .012	.630 ± .012	–	–	–
4	.018 ± .004	.242 ± .013	.441 ± .015	.495 ± .015	.527 ± .015	.545 ± .015	.555 ± .015	.559 ± .015	.560 ± .015	–
5	.009 ± .002	.121 ± .006	.259 ± .008	.308 ± .009	.339 ± .009	.355 ± .009	.366 ± .009	.371 ± .009	.376 ± .009	.389 ± .009

Table 3. Prevalence of lifetime DSM-IV alcohol dependence in the total sample and among lifetime alcohol users, by sex and age (mean ± SE)

	Male		Female		Total	
Age (cohort)	Total sample	Lifetime users	Total sample	Lifetime users	Total sample	Lifetime users
Total	18.55 ± 0.36	23.70 ± 0.43	8.43 ± 0.23	15.42 ± 0.38	13.29 ± 0.22	20.13 ± 0.31
18-24 (1)	24.87 ± 1.03	35.40 ± 1.37	13.75 ± 0.84	24.63 ± 1.31	19.32 ± 0.71	30.64 ± 1.00
25-34 (2)	23.39 ± 0.76	28.29 ± 0.87	14.63 ± 0.50	22.25 ± 0.71	18.98 ± 0.46	25.59 ± 0.59
35-44 (3)	20.89 ± 0.73	25.19 ± 0.87	8.59 ± 0.46	13.63 ± 0.71	14.66 ± 0.45	20.13 ± 0.60
45-54 (4)	17.56 ± 0.92	21.53 ± 1.10	6.77 ± 0.55	12.05 ± 0.94	12.05 ± 0.53	17.56 ± 0.75
55+ (5)	8.75 ± 0.44	12.02 ± 0.60	1.98 ± 0.16	5.08 ± 0.40	4.95 ± 0.21	9.19 ± 0.39

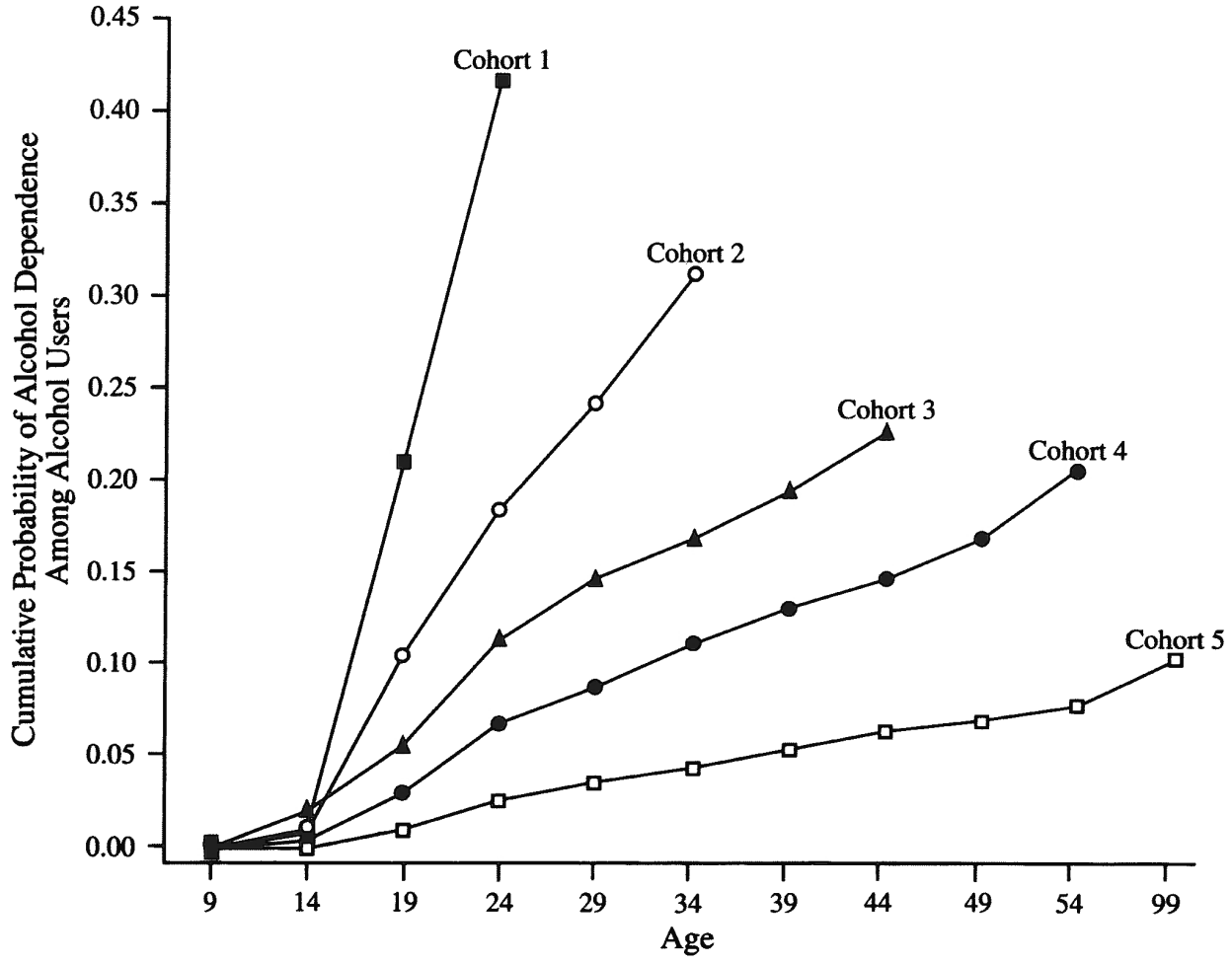
Other sociodemographic correlates

Table 6 displays a larger set of sociodemographic correlates relative to alcohol use and dependence. The contrasts presented in the three columns of Table 6 focus on three stages of progression: (1) predictors of first use (lifetime alcohol use in the total sample); (2) predictors of first onset of dependence (lifetime alcohol dependence in the subsample of lifetime alcohol users); and (3) predictors of dependence persistence (12-month alcohol dependence in the subsample of respondents with prior to the past year alcohol dependence).

Whites were significantly more likely than either blacks or Hispanics to have used alcohol at some time in their lives but were more likely than blacks, but no more likely than Hispanics, to develop dependence. However, once alcohol dependence occurred, blacks and Hispanics were more likely than whites to persist in their dependence. Education showed a similar pattern. Although the more highly educated respondents (>16 years) were more likely to use alcohol than those respondents with less than 16 years of education, the least educated respondents (<12 years) were more likely than the most educated to develop dependence, and the respondents with less than 16 years of education were more likely than the most educated to persist in dependence once it had developed. In a similar vein, married respondents were more likely than separated, divorced, widowed and never married respondents to use alcohol but were less likely to develop dependence or persist with dependence once it had developed.

Respondents having the greatest family income (≥$71,989) were more likely than respondents of lower income to use alcohol, but this pattern was reversed in predicting the progression to dependence, with lower income respondents being more likely to develop dependence than the wealthiest respondent

Figure 2. Cumulative probability of alcohol dependence among alcohol users, by cohort



group. However, only those respondents with a family income of less than $20,400 were found to be more likely to persist in dependence compared to the wealthiest subgroup.

The odds of lifetime alcohol use were significantly greater in the northeast, west and midwest compared to the south, but respondents residing in the northeast were less likely to develop dependence and the respondents residing in the west and midwest were more likely to develop dependence compared to respondents living in the south. There was no increased risk of developing dependence among midwest residents relative to those living in the south and no differences across regions in the odds of persisting in dependence. Respondents residing in urban locales were significantly more likely to use alcohol than respondents in rural locales, but they were no more likely to develop dependence or persist in dependence compared to their rural counterparts.

DISCUSSION

Prevalence of alcohol use and dependence

The NLAES alcohol use prevalence estimates were lower than those reported in NIDA's 1992 National Household Survey on Drug Abuse (NHSDA) (Department of Health and Human Services, 1993) primarily because the NHSDA measures of 12-month and lifetime use included respondents who had one or more drinks in those respective time periods. The corresponding NLAES measures included only those respondents who reported drinking at least 12 drinks during the past year or during any 1 year of their lives. The NLAES estimate for 12-month alcohol use (44.4%) was about two-thirds of the estimate reported in the NHSDA (64.7%), while the NLAES lifetime rate (66.0%) was approximately 20% less than the NHSDA rate (83.0%). In contrast, it is difficult to reconcile the differences between the NLAES and NCS estimates for 12-month and lifetime alcohol use. The prevalences rates for 12-month use (NLAES: 44.4% vs NCS: 67.5%) and lifetime use (NLAES: 66.0% vs NCS: 91.5%) were quite different between these two surveys despite the fact that similar definitions were used to define use. One explanation for the higher rates of alcohol use observed in the NCS relative to the NLAES is that the NCS included respondents aged 15 to 54 years old while the NLAES rates included adults aged 18 years and older.

Unlike the alcohol use measures, estimates of 12-month and lifetime alcohol dependence, for which comparable data exist across surveys, were remarkably similar. The 12-month prevalences of alcohol dependence in the NLAES and NCS were 4.4% and 7.2%, respectively, and rates for lifetime dependence were 13.3%, 14.1% and 7.9%, respectively, for the NLAES, NCS and ECA. The NLAES estimate of lifetime alcohol dependence among lifetime alcohol users (20.1%) was slightly higher than those of the NCS (15.4%) and ECA (15.4%). The lower rate of lifetime dependence reported in the ECA, relative to the rates observed in the NLAES and NCS, is most probably due to the DSM-III diagnostic criteria for dependence used in the ECA. The DSM-III criteria for dependence were far

Table 4. Cumulative probability of alcohol dependence among lifetime alcohol users by sex and cohort

	Age category (years)									
Sex/cohort	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-98
Total										
1	.010 ± .002	.221 ± .010	.436 ± .015	–	–	–	–	–	–	–
2	.007 ± .001	.110 ± .005	.194 ± .006	.253 ± .007	.327 ± .010	–	–	–	–	–
3	.020 ± .001	.057 ± .004	.119 ± .005	.154 ± .006	.777 ± .006	.204 ± .007	.237 ± .009	–	–	–
4	.003 ± .001	.031 ± .004	.071 ± .005	.091 ± .006	.116 ± .007	.137 ± .007	.153 ± .008	.177 ± .009	.215 ± .012	–
5	.000 ± .000	.010 ± .002	.026 ± .003	.036 ± .003	.046 ± .003	.056 ± .004	.065 ± .004	.072 ± .004	.080 ± .004	.108 ± .006
Male										
1	.007 ± .003	.251 ± .015	.497 ± .021	–	–	–	–	–	–	–
2	.006 ± .002	.124 ± .007	.214 ± .009	.280 ± .011	.359 ± .015	–	–	–	–	–
3	.002 ± .001	.070 ± .006	.150 ± .008	.195 ± .009	.224 ± .010	.253 ± .018	.296 ± .013	–	–	–
4	.005 ± .002	.047 ± .006	.102 ± .009	.127 ± .010	.155 ± .011	.177 ± .011	.194 ± .012	.218 ± .013	.258 ± .043	–
5	.000 ± .000	.015 ± .003	.039 ± .004	.052 ± .005	.066 ± .005	.080 ± .006	.090 ± .006	.096 ± .006	.106 ± .007	.141 ± .009
Female										
1	.013 ± .003	.183 ± .012	.353 ± .019							
2	.007 ± .002	.093 ± .006	.166 ± .007	.219 ± .008	.286 ± .012	–	–	–	–	–
3	.001 ± .001	.038 ± .004	.079 ± .005	.101 ± .006	.117 ± .007	.141 ± .007	.162 ± .009	–	–	–
4	.000 ± .000	.009 ± .003	.029 ± .005	.042 ± .006	.063 ± .007	.083 ± .008	.097 ± .008	.122 ± .010	.156 ± .015	–
5	.000 ± .000	.003 ± .001	.008 ± .002	.013 ± .002	.017 ± .003	.023 ± .003	.030 ± .004	.038 ± .004	.042 ± .004	.062 ± .006

Table 5. Prevalence of 12-month DSM-IV alcohol dependence in the total sample, among 12-month alcohol users and among respondents with prior to the past year alcohol dependence

Sex/age	Total sample	12-month user subsample	Prior to the past year dependent subsample
Male	6.33 ± 0.24	11.34 ± 0.41	22.59 ± 0.96
18-24	15.06 ± 0.83	24.58 ± 1.31	41.90 ± 2.87
25-34	8.75 ± 0.54	13.32 ± 0.78	27.33 ± 1.83
35-44	5.17 ± 0.41	8.73 ± 0.69	17.26 ± 1.64
45-54	3.70 ± 0.47	6.81 ± 0.84	13.77 ± 2.14
55+	1.45 ± 0.18	3.56 ± 0.45	8.86 ± 1.50
Female	2.58 ± 0.14	7.61 ± 0.37	15.96 ± 1.08
18-24	7.04 ± 0.66	17.00 ± 1.39	28.07 ± 3.15
25-34	3.99 ± 0.27	9.40 ± 0.63	15.08 ± 1.42
35-44	2.00 ± 0.22	5.10 ± 0.55	13.79 ± 2.07
45-54	1.51 ± 0.25	4.58 ± 0.73	10.10 ± 2.24
55+	0.38 ± 0.08	1.85 ± 0.36	7.12 ± 2.43
Total	4.38 ± 0.15	9.86 ± 0.30	20.44 ± 0.74
18-24	11.06 ± 0.57	21.53 ± 0.98	36.99 ± 2.12
25-34	6.35 ± 0.31	11.77 ± 0.55	22.59 ± 1.26
35-44	3.57 ± 0.23	7.26 ± 0.47	16.25 ± 1.30
45-54	2.58 ± 0.26	5.94 ± 0.59	12.76 ± 1.67
55+	0.85 ± 0.09	2.89 ± 0.31	8.48 ± 1.29

less inclusive than either the DSM-III-R or DSM-IV criteria used in the NCS and NLAES, respectively.

Sex, age and cohort effects

The finding that men were more likely than women to use alcohol was consistent with the majority of the empirical literature (Anthony et al., 1994; Helzer et al., 1991). That male alcohol users were also more likely to become dependent than female users is also consistent with the NCS and ECA findings (Anthony et al., 1994; Helzer et al., 1991). In addition, men were more likely than women to persist in dependence once it had developed. This finding has not heretofore been reported in the alcohol literature, primarily because other psychiatric interviews, such as those used in the NCS and ECA, did not yield diagnoses within both past year and prior to the past year time frames which are necessary to derive continuation or persistence in dependence rates. For example, persistence in dependence in the NCS was defined incorrectly as the prevalence of past year dependence among the lifetime

Table 6. Demographic correlates of lifetime alcohol use in the total sample, lifetime alcohol dependence among lifetime alcohol users and 12-month alcohol dependence among respondents with prior to the past year alcohol dependence

Demographic characteristic	Lifetime use (total sample)		Lifetime dependence (lifetime user subsample)		12-month dependence (prior to the past year dependence subsample)	
	OR	95% CI	OR	95% CI	OR	95% CI
Ethnicity						
White	1.00	–	1.00	–	1.00	–
Black	0.47†	0.43, 0.51	0.79†	0.68, 0.91	1.61†	1.21, 2.15
Hispanic	0.50†	0.46, 0.56	1.18	0.99, 1.38	1.87†	1.31, 2.66
Education (years)						
Less than 12	0.33†	0.31, 0.36	1.24†	1.11, 1.39	2.47†	1.86, 3.28
12	0.57†	0.53, 0.62	1.08	0.99, 1.19	1.81†	1.43, 2.30
13-15	0.83†	0.77, 0.89	1.30	0.18, 1.43	1.59†	1.24, 2.04
16+	1.00	–	1.00	–	1.00	–
Marital status						
Married	1.00	–	1.00	–	1.00	–
Separated/divorced/widowed	0.64†	0.61, 0.68	1.21†	1.10, 1.32	1.44†	1.13, 1.83
Never married	0.89†	0.82, 0.96	2.02†	1.86, 2.19	3.12†	2.54, 3.84
Family income						
≤ $20,400.00	0.35†	0.31, 0.39	1.44†	1.27, 1.63	2.20†	1.59, 3.04
$20,401.00-$35,988.00	0.57†	0.51, 0.63	1.22†	1.07, 1.39	1.32	0.93, 1.85
$35,989.00-$71,988.00	0.77†	0.68, 0.85	1.18†	1.03, 1.34	1.21	0.85, 1.72
$71,989.00+	1.00	–	1.00	–	1.00	–
Region						
Northeast	1.34†	1.23, 1.45	0.82†	0.73, 0.92	1.08	0.82, 1.43
Midwest	1.71†	1.52, 1.90	1.12†	1.02, 1.25	0.95	0.75, 1.20
South	1.00	–	1.00	–	1.00	–
West	1.55†	1.41, 1.72	1.33†	1.20, 1.48	0.88	0.69, 1.13
Urbanicity						
Rural	1.00	–	1.00	–	1.00	–
Urban	1.22†	1.13, 1.31	1.00	0.92, 1.10	1.15	0.93, 1.42

Note: OR indicates odds ratio; CI indicates confidence interval.
†An odds ratio with a confidence interval that does not include 1.0 is statistically significant.

dependent subsample when, in fact, persistence in dependence should have been defined as the prevalence of past year dependence among the prior to the past year dependent subsample (Warner et al., 1995). The incorrect formulation used in the NCS included past year dependent respondents in both the numerator and the denominator, thereby precluding the derivation of persistence in dependence rates.

The inverse relationships between age and both use and dependence found in this study were also consistent with previous research, as was the finding that use and dependence were both much more common in cohorts born after Prohibition and World War II (Anthony et al., 1994). In addition the conditional probability of dependence among users after early adolescence was greater in the youngest cohort (Cohort 1), compared to Cohort 2, despite the finding that the probability of lifetime use was lower in Cohort 1 compared to Cohort 2. No study has documented this finding primarily because the survival analytic techniques used in this study had not previously been applied to alcohol data. Although this result may be attributed to differential recall bias, or differential response sets (e.g., cohort differences in willingness to admit use and dependence), an alternative substantive interpretation is also possible. That is, it may be the case that respondents in the younger cohort, who were selected into alcohol use during a time when use was becoming less prevalent among youth (Department of Health and Human Services, 1992; Johnston et al., 1993), may be more vulnerable to dependence as the result of being more likely to have a history of other forms of psychopathology.

A related finding was that, compared to the older cohorts, there was a significantly greater probability of 12-month dependence in Cohort 1 that was due to younger persons being more likely to be recent users, more likely to become dependent and more likely to persist in dependence once it had developed. In contrast, respondents in Cohort 2 were more likely to be recent users and to be dependent, but no more likely to persist in dependence than respondents in the three oldest cohorts.

The findings that the conditional probability of dependence among users was greater in Cohort 1 compared to Cohort 2 and that Cohort 1 was more likely to persist in dependence once developed compared to Cohort 2 may be attributed to intercohort differences in drinking patterns. Since the cumulative probability of lifetime alcohol use did not differ between Cohorts 1 and 2, the increased dependence liability among respondents in Cohort 1 cannot be the result of increased use, but may well be the result of a drinking pattern in Cohort 1 that results in greater alcohol dependence liability. Recall that the drinking patterns in Cohort 2 were likely to be markedly different from those in Cohort 1 whose members consumed alcohol in conjunction with other drugs during the height of the widespread introduction of drugs into the youth subculture.

Other sociodemographic correlates

Consistent with previous research, alcohol use was found to be more likely among whites than among blacks or Hispanics, among the more highly educated than the least highly educated, among the married than among those never married, divorced, separated or widowed, and among the wealthiest than the least wealthy respondents (Dawson et al., 1995; Department of Health and Human Services, 1992). Urban residents were more likely to use alcohol than their rural counterparts, and respondents in the south were less likely to use alcohol than residents of the northwest, midwest and west.

The relationship between the alcohol measures and the various sociodemographic correlates varied as a function of the stage of the disorder. For example, whites were more likely to have used alcohol than either blacks or Hispanics. Whites were also more likely than blacks to become dependent, but equally likely to become dependent as Hispanics, although less likely to persist in dependence than either blacks or Hispanics. The most highly educated, married and wealthiest respondents were more likely to use alcohol, but less likely to become dependent and to persist in dependence compared to respondents of lower education, respondents who were never married, separated, divorced or widowed and respondents with lower incomes, respectively. Although residents of the south were less likely than those of any other region to use alcohol, the south could not be distinguished with regard to its transition to dependence from respondents in the northeast, nor from any other region with regard to persistence in dependence.

The relationships found in this study between lifetime alcohol dependence and sociodemographic correlates were not entirely consistent with those found in the NCS (Anthony et al., 1994). In the NCS, respondents who were married were not found to be less likely than never married, separated, divorced and widowed respondents to be dependent. Only respondents of the lowest income level (<$20,000/year) were found to be at higher risk of dependence relative to the wealthiest respondents.

The results of this study underscore the importance of collecting data on alcohol dependence as well as alcohol use as the basis for an adequate understanding of transitions from one stage of alcohol use disorders to the next. However, the NLAES prevalence estimates of alcohol use and dependence probably underestimate the associated true prevalences as a result of excluding high-risk subgroups of the population such as the homeless and residents of institutional and group housing. Although the AUDADIS measures of alcohol use and dependence and their associated onsets were designed to be reliable and accurate, the nature of these self-report measures are always subject to recall bias. Differential recall bias may have distorted the intercohort comparisons and caution should be used when interpreting results related to cohort differences. Finally, this study represents a descriptive epidemiologic approach focusing on bivariate relationships between sociodemographic correlates and alcohol use and dependence. Future analyses conducted within a multivariate environment should increase our understanding of the confounding and interaction between correlates of alcohol use and dependence for each progressive stage of the disorder.

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Gender Differences in the Risk of Alcohol Dependence: United States, 1992

Deborah Dawson

Data from a representative sample of U.S. adults revealed that 24% of male life-time drinkers and 15% of female life-time drinkers met the DSM-IV criteria for life-time alcohol dependence, i.e. dependence during the year preceding interview or in any 12-month period prior to that year. The median interval from first drink to onset of dependence was 3.6 years for men and 3.0 years for women. After using survival techniques to adjust for potential gender differences in the exposure to risk of developing alcohol dependence, the cumulative conditional probability of having experienced onset of dependence was 35.1% for men and 24.6% for women. The conditional probability of onset of dependence was equal for men and women in the first year after initiation of drinking, about 30% higher for men in the period 1–4 years after the first drink, and about 45% higher for men thereafter. After using proportional hazards models to adjust for the effects of age cohort, race and ethnicity, family history of alcoholism and age at first drink, these period-specific risk ratios remained virtually unchanged. Including a measure of average daily ethanol intake during periods of heaviest consumption rendered most of the gender differences statistically insignificant, revealing a slight excess risk of female dependence within the first year after initiation of drinking among the heaviest drinkers and leaving an excess male risk of dependence mostly among individuals with average daily intakes of less than one ounce of ethanol. The results suggest that different frequencies of binge drinking might help to account for these remaining gender differences and that men's and women's relative risks of developing alcohol dependence may vary as a function of life cycle stage, with men's excess risk greatest in the college/young adult years.

INTRODUCTION

Gender differences are of special significance in examining the prevalence of alcohol dependence in the general population. The usual adverse health and economic correlates of alcohol dependence are magnified in women because of the risk of fetal alcohol syndrome associated with heavy drinking during pregnancy (U.S. Department of Health and Human Services, 1993). Differences in men's and women's exposures to drinking opportunities, in how often, in what quantity and with whom they drink, in their physiological responses to ethanol intake and in the social consequences provoked by their drinking all could be expected to yield a gender differential in the prevalence of dependence. This paper applies survival analysis techniques to data from a recent sample of the U.S. population to investigate the effects of exposure and other factors on the gender differential in life-time alcohol dependence.

Studies of past-year alcohol dependence within the general population have shown that men are more likely than women to meet the criteria for past-year dependence. Using the DSM-III-R criteria for alcohol abuse and dependence (American Psychiatric Association, 1987) and a U.S. national population sample drawn as a part of the 1988 National Health Interview Survey, Grant *et al.* (1991) found that 9.6% of males 18 years of age and over were classified as dependent in the year preceding interview, compared with 3.2% of women the same age. The revisions incorporated into the DSM-IV diagnostic criteria for alcohol use disorders (American Psychiatric Association, 1994) reduced the gender differential in dependence to some degree. When applied to U.S. data from the National Longitudinal Alcohol Epidemiologic Study (NLAES), these criteria yielded past-year prevalence estimates of 6.3% and 4.4%, respectively, for men and women (Grant *et al.,* 1994a).

Little has been published comparing men's and women's risks of alcohol dependence for periods other than the past year, despite the fact that past-year data might fail to approximate life-time gender differences if men and women differed in terms of their duration of dependence, i.e., in their success in overcoming al-

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cohol use disorders. Using data from the Epidemiologic Catchment Area (ECA) program, Robins *et al.* (1984) examined life-time rates of DSM-III alcohol abuse and dependence in three U.S. sites and found male-to-female ratios of more than 4:1. However, a positive life-time diagnosis as operationalized in that study did not require the clustering of symptoms specified in the DSM-III-R and DSM-IV diagnostic criteria for alcohol use disorders; that is, there was no assurance that the symptoms had overlapped. Until the recent completion of the NLAES, no large-scale national population survey had included the array of questions necessary to establish a diagnosis of life-time alcohol dependence that accounted for the requirement of clustering (i.e., co-occurrence) of dependence criteria in the past year and/or in any other 12-month period, nor to identify the onset of dependence in periods prior to the past year.

One obvious reason why men's rates of alcohol dependence exceed those of women is that women are less likely than men to drink. In 1992, 45% of U.S. women 18 years of age and older were life-time abstainers who had never consumed at least 12 alcoholic drinks in a year. In contrast, only 22% of U.S. adult men were classified as life-time abstainers (Dawson, Grant & Chou, 1995). Analyses of earlier U.S. alcohol studies reported similar findings (Hilton, 1991a). Studies based only on current drinkers have found male-to-female ratios of past-year dependence or alcohol problems that are smaller than those observed in the general population but still indicate an excess prevalence of dependence among men (Clark & Midanik, 1982; Hilton, 1991b).

Even if based on current or life-time drinkers (i.e., people who have ever consumed alcoholic drinks), prevalence estimates do not accurately reflect the probability of developing alcohol dependence, particularly for time periods whose duration is longer than 1 year. They fail to adjust for the fact that not all drinkers *remain* at risk of developing dependence. Many life-time drinkers report having stopped drinking, and the reasons they cite for having stopped are not restricted to alcohol use disorders. Data from the 1988 U.S. NHIS indicated that 24.2% of male life-time drinkers had stopped drinking, but only 4.3% reportedly had stopped drinking because of alcohol problems. Thirty-one per cent of female life-time drinkers had stopped drinking, with just 2.3% citing alcohol problems. Other reasons reported for stopping included medical problems, alcohol problems in a family member, cost and reduced social activities (Dawson, 1994). Because these data suggest that the proportion of drinkers who stop drinking without experiencing alcohol problems may be higher for women than men, failure to account for this removal from risk means that simple prevalence figures might overestimate the male-to-female ratio in the risk of life-time dependence.

In this analysis, men's and women's risks of life-time alcohol dependence were compared after adjusting for variation in their exposure to risk arising from different ages at first drink, different ages at interview and different likelihoods of stopping drinking before becoming dependent. Multivariate proportional hazards models were used to further control for background factors such as race and family history of alcoholism and for level of alcohol consumption, taking into account interactions between gender and these factors. The outcome measure of DSM-IV life-time alcohol dependence was constructed so as to account for the requisite clustering of symptoms during both the year preceding interview and earlier time periods.

METHODS

SAMPLE

The data used in this analysis were derived from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and conducted by the U.S. Bureau of the Census. The NLAES collected information on alcohol consumption and problems, drug use and problems, periods of low mood, utilization of substance abuse treatment and family history of alcoholism from adults 18 years of age and over, selected at random from a nationally representative sample of U.S. households. Interviews were conducted in respondents' homes, and proxies were not permitted. The household and sample-person response rates for the NLAES were 92% and 97%, respectively, yielding a total sample size of 42,862. This analysis was based on the 27,616 respondents who were classified as life-time drinkers; that is, who had ever drunk at least 12 alcoholic drinks in any single year.

The NLAES featured a complex, multi-stage sample design in which primary sampling units were chosen with probability proportional to size and blacks and young adults (ages 18–29 years) were oversampled (Grant *et al.,* 1994b). In order to account for the effects of the sample design on the precision of estimates, the results presented in this paper were produced using SUDAAN (Research Triangle Institute, 1993), a software package specifically designed to accommodate complex samples by using Taylor series linearization techniques for variance estimation.

Analysis

Two steps were taken to ensure that the estimates presented in this paper accurately reflected the probability of dependence among people at risk by virtue of being drinkers. First, life-time abstainers who had never been at risk were excluded from the analysis. Secondly, survival techniques (Lee, 1980; Cox & Oates, 1984) were employed to adjust for the "censoring" effect of the fact that some individuals were removed from the risk of becoming dependent by virtue of having stopped drinking or by having had their period of observation truncated by the interview while still having many drinking years ahead of them.

In the first stage of the analysis, separate estimates were derived for men's and women's cumulative probabilities of becoming dependent, conditional upon remaining at risk (i.e., continuing to drink without yet having become dependent), within selected intervals of time since first drink. The simple (i.e., non-cumulative) conditional probability of dependence within an interval q_i, is defined as the proportion of people at risk at the beginning of the interval—those who had not stopped drinking, reached age at interview or already experienced dependence—for whom the onset of dependence occurred during the interval. The complement of this probability, $p_i=1-q_i$, indicates the conditional probability of surviving; that is, of remaining non-dependent, from the start to the end of that interval. At each interval since first drink, the *cumulative* survival function, S_i, is equal to the product of the survival probabilities for all intervals preceding and including the interval in question; that is, $S_i = (p_i)(p_i-1) \ldots (p_0)$. The cumulative conditional probability of having experienced the onset of dependence is the complement of the survival function, $1- S_i$. Male-to-female ratios of the cumulative conditional probabilities were calculated, and their associated variances were estimated using the delta method (Stuart & Ord, 1987).

In the second stage of the analysis, proportional hazards models were used to estimate the associations between various predictor variables and the hazard of experiencing the onset of alcohol dependence, as indicated by the length of the interval from first drink to either onset of dependence (for those who ever experienced DSM-IV alcohol dependence) or to withdrawal from risk of dependence (for those who never experienced dependence). Withdrawal from risk occurred at either the time of interview or, for those who had stopped drinking without having experienced dependence, at the time of last drink. In discrete time proportional hazards models, the hazard function is similar to the conditional probability of event occurrence (q_i) and can be thought of as the probability of the onset of dependence occurring within a given time interval, conditional upon the individual still being at risk of dependence at the beginning of that interval (Allison, 1984). Proportional hazards models assume that the associations between the various predictor variables and the outcome event remain constant over time even though the underlying hazard function of event occurrence varies across time intervals. The beta coefficients produced by proportional hazards models can be exponentiated to calculate hazards ratios that are analogous to the odds ratios yielded by logistic regression models.

Two separate proportional hazards models were estimated for this analysis, one that excluded alcohol consumption and one that included a measure of average daily ethanol intake during period of heaviest consumption. Other predictor variables were restricted to those whose values were fixed over the period of exposure being examined, so as to avoid erroneously attributing causal effects to variables that might actually have resulted from dependence. In addition to the main exposure variable of gender, the other predictor variables included in the models were age cohort (ages 30–54 and age 55 and over, with ages 18–29 as the reference category), race (black vs. non-black), ethnicity (Hispanic vs. non-Hispanic), family history of alcoholism (positive vs. negative) and age at first drink. Natural log transforms were applied to both age at first drink and average daily ethanol intake to improve the fit of the models. Total body water was included in the models containing ethanol intake to adjust for its modifying effect on the blood alcohol concentration caused by different levels of ethanol intake (Goist & Sutker, 1985). Both models tested for significant first-order interactions between gender and the other predictor variables.

Measures

The measure of life-time alcohol dependence used in this analysis was derived from a list of symptom item indicators designed to operationalize the seven DSM-IV dependence criteria. Although the NLAES questionnaire went into the field prior to the publication of the DSM-IV (American Psychiatric Association, 1994), the proposed DSM-IV criteria were known prior to the finalization of the NLAES questionnaire (American Psychiatric Association, 1991) and thus were fully incorporated into its design. People received a positive diagnosis for life-time alcohol dependence if they met the DSM-IV criteria for dependence in the year preceding interview *or* in any other 12-month pe-

riod. The definition of past-year dependence (i.e., dependence in the year preceding interview) has been fully described elsewhere (Grant, 1994a). Briefly, a person was considered as dependent if he or she met at least three of the seven criteria for DSM-IV dependence: tolerance; withdrawal (including relief or avoidance of withdrawal); persistent desire or unsuccessful attempts to cut down or stop drinking; much time spent drinking or recovering from its effects; important activities reduced or given up in favor of drinking; impaired control over drinking; and continued drinking despite a physical or psychological problem caused or exacerbated by drinking. Under the DSM-IV guidelines, duration qualifiers are imposed on a criterion-specific basis rather than for the overall category of dependence as was done under the DSM-III-R. For the year preceding interview, criteria not associated with duration qualifiers were satisfied if a person reported one or more positive symptoms of the criterion over the year. In order to satisfy a criterion associated with a duration qualifier, a person had to report having experienced at least one symptom two or more times over the year *or* two or more symptoms of the criterion during the same time period. To meet the criterion for withdrawal, which is defined as a syndrome or cluster of symptoms, two or more positive symptoms were required in addition to satisfaction of the duration qualifier.

To be considered dependent during a 12-month period prior to the year preceding interview, a person had to have met three or more of the seven criteria described above by reporting symptoms that occurred at some time prior to the 12 months preceding interview. Clustering and duration for periods prior to the year preceding interview were ascertained by means of a series of direct questions that recapitulated the life-time symptoms and determined that they occurred at around the same time (clustering), repeatedly for a few months or longer or most days for at least a month (duration). For people who met the criteria for dependence during a 12-month period prior to the year preceding interview, onset of dependence was ascertained by asking the age at which some of the symptoms began to happen at around the same time. For people who first experienced alcohol dependence in the year immediately preceding interview, age at onset of dependence was assigned as equal to age at interview.

Age at first drink was asked directly in the NLAES interview: "About how old were you when you first started drinking, not counting small tastes or sips of alcohol?" For former drinkers, i.e., people who had not consumed at least 12 drinks in the year preceding interview but had done so in a prior year, age at last drink was determined by means of a question that asked how long it had been since their last drink. The length of the interval representing the period of exposure to risk of dependence, which served as the dependent variable in the proportional hazards models, was estimated by subtracting age at first drink from either age at onset of dependence (for those who became dependent) or the earlier of age when stopped drinking or age at interview (when nondependent respondents were removed from the risk of becoming dependent).

Family history of alcoholism was obtained in a lengthy series of questions that asked about alcohol problems in 18 different types of blood relatives in addition to step, foster or adoptive relatives. For each type of relative, the respondent was asked "In your judgment, has (your/any of your——) been an alcoholic or problem drinker at any time in his/her life?" The following definition was provided at the outset of the questions: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hung over." For this analysis, people with a positive family history were those who identified one or more first- or second-degree biological relatives as having been alcoholics or problem drinkers.

Average daily ethanol intake was based on a series of questions concerning the period of heaviest drinking, the only period for which comparable consumption data were collected for current and former drinkers. For that period, the NLAES first asked total frequency of drinking any type of beverage, which was converted to days per year, and then obtained separate measures of usual quantity and drink size consumed on those occasions for beer, wine and liquor. The questionnaire also asked for the largest quantity and drink size ever consumed for each of the three beverages and the frequency with which this heaviest intake occurred. The annual volume of ethanol for each beverage type was calculated as follows: oz. (1 oz = 29.58 ml = 23.22 g) $\text{ethanol}_{\text{beverage}}$ = (total minus heavy drinking days per year x number of drinks per usual drinking $\text{day}_{\text{beverage}}$ x ounces of beverage in typical drink consumed on usual drinking $\text{day}_{\text{beverage}}$ x ethanol conversion factor $_{\text{beverage}}$) + (heavy drinking days per year x number of drinks per heavy drinking $\text{day}_{\text{beverage}}$ x oz. of beverage in typical drink consumed on heavy drinking $\text{day}_{\text{beverage}}$ x ethanol conversion $\text{factor}_{\text{beverage}}$). Ounces of beverage were converted to ounces of ethanol using the following conversion factors: 0.045 for beer, 0.121 for wine and

0.409 for liquor (DISCUS, 1985; Kling, 1989; Turner, 1990; Modern Brewery Age, 1992; Williams, Clem & Dufour, 1993). The beverage-specific volumes were summed over the three beverage types, and the resulting volume was divided by 365 to yield average daily intake during period of heaviest consumption. Total body water, which was included in the models adjusting for ethanol intake, was estimated for each individual on the basis of his or her age, sex, and self-reported height and weight (Moore *et al.*, 1963).

RESULTS

The proportions of life-time drinkers who met the DSM-IV criteria for alcohol dependence at some point during their lives were 23.7% for men and 15.4% for women (Table 1). The median interval from first drink to onset of dependence was slightly longer for men than women, 3.6 vs. 3.0 years. Table 1 also presents variables that might have affected the risk of dependence if they differed for men and women, i.e., potential confounders of the gender differential in that risk. More women than men had stopped drinking by the time of the NLAES interview (38.1% vs. 28.8%), but this difference was attributable totally to more women who stopped drinking after becoming dependent. However, the distribution by age at first drink indicated that men started drinking earlier than women, which implies a slightly shorter period of exposure to risk for women than for men. Finally, Table 1 shows that women consumed less ethanol than men during their periods of heaviest consumption. Although body water differences that affect the absorption of ethanol into the bloodstream (Goist & Sutker, 1985) would counteract some of the gender difference in intake, these data indicate the need to adjust for effective con-

Table 1. Selected characteristics of male and female life-time drinkers: United States, 1992

	Male	Female
n of cases	13 990	13 626
Percentage with life-time alcohol dependence	23.7 (0.4)	15.4 (0.4)
Percentage distribution by age at onset of dependence		
16 years or younger	8.5 (0.3)	10.8 (0.3)
17 or 18 years	22.6 (0.4)	21.0 (0.4)
19 or 20 years	20.2 (0.4)	17.6 (0.4)
21–24 years	20.8 (0.4)	20.1 (0.4)
25–29 years	11.1 (0.3)	11.7 (0.3)
30–39 years	10.5 (0.3)	12.2 (0.4)
40 years or older	6.4 (0.2)	6.6 (0.2)
Median number of years from first drink to onset of dependence	3.6 (0.2)	3.0 (0.2)
Percentage who stopped drinking prior to year preceding interview		
Including stop after onset of dependence	28.8 (0.5)	38.1 (0.6)
Excluding stop after onset of dependence	5.1 (0.2)	6.5 (0.2)
Percentage with a positive family history of alcoholism	54.8 (0.5)	63.2 (0.5)
Percentage distribution by age at first drink		
14 years or younger	8.9 (0.3)	5.8 (0.2)
15 years	6.6 (0.2)	5.0 (0.2)
16 years	13.8 (0.4)	10.7 (0.3)
17 years	12.6 (0.3)	9.8 (0.3)
18 years	22.7 (0.4)	19.8 (0.4)
19 years	8.1 (0.3)	8.5 (0.3)
20 years	7.2 (0.2)	7.9 (0.3)
21 years or older	20.1 (0.4)	32.6 (0.5)
Percentage distribution by average daily ethanol intake during period of heaviest consumption		
Less than 0.10 oz.[a]	10.7 (0.3)	23.1 (0.5)
0.10–0.24 oz.	11.4 (0.3)	18.8 (0.4)
0.25–0.49 oz.	13.4 (0.3)	18.4 (0.4)
0.50–0.99 oz.	18.2 (0.4)	16.5 (0.4)
1.00–1.99 oz.	17.6 (0.4)	12.0 (0.4)
2.00 oz. or more	28.7 (0.5)	11.2 (0.3)

Figures in parentheses are standard errors of estimates.[a] 1 oz. = 29.58 ml = 23.22 g.

sumption levels in order to determine whether men's greater risk of dependence stems solely from their greater alcohol intake or whether differential responses to consumption are also involved.

Table 2 shows the cumulative conditional probabilities of onset of alcohol dependence for men and women within selected intervals since first drink. Within the first year after initiation of drinking, there was no significant difference by gender in the probability of having experienced the onset of dependence. Within years 1–4, the male-to-female ratio of dependence was 1.33, with cumulative conditional probabilities of 9.6% for men and 7.2% for women at the end of that interval. Within subsequent intervals, the male-to-female ratio of dependence remained stable at about 1.45. Ultimately, the cumulative conditional probabilities of having experienced onset of alcohol dependence reached 35.1%, for men and 24.6% for women.

Since the male-to-female ratios shown in Table 2 indicated that the effect of gender changed over time since first drink, thus violating the central assumption of proportional hazards models, separate models were estimated for three time periods: within the first year after initiation of drinking, e.g., at the same age as at first drink; within 1–4 years after first drink; and 5 or more years after initiation of drinking. Table 3 shows two logistic regression models for each of these three time periods, the first excluding the effects of alcohol consumption and the second incorporating this effect.

Because of the log transforms applied to age at first drink and average daily ethanol intake and the interactions of both of these variables with gender, it is difficult to infer the magnitude or even the direction of the effects of these variables from the model parameters themselves. Parameters that *are* directly interpretable include those for age cohort, which indicated an inverse relationship with the probability of having experienced dependence, and positive family history, which was associated with an increased risk of dependence. Neither black race nor Hispanic ethnic origin showed a consistent relationship with the risk of dependence.

Table 4 presents male-to-female hazard ratios for dependence among people with various ages at first drink and various levels of average daily ethanol intake. These ratios were obtained by exponentiating the appropriate sums of the model parameters. For example, the male-to-female ratio of the hazard of dependence within 1–4 years after initiation of drinking among persons who had their first drink at age 17 and consumed an average of 0.25 oz of ethanol per day during their period of heaviest consumption was equal to $e^{-1.827 + [\log(17)](0.683)+[\log(0.25)](-0.111)}$, or 1.41.

When alcohol consumption was not taken into account, men generally exhibited greater probabilities of dependence than did women, although the gender difference was not significant within the first year after initiation of drinking except for those individuals who started drinking at late ages. Overall, the adjusted male-to-female hazard ratios were similar to but slightly larger than the unadjusted ratios shown in Table 2. Within the first 4 years after initiation of drinking, the male-to-female hazard ratio for dependence increased with age at first drink; within later intervals, age at first drink had no effect on the ratio.

When alcohol consumption was taken into account, gender differences in the risk of lifetime alcohol dependence were often statistically insignificant. In general, an excess risk of male dependence was noted only among people who drank an average of one drink per day (0.5 oz of ethanol) or less, and not within the first year after initiation of drinking regardless of consumption level. There were two exceptions to this pattern.

Table 2. Life table estimates of cumulative conditional probability of having experienced onset of DSM-IV alcohol dependence within selected intervals since first drink, by gender

	Male		Female		
Interval since first drink	Prob.	SE	Prob.	SE	Male-to-female ratio & 95% CI
Less than 1 year	0.008	(0.001)	0.004	(0.001)	NS (0.90–3.10)
1–< 5 years	0.096	(0.004)	0.072	(0.003)	1.33 (1.17–1.49)
5–< 10 years	0.145	(0.005)	0.101	(0.004)	1.44 (1.29–1.58)
10–< 15 years	0.204	(0.005)	0.137	(0.004)	1.49 (1.38–1.60)
15–< 20 years	0.242	(0.006)	0.160	(0.005)	1.51 (1.39–1.63)
20–< 25 years	0.266	(0.006)	0.180	(0.005)	1.48 (1.37–1.58)
25–< 30 years	0.286	(0.007)	0.194	(0.006)	1.47 (1.36–1.59)
30–< 25 years	0.302	(0.007)	0.205	(0.006)	1.47 (1.37–1.58)
35–< 40 years	0.313	(0.008)	0.212	(0.007)	1.48 (1.37–1.59)
40–< 45 years	0.321	(0.008)	0.218	(0.007)	1.47 (1.35–1.59)
45–< 50 years	0.333	(0.009)	0.232	(0.009)	1.44 (1.30–1.57)
50 or more years	0.351	(0.012)	0.246	(0.013)	1.43 (1.25–1.60)

First, among people who drank an average of four drinks per day or more (2.00 + oz of ethanol), the risk of dependence was *lower* for men than women within the first year after initiation of drinking. Secondly, within 1–4 years after drinking, men who started drinking at age 20 or later had a slightly higher risk of dependence than women regardless of their consumption level.

DISCUSSION

These data indicated that men and women experienced nearly equal (and minimal) risks of dependence within the first year after initiation of drinking. Over time the gender differential increased so that men's risks exceeded those of women by about 45% after 5 or more years of drinking. After adjusting for age, race and ethnicity, age at first drink and ethanol consumption during period of heaviest consumption, men's excess risk of dependence was evident mostly among individuals with low to moderate levels of ethanol intake. This probably reflects different drinking patterns among U.S. men and women in these consumption categories, with men's drinking more often characterized by infrequent binge drinking and women's drinking more often characterized by higher frequencies and lower volumes of intake per drinking occasion. While the NLAES did not collect information on binge drinking during period of heaviest consumption, data for the year immediately preceding interview provided support for this hypothesis. During the year preceding interview, the ratio of heavy-drinking days (days when five or more drinks were consumed) to average daily intake was 30% to 60% higher among men than women for those whose average daily ethanol intakes were less than 1.0 oz, but this ratio was only 15% higher for men among those with average daily intakes of 2.0 oz or more. Thus, drinking patterns may be a major factor in explaining the gender differences in risk of dependence that remain after adjusting for the factors examined in this analysis.

The findings of this analysis also suggested that differences in the male-to-female ratio of risk might reflect characteristics of different stages of the life cycle, e.g., adolescence and college ages/young adulthood. From the data presented in Table 4, age-specific relative hazards can be approximated using the information on age at first drink and the interval since first drink. For example, for the adolescent period (represented by the

Table 3. Proportional hazards predicting life-time alcohol dependence, by interval since first drink

	Within first year after first drink			1–4 years after first drink			5 or more years after first drink		
	Beta	SE	p^a	Beta	SE	p^a	Beta	SE	p^a
Models excluding consumption									
Main effects									
Male	−2.581	1.344	0.059	−2.356	0.704	0.001	0.491	0.052	0.000
Age30–54	−0.526	0.093	0.000	−0.983	0.051	0.000	−1.681	0.074	0.000
Age 55 +	−1.464	0.163	0.000	−2.498	0.133	0.000	−2.915	0.104	0.000
Black	−0.317	0.176	0.077	−0.618	0.119	0.000	0.229	0.077	0.005
Hispanic	−0.034	0.224	0.879	−0.190	0.107	0.079	0.199	0.106	0.065
Positive family history	0.583	0.099	0.000	0.685	0.061	0.000	0.898	0.060	0.000
Age at first drink[b]	−0.180	0.348	0.606	−1.906	0.192	0.000	−1.422	0.128	0.000
Interactions									
Male * age at first drink[b]	0.959	0.461	0.041	1.005	0.247	0.000	—	—	—
Models including consumption									
Main effects									
Male	−0.202	0.169	0.236	−1.827	0.729	0.015	0.158	0.095	0.009
Age 30–54	−0.602	0.100	0.000	−1.060	0.053	0.000	−1.727	0.080	0.000
Age 55 +	−1.559	0.178	0.000	−2.521	0.139	0.000	−2.995	0.116	0.000
Black	−0.356	0.185	0.058	−0.625	0.115	0.000	0.097	0.083	0.243
Hispanic	0.080	0.231	0.731	−0.044	0.108	0.681	0.209	0.147	0.161
Positive family history	0.348	0.100	0.001	0.513	0.064	0.000	0.666	0.062	0.000
Age at first drink[b]	1.353	0.257	0.000	−0.470	0.198	0.020	−0.236	0.140	0.096
Total body water[c]	0.006	0.010	0.579	0.003	0.005	0.665	−0.018	0.006	0.006
Average daily intake[d]	0.589	0.044	0.000	0.536	0.027	0.000	0.771	0.032	0.000
Interactions									
Male * age at first drink[b]	—	—	—	0.683	0.252	0.009	—	—	—
Male * average daily intake[d]	−0.225	0.058	0.000	−0.111	0.034	0.002	−0.106	0.042	0.013

[a]Rounded to three decimal places, i.e. 0.000 indicates a probability of less than 0.0005.[b]Age in years, on a natural log scale.[c]Measured in deciliters.[d]Ounces of ethanol, measured on a natural log scale (1 oz. = 29.58 ml = 23.22 g).

odds ratios for the first year and years 1–4 after first drink for those who started drinking at age 14 and by the odds ratios for the first year after first drink for those who started drinking at age 17), the only significant gender difference in the risk of dependence indicated an excess risk of dependence among women rather than men. The male excess risk of onset of dependence appeared to peak during college years/early adulthood (in the early 20s) and decrease thereafter.

Historically, studies of adolescent drinking in the United States indicated that boys were more likely than girls to drink and to drink heavily, although simple prevalence measures of ever having drunk alcohol or having consumed alcohol within a specified time period converged during the 1970s and again in recent years (see review in Johnstone, 1994). Heavy drinking is still more common among boys, but the magnitude of this differential shows an interesting variation with age. Data from the U.S. 1992 Monitoring the Future Study showed that the male-to-female ratio in the proportion of adolescents who drank 5+ drinks during the 2 weeks preceding interview rose from a low of 1.08 among 8th graders to a high of 1.75 among 12th graders (National Institute on Drug Abuse, 1993). The NLAES data reported in this paper showed that boys were 50% more likely than girls to have started drinking by age 14. Together, these findings suggest a process of selection that could help to account for the excess risk of dependence among some girls within the first year after initiation of drinking. That is, girls who start drinking at young ages or whose consumption in the later teen years reaches levels more commonly reported by boys may differ from their male counterparts in the severity of characteristics associated with the risk of early dependence, such as risk taking and behavioral undercontrol. Looking at the reversal that occurs during the college/young adult years, wherein the risk of dependence becomes greater among young men than among young women, this may reflect the trend with increasing age toward more consumption of alcohol in public places, with the concomitant additional risk of certain types of consequences that favor males, such as fighting.

It has been stated that U.S. cultural norms are less tolerant of drinking and intoxication in women than in men (Knupfer, 1984). It is interesting to note that men's excess risk of dependence, after accounting for level of intake, appeared to decline slightly in the later adult years. One might assume that the opposite would be true, because entry into adult roles such as those of spouse and parent would increase the adverse social consequences of women's drinking. However, many studies have reported that, with advancing age, women do much of their drinking in hidden contexts, such as in the home when alone (Wanberg & Knapp, 1970). Alcohol-related problems may even be covered up by family members. Thus the trend in these NLAES data may reflect this increasingly private aspect of women's drinking over time. As with all of the find-

Table 4. Male-to-female ratios for hazard of life-time alcohol dependence, by interval since first drink, for selected population subgroups defined by age at interview, age at first drink and average daily ethanol intake[a]

	Within first year after first drink	1–4 years after first drink	5 or more years after first drink
Based on models excluding consumption			
Age 14 at first drink	NS (0.71–1.27)	1.35 (1.17–1.56)	1.63 (1.47–1.81)
Age 17 at first drink	NS (0.95–1.36)	1.64 (1.48–1.82)	1.63 (1.47–1.81)
Age 20 at first drink	1.34 (1.12–1.61)	1.93(1.93–2.19)	1.63 (1.47–1.81)
Based on models including consumption			
Age 14 at first drink			
Average intake[a] = 0.25 oz.	NS (0.74–1.69)	NS (0.83–1.59)	1.36 (1.08–1.72)
Average intake[a] = 0.50 oz.	NS (0.66–1.37)	NS (0.78–1.41)	1.26 (1.03–1.54)
Average intake[a] = 1.00 oz.	NS (0.59–1.14)	NS (0.73–1.26)	NS (0.97–1.41)
Average intake[a] = 2.00 oz.	0.70 (0.51–0.96)	NS (0.68–1.15)	NS (0.91–1.31)
Age 17 at first drink			
Average intake[a] = 0.25 oz.	NS (0.74–1.69)	1.41 (1.08–1.84)	1.36 (1.08–1.72)
Average intake[a] = 0.50 oz.	NS (0.66–1.37)	1.29 (1.02–1.64)	1.26 (1.03–1.54)
Average intake[a] = 1.00 oz.	NS (0.59–1.14)	NS (0.94–1.48)	NS (0.97–1.41)
Average intake[a] = 2.00 oz.	0.70 (0.51–0.96)	NS (0.86–1.35)	NS (0.91–1.31)
Age 20 at first drink			
Average intake[a] = 0.25 oz.	NS (0.74–1.64)	1.67 (1.29–2.16)	1.36 (1.08–1.72)
Average intake[a] = 0.50 oz.	NS (0.66–1.37)	1.53 (1.21–1.94)	1.26 (1.03–1.54)
Average intake[a] = 1.00 oz.	NS (0.59–1.14)	1.40 (1.11–1.77)	NS (0.97–1.41)
Average intake[a] = 2.00 oz.	0.70 (0.51–0.96)	1.28 (1.01–1.63)	NS (0.91–1.31)

[a]During period of heaviest consumption (1 oz. = 29.58 ml = 23.22 g).

ings of this study, the generalizability of this conclusion across cultures may be limited by cultural differences in norms pertaining to men's and women's drinking and the context in which men's and women's drinking takes place.

As Table 1 indicated, much of the dependence reported by the NLAES sample occurred soon after initiation of drinking. In fact, fully half of the ages at onset of dependence reported by the NLAES respondents were younger than age 21, that is, before reaching adulthood or the legal drinking age. Forthcoming research will address the question of whether the symptom arrays of individuals with early- and late-onset dependence vary systematically, with the former comprising problems that might be better characterized as short-term sequelae of heavy or binge drinking (Room, 1977) rather than indicators of clinical alcohol dependence. Some researchers have argued that adolescent drinking problems may be part of a broader spectrum of behavioral problems that may resolve themselves with increasing age and maturity (Donovan, Jessor & Jessor, 1983; White, 1987; Jessor, Donovan & Costa, 1991). It would be interesting to see how the gender differentials in onset of dependence might vary if only adult onset of dependence was considered. However, with the NLAES data, such an analysis could only be undertaken for a very non-representative sample, i.e., those who did *not* report onset of dependence prior to adulthood. Changes in onset and remission of alcohol dependence could be measured most easily in a longitudinal context (see, for example, the longitudinal analysis of the stability of alcohol consumption conducted by Grant, Harford & Grigson, 1988).

The inability of the NLAES data to account for changing marital status, education, occupation and income over the exposure period from time of first drink was a limiting factor in this analysis. Marital status and especially occupation might prove to be interesting confounders or modifiers of gender differences in the risk of developing alcohol dependence, as many studies have found support for the argument that certain types of occupations encourage women to adopt a style of heavy drinking that increases their risk of alcohol problems (Shore, 1985; Wilsnack & Wilsnack, 1991). The detailed marital, educational and work histories required to measure changes in dynamic variables such as these are difficult to obtain in retrospective surveys, yet their utility is indicated by the richness of analyses that have been conducted with longitudinal datasets. Yet another advantage of longitudinal data would be a more detailed drinking history. The NLAES did not collect information on intermittent periods of abstinence. People were counted as having stopped drinking only if they were still abstinent at the time of interview, and they met the definition for past-year abstinence if they had consumed less than 12 drinks in the year preceding interview. Any gender-related differences in discontinuity of drinking thus were not accounted for in the period of exposure to risk that was used in this analysis, nor did this study account for gender differences in the possibility of developing dependence during the year preceding interview for persons with very low levels of consumption.

Finally, the strong negative association between age cohort and the risk of dependence suggests that this study may have underestimated the overall risk of dependence for both men and women. Since this analysis was conditional upon having initiated drinking, there would be no reason to expect large legitimate cohort effects in the risk of dependence such as those that might have derived from changing rates of abstention across cohorts. While the gender effect might have been expected to vary across cohorts in response to changing women's roles and employment experience, this expectation was not supported by any significant interaction between age cohort and gender. Rather, it seems likely that with advancing age, the saliency of past symptoms (often occurring in adolescence or early adulthood) may have decreased, leading to their being under-reported. Alternatively, changes over time in the perceived acceptability of acknowledging alcohol problems may be associated with more accurate reporting among younger people. While there is no reason to expect that under-reporting would have differed for men and women, i.e., that the gender differentials derived from this analysis would have been biased by this phenomenon, this possibility cannot be precluded. Again, the use of longitudinal data might have helped to overcome this source of under-reporting, reducing at least the loss of salience over time if not cohort effects in deliberate under-reporting.

In summary, other than by excluding life-time abstainers, adjustment for factors moderating exposure to the risk of alcohol problems did little to reduce the apparent gender difference in the risk of lifetime alcohol dependence. Adjusting for differences in total volume of ethanol intake had a somewhat greater effect in terms of reducing the gender differential, but left unexplained an excess risk of male dependence among individuals with relatively low levels of intake and in the young adult years regardless of consumption level. These remaining differences point toward the need for further exploration of factors not considered in this

analysis, notably drinking patterns and social reactions to drinking. A more complex measure of intake, one which incorporated both aspects of quantity and frequency of drinking, would surely lead to a greater understanding of the remaining gender differential, as would consideration of the context in which drinking occurs. These findings suggest the need for additional study of the joint and individual influences of factors such as life cycle stage, where and with whom drinking takes place, and dynamic measures of variables associated with role responsibilities.

ACKNOWLEDGMENTS

The author gratefully acknowledges the many worthwhile suggestions made by the anonymous reviewers of this manuscript. Their input led to substantial improvements in the quality of the analysis.

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Age at Onset of Alcohol Use and Its Association With DSM–IV Alcohol Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant and Deborah A. Dawson

Data from 27,616 current and former drinkers interviewed in the 1992 National Longitudinal Alcohol Epidemiologic Survey were used to examine the relationship between age at first use of alcohol and the prevalence of lifetime alcohol abuse and alcohol dependence, among all U.S. adults 18 years of age and over and within subgroups defined by sex and race. The rates of lifetime dependence declined from more than 40% among individuals who started drinking at ages 14 or younger to roughly 10% among those who started drinking at ages 20 and older. The rates of lifetime abuse declined from just over 11% among those who initiated use of alcohol at ages 16 or younger to approximately 4% among those whose onset of use was at ages 20 or older. After using multivariate logistic regression models to adjust for potential confounders, the odds of dependence decreased by 14% with each increasing year of age at onset of use, and the odds of abuse decreased by 8%. These findings are discussed with respect to their implications for prevention policies and the need to integrate epidemiological and intervention research.

Although early onset of alcohol use has been closely associated with numerous adverse short-term and long-term consequences, very little is known about the relationship between early onset and the subsequent development of alcohol abuse and dependence. Early onset of alcohol use is a major public health concern in terms of its impact on adolescent morbidity and mortality. Among adolescents and young adults, early onset of alcohol use has been associated with motor vehicle crashes (Millstein & Irwin, 1988; U. S. Congress, 1991), tobacco and other drug use (Schuckit & Russell, 1983), sexual intercourse, infrequent condom use and pregnancy (DiClemente, 1992; Epstein & Tamar, 1984), sexually-transmitted diseases (Shafer & Boyd, 1991), violence (Choquet, Menke, & Manfredi, 1991), depression and suicide (Kaplan, Landa, Weinhold, & Shenker, 1984; Deykin, Levy, & Wells, 1987; Robbins & Alessi, 1985), and alcohol abuse and dependence symptomatology (Gruber, DiClemente, Anderson, & Lodico, 1996).

Long-term consequences of early onset of alcohol use among adolescents and adults have also been documented in the literature. Early onset of alcohol use is associated with greater risks of other drug use and abuse (Irwin, Schuckit, & Smith, 1990; Robbins & Przybeck, 1985), early onset of alcohol abuse and dependence (Andersson & Magnusson, 1988: Von Knorring, Palm, & Andersson, 1985), and psychopathology (Buydens-Branchy, Branchy, & Noumair, 1989; Rich, Young, & Fowler, 1986).

Important limitations of most of this research on alcohol use relate to their small sample sizes and the absence of generalizability either to other adolescent populations or to the general population (including untreated individuals).

The purpose of this study is to extend the body of research on alcohol use disorders conducted over recent years by directly examining the relationship between age at onset of alcohol use and the prevalence of alcohol abuse and dependence in late adolescence and adulthood. To our knowledge, no other study has determined the odds of alcohol abuse and dependence as a function of age at onset of alcohol use in a large representative sample of the United States population.

METHOD

Study Sample

This study was based on the National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national

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probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). Field work for the study was conducted by the United States Bureau of the Census in 1992. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. Approximately 92% of the selected households participated in this survey, and 97.4% of the randomly selected respondents in these households participated in this survey.

The NLAES utilized a complex multistage design that featured sampling of primary sampling units with probability proportional to size and oversampling of the black and young adult (18 to 29 years) populations. The NLAES design has been described in detail elsewhere (Grant, et al., 1994; Massey, Moore, Parsons, & Tadros, 1989).

Measures

Diagnoses of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV: American Psychiatric Association, 1994) alcohol use disorders were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained interviewers who were not clinicians (Grant & Hasin, 1992). The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV criteria for alcohol abuse and dependence.

The AUDADIS diagnoses of alcohol abuse and dependence satisfied the clustering and duration criteria of the DSM-IV definitions. The criteria of the DSM-IV include the requirement for a clustering of symptoms within any 1-year period. The duration criterion is defined as the repetitiveness with which symptoms must occur to be counted as positive toward a diagnosis. They are represented by the terms 'recurrent', 'often' and 'persistent' appearing in the description of most of the abuse and dependence diagnostic criteria. Not only were the clustering criteria represented in past year AUDADIS diagnoses of abuse and dependence diagnostic criteria, but the corresponding past diagnoses (before the past year) also were measured as syndromes, or the clustering of the required number of symptoms necessary to achieve a diagnosis: (1) at the same time; (2) continuously for at least 1 month; or (3) repeatedly for at least 1 month. For the purposes of the present study, respondents were classified with a lifetime alcohol use disorder if they had experienced an episode of abuse or dependence in past year and/or before the past year. The DSM-IV abuse and dependence disorders diagnostic groups were mutually exclusive. Respondents classified as lifetime alcohol abusers did not meet criteria for lifetime dependence. Respondents classified with lifetime dependence included those with and without abuse diagnoses. Reliabilities of past year and prior to past year alcohol use disorders were 0.76 and 0.73 as determined in an independent test-retest study conducted in the general population prior to fielding the full survey (Grant et al., 1995).

Age of drinking onset was ascertained by asking respondents how old they were when they first started drinking, not counting small tastes or sips of alcohol. Measures selected as control variables for multivariate analyses were demographic and alcohol-related items that have been shown to affect the risk of alcohol abuse and dependence. These included race (black vs. nonblack), sex, age (18–24 years; 25–44 years; 45–64 years; 65+ years) and duration of drinking in years. Duration of drinking was estimated by subtracting the age at onset of drinking from either the age at last drink (for former drinkers) or age at interview (for past year or current drinkers). When age at drinking onset equaled age at last drink, duration of drinking variable was set at 0.5 years. The test-retest reliability of the drinking onset variable was 0.72 (Grant et al., 1995).

Family history of alcoholism was ascertained through a series of questions that asked about 18 different types of first- and second-degree biological relatives. For each type of relative, the respondent was asked how many relatives of that type lived to be at least ten years old and how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM-IV criteria for alcohol use disorders: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover." In a test-retest study conducted in conjunction with the pretest for the NLAES, the family history items generally showed good to excellent reliability, with kappas of 0.70 or higher for most types of first- and second-degree relatives (e.g., 0.72 for fathers, 1.00 for mothers, 0.90 for brothers, 0.73 for sisters, and 0.77 for both maternal and paternal grandparents). Slightly lower kappa values were obtained for sons and daughters (0.65 for each). The family history measure in this study was considered as positive if any first- or second-degree relatives were reported as having been alcoholics or problem drinkers.

STATISTICAL ANALYSIS

The analysis consisted of two parts. First, the prevalences of lifetime alcohol abuse and dependence were estimated for each year of age at onset of alcohol use from ages 12 to 25 years for the overall sample and separately by sex and race. Second, linear logistic regression analyses were used to assess the relationship between age at onset of alcohol use and the odds of alcohol abuse or dependence in late adolescence and adulthood, controlling for the effects of sex, race, age, duration of drinking, family history of alcoholism and current drinking status, that is, current (past year) drinker versus former drinker (drank in the past, but not in the past year). All analyses were conducted using SUDAAN, a software package that uses Taylor series linearization to adjust for the complex design of the NLAES (Research Triangle Institute, 1996).

RESULTS

Sixty-six percent (n=27,616) of the NLAES sample was composed of current (18,352) and former (9,264) drinkers. Fifty-one percent of the drinkers were male, and 49% were female. Mirroring the distribution found in the general population, 88.9% of the drinkers were nonblack, and 11.1% were black.

Table 1 shows the prevalence of lifetime alcohol dependence for each year of age at onset of alcohol use from age 12 or less to age 25 or older. The prevalence of lifetime alcohol dependence decreased steeply as a function of increasing age at onset of drinking. In the total sample, more than 40% of respondents who initiated drinking before 15 years of age were classified with alcohol dependence at some time in their lives. Corresponding prevalences among those who started drinking at ages 15 and 16 were 38.7% and 30.6%, respectively. The prevalence of lifetime alcohol dependence among those who started drinking at age 17 was 24.5%, decreasing steadily to approximately 10% among those who started drinking at ages 21 and 22. Interestingly, the prevalence of dependence increased slightly among those respondents who initiated drinking at ages 22 and 23 years after which the prevalence of dependence resumed its decline. The downward trend of alcohol dependence as a function of increasing age at onset of alcohol use that was observed in the total sample of drinkers was similar to that observed within each sex and racial subgroup.

Table 2 presents the prevalence of lifetime alcohol abuse as a function of age at initiation of drinking. Similar to the results for alcohol dependence, the prevalence of abuse generally declined with each increasing year of age at onset of drinking. The prevalence of lifetime abuse peaked among respondents who began drinking at age 14 years (between 11.6% and 14.7%, depending on sex and race), declining slowly to between 1.8% and 3.6% among respondents 25 years and older at initiation of drinking.

Multivariate linear logistic analyses were conducted to assess the contribution of age at onset of drinking

Table 1. Age at First Alcohol Use and the Prevalence of Lifetime Alcohol Dependence

Age at First Alcohol Use (in Years)	Prevalence[1] of Lifetime Dependence				
	Male n=13,990	Female n=13,626	Black n=3,062	Nonblack n=24,554	Total n=27,616
12 or Younger	41.3 (2.7)	39.1 (3.7)	36.3 (6.7)	41.1 (2.2)	40.6 (2.1)
13	49.4 (3.4)	43.2 (3.9)	44.4 (9.9)	47.5 (2.7)	47.3 (2.7)
14	43.2 (2.5)	36.2 (2.9)	31.1 (6.4)	41.5 (2.0)	40.8 (1.9)
15	39.8 (1.8)	36.6 (2.2)	27.4 (4.5)	39.5 (1.5)	38.7 (1.4)
16	33.0 (1.2)	26.6 (1.4)	25.4 (3.4)	31.0 (1.0)	30.6 (0.9)
17	26.8 (1.3)	20.7 (1.3)	23.4 (3.2)	24.6 (1.0)	24.5 (1.0)
18	19.4 (0.8)	12.5 (0.7)	13.9 (1.6)	16.9 (0.6)	16.6 (0.6)
19	19.9 (1.5)	12.2 (1.1)	14.3 (3.2)	16.7 (1.1)	16.5 (1.0)
20	14.7 (1.3)	7.4 (0.8)	12.5 (2.6)	11.3 (0.8)	11.4 (0.8)
21	11.9 (1.0)	8.1 (0.7)	10.6 (2.3)	9.9 (0.6)	10.0 (0.6)
22	11.3 (1.7)	7.6 (1.3)	9.5 (3.3)	9.5 (1.1)	9.5 (1.1)
23	15.7 (3.1)	13.8 (2.2)	15.0 (4.8)	14.7 ((2.0)	14.7 (1.9)
24	20.4 (3.7)	8.2 (2.2)	16.0 (5.1)	13.2 (2.3)	13.6 (2.1)
25 or Older	9.7 (1.2)	6.9 (0.7)	9.6 (1.5)	7.7 (0.7)	7.9 (0.6)

[1] Prevalence expressed as a weighted percentage; ns presented as unweighted figures.

Note: Standard errors appear in parentheses

to the odds of lifetime abuse and dependence, controlling for the effects of sex, race, age, duration of drinking, current drinking status, and family history of alcoholism. Age at first alcohol use was entered into this analysis as a continuous measure. After adjusting for the other model covariates, age at onset of alcohol use remained a major contributor to the development of alcohol abuse and alcoholism. Specifically the odds of lifetime alcohol dependence were reduced by 14% with each increasing year of age at first use. Similarly, the odds of lifetime alcohol abuse were reduced 8% with each increasing year that drinking onset was delayed.

The contribution of age at onset of alcohol use to the odds of lifetime alcohol abuse and dependence varied little across sex and race subgroups. Among males, females and nonblacks the odds of lifetime alcohol dependence were reduced 14.7%, 13.2%, and 14.5%, respectively, with each increasing year of age at onset of alcohol use, while the corresponding reduction among blacks was somewhat lower (8.1%). The odds of lifetime abuse were reduced 7.0%, 9.1%, 6.7% and 7.8% among males, females, blacks and nonblacks, respectively.

DISCUSSION

Age at first use of alcohol is a powerful predictor of lifetime alcohol abuse and dependence. The prevalence of each of these disorders showed a striking decrease with increasing age at onset of use. After using multivariate techniques to adjust for potential confounders, the odds of lifetime alcohol dependence and abuse were reduced by 14% and 8%, respectively, for each increasing year of age at initiation.

The prevalence of alcohol abuse showed a steady decline with postponement of age at first use. However, the trend for dependence was not as uniform. Specifically, the prevalence of dependence increased from 9.5% for initiation at age 22 to 14.7% and 13.6% for initiation at ages 23 and 24, respectively. A possible explanation for this finding is that initiation of alcohol use abnormally late (i.e., after a large majority of the population initiates use) may be an indicator of the presence of other psychopathology and perhaps efforts at self-medication in response to those disorders. Although the present study cannot conclusively confirm this interpretation, the perturbation in the decline of the prevalence of alcohol dependence occurring during these ages deserves further study. Very little variation in risk was noted across sex and race subgroups of the population. However, the reduction in risk of dependence, but not abuse, with each increasing year of age of onset of use was lower among blacks than nonblacks. Although the interpretation of this finding is unclear, this result highlights the need for research on racial minorities and other groups whose unique cultural traditions and life experiences may contribute to different patterns of risk for alcohol dependence.

The findings of this study identified preadolescence and early adolescence (ages 16 and younger) as a particularly vulnerable period for initiation of drinking,

Table 2. Age at First Alcohol Use and the Prevalence of Lifetime Alcohol Abuse

Age at First Alcohol Use (in Years)	Prevalence[1] of Lifetime Abuse				
	Male n=13,990	Female n=13,626	Black n=3,062	Nonblack n=24,554	Total n=27,616
12 or Younger	8.1 (1.4)	8.6 (1.9)	8.0 (4.1)	8.3 (1.2)	8.3 (1.1)
13	13.6 (2.6)	7.4 (2.3)	2.5 (2.5)	12.1 (1.9)	11.5 (1.8)
14	14.7 (2.0)	12.2(2.0)	11.6 (4.7)	14.0 (1.5)	13.8 (1.5)
15	12.4 (1.2)	11.1 (1.4)	9.7 (4.1)	12.1 (0.9)	11.9 (0.9)
16	12.3 (0.9)	7.7 (0.8)	8.7 (2.5)	10.7 (0.7)	10.6 (0.7)
17	10.3 (0.8)	8.1 (0.9)	5.8 (1.7)	9.8 (0.6)	9.5 (0.6)
18	9.1 (0.6)	5.7 (0.5)	3.3 (0.9)	8.1 (0.4)	7.8 (0.4)
19	6.9 (0.8)	5.6 (0.7)	3.6 (1.2)	6.6 (0.6)	6.3 (0.6)
20	6.4 (0.8)	2.8 (0.5)	2.2 (1.0)	5.1 (0.5)	4.8 (0.5)
21	6.5 (0.8)	3.1 (0.5)	5.1 (1.9)	4.7 (0.5)	4.8 (0.4)
22	5.9 (1.7)	3.8 (1.0)	2.6 (1.7)	5.2 (1.1)	4.9 (1.0)
23	3.2 (1.0)	3.9 (1.4)	0.0 (0.0)	4.0 (1.0)	3.6 (0.9)
24	4.3 (1.9)	2.2 (1.1)	1.1 (1.1)	3.5 (1.2)	3.1 (1.0)
25 or Older	3.6 (0.8)	2.0 (0.4)	1.8 (0.9)	2.7 (0.4)	2.5 (0.4)

[1] Prevalence expressed as a weighted percentage; ns presented as unweighted figures.

Note: Standard errors appear in parentheses

one that is strongly associated with an elevated risk of developing an alcohol use disorder. Although these results suggest that preventive efforts should be targeted toward the delay of alcohol use onset until after ages 18 or 19 when the associated risk of alcohol abuse and dependence has dramatically dropped, such a recommendation should be considered cautiously. The strength of such a preventive strategy lies in its focus on the prevention of alcohol abuse and dependence rather than alcohol use, a strategy that recognizes that the use of alcohol is commonplace among American adolescents and youth. However, the weakness of such a preventive strategy is the lack of a complete understanding as to why the onset of alcohol use is related to the development of alcohol abuse and dependence.

The most significant contribution of this study is the focus it provides for the direction of future research and preventive efforts. There exists an urgent need to integrate epidemiological and etiologic research with intervention research, with a view toward the prevention of alcohol use disorders. Such an integration should take the form of a prospective study incorporating prevention efforts targeted toward early onset alcohol users. This research could ascertain if it is the delay in alcohol use or, more likely, other associated factors that account for the inverse relationship between age at first drink and the risk of lifetime alcohol use disorders. Within this paradigm, another central research question is to determine the status of early onset use as either a critical and potentially modifiable risk factor in the development of alcohol use disorders, or alternatively, as a marker or early indicator of the inevitable, and perhaps unmodifiable development of alcohol use disorders. It will also be possible to determine whether delaying onset of alcohol use has any adverse or unintended effects, for example, increasing the prevalence of other drug use and experimentation among adolescents. From a methodological point of view, a prospective longitudinal study could also reduce the extent of recall bias inherent in cross-sectional designs (including the present study) and increase our ability to disentangle the importance of the contribution of age of onset of alcohol use and duration of drinking on the development of alcohol use disorders.

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Comorbidity Between DSM–IV Alcohol and Drug Use Disorders: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant and Roger P. Pickering

Research has not yet determined the answers to many questions regarding the comorbidity of alcohol and drug use disorders. Past studies often have not distinguished abuse from dependence and use and have not made diagnoses according to psychiatric classifications. This study relies on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey, which attempts to address these concerns. The study demonstrates a pervasive co-occurrence of alcohol and drug use disorders in the general population. Further, the comorbidity of alcohol and drug dependence is found to be significantly greater than the comorbidity of alcohol and drug abuse.

Although much has been learned since the early 1970's about the use of alcohol with other drugs, gaps remain in our understanding of the comorbidity of alcohol and drug use disorders (i.e., abuse and dependence). Our lack of knowledge concerning the relationship between alcohol and drug use disorders can be attributed to several factors. First, most studies have failed to differentiate alcohol and drug use from either abuse or dependence and, in fact, have used all three terms interchangeably. Second, few studies have provided for the requisite assessment of alcohol and drug use diagnoses according to current psychiatric classifications. Of these studies, the majority have been conducted among clinical or treated samples that are not well suited to the study of comorbidity (Rounsaville et al. 1982; Weiss et al. 1988). People in treatment are more likely to have multiple disorders than are people in the general population, thereby spuriously inflating estimates of the prevalence of comorbidity, a phenomenon referred to as Berkson's bias (Berkson 1946).

Despite the bias inherent in the study of comorbidity in clinical samples, large general population surveys of the distribution of psychiatric comorbidity are rare. To date, only one general population survey has produced results bearing specifically on the co-occurrence of alcohol and drug use disorders (Helzer and Pryzbeck 1988). In this survey, entitled the Epidemiologic Catchment Area (ECA) study, 18,571 respondents were interviewed during the early 1980's in a series of five community-based epidemiologic studies (Regier et al. 1984).

The ECA, however, has raised several historical and methodological issues that support further investigation of comorbid alcohol and drug use disorders. First, the study was conducted in the early 1980's, and changes in the epidemiology of alcohol and drug use disorders and their associated comorbidity have rendered the ECA results outdated. In addition, the ECA utilized diagnostic criteria for alcohol and drug use disorders from the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM–III) (American Psychiatric Association 1980), a psychiatric classification that is no longer used in the field. Furthermore, as highlighted by Helzer and Pryzbeck (1988), the ECA was not a nationally representative sample of the U.S. population, and the measurement of alcohol and drug use disorders was not adequate. Alcohol and drug use disorders, as measured in the ECA, failed to account for the clustering requirement of the DSM–III. For example, in the ECA, a lifetime diagnosis of alcohol dependence was defined as the occurrence of a minimum of two alcohol symptoms on a lifetime basis. These symptoms, according to Helzer and Pryzbeck (1988), "may have been separated by a period of several years and there is no guarantee that there was ever a cluster of symptoms or alcohol problems occurring together."

The purpose of the current study was to examine the comorbidity of alcohol and drug use disorders in a large representative sample of the United States popu-

Reprinted from Alcohol Health & Research World *(now titled* Alcohol Research & Health), *Volume 20, Number 1, pp.67–72, 1996, National Institute on Alcohol Abuse and Alcoholism.*

lation. In this study, alcohol and drug use disorders were classified according to the most recent psychiatric classification, the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM–IV) (American Psychiatric Association 1994), using a psychiatric assessment instrument that, importantly, defined alcohol and drug use disorders as syndromes, or the occurrence or clustering of symptoms within a period of time necessary to achieve a diagnosis.

METHODS

Study sample

This study is based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism, with fieldwork conducted by the Bureau of the Census. The NLAES consisted of direct face-to-face interviews with 42,862 adults, age 18 and older, who were randomly selected from a nationally representative sample of households. Interviews were conducted in the respondents' homes, and proxies were not permitted. The study sample design of the NLAES included stratification and clustering[1] as well as oversampling of blacks and young adults (ages 18 to 29) and is more fully described elsewhere (Grant et al. 1994). The household and sample person response rates for the NLAES were 91.9 percent and 97.4 percent, respectively.

Diagnostic assessment

Diagnoses of DSM–IV alcohol and drug use disorders were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained interviewers who are not clinicians (Grant and Hasin 1992). The AUDADIS includes an extensive list of symptom questions that operationalize the DSM–IV criteria for substance use disorders. Substance-specific diagnoses of abuse and dependence can be derived separately for alcohol, sedatives, tranquilizers, opioids (other than heroin), amphetamines, cocaine, cannabis (as well as tetrahydrocannabinol [THC] and hashish), heroin, methadone, and hallucinogens. A prescription drug use disorder measure also was constructed to represent abuse of and/or dependence on sedatives, tranquilizers, opiates, and/or amphetamines. Similarly, any drug abuse and/or dependence measure was constructed to represent abuse and/or dependence on any drug excluding alcohol. Although the DSM–IV was not published until 1994, the specific diagnostic criteria of interest were known prior to conducting the NLAES (American Psychiatric Association 1991) and therefore were incorporated in their entirety within the AUDADIS.

Consistent with the DSM–IV, an AUDADIS diagnosis of past-year substance abuse required that a person exhibit a maladaptive pattern of substance use leading to clinically significant impairment or distress, as demonstrated by at least one of the following during the past year: (1) continued use despite a social or interpersonal problem caused or exacerbated by the effects of use, (2) recurrent use in situations in which substance use is physically hazardous, (3) recurrent use resulting in a failure to fulfill major role obligations, or

Table 1. Prevalence of Lifetime DSM–IV[1] Alcohol and Drug Use Disorders: United States, 1992

Disorder	Prevalence (%)	S.E.[2]
Alcohol abuse and/or dependence	18.17	(0.27)
Alcohol abuse only	4.88	(0.13)
Alcohol dependence	13.29	(0.22)
Any drug abuse and/or dependence	6.05	(0.15)
Any drug abuse only	3.14	(0.11)
Any drug dependence	2.91	(0.10)
Prescription drug abuse and/or dependence	2.01	(0.08)
Prescription drug abuse only	0.98	(0.06)
Prescription drug dependence	1.03	(0.06)
Sedative abuse and/or dependence	0.64	(0.04)
Sedative abuse only	0.30	(0.03)
Sedative dependence	0.34	(0.03)
Tranquilizer abuse and/or dependence	0.63	(0.05)
Tranquilizer abuse only	0.31	(0.03)
Tranquilizer dependence	0.32	(0.03)
Amphetamine abuse and/or dependence	1.48	(0.07)
Amphetamine abuse only	0.76	(0.05)
Amphetamine dependence	0.72	(0.05)
Cannabis abuse and/or dependence	4.64	(0.14)
Cannabis abuse only	2.86	(0.11)
Cannabis dependence	1.78	(0.08)
Cocaine abuse and/or dependence	1.66	(0.07)
Cocaine abuse only	0.64	(0.04)
Cocaine dependence	1.02	(0.06)
Hallucinogen abuse and/or dependence	0.59	(0.04)
Hallucinogen abuse only	0.30	(0.03)
Hallucinogen dependence	0.29	(0.03)

[1]DSM–IV = *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.*
[2]SE = standard error.

[1]For a definition of these and other technical terms, see glossary, p. 94.

(4) recurrent substance-related legal problems. An AUDADIS diagnosis of past-year substance dependence required that a person meet at least three of seven criteria defined for dependence during the past year, including the following: (1) tolerance; (2) withdrawal, or relief of or avoidance of withdrawal; (3) persistent desire or unsuccessful attempts to cut down or stop using; (4) spending much time obtaining a substance, using it, or recovering from its effects; (5) giving up or reducing occupational, social, or recreational activities in favor of substance use; (6) impaired control over use; and (7) continued use despite a physical or psychological problem caused or exacerbated by substance use.

Past-year diagnoses of alcohol and drug use disorders also satisfied the duration criteria of the DSM–IV. According to the DSM–IV, duration qualifiers associated with some, but not all, abuse and dependence criteria define the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. Duration qualifiers are represented by the terms "recurrent," "often," and "persistent." To satisfy a criterion associated with a duration qualifier, a respondent had to report having experienced at least one symptom two or more times during the past year or two or more symptoms of the criterion at least once during the same time period. To meet the criterion for withdrawal, which is defined as a syndrome or cluster of symptoms, two or more positive symptoms of withdrawal were required, each of which had to occur on at least two occasions. Corresponding prior to the past-year diagnoses of DSM–IV, alcohol and drug use disorders also were measured as syndromes, that is, the clustering of the required number of symptoms either (1) on most days for at least 1 month, (2) on and off for a few months or longer, or (3) at about the same time. Respondents classified with an alcohol- or drug-specific DSM–IV lifetime diagnosis encompassed all those who had ever experienced an episode of abuse and/or dependence either during the past year or prior to the past year.

Statistical analysis

Because of the complex survey design of the NLAES, variance estimation procedures that assume a simple random sample cannot be employed. To take into account the NLAES sample design, all standard errors of the prevalence estimates and comorbidity rates presented in this report were generated using Survey Data Analysis (SUDAAN) (Research Triangle Institute 1994), a software program that uses appropriate statistical techniques to adjust for sample design characteristics.

Associations between alcohol and drug use disorders were expressed in terms of odds ratios. Odds ratios and their 95-percent confidence intervals were derived from separate logistic regression analyses using the SUDAAN LOGISTIC program, which also adjusted for the complex sampling design of the NLAES. Because comorbidity is strongly influenced by important sociodemographic factors, two sets of odds ratios are presented: one set that has been adjusted for age, ethnicity, and sex and one set that consists of crude, or unadjusted, odds ratios. An odds ratio of greater than 1.0 reflects a positive association between the comorbid disorders and is statistically significant if its 95-percent confidence interval does not encompass the value of 1.0. In this report, comorbidity rates and odds ratios are not presented separately for opioids, heroin, or methadone because of their extremely low prevalence, and therefore imprecision, in the study sample.

The current analyses focused on what has been termed episode, or period, comorbidity, which is the co-occurrence of two or more psychiatric disorders during the same time period. Episode comorbidity should be contrasted with comorbidity viewed from the primary-secondary distinction, in which one of two or more comorbid disorders is designated as primary, usually on the basis of its onset at an earlier age. An important consequence of examining the co-occurrence of disorders from a period comorbidity rather than a primary-secondary perspective is that the odds ratios are equivalent regardless of whether an alcohol use disorder or a drug use disorder is designated as the index, or focal, disorder.

RESULTS

Table 1 shows the prevalence rates of lifetime DSM–IV alcohol and drug use disorders. The prevalence of lifetime alcohol use disorders was 18.2 percent, with 4.9 percent and 13.3 percent of the respondents classified as alcohol abusers and alcohol dependent, respectively. For all drugs combined, slightly more respondents were classified with abuse (3.1 percent) than dependence (2.9 percent). Prevalences of lifetime sedative, tranquilizer, and hallucinogen abuse and dependence combined were less that 1.0 percent. For the remainder of the drugs, lifetime abuse and/or dependence was approximately 1.5 percent for amphetamines and cocaine, 2.0 percent for prescription drugs, and 4.6 percent for cannabis. With the exception of cocaine and alcohol, the prevalences of abuse diagnoses equaled or exceeded the corresponding dependence diagnoses.

Table 2 shows the comorbidity rates between DSM–IV alcohol and drug use disorders. Among respondents with any drug use disorder, 69.4 percent were classified with an alcohol use disorder, compared with 14.9 percent of the respondents with no history of a drug use disorder. The prevalences of alcohol abuse and alcohol dependence combined among respondents with prescription drug, sedative, tranquilizer, amphetamine, cannabis, cocaine, and hallucinogen abuse or dependence combined were quite high, ranging from approximately 60.0 to 80.0 percent. In general, the prevalences of alcohol abuse among respondents with drug abuse (11.0 to 16.4 percent) were much lower than those associated with drug dependence (70 to 85 percent).

The results of table 2 were calculated under the assumption that respondents with drug use disorders represent the exposed group. One could easily designate, however, respondents with alcohol use disorders as the exposed group, as indicated in table 3, and then calculate the odds of a drug use disorder in that exposed group relative to the odds of a drug use disorder in the unexposed group or in those without an alcohol use disorder. Regardless of the specification of exposed and unexposed groups, the odds ratios calculated from the results presented in tables 2 and 3 will be identical.

As seen in table 3, the prevalence of any drug use disorder among respondents with a history of an alcohol use disorder was 23.1 percent, compared with 2.3 percent among respondents who had not had an alcohol use disorder. Respondents classified with an alco-

[2]Expressed in terms of odds ratios (OR) adjusted for age, sex, and ethnicity.

Table 2. Lifetime Prevalence of Selected DSM–IV[1] Alcohol Use Disorders Among Respondents With and Without Corresponding DSM–IV[1] Drug Use Disorder; United States, 1992

	Prevalence of Alcohol Use Disorders[2] Among Respondents			
	With Corresponding Drug Use Disorder[3]		Without Corresponding Drug Use Disorder[3]	
Drug Use Disorder	%	S.E.[4]	%	S.E.[4]
Any drug abuse and/or dependence	69.4	(1.04)	14.9	(0.25)
Any drug abuse only	16.4	(1.17)	4.5	(0.13)
Any drug dependence	65.3	(1.53)	11.7	(0.21)
Prescription drug abuse and/or dependence	73.4	(1.74)	17.0	(0.27)
Prescription drug abuse only	13.4	(2.27)	4.8	(0.13)
Prescription drug dependence	68.9	(2.50)	12.7	(0.22)
Sedative abuse and/or dependence	80.1	(2.57)	17.8	(0.27)
Sedative abuse only	13.0	(3.81)	4.9	(0.13)
Sedative dependence	80.6	(3.38)	13.1	(0.22)
Tranquilizer abuse and/or dependence	80.7	(3.61)	17.8	(0.27)
Tranquilizer abuse only	11.2	(3.52)	4.9	(0.13)
Tranquilizer dependence	79.3	(4.06)	13.1	(0.22)
Amphetamine abuse and/or dependence	75.9	(2.05)	17.3	(0.27)
Amphetamine abuse only	13.5	(2.67)	4.8	(0.13)
Amphetamine dependence	70.0	(3.07)	12.9	(0.22)
Cannabis abuse and/or dependence	71.0	(1.22)	15.6	(0.25)
Cannabis abuse only	16.6	(1.19)	4.5	(0.13)
Cannabis dependence	69.5	(2.00)	12.3	(0.21)
Cocaine abuse and/or dependence	76.4	(1.86)	17.2	(0.26)
Cocaine abuse only	15.3	(2.80)	4.8	(0.13)
Cocaine dependence	71.2	(2.42)	12.7	(0.22)
Hallucinogen abuse and/or dependence	85.4	(2.25)	17.8	(0.27)
Hallucinogen abuse only	11.0	(3.62)	4.9	(0.13)
Hallucinogen dependence	84.7	(3.43)	13.1	(0.22)

[1]DSM–IV = *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.*
[2]Abuse and/or dependence, abuse only, or dependence only.
[3]Comparisons between alcohol abuse versus drug-specific abuse, alcohol dependence versus drug-specific dependence, and alcohol abuse and/or dependence versus drug-specific abuse and/or dependence.
[4]SE = standard error.

hol use disorder were at approximately 10 times the risk of having a drug use disorder as were those with no alcohol use disorder. The associations[2] between hallucinogen (OR = 17.1) and tranquilizer (OR = 16.5) abuse and/or dependence and alcohol abuse and/or dependence were greater than those for sedatives (OR = 14.6), cocaine (OR = 12.0), amphetamines (OR = 11.4), cannabis (OR = 9.3), or overall prescription drug abuse and/or dependence (OR = 10.6). For all drug categories examined, the associations between drug abuse and alcohol abuse were much smaller than the corresponding dependence association. In all cases, respondents classified with alcohol abuse were at approximately two times the risk of having a drug use disorder as were those without a history of alcohol abuse.

DISCUSSION

Virtually all the odds ratios presented in this study were significantly greater than 1.0, demonstrating that the comorbidity of alcohol and drug use disorders is pervasive in the general population. Among those with a lifetime DSM–IV drug use disorder, 69.4 percent experienced an alcohol use disorder, a comorbidity rate significantly greater than the population base rate of

Table 3. Lifetime Prevalence and Odds Ratios of Selected DSM–IV[1] Drug Use Disorders Among Respondents With and Without Corresponding DSM–IV[1] Alcohol Use Disorder: United States, 1992

	Prevalence of Drug Use Disorders[2] Among Respondents						
	With Corresponding Alcohol Use Disorder[3]		Without Corresponding Alcohol Use Disorder[3]				
Drug Use Disorder	%	S.E.[4]	%	S.E.[4]	Odds Ratio	Adjusted Odds Ratio[5]	95% Confidence Interval
Any drug abuse and/or dependence	23.1	(0.61)	2.3	(0.09)	13.0	9.6	(8.6, 10.6)
Any drug abuse only	10.6	(0.79)	2.8	(0.10)	4.2	2.9	(2.4, 3.5)
Any drug dependence	14.3	(0.55)	1.7	(0.70)	14.2	10.9	(9.5, 12.6)
Prescription drug abuse and/or dependence	8.1	(0.38)	0.7	(0.05)	13.4	10.6	(8.8, 12.9)
Prescription drug abuse only	2.7	(0.49)	0.9	(0.06)	3.1	2.2	(1.5, 3.2)
Prescription drug dependence	5.3	(0.34)	0.4	(0.04)	15.2	12.6	(9.9, 16.1)
Sedative abuse and/or dependence	2.8	(0.22)	0.2	(0.02)	18.6	14.6	(10.6, 20.2)
Sedative abuse only	0.8	(0.25)	0.3	(0.03)	2.9	2.1	(1.1, 4.1)
Sedative dependence	2.0	(0.22)	0.1	(0.01)	27.6	22.0	(14.5, 33.5)
Tranquilizer abuse and/or dependence	2.8	(0.23)	0.2	(0.02)	19.4	16.5	(11.5, 23.6)
Tranquilizer abuse only	0.7	(0.21)	0.3	(0.03)	2.5	1.8	(0.9, 3.7)
Tranquilizer dependence	1.9	(0.21)	0.1	(0.02)	25.5	22.2	(13.5, 36.5)
Amphetamine abuse and/or dependence	6.2	(0.33)	0.4	(0.04)	15.0	11.4	(9.1, 14.5)
Amphetamine abuse only	2.1	(0.45)	0.7	(0.05)	3.1	2.2	(1.4 3.4)
Amphetamine dependence	3.8	(0.30)	0.3	(0.03)	15.8	12.3	(9.1, 16.6)
Cannabis abuse and/or dependence	18.1	(0.55)	1.6	(0.08)	13.3	9.3	(8.2, 10.5)
Cannabis abuse only	9.7	(0.72)	2.5	(0.10)	4.2	2.9	(2.4, 3.5)
Cannabis dependence	9.3	(0.48)	0.6	(0.05)	16.3	11.7	(9.7, 14.2)
Cocaine abuse and/or dependence	7.0	(0.33)	0.5	(0.04)	15.6	12.0	(9.6, 15.0)
Cocaine abuse only	2.2	(0.54)	0.8	(0.07)	3.6	2.5	(1.6, 3.9)
Cocaine dependence	5.5	(0.34)	0.3	(0.03)	17.0	13.5	(10.6, 17.2)
Hallucinogen abuse and/or dependence	2.8	(0.22)	0.1	(0.01)	27.1	17.1	(12.0, 24.6)
Hallucinogen abuse only	0.7	(0.24)	0.3	(0.03)	2.4	1.6	(0.8, 3.2)
Hallucinogen dependence	1.8	(0.22)	0.1	(0.01)	36.8	23.2	(13.5, 40.0)

[1]DSM–IV = *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.*
[2]Abuse and/or dependence, abuse only, or dependence only.
[3]Comparisons between alcohol abuse versus drug-specific abuse, alcohol dependence versus drug-specific dependence, and alcohol abuse and/or dependence versus drug-specific abuse and/or dependence.
[4]SE = standard error.
[5]Odds ratio adjusted for age, sex, and ethnicity.

alcohol use disorders (18.17 percent). Conversely, 23.1 percent of the respondents with an alcohol use disorder also reported a drug use disorder, a comorbidity rate significantly greater than the population base rate of drug abuse and dependence combined (6.1 percent). These results strongly support the growing trend in recent years toward integrating drug abuse and alcoholism treatment programs.

The findings from this study largely confirm the results of the ECA conducted in the early 1980's. The associations between alcohol and drug abuse, dependence, and abuse and/or dependence were 2.9, 10.9, and 9.6, respectively. These odds ratios were strikingly similar to those reported by Helzer and Pryzbeck (1988) from the ECA (OR's = 3.9, 11.2, and 7.2, respectively). Although not strictly comparable, the associations found in the study for abuse and/or dependence closely corresponded to those reported from the ECA for sedatives (NLAES: OR = 14.6; ECA: OR = 16.9), amphetamines (NLAES: OR = 11.4; ECA: OR = 11.1), and cannabis (NLAES: OR = 9.3; ECA: OR = 6.0). In contrast, the association between alcohol abuse and/or dependence and hallucinogen abuse and/or dependence was greater in the NLAES (OR = 17.1) than in the ECA (OR = 10.9), whereas the corresponding association for cocaine was greater in the ECA (OR = 36.3) than in the NLAES (OR = 12.0) (Regier et al. 1990). Discrepancies between the NLAES and ECA findings may result from differences in the sampling frame and sample size, the diagnostic interview schedules, or the diagnostic criteria used to formulate the diagnoses. It is also likely that changes in alcohol and drug use practices in the United States during the 10-year period since the ECA was conducted have altered the relationships between alcohol and drug use disorders.

The associations between alcohol abuse and drug abuse, regardless of drug type, were consistently found to be smaller than those between alcohol and drug dependence. These results provide some support for the widespread view that abuse is a less severe form of the disorder than dependence and that fewer complications, such as comorbid drug abuse, would be expected with a less severe disorder. Recall that the DSM–IV defines substance abuse separately from dependence, as social, occupational, legal, and interpersonal consequences arising from drinking. The manual relegates indicators of patterns of compulsive drinking (e.g., impaired control over drinking, giving up important activities to drink) and tolerance and withdrawal to the dependence category. Alternatively, the abuse associations may be smaller than the dependence associations because the dependence construct may be defined more diffusely than the abuse construct.

GLOSSARY

Cluster sampling: A sampling method in which each sampling unit is a collection of persons, units, or elements of interest.

Confidence interval for an odds ratio: When 95-percent confidence intervals are constructed around a sample estimate of an *odds ratio,* it means that we are 95-percent certain that the true population odds ratio (which is unknown) lies within the confidence interval. With respect to the intervals surrounding an odds ratio, the association is positive and statistically significant if the interval values are both positive and the interval does not encompass the value of 1.0 (i.e., the value of no association). Conversely, if the interval values are both negative, the association is negative and statistically significant.

Odds ratio: A measure of association between two variables (e.g., an alcohol use disorder and a drug use disorder).

Simple random sample: A method of drawing samples such that each person, element, or unit has an equal probability of being selected.

Stratification: The classification of all persons, units, or elements of interest into comprehensive, mutually exclusive categories.

Variance estimation procedures: A technique that allows estimation of the amount of dispersion around a measure of data, such as a percentage or mean.

The results of this study highlight the importance of comorbid alcohol use disorders and drug use disorders. Alcohol use disorders have been documented to increase both morbidity and mortality among persons with drug use disorders, primarily due to liver dysfunction (Maddux and Elliott 1975), overdose deaths (Baden 1970), and continued drug use (Mezritz et al. 1975). In view of the extent of comorbid alcohol and drug use disorders in the general population and the resultant adverse consequences, it would seem imperative to sharpen our efforts with regard to identifying comorbid patients and designing specific treatment modalities for them. With reference to detection, health professionals in all treatment sectors should be alert to comorbid alcohol and drug use disorders, particularly professionals in primary care, in which physicians have been shown to be less successful in diagnosing patients with alcohol and drug use disorders (Gold and Dackis 1986).

Although the results of this study have answered basic questions about the distribution of comorbidity between alcohol and drug use disorders, future research using the NLAES data will focus on defining the heterogeneity of people with alcohol and drug use disorders. This research will compare respondents with

comorbid alcohol and drug use disorders with respondents who have alcohol use disorders but no history of drug use disorders and respondents who have drug use disorders but no history of alcohol use disorders. This comparison would identify important subgroups of substance use disorders that may be distinguished by differences in sociodemographic factors, family history profiles, and associated psychopathology. The identification of important subgroups of substance use disorders should both further our efforts to more readily identify those with alcohol and drug use problems and aid in specific treatment planning.

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Alcohol and Drug Use, Abuse, and Dependence Among Welfare Recipients

Bridget F. Grant and Deborah A. Dawson

Objectives: *This paper presents national estimates of heavy drinking, drug use, and alcohol and drug abuse and/or dependence among recipients of selected welfare programs.* Methods: *Data from the 1992 National Longitudinal Alcohol Epidemiologic Survey were analyzed.* Results: *The percentages of welfare recipients using, abusing, or dependent on alcohol or drugs were relatively small and consistent with the general U.S. population and those not receiving welfare benefits.* Conclusions: *Although a minority of welfare recipients have alcohol or drug problems, substance abuse prevention and treatment services are needed among high-risk subgroups.*

INTRODUCTION

The network of federal programs designed to help the nation's needy has rapidly grown since the Great Depression, most notably as the result of the War on Poverty. Recent concerns regarding these programs, referred to collectively as welfare, have generated great debate in the current administration and among lawmakers. At the center of this often intense and emotional political debate are characterizations of welfare recipients that are usually not supported by empirical data. One such characterization depicts the welfare mother in particular as having an alcohol or drug problem. It was the objective of the present study to provide the most recent national estimates of the prevalence of heavy alcohol use, drug use, and alcohol and drug abuse and dependence among welfare recipients participating in five social services programs: Aid to Families with Dependent Children (AFDC); the Special Supplemental Food Program for Women, Infants, and Children (WIC); food stamps; supplemental security income (SSI); and Medicaid. The major goal of the study was to identify high-risk subgroups of the welfare population in need of prevention, intervention, and treatment of alcohol and drug problems.

METHODS

The data presented in this report were collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES) designed by the National Institute on Alcohol Abuse and Alcoholism (NIAAA), with fieldwork conducted by the Bureau of the Census. Direct face-to-face interviews were conducted with one randomly selected respondent, 18 years of age or older, in each of 42,862 households within the contiguous United States and the District of Columbia. The household response rate for the NLAES was 91.90%, and the individual response rate was 97.4%. The sampling design of the survey involved stratification and clustering, along with oversampling of blacks and young adults (18 to 29 years of age); the design has been described more fully elsewhere.[1,2] Because of the complex multistage nature of the survey, SUDAAN,[3] a software program that uses appropriate statistical techniques to adjust for sample design characteristics, was used to generate the prevalence estimates and statistical tests presented in this report. Statistical comparisons among sociodemographic subgroups of the welfare population were accomplished by means of *t* tests ($P > .01$ denoting significance due to multiple comparisons).

NLAES respondents were asked whether they received AFDC, WIC, SSI or Medicaid payments, or food stamps during the month prior to the interview. The survey estimates of the number of adults covered by these programs were very similar to those derived from the 1992 Current Population Survey[4] and 1992 program statistics derived from the federal agencies that administer such programs: the Department of Health and Human Services (HHS) for AFDC (L. Carrera, written communication, Administration for

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Children and Families, Office of Family Assistance), Medicaid[5] and SSI (J. Schmulowitz, written communication, Office of Research Statistics, Social Security Administration), and the Department of Agriculture for WIC[6] and food stamps.[7] It should be noted that the NLAES estimates of program participation presented in this report do not include recipients under the age of 18 years or recipients residing in institutions, nor do they reflect multiple recipients of welfare programs residing in the same household. Furthermore, recipients may also have been receiving support from more than one welfare program during the previous year.

In this report, heavy drinking was operationalized as an average daily ethanol consumption exceeding 1 oz (28 g) (i.e., more than two drinks per day) or consumption of five or more drinks on at least 12 occasions (i.e., once a month or more) during the previous year. Any drug use was defined as taking of any of the following medicines or drugs "on your own" (i.e., without a prescription) at least 12 times during the previous year: sedatives, tranquilizers, opioids other than heroin, amphetamines, cannabis (including hashish), methadone, heroin, or other drugs such as hallucinogens, inhalants, or solvents. Diagnoses of alcohol or drug abuse and/or dependence were obtained from lists of symptom items operationalizing definitions appearing in the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; DSM-IV).[8] Respondents classified as abusers were required to meet at least one of the following criteria: continued use despite social or interpersonal problems, hazardous use, legal problems, and neglect of role obligations. A diagnosis of dependence required affirmative responses to three or more of the following seven criteria: tolerance; withdrawal; unsuccessful attempts or persistent desire to stop use; use for longer or in larger amounts than intended; activities given up in favor of use; time spent in obtaining, using, or recovering from substance effects; and continued use despite physical or psychological problems. The complex algorithms designed to yield these DSM-IV diagnoses have been defined in detail elsewhere.[9] The reliabilities of alcohol use, abuse and dependence measures used in this study exceeded .73 (kappa coefficients), as ascertained from an independent test-retest study conducted in a general population sample.[10] Similarly, kappa coefficients associated with drug use, abuse, and dependence measures exceeded .79.

RESULTS

The prevalence of heavy drinking was similar among recipients of AFDC (13.2%), WIC (11.5%), and food stamps (13.8%) (Table 1) and somewhat lower among recipients of SSI (6.4%) and Medicaid (10.3%) (Table 2). Prevalences of DSM-IV alcohol abuse and/or dependence among recipients of AFDC (7.6%), WIC

Table 1. Prevalence of Past Year Heavy Drinking and DSM-IV Alcohol Abuse and/or Dependence among Recipients of Aid to Families with Dependent Children (AFDC), Supplemental Food Program for Women, Infants, and Children (WIC), and Food Stamps, by Sex, Race, and Age

	AFDC[a]			WIC[b]			Food Stamps[c]		
	Population Estimate (Thousands)	Heavy Drinking, % (SE)	Alcohol Abuse/ Dependence, % (SE)	Population Estimate (Thousands)	Heavy Drinking, % (SE)	Alcohol Abuse/ Dependence, % (SE)	Population Estimate (Thousands)	Heavy Drinking, % (SE)	Alcohol Abuse/ Dependence, % (SE)
Sex									
Male	559.7	20.9 (4.4)	9.6 (2.9)	974.3	26.0 (3.8)	18.6 (3.3)	2 818.9	24.9 (2.1)	13.9 (1.7)
Female	3 523.0	12.0 (1.2)	7.3 (1.0)	3 250.3	7.1 (0.9)	4.7 (0.8)	7 256.6	9.5 (0.7)	5.9 (0.6)
Race									
Black	1 495.8	10.2 (1.4)	7.2 (1.3)	1 077.9	7.9 (2.1)	7.4 (2.1)	3 210.1	13.0 (1.3)	8.6 (1.1)
Non-Black	2 586.9	15.0 (1.8)	7.8 (1.3)	3 146.6	12.7 (1.3)	8.1 (1.1)	6 865.4	14.2 (1.0)	8.0 (0.8)
Age, y									
18–24	1 113.6	11.5 (2.4)	7.2 (1.6)	1 778.8	10.7 (1.7)	6.6 (1.4)	2 035.9	13.2 (1.6)	8.1 (1.2)
25–34	1 655.0	17.6 (1.9)	10.7 (1.9)	1 850.3	12.9 (1.8)	9.1 (1.8)	3 227.4	17.8 (1.5)	11.1 (1.4)
35+	1 314.1	9.2 (1.7)	3.9 (1.0)	595.4	9.4 (2.4)	6.2 (2.4)	4 812.2	11.4 (1.2)	6.2 (0.8)
Total	4 082.8	13.2 (1.3)	7.6 (0.9)	4 224.5	11.5 (1.1)	7.9 (1.0)	10 075.5	13.8 (0.8)	8.2 (0.6)

Note. As a result of rounding, components may not add to totals.
[a]Cash assistance to needy children who lack the financial support of one parent because that parent is continuously absent from the home, incapacitated, dead, or unemployed.
[b]Nutrition program to improve the nourishment of pregnant and postpartum women, infants, and children under 6 years of age.
[c]Used to purchase food; intended to permit members of low-income households to obtain a more nutritious diet.

(7.9%), and food stamps (8.2%) (Table 1) were also greater than comparable prevalences among SSI (4.3%) and Medicaid (5.2%) recipients (Table 2). Similarly, rates of any drug use ranged from 7.2% to 9.8% for AFDC, WIC and food stamp recipients but were lower among recipients of SSI (3.8%) and Medicaid (6.0%). Overall, the rates of drug abuse and/or dependence were also greater for recipients of AFDC, WIC, and food stamps (2.5% to 3.6%) than for SSI (1.3%) and Medicaid (2.0%) recipients.

Prevalences of heavy drinking and of alcohol abuse and/or dependence were significantly greater for men than for women ($P < .01$) in each welfare program except AFDC. There were no sex differences found for drug use or drug use disorder measures across welfare programs. Rates of heavy drinking, drug use, and alcohol and drug abuse and or dependence were also not significantly different between nonblack and black recipients of AFDC, WIC, food stamps, SSI, and Medicaid.

Among AFDC and food stamp recipients, but not WIC recipients, heavy drinking, drug use, and alcohol and drug abuse and/or dependence were more prevalent ($P < .01$) in the 25- to 34-year age group than in

Table 2. Prevalence of Past Year Heavy Drinking and DSM-IV Alcohol Abuse and/or Dependence among Recipients of Supplemental Security Income (SSI) and Medicaid, by Sex, Race, and Age

	SSI[a]			Medicaid[b]		
	Population Estimate (Thousands)	Heavy Drinking, % (SE)	Alcohol Abuse/ Dependence, % (SE)	Population Estimate (Thousands)	Heavy Drinking, % (SE)	Alcohol Abuse/ Dependence, % (SE)
Sex						
Male	1 545.5	11.3 (2.2)	7.8 (1.9)	3 620.8	15.8 (1.5)	7.5 (1.1)
Female	2 670.7	3.6 (0.7)	2.2 (0.5)	8 447.9	7.9 (0.7)	4.3 (0.4)
Race						
Black	1 140.9	7.9 (1.6)	4.8 (1.2)	3 145.1	11.0 (1.1)	5.9 (0.8)
Non-Black	3 074.5	5.9 (1.1)	4.1 (0.9)	8 923.6	9.9 (0.8)	4.9 (0.6)
Age, y						
18–29	593.4	6.8 (3.1)	5.4 (2.9)	3 720.9	12.9 (1.2)	8.4 (0.9)
30–54	1 332.2	9.4 (1.8)	8.1 (1.7)	3 667.0	13.8 (1.3)	7.7 (1.0)
55+	2 290.6	4.6 (1.0)	1.8 (0.6)	506.8	5.3 (0.7)	0.8 (0.2)
Total	4 216.3	6.4 (0.9)	4.3 (0.8)	12 068.7	10.3 (0.6)	5.2 (0.5)

[a]Provides cash benefits, paid monthly, to elderly, blind, and disabled persons who are financially needy.
[b]Furnishes medical assistance on behalf of needy families with dependent children and on behalf of elderly, blind, or permanently and totally disabled individuals whose incomes and resources are insufficient to meet the costs of necessary medical services.

Table 3. Prevalence of Past Year Drug Use and DSM-IV Drug Abuse and/or Dependence among Recipients of Aid to Families with Dependent Children (AFDC), Supplemental Food Program for Women, Infants, and Children (WIC), and Food Stamps, by Sex, Race, and Age

	AFDC			WIC			Food Stamps		
	Population Estimate (Thousands)	Any Drug Use, % (SE)	Drug Abuse/ Dependence, % (SE)	Population Estimate (Thousands)	Any Drug Use, % (SE)	Drug Abuse/ Dependence, % (SE)	Population Estimate (Thousands)	Any Drug Use, % (SE)	Drug Abuse/ Dependence, % (SE)
Sex									
Male	559.8	10.3 (3.1)	5.6 (2.3)	974.3	11.0 (3.0)	18.6 (3.3)	2 818.9	11.6 (1.5)	3.6 (0.8)
Female	3 523.0	9.7 (0.9)	3.3 (0.5)	3 250.3	6.0 (0.8)	4.7 (0.8)	7 256.6	7.2 (0.6)	2.4 (0.4)
Race									
Black	1 495.8	7.4 (1.0)	2.86 (0.7)	1 077.9	4.5 (1.2)	1.2 (0.5)	3 210.1	7.5 (1.0)	1.9 (0.4)
Non-Black	2 586.9	11.1 (1.2)	4.09 (0.7)	3 146.6	8.1 (1.1)	2.9 (0.7)	6 865.4	8.8 (0.7)	3.1 (0.4)
Age, y									
18–24	1 113.6	14.6 (2.2)	3.9 (1.2)	1 778.8	8.7 (1.5)	3.5 (1.1)	2 035.9	13.9 (1.7)	3.6 (0.9)
25–34	1 655.1	10.7 (1.5)	4.3 (0.9)	1 850.3	5.9 (1.2)	1.7 (0.6)	3 227.4	11.3 (1.2)	3.8 (0.7)
35+	1 314.0	4.7 (1.0)	2.5 (0.8)	595.4	6.7 (2.3)	2.0 (1.1)	4 812.2	4.1 (0.6)	1.6 (0.4)
Total	4 082.8	9.8 (0.9)	3.6 (0.5)	4 224.5	7.2 (0.9)	2.5 (0.6)	10 075.5	8.4 (0.6)	2.7 (0.3)

Note. As a result of rounding, components may not add to totals. See Table 1 for program descriptions.

the oldest (35 years and older) age group. For SSI and Medicaid recipients, the rates of heavy drinking, drug use and alcohol and drug abuse and/or dependence were greatest among 30- to 54-year-olds. For Medicaid recipients, rates of all four alcohol and drug problem measures were also greater ($P < .01$) among 18- to 29-year-olds than among those in the oldest age group.

DISCUSSION

Contrary to common characterizations of the welfare population as having alcohol and drug problems, the results of this study indicate that small percentages of AFDC, WIC, food stamp, SSI, and Medicaid recipients are heavy drinkers (6.4% to 13.8%), use drugs (3.8% to 9.8%), or abuse or are dependent on alcohol (4.3% to 8.2%) or other drugs (1.3% to 3.6%). These rates among welfare recipients were similar to national estimates derived from the NLAES survey for heavy drinking (14.5%), any drug use (5.0%), alcohol abuse and/or dependence (7.4%), and drug abuse and, or dependence (1.5%).[9] Also, they are comparable to rates of heavy drinking (14.8%), drug use (5.1%), alcohol abuse and/or dependence (7.5%), and drug abuse and/or dependence (1.5%) among the subpopulation of the United States not receiving welfare benefits.

In general, the sociodemographic differentials associated with heavy drinking and alcohol abuse and/or dependence observed in the U.S. general population were not entirely preserved within the subgroup of welfare recipients. In the general population, rates for all four alcohol and drug problem indicators are greater among men than among women. Among welfare recipients, indicators of alcohol problems were greater for men than for women, except AFDC recipients, but no sex differences were found in the rates of drug problem indicators. Although the prevalences of heavy drinking, drug use, and alcohol and drug abuse and/or dependence are generally greater among nonblacks than among blacks in the general population, no ethnic differences were found in these measures among welfare recipients. There was, however, a trend for the rates of each problem indicator to be greater among nonblacks than among blacks, except among SSI and Medicaid recipients. In addition, its comparisons with those in the older age group, heavy drinking, drug use, and alcohol and drug abuse and/or dependence were significantly greater among 25- to 34-year-old recipients of AFDC and food stamps, but not WIC, and among 30- to 54-year-old SSI and Medicaid recipients.

Although the reasons for the observed sex, ethnic, and age differentials in terms of alcohol and drug problem indicators among welfare recipients remain unclear, more substantive future analyses carried out within a multivariate environment may clarify the questions raised in this study. However, this study has achieved its purpose of identifying high-risk subgroups of the welfare populations in need of alcohol and drug prevention and treatment programs. Education programs, screening efforts, and provisions for treatment of alcohol and drug problems could be incorporated into the welfare system of social services, just as provisions currently exist for training and employment services for AFDC recipients and nutrition education for

Table 4. Prevalence of Past Year Drug Use and DSM-IV Drug Abuse and/or Dependence among Recipients of Supplemental Security Income (SSI) and Medicaid, by Sex, Race, and Age

	SSI			Medicaid		
	Population Estimate (Thousands)	Any Drug Use, % (SE)	Drug Abuse/ Dependence, % (SE)	Population Estimate (Thousands)	Any Drug Use, % (SE)	Drug Abuse/ Dependence, % (SE)
Sex						
Male	1 545.5	4.3 (1.2)	1.3 (0.4)	3 620.7	5.9 (0.9)	2.4 (0.6)
Female	2 678.7	3.5 (0.9)	1.3 (0.4)	8 447.9	6.0 (0.5)	1.9 (0.3)
Race						
Black	1 140.9	5.4 (1.9)	1.5 (0.8)	3 145.1	6.1 (0.9)	1.9 (0.4)
Non-Black	3 075.4	3.2 (0.7)	1.2 (0.3)	8 923.6	6.0 (0.6)	2.1 (0.3)
Age, y						
18–29	593.1	4.0 (1.6)	1.1 (0.7)	3 720.9	11.5 (1.0)	3.4 (0.6)
30–54	1 332.3	8.8 (1.8)	3.6 (0.8)	3 667.1	7.4 (0.9)	3.3 (0.6)
55+	2 290.6	0.8 (0.4)	0.0 (0.0)	4 680.7	0.5 (0.2)	0.0 (0.0)
Total	4 216.3	3.8 (0.7)	1.3 (0.3)	12 068.7	6.0 (0.5)	2.0 (0.3)

Note. As a result of rounding, components may not add to totals. See Table 2 for program descriptions.

WIC recipients. The implementation of prevention programs and treatment services for alcohol and drug problems among welfare recipients would foster and facilitate the major goals of work, responsibility, and reduction of dependency that form the basis of the proposed welfare reform.

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CHAPTER 3
MEDICAL CONSEQUENCES OF ALCOHOL CONSUMPTION

Medical Consequences of Alcohol Consumption—United States, 1992

S. Patricia Chou, Bridget F. Grant, and Deborah A. Dawson

There is plenty of evidence in the alcohol literature that chronic excessive use of alcohol poses a threat to every organ system in the body. At the same time, there is a growing consensus that drinking in moderation protects against cardiovascular disease. This study was based on the most recent national household survey of the United States general population on drinking practices, alcohol use disorders, and their associated disabilities. The prevalences of major alcohol-related diseases were examined across different categories of drinking status. Excess morbidity caused by heavy intake of alcohol was also studied. Results were generally in agreement with the popular belief that light or moderate drinking is beneficial relative to abstention, particularly that moderate alcohol consumption confers a beneficial cardiovascular effect. Our findings also pointed toward the injurious effect of heavy alcohol use. However, results on benefits of drinking must be interpreted with caution.

Over the past three decades, an impressive body of literature has provided evidence that alcohol use is responsible for considerable morbidity and mortality, as well as social or legal problems. Injuries, especially motor vehicle crashes caused by alcohol use, also contribute substantially to medical costs and premature death in the United States (U.S.). The adverse medical consequences directly related to alcohol consumption can be physical illnesses or psychiatric conditions, including drug use disorders and alcoholism. Although there is a diverse range of alcohol-related problems, this study only focused on physical morbidity associated with the adverse effects of alcohol.

Among the wide range of alcohol-induced disorders, alcohol has been shown to be directly toxic to the liver. Being the primary site for detoxification of alcohol by oxidation to its metabolites, the level and duration of alcohol consumption are important determinants in the development of liver pathology, including fatty liver, alcoholic hepatitis, and cirrhosis. The adverse effects of alcohol on the pancreas have also long been recognized. Being one of the leading causes of acute and chronic pancreatitis,[1,2] alcohol accounts for ~65% of all cases of pancreatitis.[3] Furthermore, alcohol impairs or causes structural or functional changes of the mucosa of the stomach or intestine.

Alcohol affects the cardiovascular system in many different ways. Even though there is considerable evidence that moderate drinking protects against mortality and morbidity from coronary heart disease,[4–6] heavy consumption is shown to have deleterious cardiovascular effects. It exerts its adverse effects by increasing the risks of cardiomyopathy, hypertension, arrhythmias, and cerebrovascular hemorrhage.[7] Among these conditions, hypertension is identified as a major risk factor for hemorrhagic stroke and myocardial infarction.[8]

In studying alcohol's link to cancer, epidemiological and basic research into the underlying mechanism have demonstrated that alcohol increases the risk for various cancers. Specifically, evidence has suggested a strong association of alcohol and cancers of the upper digestive tract, including the esophagus, mouth, pharynx, and larynx.[9,10] There are somewhat weaker associations relating alcohol to cancers of the liver, breast, and colon.[9]

There are additional medical conditions that are more prevalent among alcoholics than in the general population. Alcohol interferes with the metabolism of most vitamins and some nutrients. The deficiencies of iron, thiamine, folate, vitamin A, and zinc may result from impaired absorption, as well as from poor nutrition.[11,12] For lung diseases, Burch and De Pasquale[13] first suggested that alcohol might have an injurious effect on the lungs by virtue of excreting alcohol through the lungs. Found more commonly among alcoholics than in the general population are certain nontuberculous lung diseases, including emphysema with mucus hypersecretion, airway obstruction, and impairment of diffusion.[14,15]

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Despite the potential health hazards associated with alcohol use, the putative beneficial aspects of light and moderate consumption with respect to coronary heart disease have been widely disseminated in the media and among the general public that light and moderate drinking protects against cardiovascular disease. Ongoing debate has centered on whether such a phenomenon is genuine or artifactual, and has led to a series of investigations into the biological mechanism underlying it.[16–18] However, amidst the controversy surrounding the benefits of drinking, there is unequivocal evidence that prolonged excessive alcohol consumption can cause serious health consequences, because virtually no part of the body is spared from its adverse effects.[12]

The major purpose of this study was to present the current prevalence rates of major alcohol-related diseases, using the most recent nationally representative sample of the U.S. general population. The prevalence rates of alcohol-related diseases were examined across different drinking statuses. The putative protective effects of light and moderate consumption and excess morbidity associated with heavy alcohol use were also examined.

METHODS

Data source

The National Longitudinal Alcohol Epidemiologic Survey (NLAES) is a multipurpose survey conducted to comply with the Anti-Drug Abuse Act of 1988. One major purpose of this survey was to measure the prevalence of alcohol use disorders (i.e., alcohol abuse and dependence) and their associated disabilities (drug-specific abuse and dependence, major depression, and physical disorders). This survey was targeted at the civilian noninstitutionalized population of the contiguous United States, 18 years and older. The multistage-stratified sample design was similar to the 1985 redesign of the National Health Interview Survey.[19, 20] Primary sampling units (PSUs) were stratified according to major sociodemographic characteristics and selected with probability proportional to size. In all, 198 of a sampling frame of ~2,000 PSUs were selected. Within PSUs, geographically defined secondary sampling units (segments) were selected systematically. Segments then were divided into clusters of ~4 to 8 housing units, and all occupied housing units were included in the sample. In oversampling, the sampling intervals were increased in areas where there were the highest concentration of Blacks. To compensate for the effect of oversampling, the sampling intervals in other areas were decreased. Young adults (ages 18 through 29) were also oversampled at the household level. Overall, there were 42,862 completed interviews, with 92% and 97% response rates, respectively, at the household and sample person levels.

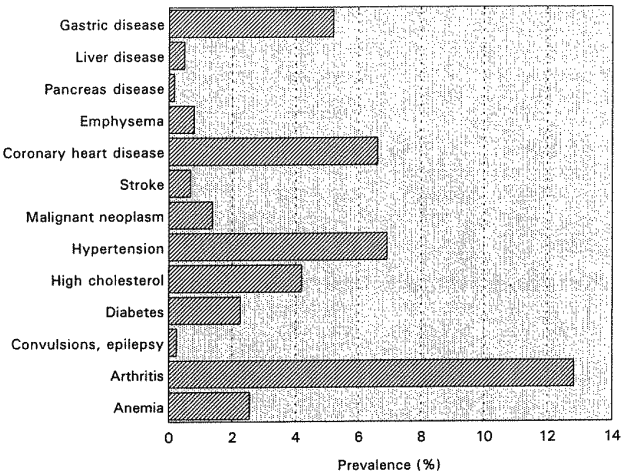
Disease-specific morbidity

There were 24 survey questions dealing with various medical conditions. To estimate the 12-month prevalence of physical morbidity, we excluded mental or emotional problems and considered only conditions that were reported to have caused any problems during the last 12 months, and that have ever been diagnosed by a doctor or other health professional. The opening statement of the section on medical conditions reads, "Now I am going to read you a list of medical conditions that you may have had. As I read each one, please tell me whether you have ever had it." Then for each condition, the respondent was asked, "Have you EVER had (condition)?" To ascertain the time frame when the specific morbid condition occurred, the respondent was asked, "Did (condition) cause you any problems during the last 12 months?" As to whether it was ever diagnosed, the respondent was asked, "Did a doctor or other health professional tell you that you had (condition)?" In calculating the odds ratios, we used the disease-specific morbidity as the outcome measure and the drinking status as the exposure measure. When prevalence rates of certain conditions were too low to generate meaningful results, similar conditions were grouped into broader categories. For example, coronary heart disease consisted of the conditions of arteriosclerosis, angina pectoris, myocardial infarction, and other heart disease. Liver disease encompassed the conditions of enlarged liver, yellow jaundice, liver cirrhosis, hepatitis, and other liver disease. All of the alcohol-related medical conditions were grouped according to the major ICD-10[21] categories as shown in "Results."

Consumption measure

We used a summary consumption measure, the past-year average daily intake of ethanol. This measure was derived based on usual and heaviest past-year consumption,[22] as described herein. The annual volume for each beverage type was the sum of two consumption items: one based on usual intake and one based on heaviest intake. The usual frequency of intake was adjusted by subtracting out the frequency of heaviest intake to avoid double-counting on drinking days. The total volume of ethanol intake was aggregated over the

Figure 1. 12-Month Prevalence of Alcohol-Related Medical Conditions



separate consumption items for beer, wine, and liquor, and was divided by 365 to derive an average daily intake of ethanol. Ounces of beverage were converted to ounces of ethanol using the conversion factors 0.045 for beer, 0.121 for wine, and 0.409 for liquor.[23–25] The average ounces of beverage for beer, wine, and liquor were 12, 8, and 1.5, respectively.

Drinking status

Among all NLAES respondents, 44% were current drinkers, 34% were lifetime abstainers, and 22% were former drinkers. For this analysis, current drinkers were defined as individuals who consumed at least 12 drinks during the past 12 months. Former drinkers were persons who had not consumed 12 or more drinks in the past year, but had done so in previous years. Lifetime abstainers were those who had never consumed 12 or more drinks in any 1 year. Among current drinkers, light drinkers were defined as those who drank <0.22 oz of ethanol/day on average.[26] This level is equivalent to <3 drinks/week. Moderate drinkers were those who drank an average of 0.22 to 1.0 oz of ethanol/day (equivalent to 3 drinks/week to 2 drinks/day). Moderately heavy drinkers consumed >1.0 oz of ethanol (two standard drinks), but no more than 2.5 oz/day on average. Very heavy drinkers consumed >5 standard drinks/day. Lastly, alcoholics roughly refer to those individuals who met the diagnostic criteria for alcohol use disorders.

Statistical analysis

Morbidity rates frequently vary by age and sex, and occasionally by race as well. These factors are also associated with drinking patterns. In order that demographic differences do not confound the apparent associations between consumption and morbidity, all prevalence rates reported herein were standardized to reflect the distribution of the U.S. adult population in terms of sex, race (Black versus non-Black), and age (18 to 29, 30 to 44, 45 to 64, and 65 or older). In estimating the alcohol-morbidity association, we restricted the odds ratios calculation to current drinkers so that the disease-specific morbidity and the consumption measure covered the same time period. In addition, all standard errors of prevalence or odds ratios were computed using the SUDAAN software.[27] Its statistical procedures use the first-order Taylor series approximation to take into account the stratified and clustered sampling characteristics of the NLAES.

RESULTS

The overall 12-month prevalences of various medical conditions are shown in Fig. 1 and presented as the total column in Table 1. The most prevalent medical condition in the general population was arthritis (12.85%), followed by hypertension (6.91%), and coronary heart disease (6.6%). A substantial proportion of the U.S. population reported diseases of the stomach (5.20%) and high cholesterol or lipid content (4.18%). The prevalences of several physical disorders were very low (e.g., <0.5%) for liver disease, diseases of the pancreas, and convulsions or epilepsy.

To account for the potential confounding of smoking on health outcomes,[28] the prevalence rates of alcohol-related medical conditions were first generated based on stratifying the respondents according to smoking (ever versus never smoker) and drinking status. The results indicated that there was no significant interaction between smoking and drinking (data not shown). Further, to ensure that there were an adequate number of cases in each resultant stratum for analytical purposes, the prevalence rates or odds ratios presented in Tables 1 and 2 were based on stratifying the drinking status alone.

There were about the same number of light and moderate drinkers, each comprising ~40% of all current drinkers. Fourteen percent of current drinkers were classified as moderately heavy drinkers, whereas only half of that were very heavy drinkers. Table 1 presents the 12-month prevalence rates of alcohol-related medical conditions across different categories of drinking status. The lowest prevalence rates were observed among light and moderate drinkers. Furthermore, the various prevalences between these two groups were similar to each other except for the conditions of high

cholesterol or lipid content, diseases of the stomach, and arthritis, for which moderate drinking showed beneficial effects. Abstainers generally had higher prevalence rates of various medical conditions than did light or moderate drinkers. Most significantly, the rates of stomach disease, coronary heart disease, cerebrovascular accident, and hypertension among abstainers were substantially higher than those among light and moderate drinkers. In addition, the rates among abstainers were comparable with those among moderately heavy drinkers, with the exception of coronary heart disease and hypertension, for which the rates among abstainers were substantially greater than those among moderately heavy drinkers. The elevated prevalence of physical disorders among very heavy drinkers, compared with light, moderate or moderately heavy drinkers, was also apparent. In all, former drinkers and very heavy drinkers manifested the most physical symptoms among all types of drinkers.

Table 2 addresses both concerns that heavy alcohol consumption poses special dangers for physical health, at the same time light-to-moderate drinking affords protection for various physical disorders. Because of the similarity of the resulting odds ratios of the two drinking groups, light and moderate drinkers were combined. Using the combined drinking group as the reference category, Table 2 shows odds ratios for various disorders, first in relation to lifetime abstainers and second in relation to very heavy drinkers. The 95% confidence limits provide a range within which the true odds ratios might actually take values at a 95% confidence level. The confidence limits encompassing 1.0 indicate that the odds of experiencing certain morbid conditions of the given drinking category (abstention or very heavy) in relation to light-to-moderate drinking are not significantly different than one. As shown in column 1, all odds ratios but that of liver disease were significantly >1 suggesting the beneficial health effects of light-to-moderate drinking. On the other hand, when alcohol intake exceeded 5 drinks/day among very heavy drinkers, only diseases of the digestive, respiratory, and circulatory (except cerebrovascular accident) systems and malignant neoplasm exhibited greater risks than among light-to-moderate drinkers. As expected, those diseases with extremely low prevalences (i.e., diseases of the pancreas and convulsions) yielded fairly wide confidence limits. However, the results distinctly indicated that heavy alcohol intake poses serious health concerns for diseases of the digestive system. This was particularly true for diseases of the liver and pancreas. For liver disease, the harmful effect of heavy alcohol use was reflected in elevated odds that were six times greater than those of light-to-moderate drinking. Likewise, the magnitude

Table 1. 12-Month Prevalence Rates* of Alcohol-Related Medical Conditions by Drinking Status

					Current drinker			
Medical condition	*n*= Total (42,862)	Abstainer (15,246)	Former drinker (9,264)	Light (7,427)	Moderate (7,124)	Moderately heavy (2,577)	Very heavy (1,224)
Digestive disease							
Stomach	5.20 (0.13)†	5.00 (0.23)	6.70 (0.31)	4.58 (0.30)	3.85 (0.26)	5.09 (0.60)	7.04 (1.60)
Liver	0.49 (0.04)	0.39 (0.06)	0.62 (0.09)	0.30 (0.07)	0.33 (0.08)	0.51 (0.14)	2.11 (0.66)
Pancreas	0.17 (0.03)	0.12 (0.04)	0.30 (0.07)	0.08 (0.03)	0.07 (0.04)	0.01 (0.01)	0.57 (0.43)
Respiratory disease							
Emphysema	0.79 (0.05)	0.55 (0.07)	1.42 (0.13)	0.52 (0.10)	0.65 (0.17)	0.60 (0.16)	0.96 (0.43)
Circulatory disease							
Coronary heart disease	6.60 (0.15)	6.40 (0.25)	8.57 (0.33)	5.27 (0.32)	5.15 (0.36)	4.46 (0.47)	7.69 (1.74)
Cerebrovascular accident	0.68 (0.04)	0.75 (0.08)	0.84 (0.10)	0.29 (0.07)	0.50 (0.11)	0.65 (0.22)	0.95 (0.70)
Hypertension	6.91 (0.17)	7.32 (0.29)	7.79 (0.31)	5.60 (0.35)	5.39 (0.35)	5.99 (0.63)	7.52 (1.68)
Neoplasm							
Malignant neoplasm	1.38 (0.07)	1.14 (0.10)	1.82 (0.15)	1.16 (0.15)	1.31 (0.20)	2.02 (0.39)	4.18 (1.49)
Endocrine, nutritional, and metabolic disease							
High cholesterol or lipid contents	4.18 (0.13)	3.69 (0.21)	5.06 (0.25)	4.04 (0.28)	3.34 (0.26)	4.35 (0.60)	3.96 (0.82)
Diabetes	2.26 (0.09)	2.71 (0.18)	3.21 (0.21)	1.06 (0.15)	1.06 (0.17)	1.24 (0.30)	0.97 (0.39)
Disease of the nervous system							
Convulsions or epilepsy	0.26 (0.03)	0.45 (0.08)	0.29 (0.06)	0.13 (0.05)	0.18 (0.06)	0.06 (0.05)	0.48 (0.28)
Disease of the musculoskeletal system and connective tissue							
Arthritis	12.85 (0.19)	11.62 (0.27)	16.06 (0.45)	11.80 (0.49)	10.28 (0.44)	11.77 (0.88)	11.58 (1.84)
Disease of the blood and blood-forming organs, and certain disorders involving the immune mechanism							
Anemia or vitamin deficiencies	2.56 (0.09)	2.37 (0.13)	3.36 (0.22)	2.05 (0.17)	2.25 (0.23)	1.87 (0.34)	3.75 (1.13)

* Prevalence rates were standardized according to the distribution of the U.S. general population in terms of sex, race, and age.

† Numbers in parentheses are standard errors of the prevalences.

of the odds ratio for disease of the pancreas strongly suggested a detrimental effect of heavy alcohol use.

DISCUSSION

This study examined the prevalence rates of various medical conditions among different categories of drinking status. The evidence provided herein was generally in agreement with the popular belief that light or moderate drinking is beneficial relative to abstention, and that heavy alcohol consumption increases the risks for various medical complications.

The excess cardiovascular morbidity observed among abstainers, as well as among very heavy drinkers in relation to light, moderate, and moderately heavy drinkers, seemed to support the U-shaped relation between alcohol and cardiovascular morbidity. Thus, the results were in favor of the growing public perception that light-to-moderate drinking confers a protective cardiovascular effect.

The prevalence rates among former drinkers were among the greatest for many medical conditions. This finding is consistent with other studies that nondrinkers are not all alike. Because of the inclusion of sick quitters who stopped drinking due to health or drinking problems, former drinkers tended to exhibit more physical symptoms than among lifetime abstainers, and light or moderate drinkers.[29–31] This morbidity excess could also be attributed to higher consciousness about health issues or greater willingness to comply with medical advice among former drinkers, thus resulting in more diagnosed medical conditions. On the other hand, heavy drinkers, especially alcoholics, tend to avoid any encounter with medical professionals unless a crisis strikes. Both factors could potentially confound the prevalence rates observed among former drinkers. In his review, Shaper[32] argued that failure to distinguish lifelong teetotalers from former drinkers in major prospective studies leads to the controversial U-shaped curve being interpreted as the protective effect of moderate drinking.[28,29,33] However, this study showed that the prevalence rates among lifetime abstainers were generally higher than those among light or moderate drinkers, suggesting that the inclusion of sick quitters in the nondrinker group does not fully account for the higher prevalence rates observed in nondrinkers. An alternative argument[6] is that sick people may not develop the drinking habit. In this sense, lifetime abstainers are a self-select group. They choose not to engage in drinking activities because of their burden of ill health, and this burden is reflected in higher morbidity rates. Others disagree with this speculative explanation. Studies reported that a majority of abstainers gave "did not care to drink," "religious/moral reasons," or "brought up not to drink" as primary reasons for not to engage in drinking.[34]

Data from morbidity studies have shown that excess morbidity among alcoholics is most profound for diseases of the respiratory, circulatory, and digestive systems.[35] For coronary heart disease, alcohol has been found to be an important factor—at high levels in promoting and at moderate levels in protecting against

Table 2. Odds Ratios* of Alcohol-Related Medical Conditions among Abstainers and Very Heavy Drinkers in Reference to Light-to-Moderate Drinkers

Medical condition	Abstain/light to moderate	Very heavy/light to moderate
Digestive disease		
Stomach	1.39 (1.23, 1.57)	2.03 (1.50, 2.76)
Liver	1.43 (0.94, 2.19)	5.82 (3.16, 10.7)
Pancreas	2.22 (1.03, 4.78)	16.68 (3.60, 77.2)
Respiratory disease		
Emphysema	1.63 (1.12, 2.38)	3.42 (1.32, 8.88)
Circulatory disease		
Coronary heart disease	1.84 (1.63, 2.07)	1.59 (1.12, 2.25)
Cerebrovascular accident	3.56 (2.47, 5.14)	1.74 (0.60, 5.00)
Hypertension	2.22 (1.96, 2.51)	1.50 (1.11, 2.01)
Neoplasm		
Malignant neoplasm	1.59 (1.23, 2.05)	2.16 (1.23, 3.79)
Endocrine, nutritional, and metabolic disease		
High cholesterol or lipid content	1.45 (1.26, 1.67)	1.15 (0.78, 1.70)
Diabetes	4.10 (3.24, 5.20)	1.16 (0.58, 2.33)
Disease of the nervous system		
Convulsions or epilepsy	2.74 (1.52, 4.96)	4.35 (0.84, 22.5)
Disease of the musculoskeletal system and connective tissue		
Arthritis	1.98 (1.81, 2.15)	0.93 (0.71, 1.22)
Disease of the blood and blood-forming organ and certain disorders involving the immune mechanism		
Anemia or vitamin deficiencies	1.52 (1.28, 1.79)	0.87 (0.51, 1.47)

* Numbers in parentheses are 95% confidence intervals.

the disease. Our finding is consistent with earlier studies that the prevalence of coronary heart disease among light, moderate, or moderately heavy drinkers was significantly lower than that among lifetime abstainers or former drinkers.[36–39] However, we observed an elevated risk of coronary heart disease among those who drank >5 drinks/day. This finding is consistent with previous studies that excessive drinking may increase the risk of coronary heart disease.[5,40]

Chronic excessive use of alcohol has been found to be the single most important cause of morbidity and mortality from liver disease.[41] The risk of liver disease increases exponentially at a certain level of continuous ethanol ingestion.[42–44] Our results indicated that the odds of liver disease associated with heavy drinking could be as high as six times those observed at a low or moderate drinking level. The observed excess odds of disease of the pancreas because of heavy use of alcohol were also consistent with the evidence that there is a linear relationship between average daily alcohol intake and the risk for pancreatitis.[45]

It has been reported in cancer research that an estimated 2 to 4% of all cancer cases have been thought to be induced directly or indirectly by alcohol.[9] Even though specific types of cancer were not identified in this study, our findings support the notion that heavy alcohol use can contribute to cancer initiation or development.

There are two important reasons why the findings of this study should be considered preliminary. First, being a descriptive presentation of alcohol-related morbidity data, this study did not account for all potential confounding variables in the analysis. Smoking, which is recognized as a major contributory risk factor for cardiovascular diseases and various cancers, was taken into account in the preliminary analysis. The results presented, however, were not stratified on smoking because of nonsignificant interaction between smoking and drinking. Also, we wanted to ensure that the resultant strata contained an adequate number of cases for the stratified analysis. Separate papers addressing the confounding of smoking and various sociodemographic characteristics are currently being prepared by the authors for different medical diagnoses. Next, one might also argue that the lack of scientifically proper definitions of "light," "moderate," and "heavy" drinking might render the results inconclusive. More scientific research needs to be conducted to quantify more precisely the two consumption thresholds: (1) that at which certain beneficial outcomes would come forth; and (2) that at which the adverse effects of drinking would become apparent. Additional research is also required to determine whether these thresholds vary for different diseases or conditions.

There are additional limitations inherent in this study. In this analysis, past-year consumption and disease-specific morbidity were used to examine the association of alcohol and various physical disorders. Presumably, the average daily intake of alcohol was a good indicator of the typical amount of alcohol consumed last year. However, some diseases might take longer than 1 year to develop. In addition, like all behavioral characteristics, drinking behavior does change over time. Conceivably, one might reduce his/her drinking level as a result of accumulating ill health, thus obfuscating the association of alcohol consumption and morbidity. Furthermore, the analysis was based on cross-sectional data. Therefore, it is inappropriate to address causal effects.

Reliability and validity have been important issues when dealing with self-report data similar to that collected in the NLAES. Studies[46,47] on test–retest reliability of various NLAES variables indicated that the alcohol consumption and major sociodemographic items were moderately to highly reliable (ranging from 0.70 to 0.99). Self-report of medical diagnoses demonstrated fair to excellent reliability (0.55 to 0.90). As for validity, studies of self-report of alcohol consumption and medical conditions were limited because of methodological concerns. However, evidence indicates that self-report data are highly correlated with actual consumption levels, or at a minimum, provide an accurate description of relative consumption levels.[48]

Substances other than alcohol found in each type of alcoholic beverage may have differential effects on health. Several studies that isolated differential effects for beer, wine, and liquor consumption have provided evidence that drinking wine has a distinct protective effect over beer and liquor.[31,49] Contrary to these finding, others[29,50] have shown that the three beverage types have remarkably similar health effects. Our results indicated that, whereas a substantial number (43%) of current drinkers drank all three beverage types, most of them had beer as the predominant beverage type, followed by wine, then liquor. Even though our findings showed an increased health risk associated with heavy intake of alcohol, the nature of this association can be clarified further when differential beverage effects are taken into account.

In conclusion, we need to be especially cautious in interpreting the findings pertaining to the beneficial effects of alcohol. Our prevalence study indicated that light/moderate drinking was associated with lower

prevalence rates for various conditions. Despite the potential values of the benefits of moderation, there are risks that might offset the benefits.[51] There is epidemiological evidence that the risk of stroke caused by bleeding (hemorrhagic stroke) was increased at a moderate level of alcohol.[52] Also, female breast cancer is found to be related to moderate drinking.[53] In addition, other adverse consequences include impairment of driving-related skills[54] and harmful interaction with >100 medications, among which some are over-the counter medicines.[55] In animal studies, the harmful fetal effects were found at low levels of drinking.[56] Thus, the results can never be interpreted as suggesting that people drinking less than moderate amounts should increase their drinking level, nor that abstainers should initiate drinking.

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Alcoholic Beverage Preference and Risks of Alcohol-Related Medical Consequences: A Preliminary Report From the National Longitudinal Alcohol Epidemiologic Survey

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In studying the alcohol-morbidity association, a substantial amount of attention and effort has been focused on volume of alcohol intake. Considerably less is known about the differential health effects of beverage types. The present study used a most recent national household survey of the U.S. general population on drinking practices, alcohol use disorders, and their associated disabilities. The prevalence of a broad range of alcohol-related diseases was examined with respect to preferred beverage type, as well as consumption level. Our findings showed a reduced health risk associated with beer and wine drinking for a number of physical disorders, and a somewhat favorable cardiovascular effect of these two beverage types in relation to abstention. Among preferrers of beer, wine, and liquor, the results indicate that liquor preference is associated with elevated morbidity for several medical consequences. However, interpretation of results and the public health implications of these findings need to be taken cautiously, because sociodemographic and other behavioral characteristics were not considered in this preliminary report.

It has long been documented in the alcohol literature that alcohol use is responsible for considerable morbidity and mortality, as well as social and legal problems. Evidence provided in epidemiological and basic research has reported that prolonged excessive alcohol consumption poses a serious threat to human body systems.[1] The adverse medical consequences directly related to alcohol consumption include acute and chronic pancreatitis, alcoholic hepatitis and liver cirrhosis, cardiomyopathy, hypertension, arrhythmias, cerebrovascular hemorrhage, and cancers of the upper digestive tract. With respect to the acute biological effects of alcohol, results based on the studies of the general population and emergency room visits suggest that alcohol use is one of the most important risk factors for injury occurrence.[2,3] All of these alcohol-related adverse consequences have taken their toll in premature deaths and hospital and medical costs, and thus have become major public health concerns over the years.

Conceivably, any nonethanol components found in each type of alcoholic beverage may have differential effects on health. Morbidity and mortality studies have documented the protective effects of moderate wine consumption, specifically in reducing the risk of death from coronary artery disease.[4] The so-called "French paradox," which describes the phenomenon that, despite high saturated fat in their diet, French people generally exhibit low mortality rate from coronary heart disease, has received much media attention (e.g., "60 Minutes," CBS Television Network, November 17, 1991 and November 5, 1995; Health Section, *The New York Times,* December 28, 1994), and promoted the general awareness of benefits of wine consumption. Renaud and de Lorgeril[5] further attributed the French paradox to red wine consumption. Isolated reports also suggest that tannins,[6–7] phenolic compounds,[8] or flavenoids[7] contained in red wine might be responsible for the potential benefits of wine use, possibly through the mechanism of inhibiting the oxidation of low-density lipoprotein cholesterol[9] or inhibiting the formation of blood clots. However, most morbidity studies have not reached any consensus on differential health effects of beer, wine, and liquor. Moreover, their relative effects might even vary by physical disorders. Some reported an inverse association between the risk of pancreatic cancer and the intake of white wine.[10–12] Others have suggested a

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causal role of drinking alcohol, most often beer consumption, in rectal cancer.[13,14]

Among alcohol-related physical disorders, previous studies have shown that drinkers of beer, wine, and liquor all are at reduced risk of coronary artery disease[15,16] when compared with abstainers. In addition, findings from the authors of the present study[17] reported that light or moderate drinkers in general fared better than lifetime abstainers for various medical conditions. The nature of the seemingly protective effects of drinking can be clarified further when differential beverage effects are taken into account.

The purpose of this study was to address differential effects of beer, wine, and liquor consumption on physical morbidity, using a nationally representative sample of the U.S. general population. The prevalence of a broad range of alcohol-related diseases was examined with respect to predominant beverage preference and consumption level.

METHODS

Data source

The National Longitudinal Alcohol Epidemiologic Survey (NLAES) is a multipurpose survey conducted to comply with the Anti-Drug Abuse Act of 1988. One major purpose of this survey was to measure the prevalence of alcohol use disorders (i.e., alcohol abuse and dependence), and their associated disabilities (drug-specific abuse and dependence, major depression, and physical disorders). This survey was targeted at the civilian noninstitutionalized population of the contiguous United States, 18 years and older. The multistage stratified sample design was similar to the 1985 redesign of the National Health Interview Survey.[18,19] This sample design featured first-stage sampling of primary sampling units, with probability proportional to size and oversampling of blacks. Interviewers hired and trained by the U.S. Bureau of the Census conducted personal interviews at the respondent's homes. Proxy respondents were not permitted. In oversampling, the sampling intervals were increased in areas where there were the highest concentrations of blacks. To compensate for the effect of oversampling, the sampling intervals in other areas were decreased. Young adults (ages 18 through 29) were also oversampled at the household level. Overall, there were 42,862 completed interviews, with 92% and 97% response rates at the household and sample person level, respectively.

Disease-specific morbidity

There were 24 survey questions dealing with various medical conditions. As a reference, these 24 survey questions are listed in the Appendix. To estimate the 12-month prevalence rates of disease-specific morbidity, we excluded the last two questions on other health problems, and mental or emotional problems. Furthermore, we considered only those conditions that caused any problems during the last 12 months and were diagnosed by a doctor or other health professional. When prevalence rates of certain conditions were too low to produce meaningful results, similar conditions were grouped into broader categories. For example, coronary heart disease consisted of the conditions of arteriosclerosis, angina pectoris, tachycardia, myocardial infarction, and other heart disease. Liver disease encompassed the conditions of enlarged liver, yellow jaundice, liver cirrhosis, hepatitis, and other liver disease. All alcohol-related medical conditions were grouped according to the ICD-10[20] categories as shown in "Results."

Drinking status and predominant beverage preference

Current drinkers were those respondents who consumed at least 12 drinks during the 12 months preceding the interview. These individuals were classified as light-to-moderate, heavy, and very heavy drinkers according to their levels of past-year average daily intake of ethanol. Light and moderate drinkers were combined into one group in this study because previous studies indicate that they render comparable health outcomes. The consumption measure of past-year average daily intake of ethanol was derived based on usual and heaviest past-year consumption.[21] The overall annual volume of ethanol was aggregated over the separate consumption items for beer, wine, and liquor, and the resulting volume was divided by 365 to derive an average daily ethanol intake. Ounces of beverages were converted to ounces of ethanol using the conversion factors 0.045 for beer, 0.121 for wine, and 0.409 for liquor.[22–24]

To reflect the gender differences in body composition and to be consistent with the U.S. health guidelines for moderate drinking, light-to-moderate drinkers were defined as those who drank no more than an average of 1.0 oz of ethanol/day (equivalent to 2 standard drinks/day) for males, and 0.5 oz of ethanol (one standard drink) for females. Heavy drinkers consumed >1.0, but no more than 2.5 oz, and >0.5 but no more than 1.67 oz of ethanol for males and females, respectively. Any drinkers who consumed beyond their respective gender-specific levels of alcohol intake were classified as very heavy drinkers. We also defined the respondent's predominant beverage preference as the beverage type that accounted for >85% of his/her total

ethanol intake. In addition, for comparison purposes, the prevalence rates of various alcohol-related medical conditions of the U.S. adult population and lifetime abstainers were also included.

Statistical analysis

Morbidity rates frequently vary by age and sex, and occasionally by race as well. These factors also are associated with drinking patterns. In order that demographic differences not confound the apparent associations between consumption and morbidity, all prevalence rates reported in this paper were standardized to reflect the distribution of the U.S. adult population in terms of sex, race (black versus non-black), and age (18–29, 30–44, 45–64, and 65 or older). Pairwise comparisons[25] were conducted to examine the statistical significance of the observed differences among drinkers of various beverage preferences, as well as lifelong abstainers. The overall type I error rate was controlled at 5% for each medical consequence. That is, the probability of one such comparison among all tests turns out to be significant by chance alone was at most 5%.[26] In addition, all standard errors of prevalences were computed using SUDAAN Software.[27] Its statistical procedures use the first-order Taylor series approximation to take into account the stratified and clustered sampling characteristics of the NLAES.

RESULTS

Light-to-moderate drinkers comprised ~74% of all current drinkers. Only one in five current drinkers were heavy drinkers. The remaining 6% were those individuals who drank very heavily. Among those individuals who had a clear beverage preference, most preferred beer (66.3%), followed by wine (19.7%), then liquor (14.1%).

Table 1 presents the overall effects of preferred beverage (i.e., the type of beverage that contributed >85% toward overall intake) on the prevalence of various physical disorders. For a frame of reference, prevalences of alcohol-related medical conditions among the U.S. adult population and lifetime abstainers are also shown (columns 1 and 2 of Table 1, respectively). To further examine the data, significant tests of pairwise comparisons of various prevalence rates were conducted among abstainers and drinkers of the different alcoholic beverages (Table 2). Each pair presented in Table 2 represents a statistically significant comparison for which the overall type I error rate was controlled at 5% for a given medical condition. As indicated in Tables 1 and 2, beer and wine drinkers fared better than abstainers for several medical conditions, including coronary heart disease and hypertension. Even though liquor preference appeared to be associated with greater prevalence of various medical conditions

Table 1. Twelve-Month Prevalence Rates* of Alcohol-Related Medical Conditions Among the Total Population, Abstainers, and Current Drinkers by Beverage Preference

Medical condition	Total	Abstainer	Current drinker		
			Beer	Wine	Liquor
Digestive disease					
Stomach	5.20 (0.13)	5.00 (0.23)	4.22 (0.36)	3.15 (0.43)	7.37 (0.89)
Liver	0.49 (0.04)	0.39 (0.06)	0.35 (0.09)	0.27 (0.19)	0.87 (0.31)
Pancreas	0.17 (0.03)	0.12 (0.04)	0.15 (0.09)	0.06 (0.04)	0.44 (0.29)
Respiratory disease					
Emphysema	0.79 (0.05)	0.55 (0.07)	0.90 (0.22)	0.16 (0.08)	0.87 (0.26)
Circulatory disease					
Coronary heart disease	6.60 (0.15)	6.40 (0.25)	4.84 (0.42)	4.70 (0.63)	7.50 (0.88)
Cerebrovascular accident	0.68 (0.04)	0.75 (0.08)	0.65 (0.19)	0.49 (0.20)	0.44 (0.14)
Hypertension	6.91 (0.17)	7.32 (0.29)	5.80 (0.50)	5.06 (0.49)	7.42 (0.81)
Neoplasm					
Malignant neoplasm	1.38 (0.06)	1.14 (0.10)	1.22 (0.24)	1.13 (0.37)	1.73 (0.30)
Endocrine, nutritional, and metabolic disease					
High cholesterol or lipid content	4.18 (0.13)	3.68 (0.21)	3.30 (0.35)	3.41 (0.46)	4.68 (0.61)
Diabetes	2.26 (0.09)	2.71 (0.18)	1.04 (0.23)	1.38 (0.16)	1.64 (0.40)
Disease of the nervous system					
Convulsions or epilepsy	0.25 (0.03)	0.45 (0.08)	0.18 (0.10)	0.22 (0.16)	0.14 (0.08)
Disease of the musculoskeletal system and connective tissue					
Arthritis	12.85 (0.19)	11.62 (0.27)	10.94 (0.63)	8.29 (0.70)	15.60 (1.05)
Disease of the blood and blood-forming organs, and certain disorders involving the immune mechanism					
Anemia or vitamin deficiencies	2.55 (0.09)	2.37 (0.13)	1.86 (0.26)	3.11 (0.48)	2.05 (0.45)

Numbers in parentheses are standard errors of the prevalences.

* Prevalence rates were standardized according to the distribution of the U.S. general population in terms of sex, race, and age.

than that of either beer or wine preference, this elevated morbidity was statistically significant for gastric disease, coronary heart disease, and arthritis. Despite the observed differences, Table 2 also showed that the beverage type did not render statistically significant differences in prevalence for some medical conditions. Furthermore, beer and wine preferrers seemed to have comparable prevalence rates.

Different beverage preferences could be associated with level of consumption, thus obscuring the effect of beverage type per se on the prevalence rates of medical conditions. Accordingly, Table 3 shows the 12-month prevalence rates by drinking status and preferred beverage. Notably, sampling fluctuation resulted in fairly large standard errors for prevalence rates of various physical disorders among liquor preferrers who also drank very heavily due to the small number of respondents falling into that category. Similar to Table 2, significant tests of pairwise comparisons were conducted by drinking status. The results (data not shown) indicated that, for a number of medical conditions, liquor preferrers exhibited significantly greater prevalence than that of wine preferrers. Again, no consistent pattern was noted to determine whether beer or wine preference was superior over the other.

Table 2. Pairwise Comparisions of 12-Month Prevalence Rates of Alcohol-Related Medical Conditions Among Abstainers and Current Drinkers With Predominant Beverage Preference

Medical condition	Pairwise comparison[a]
Digestive disease	
Stomach	(A,W) (B,L) (W,L)[b]
Liver	NS
Pancreas	NS
Respiratory disease	
Emphysema	(A,W) (B,W)
Circulatory disease	NS
Coronary heart disease	(A,B) (B,L)
Cerebrovascular accident	NS
Hypertension	(A,W)
Neoplasm	
Malignant neoplasm	NS
Endocrine, nutritional, and metabolic disease	
High cholesterol or lipid content	NS
Diabetes	(A,B) (A,W)
Disease of the nervous system	
Convulsions or epilepsy	NS
Disease of the musculoskeletal system and connective tissue	
Arthritis	(A,W) (A,L) (B,W) (B,L) (W,L)
Disease of the blood and blood-forming organs, and certain disorders involving the immune mechanism	
Anemia or vitamin deficiencies	NS

[a] Only significant comparisons are shown.
[b] A, abstainers; B, beer preferrers; W, wine preferrers; L, liquor preferrers; NS, nonsignificant pairwise comparison.

DISCUSSION

This study examined the prevalence rates of various medical conditions by preferred beverage type. Level of alcohol consumption was also controlled for in studying this differential beverage effect. The evidence provided herein indicates that liquor preference was associated with increased risks for several physical disorders. Beer and wine preferences were comparable, because there was no consistent pattern to differentiate their health effects.

Our findings pointed toward reduced risks of several physical disorders associated with beer and wine drinking in relation to abstention. For cardiovascular effects, drinkers of beer and wine in particular exhibited significantly lower morbidity rates than among lifelong abstainers for coronary heart disease and hypertension. This seemed to support the public notion that drinking might confer a protective cardiovascular effect. However, the nature of the observed cardiovascular differences between drinkers and lifelong abstainers is not totally clear; interpretation of results needs to be taken carefully. In addition, risks of accidental injuries of all types have been shown to increase even at relatively low levels of alcohol consumption.[28] Other problems, such as breast cancer[29] and interaction with numerous medications,[30] are also found to be related to moderate drinking. Thus, any findings indicating benefit of drinking need to be interpreted with extreme caution.

The higher prevalence of several alcohol-related medical consequences associated with predominant liquor use was fairly consistent across drinking categories. Given the acknowledged fact that heavy drinking poses serious threats to human health, the liquor effects on health have special implications to those heavy drinkers who prefer liquor. Even though the nature of the NLAES data does not lend itself to investigating causal relationships, for public education and preventive purposes, the observed excess morbidity associated with liquor use warrants further examination.

One strength of the present study lies in the fact that we were able to include a variety of medical conditions in examining differential beverage effects. However, several methodological limitations inherent in this study bear mentioning. First, the NLAES data were cross-sectional. It is inadequate to address any causal associations. Next, the preferred beverage type and morbidity reported during the 12 months were examined to distinguish beverage-specific health effects. However, some diseases might take longer than a year to develop, hence rendering the results inclusive.

Furthermore, as a preliminary study of different beverage effects on physical health, a multivariate approach was not taken in this study. Consequently, potential confounding variables (such as smoking, sex, race, and other sociodemographic variables) were not adequately controlled. It is possible that the observed beverage differentials might attribute to differences in the relative ethanol content or substances other than ethanol contained in each beverage type, or external factors such as personality traits, lifestyle characteristics, or other factors leading different people to different types of alcoholic beverages. To address the potential confounding of smoking and other sociodemographic characteristics, several disease-specific multivariate analyses (e.g., coronary heart disease and hypertension) were being undertaken by the authors. In addition, to further clarify the nature of the beverage effects, other dietary intake and external factors rather than the alcoholic beverages per se need to be taken into account. However, to provide adequate explication or to test competing explanations of the findings is beyond the scope of the present study.

Within the scientific community, a substantial amount of attention and effort has been focused on volume of alcohol intake when studying the health effects of alcohol. Considerably less is known about the differential effects of beverage types on morbidity. Future morbidity research should address this issue by relating beverage-specific consumption with morbidity data, while controlling for potential confounders. For disease-specific beverage effects, a characterization of the dose-response relationship would be useful in describing the beverage differentials. In addition, differences in congeners and electrolytes contained in alcoholic beverages, and external factors (including personality type, lifestyle characteristics, usual consumption pattern, and dietary intake) should also be taken into account in studying beverage effects.[4,31,32] Additional efforts should also promote greater understanding of the basic biological mechanisms, and psychological, social, and cultural processes underlying different beverage effects. After careful consideration of these relevant factors, beverage-specific effects can be clarified further. Implications of such findings will have impact on the alcoholic beverage industry, the public health policymakers, and the consumers at large.

APPENDIX: 24 SURVEY QUESTIONS DEALING WITH ALCOHOL-RELATED MEDICAL CONDITIONS

The opening statement of this section reads: "Now I am going to read you a list of medical conditions that you may have had. As I read each one, please tell me whether you have ever had it. Have you EVER had...

1. A stomach ulcer?

Table 3. Twelve-Month Prevalence Rates* of Alcohol-Related Medical Conditions Among Current Drinkers by Drinking Status and Preferred Beverage Type

Medical condition	Light/Moderate			Heavy			Very Heavy		
	Beer	Wine	Liquor	Beer	Wine	Liquor	Beer	Wine	Liquor
Digestive disease									
Stomach	4.29 (0.50)	2.99 (0.46)	6.00 (0.96)	3.91 (0.66)	3.06 (0.94)	9.08 (2.07)	4.58 (1.03)	3.90 (1.31)	13.01 (4.46)
Liver	0.30 (0.11)	0.30 (0.24)	0.26 (0.14)	0.25 (0.12)	0.00 (0.00)	1.34 (0.51)	1.03 (0.55)	0.81 (0.80)	2.96 (1.30)
Pancreas	0.07 (0.04)	0.00 (0.00)	0.20 (0.15)	0.03 (0.03)	0.10 (0.08)	0.00 (0.00)	0.90 (0.84)	0.81 (0.80)	0.53 (0.45)
Respiratory disease									
Emphysema	0.85 (0.31)	0.05 (0.05)	0.86 (0.35)	1.03 (0.35)	0.36 (0.33)	0.74 (0.33)	0.43 (0.24)	1.42 (0.97)	1.37 (1.09)
Circulatory disease									
Coronary heart disease	4.96 (0.57)	4.14 (0.60)	6.70 (0.92)	4.76 (0.73)	4.05 (1.05)	7.98 (1.73)	4.27 (1.13)	8.08 (0.98)	11.91 (4.57)
Cerebrovascular accident	0.66 (0.24)	0.34 (0.19)	0.51 (0.18)	0.71 (0.42)	0.61 (0.37)	0.32 (0.22)	0.06 (0.06)	0.81 (0.80)	0.00 (0.00)
Hypertension	6.60 (0.72)	5.03 (0.55)	7.55 (0.96)	4.79 (0.68)	3.05 (0.89)	8.80 (1.63)	4.92 (1.56)	7.32 (1.99)	4.03 (1.17)
Neoplasm									
Malignant neoplasm	1.33 (0.33)	0.40 (0.15)	1.92 (0.39)	0.83 (0.33)	1.54 (0.56)	1.21 (0.40)	1.79 (0.90)	2.46 (0.66)	0.26 (0.17)
Endocrine, nutritional, and metabolic disease									
High cholesterol or lipid content	3.56 (0.53)	3.44 (0.55)	4.83 (0.73)	2.90 (0.51)	2.61 (0.70)	4.14 (0.92)	4.26 (1.33)	5.97 (2.01)	1.84 (0.63)
Diabetes	1.14 (0.31)	1.24 (0.18)	1.31 (0.35)	0.71 (0.28)	0.90 (0.44)	5.23 (2.19)	1.51 (1.05)	0.80 (0.56)	1.06 (0.65)
Disease of the nervous system									
Convulsions or epilepsy	0.20 (0.14)	0.18 (0.17)	0.12 (0.09)	0.10 (0.06)	0.16 (0.16)	0.16 (0.16)	0.27 (0.31)	0.34 (0.36)	0.00 (0.00)
Disease of the musculoskeletal system and connective tissue									
Arthritis	10.28 (0.82)	8.91 (0.88)	15.64 (1.23)	12.42 (1.23)	6.76 (0.99)	14.92 (1.80)	11.18 (2.92)	6.22 (1.43)	15.27 (4.66)
Disease of the blood and blood-forming organs, and certain disorders involving the immune mechanism									
Anemia or vitamin deficiencies	2.04 (0.35)	2.63 (0.48)	1.73 (0.44)	0.86 (0.27)	3.45 (0.85)	3.42 (1.36)	2.80 (0.85)	5.23 (0.93)	0.61 (0.60)

* Prevalence rates were standardized according to the distribution of the U.S. general population in terms of sex, race, and age. Numbers in parentheses are standard errors of the prevalences.

2. An enlarged liver?
3. Yellow jaundice?
4. Cirrhosis of the liver?
5. Hepatitis?
6. Some other liver disease? Specify
7. High blood cholesterol, high blood fat, or high lipid content?
8. Diabetes?
9. Gastritis?
10. Convulsions or epilepsy?
11. Hardening of the arteries or arteriosclerosis?
12. High blood pressure?
13. Chest pain or angina pectoris?
14. Rapid heart beat or tachycardia?
15. Heart attack or myocardial infarction?
16. Other heart disease?
17. A stroke or cerebrovascular accident?
18. Emphysema?
19. Arthritis, osteoporosis, or any other joint or bone disease?
20. Vitamin deficiencies or anemia?
21. Pancreatitis or any other disease of the pancreas?
22. Cancer? Specify type
23. Any other physical health problems? Specify type
24. A mental or emotional problem? Specify type or problem

To ascertain the timeframe when the specific morbid condition occurred, the respondent was asked, "Did (condition) cause you any problems during the last 12 months?" As to whether each morbid condition was ever diagnosed, the respondent was asked the follow-up question, "Did a doctor or other health professional tell you that you had (condition)?"

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CHAPTER 4
FAMILY HISTORY OF ALCOHOL PROBLEMS

Estimates of U.S. Children Exposed to Alcohol Abuse and Dependence in the Family

Bridget F. Grant

Objectives: *This study sought to provide direct estimates of the number of U.S. children younger than 18 years who are exposed to alcohol abuse or alcohol dependence in the family.* Methods: *Data were derived from the National Longitudinal Alcohol Epidemiologic Survey.* Results: *Approximately 1 in 4 children younger than 18 years in the United States is exposed to alcohol abuse or alcohol dependence in the family.* Conclusions: *There is a need for approaches that integrate systems of services to enhance the lives of these children.*

Alcohol abuse and alcohol dependence are the two most prevalent and deleterious psychiatric disorders not only in the United States but in the world.[1] In 1992, an estimated 14 million adults in the United States abused alcohol or were dependent on it.[2] Beyond the enormous, devastating effects on both these individuals and society, immediate family members, particularly children, suffer the burdens inflicted by such disorders.

There is considerable evidence that both genetic exposure and environmental exposure to alcoholism predispose children to become alcoholics themselves. Evidence of familial aggregation of alcoholism has been strongly supported by family, twin, and adoption studies.[3–5] Despite a number of methodological shortcomings,[6] there is also abundant evidence from studies of environmental exposure that children of alcoholic parents are at risk. Children of alcoholics are often subjected to an extremely disorganized milieu, negligent and abusive rearing, economic hardship, and social isolation that accompanies attempts to hide the disorder from friends, relatives, and others.[7,8]

Studies using self-report or behavioral observations have shown that alcoholic families, relative to nonalcoholic families, are characterized by higher levels of conflict, lower levels of cohesion, more impaired problem solving, and more hostile communications.[6–9] Many studies have shown as well that parental alcoholism is associated with a range of psychopathology and other behavioral and medical problems in offspring, including conduct disorder and delinquency,[10–12] use and abuse of alcohol and other drugs in adolesence,[13–15] anxiety disorders,[6] and impaired physical health.[16]

Because of the important consequences of exposure to alcohol in the family, this study was conducted to provide estimates of the number of children (i.e., younger than 18 years) who are exposed to alcohol abuse and/or dependence in the family environment. This study provides, for the first time, a direct estimate of the number of children living with at least one adult classified with alcohol abuse or dependence, considering both past-year and lifetime diagnoses.

The present study represents a significant advance over previous estimates of the number of children of alcoholics in the United States, which have ranged from 5 to 6 million.[17–19] These previous estimates were based on extrapolating the ratio of children to adults from the general population to subpopulations of alcoholics.[17–19] Other studies have questioned adults about exposure in their childhoods.[20]

This study avoids several limitations of previous research, including exclusion of certain subpopulations, absence of standardized definitions of alcohol abuse and dependence, recall bias, and the untenable assumptions that alcoholism is randomly distributed in the general population and that the ratios of children in alcoholic families are consistent with those in the general population. Unlike previous research, the present study provides a description of children at risk by virtue of exposure to alcohol in the family in terms of sex, race, and age.

METHODS

Sample

This study was based on the 1992 National Longitudinal Alcohol Epidemiologic Survey, spon-

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sored by the National Institute on Alcohol Abuse and Alcoholism.[21] Data were collected via personal interviews conducted in respondents' homes by U.S. Bureau of the Census interviewers. The survey sample consisted of adults 18 years or older who were selected at random from a nationally representative sample of households. The multistage sampling design featured the selection of primary sampling units via probability—proportional-to-size techniques, oversampling of segments with high proportions of Black residents, and oversampling of young adults (i.e., those aged 18 to 29 years) at the household level. The household-response rate was 92%, and the individual-response rate was 97%, yielding a total sample size of 42,862.

Diagnostic assessment

Diagnoses of alcohol use disorders, as classified in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV),[22] were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule, a fully structured psychiatric interview designed to be administered by trained lay interviewers.[23] The interview schedule included an extensive list of symptom items that operationalized the DSM-IV criteria for alcohol abuse and dependence.

Respondents were classified with past-year dependence if they met at least 3 of the 7 DSM-IV criteria for dependence within the 1-year period preceding the interview: tolerance; withdrawal or avoidance of withdrawal; desire or attempts to cut down or stop drinking; much time spent on drinking, obtaining alcohol, or recovering from its effects; reduction/cessation of important activities in favor of drinking; impaired control; and continued drinking despite physical or psychological problems caused or exacerbated by drinking. Respondents were classified with past-year alcohol abuse if they met at least 1 of the 4 DSM-IV criteria for abuse in the 1-year period preceding the interview: alcohol-related legal problems, continued drinking despite interpersonal problems, neglect of role responsibilities as a result of drinking, and drinking in hazardous situations.

The Alcohol Use Disorders and Associated Disabilities Interview Schedule diagnoses of past-year alcohol abuse and dependence also satisfied the clustering and duration criteria of the DSM-IV definitions. The criteria of the DSM-IV included clustering of symptoms for each diagnosis within the 1-year period preceding the interview, along with associating duration qualifiers with certain abuse and dependence criteria. Duration qualifiers are defined in DSM-IV as the repetitiveness with which symptoms must occur to be classified as positive toward a diagnosis. They are represented by the terms *recurrent, often,* and *persistent* appearing in the description of the diagnostic criteria.

Lifetime diagnoses were also measured as syndromes, or the clustering of the number of simultaneous symptoms required for a diagnosis of abuse or dependence. Respondents classified with a lifetime diagnosis encompassed all who had experienced an episode of abuse or dependence at any point in their lives. The reliabilities of past-year and lifetime DSM-IV alcohol abuse and dependence diagnoses were 0.73 and 0.76, as ascertained from an independent test-retest study conducted in the general population.[24]

Statistical analyses

SUDAAN,[25] a software program that uses Taylor series linearization to adjust for sample design characteristics in complex sample surveys, was used in conducting all of the analyses presented here to take into account the complex sample design of the National Longitudinal Alcohol Epidemiologic Survey. Two estimates of the number of children living with at least 1 adult classified with alcohol abuse or dependence were derived from the study data: (1) the number of children living in households with 1 or more adults classified with past-year alcohol abuse and/or dependence, and (2) the number of children living in households with 1 or more adults classified with a diagnosis of alcohol abuse, alcohol dependence, or both, at any point during their lives.

The number of children living in households with 1 or more adults who abused or were dependent on alcohol in the past year was estimated by multiplying the total estimated number of adults 18 years and older who met DSM-IV criteria for past-year abuse and/or dependence by the average number of children from birth to 17 years of age living in their households. This average number of children was obtained by linking National Longitudinal Alcohol Epidemiologic Survey respondents' files with those of all other individuals in the same household, by taking a count of children based on the ages of each other household member, and then calculating the weighted mean of this count for adults who were classified with past-year abuse and/or dependence.

The weight factor for each adult sample member included a multiplier to account for all other adults in the household represented by the sample member. Thus, weighting the number of children in the household by the weight factor for this sample member accounted for the probability that at least one of the adults in the household abused or was dependent on

alcohol. The same procedure was used to estimate the number of children living in households with one or more adults classified with a diagnosis of alcohol abuse or dependence on a lifetime basis; the total number of adults classified with a past-year diagnosis of abuse or dependence was replaced with the estimated number of adults classified with a lifetime diagnosis.

The first estimate of children living in households with at least one abusing/dependent adult represents an underestimation of the number of exposed children. The reason is that this estimate does not account for adults who were not abusing or dependent on alcohol during the year preceding the interview but had been at some earlier time that may have coincided with the time the child was living in the household.

In contrast, the estimate based on adults with lifetime diagnoses represents an overestimation of the number of exposed children to the extent that an unknown proportion of these adults had not abused or been dependent on alcohol during a period that coincided with the time the child was living in the household. Both estimates are also overrepresentations to the extent that households with multiple alcoholics increase the probability that children will be categorized as living in an alcoholic household.

RESULTS

Approximately 7.4% (13,760,000) of U.S. adults were classified with a past-year diagnosis of DSM-IV alcohol abuse or dependence. Of these individuals, 9,806,000 were men and 3,954,000 were women. An estimated 9,667,473 children were living in households with 1 or more adults classified with a past-year diagnosis of alcohol abuse or dependence (Table 1). Approximately 49.0% of these children were male, 11.9% were Black, and 88.1% were non-Black. Slightly more than one-third (36.3%) of the children were 5 years or younger, 33.7% were aged 6 to 11 years, and 30% were aged 12 to 17 years.

Most of the children were identified as biological, foster, or adopted children or stepchildren (70.4%); 11.6% were siblings of the adults classified with a past-year diagnoses of DSM-IV alcohol abuse and/or dependence. The remainder (8.8%) were classified as other biological relatives (e.g., cousins, grandchildren, nieces, nephews), non-relatives with or without their own relatives in the household (6.3%), and children with an unspecified relationship to the adult classified with past-year abuse or dependence (2.9%).

Approximately 18.2% (n= 33,761,710) of the adults were classified with a lifetime diagnosis of DSM-IV alcohol abuse or dependence. An estimated 28,046,258 children were living in households with 1 or more adults classified as having such a diagnosis (Table 2). Among these children, 51.1% were male, 8.4% were Black, and 91.6% were non-Black. Again, slightly more than one-third (36.8%) were 5 years or younger; 34% were aged 6 to 8 years, and 29.2% were aged 12 to 17 years.

Table 1. Numbers of Children Living in Households With 1 or More Adults Who Abused or Were Dependent on Alcohol in the Previous Year: National Longitudinal Alcohol Epidemiologic Survey, 1992

Characteristic	No.
Sex	
Male	4 740 904
Female	4 926 569
Race/ethnicity	
Black	1 146 485
Non-Black	8 520 988
Age, y	
0–2	1 842 237
3–5	1 668 656
6–8	1 741 220
9–11	1 512 953
12–14	1 408 891
15–17	1 493 516
Total	9 667 473

Most of the children were the biological, foster, or adopted children or stepchildren (82.5%) of the adults classified with lifetime abuse or dependence. The remaining children were identified as siblings (5.1%), other biological relatives (5.6%), nonrelatives living in the household (4.2%), and children with an unspecified relationship to the adult with an abuse and/or dependence diagnosis (2.6%).

DISCUSSION

In 1992, an estimated 9,667,473 children, representing approximately 15% of the 66 million U.S. children 17 years or younger, were living in households with 1 or more adults who were abusing or dependent on alcohol. Nearly 43% (n=28,046,258) of U.S. children were members of households with 1 or more adults who, at some time in their lives, had abused or were dependent on alcohol. The true estimate of the number of children exposed to alcohol abuse or dependence lies somewhere between these two figures.

Given the more conservative estimate involving past-year diagnoses, about 1 in every 6.6 children in the United States is exposed to alcohol abuse or dependence in the family. Assuming the less conservative

Table 2. Numbers of Children Living in Households With 1 or More Adults Who Had Abused or Been Dependent on Alcohol at Some Time in Their Lives: National Longitudinal Alcohol Epidemiologic Survey, 1992

Characteristic	No.
Sex	
Male	14 327 666
Female	13 718 592
Race/ethnicity	
Black	2 359 344
Non-Black	25 686 914
Age, y	
0–2	5 252 965
3–5	5 064 018
6–8	4 983 415
9–11	4 556 563
12–14	4 276 707
15–17	3 912 590
Total	28 046 258

estimate involving lifetime diagnoses, 1 in every 2.3 children is exposed to alcohol abuse or dependence in the family. Consider that neither estimate includes children's exposure to biological and nonbiological family and friends who do not reside in the household. Also, assume that 50% of the 18,378,785 children living with 1 or more adults with diagnoses of abuse and/or dependence only prior to the past-year are indeed affected before the age of 18 years by that adult's abuse and/or dependence. With these considerations, it can conservatively be estimated that approximately 1 in every 4 (28.6%) children in the United States is exposed to alcohol abuse or dependence in the family. Such figures represent underestimations to the extent that they do not include children who are homeless and otherwise not residing in households.

As is the case with most research, this study raises a number of critical questions and issues. Foremost, all of the children exposed to alcohol abuse or dependence in the family are at risk of adverse developmental, social, and health outcomes, but not all exposed children will manifest the effects of these threats to their health, well-being, and ability to achieve their full potential in life. More research is necessary to explore the resiliency found in some children who manage to cope positively despite their exposure to alcohol abuse and dependence in the family and an environment that can, at best, be characterized as stressful, chaotic, and frightening. More needs to be known about these individual differences and the impact of mediating factors (e.g., sex of child and affected adult, personality features, comorbidity, social support) on the development and outcomes of children exposed to alcohol abuse or dependence in the family.

The extraordinary number of children in this country who are exposed to alcohol abuse and dependence defines one of today's major public health problems and demands a comprehensive public policy directed toward prevention and intervention. Extant social health and treatment services designed to improve the lives of children from a variety of types of dysfunctional families have ignored the wide-ranging problems experienced by children exposed to alcohol abuse and dependence in the family. There are simply too many of these children at risk to rely on existing fragmented, incomplete, and compartmentalized health, social, and treatment services.

What is urgently needed is a comprehensive strategy that integrates all systems oriented toward the provision of health, social, and treatment services, designed to improve the lives of children at risk from their exposure to alcohol abuse and dependence in the family. Such a strategy must include a broadening of an array of services targeted to the needs of these children at every developmental stage, coupled with aggressive interventions to enhance their lives and protect their safety.

Children exposed, through no fault of their own, to alcohol abuse and dependence during their critical developmental years are thrust into families and environments that pose extraordinary risks to their immediate and future well-being and that threaten the achievement of their fullest potential. Unless comprehensive and intensive interventions are provided to address the full range of needs of children exposed to abuse and dependence, along with the needs of their families, the potential costs to human services, health, education, social services, and correctional systems will quickly become overwhelming.

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The Impact of a Family History of Alcoholism on the Relationship Between Age at Onset of Alcohol Use and DSM–IV Alcohol Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant

Both the age at onset of alcohol use and a family history of alcoholism can influence a person's risk of becoming alcohol dependent. The relationship between lifetime alcohol dependence, age at first alcohol use, and a family history of alcoholism was investigated using data obtained in the 1992 National Longitudinal Alcohol Epidemiologic Survey. This analysis demonstrated that regardless of the family history of alcoholism, respondents with an earlier age of drinking onset were more likely to become alcohol dependent compared with respondents with a later age of drinking onset. Among all age, race, and gender subgroups studied, however, people with a family history of alcoholism had a higher prevalence of lifetime alcohol dependence than did people without such a history.

Various factors can influence a person's risk of developing alcohol dependence during his or her lifetime, including the age at which alcohol use first occurred and a family history of alcoholism. Epidemiologic analyses have found that people who started drinking at age 14 and younger are approximately four times as likely to become alcohol dependent as are those who began drinking at age 20 and older (Grant and Dawson 1997). Similarly, numerous studies have demonstrated that first-degree relatives of alcoholics are two to seven times more likely than people with nonalcoholic relatives to develop problems with alcohol at some time in their lives (National Institute on Alcohol Abuse and Alcoholism [NIAAA] 1997). This Epidemiologic Bulletin explores the relationship between lifetime alcohol dependence and the age at onset of alcohol use as a function of a family history of alcoholism. The article includes an analysis of the U.S. drinking population, classified by race and gender (i.e., race-gender subgroups). This study extends the findings of an earlier study that investigated the relationship between age at onset of drinking and alcohol use disorders (Grant and Dawson 1997).

BACKGROUND AND PROCEDURES

Lifetime prevalence estimates of alcohol dependence in this study were based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), a nationwide household survey sponsored by NIAAA (Grant et al. 1994). The survey consisted of face-to-face interviews with 42,862 respondents, age 18 and older, in the contiguous United States and the District of Columbia. The household response rate for the NLAES was 91.9 percent, and the person response rate was 97.4 percent. Fieldwork for the study was conducted by the Bureau of the Census.

The NLAES featured a complex multistage design (Massey et al. 1989). Primary sampling units (PSUs)[1] were stratified according to sociodemographic criteria and were selected with probability proportional to size. The NLAES sample included approximately 200 PSUs, 52 of which were self-representing—that is, selected with certainty.[2] Within PSUs, geographically defined secondary sampling units, called segments, were selected systematically for each sample. Because blacks experience higher rates of alcohol-related disease (e.g., liver cirrhosis) than do other population

[1]For a definition of this and other technical terms used in this article, see glossary, p. 133.

[2]This means that the 52 largest PSUs were automatically included in the study.

Reprinted from Alcohol Health & Research World *(now titled* Alcohol Research & Health), *Volume 22, Number 2, pp. 144–148, 1998, National Institute on Alcohol Abuse and Alcoholism.*

subgroups, oversampling of the black population was accomplished at this stage of sample selection.

Segments then were divided into clusters of approximately four to eight housing units, and all occupied housing units were included in the survey. Within each household, one person age 18 or older was randomly selected to participate in the survey. Oversampling of young adults (i.e., people ages 18 to 29) was accomplished at that stage of sample selection to increase the representation of this heavy-drinking population subgroup. Thus, young adults were sampled at a ratio of 2.25 percent to 1.00 percent.

Because of the complex survey design of the NLAES, variance estimation procedures that assume a simple random sample cannot be employed. Research has shown that the stratification and clustering strategies of the NLAES sample selection may result in standard errors much larger than those that would be obtained with a simple random sample of equal size. To take into account the NLAES sample design, all standard errors of the prevalence estimates presented here were calculated using SUDAAN (Research Triangle Institute 1997), a software program that uses appropriate statistical techniques to adjust for sample design characteristics.

MEASURES

The definition of lifetime alcohol dependence was based on the diagnostic criteria of the *Diagnostic and Statistical Manual, Fourth Edition* (DSM–IV) (American Psychiatric Association 1994). DSM–IV-based diagnoses were established using the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained interviewers who are not clinicians (Grant and Hasin 1992). The AUDADIS includes an extensive list of symptom questions that operationalize the DSM–IV criteria for alcohol dependence.

The AUDADIS diagnosis of alcohol dependence satisfied the clustering and duration criteria of the DSM–IV definition. The criteria of the DSM–IV include the requirement for a clustering of symptoms within any 1-year period. The duration criterion is defined as the repetitiveness with which symptoms must occur to be counted as positive toward a diagnosis. The duration criterion is represented by the terms "recurrent" and "persistent" that appear in the description of most of the dependence diagnostic criteria. Not only was the clustering criterion represented in past-year AUDADIS diagnoses of dependence, but also the corresponding past diagnoses (i.e., before the past year) were measured as syndromes, or the clustering of the required number of symptoms necessary to achieve a diagnosis as follows: (1) at the same time, (2) continuously for at least 1 month, or (3) repeatedly for several months.

For the purposes of the current study, respondents were classified as having a lifetime alcohol dependence diagnosis if they had experienced an episode of dependence in the past year or at any time before the past year. Respondents classified with lifetime dependence included those with and without abuse diagnoses. To determine the reliability of alcohol dependence diagnoses established using this approach, an independent

Table 1. Age at First Alcohol Use and the Prevalence of Lifetime Alcohol Dependence by Race and Family History of Alcoholism

Age at First Alcohol Use (years)	Prevalence of Lifetime Dependence					
	White		Black		Total	
	FHP[1]	FHN[1]	FHP	FHN	FHP	FHN
≤ 13	57.9 (2.2)	26.6 (2.3)	51.5 (7.7)	23.4 (7.0)	57.3 (2.1)	26.4 (2.1)
14	51.9 (2.9)	31.1 (2.7)	27.0 (8.4)	34.9 (9.0)	50.4 (2.8)	31.4 (2.6)
15	47.3 (2.3)	33.1 (2.0)	35.0 (7.3)	22.0 (5.4)	46.5 (2.2)	32.3 (1.9)
16	37.3 (1.6)	26.3 (1.2)	32.1 (6.1)	20.4 (3.8)	36.9 (1.5)	25.9 (1.2)
17	33.7 (1.7)	18.8 (1.2)	35.2 (6.0)	14.9 (3.1)	33.9 (1.6)	18.5 (1.1)
18	23.4 (1.1)	13.3 (0.7)	17.9 (3.1)	11.7 (1.9)	22.9 (1.0)	13.1 (0.6)
19	22.1 (1.8)	13.5 (1.3)	17.4 (5.0)	12.7 (4.0)	21.6 (1.7)	13.4 (1.2)
20	15.6 (1.7)	9.2 (0.9)	19.1 (4.6)	9.6 (3.1)	15.9 (1.6)	9.2 (0.9)
≥ 21	15.3 (0.9)	6.4 (0.5)	18.1 (2.6)	6.9 (1.2)	15.6 (0.8)	6.5 (0.4)

NOTE: Standard errors appear in parentheses; prevalences are presented as weighted figures.
[1]FHP = family history positive; FHN = family history negative.

test-retest study was conducted in the general population before fielding the full NLAES (Grant et al. 1995). The analysis determined good reliabilities, with kappas of 0.76 and 0.73 for past-year and prior-to-past-year dependence diagnoses, respectively.

The age of drinking onset was ascertained by asking respondents how old they were when they first started drinking, not counting small tastes or sips of alcohol. Drinking onset data were collected from respondents who were classified as current drinkers (i.e., who had consumed at least 12 drinks in the past 12 months) and former drinkers (i.e., who had consumed at least 12 drinks in any 1 year of their lives but not during the year preceding the interview). The test-retest reliability of the drinking onset variable was good, with a kappa of 0.72 (Grant et al. 1995).

Measures selected as control variables included race (i.e., black versus white), gender, and family history of alcoholism (i.e., family-history positive [FHP] versus family-history negative [FHN]). The latter was ascertained through a series of questions that asked about different types of first-degree biological relatives (i.e., parents, children, and siblings). For each type of relative, the respondent was asked how many relatives of that type lived to be at least 10 years old and how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM–IV criteria for alcohol use disorders:

> By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking; problems with a spouse, family, or friends because of drinking; problems at work because of drinking; problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hung over.

In a test-retest study conducted in conjunction with the pretest for the NLAES, the family history items generally showed good to excellent reliability, with kappas of 0.70 or higher for most types of first-degree relatives (e.g., 0.72 for fathers, 1.00 for mothers, 0.90 for brothers, and 0.73 for sisters). Slightly lower kappa values were obtained for sons and daughters (0.65 for each). For the purpose of this study, a respondent was classified as FHP if any first-degree relative of the respondent was reported as having been alcoholic or a problem drinker.

SUMMARY OF FINDINGS

Of the entire NLAES sample, 66 percent (n = 27,616) were classified as lifetime drinkers, including current (n = 18,352) and former (n = 9,264) drinkers. Approximately 50.6 percent (n = 13,990) of the lifetime drinkers were male and 49.4 percent (n = 13,626) were female. With respect to the racial distribution, 11.1 percent (n = 3,062) of lifetime drinkers

Table 2. Age at First Alcohol Use and the Prevalence of Lifetime Alcohol Dependence by Race and Family History of Alcoholism Among Males

	Prevalence of Lifetime Dependence					
Age at First Alcohol Use (years)	White Male		Black Male		Total Male	
	FHP[1]	FHN[1]	FHP	FHN	FHP	FHN
≤ 13	58.8 (2.8)	30.4 (2.9)	58.8 (10.9)	21.7 (8.8)	58.8 (2.8)	29.7 (2.7)
14	56.9 (3.9)	33.3 (3.4)	20.8 (8.6)	30.9 (10.6)	54.7 (3.8)	33.2 (3.2)
15	50.6 (3.2)	33.6 (2.4)	42.4 (9.8)	21.7 (6.1)	50.1 (3.1)	32.7 (2.3)
16	40.8 (2.1)	27.8 (1.5)	45.3 (8.7)	25.1 (5.2)	41.1 (2.1)	27.7 (1.4)
17	40.8 (2.3)	19.6 (1.5)	43.6 (8.8)	15.5 (4.1)	41.1 (2.3)	19.4 (1.4)
18	28.1 (1.7)	15.9 (0.9)	17.1 (4.1)	12.9 (2.6)	27.1 (1.6)	15.7 (0.9)
19	27.8 (2.8)	15.9 (1.9)	20.7 (6.6)	16.9 (5.7)	27.1 (2.6)	16.1 (1.8)
20	22.6 (2.9)	10.9 (1.4)	20.8 (8.2)	17.4 (5.7)	22.5 (2.8)	11.6 (1.4)
≥ 21	20.1 (1.6)	8.3 (0.8)	21.2 (4.1)	8.4 (1.2)	20.3 (1.5)	8.4 (0.7)

NOTE: Standard errors appear in parentheses; prevalences are presented as weighted figures.
[1]FHP = family history positive; FHN = family history negative.

were black and 88.9 percent (n = 24,554) were white. Among the lifetime drinkers in the NLAES study, classification according to both race and gender resulted in 12,518 (45.3 percent) white males; 12,036 (43.6 percent) white females; 1,472 (5.3 percent) black males; and 1,590 (5.8 percent) black females.

Tables 1 through 3 present the lifetime prevalence of alcohol dependence by age of drinking onset (for ages ≤13 and ≥21), race, and gender. For the total sample of lifetime drinkers, prevalence of lifetime alcohol dependence decreased substantially with increasing age at drinking onset, regardless of family history of alcoholism (see table 1). However, the prevalence of lifetime dependence generally was far greater among FHP respondents than among FHN respondents. For example, the prevalence of lifetime dependence among respondents who began drinking at age 21 or older was two to three times greater among those classified as FHP compared with those classified as FHN. All the trends noted for the total sample of drinkers also were observed for each race-gender subgroup of lifetime drinkers (see tables 2 and 3). Furthermore, consistent with the distribution of dependence among lifetime drinkers, the prevalence of lifetime alcohol dependence was greater among whites than among blacks and greater among male respondents than among female respondents at almost all ages of onset of drinking, regardless of family history of alcoholism.

DISCUSSION

As expected from previous studies, a family history of alcoholism in this analysis was shown to have a substantial effect on the development of alcohol dependence over the life span. In addition, the age at onset of drinking was a powerful predictor of lifetime alcohol dependence, regardless of family history status, race, or gender. Although these findings highlight the importance of early onset drinking and a family history of alcoholism in the development of subsequent alcohol dependence, they cannot explain why or how these two factors relate to alcohol dependence.

Another significant finding of this study is that early onset drinking cannot be viewed solely as a marker or early indicator of a family history of alcoholism (i.e., not only FHP respondents but also FHN began to drink early and therefore were at increased risk for lifetime alcohol dependence). For both FHP and FHN respondents, the likelihood of lifetime alcohol dependence decreased with increasing age at drinking onset. Those findings indicate that early onset drinking implies an increased risk of dependence, regardless of family history, and that people who drink at an early age are not necessarily destined to become alcohol dependent by virtue of having a positive family history. Moreover, a family history of alcoholism may be an indicator of shared or common environmental factors; genetic influences; or, more likely, a combination of both. This suggests that a family history of alcoholism may be, at least in part, a modifiable risk factor. The

Table 3. Age at First Alcohol Use and the Prevalence of Lifetime Alcohol Dependence by Race and Family History of Alcoholism Among Females

Age at First Alcohol Use (years)	Prevalence of Lifetime Dependence					
	White Female		Black Female		Total Female	
	FHP[1]	FHN[1]	FHP	FHN	FHP	FHN
≤ 13	56.4 (3.6)	16.4 (3.0)	39.4 (10.6)	27.5 (10.2)	54.9 (3.4)	17.4 (2.9)
14	43.9 (4.0)	26.2 (4.1)	37.0 (10.9)	51.2 (14.9)	43.5 (3.9)	27.3 (4.0)
15	42.5 (3.2)	32.1 (3.4)	22.6 (9.4)	22.9 (9.6)	41.4 (3.1)	31.7 (3.3)
16	32.3 (2.2)	23.1 (1.8)	18.2 (5.1)	7.0 (3.5)	31.2 (2.1)	22.3 (1.7)
17	25.1 (2.0)	16.9 (1.8)	23.9 (6.9)	13.4 (5.3)	25.1 (1.9)	16.7 (1.7)
18	17.8 (1.3)	8.7 (0.8)	19.6 (4.8)	9.3 (2.5)	17.9 (1.3)	8.7 (0.8)
19	16.1 (1.9)	10.2 (1.4)	13.1 (7.7)	4.5 (2.4)	15.8 (1.9)	9.7 (1.3)
20	8.7 (1.5)	6.7 (1.1)	18.0 (5.3)	1.3 (0.8)	9.8 (1.5)	6.0 (0.9)
≥ 21	12.4 (0.9)	4.7 (0.5)	16.2 (3.2)	5.1 (1.3)	12.8 (0.9)	4.7 (0.4)

NOTE: Standard errors appear in parentheses; prevalences are presented as weighted figures.
[1]FHP = family history positive; FHN = family history negative.

extent to which this is true, however, will have to be determined in future studies specifically aimed at clarifying and defining the contributions of environmental and genetic influences that are manifested in consistent findings of familial aggregation of alcoholism.

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GLOSSARY

Cluster sampling: A sampling method in which each sampling unit is a collection of persons, units, or elements of interest.

Kappa: A coefficient that serves as a measure of *test-retest reliability*. A kappa of 1.0 indicates that in all cases both the test and the retest produce the same result.

Oversampling: A sampling technique used to bolster the numbers of low-prevalence subgroups of the population in order to achieve adequate numbers suitable for statistical analysis.

Primary sampling units: Comprehensive, mutually exclusive categories, consisting of all persons, units, or elements of interest, usually identified in the first stage of a multistage sampling design. For example, *primary sampling units* can consist of geographic regions of the United States (e.g., cities) defined in terms of sociodemographic criteria.

Selected with certainty: This typically refers to the selection of sampling units with a probability of 1.0. For example, if *primary sampling units* are designated to be selected in proportion to their size, it follows that the largest of the units will be selected with certainty.

Selected with probability: This typically refers to the selection of sampling units according to predetermined probabilities. For example, *primary sampling units* may be selected that have probabilities proportional to size (i.e., larger primary sampling units have a greater probability of being selected than do smaller primary sampling units).

Simple random sample: A method of drawing samples such that each person, element, or unit has an equal probability of being selected.

Stratification: The classification of all persons, units, or elements of interest into comprehensive, mutually exclusive categories.

Test-retest reliability: A measure of the likelihood with which two independent tests of the same variable will produce the same result.

Variance estimation procedures: A technique that allows estimation of the amount of variability (i.e., dispersion) around a measure of data, such as a percentage or mean.

Weighted percentage: Percentages that have been adjusted to account for all aspects of the sample design (e.g., different

The Link Between Family History and Early Onset Alcoholism: Earlier Initiation of Drinking or More Rapid Development of Dependence?

Deborah A. Dawson

Objective: *This study examines the associations between early onset alcoholism and family history to determine whether family history of alcoholism is predictive of earlier initiation of drinking, more rapid onset of dependence once drinking has begun, or both.* Method: *Using cross-sectional, retrospective data from a large, nationally representative sample of U.S. adults, discrete time proportional hazards models were used to assess the effects of family history saturation (% of alcoholic first- and second-degree relatives) on: (1) the risk of initiating drinking among all adults (*N *= 42,862; 58.4% female) and (2) the risk of progressing from initiation of drinking to onset of dependence among lifetime drinkers (*N *= 27,616; 50.7% male). Models were estimated for different time periods to see if the effect of family history saturation varied over time in a manner suggestive of a stronger association with early onset dependence.* Results: *The positive effect of family history saturation on the risk of initiating drinking was strongest prior to age 15 and declined steadily with increasing age. It was slightly weaker for men than women. After controlling for early initiation of drinking, the direct positive effect of family history saturation on the risk of progressing to dependence increased over time and was slightly reduced among individuals who started drinking before age 18. The indirect effect of family history on the risk of developing dependence, via its effect on early drinking as a risk factor for dependence, was strongest in the interval from 3 to 9 years after initiation of drinking.* Conclusions: *The associations between family history and early onset alcoholism appear to be driven most clearly by family history predicting earlier initiation of drinking. The weak effect of family history on the development of dependence within the first 3 years after initiation of drinking may reflect the preponderance of developmentally limited dependence during this time period. The data are consistent with the links established between novelty seeking, impulsivity and early onset alcoholism. While supporting the possibility of genetic effects via dopaminergic and serotonergic function, these findings also suggest that environmental factors may play an important part in helping to explain the association between family history and early onset alcoholism.*

Among the numerous alcoholism typologies that have been proposed in recent decades (Babor, 1996), many have included subtypes whose features include both a positive family history of alcohol problems and an early onset of alcoholism. These include Type II alcoholism (see review in Goodwin, 1984; see also Cloninger, 1981, 1987), Type B alcoholism (Babor et al., 1992; Schuckit et al., 1995) and early onset alcoholism (Buydens-Branchey et al., 1989; Parrella and Filstead, 1988). In addition to early onset and familial aggregation, these subtypes of alcoholism are characterized by the presence of social and legal problems related to alcohol use, including criminality and a history of adolescent conduct disorder or antisocial personality disorder (ASPD).

In fact, Zucker (1987) cited antisocial behavior as the primary factor distinguishing two groups of early onset alcoholics, only one of which shared the genetic predisposition of the Type II/Type B/early onset alcoholics. He labeled members of this group, with early onset of both antisocial behavior and alcohol problems, "antisocial alcoholics." Zucker referred to the other, nonfamilial type of early onset alcoholism as "developmentally limited," because it tended to resolve itself, often without treatment, after the assumption of adult roles and responsibilities. Recent population-based studies (Dawson, 1996, 1998) have suggested that developmentally limited alcoholism may account for a far greater proportion of all alcohol dependence than would be expected based on studies of clinical samples.

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Research into heritable personality characteristics has provided a theoretical basis for the linkage between family history and early onset alcoholism. Cloninger (1987) proposed that the risk of developing alcoholism was influenced by three personality characteristics: novelty seeking, harm avoidance and reward dependence. He predicted that Type II alcoholics would be characterized by high novelty seeking, low harm avoidance and low reward dependence. Based on a review of studies conducted between 1986 and mid-1995, Howard et al. (1997) concluded that novelty seeking has been shown to successfully distinguish Type I/Type II and Type A/Type B alcoholics, in addition to distinguishing alcoholics from nonalcoholics and substance users from nonusers. However, there was little consistent support for the effects of either harm avoidance or reward dependence, or for the superiority of the tridimensional model in general. More recent studies have continued to confirm the importance of novelty seeking (Gabel et al., 1997; Galen et al., 1997; Meszaros et al., 1996), while providing mixed evidence for the role of harm avoidance in the etiology of alcohol dependence. However, a few studies based on population samples have demonstrated an effect of harm avoidance as well as novelty seeking (Cloninger et al., 1995; Mâsse and Tremblay, 1997). None of the recent studies have found a significant effect of reward dependence.

Cloninger (1987) linked novelty seeking and harm avoidance to the production and regulation of dopamine and serotonin. Drug challenge studies attempting to demonstrate an association between dopaminergic function and novelty seeking have reported inconsistent findings. For example, Wiesbeck et al. (1995) reported a significant association, whereas Heinz et al. (1996) failed to replicate this finding with a similar study design. Some studies have reported evidence of an association between polymorphisms at the DRD2 and DRD4 dopamine receptor sites and either novelty seeking (Benjamin et al., 1996; Ebstein et al., 1996), alcoholism (Blum et al., 1990) or early onset alcoholism (Kono et al., 1997); however, neither the majority of individual studies nor a meta-analysis of published reports has found significant associations (Berrettini and Persico, 1996; Chang et al., 1997; Finckh et al., 1996; Geijer et al., 1997; Gelernter et al., 1991, 1993; Higuchi et al., 1996; Sander et al., 1997).

The association between serotonergic function and harm avoidance has received inconsistent support (Limson et al., 1991), but there has been ample evidence of a strong association between a low serotonin turnover rate and impaired impulse control, antisocial personality and early onset alcoholism (see review in Virkkunen and Linnoila, 1997). To date, the genetic evidence for an association between serotonergic activity and age at onset of alcoholism is inconclusive (Virkkunen and Linnoila, 1997).

Of course, not all of the association between family history and early onset alcoholism can be attributed to genetic causes. Twin studies have been used to distinguish genetic from shared environmental effects. On the basis of a review and reanalysis of a number of studies of large twin samples, Heath (1995) concluded that the evidence for environmental effects was weaker than that for genetic effects, but that several studies did find environmental contributions to variance in alcohol consumption and heavy drinking. Similar findings, including evidence of interaction between genetic and environmental effects, have been reported by Rose et al. (1997), based on data from a sample of Finnish twins. Based on studies from a variety of countries, Rose (1998) concluded that the influence of genetic factors on initiation of drinking was negligible relative to environmental effects, but that after initiation of drinking, genetic influence played a significant role in drinking behavior.

The association between family history and early onset alcoholism would be better understood, and the specific genetic and environmental mechanisms could be more appropriately evaluated, if there were more information on *how* early onset alcoholism develops. Does the early onset result from earlier initiation of drinking, from more rapid development of alcohol problems once drinking has begun, or both? Surprisingly few studies have distinguished these two components of the risk of early onset alcoholism. In an early study of 155 male alcoholics in treatment, Penick et al. (1978) found that those with the strongest family histories of alcoholism tended to initiate drinking at a somewhat younger age ($p < .10$). These subjects also reported having experienced, at an earlier age, problems related to excessive drinking ($p < .03$). Schuckit et al. (1995) reported that the significantly earlier onset of dependence found among Type B compared to Type A subjects was based on both drinking and dependence beginning earlier, although the difference in initiation of drinking was significant only for men. The mean ages at initiation of drinking and onset of dependence that were published in that study suggested that the interval between the two was about 2 years shorter for the Type B subjects, but these differences in interval length were not presented directly nor tested for significance. A study of adults with a lifetime history of drug use disorders found that those with adolescent onsets of a substance use disorder had a significantly shorter interval from alcohol use to alcohol depen-

dence than did those with late adult onset of a substance use disorder (Clark et al., 1998). Hill and Yuan (1999) recently reported that high-risk children, as defined by familial density of alcoholism, were more likely to start drinking at younger ages than low-risk children; however, this basically reflected their greater likelihood of drinking at any age. The proportional hazards model used in their analysis assumed that the high-risk children's excess likelihood of initiating drinking was invariant across age and, thus, did not determine whether there were differences by risk status in age at first drink among those who *did* drink.

This study was designed to address the two components of risk involved in early onset alcoholism. It examines the association between family history saturation (the proportion of alcoholic first- and second-degree relatives) and both (1) the risk of initiating drinking and (2) the risk of progressing from drinking initiation to onset of dependence. In addition to using proportional hazards models to adjust for factors that censor an individual's exposure to risk (e.g., stopping drinking), as well as other confounders, the study tests the proportionality assumption to see if the effect of family history saturation in fact varies over time, as opposed to remaining constant. Because the subtype of dependence associated with early onset has been described as occurring predominantly among men, interactions between family history saturation and male gender, as well as other covariates, are tested for statistical significance. In addition, the models testing progression from initiation of drinking to dependence were repeated for that subset of dependence ultimately resulting in treatment seeking. This was done in order to compare the results obtained in this analysis of a large representative national sample of adults with those that might be obtained from a clinical sample.

METHOD

Sample

This study is based on data collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and conducted by the Bureau of the Census. The NLAES sample was representative of U.S. adults 18 years of age and over, residing in households in the civilian, noninstitutionalized population. Data were obtained in personal interviews conducted in respondents' homes and proxy respondents were not permitted. The household and sample person response rates were 92% and 97%, respectively. This yielded a total sample size of 42,862 individuals (58.4% female), among whom the onset of drinking was examined. Of these, 27,616 individuals (50.7% male) were classified as lifetime drinkers who had consumed at least 12 drinks in 1 or more years. The onset of dependence was examined among this group of lifetime drinkers.

The complex, multistage NLAES sample design (Grant et al., 1994) featured selection of primary sampling units with probability proportional to size, and oversampling of blacks and of young adults between the ages of 18 and 29. To adequately incorporate these design effects into variance estimation procedures, the estimates presented in this article were generated by SUDAAN (Shah et al., 1996), a software package that accommodates the design features of complex sample designs.

Measures

Age at onset of drinking was established by asking respondents when they first started drinking, not counting small sips or tastes. In a test-retest conducted in a community sample, the reliability of the age-at-onset variable was 0.72 (Grant et al., 1995). A dichotomous measure of early drinking, used as a control in the models predicting interval from onset of drinking to dependence, was based on age at initiation being less than 18 years versus 18 years or older.

To be classified with lifetime DSM–IV (American Psychiatric Association, 1994) alcohol dependence (i.e., dependence within the year immediately preceding the NLAES interview or any other 1-year period), an individual had to meet at least three of the seven DSM–IV criteria for dependence: (1) tolerance; (2) withdrawal (including relief or avoidance of withdrawal); (3) persistent desire or unsuccessful attempts to cut down on or stop drinking; (4) much time spent drinking, obtaining alcohol or recovering from its effects; (5) reduction or cessation of important activities in favor of drinking; (6) impaired control over drinking; and (7) continued use despite physical or psychological problems caused by drinking.

For the year immediately preceding the interview, criteria with duration qualifiers (stipulating the repetitiveness with which they must occur) were satisfied if one symptom had occurred two or more times or if two or more symptoms had occurred one or more times. Criteria not associated with duration qualifiers were satisfied if a person reported one or more positive past-year symptoms. To be consistent with the syndromal definition of the withdrawal criterion, two or more positive symptoms were required in addition to satis-

faction of the duration qualifier. For the period prior to the past year, respondents had to report that some of the symptoms of dependence occurred "at around the same time" *or* "at around the same time, on and off for a few months or longer" *or* "at around the same time, most days for at least a month." In addition to establishing that duration qualifiers were satisfied, these questions were designed to distinguish symptoms that were clustered at a period in time (as is required for the classification of dependence) from those that may have occurred at different times over the life span. Reliabilities of alcohol use disorders for the past year and the period prior to the past year were 0.76 and 0.73, respectively (Grant et al., 1995).

For individuals who met the criteria for past year alcohol dependence during the year immediately preceding interview but *not* prior to the past year, age at onset of dependence was defined as age at interview. For individuals who met the criteria for prior to past year dependence, age at onset of dependence was established by asking the age at which symptoms first began to co-occur as defined above.

Family history saturation was derived from respondents' reports of alcoholism/problem drinking (defined as drinking-related physical or emotional problems; problems with a spouse, family or friends; problems at work; problems with the police, like drunk driving; or spending a lot of time drinking or being hungover) in 18 types of first- and second-degree relatives. The proportion of alcoholic first- and second-degree relatives was obtained by dividing the number of alcoholic relatives by the total number of relatives who lived to be at least 10 years of age. Reliabilities for family history of alcoholism exceeded 0.70 for all types of relatives except sons and daughters, for whom they were 0.65 each (Grant et al., 1995).

Consumption measures were defined for the period identified by each respondent as his or her period of heaviest drinking. Average daily ethanol intake was derived by dividing the annual volume by 365, where annual volume was the sum over beer, wine and distilled spirits of the following: (number of drinking days per $year_{usual\ -\ heavy}$) (number of drinks per drinking day_{usual}) (size of drink in $ounces_{usual}$) (ethanol conversion factor) + (number of drinking days per $year_{heavy}$) (number of drinks per drinking day_{heavy}) (size of drink in $ounces_{heavy}$) (ethanol conversion factor). Ethanol conversion factors were defined as 0.045 for beer, 0.121

Table 1. Number and percentage distribution of adults 18 years of age and over and average percentage of first- and second-degree relatives who were alcoholics or problem drinkers, by age at onset of alcohol dependence, age at initiation of drinking and interval from onset of drinking to dependence

		Distribution of			
	Number of cases	All adults % (± SE)	Lifetime drinkers % (± SE)	Individuals with lifetime dependence % (± SE)	Average % of alcoholic relatives % (± SE)
Total[a]	42,862	100.0 (0.0)	100.0 (0.0)	100.0 (0.0)	7.9 (0.1)
Age at onset of dependence					
<18 years	773	1.9 (0.1)	3.1 (0.1)	15.4 (0.6)	20.0 (0.8)
18-20 years	1,600	4.0 (0.1)	6.4 (0.2)	31.6 (0.8)	14.3 (0.4)
21-24 years	1,061	2.6 (0.1)	4.0 (0.1)	20.2 (0.6)	15.3 (0.5)
≥25 years	1,888	4.1 (0.1)	6.6 (0.2)	32.8 (0.8)	14.6 (0.4)
Never dependent	37,450	87.4 (0.2)	79.9 (0.3)	0.0 (0.0)	6.8 (0.1)
Age at initiation of drinking					
<15 years	1,999	4.8 (0.1)	7.5 (0.2)	15.7 (0.6)	15.7 (0.4)
15-17 years	7,602	19.2 (0.3)	29.8 (0.4)	43.7 (0.8)	11.4 (0.2)
18-20 years	10,079	24.0 (0.3)	37.2 (0.4)	28.4 (0.7)	8.1 (0.1)
≥21 years	7,401	16.4 (0.2)	25.5 (0.3)	12.2 (0.5)	7.2 (0.1)
Lifetime abstainer	15,426	35.6 (0.4)	0.0 (0.0)	0.0 (0.0)	5.2 (0.1)
Interval from initiation of drinking to onset of dependence					
<3 years	1,883	NA	7.6 (0.2)	36.8 (0.8)	14.7 (0.5)
3-4 years	920	NA	3.7 (0.1)	17.9 (0.6)	16.1 (0.6)
5-9 years	1,040	NA	4.1 (0.1)	19.9 (0.6)	17.4 (0.6)
≥10 years	1,437	NA	5.2 (0.2)	25.4 (0.7)	14.8 (0.5)
Never dependent[b]	22,204	NA	79.4 (0.3)	0.0 (0.0)	7.8 (0.1)

[a]Including individuals with unknown age at onset of dependence and drinking and unknown duration from onset of drinking to dependence.
[b]Excluding lifetime abstainers.

for wine and 0.409 for distilled spirits ("Beer analysis," 1992; DISCUS, 1985; Kling, 1989; Turner, 1990; Williams et al., 1993). The test-retest reliability of average daily ethanol intake during period of heaviest consumption was 0.71 (Grant et al., 1995).

Analysis

The risks of (1) initiating drinking and (2) progressing from drinking to dependence were ascertained using SUDAAN discrete time proportional hazards models (Shah et al., 1996). In the models estimating initiation of drinking, individuals were followed from age 10 to the age at which they started drinking and were withdrawn from risk (censored) at their age at interview if they were lifetime abstainers. Individuals who initiated drinking at or before age 10 were assumed to have started drinking in the first year of exposure. In the models estimating progression to dependence, individuals were followed from age at first drink to age at onset of dependence, and those who never became dependent were withdrawn (censored) at the earlier of either age at interview or age at cessation of drinking.

Proportional hazards models assume that the associations between the predictor variables (e.g., family history) and the outcome events remain constant over time even though the underlying hazard functions of event occurrence change. In order to test this assumption, separate SUDAAN models were run for different intervals of the exposure periods in question, and the Beta coefficients for the effect of family history saturation were compared across these intervals. In addition, the assumption of proportionality was tested directly using the SAS procedure PHREG (SAS, 1996), which measures the magnitude and significance of parameter changes over time (but does not account for complex sample design effects).

RESULTS

The majority of U.S. adults (87.4% of all adults 18 years of age and over, 79.9% of lifetime drinkers) never met the criteria for lifetime alcohol dependence (Table 1). Among those classified as positive for lifetime dependence, almost half experienced its onset before reaching the legal drinking age (15.4% before age 18 and 31.6% between ages 18 and 20). Average family history saturation (% of alcoholic first- and second-degree relatives) declined from 20.0% among individuals with onset of alcohol dependence prior to 18 years to between 14.3% and 15.3% of those with later onsets of dependence and 6.8% among those without alcohol dependence.

The percentage of alcoholic relatives showed a more clearly linear decrease with age at initiation of drinking, falling from 15.7% among those who started drinking before age 15 to 7.2% of those who started drinking at age 21 or later. Lifetime abstainers reported the lowest levels of familial alcoholism, an average of 5.2% of their relatives. The proportion of alcoholic relatives showed far less variation with interval from initiation of drinking to onset of dependence. It was slightly higher for those with intervals of 3–4 and 5–9 years (16.1% and 17.4%, respectively) than for those with durations of either <3 years (14.7%) or ≥10 years (14.8%).

Table 2 presents the Beta parameters and standard errors (SEs) from multivariate proportional hazards models predicting the hazard of initiating drinking from age 10 onward. The model on the left assumes a constant effect of the independent variables at all subsequent ages; the models on the right test for varying effects at different ages. All of the models indicated that the hazard of initiating drinking declined with age

Table 2. Survival models predicting hazard of initiating drinking at various ages

	Models predicting hazard of initiating drinking at				
	All ages Beta (±SE)	<15 years Beta (±SE)	15–17 years Beta (±SE)	18–20 years Beta (±SE)	≥21 years Beta (±SE)
Main effects					
Age	-0.0212 ± .0005‡	-0.0229 ± .0018‡	-0.0348 ± .0011‡	-0.0201 ± .0007‡	-0.0094 ± .0008‡
Male	0.7575 ± .0194‡	0.9450 ± .0680‡	0.8138 ± .0360‡	0.8035 ± .0295‡	0.6543 ± .0340‡
Black	-0.5756 ± .0299‡	-0.5052 ± .0995‡	-0.7607 ± .0576‡	-0.5596 ± .0422‡	-0.4027 ± .0480‡
Hispanic	-0.5017 ± .0370‡	-0.0914 ± .1090	-0.3731 ± .0598‡	-0.6168 ± .0532‡	-0.5929 ± .0684‡
Not 2-parent household	0.0865 ± .0212‡	0.3684 ± .0593‡	0.1475 ± .0343‡	-0.0379 ± .0327	0.0437 ± .0393
% of ARs	0.0220 ± .0009‡	0.0371 ± .0020‡	0.0255 ± 0.013‡	0.0155 ± .0013‡	0.0220 ± .0015‡
Interaction terms					
Male × % of ARs	-0.0038 ± .0013*	-0.0056 ± .0027)*	-0.0067 ± .0018‡	-0.0039 ± .0019*	-0.0090 ± .0027‡

*p <.05; ‡p <.001.
Notes: SE = standard error; ARs = alcoholic relatives.

(i.e., that older cohorts had been less likely to drink), was greater for men than women and was smaller for blacks than for whites. At ages 15 and older (but not before age 15), Hispanic origin was associated with a reduced likelihood of initiating drinking, and prior to age 18 (but not thereafter) the likelihood of starting to drink was higher among individuals who did not grow up with both biological parents.

The percentage of alcoholic relatives was positively associated with the hazard of initiating drinking; this positive effect was slightly lower among men, however, as indicated by the negative association between family history and male gender. The Beta coefficient representing the association between familial alcoholism and the hazard of initiating drinking was greatest (Beta = 0.0371) in the model predicting initiation of drinking prior to age 15; it decreased in subsequent models, except for a slight upturn in the model predicting initiation of drinking at ages 21 and older. The coefficient for the interaction between family history and male gender became increasingly negative with time. (When tested using the SAS procedure PHREG, the changes in the Beta coefficients for both family history saturation and its interaction with male gender were highly significant: $p < .0001$ and $p = .002$, respectively.)

Table 3 presents the hazard rate ratios (HRR) associated with these model parameters. Compared to a man with no alcoholic relatives, a man with 25% alcoholic relatives would be 2.20 times as likely to initiate drinking prior to age 15. By ages 21 and older, his excess risk of initiating drinking would have declined to 1.38 times that of a man with no alcoholic relatives. Among women, the respective decline in the hazard rate ratio was from 2.53 before age 15 to 1.73 at ages 21 and older. Lower proportions of alcoholic relatives yielded lower increases in the risk of initiating drinking, but even among individuals with only 5% alcoholic relatives the increased likelihood of starting to drink was statistically significant ($p < .05$).

The models in Table 4 examine the hazard of developing alcohol dependence among drinkers (i.e., conditional upon having initiated drinking). In all models, the likelihood of becoming dependent was lower for the older age cohorts and was higher for men than women. Compared to whites, blacks had a lower likelihood of becoming dependent in the intervals shortly after initiation of drinking, but a greater likelihood in the period 10 years or more after drinking began. Hispanics also demonstrated an increased hazard of dependence in the interval 10 or more years after initiation of drinking, but did not differ from non-Hispanics in earlier intervals. Increasing average daily ethanol intake and being a daily drinker were both associated with an elevated likelihood of becoming dependent, and the positive effects of both of these drinking behaviors increased over time, suggesting a cumulative effect. Individuals who started to drink before age 18 had an increased risk of dependence in all intervals except the first 3 years after initiation of drinking. The effect was strongest, a more than two-fold increase in the risk of becoming dependent, in the periods 3–4 years and 5–9 years after initiation of drinking.

The percentage of alcoholic relatives was positively associated with the likelihood of developing alcohol dependence, but this positive effect was substantially reduced among persons who initiated drinking before age 18. The underlying effect of family history revealed a significant increase over time ($p < .0001$ as tested using the SAS procedure PHREG). Although the interaction between early drinking and familial alcoholism appeared to become increasingly negative with time, this change was not significant when tested in PHREG ($p = .72$).

Table 3. Hazard rate ratios for initiation of drinking for individuals with selected percentages of alcoholic first- and second-degree relatives (ARs)

	Models predicting hazard of initiating drinking at				
	All ages	<15 years	15-17 years	18-20 years	≥21 years
	HRR (95% CI)	HRR (95% CI)	HRR (95% CI)	HRR (95% CI)	HRR (95% CI)
Male					
0% ARs	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
5% ARs	1.10 (1.09-1.11)	1.17 (1.15-1.19)	1.10 (1.09-1.11)	1.06 (1.04-1.08)	1.07 (1.05-1.10)
10% ARs	1.20 (1.18-1.23)	1.37 (1.32-1.42)	1.21 (1.18-1.24)	1.12 (1.09-1.15)	1.14 (1.09-1.19)
25% ARs	1.58 (1.50-1.66)	2.20 (2.00-2.42)	1.60 (1.50-1.71)	1.34 (1.24-1.44)	1.38 (1.23-1.55)
Female					
0% ARs	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
5% ARs	1.12 (1.11-1.13)	1.20 (1.18-1.22)	1.14 (1.13-1.15)	1.08 (1.07-1.09)	1.12 (1.10-1.14)
10% ARs	1.25 (1.23-1.27)	1.45 (1.39-1.51)	1.29 (1.26-1.32)	1.17 (1.14-1.20)	1.25 (1.21-1.29)
25% ARs	1.73 (1.66-1.81)	2.53 (2.30-2.79)	1.89 (1.77-2.02)	1.47 (1.38-1.57)	1.73 (1.61-1.86)

As shown in Table 5, among individuals who started drinking before age 18, the effect of having 25% alcoholic relatives compared to none (a negative family history) was a 34% increase (HRR = 1.34) in the likelihood of becoming dependent within the first 3 years after initiation of drinking. By 10 years or more after first drink, the effect had grown to an 82% increase (HRR = 1.82) in the risk of becoming dependent. Among individuals who started drinking later, the hazard rate ratio increased from 1.65 in the first 3 years after initiation of drinking to 2.50 in the period 10 years or more after drinking commenced. As with the models predicting initiation of drinking, the excess hazards associated with even low levels of familial alcoholism were all significant ($p < .05$).

When the models from Tables 4 and 5 were rerun using a continuous measure of age at initiation of drinking or a different cutpoint for early drinking (<15 years), the patterns remained the same as when the dichotomous measure of initiating drinking before or after 18 years of age was used (data not shown). When separate sets of models were run for dependence ultimately resulting and not resulting in treatment, the positive Beta parameters for family history saturation increased over time and were offset by a negative interaction with early initiation of drinking in both sets of models. However, in the models predicting onset of dependence resulting in treatment, the Beta coefficients for family history saturation were nearly twice as large and the negative interactions with early initiation of drinking only about half as large as in the models predicting onset of dependence not resulting in treatment (data not shown).

DISCUSSION

In a general population of U.S. adults 18 years of age and over, survival analyses indicated that the positive association between the percentage of alcoholic first- and second-degree relatives and early onset of alcoholism was a result of earlier initiation of drinking rather than a more rapid development of dependence once drinking had begun. In fact, after controlling for early initiation of drinking, the direct effect of familial alcoholism on the likelihood of developing dependence actually showed an increasingly positive value over time, suggesting a stronger link with more slowly developing rather than rapidly developing dependence. The same pattern was observed when early initiation of drinking was *not* included as a control, both in multivariate models that excluded this factor (data not shown) and in the simple bivariate associations between family history saturation and interval from first drink to dependence.

However, the indirect effects of family history on the development of dependence, via its association with early initiation of drinking, must also be born in mind. Early initiation of drinking, itself positively associated

Table 4. Survival models predicting hazard of developing alcohol dependence within various intervals following initiation of drinking

	Models predicting hazard of developing dependence following initiation of drinking				
	All intervals after Beta (± SE)	<3 years after Beta (± SE)	3-4 years after Beta (± SE)	5-9 years after Beta (± SE)	≥10 years after Beta (± SE)
Main effects					
Age	-0.0650 ± .0021‡	-0.0578 ± .0027‡	-0.0682 ± .0048‡	-0.0688 ± .0049‡	-0.0696 ± .0048‡
Male	0.2846 ± .0323‡	0.2254 ± .0560‡	0.3942 ± .0850‡	0.3946 ± .0850‡	0.2112 ± .0703‡
Black	-0.1812 ± .0621*	-0.6404 ± .1189‡	-0.3122 ± .1593	-0.0902 ± .1367	0.2612 ± .1147*
Hispanic	0.0919 ± .0734	-0.1985 ± .1353	0.1149 ± .0598	-0.0161 ± .1564	0.4154 ± .1493*
Average daily ethanol intake	0.0221 ± .0036‡	0.0183 ± .0041‡	0.0140 ± .0041‡	0.0281 ± .0052‡	0.0369 ± .0055‡
Daily drinker	0.9205 ± .0393‡	0.6021 ± .0788‡	0.8796 ± .0968‡	1.0234 ± .0808‡	1.2572 ± .0713‡
Early initiation of drinking[a]	0.4096 ± .0493‡	0.0856 ± .0734	0.7556 ± .1147‡	0.7690 ± .1135‡	0.3261 ± .0959‡
% of ARs	0.0284 ± .0019‡	0.0201 ± .0026‡	0.0272 ± 0.042‡	0.0347 ± .0041‡	0.0367 ± .0033‡
Interaction terms					
Early initiation of drinking[a] × % of ARs	-0.0112 ± .0022‡	-0.0084 ± .0031*	-0.0107 ± .0047‡	-0.0122 ± .0047*	-0.0127 ± .0046‡

[a]Before 18 years of age.
*$p < .05$; ‡$p < .001$.
Notes: SE = standard error; ARs = alcoholic relatives.

with familial alcoholism, was a powerful risk factor for the development of dependence, and the positive effect of early drinking was far stronger in the intervals 3–9 years after initiation of drinking than in either earlier or later periods. As an indirect reflection of family history, the reduction in the effect of early drinking in the period 10 or more years after initiation of drinking would be consistent with the linkage between family history and early onset of dependence. For this to be so, however, there needs to be some explanation for why the effect of early drinking is also relatively low in the first 3 years after initiation of drinking.

There are several reasons why alcoholism developing in the first 3 years of drinking might *not* show a strong association with family history of alcohol problems. These first 3 years typically occur during the teen years or early twenties, when alcohol use per se is illegal. Because it is illegal use, it is likely to take place in unregulated circumstances in which there are few social constraints preventing intoxication and associated problems. This type of illegal, uncontrolled drinking is particularly conducive to the development of alcohol abuse symptoms. However, it may also be associated with dependence criteria, including: excessive time required to obtain alcohol (because of being underage) or to recover from its effects, drinking more or for longer than intended and hangover symptoms confused with symptoms of withdrawal. Thus, there may be problems associated with early use of alcohol that would be sufficient to result in a classification of alcohol dependence even in the absence of any genetic predisposition to the disorder.

Type II early onset alcoholism was originally described as male limited (Cloninger, 1987) and is thought to occur much more frequently among men than women. In the models used for this analysis, male gender was a positive risk factor both for initiating drinking and for developing alcohol dependence. As a predictor of initiation of drinking, male gender had a negative interaction with family history, indicating that familial alcoholism was actually a stronger risk factor for women than men. This may reflect the fact that drinking, especially at very young ages, is less common among women than men and therefore less likely to occur in the absence of some genetic predisposition or psychopathology that might be associated with family history of alcoholism. It is interesting to note, however, that the reduction in family history effect caused by male gender was smallest at the youngest ages of drinking initiation. This would be consistent with familially transmitted alcoholism being more strongly male-limited among early onset alcoholics than among later onset alcoholics.

The evidence showing the association of family history with early onset alcoholism as, primarily, the result of an earlier age at initiation of drinking is consistent with studies indicating the possible role of the dopaminergic and serotonergic systems in the genetics of alcoholism. Both novelty seeking and impulsivity may be predictive of starting to drink at an early age and have been associated with these systems. However, the fact that serotonin function is also associated with anxiety, a factor often associated with Type I/Type A alcoholism, might make it an unlikely candidate for discriminating between early and late onset alcohol dependence. In addition, the evidence from twin studies suggests that initiation of drinking is more strongly influenced by shared environmental than genetic factors. Children of alcoholics might simply start drinking earlier because of easier access to alcohol in

Table 5. Hazard rate ratios for onset of alcohol dependence for individuals with selected percentages of alcoholic first- and second-degree relatives (ARs)

	Models predicting hazard of developing dependence following initiation of drinking									
	All intervals after		<3 years after		3-4 years after		5-9 years after		≥10 years after	
	HRR	(95% CI)	HRR	(95% CI)	HRR	(95% CI)	HRR	(95% CI)	HRR	(95% CI)
Initiation of drinking at <18 years										
0% ARs	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
5% ARs	1.09	(1.02-1.17)	1.06	(1.04-1.08)	1.09	(1.07-1.11)	1.12	(1.08-1.15)	1.13	(1.10-1.16)
10% ARs	1.19	(1.04-1.36)	1.12	(1.07-1.17)	1.18	(1.13-1.23)	1.25	(1.19-1.31)	1.27	(1.20-1.35)
25% ARs	1.54	(1.10-2.15)	1.34	(1.21-1.49)	1.51	(1.35-1.69)	1.76	(1.56-1.78)	1.82	(1.57-2.11)
Initiation of drinking at 18+ years										
0% ARs	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
5% ARs	1.15	(1.13-1.17)	1.11	(1.08-1.14)	1.15	(1.10-1.20)	1.19	(1.14-1.24)	1.20	(1.16-1.24)
10% ARs	1.33	(1.28-1.38)	1.22	(1.16-1.29)	1.31	(1.21-1.42)	1.41	(1.30-1.53)	1.44	(1.35-1.54)
25% ARs	2.03	(1.85-2.23)	1.65	(1.45-1.88)	1.97	(1.60-2.43)	2.38	(1.95-2.91)	2.50	(2.13-2.94)

the home, familial acceptance of drinking or poor monitoring of children's behavior by alcoholic parents.

It must be stressed that the family history measure used in this analysis does not differentiate between genetic and environmental effects. If these effects varied over time in offsetting ways, then this study's estimates of the overall family history effect might not adequately represent the variation over time in either its genetic or its environmental components. Moreover, the family history saturation measure is not a true reflection of genetic loading or liability, in that it gives equal weight to both first- and second-degree relatives. However, when the analyses were repeated using a family history saturation measure based on first-degree relatives only, the changes over time in the effect of family history remained unchanged, and the same was true when a dichotomous measure of family history of alcoholism was used (data not shown). Finally, the measure of family history saturation provides only a crude indication of environmental exposure, as it is unknown whether the alcoholic relatives lived with the respondents or even whether the relatives' alcohol problems occurred during the respondents' lifetimes. In addition, the family history measure was based on a count of all relatives who had ever been alcoholic as of the time of the NLAES interview. Thus, the proportion of alcoholic relatives did not change, over the exposure periods, for the risks of initiating drinking and becoming dependent, as it would in real life (e.g., if a parent did not develop alcohol problems until the respondent was in his or her 30s). To whatever extent this may have resulted in overestimation of family history saturation at the respondents' early ages, it may have underestimated the positive association of family history with the risks of initiating drinking and becoming dependent during those years.

Among the strengths of this study is the large national population sample, which included individuals old enough to have been able to develop late onset alcoholism (with age at interview having been controlled as a possible confounder). The study's deconstruction of the risk of dependence into the component risks of initiating drinking and of going on to develop dependence is also important. However, the results need to be interpreted with caution due to a number of limitations, many of which are common to cross-sectional retrospective studies. Primary among these is recall error, compounded by the possibility that, over time, increases in recall error (e.g., telescoping of events) could confound the identification of genuine cohort changes in ages at initiation of drinking and onset of dependence, and their association with familial alcoholism.

The developmental literature has suggested that early drinking and subsequent alcohol problems are linked, both reflecting a broader range of deviant behaviors commonly influenced by a host of personality factors and perceived environmental factors (Jessor and Jessor, 1977; Donovan et al., 1983). Data from both the ongoing Monitoring the Future study (Johnston et al., 1999) and from cohort analyses of the NLAES data (Grant, 1997) have demonstrated that early initiation of drinking has become progressively more normative among recent age cohorts. In view of this trend, one might expect that the association of age at initiation of drinking with both familial alcoholism and the risk of developing alcohol dependence might be weaker among the younger cohorts included in this analysis. In fact, Donovan et al. (1999) did find some significant variation over time (from 1972 to 1992) in the correlations of problem drinking with perceived parental and friends' approval of drinking, although the changes did not form a clearly linear trend. A logical and important extension of this research would be to replicate the findings in different age cohorts and to assess the effect of using a relative measure of early initiation of drinking (e.g., above or below the median age for the respondent's age cohort) rather than a measure based on a fixed age.

This analysis also suffered from the two important limitations for which earlier studies have been criticized (Vaillant, 1994): lack of control for environmental influences (as discussed above) and lack of control for childhood conduct and/or antisocial personality disorder in the respondents themselves and in their relatives. Each of these has the potential to bias interpretations of the data linking family history and early onset alcoholism. In the case of the former, the environmental effects of alcoholic parents may be stronger at younger ages while the individual is still living with his or her parents. In the latter, the considerable overlap of the characteristics of early onset alcoholism and ASPD could affect interpretation of the data. A better understanding of whether and how family history affects the risk of early onset alcoholism will need to account for these factors while retaining the positive aspects of this analysis.

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CHAPTER 5
ALCOHOL AND DEPRESSION

Comorbidity Between DSM–IV Alcohol Use Disorders and Major Depression: Results of a National Survey

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The purpose of this study was to describe detailed patterns of comorbidity between Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM–IV) alcohol use disorders and major depression using a representative sample of the United States. Comorbidity rates and associations between DSM–IV alcohol use disorders and major depression were expressed as odds ratios with confidence intervals adjusted for the complex design characteristics of the NLAES. Comorbidity analyses were presented by sex, ethnicity and age for past year, prior to past year and lifetime diagnoses. Virtually all odds ratios were significantly greater than 1.0, demonstrating that comorbidity of alcohol use disorders and major depression is pervasive in the general population. The magnitude of the association remained stable across the three time frames but diagnostic and subgroup variations in comorbidity were noted. The association between alcohol dependence and major depression was greater than the association between abuse and major depression and the association between alcohol abuse and major depression was consistently greater for females and blacks, compared to their male and non-black counterparts. Implications of the results are discussed in terms of professional help seeking, the self-medication hypothesis, and differential social control theory.

INTRODUCTION

The co-occurrence of alcohol use disorders and major depression has frequently been reported in alcoholic and psychiatric patient samples (Allen and Francis, 1986; Demitio, 1989; Keeler et al., 1979; Petty, 1992). Research conducted in treated samples has also highlighted the clinical relevance of such comorbidity as adversely affecting the course, treatment and prognosis of both alcohol use disorders and major depression (Keitner et al., 1991; Pottenger et al., 1978; Rounsaville et al., 1987). However, studies of patients in treatment are not well suited to the study of the distribution of comorbidity, since patterns of comorbidity found in clinical samples are not representative of those found in the general population. Individuals in treatment are more likely to have multiple disorders than cases in the general population (Ross et al., 1988; Rounsaville et al., 1991; Wolf et al., 1988), thus spuriously inflating estimates of the prevalence of comorbidity (Berkson, 1946).

General population surveys designed to reliably study the distribution of comorbidity are rare. To date, only two major studies have considered psychiatric comorbidity, including alcohol use disorders and major depression, in the United States. The first was the Epidemiologic Catchment Area (ECA) Survey (Robins et al., 1991), in which 18,571 respondents were interviewed in a series of five community-based epidemiologic studies in the early 1980s. The second was the National Comorbidity Survey (NCS), a nationally representative sample of 8,098 respondents conducted in 1991 (Kessler et al., 1994).

The purpose of the present report was to present detailed descriptive epidemiological data on the association between alcohol use disorders and major depression in the third and most recent national comorbidity study of 42,862 respondents, the National Institute on Alcohol Abuse and Alcoholism's (NIAAA) National Longitudinal Alcohol Epidemiologic Survey (NLAES) (Grant et al., 1994; Massey et al., 1989). Several methodological advantages of the NLAES and its psychiatric assessment instrument are noteworthy. First, the diagnostic interview used in the NLAES, the Alcohol Use Disorder and Associated Disabilities Interview Schedule (Grant and Hasin, 1992) yielded diagnoses based on the most current psychiatric classification of the Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition (DSM–IV; American Psychiatric Association, 1992) and not its

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predecessors, the Third Edition Revised (DSM–III–R; American Psychiatric Association, 1987) used in the NCS or the Third Edition (DSM–III; American Psychiatric Association, 1980) used in the ECA. Second, unlike the NLAES, the ECA was not a nationally representative sample of the United States population and the relatively small sample size of the NCS could not provide for reliable and detailed analyses of comorbidity among major subgroups of the population defined by sex, ethnicity or age. This is a serious limitation since clinical studies have shown comorbidity rates to vary by important sociodemographic variables (Roy et al., 1991a, 1991b; Woodruff et al., 1979).

One of the unique aspects of the AUDADIS was its syndromal measurement of alcohol use disorders as defined in the DSM–IV. The Diagnostic Interview Schedule (DIS; Robins et al., 1981) used in the ECA and the University of Michigan-Composite International Diagnostic Interview (UM–CIDI; Wittchen and Kessler, 1991) used in the NCS both failed to measure the syndromal aspects of alcohol use disorders as defined by the DSM–III and DSM–III–R. For example, to achieve a DIS and UM–CIDI lifetime diagnosis of DSM–III or DSM–III–R alcohol dependence, a respondent only needed to report two or three positive dependence symptoms, respectively, over the entire life course. In both interviews, only one positive symptom was necessary to meet diagnostic criteria for an alcohol abuse diagnosis on a lifetime basis. Similarly, the DIS and UM–CIDI defined current or past year alcohol dependence as meeting the criteria for a lifetime diagnosis with the occurrence of at least one dependence symptom during the past year. Unlike the DIS and UM–CIDI, the AUDADIS alcohol use disorders are represented syndromally, that is, as the clustering of enough symptoms of abuse or dependence at the same time to achieve a diagnosis. Alcohol use disorders are defined for three time frames in the AUDADIS, the past year, prior to the past year and on a lifetime basis. It is important to note the AUDADIS redefined the current operationalization of lifetime diagnosis as described in the ECA and NCS. With the AUDADIS, the prevalence of lifetime diagnosis was now defined as the occurrence of an episode of either alcohol abuse or dependence at any time in a respondent's life, rather than a loose collection of abuse or dependence symptoms accumulating over the life course.

The NLAES overcomes many of the methodological problems inherent in the ECA and NCS, including sampling difficulties and small sample sizes, the lack of current definitions of alcohol use disorders and major depression, the absence of detailed comorbidity analyses among important subgroups of the population, and the failure to represent alcohol use disorders as syndromes. Thus, the basic comorbidity rates and associations between alcohol use disorders and major depression derived from the NLAES are likely to be more reliable and precise than those reported from either the ECA and NCS.

METHODS

Study sample

Prevalence and comorbidity estimates of DSM–IV alcohol use disorders and major depression were based on the 1992 NLAES, a national probability survey sponsored by the NIAAA. Field work for the study was conducted by the United States Bureau of the Census. Direct face-to-face interviews were conducted with 42,862 respondents, 18 years of age and older, residing in the non-institutionalized population of the contiguous United States, including the District of Columbia. The household response rate was 91.9% and the sample person response rate was 97.4%.

The NLAES featured a complex multistage design (Grant et al., 1994). Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probability proportional to size. From a sampling frame of approximately 2,000 PSUs, 198 were selected for inclusion in the 1992 NLAES sample, including 52 which were self-representing — that is, selected with certainty. Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically for sample. Oversampling of the black population was accomplished at this stage of sample selection to secure adequate numbers for analytic purposes. Segments then were divided into clusters of approximately 4–8 housing units, and all occupied housing units were included in NLAES. Within each household, 1 randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18–29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavy drinking population subgroup. This subgroup of young adults was randomly sampled at a ratio of 2.25% to 1.00%.

Diagnostic assessment

Diagnoses of DSM–IV alcohol use disorders and major depression were derived from the AUDADIS, a fully structured psychiatric interview designed to be administered by trained interviewers who were not clinicians.

The AUDADIS included an extensive list of symptom questions that operationalized the DSM–IV criteria for alcohol use disorders and major depression. These questions are described in detail elsewhere (Grant et al., 1994). Although the DSM–IV was not published until 1994, the specific diagnostic criteria of interest were known prior to the conduct of the NLAES (American Psychiatric Association, 1991) and, therefore, incorporated in their entirety within the AUDADIS. In an independent test-retest study conducted in the general population AUDADIS diagnoses of alcohol use disorders and major depression were shown to be highly reliable, achieving reliability (κ) coefficients ranging between 0.73–0.76 and 0.60–0.65 for the various time frames, respectively (Grant et al., 1995).

Consistent with the DSM–IV, an AUDADIS diagnosis of alcohol abuse required that a person exhibit a maladaptive pattern of alcohol use, leading to clinically significant impairment or distress, as demonstrated by at least one of the following in any one year: (1) continuing to drink despite a social or interpersonal problem caused or exacerbated by the effects of drinking; (2) recurrent drinking in situations in which alcohol use is physically hazardous; (3) recurrent drinking resulting in a failure to fulfill major role obligations; or (4) recurrent alcohol-related legal problems. An AUDADIS diagnosis of alcohol dependence required that a person meet at least 3 of 7 criteria defined for dependence in any 1 year including: (1) tolerance; (2) withdrawal or relief or avoidance of withdrawal; (3) persistent desire or unsuccessful attempts to cut down or stop drinking; (4) spending much time drinking or recovering from its effects; (5) giving up or reducing occupational, social or recreational activities in favor of drinking; (6) impaired control over drinking; and (7) continuing to drink despite a physical or psychological problem caused or exacerbated by drinking.

Unlike the DIS and UM–CIDI, the AUDADIS diagnoses of alcohol abuse and dependence also satisfied the clustering or duration criteria of the DSM–IV definition. In the DSM–III, the duration criterion associated with abuse and dependence specified each disturbance to persist for at least 1 month, a requirement not met in the DIS. In the DSM–III–R, the duration or clustering criterion for symptoms was directly applied to the categories of alcohol abuse and dependence (i.e. some symptoms of the disorder needed to occur consistently for at least a month or repeatedly over a longer period of time). This aspect of the DSM–III–R definition of abuse and dependence was ignored in the UM–CIDI. In contrast, the duration criteria of the DSM–IV included the requirement for a clustering of symptoms within any 1 year period, in addition to associating duration qualifiers with certain abuse and dependence symptoms. The duration qualifiers are defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often" and "persistent" appearing in the diagnostic criteria.

Not only were the duration criteria represented in past year AUDADIS diagnoses of abuse and dependence, but the corresponding prior to the past year diagnoses were also measured as syndromes, or the clustering of the required number of symptoms necessary to achieve a diagnosis. The method used to establish the clustering of symptoms for prior to the past year diagnoses was that of recapitulation. This entails the summarization of symptoms occurring in the past by the interviewer who then separately determines through a series of additional questions whether the required number of symptoms of abuse and dependence occurred at the same time in the past or either (1) continuously for a month or longer, (2) repeatedly over the period of a month or longer. Respondents classified with a lifetime diagnosis encompassed all those who had ever experienced an episode of abuse or dependence, in the past year and/or prior to the past year rather than those demonstrating the required number of symptoms of these disorders over the life course.

Episodes of DSM–IV major depressive disorder were also constructed for the past year, prior to the past year, and lifetime in order to allow for comorbidity analyses within each time frame. Consistent with the DSM–IV, the AUDADIS diagnoses of major depression required the presence of at least 5 depressive symptoms (inclusive of depressed mood or loss of pleasure and interest) nearly every day for most of the day for at least the same 2 week period. In contrast to the DSM–III–R definition of major depression, social and/or occupational dysfunction must also have been present during the disturbance, and episodes of DSM–IV major depression exclusively due to bereavement and physical illness were ruled out.

Statistical analysis

Because of the complex survey design of the NLAES, variance estimation procedures that assume a simple random sample cannot be employed. Statistical research has shown that clustering and stratification specifications of the NLAES sample may result, in certain instances, in standard errors somewhat larger than those that would be obtained with a simple random sample of equal size (Massey et al., 1989). To take

into account the NLAES sample design, all standard errors of the prevalence estimates and comorbidity rates (expressed as weighted percentages) presented here were generated using SUDAAN (Research Triangle Institute, 1994), a software program that uses Taylor series linearization to adjust for sample design characteristics.

Associations between alcohol use disorders and major depression were expressed in terms of odds ratios. Odds ratios and their 95% confidence intervals were derived from separate logistic regression analyses using the SUDAAN LOGISTIC program that also adjusted for the complex sampling design of the NLAES. An odds ratio of greater than 1.0 reflects a positive association between the comorbid disorders and is statistically significant if its 95% confidence interval does not encompass the value of 1.0. An odds ratio of 1.0, or indistinguishable from 1.0 as bounded by the 95% confidence intervals, was considered non-significant.

It is important to note that the present analyses focused on what has been termed episode or period comorbidity, or the co-occurrence of two or more psychiatric disorders at the same time or during the same interval. Episode comorbidity should be contrasted with comorbidity viewed from the primary-secondary distinction in which one of two or more comorbid disorders is designated as primary, usually on the basis of its first occurrence or onset at an earlier age. An important consequence of examining the co-occurrence of disorders from a period comorbidity perspective, rather than a primary-secondary perspective is that the odds ratios are equivalent regardless of whether alcohol use disorders or major depression is designated as the index or focal disorder.

RESULTS

Prevalence rates

Table 1 shows the prevalence rates of DSM-IV major depression for each of the three time frames and by each sex, ethnic and age subgroup of the population in which comorbidity rates were examined. Nearly 10.0% of the respondents had a history of major depression, with 3.33% and 7.73% having an episode in the past 12 months and prior to that time, respectively. Rates of major depression in the past year, prior to the past year and lifetime were 29.4%, 19.7% and 21.3% greater among females than males. Rates among non-blacks also exceeded those of their black counterparts by 25.6% for the past year and about 40.0% for episodes occurring prior to the past year and on a lifetime basis.

Table 1. Prevalence of DSM-IV major depression by sex, ethnicity and age: United States, 1992

Sociodemographic characteristic	Past year % (S.E.)	Prior to past year % (S.E.)	Lifetime % (S.E.)
Total	3.33 (0.10)	7.73 (0.16)	9.86 (0.18)
Sex			
Male	2.74 (0.16)	6.85 (0.23)	8.64 (0.26)
Female	3.88 (0.14)	8.54 (0.21)	10.99 (0.23)
Ethnicity			
Black	2.55 (0.25)	4.65 (0.35)	6.52 (0.41)
Non-black	3.43 (0.11)	8.13 (0.18)	10.29 (0.19)
Age			
18–29 years	5.99 (0.27)	10.38 (0.33)	14.28 (0.39)
30–44 years	3.86 (0.18)	9.48 (0.31)	11.87 (0.34)
45–64 years	1.80 (0.14)	6.79 (0.30)	8.00 (0.32)
65+ years	0.55 (0.09)	1.50 (0.16)	1.79 (0.17)

Prevalences of major depression decreased with age within each time frame.

The prevalences of DSM–IV alcohol abuse and dependence are summarized in Table 2 by sex, ethnicity and age according to the time of occurrence of the episode. The prevalence of combined abuse and dependence was 18.17% on a lifetime basis, with 7.41% and 14.60% of the respondents experiencing an episode within the previous year and prior to that time, respectively. Slightly more respondents were classified as meeting DSM–IV criteria for dependence rather than abuse, a pattern consistent for all three time frames and among each sociodemographic subgroup of the population. Regardless of time frame examined, the prevalence of abuse and dependence was greater among males than females, greater among non-blacks compared to blacks, and generally decreased with age.

Comorbidity

Table 3 shows the comorbidity rates and associations between alcohol abuse and dependence combined, and separately for abuse and dependence and major depression for the three time frames of interest. The analyses shown in Table 3 assume that major depression represents the exposed group with the odds ratios representing the odds of an alcohol use disorder in the exposed group relative to the odds in the unexposed group or among those with no major depression. Among respondents with an episode of major depression in the past year, 21.36% were additionally classified with an alcohol abuse or dependence diagnosis compared to 6.92% of the respondents with no evi-

Table 2. Prevalence of DSM-IV alcohol abuse and dependence by sex, ethnicity and age: United States 1992

Sociodemographic characteristic	Alcohol abuse only			Alcohol dependence			Alcohol abuse and/or dependence		
	Past year % (S.E.)	Prior to past year % (S.E.)	Lifetime % (S.E.)	Past year % (S.E.)	Prior to past year % (S.E.)	Lifetime % (S.E.)	Past year % (S.E.)	Prior to past year % (S.E.)	Lifetime % (S.E.)
Total	3.03 (0.1)	3.39 (0.11)	4.88 (0.13)	4.38 (0.15)	11.20 (0.20)	13.29 (0.22)	7.41 (0.20)	14.60 (0.20)	18.17 (0.27)
Sex									
Male	4.67 (0.19)	4.76 (0.18)	6.99 (0.22)	6.33 (0.24)	15.80 (0.34)	18.55 (0.36)	11.00 (0.32)	20.56 (0.36)	25.54 (0.40)
Female	2.13 (0.10)	2.13 (0.11)	2.93 (0.13)	2.58 (0.14)	6.97 (0.20)	8.43 (0.23)	4.09 (0.18)	9.10 (0.24)	11.36 (0.28)
Ethnicity									
Black	1.51 (0.22)	1.15 (0.21)	2.19 (0.26)	3.77 (0.32)	6.57 (0.45)	8.57 (0.53)	5.28 (0.39)	8.08 (0.49)	10.76 (0.60)
Non-black	3.22 (0.13)	3.63 (0.12)	5.22 (0.15)	4.54 (0.16)	11.80 (0.22)	13.90 (0.24)	7.68 (0.22)	15.44 (0.25)	19.12 (0.30)
Age									
18–29 years	6.54 (0.33)	3.60 (0.21)	6.69 (0.31)	9.40 (0.38)	15.05 (0.46)	19.88 (0.52)	15.94 (0.53)	18.64 (0.49)	26.57 (0.61)
30–44 years	3.02 (0.16)	4.64 (0.20)	6.17 (0.23)	4.25 (0.21)	13.95 (0.36)	15.68 (0.37)	7.28 (0.26)	18.60 (0.41)	21.85 (0.43)
45–64 years	1.35 (0.16)	3.01 (0.19)	3.70 (0.23)	2.12 (0.17)	8.92 (0.34)	9.95 (0.35)	3.47 (0.22)	11.92 (0.39)	13.66 (0.42)
65+ years	0.25 (0.08)	1.10 (0.14)	1.25 (0.15)	0.39 (0.07)	3.18 (0.22)	3.41 (0.23)	0.65 (0.10)	4.28 (0.25)	4.66 (0.26)

Table 3. Prevalence and odds ratios of DSM-IV alcohol abuse and dependence and DSM-IV major depression by time frame: United States, 1992

Alcohol use disorder	Major depression % (S.E.)	No major depression % (S.E.)	Odds ratio	(95% Confidence limits)
		Past year		
Abuse and/or dependence	21.36 (1.38)	6.92 (0.19)	3.65	(3.12, 4.27)
Abuse only	6.31 (0.74)	2.92 (0.11)	2.24	(1.74, 2.88)
Dependence	15.05 (1.23)	4.01 (0.14)	4.24	(3.51, 5.13)
		Prior to past year		
Abuse and/or dependence	36.31 (0.58)	12.78 (0.22)	3.89	(3.55, 4.27)
Abuse only	6.26 (0.49)	3.15 (0.11)	2.05	(1.71, 2.46)
Dependence	30.05 (0.99)	9.63 (0.19)	4.03	(3.65, 4.45)
		Lifetime		
Abuse and/or dependence	40.03 (0.95)	15.78 (0.26)	3.56	(3.29, 3.86)
Abuse only	7.54 (0.44)	4.59 (0.13)	1.69	(1.49, 1.94)
Dependence	32.49 (0.90)	11.19 (0.20)	3.82	(3.51, 4.16)

dence of major depression during this time period. Regardless of time frame, the increased risk of having an alcohol use disorder among those with major depression was about 4 times (ORs = 3.65–3.89) greater than those without major depression. The association between alcohol dependence and major depression was stronger than the association between alcohol abuse and major depression. The odds of abuse among respondents with major depression was ~ 2 times greater than those experiencing no major depression within each time frame.

Tables 4–6 present comorbidity rates and associated odds ratios of alcohol use disorders among those with and without a co-occurring major depression for each time frame separately for each sex, ethnic and age subgroup of the population. The risk of alcohol abuse and major depression was consistently greater among females and blacks compared to their male and non-black counterparts, respectively. The opposite was true, to a lesser extent, for the association between dependence and major depression. Males and non-blacks demonstrated a greater association between alcohol dependence and major depression than either females or blacks, respectively, particularly within the past year and on a lifetime basis. Although not entirely consistent for abuse only diagnoses, the odds ratios associated with dependence and combined abuse and dependence had a tendency to increase with age most predominantly in terms of lifetime comorbidity.

DISCUSSION

The most striking result from this study was that virtually all the odds ratios were significantly greater than 1.0, demonstrating that the comorbidity of alcohol use disorders and major depression is pervasive in the general population. The results largely confirm, in a general population sample, a number of reports in the literature conducted in clinical samples. Among those with a current or past year alcohol use disorder, 9.61% experienced a major depression, a comorbidity rate significantly greater than the population base rate of current major depression (3.33%). Conversely, 21.36% of the respondents with major depression reported an alcohol use disorder during the past year, a comorbidity rate significantly greater than the population base rate of alcohol abuse and dependence combined (7.41%). These comorbidity rates are consistent with, but lower than, those reported in treatment samples (8.0–53.0%), suggesting that the comorbidity among alcohol use disorders and major depression is related to professional help seeking.

The associations between lifetime depression and lifetime alcohol use disorders were 3.56, 1.69 and 3.82 for alcohol abuse and dependence combined, alcohol abuse only and alcohol dependence, respectively. The corresponding lifetime ratios reported in the ECA (ORs = 1.3, 0.9 and 1.6) were not statistically significant (Regier et al., 1990). Although not strictly comparable, the risks found for comorbid alcohol use disorders and major depression during the past year (OR 3.65) exceeded the corresponding 6-month odds ratios reported from the ECA (OR = 2.7) and NCS (OR = 2.6) (Kessler et al., 1995; Regier et al., 1990). These findings may result from a variety of factors that preclude direct comparisons between surveys, including differences in sampling frame and sample size, diagnostic interview schedules, or diagnostic criteria used

to formulate diagnoses. Alternatively, the AUDADIS representation of alcohol use disorders as syndromes, as opposed to alcohol symptoms occurring over the life course derived from the DIS or UM–CIDI, could, in part, be responsible for the higher comorbidity rates observed in the present study. That is, the AUDADIS measurement of alcohol use disorders as cohesive constellations of symptoms clustering within specified time frames, might be more highly associated with other psychiatric disorders also measured as syndromes, than are operationalization of alcohol use disorders as loose collections of alcohol symptoms accumulating over the life course.

There was little variation in comorbidity rates and odds ratios across the three time frames of interest. This stability is consistent with evidence from clinical studies that alcohol is often used to self-medicate major depression. Specifically, the mood effects of alcohol have been shown to be variable, initially causing euphoria but producing dysphoria particularly with prolonged use among chronic users. The similarity of the odds ratios associated with past year and lifetime diagnoses, and prior to the past year and lifetime diagnoses suggests that self-medication for depression with alcohol may be effective in the short term but that chronic,

Table 4. Past year prevalence and odds ratios of DSM-IV alcohol abuse and dependence and DSM-IV major depression by sex, ethnicity and age: United States, 1992

Alcohol use disorder	Major depression % (S.E.)	No major depression % (S.E.)	Odds ratio (95% Confidence limits)
		Male	
Abuse and/or dependence	34.55 (2.48)	10.33 (0.30)	4.58 (3.69, 5.69)
Abuse only	9.17 (1.42)	4.54 (0.20)	2.12 (1.49, 3.02)
Dependence	25.38 (2.39)	5.79 (0.23)	5.54 (4.31, 7.12)
		Female	
Abuse and/or dependence	12.75 (1.26)	3.74 (0.17)	3.76 (3.01, 4.69)
Abuse only	4.44 (0.76)	1.40 (0.09)	3.28 (2.28, 4.72)
Dependence	8.31 (9.98)	2.34 (0.13)	3.78 (2.91, 4.89)
		Black	
Abuse and/or dependence	12.14 (3.15)	5.10 (0.40)	2.57 (1.41, 4.69)
Abuse only	5.93 (2.50)	1.40 (0.20)	4.45 (1.78, 11.14)
Dependence	6.21 (2.14)	3.71 (0.32)	1.72 (1.01, 3.63)
		Non-black	
Abuse and/or dependence	22.25 (1.47)	7.16 (0.21)	3.71 (3.15, 4.36)
Abuse only	6.34 (0.78)	3.11 (0.12)	2.11 (1.62, 2.74)
Dependence	15.90 (1.32)	4.05 (0.15)	4.48 (3.70, 5.44)
		18–29 years	
Abuse and/or dependence	30.58 (2.23)	15.01 (0.51)	2.49 (2.03, 3.07)
Abuse only	10.52 (1.47)	6.28 (0.33)	1.75 (1.27, 2.41)
Dependence	20.07 (1.93)	8.72 (0.38)	2.63 (2.05, 3.36)
		30–44 years	
Abuse and/or dependence	15.78 (1.83)	6.94 (0.26)	2.51 (1.90, 3.33)
Abuse only	2.90 (0.71)	3.03 (0.16)	1.04 (0.63, 1.72)
Dependence	12.88 (1.74)	3.91 (0.21)	3.63 (2.63, 5.03)
		45–64 years	
Abuse and/or dependence	9.19 (2.02)	3.36 (0.22)	2.91 (1.77, 4.76)
Abuse only	2.74 (1.05)	1.32 (0.15)	2.10 (0.94, 4.70)
Dependence	6.45 (1.76)	2.04 (0.17)	3.31 (1.83, 5.97)
		65+ years	
Abuse and/or dependence	—	—	—
Abuse only	—	—	—
Dependence	—	—	—

Note: Past year prevalences of major depression among 65+ year old respondents was too low for reliable estimation of comorbidity rates.

prolonged self-medication may lead to increased dysphoria and exacerbation of depressive symptoms in the long-term. If self-medication was successful in the long-term, we would have expected the association to be lower in the past year time frame relative to the lifetime and prior to the past year time frames. Alternatively, these findings suggest that having a comorbid diagnosis may impede recovery from either disorder through a mechanism other than self-medication. More conclusive evidence supporting the self-medication or other hypothesis must await the reanalysis of NLAES data from a primary secondary comorbidity perspective, as opposed to the episodic comorbidity perspective presented here.

Although the relationship between alcohol dependence and major depression was greater than the abuse-major depression association at the aggregate level, subgroup variations in the strength of the abuse and dependence relationships with major depression were noted. There was a slight trend for the magnitude of the association between alcohol use disorders and major depression to increase with age for prior to the past year and lifetime diagnoses. These findings may reflect age differences in the lifetime risk of both alcohol use disorders and major depression.

The associations between alcohol abuse and major depression were also consistently greater across time

Table 5. Prior to past year prevalence and odds ratios of DSM-IV alcohol abuse and dependence and DSM-IV major depression by sex, ethnicity and age: United States, 1992

Alcohol use disorder	Major depression % (S.E.)	No major depression % (S.E.)	Odds ratio (95% Confidence limits)
	Male		
Abuse and/or dependence	49.51 (1.73)	18.43 (0.36)	4.34 (3.76, 5.01)
Abuse only	8.17 (0.91)	4.51 (0.18)	1.88 (1.47, 2.42)
Dependence	41.33 (1.63)	13.62 (0.32)	4.36 (3.79, 5.01)
	Female		
Abuse and/or dependence	26.55 (1.10)	7.47 (0.22)	4.48 (3.97, 5.06)
Abuse only	4.84 (0.52)	1.87 (0.11)	2.66 (2.07, 3.42)
Dependence	21.71 (1.08)	5.59 (0.18)	4.68 (4.08, 5.37)
	Black		
Abuse and/or dependence	25.09 (3.16)	7.26 (0.47)	4.27 (3.02, 6.06)
Abuse only	3.88 (1.67)	1.39 (0.21)	2.86 (1.13, 7.25)
Dependence	21.20 (2.97)	5.87 (0.43)	4.32 (3.00, 6.27)
	Non-black		
Abuse and/or dependence	37.14 (1.08)	13.52 (0.24)	3.78 (3.43, 4.16)
Abuse only	6.43 (0.52)	3.39 (0.12)	1.96 (1.63, 2.36)
Dependence	30.70 (1.05)	10.13 (0.20)	3.93 (3.54, 4.36)
	18–29 years		
Abuse and/or dependence	39.89 (1.69)	16.18 (0.48)	3.44 (2.96, 3.99)
Abuse only	5.60 (0.80)	3.36 (0.21)	1.70 (1.23, 2.37)
Dependence	34.00 (1.65)	12.82 (0.46)	3.55 (3.02, 4.17)
	30–44 years		
Abuse and/or dependence	38.36 (1.58)	16.53 (0.40)	3.14 (2.73, 3.62)
Abuse only	7.73 (0.80)	4.32 (0.21)	1.86 (1.46, 2.37)
Dependence	30.62 (1.51)	12.21 (0.34)	3.17 (2.73, 3.69)
	45–64 years		
Abuse and/or dependence	30.50 (1.94)	10.57 (0.39)	3.71 (3.06, 4.51)
Abuse only	5.25 (1.04)	2.84 (0.19)	1.89 (1.23, 2.91)
Dependence	25.05 (1.81)	7.73 (0.33)	4.03 (3.28, 4.96)
	65+ years		
Abuse and/or dependence	12.82 (1.13)	4.15 (0.25)	3.40 (1.94, 5.95)
Abuse only	1.44 (0.86)	1.09 (0.14)	1.32 (0.26, 6.66)
Dependence	11.38 (2.94)	3.06 (0.22)	4.07 (2.26, 7.35)

frames among females and blacks compared to their male and non-black counterparts, respectively. One reason for this observed risk differential may be implicated in the definition of abuse underlying the comorbidity rates. The DSM–IV defines alcohol abuse, separately from dependence, as social, occupational, legal and interpersonal consequences arising from drinking. Indicators of patterns of compulsive drinking (e.g. impaired control over drinking, giving up important activities to drink) and tolerance and withdrawal symptomatology were relegated to the dependence category. Unlike the physiological and compulsive use indicators of DSM–IV dependence, the DSM–IV abuse criteria may be viewed as societal reactions to drinking behavior. As socially subordinate subgroups of the population, female's and black's drinking behavior may be more heavily sanctioned than that of males and non-blacks (Makela, 1987; Park, 1983), thereby increasing their vulnerability to societal reaction as reflected in the DSM–IV formulation of alcohol abuse. The increased risk of major depression among females and blacks diagnosed as alcohol abusers, may therefore reflect the development of major depression among these subgroups of the population as the result of a more adverse societal reaction to their drinking behavior than experienced by either males or non-blacks.

Although the results of the present study have answered basic questions about the descriptive epidemiol-

Table 6. Lifetime prevalence and odds ratios of DSM-IV alcohol abuse and dependence and DSM-IV major depression by sex, ethnicity and age: United States, 1992

Alcohol use disorder	Major depression % (S.E.)	No major depression % (S.E.)	Odds ratio (95% Confidence limits)
	Male		
Abuse and/or dependence	55.21 (1.50)	22.74 (0.40)	4.19 (3.69, 4.76)
Abuse only	9.97 (0.85)	6.71 (0.23)	1.54 (1.27, 1.87)
Dependence	45.24 (1.46)	16.03 (0.34)	4.33 (3.83, 4.89)
	Female		
Abuse and/or dependence	29.02 (1.03)	9.18 (0.26)	4.04 (3.63, 4.51)
Abuse only	5.78 (0.46)	2.58 (0.13)	2.14 (1.92, 2.79)
Dependence	23.25 (0.97)	6.60 (0.21)	4.28 (3.79, 4.84)
	Black		
Abuse and/or dependence	25.54 (2.57)	9.73 (0.60)	3.18 (2.38, 4.25)
Abuse only	4.64 (1.43)	2.02 (0.26)	2.36 (1.20, 4.66)
Dependence	20.89 (2.39)	7.71 (0.53)	3.16 (2.31, 4.32)
	Non-black		
Abuse and/or dependence	41.22 (1.01)	16.59 (0.28)	3.52 (3.24, 3.84)
Abuse only	7.77 (0.47)	4.93 (0.15)	1.62 (1.42, 1.86)
Dependence	33.44 (0.96)	11.66 (0.22)	3.81 (3.40, 1.17)
	18–29 years		
Abuse and/or dependence	45.70 (1.51)	23.38 (0.63)	2.75 (2.42, 3.15)
Abuse only	8.62 (0.79)	6.37 (0.33)	1.39 (1.11, 1.73)
Dependence	37.08 (1.47)	17.01 (0.52)	2.88 (2.50, 3.30)
	30–44 years		
Abuse and/or dependence	40.88 (1.41)	19.29 (0.42)	2.89 (2.56, 3.27)
Abuse only	7.87 (0.69)	5.94 (0.24)	1.35 (1.10, 1.66)
Dependence	33.01 (1.33)	13.35 (0.36)	3.20 (2.81, 3.64)
	45–64 years		
Abuse and/or dependence	32.24 (1.82)	12.04 (0.42)	3.48 (2.91, 4.15)
Abuse only	5.89 (1.00)	3.51 (0.23)	1.72 (1.19, 2.49)
Dependence	26.35 (1.70)	8.53 (0.35)	3.84 (3.18, 4.64)
	65+ years		
Abuse and/or dependence	13.30 (2.86)	4.50 (0.26)	3.25 (2.00, 5.36)
Abuse only	1.20 (0.78)	1.25 (0. 15)	1.05 (0.54, 5.26)
Dependence	12.10 (2.72)	3.25 (0.22)	4.10 (2.43, 6.89)

ogy of episode comorbidity in the general population, future research using the NLAES data will focus on differentiating competing hypotheses or models underlying the comorbidity of alcohol use disorders and major depression. Central to this research was the measurement of age of onset of each disorder that would allow comorbidity analyses from a primary and secondary perspective. The AUDADIS uniquely provides for this requirement particularly in its measurement of age of onset of episodes of alcohol use disorders as opposed to age of onset of the first symptom of abuse or dependence. Despite the need for longitudinal studies of comorbidity that are eminently more suited to elucidate causes, recent advances in multivariate statistical procedures (e.g. survival analysis, structural equation modeling combined with logistic regression) will enable testing of competing causal hypotheses underlying comorbidity with this retrospective data.

The consistent and significant associations between major depression and alcohol use disorder shown in this study provide persuasive evidence that major depression must be addressed as a critical component of alcohol abuse and dependence prevention efforts. Early recognition and appropriate treatment of major depression should be a promising addition to the battery of existing prevention strategies for alcohol abuse and dependence. With regard to the treatment implications, tailoring alcohol programs to individuals with mood (and other psychiatric disorders) and substance use disorders should facilitate meeting the specific management needs of this important subgroup of comorbid individuals. From a scientific perspective, future research on comorbidity should be encouraged and supported with a view that the study of the interrelationships between two or more disorders may help elucidate the causes of both.

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The Relationship Between DSM–IV Alcohol Use Disorders and DSM–IV Major Depression: Examination of the Primary-Secondary Distinction in a General Population Sample

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This study examined the primary-secondary distinction with respect to DSM–IV alcohol use disorders and major depression in a large representative sample of the United States. Primary, secondary and concurrent depressives (i.e. the comorbid groups), respondents classified with major depression only and respondents classified with alcohol use disorders only were compared on a large number of sociodemographic, alcohol-related, depression-related and drug-related variables. Results were at variance with a majority of findings from the treatment literature with regard to familial aggregation of alcohol use disorders and sociodemographic, alcohol and depression profiles. Implications of the results are discussed in terms of the diagnostic and prognostic significance of the primary-secondary distinction.

INTRODUCTION

Recent reviews of the literature have focused on numerous studies that aimed to examine the primary-secondary distinction as it relates alcoholism and major depression (Bronisch, 1990; Davidson and Ritson, 1993; Merikangas and Gelernter, 1990). In these studies, the index cases were psychiatric patients in treatment for major depression and, more commonly, alcoholics in clinical samples. Depending on the selection of index cases in this literature, the obvious heuristic value of the primary-secondary distinction was to identify relatively pure or homogeneous subtypes of major depression or, alternatively, alcoholism for the purpose of improved classification and diagnosis, management and outcome. Each of these studies sought to identify subgroups of patients defined in terms of the primary-secondary distinction that could be differentiated with respect to sociodemographic profiles, phenomenology of alcoholism and/or major depression and the occurrence of other psychopathology. Of particular interest in this literature was the clinical utility of the primary-secondary distinction in predicting one particular independent validator of diagnosis, i.e., familial aggregation of alcoholism and major depression.

There is great variation in the literature in both the definitions of primary and secondary disorder used and the actual subgroups of patients with and without alcoholism and major depression examined. In studies conducted among alcoholics or mixed samples of alcoholics and psychiatric patients, subjects were usually classified with primary depression if the onset of major depression preceded the onset of alcoholism and classified with secondary depression if the onset of alcoholism preceded the onset of major depression. However, there were differences in the alcohol subgroups assessed in these studies. Some of these studies compared primary and secondary depressives to alcoholics with no history of major depression (Cadoret and Winokur, 1974; Hasegawa et al., 1991; O'Sullivan et al., 1983; Roy et al., 1991) or to major depressives with no history of alcoholism (Brown et al., 1995), while others specifically excluded primary depressives (Schuckit, 1983) or secondary depressives (Leibenluft et al., 1993) from their comparison groups. The remainder of the studies compared alcoholics with and without histories of major depression, thereby obscuring potential differences between primary and secondary depression (O'Sullivan et al., 1979; O'Sullivan et al., 1988, Winokur et al., 1971). In those studies conducted among psychiatric samples, patients with diagnoses of major depression with no histories of alcoholism were usually compared with subtypes referred to as major depressives with alcoholism that represented secondary depressives (as defined above) in one study (Woodruff et al., 1973) and primary de-

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pressives in two others (Coryell et al., 1992; Merikangas et al., 1985).

The rates of secondary depression among alcoholics reported in the extant literature also varied widely from 10 to 53% while the rates of primary depression were much less variable, in general, ranging from 2 to 16% (Bronisch, 1990). The variation in rates of primary and secondary depression was largely the result of differences in patient samples, diagnostic criteria and psychiatric assessments used to classify the disorders. Methodological limitations of some of the studies may also have contributed to the observed variation. Some of these studies measured onset of alcoholism and depression as the occurrence of the first symptom of each disorder, which may or may not coincide with the onset of the first episode of the disorder (Brown et al., 1995; Schuckit, 1983), while other research was compromised by extremely small sample sizes (Brown et al., 1995; Leibenluft et al., 1993) or the exclusion of females (Hasegawa et al., 1991; O'Sullivan et al., 1983; Schuckit, 1983).

Perhaps the most serious limitation of all previous studies examining the primary-secondary distinction relative to alcoholism and major depression is that they have been conducted in clinical or treated samples. Studies of primary and secondary depression among alcoholics, or among patients with major depression for that matter, may not be suitable for the study of comorbidity since clinical samples are naturally biased toward the more severely affected population. Comorbidity between alcoholism and major depression may be a function of severity and patients with comorbid disorders may be more likely to enter treatment (Berkson, 1946). However, only 2 previous general population surveys have collected data relevant to the primary-secondary distinction among persons classified with alcohol use disorders: the New Haven Community Survey (Weissman and Meyers, 1978) and the Epidemiologic Catchment Area Study (ECA; Helzer and Przybeck, 1987). No attempts were made to analyze the ECA results from a primary-secondary perspective while the New Haven Community survey did not compare subgroups of alcoholics primarily as the result of the extremely small size of the target comorbid groups ($n = 24$).

The present study overcomes many of the methodological limitations of prior research that has examined the primary-secondary distinction in relation to alcoholism and major depression. In this study, the primary-secondary distinction with regard to the Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition (DSM–IV; American Psychiatric Association, 1994) alcohol use disorders (i.e. abuse and dependence) and major depression was examined in a large representative sample of the USA population. This epidemiological survey also used systematic and reliable diagnostic assessment in which both disorders were assessed as syndromes and respondents were selected without regard to treatment status.

The major purpose of this study was to compare 5 subgroups of respondents with respect to sociodemographic profiles, alcohol and depressive phenomenology, familial aggregation of alcoholism and drug use and drug use disorders. Among these were 3 comorbid subgroups of respondents, defined in terms of the primary-secondary distinction: primary depressives; secondary depressives; and concurrent depressives. The latter category, which has not been examined in the past due to small sample sizes, comprised that comorbid subgroup in which the onset of alcohol use disorder and major depression occurred during the same time period. The remaining 2 subgroups consisted of respondents who were classified with major depression and no history of an alcohol use disorder (MDD only group) and respondents classified with an alcohol use disorder and no history of major depression (AUD only group). For the purposes of this study, primary depressives will refer to comorbid respondents whose onset of major depression predates their onset of alcoholism and secondary depressives will refer to respondents whose onset of alcoholism predates their onset of major depression. The definition of primary depression used in this study is at variance with the definition of the term used in much of the broader psychiatric research literature on the primary-secondary distinction. In that literature, the term primary depression is also commonly used to define patients classified with major depression without histories of alcohol use disorders. In this study, respondents with diagnoses of major depression without histories of alcohol use disorders are referred to as the major depression subgroup to cleanly differentiate them from primary depressives. Of particular interest in this study was whether the results of prior research on the primary-secondary distinction relative to major depression and alcoholism conducted exclusively in treated samples would be confirmed in a general population survey.

METHODS

SAMPLE

This study is based on data collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey

(NLAES), designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism with fieldwork conducted by the U.S. Bureau of the Census (Grant et al., 1994; Massey et al., 1989). Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older residing in the noninstitutionalized population of the contiguous U.S. including DC. The household response rate for this representative sample of the U.S. was 91.9% and the sample person response rate was 97.4%.

The NLAES featured a complex multistage design. Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probability proportional to size. From a sampling frame of ~ 2,000 PSUs, 198 were selected for inclusion in the 1992 NLAES sample, including 52 that were self-representing, i.e. selected with certainty. Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically for sample. Oversampling of the black population was accomplished at this stage of sample selection to secure adequate numbers for analytic purposes. Segments then were divided into clusters of ~ 4–8 housing units and all occupied housing units were included in the NLAES. Within each household, 1 randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18–29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavy substance using population subgroup. This subgroup of young adults was randomly sampled at a ratio of 2.25:1.00.

Diagnostic assessment

Diagnoses of DSM–IV alcohol and drug use disorders were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained interviewers who were not clinicians (Grant and Hasin, 1992). The AUDADIS included an extensive list of symptom questions that operationalized the DSM–IV criteria for substance use disorders and major depression. Drug-specific diagnoses of abuse and dependence were derived separately for alcohol, sedatives, tranquilizers, opioids (other than heroin), amphetamines, cocaine, cannabis (and THC and hashish), heroin, methadone and hallucinogens. A prescription drug use disorder measure was also constructed to represent abuse of and/or dependence on sedatives, tranquilizers, amphetamines and/or opioids.

The AUDADIS diagnoses of substance-specific abuse and dependence satisfied the clustering and duration criteria of the DSM–IV definitions. The criteria of the DSM–IV included the requirement for a clustering of symptoms within any 1-year period, in addition to associating duration qualifiers with certain abuse and dependence criteria. The duration qualifiers are defined as the repetitiveness with which symptoms must occur to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often," and "persistent" appearing in the description of the diagnostic criteria. Not only were the clustering criteria represented in past year AUDADIS diagnoses of abuse and dependence, but the corresponding past diagnoses (before the past year) were also measured as syndromes, or the clustering of the required number of symptoms necessary to achieve a diagnosis: (1) at the same time; (2) continuously for at least 1 month; or (3) repeatedly for at least 1 month. For the purposes of the present study, respondents were classified with a lifetime substance use disorder if they had experienced an episode of abuse or dependence in the past year and/or before the past year.

Episodes of DSM–IV major depressive disorder were also derived for the past year, before the past year and lifetime. Consistent with the DSM–IV, the AUDADIS diagnoses of major depression required the presence of at least 5 depressive symptoms (inclusive of depressed mood or loss of pleasure and interest) nearly every day, most of the day for at least a 2-week period. Social and/or occupational dysfunction must also have been present during the disturbance. Episodes of major depression exclusively due to bereavement or physical illness were ruled out. Other more chronic mood disorders (e.g. dysthymia) were also measured in this survey and will be the subject of another separate study.

Reliabilities of past year and before the past year alcohol use disorders, drug use disorders and major depression were 0.76 and 0.73, 0.66 and 0.91, and 0.60 and 0.65, respectively, as determined from an independent test–retest study conducted in a general population sample (Grant et al., 1995).

Age of onset for DSM–IV substance use disorders and major depression was defined as the respondent's age when the first episode of each of these disorders began, since episodes of either disorder may persist for > 1 year. The respondents in this study were all those with a lifetime diagnosis of an alcohol use disorder or major depression. Those respondents with a lifetime diagnosis of major depression without a history of an alcohol use disorder constituted the major depressive disorder (MDD) only group while respondents with a

lifetime diagnosis of an alcohol use disorder (i.e. abuse and/or dependence) without a history of major depression constituted the alcohol use disorder (AUD) only group. Among the 3 comorbid groups, respondents with an age of onset of depression preceding an alcohol use disorder were classified as primary depressives, respondents with an age of onset of an alcohol use disorder preceding major depression were classified as secondary depressives while respondents who reported the same age of onset for both an alcohol use disorder and major depression were classified as concurrent depressive.

Alcohol-related measures

All relevant groups were compared in terms of age of onset of an alcohol use disorder, age at first drink and the severity of the alcohol use disorder as measured by the number of lifetime DSM–IV symptom items that operationalized the 7 DSM–IV dependence criteria and 4 DSM–IV abuse criteria (range 1–31) and the longest duration of an episode of alcohol abuse and dependence.

Groups were also compared with respect to their average daily ethanol intake during the period of heaviest drinking during their lives. Average daily ethanol intake for beer, wine and liquor was based on patterns of usual and heaviest consumption during the period when respondents drank the most in their lives. The measures obtained for each type of beverage included frequency of drinking (converted to number of drinking days/year), typical number of drinks consumed/drinking day and typical size of drink (ounces of beer, wine or liquor). Ounces of beverage were converted to ounces of ethanol using the following conversion factors: 0.045 for beer, 0.121 for wine and 0.409 for liquor (Distilled Spirits Council of the United States, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams et al., 1993). To estimate average daily ethanol intake based on usual and heaviest consumption during this period, the volume of ethanol for each beverage type was calculated as follows: oz. $ethanol_{beverage}$ = (total minus heavy drinking $days/year_{beverage}$ x number of drinks/usual drinking $day_{beverage}$ x ounces of beverage in a typical drink consumed on usual drinking $day_{beverage}$ x ethanol conversion $factor_{beverage}$) + (heavy drinking $days/year_{beverage}$ x number of drinks/heavy drinking $day_{beverage}$ x ounces of beverage in a typical drink consumed on heavy drinking $day_{beverage}$ x ethanol conversion $factor_{beverage}$). These volumes were then summed over the 3 beverage types and the resulting volume was divided by 365.

Biological first- and second-degree relatives reported to have been an alcoholic or problem drinker constituted the family history-positive measure examined in this study. Family history of alcoholism was separately determined for each biological first- and second-degree relative. An alcoholic or problem drinker was specifically defined for respondents as a person who had physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving, or a person who seems to spend a lot of time drinking or being hung over. Relatives classified as family history positive in this study had at least 1 of these alcohol problems, a cutoff identical with the one used in the Family History-Research Diagnostic Criteria (FHRDC; Andreasen et al., 1977).

Respondents in this survey were asked if they had ever gone anywhere or seen anyone for problems related to their drinking. Respondents were specifically instructed to include any help they had received for their drinking, including help for combined alcohol and drug use if alcohol was the major problem for which they sought help. Respondents were also asked to indicate whether they ever sought help from 23 specific types of treatment sources, including inpatient alcohol and/or drug rehabilitation programs and inpatient wards of general or psychiatric hospitals, outpatient clinics and alcohol and/or drug detoxification units and 12-step groups (e.g. Alcoholics Anonymous, Narcotics or Cocaine Anonymous or Alanon). Respondents receiving help from any of these sources on a lifetime basis constituted the alcohol treatment-positive measure examined in this study.

Depression-related measures

The 4 diagnostic groups of interest that included respondents with a history of major depression in this study were compared in terms of the onset of major depression and the percentage of respondents classified as positive for each depression criterion during their worst episode of depression. Severity was represented in 3 ways: as the number of episodes of major depression; the longest duration of an episode of depression; and the mean number of depressive symptom items (range 1–19) during the respondent's worst episode of depression. In the AUDADIS, there were 19 symptom items that operationalized the DSM–IV major depression criteria. Respondents were classified as receiving treatment for a major depressive disorder if they had ever seen a doctor or other health professional to improve their mood or to feel better during an episode, were prescribed medication or were a patient in a hospital for at least 1 night because of their depression.

DRUG-RELATED MEASURES

All 5 groups were also compared with respect to lifetime histories of any drug use or drug use disorder separately for prescription drugs, sedatives, tranquilizers, stimulants, cannabis, cocaine and hallucinogens. Lifetime drug use was defined for this survey as the use of any drug at least 12 x on one's own, i.e., either without a doctor's prescription, in greater amounts, more often, or longer than prescribed, or for a reason other than the one for which the drug was prescribed. Data are not presented in this study separately for opiates, heroin and methadone because of the low prevalences of use, abuse and dependence for these drugs in this general population sample. The ever drug treatment measure was constructed exactly as defined for the alcohol treatment measure.

STATISTICAL ANALYSES

To take into account the NLAES sample design, all SE values were generated and statistical tests conducted using SUDAAN (Research Triangle Institute, 1994), a software program that uses Taylor series linearization to adjust for sample design characteristics in complex sample surveys. Overall X^2 and F statistics were computed for categorical and continuous measures, respectively. If the overall X^2 or F statistic was significant (< 0.05), separate pair-wise comparisons were conducted between the groups using partitioned X^2 analysis or t tests, respectively. To protect against type I error, due to multiple comparisons, any pairwise comparison needed to yield a test statistic value that could be declared statistically significant at the level of < 0.001.

RESULTS

Of the total population, 24.3% (unweighted $n = 9{,}985$) had a lifetime history of an alcohol use disorder and/or major depression. Among this subgroup of 9,985 respondents, 24.6% were classified with MDD only, 59.2% were classified with AUD only and 6.6, 2.7 and 6.9% (a total of 16.2%) were classified with primary, concurrent and secondary depression, respectively. Among the 16.2% of the respondents in the 3 comorbid groups, 41.0, 16.5 and 42.5% were classified with primary, concurrent and secondary depression.

SOCIODEMOGRAPHIC MEASURES

The MDD only group had significantly more females (68.6%) than the other 4 groups (29.9–47.0%) while the AUD only group was composed of significantly more males (70.1%) than the remaining 4 groups (31.4–64.3%; Table 1). Compared with the MDD

Table 1. Sociodemographic characteristics of respondents with DSM–IV major depression only, DSM–IV alcohol use disorders only, and primary, concurrent and secondary depression

Sociodemographic characteristic [a]	Major depression only (1)	Alcohol use disorder only (2)	Primary depression (3)	Concurrent depression (4)	Secondary depression (5)	Significant comparisons (χ^2 or t, $P < 0.001$)
% Male	31.4 (1.11)	70.1 (0.71)	53.0 (2.02)	53.8 (3.34)	64.3 (2.02)	1 vs. (2) (3) (4) (5); 2 vs. (3) (4) (5); 3 vs. (5)
% Black	9.4 (0.67)	7.3 (0.50)	5.1 (0.89)	4.8 (1.43)	4.5 (0.90)	1 vs. (3) (4) (5);
Current age in years ($\bar{x}$)	37.6 (0.32)	36.9 (0.26)	31.9 (0.46)	34.3 (0.79)	35.4 (0.47)	1 vs. (3) (4) (5); 3 vs. (5); 2 vs. (3) (4) (5)
% Married or living with someone as if married	54.9 (1.17)	60.2 (0.89)	42.6 (2.09)	50.6 (3.36)	54.4 (2.28)	3 vs. (1) (2) (5); 1 vs. (2)
% With less than high school education	12.4 (0.81)	14.9 (0.63)	14.8 (1.72)	14.9 (2.26)	12.2 (1.39)	
Socioeconomic Status (SES) Score ($\bar{x}$)	51.4 (0.65)	52.4 (0.41)	47.6 (1.09)	47.2 (1.7)	53.2 (1.14)	3 vs. (2) (5)

SE values appear in parentheses.

[a] Males vs. females; black vs. nonblack; married or living with someone as if married vs. separated/divorced/widowed; less than high school education vs. high school education and beyond; SES scores based on 1990 Nam-Powers-Terrie occupational status scores (Terrie and Nam, 1994).

Table 2. Alcohol-related characteristics of respondents with DSM–IV major depression only, DSM–IV alcohol use disorders only, and primary, concurrent and secondary depression

Alcohol-related characteristic	Major depression only (1)	Alcohol use disorder only (2)	Primary depression (3)	Concurrent depression (4)	Secondary depression (5)	Significant comparisons (χ^2 or t, $P < 0.001$)
Average daily ethanol intake during heaviest drinking period in ounces ($\bar{x}$)	1.0 (0.08)	3.7 (0.11)	3.8 (0.25)	5.0 (0.51)	4.8 (0.3)	1 vs. (2) (3) (4) (5)
Age at first drink ($\bar{x}$)	18.8 (0.13)	17.4 (0.05)	16.8 (0.18)	16.9 (0.22)	16.7 (0.14)	1 vs. (2) (3) (4) (5)
Onset of alcohol use disorder ($\bar{x}$)	N/A	23.1 (0.14)	23.0 (0.31)	21.9 (0.43)	19.9 (0.23)	5 vs. (2) (3) (4)
Number of lifetime alcohol symptoms ($\bar{x}$)	N/A	9.8 (0.08)	12.5 (0.27)	13.5 (0.47)	13.2 (0.29)	2 vs. (3) (4) (5)
Duration of longest episode of alcohol abuse in months ($\bar{x}$)	N/A	47.9 (1.47)	32.1 (2.25)	39.3 (4.13)	43.7 (3.06)	3 vs. (2) (5)
Duration of longest episode of alcohol dependence in months ($\bar{x}$)	N/A	37.8 (1.27)	24.7 (1.64)	32.5 (3.67)	37.3 (2.84)	3 vs. (2) (5)
% Lifetime alcohol impatient treatment	N/A	8.2 (0.41)	12.4 (1.58)	18.5 (2.53)	18.1 (1.65)	2 vs. (3) (4) (5)
% Lifetime alcohol outpatient treatment	N/A	10.8 (0.47)	19.6 (1.81)	19.5 (2.59)	22.2 (1.75)	2 vs. (3) (4) (5)
% Lifetime alcohol 12-step program	N/A	12.5 (0.53)	20.4 (1.96)	22.9 (2.79)	24.8 (1.81)	2 vs. (3) (4) (5)
% Biological mother alcoholic	8.3 (0.60)	10.3 (0.50)	19.2 (1.86)	17.8 (2.39)	14.8 (1.49)	All family history variables
% Biological father alcoholic	27.7 (1.03)	30.5 (0.70)	42.4 (2.11)	38.3 (3.28)	39.2 (2.01)	
% Either biological parent alcoholic	32.0 (1.08)	34.5 (0.74)	49.2 (2.09)	44.7 (3.16)	45.9 (2.05)	1 vs. (3) (4) (5); 2 vs. (3) (4) (5)
% Any first-degree relative alcoholic	47.8 (1.13)	48.4 (0.87)	64.3 (2.09)	61.3 (3.44)	61.7 (2.24)	
% Any first- or second-degree relative alcoholic	71.7 (1.01)	70.4 (0.76)	83.4 (1.51)	86.2 (2.23)	81.6 (1.72)	

SE values appear in parentheses.

only group, the 3 comorbid groups were younger and less likely to be black. They did not differ with respect to education, socioeconomic status or marital status, with the exception of primary depressives who were less likely to be married. The 3 comorbid groups also did not differ with respect to sex, ethnicity or education, but were significantly younger than the AUD only group. Although the concurrent and secondary depressives did not differ from the AUD only group with respect to socioeconomic and marital status, primary depressives were significantly less likely to be married and were of lower socioeconomic status than respondents in the AUD only group. Among the 3 comorbid groups, there were marked differences between primary and secondary depressives: primary depressives were more likely to be female, unmarried, younger and of lower socioeconomic status than secondary depressives.

Alcohol-related measures

One of the most striking findings in Table 2 was that the 3 comorbid groups were not shown to have any less severe alcohol use disorders than the AUD only group. The respondents in all 3 comorbid groups did not differ from the AUD only group with respect to average daily ethanol intake during their heaviest lifetime drinking period, but were more likely to have entered treatment for an alcohol problem and reported more severe alcohol use disorders, in terms of the number of lifetime alcohol symptoms than the AUD only group. The duration of the longest episode of alcohol abuse and dependence and the age of first drink did not significantly differ between concurrent and secondary depressives and the AUD only group, but primary depressives reported shorter durations of their alcohol use disorders than did secondary depressives or respondents in the AUD only group. Secondary depressives had a significantly earlier onset of an alcohol use disorder (20 years) compared with the 3 remaining groups (22–23 years).

With regard to a family history of alcoholism, the MDD only and AUD only groups were consistently less likely than respondents in all 3 comorbid groups to have a biological parent, first-degree relative or a first- and/or second-degree relative who was an alcoholic. Interestingly, when the MDD only group was compared with a normal control group, i.e., those respondents in this study who did not have histories of either major depression or alcohol use disorders (n = 32,859), the familial aggregation of alcoholism among the MDD only probands was significantly greater than among the normal controls (Table 3).

Depression-related measures

As shown in Table 4, the 3 comorbid groups were not shown to experience any less severe major depressions than the MDD only group. All 3 comorbid groups did not differ from the MDD only group with respect to the duration of their longest episode of major depression nor in the number of depressive symptoms reported during their worst episode. Concurrent and secondary depressives also did not differ from the MDD only group in terms of the number of lifetime episodes of depression, but were less likely to have received treatment for a major depressive episode. Unlike concurrent and secondary depressives, primary depressives experienced a greater number of episodes of depression and were equally likely to have received treatment for a major depression relative to the MDD only group. Primary depressives also reported more serious depressive episodes compared with the concurrent and secondary depressives in terms of a greater number of lifetime depressive episodes and an increased likelihood to receive treatment for a depressive episode.

The mean age of onset of major depression differed among the 4 subgroups of interest. Primary depressives experienced their first episode of depression at the earliest age (17 years) followed by concurrent depressives (22 years), respondents in the MDD only

Table 3. Family history of alcoholism among respondents with major depression only and respondents without major depression or alcohol use disorder

Family history variable	Major depression only (1)	No major depression no alcohol use disorder (2)	Significant comparison (χ^2 or t, $P < 0.001$)
% Biological mother alcoholic	8.3 (0.60)	3.8 (0.13)	All family history variables (1) vs. (2)
% Biological father alcoholic	27.7 (1.03)	15.6 (0.25)	
% Either biological parent alcoholic	32.0 (1.08)	17.5 (0.27)	
% Any first-degree relative alcoholic	47.8 (1.13)	29.1 (0.33)	
% Any first- or second-degree relative alcoholic	71.7 (1.07)	46.6 (0.36)	

SE values appear in parentheses.

Table 4. Depression-related characteristics of respondents with DSM–IV major depression only, DSM–IV alcohol use disorders only, and primary, concurrent, and secondary depression

Depression-related characteristic	Major depression only (1)	Alcohol use disorder only (2)	Primary depression (3)	Concurrent depression (4)	Secondary depression (5)	Significant comparisons (χ^2 or t, $P < 0.001$)
Onset of major depression ($\bar{x}$)	24.9 (0.26)	N/A	16.7 (0.25)	21.9 (0.43)	26.6 (0.38)	1 vs. (3) (4) (5); 3 vs. (4) (5); 4 vs. (5)
Number of episodes of major depression ($\bar{x}$)	5.1 (0.23)	N/A	9.4 (0.60)	7.4 (0.90)	4.3 (0.24)	3 vs. (1) (5); 4 vs. (5)
Longest duration of episode of major depression in months (x)	9.1 (0.62)	N/A	12.3 (1.38)	12.5 (2.18)	6.6 (0.65)	5 vs. (3) (4)
% Ever treated for major depression	50.5 (1.19)	N/A	50.9 (2.28)	40.9 (3.42)	42.5 (2.16)	1 vs. (4) (5); 3 vs. (4) (5)
Number of depressive symptoms during worst episode ($\bar{x}$)	10.2 (0.07)	N/A	11.4 (0.13)	11.2 (0.22)	10.7 (0.14)	1 vs. (3) (4) (5)
% Positive for major depressive diagnostic criteria during worst episode of depression						
Depressed mood	95.2 (0.76)	N/A	96.1 (0.80)	95.1 (1.31)	95.0 (1.02)	
Diminished interest/pleasure	88.9 (0.71)	N/A	90.8 (1.31)	86.8 (2.38)	90.0 (1.61)	
Insomnia/hypersomnia	85.4 (0.99)	N/A	88.3 (1.33)	87.1 (2.30)	86.4 (1.56)	
Pschomotor agitation/retardation	67.0 (1.16)	N/A	69.4 (2.04)	72.9 (3.36)	70.1 (2.12)	
Change in appetite/weight	72.1 (1.01)	N/A	77.6 (1.95)	74.4 (2.99)	76.6 (1.78)	
Fatigue	78.5 (0.94)	N/A	81.8 (1.59)	74.4 (2.81)	72.2 (2.09)	5 vs. (1) (3)
Worthlessness/guilt	83.0 (0.82)	N/A	90.1 (1.26)	86.9 (2.31)	86.4 (1.51)	
Difficulty concentration/making decisions	90.1 (0.72)	N/A	91.7 (1.62)	93.3 (1.74)	92.4 (1.16)	
Suicide ideation/plans/attempts	65.8 (1.18)	N/A	79.4 (1.75)	75.3 (2.80)	71.1 (2.04)	3 vs. (1) (5)

SE values appear in parentheses.

group (25 years) and secondary depressives (27 years). As can be seen in Table 4, the majority of respondents in each diagnostic group (between 65.0 and 95.0%) reported experiencing most of the symptoms associated with major depression criteria during their worst lifetime episode. Although there were few differences among the respondent groups' experiences with depressive symptomatology, secondary depressives reported fatigue significantly less often than either the primary depression or the MDD only groups. Importantly, suicide thoughts, ideation and/or attempts were reported more often among primary depressives (79.4%) than among respondents in the MDD only group (65.8%) or among secondary depressives (71.1%).

Drug-related measures

As can be seen in Table 5, ~ 40.0% of each of the comorbid groups had a history of a drug use disorder, a rate significantly higher than that experienced among respondents in the MDD only group (7.5%) and the AUD only group (18.8%). The rate of lifetime drug use disorders was also greater in the AUD only group (18.8%) compared with the MDD only group (7.5%). Although the mean age of onset for a drug use disorder did not differ among the 5 groups, respondents in the MDD only group were older (19 years) when they first used drugs on their own than respondents in the AUD only, primary depression and secondary depression groups (~ 17 years). Primary depressives were also significantly younger than the concurrent depressives with regard to age at first drug use. As gauged by the mean number of lifetime drug symptoms, the severity of drug use disorders was significantly less among the MDD only and AUD only groups (11 symptoms) compared with the 3 comorbid groups (~ 14 symptoms). Respondents in the 3 comorbid group were significantly more likely to have received treatment for a drug use disorder than either the MDD only group or the AUD only group.

Overall, the rates of drug use and drug use disorders within each of the 5 groups were remarkably high. The prevalences of any lifetime drug use and any drug use disorder were ~ 40.0 and 60.0% among the 3 comorbid groups, respectively. Regardless of the specific drug used or specific drug use disorder, a consistent pattern arose. Significantly more respondents in all 3 comorbid groups used drugs on their own or developed a drug use disorder compared with those in the MDD only and AUD only groups. In all comparisons, the MDD only group was significantly less likely to use drugs or to develop a drug use disorder than respondents in the AUD only group.

DISCUSSION

Among the comorbid groups in this general population sample, the rates of primary, concurrent and secondary depression were 41.0, 16.5 and 42.5%, respectively. These rates are at variance with most of the earlier treatment literature in this area in which the rates of secondary depression were usually much greater than the rates of primary depression (Cadoret and Winokur, 1974; Robins et al., 1977; Tyndel, 1974; Woodruff et al., 1973). This discrepancy is most likely due to the fact that most of the previous research was conducted among alcoholics in treatment. Other biases associated with the ascertainment of clinical samples may also be implicated in accounting for this discrepancy.

A consistent finding in prior studies is that alcoholic patients with secondary depression appear more similar to alcoholic patients without depression than to depressed patients without alcoholism when compared with respect to a large number of sociodemographic, alcohol-related and major depression measures (Cadoret and Winokur, 1974; Merikangas and Gelernter, 1990; Woodruff et al., 1973). The present study did not support this finding. Respondents in the secondary depression group did not resemble those in the AUD only group any more then they resembled respondents in the MDD only group. Secondary depressives could not be differentiated from the AUD only or MDD only groups with respect to ethnicity, marital status, education or socioeconomic status. Secondary depressives were also less likely to be male and younger than those in the AUD only group and more likely to be male, less likely to be black and younger than the MDD only group. When compared on the basis of background characteristics, primary depressives bore even less resemblance to the AUD only and MDD only groups, i.e., they were younger and less likely to be male or married. Primary depressives were also less likely to be black than the MDD only group and were of lower socioeconomic status compared with the AUD only group. The sociodemographic profile of concurrent depressives was very similar to that of the primary depressives, with the exception that they could not be distinguished from the MDD only group in terms of marital status or from the AUD only group in terms of marital and socioeconomic status.

All 3 comorbid groups reported slightly more severe alcohol use disorders and major depressive episodes than did the AUD only and MDD only groups, respectively. Secondary and concurrent depressives could not be differentiated from the MDD only group in terms of most features of depression, but did report

Table 5. Drug-related characteristics of respondents with DSM–IV major depression only, DSM–IV alcohol use disorders only, and primary, concurrent, and secondary depression

Drug-related characteristic	Major depression only (1)	Alcohol use disorder only (2)	Primary depression (3)	Concurrent Depression (4)	Secondary depression (5)	Significant comparisons (χ^2 or t, $P < 0.001$)
% Lifetime drug use disorder	7.5 (0.59)	18.8 (0.66)	41.6 (2.14)	39.4 (3.30)	36.3 (2.10)	1 vs. (2) (3) (4) (5); 2 vs. (3) (4) (5)
Age at first drug use ($\bar{x}$)	19.0 (0.36)	17.4 (0.11)	16.4 (0.24)	17.5 (0.64)	17.3 (0.30)	1 vs. (2) (3) (5); 2 vs. (3)
Onset of drug use disorder ($\bar{x}$)	20.4 (0.60)	19.7 (0.18)	19.0 (0.38)	19.8 (0.53)	19.5 (0.42)	
Number of lifetime drug symptoms ($\bar{x}$)	11.6 (0.59)	11.0 (0.24)	14.0 (0.54)	14.8 (0.70)	14.6 (0.58)	1 vs. (3) (4) (5); 2 vs. (3) (4) (5)
% Lifetime use on own						
Any drug	20.4 (0.89)	39.7 (0.78)	58.9 (2.06)	57.5 (3.25)	57.6 (2.11)	All drug use, drug use disorder, and drug treatment variables
Prescription drugs	9.0 (0.68)	17.3 (0.63)	32 2 (2.12)	33.3 (3.01)	31.6 (1.88)	
Sedatives	2.2 (0.38)	6.8 (0.42)	13.9 (1.44)	16.7 (2.40)	11.4 (1.38)	1 vs. (2) (3) (4) (5); 2 vs. (3) (4) (5)
Tranquilizers	4.0 (0.49)	7.0 (0.43)	16.0 (1.77)	16.3 (2.40)	14.6 (1.54)	
Amphetamines	4.9 (0.52)	12.8 (0.56)	25.3 (1.85)	26.2 (3.08)	24.5 (1.84)	
Cannabis	16.3 (0.80)	36.7 (0.78)	55.2 (2.06)	52.9 (3.30)	51.5 (2.16)	
Cocaine	3.8 (0.44)	12.2 (0.52)	18.8 (1.57)	20.0 (2.63)	21.1 (1.77)	
Hallucinogens	2.4 (0.38)	6.7 (0.40)	14.8 (1.54)	13.7 (2.25)	13.6 (1.61)	
% Lifetime drug use disorder						
Any drug	7.5 (0.59)	18.8 (0.66)	41.6 (2.14)	39.4 (3.30)	36.3 (2.10)	
Prescription drugs	2.5 (0.35)	5.9 (0.38)	17.1 (1.63)	17.4 (2.44)	15.3 (1.58)	
Sedatives	0.5 (0.16)	2.0 (0.22)	6.3 (1.02)	5.4 (1.43)	5.0 (1.00)	
Tranquilizers	0.5 (0.15)	2.0 (0.23)	6.8 (1.09)	6.8 (1.09)	6.6 (1.60)	
Amphetamines	1.5 (0.29)	4.4 (0.33)	12.2 (1.35)	13.6 (2.20)	12.4 (1.42)	
Cannabis	4.9 (0.50)	15.0 (0.60)	32.7 (2.07)	32.1 (3.05)	25.8 (1.97)	
Cocaine	1.8 (0.33)	5.7 (0.37)	10.9 (1.23)	12.2 (2.10)	12.4 (1.45)	
Hallucinogens	0.4 (0.12)	2.1 (0.22)	6.1 (1.01)	5.5 (1.53)	4.7 (1.06)	
% Lifetime drug inpatient treatment	1.0 (0.29)	2.2 (0.23)	6.7 (1.15)	5.8 (1.47)	6.4 (1.05)	
% Lifetime drug outpatient treatment	1.2 (0.29)	2.8 (0.30)	7.9 (1.13)	7.8 (1.73)	8.6 (1.27)	
% Lifetime drug 12-step program	0.7 (0.29)	2.4 (0.23)	8.7 (1.13)	7.3 (1.71)	7.4 (1.14)	

SE values appear in parentheses.

slightly more depressive symptoms during their worst episode. Similarly, secondary and concurrent depressives did not significantly differ from the AUD only group with respect to most alcohol-related features. Both secondary and concurrent depressives reported more lifetime alcohol abuse and dependence symptoms than the AUD only group.

One of the most significant findings from this study was that primary depressives were identified as having more serious episodes of major depression than any other group, even the MDD only group. Respondents in the primary depression group reported a greater number of episodes of depression, a greater number of depressive symptoms and were significantly more likely than the MDD only or other 2 comorbid groups to report suicide thinking, ideation or attempts during their worst episode of depression. Primary depressives resembled the secondary and concurrent depressives with regard to their alcohol profiles, but experienced shorter durations of abuse or dependence episodes compared with the AUD only group.

The drug-related profiles were extremely different when the 3 comorbid groups were compared with the AUD only and MDD only groups. Although the 3 comorbid groups did not differ in age of onset of a drug use disorder from the MDD only or AUD only groups, they were consistently and significantly more likely to have used prescription drugs, sedatives, tranquilizers, amphetamines, cannabis, cocaine and hallucinogens or developed the associated drug-specific use disorder (i.e., abuse and/or dependence) on a lifetime basis than either the MDD only or AUD only groups.

Although findings of past research regarding increased familial aggregation of alcoholism among probands with alcoholism and major depression are mixed, 2 general conclusions can be gleaned from the literature. In studies of the risk of alcoholism among probands with depression, primary depressives (Coryell et al., 1992; Merikangas et al., 1985) and secondary depressives (Spring and Rothgery, 1984; Stancer et al., 1984; Woodruff et al., 1973) had significantly more alcoholic relatives compared with the MDD only group, and there were no differences in the percentage of alcoholic relatives between the MDD only and normal control groups (Gershon et al., 1982; Merikangas et al., 1985; Weissman et al., 1984). Among alcoholic probands, no significant differences were found in family histories of alcoholism between the AUD only group and primary depressives (Leibenluft et al., 1993), secondary depressives (Cadoret and Winokur, 1974; Schuckit, 1983) and primary and secondary depressives (Hasegawa et al., 1991; O'Sullivan et al., 1979). Finally, patients in the AUD only group had significantly more alcoholic relatives than the MDD only group (Cloninger et al., 1979).

In marked contrast to the results of previous studies conducted in alcoholic samples, all 3 comorbid groups had significantly more relatives with alcoholism than the AUD only group, and no significant differences were found in the percentage of alcoholic relatives in the AUD only and MDD only groups. However, our findings were consistent with the depression literature in that primary and secondary depressives were significantly more likely to have alcoholic relatives compared with the MDD only group. In fact, the high rates observed among the comorbid groups compared with the AUD only group further suggest that alcoholism is highly familial whether or not it complicates major depression. These findings also weigh against assertions that depression results in alcoholism with any regularity since we did find that alcoholism that develops after major depression (i.e., among primary depressives) was highly familial.

Contrary to the results of previous research, the MDD only group was significantly more likely to have alcoholic relatives compared with normal controls or that subset of the NLAES sample classified without major depression or alcohol use disorders. This result strongly suggests that alcohol use disorders and major depression could be alternate manifestations of the same underlying disorder, as has been proposed by researchers in the past (Winokur and Coryell, 1991; Winokur et al., 1971). In any case, this result does not support the view that the transmissions of these 2 disorders are independent or we would not have observed elevated rates of alcoholism in the relatives of the MDD only group compared with the normal controls. Generally, the rates of alcoholism among relatives of respondents in the normal control group were 35% less than the rates observed among MDD only group. At the very least, this study confirms that familial factors are important. However, depression among alcoholics is likely multifactorial, and whether families share common genes or common environmental, biological or behavioral risk factors is yet to be determined.

The onsets of an alcohol use disorder and major depression served to differentiate the 3 comorbid groups. The much earlier onset of major depression among concurrent, and particularly primary depressives, may account for the greater severity of their depressive disorders relative to the MDD only group. Further, the onset of depression among primary and concurrent depressives did not appear to impact on the timing of the onset of an alcohol use disorder, but the onset of alcohol use disorders among secondary depressives did

seem to delay the onset of a major depressive disorder. These findings identify secondary depressives as possible successful self-medicators, i.e. secondary depressives may be successful in self-medicating depressive symptomatology in the short-term, thereby delaying the onset of a major depressive disorder. This finding is consistent with evidence from clinical studies that alcohol is often used to self-medicate depressive symptoms. Specifically, the mood effects of alcohol have been shown to be variable, initially causing euphoria in the short-term, but producing dysphoria particularly with prolonged use among chronic users (Mello and Mendelson, 1978).

Secondary and concurrent depressives were identified as having slightly more severe major depressions compared with the MDD only group and slightly more severe alcohol use disorders compared with the AUD only group. Of all the comparison groups in this study, primary depressives were identified as having the most severe episodes of major depression and experienced alcohol use disorders that were also slightly more severe than the AUD only group. This finding was consistent with the observation that primary depressives received more treatment for depression compared with the other 2 comorbid groups and no less treatment than the MDD only group. Similar to secondary and concurrent depressives, primary depressives demonstrated more drug use, drug use disorders and received more drug treatment than either the AUD only and MDD only groups. Primary depressives were further compromised by the greater prevalence of suicidal ideation, thinking and attempts during their worst episode of depression, a prevalence that exceeded that of the secondary depression and MDD only groups.

The findings from the present study suggest that the chronology-based primary-secondary distinction has diagnostic and prognostic significance among patients diagnosed with both major depression and an alcohol use disorder. The serious affective disturbance characteristic of the 3 comorbid groups strongly suggests that clinicians evaluating patients presenting with either major depression or an alcohol use disorder should determine the presence of the comorbid disorder and, importantly, the temporal chronology of the disorders if comorbidity is established. Once comorbidity is determined, whether it be primary, concurrent or secondary, careful screening for drug use and drug use disorders is imperative. If a primary depression is identified, particular vigilance should also be directed toward evaluating the patient's risk for suicide. Even in the absence of a risk of suicide, patients in the 3 comorbid groups would be candidates for admission, appropriate observation and repeated diagnostic evaluation to assess the course of depressive symptomatology.

No previous study has identified the serious affective disturbances combined with higher risks of suicidal thinking, ideation and attempts, drug use and drug use disorders that characterize primary depressives. Several reasons exist for this. Very few studies have identified and classified their comorbid groups in terms of primary depression as we have defined it. Findings from this study also implicate help-seeking patterns among primary depressives as contributing to past difficulty in identifying this important subgroup. Although primary depressives were more likely to seek treatment for an alcohol use disorder than respondents in the AUD only group, they were just as likely to seek treatment for depression as the MDD only group. ~ 50.0% of the primary depressives sought help for their depression while only 26.4% sought help for their alcohol use disorders. This result suggests that alcohol use disorders may often go unrecognized when patients are assessed in traditional psychiatric and medical settings. Even when an alcohol use disorder diagnosis is made, referral for specialized treatment may not always occur. Often, in these settings, the explicit assumption is made that the alcohol use disorder is an unsuccessful attempt to self-medicate the depression and that successful treatment of depression will result in the amelioration of the alcohol use disorder.

Although this study was useful in identifying important subgroups of alcohol use disorders and establishing the clinical relevance of the primary-secondary distinction, much more research is needed to confirm the results of this study. Future research should focus on the development of a valid classification system that considers comorbidity as the basis of subtypes for all forms of psychopathology, including alcohol use disorders and major depression. Researchers should continue to examine familial aggregation within the primary-secondary paradigm with more rigorous family study designs than presented here. Although the assessment of comorbid disorders, particularly the onset of these disorders, was more precise in this study than in previous research, there is a critical need for longitudinal research on the primary-secondary distinction that would be less subject to recall bias. Since adolescence is a high-risk period for the development of alcohol use disorders, the longitudinal study of the chronology of alcohol use disorders and major depression beginning in adolescence should prove to be the most informative. Longitudinal research also would have the potential of further refining and disaggregating important subtypes of alcohol use disorders and

major depression and elucidating accompanying risk factors helpful to prevention, treatment and follow-up. In the short-term, it will also be important to determine the most efficacious treatments for subgroups of patients with comorbid alcohol use disorders and major depression. Identification of relevant treatment factors need to be pursued through pharmacological dissection and controlled studies of psychosocial modalities.

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Gender Differences in DSM–IV Alcohol Use Disorders and Major Depression as Distributed in the General Population: Clinical Implications

Eleanor Z. Hanna and Bridget F. Grant

This study examined gender differences within and between five groups of subjects drawn from a large representative sample of the United States population and classified as having either major depression (MDD) only, alcohol use disorder (AUD) only, or primary, secondary, or concurrent depression to determine if these diagnostic profiles (1) were consistent with those drawn on clinical samples and (2) might suggest potential clinical implications. Respondents (N = 9,985) from a nationally representative survey of the United States population met DSM-IV criteria for classification into these five mutually exclusive groups that were compared within and between groups by gender on the characteristics of each disorder. The results were consistent with those of other studies; (1) gender distributions of AUD and depressive disorder remain almost mirror opposites, and (2) comorbid disorders are more severe than either of the conditions appearing singly. Findings of particular interest were that the synergistic effects of an alcohol and a depressive condition operate equally for both men and women with concurrent depression. This points to the necessity of attending carefully to gender biases when dealing with comorbid conditions, lest we fail to take alcoholism in the presence of depression seriously enough in women and vice versa in men. Additionally, women with primary depression are at high risk for suicide and thus may require special attention in the evaluative phase of treatment.

Comorbidity has long been a factor in the treatment of persons who are either alcohol abusers or alcoholics. This is especially the case in alcohol treatment units located in general hospital settings, where a single patient may present with multiple medical, psychiatric, and substance abuse problems. Although many of us have crafted effective if sometimes opposite ways of treating individual patients and they form groups or "types" over the course of a career, there has not been a systematic study of what treatments are most effective for alcohol problems in combination with other specific disorders. This requires first establishing the group or groups of disorders that are most relevant. In the last decade, a number of clinical and population studies have provided a more solid foundation for our clinical impressions regarding comorbidity among alcohol patients.[1-3] The National Comorbidity Study[4] found that substance use, depressive, and anxiety disorders were among the most commonly occurring conditions, with women twice as likely to have a major depressive disorder (MDD) and men a substance use disorder. This is consistent with findings from other national surveys.[1-3,5,6] Since depressive and alcohol use (AUD) disorders assort themselves reciprocally between the sexes, one must also determine if and, if so, what gender differences exist that might also impact the clinical course. The literature is replete with studies explicating the relationship of comorbid depressive disorders and AUD[4-15] and examining how these relationships may affect the entire clinical course of those affected,[16-23] but much of it fails to address this gender issue. Recently, Cornelius et al.[24] found that among those presenting with AUD and depressive disorders for initial evaluation at a psychiatric hospital, those in the comorbid group were not only more severely ill but also more prone to suicidality than those with either single disorder. Grant et al.[6] in a study that categorized persons with these two disorders into five possible groups based on chronological sequencing noted that primary depressives evidenced more severity of illness and suicidal ideation than those with either MDD or secondary depressive disorder. In their study,[6] the primary depressive category included more women than the secondary. Bronisch,[25] categorizing on the usual basis of placing the more severe disorder first, reported rates for primary depression that were

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much lower than for secondary depression among alcoholics. Since depressive disorders and AUDs are reciprocally distributed in the sexes and since the categorization used by Grant et al. was based on chronological ordering, the fact that the primary depressive category included more women than the secondary depressive category is consistent with the notion that women evidence more depression and thus suicidal ideation and men more completed suicide, usually accompanied by substance abuse. However, it does not permit us to determine if women who are primary depressives are more prone to suicide than men or their female counterparts in the other comorbid categories. The cited studies illustrate the importance of including both gender differences and diagnostic group differences in studying comorbid AUD and depressive disorders. Thus, it is the purpose of this study to determine whether gender differences in the characteristics associated with depressive disorders and AUD occur within and across the five diagnostic subgroups as defined by Grant et al. If so, the implications for the clinical course of men and women with these combinations of AUD and depressive disorders can be examined.

METHOD

Sample

Data from 42,862 respondents aged 18 years and older collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey were analyzed for this study. A complex multistage design was used to ensure a nationally representative sample of the United States population. The household response rate was 91.90%, and the sample person response rate was 97.4%. Oversampling of the black population to secure adequate numbers for analytic purposes and of the young adult population aged 18–29 to include greater representation of this heavy substance using segment of the population was accomplished at the segment and household stage of selection, respectively. This survey is described in detail elseswhere.[26-28]

Diagnostic Assessment

DSM-IV,[29] drug use disorder, and MDD diagnoses were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule.[26] Reliabilities (κ) for past year and prior to past year AUD, drug use disorder, and MDD were 0.76 and 0.73, 0.66 through 0.91, and 0.60 and 0.65, respectively, as ascertained from an independent test-retest study conducted in the general population.

Five mutually exclusive groups were constructed using DSM-IV diagnoses and age of onset of each diagnostic disorder to include respondents with one or more of these disorders. Briefly, they were as follows: (1) MDD only contained respondents who met criteria for a lifetime diagnosis of MDD but not AUD; (2) AUD only contained respondents who met criteria for a lifetime diagnosis of AUD but not MDD. Respondents who met lifetime criteria for both AUD and depressive disorders were classified as (3) primary depressives if the age of onset of MDD preceded that of AUD; (4) secondary depressives if the age of onset of AUD preceded that of MDD; and 5) concurrent depressives if the age at onset was the same for both MDD and AUD.

Our primary focus is on AUD and MDD alone and comorbid with one another. Given the various etiological and clinical explanations for their relationship, chronological sequencing and chronicity and periodicity of symptoms are necessary to permit more clinically useful extrapolation. This necessitates using a classification system different from the traditional one based on severity of depression.

Measures

Alcohol-related measures included age of onset, age of taking first drink, duration of longest episodes of abuse and dependence, and severity of an AUD as measured by the number of lifetime DSM-IV symptoms of abuse or dependence (range, one to 31). Groups were also compared for mean ethanol intake during the lifetime period of heaviest consumption. Daily ethanol intake for the period of heaviest drinking was computed by multiplying the number of heavy drinking days per year by the number of drinks per heavy drinking day by the ounces of ethanol consumed in a typical drink on a heavy drinking day summed over beverage types (beer, wine, and liquor) and divided by 365. Additionally, groups were compared on a number of alcohol treatment measures. Respondents had been asked if they had ever sought and received help for their drinking problem. If so, they were asked to specify from among 23 types of treatment sources including inpatient and outpatient facilities and 12-step groups.

With respect to depression-related characteristics, groups were compared on age of onset of the depressive disorder, severity in terms of the duration and number of depressive episodes and the total number (range, one to 19) and type of depressive symptoms during their worst episode of depression, in addition to the percentage receiving treatment for depression.

Drug-related measures included age of first use and onset for first drug use disorder, i.e., abuse and/or dependence, as well as percentage using and classified with a drug use disorder for each class of drugs separately.

Groups were also compared in terms of the percent receiving inpatient, outpatient, and 12-step treatment for each of these disorders using drug treatment measures similar to those constructed for alcohol treatment.

Analytic procedure

Overall chi-square and F statistics were computed for categorical and continuous measures, respectively. SUDAAN,[30] a software program that uses Taylor series linearization to adjust for sample design characteristics in complex surveys was used to conduct all statistical tests. Separate analyses were conducted for each gender to examine diagnostic group differences on each characteristic. Partitioned chi-square or *t* tests were used as appropriate for these pairwise between-group comparisons. To reduce the probability of type I error arising from multiple comparisons only test statistics significant at *P* not greater than .0001 were considered. Tables reflect descriptive statistics for men and women for pairwise comparisons only on characteristics on which the gender difference met significance. Similarly, gender differences significant at *P* not greater than .001 within each diagnostic group are also noted in these tables, indicated by an asterisk.

RESULTS

Of 9,985 respondents who met diagnostic criteria for the conditions studied herein, 52% were men and 48% women. More women than men (73% *v* 27%) were diagnosed with MDD only, with the reciprocal being true for AUD only (35% women *v* 65% men). The difference between genders for comorbid AUDs and depressive disorders was much smaller, with women comprising more of the primary depressive group (54% *v* 46%) and men more of the concurrent (53% *v* 47%) and secondary (57% v 43%) depressive groups.

Demographics

The sociodemographic profile is presented in Table 1. Gender differences were significant primarily within the AUD only group. Women with AUD only were younger (mean, 34.6 *v* 38.0 years), less likely to be married (56% *v* 62%), and of a lower socioeconomic status (SES) (mean, 49.4 v 53.6) than men. Women with secondary depressive disorder were also significantly younger than men (mean, 33.6 *v* 36.4 years), whereas those with primary depressive disorder were of a lower SES (mean, 48.8 *v* 57.0) than men.

When the genders were examined separately across diagnostic groups, men and women presented slightly different demographic profiles on age, marital status, and SES. Among men, primary depressives were significantly younger (mean, 31.7 years) than male respondents in the MDD only, AUD only, and secondary depressive groups (mean, 37, 38, and 36.4 years, respectively). Sixty-two percent of the men with AUD only were married, significantly more than those with either primary or secondary depression (41.7% and 53.8%, respectively). The SES of men in the MDD only group was significantly higher than that of men in either the primary or secondary depressive groups (mean, 56.98 *v* 50.24 and 48.94, respectively).

Women classified as MDD only were significantly older (mean, 37.9 years) than those in all other groups. Primary depressives were less likely to be married than women with either MDD only or AUD only (43.6% *v* 54.8% and 55.9%, respectively) and were of a lower SES than secondary depressives (mean, 44.55 *v* 52.0).

Alcohol-related characteristics

Gender differences within each diagnostic group and separate analyses for men and women on between-group differences in alcohol-related characteristics are presented in Table 2. There was no significant difference in age of onset for AUD between men and women in any of the five diagnostic categories. However, men in the AUD only group were younger at the time of their first drink (mean, 17.1 *v* 17.8 years), drank more (mean, 4.3 *v* 2.4 oz), and reported both more symptoms (mean, 10.1 *v* 9.1) and longer episodes of abuse and dependence (mean, 52.3 *v* 35.8 months) than women. It is of particular interest that although secondary depressives followed the same gender patterns as the AUD only group with the exception of age at first drink, and primary depressives deviated only in terms of no gender differences on number of symptoms and duration of abuse, concurrent depressives evidenced no significant gender differences even on mean daily alcohol consumption. Men in the AUD only group were also more likely than women to have received all three types of treatment. However, although men in the primary and secondary depressive groups received more 12-step treatment than women, there were no significant within-group gender differences among the concurrent depressives on treatment.

Both men and women diagnosed with MDD only consumed significantly less alcohol per day at the time of their heaviest drinking (mean, 1.66 and 0.66 oz, re-

Table 1. Sociodemographic Characteristics of Male and Female Respondents With DSM-IV MDD Only, DSM-IV AUD Only, or Primary, Concurrent, or Secondary Depression (mean ± SE)

Sociodemographic Characteristic	(1) MDD Only		(2) AUD Only		(3) Primary Depression		(4) Concurrent Depression		(5) Secondary Depression		Significant Group Comparisons†	
	Men (n = 713)	Women (n = 1,931)	Men (n = 3,666)	Women (n = 2,004)	Men (n = 322)	Women (n = 383)	Men (n = 145)	Women (n = 130)	Men (n = 395)	Women (n = 296)	Men (n = 5,241)	Women (n = 4,744)
% Black	8.8 ± 1.27	9.7 ± 0.75	7.6 ± 0.61	6.7 ± 0.61	3.8 ± 1.17	6.5 ± 1.35	4.8 ± 1.94	4.9 ± 2.12	4.8 ± 1.23	3.9 ± 1.15		
Current age (yr)	37.0 ± 0.56	37.9 ± 0.38	38.0 ± 0.31	34.6 ± 0.37*	31.7 ± 0.62	32.3 ± 0.69	34.8 ± 1.12	33.6 ± 1.12	36.4 ± 0.65	33.6 ± 0.60*	3 *v* 1, 2, 5	1 *v* 2, 3, 4, 5
% Married	55.2 ± 2.28	54.8 ± 1.35	62.0 ± 1.04	55.9 ± 1.50*	41.7 ± 3.20	43.6 ± 3.01	47.6 ± 4.67	54.1 ± 5.06	53.8 ± 2.96	55.6 ± 3.31	2 *v* 3, 5	3 *v* 1, 2
% High school education	11.1 ± 1.44	13.0 ± 0.92	15.7 ± 0.76	13.3 ± 0.95	17.1 ± 2.75	12.2 ± 2.16	16.7 ± 3.36	12.8 ± 2.90	13.0 ± 1.82	10.9 ± 2.24		
SES	57.0 ± 1.26	48.8 ± 0.70*	53.6 ± 0.50	49.4 ± 0.67*	50.2 ± 1.47	44.6 ± 1.52*	48.9 ± 2.23	49.4 ± 2.68	53.9 ± 1.56	52.0 ± 1.58	1 *v* 3, 4	3 *v* 5

*Significant ($P < .001$) gender differences within diagnostic group.
†Comparisons significant at $P < .001$.

Table 2. Alcohol-Related Characteristics of Male and Female Respondents With DSM-IV MDD Only, DSM-IV AUD Only, or Primary, Concurrent, or Secondary Depression (mean ± SE)

Alcohol-Related Characteristic	(1) MDD Only		(2) AUD Only		(3) Primary Depression		(4) Concurrent Depression		(5) Secondary Depression		Significant Group Comparisons†	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Daily ethanol intake (oz)	1.7 ± 0.20	0.7 ± 0.05*	4.3 ± 0.14	2.4 ± 0.12*	4.7 ± 0.42	2.7 ± 0.12*	5.3 ± 0.45	4.7 ± 0.97	5.8 ± 0.42	3.0 ± 0.28*	1 *v* 2, 3, 4, 5 2 *v* 5	1 *v* 2, 3, 4, 5
Age at first drink (yr)	18.1 ± 0.18	19.2 ± 0.15*	17.1 ± 0.06	17.8 ± 0.11*	16.1 ± 0.17	17.6 ± 0.29*	16.8 ± 0.28	16.9 ± 0.33	16.6 ± 0.19	16.8 ± 0.21	1 *v* 2, 3, 4, 5 2 *v* 3	1 *v* 2, 3, 4, 5 2 *v* 5
Onset AUD (yr)	N/A		23.0 ± 0.17	24.0 ± 0.24	22.5 ± 0.40	23.7 ± 0.47	22.3 ± 0.57	21.6 ± 0.65	20.0 ± 0.32	19.8 ± 0.39		5 *v* 2, 3
No. of lifetime alcohol symptoms	N/A		10.1 ± 0.11	9.1 ± 0.13*	14.0 ± 0.42	10.9 ± 0.32*	14.1 ± 0.64	12.9 ± 0.63	14.2 ± 0.40	11.5 ± 0.41*	2 *v* 3, 4, 5	2 *v* 3, 4, 5 3 *v* 4
Longest duration of abuse (mo)	N/A		52.4 ± 1.87	35.8 ± 1.87*	23.5 ± 3.25	25.8 ± 2.36	45.3 ± 6.01	32.1 ± 5.63	49.6 ± 4.36	32.4 ± 2.81*	2 *v* 3	
Longest duration of dependence (mo)	N/A		42.5 ± 1.71	26.7 ± 1.29*	25.8 ± 2.36	23.4 ± 2.24	37.5 ± 5.41	26.6 ± 4.68	42.5 ± 4.05	28.3 ± 2.83*	3 *v* 2, 5	
% Lifetime treatment												
Inpatient	N/A		9.5 ± 0.53	5.2 ± 0.65*	15.9 ± 2.51	8.5 ± 1.71	21.9 ± 3.65	14.6 ± 3.18	20.9 ± 2.32	13.0 ± 2.09	2 *v* 3, 4, 5	2 *v* 3, 4, 5
Outpatient	N/A		11.7 ± 0.59	8.6 ± 0.76*	22.4 ± 2.91	16.5 ± 2.02	20.7 ± 3.55	18.0 ± 3.52	23.4 ± 2.40	20.1 ± 2.51	2 *v* 3, 4, 5	2 *v* 3, 4, 5
12-step	N/A		14.0 ± 0.70	8.9 ± 0.78*	25.1 ± 3.07	15.0 ± 1.89*	25.5 ± 3.78	19.9 ± 3.76	29.1 ± 2.59	16.9 ± 2.27*	2 *v* 3, 4, 5	2 *v* 3, 4, 5

*Significant gender differences ($P < .001$) within diagnostic group.
†Comparisons significant at $P < .001$.

Table 3. Depression-Related Characteristics of Male and Female Respondents With DSM-IV MDD Only, DSM-IV AUD Only, or Primary, Concurrent, or Secondary Depression (mean ± SE)

Depression-Related Characteristic	(1) MDD Only		(2) AUD Only		(3) Primary Depression		(4) Concurrent Depression		(5) Secondary Depression		Significant Group Comparisons†	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Onset of major depression (yr)	25.1 ± 0.52	24.8 ± 0.29	N/A		16.9 ± 0.38	16.5 ± 0.30	22.3 ± 0.57	21.6 ± 0.65	26.9 ± 0.52	26.0 ± 0.52	1 *v* 3, 4, 3 *v* 4, 5 4 *v* 5	1 *v* 3, 4, 3 *v* 4, 5 4 *v* 5
No. of episodes of major depression	4.0 ± 0.30	5.6 ± 0.29*	N/A		10.1 ± 0.92	8.6 ± 0.76	7.9 ± 1.18	6.9 ± 1.37	4.6 ± 0.37	3.7 ± 0.33	3 *v* 1, 5	3 *v* 1, 5 3 *v* 5
Longest duration of major depression (mo)	8.0 ± 1.14	9.7 ± 0.73	N/A		8.5 ± 1.07	16.5 ± 2.70*	12.5 ± 3.19	12.6 ± 3.05	6.4 ± 0.89	6.9 ± 0.85		3 *v* 5
% Ever treated for major depression	39.4 ± 2.14	55.6 ± 1.38*	N/A		41.7 ± 3.52	61.2 ± 2.85*	32.9 ± 4.45	50.2 ± 5.21	35.2 ± 2.77	55.6 ± 3.31		
No. of depressive symptoms during worst episode	9.5 ± 0.13	10.5 ± 0.08*	N/A		11.0 ± 0.20	11.9 ± 0.16*	10.6 ± 0.31	11.8 ± 0.29*	10.3 ± 0.17	11.4 ± 0.22*	1 *v* 3, 5	1 *v* 3, 4, 5
% Positive for major depressive diagnostic criteria during worst episode of depression												
Depressed mood	93.5 ± 2.03	95.9 ± 0.57	N/A		97.0 ± 1.02	95.2 ± 1.25	91.5 ± 2.29	99.3 ± 0.74	94.6 ± 1.44	95.8 ± 1.21		
Diminished interest/pleasure	88.5 ± 1.32	89.0 ± 0.83	N/A		90.5 ± 1.95	91.2 ± 1.57	85.5 ± 3.26	88.2 ± 3.31	85.7 ± 2.34	92.0 ± 1.62		
Insomnia/hypersomnia	82.7 ± 2.21	86.6 ± 1.02	N/A		87.9 ± 1.86	88.7 ± 1.87	90.1 ± 2.60	83.6 ± 3.39	85.1 ± 2.12	88.7 ± 2.04		
Psychomotor agitation/retardation	68.1 ± 2.01	63.6 ± 1.39	N/A		70.6 ± 2.67	68.1 ± 2.89	74.8 ± 3.90	70.6 ± 5.09	70.7 ± 2.84	69.0 ± 3.16		
Change in appetite/weight	62.2 ± 2.33	82.4 ± 0.99*	N/A		69.8 ± 3.23	86.4 ± 1.96*	65.4 ± 4.49	84.8 ± 3.30*	73.9 ± 2.37	81.6 ± 2.51*	1 *v* 5	
Fatigue	71.7 ± 2.04	81.6 ± 1.01*	N/A		75.6 ± 2.55	88.8 ± 1.85*	66.7 ± 4.42	83.4 ± 3.45*	67.9 ± 2.84	80.0 ± 2.64*		
Worthlessness/guilt	82.0 ± 1.65	83.4 ± 0.98	N/A		88.0 ± 1.81	92.4 ± 1.50	85.3 ± 3.19	88.7 ± 3.38	85.7 ± 2.05	87.7 ± 2.03		1 *v* 3
Difficulty concentrating/making decisions	88.6 ± 1.47	90.8 ± 0.89	N/A		91.2 ± 2.18	92.3 ± 1.72	94.8 ± 1.67	91.6 ± 3.25	91.1 ± 1.62	94.8 ± 1.48		
Suicide ideation/plan/attempts	62.9 ± 2.41	67.2 ± 1.26	N/A		76.3 ± 2.64	83.0 ± 2.19*	69.5 ± 4.37	82.1 ± 3.47*	68.4 ± 2.69	76.1 ± 2.75*		1 *v* 3, 4

*Significant gender differences ($P < .001$) within diagnostic group.

†Comparisons significant at $P < .001$.

spectively) and were less likely to have had treatment than their counterparts in the other diagnostic categories studied. Men in the AUD only group also consumed significantly less alcohol (mean, 4.3 oz) and reported fewer symptoms (mean, 10.1) than men in any of the comorbid groups. They were also significantly older at the time of their first drink (mean, 17.1 *v* 16.1 years) and reported longer periods of abuse and dependence (mean, 52.4 *v* 23.5) than men in the primary depressive group. Women in the AUD only group and those with primary depressive disorder were older at the age of onset for this disorder than women who were secondary depressives (mean, 24.0 and 23.7, respectively, *v* 19.8 years). Women who were concurrently depressed reported more symptoms than their primary depressive counterparts (mean, 12.9 *v* 10.9).

Depression-related characteristics

Depression-related characteristics are presented in Table 3. Gender differences within groups indicated that women who are in the MDD only and primary depressive groups differed from men on indices of severity, symptomatology, and treatment participation, whereas those in the concurrent and secondary depressive groups differed from men only in terms of symptomatology. Women with MDD only reported having more depressive episodes (mean, 5.6 *v* 4.0) and symptoms (mean. 10.5 *v* 9.5) than the men. Both MDD only and primary depressive women were more likely to have been treated for a depressive episode than men. Although primary depressive women did not differ from men in terms of the number of depressive episodes reported, their episodes were of longer duration (mean, 16.5 *v* 8.5). Women in every diagnostic category reported fatigue and appetite changes significantly more frequently than the men, whereas those with comorbid AUD and depressive disorders reported more suicidal ideation, plans, and attempts than their male counterparts.

Men with MDD only had fewer depressive symptoms during their worst episode of depression than men with either primary or secondary depression (mean, 9.5 *v* 11.01 and 10.3, respectively). Primary depressives reported significantly more episodes of depression than men with MDD only and secondary depression (mean. 10.1 *v* 4.0 and 4.6. respectively). A change in appetite or weight was the only depressive symptom on which men in various diagnostic groups differed. Secondary depressive men were significantly more likely to report this than were respondents with MDD only (73.9% *v* 62.2%). Differences between women distributed among the diagnostic groups followed a similar pattern with the following additions. Women with primary depression also reported more depressive episodes than women with secondary depression (mean, 8.6 *v* 3.7). They also reported a depressive episode of longer duration than did secondary depressives (mean, 16.5 *v* 6.93). Women diagnosed as MDD only also reported fewer symptoms than women in all three comorbid groups (mean, 10.5 *v* 11.9, 11.8, and 11.4 for primary, concurrent, and secondary depressives, respectively). One symptom group significantly differentiated among women in the various diagnostic categories. Primary and concurrent depressive women reported having had suicidal ideation, plans, or attempts more frequently than those with MDD only (83% and 82.1% *v* 67.2%, respectively). Additionally, primary depressive women were significantly more likely to report feelings of guilt or worthlessness than those with MDD only (92.4% *v* 83.9%).

Drug-related characteristics

Overall, the results indicate that although men outnumber women for drug use and drug use disorders (41% *v* 34%), few significant gender differences were obtained within the diagnostic groups studied (Table 4). Among those with MDD only, men exceeded women in hallucinogen use (4% *v* 1.6%) and cannabis use (20.9% *v* 14.2%), as well as cannabis use disorder (7.7% *v* 3.7%). This same pattern held true for reported drug use disorder but not drug use among those in the AUD only group. Men in this category exceeded women only in having used hallucinogens (7.6% *v* 4.7%). With the exception of primary depressives, men and women in the comorbid groups did not differ significantly on drug-related conditions. Primary depressive men exceeded women in the percentage having used hallucinogens (18.7% *v* 10.5%) and having had a hallucinogen use disorder.

Between-group differences for men and women followed nearly identical patterns for reporting both ever having used any drug and having had a lifetime drug use disorder. Both men and women in the comorbid groups exceeded their counterparts in MDD only and AUD only groups. On the other hand, between-group differences for each gender on age of first use and number of symptoms reported were not significant. Primary depressive men were significantly younger at age of first use than their counterparts in either the MDD only or AUD only groups (mean, 16.0 *v* 18.2 and 17.3 years respectively), whereas women with MDD only were significantly older at age of first use than women in all other diagnostic groups (mean, 19.4 *v* 17.5, 17.0. 16.7, and 16.4 years for AUD only,

Table 4. Drug-Related Characteristics of Male and Female Respondents With DSM-IV MDD Only, DSM-IV AUD Only, or Primary, Concurrent, or Secondary Depression (mean ± SE)

Drug-Related Characteristic	(1) MDD Only		(2) AUD Only		(3) Primary Depression		(4) Concurrent Depression		(5) Secondary Depression		Significant Group Comparisons†	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
% Lifetime drug use disorder	10.4 ± 1.47	6.1 ± 0.56	19.5 ± 0.86	17.3 ± 1.00	44.6 ± 3.12	38.2 ± 2.94	38.9 ± 4.36	40.0 ± 4.97	38.2 ± 2.79	32.9 ± 2.99	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5	1 *v* 3, 4, 5 2 *v* 3, 4, 5
Age at first drug use (yr)	18.2 ± 0.48	19.4 ± 0.50	17.3 ± 0.13	17.5 ± 0.19	16.0 ± 0.33	17.0 ± 0.37	18.4 ± 1.14	16.7 ± 0.60	17.8 ± 0.41	16.4 ± 0.35	3 *v* 1, 2	1 *v* 2, 3, 4, 5
Onset of drug use disorder (yr)	19.4 ± 0.73	21.2 ± 0.88	19.7 ± 0.22	19.6 ± 0.32	18.3 ± 0.46	19.2 ± 0.59	19.7 ± 0.64	19.9 ± 0.99	19.9 ± 0.55	18.9 ± 0.62		
No. of lifetime drug symptoms	2.5 ± 0.10	2.5 ± 0.12	1.9 ± 0.33	2.4 ± 0.33	1.3 ± 0.25	1.5 ± 0.37	1.3 ± 0.25	1.5 ± 0.37	2.1 ± 0.25	2.1 ± 0.32	2 *v* 3, 5	4 *v* 1, 2
% Lifetime use on own												
Any drug	24.0 ± 1.95	18.7 ± 0.95	39.9 ± 0.99	39.3 ± 1.35	60.7 ± 2.82	56.8 ± 3.03	53.8 ± 4.31	61.9 ± 4.59	57.6 ± 2.78	57.6 ± 3.13	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5
Prescription drugs	10.3 ± 1.43	8.4 ± 0.72	17.1 ± 0.77	17.6 ± 1.02	32.7 ± 3.11	31.6 ± 2.85	28.5 ± 3.89	39.0 ± 4.83	33.5 ± 2.46	18.2 ± 2.90	1 *v* 2, 3, 4, 5 2 *v* 3, 4	1 *v* 3, 4, 5 2 *v* 3, 4, 5
Sedatives	3.7 ± 1.03	1.6 ± 0.29	7.1 ± 0.51	6.2 ± 0.64	14.5 ± 2.10	13.1 ± 1.87	14.6 ± 3.09	19.2 ± 3.98	12.6 ± 1.85	9.2 ± 2.05	1 *v* 3, 4, 5 2 *v* 3, 4, 5	1 *v* 3, 4, 5 2 *v* 3, 4
Tranquilizers	4.2 ± 1.09	3.9 ± 0.50	7.3 ± 0.52	6.2 ± 0.64	18.2 ± 2.43	13.5 ± 2.16	13.9 ± 3.09	19.1 ± 3.73	17.1 ± 2.16	10.2 ± 2.06	1 *v* 3, 4, 5 2 *v* 3, 4, 5	1 *v* 3, 4, 5 2 *v* 3, 4
Amphetamines	6.7 ± 1.27	4.1 ± 0.48	12.9 ± 0.69	12.8 ± 0.89	27.2 ± 2.86	23.2 ± 2.69	24.5 ± 3.78	28.3 ± 5.07	26.7 ± 2.47	20.6 ± 2.72	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5	1 *v* 3, 4, 5 2 *v* 3, 4, 5
Cannabis	20.9 ± 1.85	14.2 ± 0.82*	37.5 ± 0.99	34.8 ± 1.33	58.4 ± 2.82	51.5 ± 3.07	49.8 ± 4.39	56.5 ± 4.64	51.1 ± 2.92	52.2 ± 3.07	1 *v* 2, 3, 4, 5 2 *v* 3, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5
Cocaine	5.3 ± 1.11	3.1 ± 0.40	12.3 ± 0.64	11.9 ± 0.85	20.3 ± 2.36	17.2 ± 2.04	16.8 ± 3.41	22.8 ± 4.31	23.2 ± 2.35	17.5 ± 2.55	1 *v* 2, 3, 4, 5 2 *v* 3, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4
Hallucinogens	4.0 ± 1.02	1.6 ± 0.30*	7.6 ± 0.50	4.7 ± 0.57*	18.7 ± 2.44	10.5 ± 1.76*	14.6 ± 3.11	12.8 ± 3.25	16.1 ± 2.18	9.0 ± 2.01	1 *v* 3, 4, 5 2 *v* 3, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4
% Lifetime drug use disorder												
Prescription drugs	3.4 ± 0.86	2.1 ± 0.33	5.8 ± 0.47	6.1 ± 0.59	17.5 ± 2.44	16.7 ± 2.36	14.4 ± 3.08	20.8 ± 3.82	15.8 ± 2.16	14.4 ± 2.31	1 *v* 3, 4, 5 2 *v* 3, 4, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5
Sedatives	0.9 ± 0.43	0.3 ± 0.11	2.2 ± 0.29	1.8 ± 0.32	7.0 ± 1.53	5.5 ± 1.24	6.6 ± 2.06	4.1 ± 2.05	6.0 ± 1.42	3.1 ± 1.07	1 *v* 3, 4, 5 2 *v* 3, 4, 5	1 *v* 2, 3, 4, 5 2 *v* 3
Tranquilizers	0.4 ± 0.38	0.5 ± 0.14	2.0 ± 0.29	4.4 ± 0.49	8.5 ± 1.73	4.9 ± 1.18	6.3 ± 2.17	6.9 ± 2.44	4.7 ± 1.21	5.3 ± 1.72	1 *v* 3, 4 2 *v* 3, 4, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4
Amphetamines	2.0 ± 0.70	1.2 ± 0.27	4.4 ± 0.42	4.4 ± 0.49	12.4 ± 2.05	12.0 ± 2.72	12.7 ± 2.95	14.6 ± 3.21	13.2 ± 2.04	10.8 ± 1.89	1 *v* 3, 4, 5 2 *v* 3, 4, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5
Cannabis	7.7 ± 1.27	3.7 ± 0.43*	16.3 ± 0.80	12.1 ± 0.86*	37.2 ± 3.08	27.7 ± 2.76	33.3 ± 4.26	30.6 ± 4.49	27.6 ± 2.67	22.6 ± 2.58	1 *v* 2, 3, 4, 5	1 *v* 2, 3, 4, 5 2 *v* 3, 4, 5
Cocaine	2.4 ± 0.89	1.5 ± 0.28	5.7 ± 0.46	5.6 ± 0.57	12.1 ± 1.84	9.56 ± 1.55	12.0 ± 2.94	12.5 ± 3.08	14.1 ± 1.90	9.4 ± 2.05	1 *v* 3, 4, 5 2 *v* 3, 5	1 *v* 2, 3, 4, 5 2 *v* 4
Hallucinogens	0.8 ± 0.31	0.3 ± 0.10	2.3 ± 0.29	1.6 ± 0.31	8.7 ± 1.66	3.2 ± 1.03*	7.9 ± 2.57	2.7 ± 1.36	5.9 ± 1.48	2.6 ± 0.96	1 *v* 3, 4, 5 2 *v* 3, 4, 5	1 *v* 2, 3, 4, 5
% Lifetime drug inpatient treatment	0.8 ± 0.20	1.8 ± 0.32	2.4 ± 0.30	1.8 ± 0.32	3.0 ± 1.74	4.1 ± 1.08	6.8 ± 2.29	4.6 ± 1.64	7.4 ± 1.46	4.5 ± 1.38	1 *v* 4 2 *v* 3, 5	1 *v* 3, 4, 5
% Lifetime drug outpatient treatment	1.4 ± 0.74	1.1 ± 0.24	3.1 ± 0.39	2.2 ± 0.33	9.3 ± 1.74	6.3 ± 1.43	7.6 ± 2.41	8.0 ± 2.53	10.2 ± 1.7	5.6 ± 1.76	1 *v* 4 2 *v* 3, 5	1 *v* 3, 4, 5 2 *v* 4
% Lifetime 12-step program	1.0 ± 0.71	0.6 ± 0.16	2.4 ± 0.31	2.5 ± 0.40	11.1 ± 2.30	6.0 ± 1.40	6.0 ± 2.12	8.7 ± 2.70	7.7 ± 1.46	6.9 ± 1.84	2 *v* 3, 5	1 *v* 2, 3, 4, 5 2 *v* 4, 5

*Significant gender differences ($P < .001$) within diagnostic group.

†Comparisons significant at $P < .001$.

primary depression, concurrent depression, and secondary depression, respectively). Men with AUD only reported significantly more drug-related symptoms (mean, 1.9) than those with primary depression (mean, 1.3), but fewer than those with secondary depression (mean, 2.1). Women with MDD only and those with AUD only reported significantly more symptoms than women who were concurrent depressives (mean, 2.5 and 2.4, respectively, *v* 1.5). Women in the three comorbid groups were more likely to have had all three types of treatment than those with MDD only; however, only on 12-step treatment did women in the AUD only group differ significantly from those with MDD only. Men with concurrent depression significantly exceeded both the inpatient (7.4%) and outpatient (10.2%) treatment rates of those with MDD only (0.8% and 1.8%, respectively). Men with AUD only had significantly lower rates for all three types of treatment than men with primary and secondary depression.

DISCUSSION

The results of this large national population survey are consistent with the findings of other surveys and clinical studies: (1) gender distributions of AUD and depressive disorder remain almost mirror opposites, with males predominant in the former and females in the latter, and (2) comorbid disorders are more severe than either of the single conditions. Our failure to obtain gender differences within some of these comorbid conditions especially points to the necessity of attending carefully to traditional gender biases when dealing with comorbid patients, lest we fail to take alcoholism in the presence of depression seriously enough in women and vice versa in men. Additionally, our analysis by gender across diagnostic categories suggests that specific vulnerabilities, e.g., the high suicidal potential for women with primary depression, may require attention in the evaluative phase of treatment.

Although it is no surprise that the proportions of males with AUD only and females with MDD only are reciprocals of one another, we found that within comorbid groups, primary depressives contained 8% more women and concurrent and secondary depressives more men (6% and 14%, respectively). Contrary to Cornelius et al.,[24] who found that depressed alcoholics included more men than women, our results indicate that even if this is the case, proper classification lends a different clinical and statistical interpretation to the numbers. For example, AUDs are more severe for men than for women in all diagnostic groups but the concurrent depressives, where no gender differences, even in the amount of alcohol ingested, were obtained. In addition, women in the concurrent depressive group surpass their primary depressive counterparts on the number of symptoms of alcohol dependence reported, whereas like secondary depressives, they have an earlier age of onset for alcohol-related problems than do either AUD only or primary depressives. This suggests that for women, the problems appearing concurrently are of greater import in terms of alcohol symptomatology. These women, like Cloninger's type II male alcoholics, may have a more biological or genetic basis for their alcoholism, and perhaps depression as well. For example, female concurrent depressives comprised the one comorbid diagnostic group in which the family history of alcoholism was consistently different from the others. Biological first- and second-degree relatives only were significant in the line of transmission; alcoholism in both paternal and maternal half-siblings was not significantly different from that of normal and MDD only controls.[31] Therefore, in planning the clinical course of these women, one should be aware that their alcohol problems may be less tractable and must be dealt with in a context of the more long-standing personality traits and behaviors associated with male-pattern alcoholism rather than as an adjunct to their depression. Nor can treatment of the depression wait until the alcoholism is resolved.

Our findings may be an interesting complement to the already extensive literature on the relationship of these conditions to one another. For example, both men and women with MDD only drink less and take their first drinks at a later age than their counterparts in all other diagnostic categories. This suggests that alcohol may have no part in the development of "pure" depression in both sexes and is consistent with one of the earliest studies of MDD and substance use to make the primary-secondary distinction,[32] which found that MDD preceded alcohol abuse by 4.5 years. However, primary depressive men take their first drink at an age even younger than those with AUD only. In addition, their longest episodes of alcohol abuse and dependence were shorter than those of secondary depressives. This suggests that although they may look to alcohol for relief from depression, it proves to be an ineffective palliative agent. Alternatively, Deykin et al.[32] found that as few as two episodes of drunkenness in early life were twice as common among alcohol-abusing students who met the criteria for MDD versus abusers without depression, and suggested it be considered as a marker for later depressive disorder. It is of note that primary depressive men also have more episodes of depression than those with either MDD

only or secondary depression. Secondary depressive women, on the other hand, are younger than those with AUD only at the time of their first drink. It is possible that alcohol may have a role in the development of this type of depression, if not biochemical, then from the strain or loss suffered as a consequence of drinking, or that for secondary depressive women, like primary depressive men, early drinking serves as an indicator of later depression. Additionally, it is noteworthy that secondary depressive women report fewer episodes of briefer duration and may thus have a less severe form of depression than women with primary depression. Although this might be artifactual, it is consistent with the notion of secondary depression developing as a result of environmental stressors associated with drinking for women. Thus, noting the increased report of both suicidal ideation, plans, or attempts and feelings of guilt and worthlessness among the more severely ill primary depressive women should trigger an alarm among clinicians. Alcohol with its disinhibiting effects may exacerbate an already dangerous predilection in this group of women. Additionally, primary depressive women have fewer social supports than women in the other diagnostic groups: they are younger, less likely to be married and of a lower SES. Although our findings are based on lifetime symptoms, they are consistent with actual cases reported by Cornelius et al.,[24] who found suicidality to be the major distinguishing symptom for depressive alcoholics. In addition, Beautrais et al.[33] found that substance use disorders comorbid with mood disorders prevalent in women and antisocial or anxiety disorders among men play a similar role in suicide attempts for both men and women. Conwell et al.,[34] in a study of suicide victims who underwent autopsy, found comorbid affective and addictive disorders prevalent among victims in young adulthood and middle age. Throughout the clinical course, the possibility of suicide should be a serious consideration for primary depressive women.

Men exceed women in drug use and drug use disorders. Both men and women with AUD and depressive disorders exceed those with single disorders in terms of drug use and drug use disorder. However, respondents with AUD only exceed those with MDD only with respect to drug use and disorder. This is not surprising, given the known association between illicit drug and alcohol use and the high rates of comorbidity for drug use disorders and AUDs.[2,28] Although few gender differences exist, those that do are consistent with the propensity for males to be greater risk-takers than females and for substance users to be greater risk-takers than nonusers. Men with MDD only used cannabis and hallucinogens more than their female counterparts and had more cannabis use disorders. Men with AUD only used and reported disorders with hallucinogens more than the women. Men who are primary depressives seem to be most at risk for using drugs at an earlier age versus men in the other diagnostic groups. This may, as with their use of alcohol, be indicative of an attempt at self-medication early on, especially since these men have fewer drug use-related symptoms than men with AUD only. Female primary depressives may be more disposed to suicide, but male primary depressives are more at risk for hallucinogen use and disorder and thus the significant life-threatening danger that accompanies this high-risk drug.

We would be remiss if we failed to mention again that the method used herein for establishing the primary-secondary distinction is based on chronological sequence and not on sequencing according to severity of condition. It is thus subject to the caveats attendant to all studies that rely on self-reported information. It cannot be left unsaid that almost all diagnostic information in medicine relies to some degree on the patient as a reliable historian. We have no reason to believe that our respondents whose information proved reliable on sensitive issues such as alcohol and drug use would be any less forthcoming in recalling autobiographical sequencing of life events that are usually subject to lower levels of recall bias. Additionally, the primary depressive category even as defined chronologically seems to be a more severe depressive illness than the other comorbid conditions.

In summary, these findings underscore the fact that comorbid AUDs and MDDs are more severe on each of the dimensions studied, alcohol-, depression-, and drug-related characteristics, than is either alone. The fact that we have supported in this large national survey the findings of Cornelius et al.[24] about the increased susceptibility to suicide among those with comorbid alcoholism and depression makes it imperative that clinicians in both general psychiatric and special addictions units alter long-accepted notions about suicide and severe alcoholism being predominantly male behaviors and depression a primarily female condition when evaluating and formulating treatment plans for both men and women with comorbid depressive and alcohol problems.

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Family History of Alcoholism and Gender: Their Combined Effects on DSM–IV Alcohol Dependence and Major Depression

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Objective: *Data from a representative sample of U.S. adults were used to assess the extent of familial alcoholism, to examine its association with the odds of DSM–IV lifetime alcohol dependence, major depression, and their comorbid occurrence, and to determine whether the magnitude of this association was different for men and women.* Method: *Self-report data from a sample of 42,862 U.S. adults (25,043 women) 18 years of age and over were analyzed by means of multiple logistic regression models that predicted the odds of various combinations of DSM–IV alcohol dependence and major depression.* Results: *After adjusting for potential confounders through multiple logistic regression, family history saturation was associated with increased odds of dependence only, depression only, and all primary-secondary-concurrent combinations of these two disorders. The estimated effects were greatest for comorbid dependence and depression, next highest for dependence only, and lowest for depression only. Differences in odds ratios among these groups increased with degree of family history saturation but were statistically significant at all levels of saturation. The effects of family history were greater for men than women for the outcome of primary depression followed by secondary dependence, but only at the higher levels of saturation. Among persons with lifetime major depression, family history of alcoholism had a positive independent association with the conditional odds of having experienced comorbid alcohol dependence. It had a weaker but still significant association with the odds of comorbid depression, conditional upon having experienced dependence, and this association was stronger among men than among women. For most outcomes, family history effects were stronger for paternal male and maternal female relatives than for paternal female and maternal male relatives.* Conclusions: *These findings supported prior research showing more familial alcoholism among persons with comorbid dependence and depression than among those with dependence alone. Gender differences were supportive of the proposed distinction between pure depression and depressive-spectrum disease.*

Comorbidity for alcohol dependence and depression—the increased risk of one of these disorders being manifested in the presence of the other—has been noted in clinical and epidemiological studies spanning several decades, as summarized in recent reviews by Bronisch (1990) and Davidson and Ritson (1993). Although clinical studies have been criticized for overstating the level of comorbidity because of the greater likelihood of individuals with multiple disorders entering their samples (Berkson's bias), even population-based studies have found that persons with either dependence or depression have approximately a two- to four-fold increase in the odds of having the other disorder (Grant and Harford, 1995; Helzer and Pryzbeck, 1988). In general, the studies that have defined these disorders most narrowly or strictly have found the strongest association between the two.

Numerous family history, adoption, and twin studies have investigated the effect of genetic predisposition toward alcohol dependence, depression, and their comorbid occurrence (see reviews in Bukstein et al., 1989; Cloninger et al., 1979; Hill, 1993; Merikangas and Gelernter, 1990). At the heart of these studies is the question of whether the association between the two disorders indicates that they are alternate manifestations of a common underlying genetic factor or whether their association is etiologic (one disorder causing the other) or reflective of common environmental causes. While the consensus of reviewers has been in favor of genetic heterogeneity, the results of the studies have been inconsistent. Among those that have argued against genetic heterogeneity are clinical studies by Winokur et al. (1971) who found an in-

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creased risk for alcoholism in first-degree relatives of probands with depressive disorders, by Winokur and Coryell (1991) who found higher rates of familial alcoholism among female but not male primary unipolar depressives, by Coryell et al. (1992) who found increased rates of alcoholism in the male relatives of probands with primary unipolar depression, and by Araujo and Monteiro (1995) who found that male alcoholics had higher prevalences of both familial alcoholism and familial depression than did controls. In addition, a recent epidemiological study based on a large, representative population sample (Grant et al., 1996) found no differences in the prevalences of various measures of familial alcoholism among persons with alcohol use disorders only and persons with major depression only. Individuals with comorbid alcohol use disorders and depression had the highest rates of familial alcoholism, and these rates were equally high regardless of which disorder was designated as primary. Finally, based on a study of female twins that found significant genetic correlations between major depression and alcoholism, Kendler et al. (1993) concluded that in women there were genetic factors that influenced the risks of the co-occurrence of alcoholism and major depression as well as genetic factors that influenced the risk of only one or the other of these disorders.

Several studies that included both men and women reported gender differences in findings. For example, Coryell et al. (1992) argued that depressive-spectrum disease—a subtype of depression distinguished from pure-depressive disease by the presence of familial alcoholism, younger age at onset and more extensive social, sexual and marital problems (Winokur, 1979; Winokur and Coryell, 1991)—tends to first manifest itself as depression in women and as alcoholism in men. Thus, *secondary* alcoholism may have a different etiology in men and women, more often representing a separate disorder among the former and a secondary aspect of depressive-spectrum disease among the latter. Schuckit (1983) found that alcoholics with secondary depression when compared to persons with alcoholism only had increased rates of alcoholism in their male relatives but that there were smaller or no differences for their female relatives. Similarly, Woodruff et al. (1973) found excess familial alcoholism among men with secondary depression relative to those with alcoholism only, but did not find the same pattern for women.

Adoption and twin studies also have yielded gender differences. In a study of adopted daughters, Bohman et al. (1981) found a three-fold increase in the risk of alcohol abuse among the daughters whose biological mothers were alcoholics. The excess risk among daughters whose biological fathers were alcoholics was of smaller magnitude and was observed only if the fathers' problems were mild and not associated with criminality. Further study of both male and female adoptees led Bohman et al. (1987) to distinguish three types of alcoholic families, characterized by gender differences in both composition and transmissibility. The milieu-limited type (Type 1), which showed the greatest evidence of paternal transmission to adopted-out daughters, was characterized by adult onset of alcohol abuse in both parents and lack of criminality. The male-limited type (Type 2), highly transmissible to sons but not to daughters, was characterized by early onset alcoholism, recurrent treatment for alcoholism, and alcohol-related criminality among male family members but neither alcohol-related nor criminal problems in female family members. Women in this second type of family did exhibit slightly elevated levels of somatization. In the antisocial type (Type 3), also more transmissible to sons than daughters, the level of somatization among female family members was greatly elevated, and male family members were characterized by repeated episodes of untreated alcohol abuse, violence and criminality. In addition, two twin studies (Gurling et al., 1981; Pickens and Svikis, 1991) found greater evidence of heritability of alcoholism in men than in women; that is, the rates of concordance of alcoholism in male pairs exceeded those for female pairs.

Much of the research that has investigated the impact of family history of alcoholism has been hampered by failure to employ the important distinction between primary and secondary disorders, by either not including a control group at all or by not matching the characteristics of cases and controls, or by sample sizes so small that even large differences in family history measures were not statistically significant. In contrast, this study utilizes data from a large representative sample of U.S. adults (N = 42,862); it uses individuals with neither DSM–IV (American Psychiatric Association, 1994) lifetime alcohol dependence nor major depression as the control group against which combinations of these disorders are assessed; and it uses multivariate analytic techniques to control for the potentially confounding effects of sociodemographic characteristics. Multiple logistic regression models predict the odds of five distinct lifetime diagnostic categories, based on DSM–IV definitions: alcohol dependence only, primary dependence followed by secondary depression, concurrent dependence and major depression, primary depression followed by secondary dependence, and depression only. Family history saturation, the proportion of first- and second-degree relatives identified as

alcoholics or problem drinkers, is the exposure variable of interest, and all models test for the significance of both family history and its interaction with gender as predictors of the odds of having experienced the various categories of lifetime alcohol dependence and depression. Secondary analyses focus on differences in levels of association between the outcome categories and different types of familial alcoholism (in paternal male, paternal female, maternal male and maternal female relatives) and examine the conditional risk of developing a secondary disorder given a primary diagnosis of alcohol dependence or major depression.

METHOD

Sample

The data used in this analysis were drawn from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism. The NLAES sample consisted of 42,862 adults 18 years of age and over who were selected at random from a nationally representative sample of households. Interviewers hired and trained by the Bureau of the Census collected the NLAES data in personal interviews conducted in respondents' homes. Proxy respondents were not permitted. The household and sample person response rates were 92% and 97%, respectively.

The NLAES sample was similar in design to the 1985 redesign of the National Health Interview Study (Massey et al., 1989), featuring first-stage sampling of primary sampling units (PSUs) with probability proportional to size and over-sampling of blacks. Unlike the NHIS, the NLAES also over-sampled young adults between the ages of 18 and 29 at the household level to ensure adequate numbers for analysis among this heavy-drinking subgroup (Grant et al., 1994). Because of the complex, multistage sampling design of the NLAES, estimates derived from NLAES data have standard errors that are larger than those of estimates derived from a simple random sample of equal size. Accordingly, all estimates presented in this article were produced using SUDAAN (Shah et al., 1995), a software package that uses first-order Taylor series linearization to accurately estimate variance under the conditions of a complex sampling design.

Alcohol dependence and major depression measures. The measure of lifetime DSM–IV alcohol dependence, which has been fully described elsewhere (Grant et al., 1996), was derived from measures of this disorder for two distinct time periods, the past year (that is, the year immediately preceding interview) and the period prior to the past year. For the past year, a person was classified as dependent if he or she met at least three of the seven criteria for dependence: tolerance, withdrawal, desire/attempts to cut down on or stop drinking, much time spent on alcohol-related activities, reduction/cessation of important activities in favor of drinking, impaired control, and continued use despite physical or psychological problems. Criteria not associated with duration qualifiers were satisfied if a person reported one or more positive symptoms. To satisfy a criterion associated with a duration qualifier, a person had to report two or more symptoms of the criterion or one symptom that occurred two or more times. To satisfy the DSM–IV definition of withdrawal as a syndrome, two or more symptoms were required in addition to satisfaction of the duration qualifier. To be classified with dependence during any 1-year period prior to the past year, a person had to meet three of the seven criteria listed above. Clustering and duration of symptoms were established by determining that the number of symptoms required to achieve a diagnosis (i.e., to satisfy at least three criteria) occurred at the same time, repeatedly for a few months or longer, or most days for at least 1 month. These requirements ensured a prior-to-past-year diagnosis that reflected the requisite clustering of symptoms during a 1-year period and not merely a loose collection of symptoms over the life course. For individuals who met the criteria for alcohol dependence in the past year only, age at onset of dependence was set to the age at interview. For persons with alcohol dependence in the period prior to the past year, age at onset was determined by asking the age at which the clustering of the requisite array of symptoms first occurred.

To be classified with lifetime DSM–IV major depression, a respondent had to report five or more depressive symptoms (inclusive of low mood or lack of interest) that occurred nearly every day for at least the same 2-week period, that co-occurred with social and/or occupational dysfunction, and that were not attributed to physical illness or bereavement. Age at onset of depression was ascertained by asking for the age at the first time these criteria were satisfied.

Information on the presence or absence of lifetime alcohol dependence and depression and on the relative ages at onset of each disorder were used to construct an outcome measure of diagnostic status that comprised six different categories: (1) neither dependence nor depression, (2) dependence only, (3) primary dependence followed by secondary depression, (4) con-

current dependence and depression, (5) primary depression followed by secondary dependence, and (6) depression only. A small proportion (less than 1%) of the total sample met the criteria for both dependence and depression but could not be assigned to any of the outcome categories because of missing ages at onset for one or both disorders. These cases were removed from the analyses that incorporated the primary/secondary/concurrent distinction.

Family history of alcoholism measures. Family history of alcoholism was ascertained through a series of questions that asked about 18 different types of first- and second-degree biological relatives. The former consisted of the respondents' parents, full siblings and children, the latter of the respondents' half-siblings and maternal and paternal grandparents, aunts and uncles (full biological siblings of the respondents' parents). For each type of relative, the respondent was asked how many relatives of that type lived to be at least 10 years old and how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM–IV criteria for alcohol use disorders: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover." In a test-retest study conducted in conjunction with the pretest for the NLAES, the family history items generally showed good to excellent reliability, with kappas of .70 or higher for most types of first- and second-degree relatives (e.g., .72 for fathers, 1.00 for mothers, .90 for brothers, .73 for sisters and .77 for both maternal and paternal grandfathers). Slightly lower kappa values were obtained for sons and daughters (.65 for each).

A simple dichotomous measure of family history positive (FHP) versus family history negative (FHN) was based on one or more versus no relatives identified as alcoholics. A second family history measure was obtained by dividing the number of relatives who were alcoholics by the total number of relatives who lived to at least 10 years of age. This measure represents family history saturation (i.e., the proportion of first- and second-degree relatives who were alcoholics). Both the dichotomous FHP/FHN and the continuous saturation measures were estimated for different types of relatives (e.g., male, female, paternal, maternal) as well as for all relatives combined. For the sake of brevity, the

Table 1. Family history of alcoholism in first- and second-degree relatives of U.S. men and women 18 years of age and over (% ± SE)[a]

	Total	Men	Women
Negative family history	47.1 ± 0.3	49.0 ± 0.5	45.4 ± 0.4
Positive family history in	52.9 ± 1.3	51.0 ± 0.5	54.6 ± 0.4
<10% of relatives	23.7 ± 0.2	23.6 ± 0.4	23.7 ± 0.4
10-24.9% of relatives	19.8 ± 0.2	19.0 ± 0.4	20.4 ± 0.3
25% or more of relatives	9.4 ± 0.2	8.4 ± 0.2	10.5 ± 0.2
Positive family history in any			
Male relatives	48.6 ± 0.3	46.9 ± 0.5	50.1 ± 0.4
Female relatives	20.2 ± 0.3	17.9 ± 0.4	22.3 ± 0.3
Paternal relatives	42.4 ± 0.3	40.3 ± 0.5	44.3 ± 0.4
Paternal male relatives	39.4 ± 0.3	37.7 ± 0.5	40.9 ± 0.4
Paternal female relatives	12.1 ± 0.2	10.3 ± 0.3	13.8 ± 0.3
Maternal relatives	39.4 ± 0.3	37.0 ± 0.5	41.6 ± 0.4
Maternal male relatives	33.9 ± 0.3	31.9 ± 0.5	35.7 ± 0.4
Maternal female relatives	16.2 ± 0.2	14.2 ± 0.3	18.1 ± 0.3
Average proportion of alcoholics among			
All relatives	7.9 ± 0.1	7.4 ± 0.1	8.5 ± 0.1
Male relatives	12.1 ± 0.1	11.4 ± 0.2	12.7 ± 0.1
Female relatives	3.7 ± 0.1	3.3 ± 0.1	4.2 ± 0.1
Paternal relatives	8.4 ± 0.1	7.8 ± 0.1	9.0 ± 0.1
Paternal male relatives	12.4 ± 0.1	11.7 ± 0.2	13.1 ± 0.2
Paternal female relatives	3.5 ± 0.1	2.9 ± 0.1	4.0 ± 0.1
Maternal relatives	7.4 ± 0.1	6.8 ± 0.1	7.9 ± 0.1
Maternal male relatives	11.5 ± 0.1	10.7 ± 0.2	12.1 ± 0.2
Maternal female relatives	4.1 ± 0.1	3.7 ± 0.1	4.5 ± 0.1
N of cases	42,862	17,819	25,043

[a]All figures are based on 1st- or 2nd-degree relatives who lived to at least 10 years of age. Men reported an average of 18.5 such relatives (51% male); women reported an average of 19.0 (51% male).

terms "alcoholism" and "alcoholics" as used in the remainder of this report should be understood to refer to relatives identified as alcoholics *or* problem drinkers, and the term "relatives" should be understood to refer to biological first- and second-degree relatives. In the following description of findings, all reported differences in family history saturations and effects refer to statistically significant differences unless explicitly identified as nonsignificant.

RESULTS

Slightly more than 50% of U.S. adults 18 years of age and over had positive family histories of alcoholism (Table 1); that is, they reported that one or more of their relatives had ever been alcoholics. This included nearly 25% who reported alcoholism in less than 10% of their relatives, 20% who reported alcoholism in 10.0–24.9% of their relatives and about 10% who reported alcoholism in 25% or more of their relatives. Nearly 50% of U.S. adults reported a history of alcoholism in their male relatives; only 20% reported a history of alcoholism in their female relatives. The prevalence of familial alcoholism was similar among paternal and maternal relatives. On average, U.S. adults reported that 7.9% of their relatives—12.1% of male and 3.7% of female relatives—were alcoholics. These saturation levels were about the same regardless of whether all relatives, paternal relatives or maternal relatives were considered.

Women were slightly more likely than men to report a positive family history of alcoholism (54.6% vs. 51.0%), and they reported a slightly heavier family history saturation (8.5% vs. 7.4%). The magnitudes of these differentials were similar for paternal and maternal relatives, but women's excess reporting of familial alcoholism was proportionately greater for female (4.2% vs. 3.3%) than for male (12.7% vs. 11.4%) relatives.

The majority of U.S. adults, 80.1%, had experienced neither lifetime alcohol dependence nor major depression (Table 2). One tenth (10.1%) had experienced dependence only, 6.7% had experienced depression only, and 3.1% had experienced both disorders. The comorbid group was comprised of 1.1% with primary dependence followed by secondary depression, 0.5% with concurrent dependence and depression and 1.5% with primary depression followed by secondary dependence. Men were far more likely than women to have experienced alcohol dependence only (14.6% vs. 5.9%) or primary dependence followed by secondary depression (1.6% vs. 0.8%) and were slightly more likely to have experienced primary depression with secondary dependence (1.8% vs. 1.3%). Men (0.6%) and women (0.5%) were equally likely to have experienced concurrent dependence and depression, and women were more likely than men to have experienced depression only (8.4% vs. 4.7%).

Table 2. Percentage distribution of U.S. men and women 18 years of age and over by lifetime diagnostic status for DSM–IV alcohol dependence and major depression, according to family history of alcoholism in first- and second-degree relatives (% ± SE)

				Comorbid dependence and depression				
	N	Neither dependence nor depression	Dependence only	Total comorbidity	Primary dependence, secondary depression	Concurrent dependence and depression	Primary depression, secondary dependence	Depression only
Total	42,862	80.1 ± 0.3	10.1 ± 0.2	3.1 ± 0.1	1.1 ± 0.1	0.5 ± <0.1	1.5 ± 0.1	6.7 ± 0.1
Negative family history	20,402	89.3 ± 0.3	5.7 ± 0.2	1.1 ± 0.1	0.4 ± 0.1	0.1 ± 0.1	0.6 ± 0.1	3.9 ± 0.2
Positive family history	22,460	71.9 ± 0.4	13.9 ± 0.3	5.1 ± 0.1	1.8 ± 0.1	0.9 ± 0.1	2.4 ± 0.1	9.1 ± 0.2
<10%[a]	10,084	80.4 ± 0.5	9.7 ± 0.4	2.4 ± 0.1	1.0 ± 0.1	0.4 ± 0.1	1.0 ± 0.1	7.5 ± 0.3
10-24.9%[a]	8,344	69.7 ± 0.6	15.4 ± 0.5	5.2 ± 0.3	1.9 ± 0.1	0.8 ± 0.1	2.5 ± 0.2	9.7 ± 0.4
25% or more[a]	4,032	54.8 ± 0.9	21.6 ± 0.8	11.5 ± 0.6	3.8 ± 0.3	2.0 ± 0.3	5.7 ± 0.5	12.1 ± 0.6
Men	17,819	76.7 ± 0.4	14.6 ± 0.3	4.0 ± 0.2	1.6 ± 0.1	0.6 ± 0.1	1.8 ± 0.1	4.7 ± 0.2
Negative family history	8,795	86.8 ± 0.4	8.7 ± 0.4	1.4 ± 0.1	0.5 ± 0.1	0.2 ± <0.1	0.7 ± 0.1	3.1 ± 0.2
Positive family history	9,024	67.1 ± 0.6	20.3 ± 0.5	6.3 ± 0.3	2.4 ± 0.2	1.0 ± 0.1	2.9 ± 0.2	6.3 ± 0.3
<10%[a]	4,182	83.3 ± 0.4	10.8 ± 0.3	1.9 ± 0.1	0.7 ± 0.1	0.3 ± <0.1	0.9 ± 0.1	4.0 ± 0.2
10-24.9%[a]	3,349	64.0 ± 1.0	23.1 ± 0.8	6.7 ± 0.5	2.6 ± 0.3	1.2 ± 0.2	2.9 ± 0.3	6.2 ± 0.5
25% or more[a]	1,493	48.3 ± 1.4	29.3 ± 1.3	14.5 ± 1.0	5.4 ± 0.6	2.0 ± 0.4	7.1 ± 0.8	7.9 ± 0.8
Women	25,043	83.1 ± 0.3	5.9 ± 0.2	2.6 ± 0.1	0.8 ± 0.1	0.5 ± 0.1	1.3 ± 0.1	8.4 ± 0.2
Negative family history	11,607	91.8 ± 0.3	2.8 ± 0.2	0.8 ± 0.1	0.3 ± 0.1	0.1 ± <0.1	0.4 ± 0.1	4.6 ± 0.3
Positive family history	13,436	76.0 ± 0.4	8.4 ± 0.3	4.0 ± 0.2	1.2 ± 0.1	0.8 ± 0.1	2.0 ± 0.1	11.6 ± 0.3
<10%[a]	5,902	89.2 ± 0.3	3.5 ± 0.2	1.1 ± 0.1	0.4 ± 0.1	0.2 ± <0.1	0.5 ± 0.1	6.2 ± 0.2
10-24.9%[a]	4,995	74.6 ± 0.7	8.8 ± 0.5	4.0 ± 0.3	1.3 ± 0.2	0.5 ± 0.1	2.2 ± 0.2	12.6 ± 0.5
25% or more[a]	2,539	59.6 ± 1.3	15.9 ± 0.9	9.3 ± 0.8	2.7 ± 0.3	1.9 ± 0.3	4.7 ± 0.5	15.2 ± 0.8

[a]Proportion of alcoholics or problem drinkers among 1st- and 2nd-degree relatives.

A positive family history of alcoholism was associated with an increased probability of having experienced alcohol dependence and/or depression among both men and women. Having had *any* alcoholic first- or second-degree relatives increased the risk of dependence only by a factor of 2.3 among men and 3.0 among women and increased the risk of depression only by a factor of 2.0 among men and 2.5 among women. (These relative risks were calculated by dividing the prevalence of the disorder in question among individuals with positive family histories by the prevalence of the disorder among those with negative family histories.) The relative risks were considerably higher for comorbid dependence and depression (4.5 among men and 5.0 among women) and highest for comorbidity with concurrent age at onset of dependence and depression (5.0 among men and 8.0 among women). The levels of association increased directly with family history saturation.

Table 3 presents the results of the logistic regression models used to estimate the odds of the various diagnostic outcome categories relative to the odds of having experienced neither dependence nor depression. Several of the sociodemographic factors included as controls in the model demonstrated a consistent relationship across the five outcomes. For instance, each of the five outcomes was negatively associated with age and black race and positively associated with ever having been divorced. Hispanic origin, never having been married, and college education each showed significant associations with three of the five outcomes and similar but nonsignificant associations with the others. Hispanic origin had a significant negative association with primary dependence, concurrent dependence and depression, and depression only; never having been married had a significant positive association with dependence only, primary depression, and depression only; and having attended/completed college had a significant positive association with dependence only, primary dependence, and depression only. Male gender was associated with increased odds of all of the outcomes involving dependence, with the strongest apparent effect on the outcome of dependence only. (For the outcome of primary depression, the effect of gender is not readily interpretable because of its interaction with family history saturation.) However, male gender had a *negative* association with the odds of depression only. Having been widowed decreased the odds of most of the outcomes that involved depression, but was not associated with the outcome of dependence only. Having had children had no effect on any of the outcomes.

Family history saturation (i.e., the proportion of alcoholic first- and second-degree relatives) had a positive association with all of the outcomes. Its effect was strongest for the comorbid outcomes (among which there were no significant differences), slightly lower for dependence only, and lowest for depression only. As indicated by the positive interaction term, male gender increased the effect of family history saturation for the outcome of primary depression followed by secondary dependence. An interaction of similar magnitude fell just short of significance ($p = .07$) for the outcome of primary dependence followed by secondary depression. There was no significant (or near-significant) interaction between gender and family history for concurrent dependence and depression, dependence only, or depression only.

Table 4 shows the odds ratios (ORs) derived from the models presented in Table 3. These odds ratios are presented separately for men and women to accommodate the interaction between family history and gender that was observed for the outcome of primary depression; for the other four outcomes, the odds ratios are identical for men and women. The excess odds of each type of outcome increased directly with family history saturation. For example, alcoholism in 5% of a person's first- and second-degree relatives was associated with a 26% increase in his or her odds of dependence only (OR = 1.26), whereas alcoholism in 50% of these relatives was associated with an almost ten-fold increase in the odds of this outcome (OR = 9.80). The odds ratios were consistently lowest for the outcome of depression only and highest for the comorbid disorders. For the outcome of primary depression, men's odds ratios were higher than those of women, but the difference was only significant at the highest levels of family history saturation.

Table 5 shows the family history parameters that resulted when the single measure of family history saturation was partitioned into four components: saturation among paternal male relatives, saturation among paternal female relatives, saturation among maternal male relatives, and saturation among maternal female relatives. Two of the outcomes indicated gender interactions. For the outcome of dependence only, the effect of alcoholism among maternal female relatives was weaker among men than among women. For the outcome of primary dependence, the effect of alcoholism among paternal male relatives was stronger for men than women.

Family history saturation among paternal male relatives was more strongly associated with the outcomes involving dependence than with depression only,

Table 3. Logistic regression models predicting the odds of selected lifetime diagnoses relative to the odds of having had neither dependence nor depression

	Dependence only (DPNO)			Primary dependence, secondary depression (PDPN)			Concurrent dependence and depression (CDD)			Primary depression, secondary dependence (PDPR)			Depression only (DPRO)		
	Beta	SE	*p*	Beta	SE	*p*	Beta	SE	*p*	Beta	SE	*p*	Beta	SE	*p*
Intercept	−2.182	0.107	.000	−4.540	0.249	.000	−4.964	0.401	.000	−3.879	0.246	.000	−2.045	0.102	.000
Main effects															
Age	−0.026	0.002	.000	−0.031	0.005	.000	−0.039	0.008	.000	−0.036	0.004	.000	−0.026	0.002	.000
Male	1.112	0.046	.000	0.882	0.118	.000	0.475	0.171	.007	0.229	0.121	.062	−0.491	0.051	.000
Black	−0.445	0.082	.000	−1.157	0.280	.000	−1.271	0.410	.003	−1.148	0.213	.000	−0.545	0.084	.000
Hispanic	−0.117	0.094	.216	−0.903	0.277	.002	−1.421	0.399	.001	−0.317	0.200	.117	−0.286	0.105	.008
Never married	0.150	0.069	.034	0.282	0.182	.126	0.372	0.254	.148	1.044	0.166	.000	0.461	0.072	.000
Ever divorced	0.487	0.051	.000	0.974	0.128	.000	1.189	0.186	.000	0.991	0.130	.000	0.840	0.062	.000
Ever widowed	0.009	0.106	.932	−0.856	0.412	.040	−0.726	0.454	.114	−0.644	0.341	.063	−0.514	0.129	.000
Ever had children	−0.083	0.067	.223	0.096	0.173	.582	0.211	0.216	.331	0.015	0.151	.923	0.209	0.070	.004
Attended/completed college	0.119	0.042	.006	0.305	0.118	.012	0.189	0.148	.206	0.134	0.112	.236	0.343	0.051	.000
Proportion of alcoholic 1st- and 2nd-degree relatives	4.565	0.160	.000	5.684	0.302	.000	6.007	0.431	.000	5.231	0.299	.000	3.125	0.165	.000
Interactions															
Male × Proportion of Alcoholic 1st- and 2nd-Degree Relatives	NS	NS	NS	NS	NS	NS	NS	NS	NS	1.083	0.469	.024	NS	NS	NS
Goodness of fit statistics															
Satterthswaite adj. *F*	197.5, 9.4/68 df, $p = .000$			75.2, 9.5/68 df, $p = .000$			48.1, 9.2/68 df, $p = .000$			87.2, 10.2/68 df, $p = .000$			121.5, 9.4/68 df, $p = .000$		
N of cases	36,332			32,950			32,712			33,122			35,309		

Note: Beta parameters for proportion of alcoholic 1st- and 2nd-degree relatives were significantly ($p < .05$) different for the following pairs of lifetime diagnoses: DPNO vs PDPN, CDD and PDPR; DPRO vs all others.

whereas among paternal female relatives it was equally associated with all five of the outcomes (no significant differences). Among maternal male relatives, family history was more strongly associated with primary dependence and concurrent dependence and depression than with the other outcomes; among maternal female relatives the association with depression only was weaker than the other associations while the association with primary depression was stronger.

Another way of looking at these data is to compare the effects of the different types of relatives *within* an outcome category. For the outcome of dependence only, the effect of alcoholism among paternal female relatives was weaker than the effects of alcoholism among the other types of relatives. Similarly, alcoholism among paternal female relatives was less strongly predictive of primary dependence than was alcoholism among maternal male or female relatives. For

Table 4. Odds of selected lifetime diagnoses among persons with various proportions of alcoholic first- and second-degree relatives compared to odds among persons with negative histories of alcoholism: U.S. adults 18 years of age and over

	Dependence only		Primary dependence, secondary depression		Concurrent dependence and depression		Primary depression, secondary dependence		Depression only	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Odds ratios for men										
5% of relatives alcoholic	1.26	1.24-1.28	1.33	1.29-1.37	1.35	1.29-1.41	1.37	1.32-1.42	1.17	1.15-1.19
10% of relatives alcoholic	1.58	1.53-1.63	1.77	1.66-1.87	1.82	1.68-1.98	1.88	1.75-2.02	1.37	1.32-1.41
25% of relatives alcoholic	3.13	2.90-3.39	4.14	3.57-4.80	4.49	3.63-5.55	4.85	4.04-5.82	2.18	2.02-2.37
50% of relatives alcoholic	9.80	8.38-11.47	17.15	12.75-23.06	20.15	13.21-30.75	23.51	16.31-33.88	4.77	4.06-5.61
Odds ratios for women										
5% of relatives alcoholic	1.26	1.24-1.28	1.33	1.29-1.37	1.35	1.29-1.41	1.30	1.26-1.34	1.17	1.15-1.19
10% of relatives alcoholic	1.58	1.53-1.63	1.77	1.66-1.87	1.82	1.68-1.98	1.69	1.59-1.79	1.37	1.32-1.41
25% of relatives alcoholic	3.13	2.90-3.39	4.14	3.57-4.80	4.49	3.63-5.55	3.70	3.20-4.28	2.18	2.02-2.37
50% of relatives alcoholic	9.80	8.38-11.47	17.15	12.75-23.06	20.15	13.21-30.75	13.68	10.21-18.34	4.77	4.06-5.61
N of cases	36,332		32,950		32,712		33,122		35,309	

Table 5. Disaggregated family history parameters from logistic regression models predicting the odds of selected lifetime diagnoses relative to the odds of having had neither dependence nor depression

	Dependence only (DPNO)			Primary dependence, secondary depression (PDPN)			Concurrent dependence and depression (CDD)			Primary depression, secondary dependence (PDPR)			Depression only (DPRO)		
	Beta	SE	*p*	Beta	SE	*p*	Beta	SE	*p*	Beta	SE	*p*	Beta	SE	*p*
Main effects															
Proportion of alcoholic 1st- and 2nd-degree:															
Paternal male relatives	1.374	0.100	.000	1.128	0.294	.000	1.439	0.326	.000	1.698	0.192	.000	0.911	0.116	.000
Paternal female relatives	0.611	0.165	.000	0.662	0.338	.054	1.443	0.414	.001	0.790	0.276	.006	0.495	0.206	.019
Maternal male relatives	1.103	0.095	.000	1.646	0.228	.000	1.787	0.280	.000	0.954	0.203	.000	0.677	0.113	.000
Maternal female relatives	1.873	0.238	.000	1.595	0.355	.000	1.526	0.382	.000	2.611	0.294	.000	1.099	0.184	.000
Interactions															
Male × Proportion of Alcoholic 1st- and 2nd-degree:															
Paternal male relatives	NS	NS	NS	0.991	0.345	.005	NS	NS	NS	NS	NS	NS	NS	NS	NS
Paternal female relatives	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Maternal male relatives	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Maternal female relatives	−0.751	0.309	.018	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
N of cases	36,332			32,950			32,712			33,122			35,309		

Note: The following differences in beta parameters were statistically significant ($p < .05$): For paternal male relatives, DPRO vs DPNO, PDPN (male probands only), CDD and PDPR; For paternal female relatives, none significant; For maternal male relatives, PDPN vs DPNO, PDPR and DPRO, and CDD vs PDNO, PDPR and DPRO; For maternal female relatives, PDPR vs all others, and DPNO vs DPRO (female probands only); Within the outcome of DPNO, paternal female relatives vs all other types; Within the outcome of PDPN, paternal female vs maternal male and maternal female relatives; Within the outcome of CDD, none significant; Within the outcome of PDPR, paternal male vs paternal female and maternal male relatives, and maternal female relatives vs all other types; Within the outcome of DPRO, paternal male vs paternal female and maternal male relatives, and maternal female vs paternal female and maternal male relatives.

the outcome of concurrent dependence and depression, there were no significant differences among the beta parameters for the different types of relatives. Alcoholism among maternal female relatives was far more predictive of primary depression than was alcoholism among other types of relatives; alcoholism among paternal male relatives also had a stronger effect than did alcoholism among paternal female or maternal male relatives. Similarly, the strongest predictors of depression only were alcoholism among paternal male and maternal female relatives.

Table 6 summarizes the family history regression coefficients yielded by the conditional models that examined the odds of comorbid depression conditional upon having had dependence and the odds of comorbid dependence conditional upon having had depression. These results indicate that even after having conditioned upon the presence of either alcohol dependence or major depression, family history saturation (for all types of relatives combined) increased the odds of comorbidity, that is, of having experienced primary, concurrent or secondary onset of the other disorder. The coefficients for comorbid dependence exceeded those for comorbid depression, even after taking into account the gender interactions. Consistent with the unconditional models, male gender increased the effects of family history on the odds of having had primary depression and came close to increasing the effects of family history on secondary depression among persons with lifetime alcohol dependence. However, among persons with lifetime depression, gender did not modify the effect of family history on the odds of comorbid dependence.

DISCUSSION

This study provided valuable data with which to address the question of genetic heterogeneity in the etiology of alcohol dependence and major depression. It found the expected evidence of familial transmission of alcoholism, with the odds of having experienced lifetime dependence—alone or in conjunction with major depression—increasing in direct proportion to the percentage of first- and second-degree relatives identified as alcoholics. Consistent with a number of prior studies that found more familial alcoholism among cases with comorbid alcohol dependence and depression than among cases with alcoholism or depression alone (Hensel et al., 1979; Hesselbrock, 1991; Leibenluft et al., 1993; Penick et al., 1987; Schuckit, 1983; Woodruff et al., 1973), this study found that the estimated effect of family history saturation was greatest for the comorbid outcomes. This was demonstrated not only by the larger family history coefficients for comorbid outcomes in the models that used individuals with no diagnosis as the reference group, but also by the models that showed an incremental effect of family history on the odds of comorbid outcomes conditional upon having had either dependence or depression.

The effect of family history saturation on the odds of having had alcohol dependence alone was greater than its effect on major depression alone. Likewise, its effects on the odds of the comorbid dependence outcomes (conditional upon having had depression) were larger than its effects on the odds of the comorbid depression outcomes (conditional upon having had dependence). These two findings provide qualified support for previous research favoring genetic heterogeneity of the two disorders. If alcohol dependence

Table 6. Regression parameters from conditional models: Coefficients for family history saturation and interaction between male gender and family history saturation

		Family history saturation			Male × Family History Saturation		
	N	Beta	SE	*p*	Beta	SE	*p*
Models conditional upon having had lifetime alcohol dependence							
Any comorbid depression vs dependence only	5,149	1.397	0.304	<.001	1.104	0.408	.009
Primary depression vs dependence only	4,485	1.310	0.377	.009	1.363	0.539	.014
Concurrent depression vs dependence only	4,048	2.235	0.468	<.001	NS	NS	NS
Secondary depression vs dependence only	4,286	1.127	0.485	.023	1.201	0.613	.054
Models conditional upon having had lifetime major depression							
Any comorbid dependence vs depression only	4,126	3.465	0.257	<.001	NS	NS	NS
Primary dependence vs depression only	3,263	3.383	0.394	<.001	NS	NS	NS
Concurrent dependence vs depression only	3,025	3.934	0.527	<.001	NS	NS	NS
Secondary dependence vs depression only	3,435	3.459	0.315	<.001	NS	NS	NS

and depression were different manifestations of a common underlying genetic factor, there should be no differences among these estimated parameters; that is, the effects on depression should be as great as those on dependence. Instead, the nature and magnitude of the differences that were observed suggest that, when predicting depression, family history of alcoholism is either acting as an etiologic agent (e.g., as in the development of adolescent depression in response to the pressures of living with an alcoholic parent) or acting as an imperfect proxy for family history of depression. The increased odds of an alcoholic relative having had major depression himself or herself and the fact that the presence of alcoholism in some relatives may have caused depression in others (e.g., alcoholism in an individual's father having resulted in major depression in that individual's mother) probably accounts for much of the observed association between depression and familial alcoholism. However, in the absence of a measure of familial depression, this conclusion remains only speculative, and the possibility of a more direct link between the transmission of the two disorders cannot be ruled out.

This study's finding of an increased effect of family history on the risk of secondary dependence among men provides support for the proposed distinction between depressive-spectrum disease and pure depression (Coryell et al., 1992). If women with depressive relatives were likely to develop alcoholism even in the absence of alcoholic relatives, simply because alcoholism is symptomatic of depressive-spectrum disease, then one would expect women to be less responsive to familial alcoholism than men with respect to the development of secondary alcohol dependence. This expectation was supported by these data showing greater responsiveness to familial alcoholism among men for this outcome.

This study was able to distinguish male, female, paternal, and maternal relatives and found that the effects of family history saturation varied according to the type of alcoholic relative. Although patterns varied according to outcome category, the most common finding was that alcoholism among paternal male and maternal female relatives was more strongly associated with dependence and/or depression than was alcoholism among paternal female and maternal male relatives. In all likelihood, this merely reflects the fact that these two categories, which contain the biological father and mother, are the most heavily composed of first-degree relatives. Thus, they would be expected to show the strongest levels of association based on genetic proximity alone. Of greater interest are deviations from this pattern; for example, the equally strong effect of all types of relatives with respect to the outcome of concurrent dependence and depression. This finding suggests that the overall strong effect of family history saturation on this outcome is caused not by a greater prevalence of alcoholism among first-degree relatives but by a greater concentration of alcoholism—extending to both first- and second-degree relatives—within affected families.

One of the striking findings of this study was the widespread prevalence of familial alcoholism, irrespective of the evidence of its transmissibility. The percentage of adults with any positive family history of alcoholism, 53%, was more than twice as high as that reported by Midanik (1983), who also examined a representative sample of U.S. adults but considered only first-degree relatives, excluding children. It also exceeded the 40% of U.S. adults who reported alcoholism or problem drinking in any of their first-, second-, or third-degree relatives in the 1988 National Health Interview Survey (Dawson et al., 1992). While consistent with evidence of a secular trend toward increasing prevalence of familial alcoholism (Reich et al., 1988), it is more likely that the NLAES questionnaire increased positive reporting of alcoholic relatives both by focusing attention on the specific types of relatives to be considered and by providing examples of the types of behaviors that constitute the DSM–IV criteria for alcohol use disorders. Another likely result of providing such definitions is the reduction of bias arising from alcohol-dependent respondents being more likely than other respondents to identify the signs of alcoholism in their relatives. At the same time, it should be recognized that misclassification of alcoholism in relatives may exist even when similar levels of false positives and false negatives combine to yield a reasonable estimate of the prevalence of familial alcoholism. Since any such misclassification would reduce the estimated effect of family history saturation on the risk of alcohol dependence, the measures of association presented in this analysis are probably somewhat conservative.

In interpreting the findings of this study, it is important to bear in mind any bias that may have resulted from the fact that women reported slightly more alcoholic relatives than did men—a pattern of reporting that has been found in other general population and clinical samples (Dawson et al., 1992; Hesselbrock, 1991). Because the multivariate analyses controlled for gender, this reporting differential should affect neither the estimated effect of family history saturation itself, nor comparisons of this effect across outcome categories. It could affect interactions between

male gender and family history, however, slightly reducing the magnitude of positive interactions or inflating the magnitude of negative interactions. This risk would be greatest with respect to familial alcoholism in female relatives, among whom the gender differential in the reported prevalence of alcoholism was greatest.

This study's unique contribution lies in its having used a sample large enough to both adjust for sociodemographic factors and test for conditional as well as absolute effects of family history of alcoholism. In doing so, it was able to clarify the incremental effect of family history on the conditional risks of comorbidity, which exceeded its underlying association with the odds of having experienced alcohol dependence and major depression and demonstrated gender differentials that need to be considered in evaluating treatment modalities. This study also provided some interesting findings for further exploration in clinical samples, namely the multigenerational concentration of alcoholism in individuals with concurrent onsets of dependence and depression and the indication of matrilineal heritability of depressive-spectrum disease.

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Familial Aggregation of DSM–IV Alcohol Use Disorders: Examination of the Primary-Secondary Distinction in a General Population Sample

Bridget F. Grant and Roger Paul Pickering

This study examined the familial aggregation of alcoholism in subgroups of respondents classified with respect to the primary-secondary distinction as it is related to DSM-IV major depression and alcohol use disorders. Rates of alcoholism among specific first- and second-degree relatives of male and female probands with primary, secondary, and concurrent depression (i.e., the comorbid groups) and with major depression only were compared with one another and with a normal control group. The results of this general population survey that uses a large representative sample of the U.S. were at variance with some findings from the clinical literature with regard to familial aggregation. Greater rates of alcoholism were found among first- and second-degree relatives of the major depression only group compared with normal controls. Male and female probands of all three comorbid groups were not shown to convey a greater risk of alcoholism to their offspring compared with the normal control group or the major depression only group. The discrepancy between clinical research findings and those of this general population study were discussed in terms of methodological considerations.

Over the past 30 years, there have been several studies that have focused on the familial aggregation of major depression and alcohol use disorders among probands defined in terms of the primary-secondary distinction (Bronisch, 1990; Davidson and Ritson, 1993; Merikangas and Gelernter, 1990). The obvious heuristic value of studying familial aggregation within the context of the primary-secondary distinction was to refine diagnostic criteria and identify meaningful homogeneous subtypes for the purpose of improving classification, diagnosis, management, and outcome.

Of particular interest in this literature was determining whether depression and alcoholism were manifestations of the same underlying disorder or, alternatively, were transmitted independently. The data clearly favored the independence of the transmission of major depression and alcoholism among clinical samples of depressed probands (Gershon et al., 1982; Merikangas et. al. 1985 a and b; Weissman et al., 1984). In all these studies, the rates of alcoholism among first-degree relatives of probands with major depression were not greater than those observed among relatives of controls. Among the studies that have argued against heterogeneity are the clinical studies of Winokur and Coryell (1991) who found higher rates of alcoholism among first-degree relatives of depressed probands compared with controls and by Puig-Antich et al. (1989) who found that the prevalence of alcoholism was double among first-degree relatives of children with major depression compared with relatives of control children.

However, the results become mixed when the two disorders are broken down according to sex and/or the order of onset of the disorders. Winokur and Coryell (1991) found higher rates of alcoholism among first-degree relatives of depressed women, but not men, compared with normal controls, whereas no such corresponding sex differences were found in the study by Merikangas et al. (1985a). Puig-Antich et al. (1989) found a similar result for first-degree relatives of depressed children, but among their second-degree relatives, the rates of alcoholism were greater among male and female probands relative to male and female normal controls, respectively.

Although no one study to date has compared the rates of alcoholism in the relatives of a depressed only proband and probands with primary depression (in which the onset of depression precedes the onset of alcoholism), secondary depression (in which the onset of alcoholism precedes the onset of depression), and con-

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current depression (in which the onset of alcoholism and major depression occur during the same time), the results from those studies that have examined familial aggregation broken down by the primary-secondary distinction are relatively consistent. In general, primary depressives have higher rates of alcoholism in first-degree relatives than major depression only probands (Leibenluft et al. 1993; Merikangas et al., 1985a). Woodruff et al. (1973) found higher rates of alcoholism among male, but not female, secondary depressives compared with major depression only probands. Regardless of depressed proband sex, male first-degree relatives of primary depressives had a much higher risk for alcoholism than did the male relatives of the major depression only proband. Together, these results support the independence of the transmission of alcoholism and major depression.

These earlier studies had several limitations. Foremost, all were conducted in clinical samples of alcoholics or among patients with major depression. These clinical samples may not be suitable for the study of familial aggregation across probands defined in terms of the primary-secondary distinction, because such samples are biased toward the more severely affected population. Thus, there is a greater likelihood of probands entering these samples with multiple disorders (Berkson, 1946). Much of this research was also restricted to examining rates of alcoholism among aggregate measures of first-degree relatives, neglecting potential variation among specific first-degree relative types (*e.g.,* mothers, brothers). Their results were further compromised by small sample sizes that may have obscured significant and meaningful differences in family history measures and precluded analyses by sex of proband and the whole range of depression diagnoses defined in terms of the primary-secondary distinction.

This study overcomes many of the methodological limitations of prior research examining the familial aggregation of probands defined in terms of alcoholism and major depression. This study uses data from a large representative sample of the U.S. (N = 42,862). The sample is large enough to examine the risk of alcoholism among specific types of first- and second-degree relatives by sex of proband and by subgroups defined in terms of the primary-secondary distinction. Among these were three comorbid subgroups: primary depressives, secondary depressives, and concurrent depressives. The latter category, which has not been examined in past research because of small sample sizes, comprised that subgroup of respondents in which the onsets of an alcohol use disorder (*i.e.,* alcohol abuse and/or dependence) and major depression occurred during the same time. For the purposes of this study, primary depressives will refer to comorbid respondents whose onset of major depression predated their onset of an alcohol use disorder, and secondary depressives will refer to comorbid respondents whose onset of an alcohol use disorder predated their onset of major depression. The remaining two subgroups consisted of respondents who were classified with major depression and no history of an alcohol use disorder (major depressive disorder [MDD] only group) and respondents with no lifetime histories of an alcohol use disorder or major depression (control group). Of particular interest in this study is whether the results of familial aggregation studies supporting the independent transmission of alcoholism and major depression conducted exclusively in clinical samples would be confirmed in this general population sample.

METHODS

Sample

The data used in this analysis were drawn from the 1992 National Longitudinal Alcohol Epidemiologic Study (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism (NIAAA; Grant et al., 1994; Massey et al., 1989). The NLAES sample consisted of 42,862 adults, 18 years of age and over, who were selected at random from a nationally representative sample of households. Interviewers underwent a 2-week extensive and standardized training course, including extensive reliability checks of their first 50 interviews. Personal interviews were conducted in respondents' homes, and proxy interviews were not permitted. The household and sample person response rates were 92% and 97%, respectively.

The NLAES sample was similar in design to the 1985 redesign of the National Health Interview Survey, featuring first-stage sampling of primary sampling units (PSUs) with probability proportional to size and oversampling of blacks. The NLAES also oversampled young adults between the ages of 18 and 29 at the household level to ensure adequate numbers for analysis among this heavy drinking subgroup.

Diagnostic assessment

Diagnoses of DSM-IV (American Psychiatric Association, 1994) alcohol use disorders were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully

structured psychiatric interview designed to be administered by trained interviewers who were not clinicians (Grant and Hasin, 1992). The AUDADIS included an extensive list of symptom items that operationalized the DSM-IV criteria for alcohol use disorders (*i.e.,* abuse and dependence) and major depression. For the purposes of this study, the term alcohol use disorders will be used synonymously with the term *alcoholism*. Diagnoses were derived for two periods, the past year and before the past year.

Respondents were classified as dependent if they met at least three of the seven DSM-IV criteria for dependence within any 1-year period: tolerance; withdrawal or avoidance of withdrawal; desire/attempts to cut down or stop drinking; much time spent on drinking, obtaining alcohol, or recovering from its effects; reduction/cessation of important activities in favor of drinking; impaired control; and continued drinking despite physical or psychological problems caused or exacerbated by drinking. Respondents were classified with abuse if they met at least one of the four DSM-IV criteria for abuse in any 1-year period: alcohol-related legal problems; continued drinking despite interpersonal problems; neglect of role responsibilities due to drinking; and drinking in hazardous situations.

The AUDADIS diagnoses of alcohol abuse and dependence also satisfied the clustering and duration criteria of the DSM-IV definitions. The criteria of the DSM-IV included the requirement for a clustering of symptoms within any 1-year period, in addition to associating duration qualifiers with certain abuse and dependence criteria. The duration qualifiers are defined as the repetitiveness with which symptoms must occur to be counted as positive toward a diagnosis. They are represented by the terms recurrent, often, and persistent, appearing in the description of the diagnostic criteria. Not only were the clustering criteria represented in past-year AUDADIS diagnoses of abuse and dependence, but corresponding past diagnoses (before the past year) were also measured as syndromes, or the clustering of the required number of symptoms necessary to achieve a diagnosis: a) at the same time; b) continuously for at least 1 month; or c) repeatedly for a few months or longer. For the purposes of the present study, respondents were classified with a lifetime alcohol use disorder if they had experienced an episode of abuse or dependence in the past year and/or before the past year.

Episodes of DSM-IV major depressive disorder were also derived for the past year, before the past year, and lifetime. Consistent with the DSM-IV, the AUDADIS diagnosis of major depression required the presence of at least five depressive symptoms (including depressed mood or loss of pleasure and interest) nearly every day for most of the day for at least a 2-week period. Social and/or occupational dysfunction must also have been present during the disturbance. Episodes of major depression exclusively due to bereavement or physical illness were ruled out.

Reliabilities of the past year and before the past year alcohol use disorders and major depression were .76 and .73, and .60 and .65, respectively, as determined from an independent test-retest study conducted in a general population sample (Grant et al., 1995). The questions designed to ascertain the onsets of alcohol use disorders and major depression, which in turn defined the primary, secondary, and concurrent comorbid groups in this study, were also highly reliable: .73 for the onset of depression and .85 for the onset of an alcohol use disorder (Grant et al., 1994).

Age of onset of DSM-IV alcohol use disorders and major depression was defined as the respondent's age when the first episode of each of these disorders began, because episodes of either disorder may persist for longer than 1 year. Those respondents with a lifetime diagnosis of major depression without a history of an alcohol use disorder constituted the MDD only group. Among the three comorbid groups, respondents with an age of onset of depression preceding an alcohol use disorder were classified as primary depressives, and respondents with an age of onset of an alcohol use disorder preceding major depression were classified as secondary depressives, whereas respondents who reported the same age of onset for both an alcohol use disorder and major depression were classified as concurrent depressives. Our control group consisted of all respondents who had no lifetime history of an alcohol use disorder or major depression.

Family history of alcoholism measures

Family history of alcoholism was ascertained through a series of questions that asked about 18 different types of first- and second-degree biological relatives. For each type of relative who was 10 years old or older or, alternatively, who lived to be at least 10 years old, the respondent was asked how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM-IV criteria for alcohol use disorders:

> By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the

police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover.

Thus, relatives classified as family history positive in this study had at least one of these alcohol problems, a cutoff identical to the one used in the Family History-Research Diagnostic Criteria (FH-RDC; Andreasen et al., 1977). Similar to past research, simple dichotomous measures of family history positive versus family history negative were based on one (or more) relatives versus no relatives of each type identified as alcoholics.

The family study method in which relatives are interviewed directly was not used in this study. However, the comparisons of the family history method of this study and the family study method have shown high agreement between diagnoses of alcoholism (Andreasen et al., 1986) and better accuracy of alcohol diagnoses than for less severe disorders (Thompson et al., 1982), indicating the usefulness of the family history method in familial aggregation studies. Moreover, the test-retest reliabilities of the family history of alcoholism variables, as ascertained from an independent test-retest study conducted in the general population by the U.S. Bureau of the Census (Grant et al., 1991), were good to excellent: .72 and 1.00 for father and mother; .93 and .73 for full brothers and sisters; .65 each for sons and daughters; .71 to .81 for maternal and paternal aunts and uncles; and .64 to 1.00 for maternal and paternal grandparents.

Statistical analyses

To take into account the NLAES sample design, all standard errors were generated and statistical tests conducted with the use of Survey Data Analysis for Multi-Stage Sample Designs (SUDAAN; Research Triangle

Table 1. Number of Relatives among Probands

Relatives	Control no major depression or alcohol use disorder (N = 32,859)	Major depression only (N = 2,626)	Primary depression (N = 705)	Concurrent depression (N = 275)	Secondary depression (N = 691)
First-degree relatives					
Men	115,265	7,752	1,811	692	1,835
Women	111,851	7,647	1,789	712	1,909
Total	227,116	15,399	3,600	1,404	3,744
Second-degree relatives					
Men	206,535	16,018	4,071	1,689	4,004
Women	199,578	15,600	3,955	1,555	4,007
Total	406,113	31,618	8,026	3,244	8,011
Total men	321,429	23,770	5,882	2,381	5,839
Total women	311,429	23,247	5,744	2,267	5,916
Total	633,229	47,017	11,626	4,648	11,755
Mean pedigree size	19.3	18.0	16.5	17.0	17.0

Table 2. Sociodemographic Characteristics of Respondents with DSM-IV Major Depression Only, Primary, Concurrent, and Secondary Depression and Controls

Sociodemographic characteristic[a]	Control no major depression or alcohol use disorder (0)	Major depression only (1)	Primary depression (2)	Concurrent depression (3)	Secondary depression (4)	Significant comparisons (χ^2 or t, $p<.005$)
% Men	44.6 (0.35)	31.4 (1.11)	53.0 (2.02)	53.8 (3.34)	64.3 (2.02)	0 versus (1)(2)(3)(4); 1 versus (2)(3)(4); 2 versus (4)
% Black	12.7 (0.42)	9.4 (0.67)	5.1 (0.89)	4.8 (1.43)	4.5 (0.90)	0 versus (1)(2)(3)(4); 1 versus (2)(3)(4)
Current age in years (x)	46.0 (0.17)	37.6 (0.32)	31.9 (0.46)	34.3 (0.79)	35.4 (0.47)	0 versus (1)(2)(3)(4); 1 versus (2)(3)
Socioeconomic status (SES) score (x)	50.1 (0.22)	51.4 (0.65)	47.6 (1.09)	47.2 (1.7)	53.2 (1.14)	

Note: SE values appear in parentheses.

[a] Men versus women; African American versus non-African American; SES scores based on 1990 Nam-Powers-Terrie occupational status scores (Terrie and Nam, 1994).

Institute, 1995), a software program that uses the Taylor series linearization to adjust for the sample design characteristics in complex sample surveys. Overall chi-square and *F* statistics were computed for categorical and continuous measures, respectively. If the overall chi-square or *F* statistic was significant (<.05), separate pairwise comparisons were conducted between the groups by using partitioned chi-square analysis or *t*-tests, respectively. To protect against Type I error, due to multiple comparisons, any pairwise comparison needed to yield a test statistic value that could be declared statistically significant at a probability level of <.005. For the purpose of this study, all prevalence rates are reported as weighted percentages, and all sample sizes reflect unweighted figures.

RESULTS

There were a total of 4,315 probands: 2,644 with MDD only; 705 with primary depression; 275 with concurrent depression; and 691 with secondary depression. The normal control group consisted of 32,859 respondents. The mean pedigree size of the normal control group was 19.3 compared with 18.0, 16.5, 17.0, and 17.0 for the MDD only, primary depression, concurrent depression, and secondary depression groups, respectively (Table 1).

As shown in Table 2, the respondents in the normal control group were more likely to be African American and older than those in the four depression groups and less likely to be men than those in the three comorbid groups. The respondents in the MDD only group were older, less likely to be men, and more likely to be African American than those in three comorbid groups. There were very few significant differences between the comorbid groups, with the exception that there were more men among the secondary depressives compared with the primary depressives. Socioeconomic status did not differ between the five groups of probands. The sociodemographic differences observed between the five probands did not significantly impact on the mean pedigree size associated with each group.

Table 3. Rates of Alcohol Use Disorders among Relatives of Male Probands

Relative	Control no major depression or alcohol use disorder (0) (*N* = 12,567)	Major depression Only (1) (*N* = 713)	Primary depression (2) (*N* = 322)	Concurrent depression (3) (*N* = 145)	Secondary depression (4) (*N* = 395)
Male Relatives					
Father	13.9 (.37)	23.7 (1.85)	42.9 (3.16)	37.3 (4.35)	37.4 (2.61)
Son	2.6 (.17)	5.1 (1.00)	4.2 (1.42)	3.8 (1.59)	6.6 (1.53)
Brother	13.0 (.37)	15.1 (1.61)	22.8 (2.67)	21.8 (3.71)	26.4 (2.58)
Paternal half brother	0.9 (.09)	1.1 (.39)	3.6 (1.03)	5.2 (2.12)	2.7 (.80)
Paternal uncle	12.2 (.35)	23.0 (2.38)	28.8 (2.71)	23.7 (3.76)	28.4 (2.51)
Paternal grandfather	6.2 (.26)	15.7 (1.56)	23.5 (2.56)	15.5 (3.34)	23.1 (2.45)
Maternal half brother	0.9 (.09)	1.5 (.44)	4.2 (1.27)	3.5 (2.1)	4.2 (1.15)
Maternal uncle	12.8 (.34)	17.0 (1.53)	28.1 (2.74)	33.2 (4.46)	24.5 (2.61)
Maternal grandfather	6.4 (.25)	13.6 (1.53)	26.6 (2.64)	32.1 (4.28)	18.9 (2.25)
Any first-degree male	22.7 (.47)	35.4 (2.20)	52.2 (3.14)	51.1 (4.78)	53.1 (3.00)
Any second-degree male	28.2 (.47)	48.6 (2.33)	65.7 (2.79)	64.4 (4.32)	59.2 (2.93)
Female Relatives					
Mother	3.3 (.19)	8.1 (1.28)	17.9 (2.49)	14.0 (3.16)	12.3 (1.79)
Daughter	0.8 (.01)	1.9 (.57)	1.7 (.67)	2.3 (1.17)	0.9 (.41)
Sister	10.9 (.34)	6.7 (1.02)	15.9 (2.81)	9.6 (2.58)	10.1 (1.64)
Paternal half sister	0.3 (.05)	0.2 (.09)	1.6 (.64)	2.9 (1.70)	1.0 (.53)
Paternal aunt	3.1 (.17)	7.8 (1.38)	12.0 (1.83)	11.9 (2.83)	14.6 (1.98)
Paternal grandmother	0.9 (.10)	2.7 (.69)	6.6 (1.72)	3.5 (1.86)	3.01 (.93)
Maternal half sister	0.2 (.04)	1.6 (.51)	3.0 (.94)	2.7 (1.84)	1.93 (.84)
Maternal aunt	3.4 (.20)	7.3 (1.18)	12.6 (2.1)	13.2 (3.30)	12.4 (1.88)
Maternal grandmother	1.4 (.13)	4.6 (.99)	6.2 (1.35)	2.4 (1.45)	6.03 (1.29)
Any first-degree female	6.6 (.26)	14.9 (1.52)	29.2 (3.18)	21.2 (3.60)	22.8 (2.32)
Any second-degree female	8.0 (.28)	17.3 (1.64)	33.6 (2.79)	28.9 (4.34)	28.5 (2.62)
Any first-degree relative	25.6 (.47)	41.6 (2.22)	59.9 (3.14)	55.4 (4.75)	60.0 (2.90)
Any second-degree relative	31.8 (.48)	54.1 (2.27)	70.4 (2.83)	72.5 (3.90)	67.6 (2.94)
Any relative	43.4 (.55)	67.1 (1.96)	79.9 (2.46)	84.4 (3.14)	79.7 (2.26)

Note: SE values appear in parentheses.

Rates of alcoholism among relatives

The rates of alcoholism among relatives are presented in Tables 3 and 4 for men and women, respectively. Within each of the four depression proband groups, there were no statistically significant differences in the rates of alcoholism between male and female first- and second-degree relatives of each type.

Among men, the rates of alcoholism among the first- and second-degree relatives of the MDD only group were significantly greater than those of the normal control group, with the exception of paternal half-siblings and offspring. That is, rates of alcoholism were not different among paternal half-brothers, paternal half-sisters, sons, and daughters of probands with MDD only compared with normal controls. This result was consistent for women, with the exception that there were no significant differences in the rates of alcoholism among maternal half-siblings and daughters (but not sons) between the MDD only and control group.

When male and female probands of each of the three comorbid groups were compared separately with the men and women in the normal control group, the rates of alcoholism were greater among first- and second-degree relatives of the comorbid probands, but this was not the case consistently for sons or daughters and certain half-siblings. Compared with the normal control group, rates of alcoholism among paternal half-siblings and maternal half-brothers were not elevated in primary depressives, rates among maternal and paternal half-siblings were not elevated in concurrent depressives, and rates among paternal half-siblings and maternal half-sisters were not elevated in secondary depressives.

There were no significant differences in the rates of alcoholism among first- and second-degree relatives of male and female probands across all comorbid groups. However, some differences in the rates of alcoholism among first- and second-degree relatives did emerge when the MDD only group was compared with each

Table 4. Rates of Alcohol Use Disorders among Relatives of Female Probands

Relative	Control no major depression or alcohol use disorder (0) (N = 20,292)	Major depression only (1) (N = 1,931)	Primary depression (2) (N = 383)	Concurrent depression (3) (N = 130)	Secondary depression (4) (N = 296)
Male Relatives					
Father	16.9 (.32)	29.5 (1.17)	41.8 (2.67)	39.4 (4.58)	42.7 (3.06)
Son	4.3 (.18)	6.9 (.68)	6.5 (1.58)	5.5 (2.30)	6.3 (1.65)
Brother	14.4 (.29)	20.5 (.99)	29.6 (2.62)	33.5 (4.46)	28.4 (2.85)
Paternal half brother	1.2 (.09)	2.3 (.39)	3.3 (1.10)	3.1 (1.58)	2.3 (.90)
Paternal uncle	13.2 (.28)	21.6 (1.06)	27.2 (2.55)	37.0 (4.72)	28.4 (2.93)
Paternal grandfather	7.3 (.23)	15.2 (.96)	23.0 (2.30)	29.9 (4.88)	25.1 (2.76)
Maternal half brother	1.1 (.09)	2.0 (.36)	3.3 (.81)	5.5 (2.37)	2.4 (.93)
Maternal uncle	14.7 (.30)	25.2 (1.18)	30.0 (2.51)	27.9 (4.33)	30.2 (3.02)
Maternal grandfather	8.5 (.24)	18.9 (.99)	18.4 (2.22)	28.6 (5.03)	31.4 (3.23)
Any first-degree male	27.8 (.37)	43.8 (1.25)	59.8 (2.59)	57.1 (5.09)	56.6 (3.13)
Any second-degree male	31.8 (.40)	54.1 (1.38)	65.3 (2.61)	71.9 (4.27)	64.9 (3.09)
Female Relatives					
Mother	4.1 (.17)	8.43 (.66)	20.5 (2.43)	22.3 (3.91)	19.2 (2.65)
Daughter	1.6 (.10)	2.6 (.45)	3.2 (.97)	3.9 (1.97)	3.4 (1.19)
Sister	4.9 (.18)	9.5 (.75)	20.4 (2.37)	20.0 (3.98)	17.2 (2.74)
Paternal half sister	0.4 (.05)	1.0 (.23)	1.4 (.52)	.0 (.00)	3.1 (1.16)
Paternal aunt	4.3 (.17)	7.9 (.66)	14.6 (2.05)	16.0 (3.59)	15.6 (2.57)
Paternal grandmother	1.5 (.10)	3.4 (.54)	7.6 (1.43)	8.8 (2.58)	8.7 (1.75)
Maternal half sister	0.4 (.05)	1.7 (.39)	2.7 (.82)	1.4 (.98)	1.8 (.67)
Maternal aunt	4.9 (.17)	10.3 (.78)	17.2 (2.17)	16.3 (3.67)	17.7 (2.59)
Maternal grandmother	1.8 (.49)	4.3 (.49)	11.3 (1.96)	7.9 (2.27)	6.7 (1.66)
Any first-degree female	9.5 (.26)	17.7 (.96)	37.2 (2.95)	34.7 (4.61)	34.5 (3.42)
Any second-degree female	11.1 (.26)	22.2 (1.05)	40.2 (2.92)	34.3 (4.60)	37.0 (3.14)
Any first-degree relative	31.9 (.39)	50.6 (1.23)	69.2 (2.61)	68.2 (4.78)	64.9 (3.15)
Any second-degree relative	35.6 (.41)	60.2 (1.38)	75.3 (2.36)	74.6 (4.09)	72.8 (2.98)
Any Relative	49.1 (.42)	73.8 (1.22)	87.2 (1.78)	88.3 (3.05)	85.0 (2.53)

Note: SE values appear in parentheses.

of the three comorbid groups. These results are shown in Table 5. With few exceptions (primarily mothers and fathers), the rates of alcoholism among male and female first- and second-degree relatives were not significantly different between the probands with MDD only and the three comorbid groups. This was the case when the rates of alcoholism were compared for specific relative types (*e.g.*, brothers, sisters, sons). However, when the rates of alcoholism were aggregated to the levels of any male or female first- or second-degree relative or any relative, the comorbid groups were shown to have consistently greater rates of alcoholism than the MDD only group for both men and women (Table 5).

DISCUSSION

The sex of the proband was not associated with any significant differences in familial aggregation of alcoholism within each depression proband. This result was at variance with the findings of Merikangas et al. (1985a) who found higher rates of alcoholism among the first-degree relatives of male compared with female probands with MDD only and with primary depression.

The results of this study showed significantly greater aggregate rates of alcoholism among first-and second-degree relatives of male and female probands with MDD only compared with normal controls. This result did not confirm those of previous studies that did not find rates of alcoholism increased among the first-degree relatives of the MDD only group compared with controls (Gershon et al., 1982; Merikangas et al., 1985a; Weissman et al., 1984). Thus, at the aggregate level of first- and second-degree relatives among male and female probands, this finding suggests that alcoholism and major depression may be alternate manifestations of the same underlying disorder. However, the findings become mixed when specific types of relatives were compared. Specifically, male probands with MDD only were no more likely to transmit alcoholism to their sons and daughters and female probands with MDD only were no more likely to transmit alcoholism to their daughters than were normal controls. Although the finding of increased rates of alcoholism

Table 5. Comparisons of Rates of Alcoholic Relatives between the Major Depression Only Proband versus Primary, Concurrent, and Secondary Depression Probands by Sex of Proband and Relative

Relative	Major depression only versus primary depression		Major depression only versus concurrent depression		Major depression only versus secondary depression	
Male relatives	Men	Women	Men	Women	Men	Women
Father	*	*	*	NS	*	*
Son	NS	NS	NS	NS	NS	NS
Brother	NS	*	NS	NS	*	NS
Paternal half brother	NS	NS	NS	NS	NS	NS
Paternal uncle	NS	NS	NS	*	NS	NS
Paternal grandfather	NS	*	NS	NS	NS	NS
Maternal half brother	NS	NS	NS	NS	NS	NS
Maternal uncle	*	NS	NS	NS	NS	NS
Maternal grandfather	*	NS	*	*	NS	*
Any first-degree male	*	*	*	NS	*	*
Any second-degree male	*	*	*	*	*	*
Female relatives						
Mother	*	*	NS	*	NS	*
Daughter	NS	NS	NS	NS	NS	NS
Sister	*	*	NS	NS	*	NS
Paternal half sister	NS	NS	NS	NS	NS	NS
Paternal aunt	NS	*	NS	NS	NS	NS
Paternal grandmother	NS	NS	NS	NS	NS	NS
Maternal half sister	NS	NS	NS	NS	NS	NS
Maternal aunt	NS	*	NS	NS	NS	NS
Maternal grandmother	NS	*	NS	NS	NS	NS
Any first-degree female	*	*	NS	*	*	*
Any second-degree female	*	*	NS	NS	*	*
Any relative	*	*	*	*	*	*

* = significant χ^2, $p<.005$.
NS = not significant, χ^2, $p>.005$.

in sons of female probands with MDD is consistent with the hypothesis that alcoholism and major depression are expressions of the same underlying disorder, the absence of increased rates of alcoholism among daughters of female probands and the offspring of male probands with MDD only compared with controls is not.

Although the rates of alcoholism among relatives of male and female probands were generally greater for the three comorbid groups compared with controls, no significant differences arose between these groups when sons and daughters were compared separately. There were fewer differences found in the rates of alcoholism between the relatives of the comorbid groups and the MDD only group when individual relative types were compared. Thus, the male and female probands of all three comorbid groups did not convey a greater risk of alcoholism to their offspring compared with the normal control group or the MDD only group. This result was consistent with previous research in which proband samples were ascertained for alcoholism (Hasegawa et al., 1991; O'Sullivan et al., 1983), but it is inconsistent with past research among depressed probands (Leibenluft et al., 1993; Merikangas et al., 1985 a and b; Woodruff et al., 1973). Thus, probands with MDD only were no more or less likely to transmit alcoholism than were comorbid probands with depression and alcoholism, one further result that provides support for the hypothesis that alcoholism and depression are merely alternate manifestations of the same underlying disorder. Therefore, one cannot conclude on the basis of these results that rates of alcoholism among relatives of all three comorbid probands can be accounted for by the presence of alcoholism in the proband.

This study, in part, was premised on the belief that important sources of variation in the transmission of alcoholism may have been obscured in previous studies by subsuming probands with MDD only, with primary, concurrent, and secondary depression into a single diagnostic category. However, what we did learn was that prior familial aggregation studies conducted in clinical samples were simply too small to allow for comparisons among each specific relative type across diagnostic categories. In fact, when rates were examined in aggregated family history measures (*e.g.*, among first-degree relatives or among second-degree relatives), significant variations in the rates of alcoholism were found in this study that were consistent with previous studies. However, when rates of alcoholism were examined separately for the 18 specific relative types, such variations in the rates of alcoholism largely disappeared.

This study was useful in establishing the relevance of the primary-secondary distinction as it relates to major depression and alcoholism in studies of familial aggregation. However, much more research that uses samples of sufficient size to examine the transmission of alcoholism among specific relative types is needed to confirm the results of this study. Future research should also focus on appropriate and unbiased sample selection procedures in both familial aggregation and genetic studies in the alcohol field. Most important is the critical need for longitudinal research that would be less subject to recall bias and more conducive to studying the chronology of alcohol use disorders and major depression, preferably beginning before the age of risk for these disorders (*i.e.,* during adolescence).

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CHAPTER 6
ALCOHOL AND SMOKING

Drinking as a Risk Factor for Sustained Smoking

Deborah A. Dawson

Data from a nationally representative sample of U.S. adults (n = 42,862) were used to explore the associations between drinking and smoking, on a lifetime and past-year basis, and between drinking and smoking cessation for a subsample of past-year smokers (n = 12,586). Smoking cessation was defined as current nonsmoking (as of the time of interview) that had lasted at least 3 months. The proportions of both lifetime and past-year smokers increased with volume of alcohol intake and frequency of heavy drinking and were greater for individuals with DSM–IV alcohol abuse or dependence than for individuals without these disorders. For example, the proportion of past-year smokers rose from 22.5% of lifetime abstainers to 53.0% of heavy drinkers, from 23.8% of those who never drank 5+ drinks on any drinking day to 61.8% of those who drank 5+ drinks weekly or more often and from 27.6% of individuals without past-year DSM–IV alcohol abuse or dependence to 55.5% of those with either of these disorders. The proportion of past-year smokers who had stopped smoking decreased from 7.8% of former drinkers to 4.6% of heavy drinkers, from 7.3% of those who never drank 5+ drinks to 3.4% of those who did so weekly or more often and from 6.7% among individuals without past-year abuse or dependence compared to 4.4% among those with either disorder. In a multivariate analysis that adjusted for background variables and smoking history, average daily ethanol intake was not significantly associated with the odds of smoking cessation, but drinking 5+ drinks at least once a month reduced the odds of cessation by 42%. Having an alcohol use disorder increased the odds of smoking cessation below the age of 30 (an effect that disappeared after adjusting for the interaction between age group and having children in the home), but had an increasingly negative effect on smoking cessation at older ages.

INTRODUCTION

More than 20 years of studies have shown a strong positive association between smoking and drinking in the United States. Much of this evidence has been drawn from studies of clinical samples of alcoholics, which have shown that persons with alcohol abuse and dependence are more likely to smoke and are heavier smokers than are members of the general population (see reviews in Bien and Burge, 1990; Bobo, 1992: Shiffman and Balabanis, 1995; Toneatto et al., 1995). Studies of adolescent substance use have confirmed that drinkers are more likely than nondrinkers to be smokers and vice versa and that the use of either one of these substances increases the risk of starting to use the other (Kandel and Yamaguchi, 1993; Torabi et al., 1993; Schorling et al., 1994; Chen and Kandel, 1995). In a longitudinal study of a small sample of college students, tobacco dependence and alcohol use disorders (i.e. abuse and dependence) each predicted the other over time (Sher et al., 1996). Finally, studies of the general adult population have confirmed an association between drinking and smoking. Craig and Van Natta (1977), who studied 1,089 residents of a single Maryland county in the early 1970's, found a modest association between alcohol and nicotine consumption that became stronger with increasing levels of drinking, and Friedman et al. (1991), who studied data on 13,673 patients receiving check-ups between 1979 and 1985, found that alcohol use was strongly related to the number of cigarettes smoked per day. In some but not all population subgroups, alcohol use also was associated with duration of smoking, time from arising to first cigarette, frequency and depth of inhalation and proportion of cigarette smoked. Similarly, laboratory studies with human subjects have described ethanol-associated increases in puff volume (Nil et al. 1984; Mintz et al., 1985) and decreases in intercigarette interval (Mello et al., 1987). Some of these studies have noted ethanol-induced increases in smoking only in response to high doses of ethanol or only among individuals whose previous alcohol intake levels were in the moderate to high range (see review in Zacny, 1990).

Several mechanisms have been proposed as underlying the association between smoking and drinking. One of these is cross-tolerance, i.e. the chronic use of one substance resulting in tolerance to the effects of

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another. Partial cross-tolerance to the effects of ethanol have been found in mice chronically infused with nicotine, and partial cross-tolerance to the effects of nicotine have been found in mice chronically treated with ethanol (Burch et al., 1988; Collins et al., 1988). These studies have suggested that cross-tolerance may be associated with ethanol-induced disruption of the membrane lipids that regulate nicotine receptor function (Collins, 1990). Other proposed mechanisms include the putative effects of ethanol on nicotine metabolism (Adir et al., 1980), the ability of each substance to counteract the aversive effects of the other, e.g. the stimulant effects of nicotine offsetting the performance deficits caused by excess ethanol intake (Zacny, 1990; Kerf et al., 1991), the disinhibiting effects of ethanol overcoming restraints against smoking (Shiffman et al., 1994), shared genetic predisposition and/or environmental influences (Swan et al., 1990), and cue reactivity and expectancies that foster the concurrent use of the two substances (Gulliver et al., 1995).

In addition to being correlated with current smoking, drinking bears a relationship with smoking cessation. A number of studies have found heavy levels of baseline drinking to be negatively associated with smoking cessation. A study of World War II veteran twins interviewed at two times 16 years apart found higher levels of baseline alcohol consumption in those who continued smoking than in those who quit (Carnelli et al., 1993). In a community sample of 2,115 adults, Zimmerman et al. (1990) found that heavy drinking was negatively associated with the likelihood of having attempted to quit smoking and with the likelihood of success among those who had tried; however, their measure of drinking level was at time of interview rather than at baseline. Based on data collected in 1975 and 1981 from members of the Finnish Twin Cohort, frequency of consuming spirits and frequency of consuming beer at baseline reduced the likelihood of smoking cessation (Kaprio and Koskenvuo, 1988).

Alcohol problems, recovery from alcohol problems and having stopped drinking also are associated with smoking cessation. DiFranza and Guerrera (1990) found that only 7% of alcoholic smokers successfully stopped smoking, compared to 49% of a control group of nonalcoholics, and a study of 235 smokers recovering from noncardiac surgery found that a history of alcohol abuse was positively associated with smoking relapse (Simon et al., 1992). In a study of problem drinkers receiving behavioral self control training, Miller et al. (1983) found that having stopped drinking increased the probability of smoking cessation. Similarly, in a study of Canadian adults, Sobell et al. (1995) found that the probability of having stopped smoking was lower for individuals with unresolved alcohol problems than for those whose alcohol problems were resolved. Interestingly, rates of smoking cessation were even higher among recovered problem drinkers who were drinkers in the past year than among those who were abstinent from alcohol.

Studies of samples from smoking cessation programs or of self-quitters have provided results consistent with those described above. In a study of a nicotine gum trial, Hughes (1993) reported lower 1-year quit rates among individuals with past alcohol or drug problems than among those without a history of alcohol or other drug use disorders (those with present alcohol or drug problems were excluded from the trial), and a study of 235 self-quitters recruited from newspaper advertisements found that heavier drinking at baseline was positively associated with relapse over a 1-year period (Garvey et al., 1992). Complementing these are studies of smoking relapse episodes that have implicated alcohol consumption as a risk factor for relapse (Shiffman, 1982; Baer and Lichtenstein, 1988).

Despite the large number of studies that have linked alcohol consumption, alcohol problems and smoking cessation, other studies have reported contradictory or mixed findings. Data collected over 6 years as part of the Multiple Risk Factor Intervention Trial indicated that baseline number of drinks per week was not associated with the likelihood of attempted smoking cessation but that it was positively associated with smoking relapse among those who attempted to quit (Hymowitz et al., 1991). In a study of 3,923 participants in the Lung Health Study's smoking cessation program, baseline number of drinks was negatively associated with the initial odds of quitting for women but not for men, and drinking was not associated with relapse for either men or women (Nides et al., 1995). In a study of 220 smokers enrolled in a smoking cessation program, recovering alcoholics had success rates similar to those for nonalcoholics, except for those with comorbid alcoholism and major depression, who had lower rates of success (Covey et al., 1993). Hughes and Oliveto (1993), reporting on one sample of 105 smokers receiving physician advice and placebo gum and another sample of 630 self quitters, stated that amount of baseline alcohol intake was not predictive of relapse in either study.

Many of the existing studies of the role of alcohol in smoking cessation have been based on samples of individuals who were either in smoking cessation programs or attempting to stop smoking on their own. These provide a limited view of the effect of alcohol on

smoking cessation, because they account only for the effects of alcohol on the success of a given attempt and not on its *overall* effect, which incorporates the probability of making an attempt in the first place. Other studies have been limited by inadequate measures of drinking patterns and alcohol-related problems. None have distinguished the effects of a heavy volume of ethanol intake from those of alcohol abuse or dependence or of episodic heavy drinking, despite the importance of these distinctions in attributing support to theories such as those of common genetic susceptibility, cross tolerance, cue reactivity and disinhibition. Moreover, few studies have considered the confounding effect of major depression, a condition that is highly comorbid with alcohol dependence (Grant and Harford, 1995) and for which nicotine may be a moderately effective form of self-medication.

This study represents an attempt to remedy those limitations. It is based on a nationally representative population sample of 42,862 adults, 12,586 of whom were past-year smokers. In addition to identifying past-year drinkers, former drinkers and lifetime abstainers from alcohol, it contains measures of ethanol intake volume, frequency of heavy drinking and alcohol abuse and dependence. Bivariate analyses demonstrate the associations of these various aspects of alcohol consumption with smoking and smoking cessation, on both a lifetime and past-year basis, and their effects are further distinguished in multivariate analyses that predict the odds of smoking cessation among past-year smokers, before and after adjusting for major depression, use of other drugs, aspects of smoking history, familial alcoholism and a host of other background variables.

METHOD

Sample

The data upon which this report is based were collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism. Interviewers hired and trained by the U.S. Bureau of the Census collected the data in personal interviews conducted in respondents' homes. The sample consisted of one adult 18 years of age or older selected at random from each household in a national sample of the civilian, noninstitutionalized population residing in the coterminous United States. The total sample size was 42,862, reflecting household and sample person response rates of 92 and 97%, respectively. The estimated proportions of lifetime and past-year smokers are based on the total sample. Characteristics and cessation rates of lifetime smokers are based on individuals who reported smoking at least 100 cigarettes during their lifetimes (n = 21,239), and characteristics and cessation rates of past-year smokers are further restricted to those who reported smoking any cigarettes in the 12 months preceding interview (n = 12,586). Statistics reported in this analysis were derived using SUDAAN (Shah et al., 1995), a software package that uses Taylor series linearization techniques to adjust variance estimates for characteristics (e.g. clustering, multiple stages of selection) of complex sample designs such as that used for the NLAES (Grant et al., 1994a).

Measures

For this analysis, lifetime smokers were defined as individuals who ever smoked at least 100 cigarettes, irrespective of the quantity and frequency with which they reported smoking. Past-year smokers were defined as individuals who ever smoked at least 100 cigarettes and who reported having smoked *any* cigarettes in the year preceding interview, again irrespective of quantity and frequency of smoking. Individuals were asked directly whether they had smoked on a daily basis in the past year and at any point during their lives as well as the number of cigarettes per day that they currently or formerly smoked. In the multivariate analyses, frequency was dichotomized as daily versus nondaily smoking, with the great majority of smokers in the former category. Because a preliminary analysis of the data showed that the probability of smoking cessation formed a u-shaped curve with respect to number of cigarettes smoked, quantity was represented by two dummy variables in the multivariate analyses: smoking less than ten cigarettes per day and smoking 40 or more cigarettes per day. Duration of smoking was estimated as the difference in years between age at interview and the age when the respondent first smoked his or her first full cigarette. In the multivariate analyses, duration of smoking was represented by two variables, duration and duration squared, because of its curvilinear association with the odds of stopping smoking.

The measure of smoking cessation used in this analysis was based on responses to a question that asked "When was the *last* time you smoked a cigarette?" and was defined as an interval of 3 months or longer since last cigarette. Thus, individuals were counted as having stopped smoking if they were nonsmokers at the time of interview and had not smoked for at least 3 months. Past research has indicated that most individuals will relapse within 3 months (Hunt et

al., 1971), making this a justifiable cutpoint to use for "successful" cessation—especially for those analyses based on past-year smokers, who had to have reported at least some smoking in the past year.

In this analysis, past-year drinkers were defined as individuals who had consumed at least 12 alcoholic drinks in the 12 months preceding interview. Former drinkers were defined as individuals who had consumed at least 12 alcoholic drinks in any one 12-month period but not in the 12 months preceding interview, and lifetime abstainers were defined as individuals who never had consumed 12 alcoholic drinks in any given year.

Average daily ethanol intake was measured for two periods, the past year and the period of heaviest drinking. Period of heaviest drinking was ascertained by asking respondents whether they had ever drunk more than in the past year and, if so, when that period began and how long it lasted. Within each of these periods, volume of ethanol intake was estimated from beverage-specific questions on frequency and quantity of drinking and typical drink size. Average daily ethanol intake was derived by dividing the annualized volume by 365, where annualized volume was the sum over beer, wine and liquor of the following: [(number of drinking days per year$_{\text{usual-heavy}}$) (number of drinks per drinking day$_{\text{usual}}$) (size of drink in ounces$_{\text{usual}}$) (ethanol conversion factor) + (number of drinking days per year$_{\text{heavy}}$) (number of drinks per drinking day$_{\text{heavy}}$)(size of drink in ounces$_{\text{heavy}}$) (ethanol conversion factor)]. Ethanol conversion factors were defined as 0.045 for beer, 0.121 for wine and 0.409 for liquor (DISCUS, 1985; Kling, 1989; Turner, 1990; Modern Brewery Age, 1992; Williams et al., 1993). For the period of heaviest drinking, average daily ethanol intake was assumed to be zero ounces for lifetime abstainers; for the past year, average daily ethanol intake was assumed to be zero ounces for both former drinkers and lifetime abstainers.

Frequency of heavy drinking was defined as the annual number of occasions of drinking five or more (5+) drinks and was measured only for the year preceding interview. Respondents were offered a range of categorical responses to the question on how often they drank 5+ drinks (every day, nearly every day, 3–4 days a week... 1 day in the past year, no days in the past year), and the midpoints of these categories were converted to numbers of days per year. Former drinkers and lifetime abstainers were assumed to have no occasions of drinking 5+ drinks in the past year. For use in the multivariate analyses, this measure of frequency was dichotomized into 12+ times per year (based on the categories of "once a month" through "every day") versus 0–11 times (based on the categories of "no days in the past year" through "7–11 days in the past year"), to reduce the correlation (r = 0.71) between the continuous measure and volume of ethanol intake.

Alcohol abuse and dependence were defined in accordance with the most recent DSM–IV criteria for these disorders (American Psychiatric Association, 1994) and were measured on both a past-year and lifetime basis using the AUDADIS (Grant et al., 1994b, 1995), a structured interview schedule designed for administration by lay interviewers and embedded in the NLAES questionnaire. To be classified with alcohol dependence, an individual had to meet *three or more* of the seven DSM–IV criteria for dependence: (1) tolerance; (2) withdrawal (including relief or avoidance of withdrawal); (3) persistent desire or unsuccessful attempts to cut down on or stop drinking; (4) much time spent drinking, obtaining alcohol or recovering from its effects; (5) reduction or cessation of important activities in favor of drinking; (6) impaired control over drinking; and (7) continued use despite physical or psychological problems caused by drinking. To be classified with alcohol abuse, an individual had to satisfy *one or more* of the four DSM–IV criteria for abuse: (1) continued use despite social or interpersonal consequences; (2) hazardous use; (3) alcohol-related legal problems; and (4) neglect of role responsibilities in favor of drinking.

In determining whether or not the requisite numbers of criteria were satisfied, the following steps were taken: For past-year diagnoses of alcohol abuse and dependence, each criterion not associated with duration qualifiers was satisfied if an individual reported one or more positive symptoms of that criterion during the past year. Criteria with duration qualifiers were satisfied if a person reported two or more symptoms during the past year or one symptom that occurred at least two times during the past year. To be consistent with the syndromal definition of the withdrawal criterion, two or more positive symptoms were required in addition to satisfaction of the duration qualifier. For prior-to-past-year diagnoses, criteria were satisfied if a person reported that one or more positive symptoms (two or more symptoms for withdrawal) occurred during the period prior to the past year. In order to satisfy the duration qualifiers for the period prior to the past year, respondents additionally had to report that some of the symptoms occurred at "around the same time" *or* "at around the same time, on and off for a few months or longer" *or* "at around the same time, most days for at least a month." A positive response to one of these

questions also ensured that the symptoms were clustered in time, increasing the likelihood that the required number of criteria were satisfied during a single point in time rather than occurring at different times over the life span. A lifetime diagnosis of alcohol abuse or dependence was obtained by satisfying the conditions for that disorder during either the past year or the period prior to the past year or both. Collectively, alcohol abuse and dependence are referred to as "alcohol use disorders" throughout this paper. This term should not be construed to extend to other phenomena such as alcohol withdrawal or intoxication.

Respondents were determined to have a positive family history of alcoholism if they reported that *any* of 18 different types of first- and second-degree biological relatives were alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM–IV criteria for alcohol use disorders: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover."

To be classified with lifetime DSM–IV major depression, a respondent had to report five or more depressive symptoms, inclusive of low mood or lack of interest, that occurred nearly every day for at least the same two-week period, that co-occurred with social and or occupational dysfunction, and that were not attributed to physical illness or bereavement.

Individuals were counted as having used illicit drugs in the past year if they reported having used *any* of the following at least 12 times on their own (i.e. without or beyond the limits of a doctor's prescription) during the 12 months preceding interview: sedatives, tranquilizers, painkillers, stimulants, marijuana or hashish, cocaine or crack, heroin, methadone or "other" drugs, e.g. inhalants and hallucinogens. They were counted as having experienced a potentially smoking-related illness if they reported having had any of the following conditions in the 12 months preceding interview: high cholesterol, diabetes, arteriosclerosis, high blood pressure, angina pectora, tachycardia, myocardial infarction, any other form of heart disease, a stroke or other cerebrovascular accident, emphysema or cancer. From a question that asked for the number of past-year overnight hospitalizations for various reasons, respondents were coded positively for pregnancy-related hospitalization if they reported one or more stays related to a normal pregnancy or complications of pregnancy and positively for illness-related hospitalization if they reported one or more stays for treatment of a physical condition, accident or injury, for an operation or surgical procedure, for diagnostic tests or for a mental or emotional problem.

Age, sex and gender were ascertained from self-report. Race was dichotomized as black versus all others. Hispanic origin was determined on the basis of self-reported origin or descent, using one open-ended and 58 precoded response categories (e.g. Mexican, Mexican-American, Central American, etc.). Individuals coded as married included those who reported living with someone as if married. Education was recorded in categories and recoded to college graduates (completion of 4-year college) or higher versus all others. Income was coded from the midpoints of categories representing monthly family income (less than $550–$13,000 or more), which were multiplied times 12 to annualize them. Individuals were coded as employed if they reported working at a job or business (including unpaid work in a family business or farm) at *any* time during the 12 months preceding interview and were coded as having children in the home if their household rosters included biological, adoptive, step or foster children 18 years of age or younger. Total body water was calculated on the basis of each individual's reported height, weight, age and gender (Moore et al., 1963).

Analysis

The first stage of the analysis studied the simple bivariate associations between drinking and smoking, for both the lifetime and past-year time frames. Pairwise differences in proportions and means were tested by means of *t*-tests. In general, only differences that were significant at the $P < 0.001$ level were cited when describing the results of these bivariate analyses, because of the large number of pairwise comparisons that were performed. (When differences with P values of 0.001 to 0.05 were discussed, they were described as being of marginal significance.)

In the second stage of the analysis, a hierarchical series of multiple logistic regression models was calculated to examine the effects of drinking upon smoking cessation after accounting for the potentially confounding effects of background and smoking history variables. These models estimated the odds of smoking cessation among past-year smokers. This analysis was restricted to past-year smoking cessation because of the dynamic nature of many of the independent variables. Insofar as there was no information on dates of changes in drinking status, marital status, edu-

cation and occupational status and so forth, it would have been difficult to draw any inferences as to the direction of causality regarding lifetime associations between these factors and smoking cessation.

The first model included only those background variables that were hypothesized to affect the odds of smoking cessation: age group, sex, race, and ethnicity (because of demographic variations in the prevalence of smoking), total body water (because it might mediate both the effects of ethanol and nicotine), education (because it might affect understanding of health risks), income (because individuals with higher incomes could more easily afford to continue smoking or, alternatively, to afford nicotine patches, gum and other aids to smoking cessation), employment status (because of smoking bans in many workplaces), marital status and presence of children in the home (because concerns over the effects of second-hand smoke might promote smoking cessation), past-year major depression (because nicotine might be used for its antidepressant qualities), past-year illicit drug use (because it might be associated with smoking in much the same way as alcohol), and past-year smoking-related illness, hospitalization for any illness/testing, and hospitalization related to pregnancy or delivery (because all of these might provide a strong medical indication for smoking cessation). Smoking-related variables—frequency, quantity and duration of smoking—were added in the second stage of the model-building process. Drinking variables—average daily volume of ethanol intake, frequency of heavy drinking, former drinker status (to distinguish former drinkers from lifetime abstainers among those whose past-year volumes of intake were zero) and past-year alcohol abuse or dependence—were added in the third stage of model building. In the final stage of model building, interactions between the drinking and other variables were tested for significance. Because of the large number of interactions tested, a significance level of $P < 0.001$ was required for interaction terms to be considered as definitive effect modifiers, whereas those with P values of 0.001 to 0.05 were considered to be of marginal significance.

Four measures of fit were calculated for these models. Sattersthwaite adjusted F (Shah et al., 1995) tests the model with all covariates against the model with the intercept only. The adjusted R^2 is based on the ratio of the −2 log likelihood estimates for the full and intercept-only models. The lack of fit chi square compares the observed and predicted outcomes. For both this measure and the percentage of cases predicted concordantly, the predicted outcome is positive or negative based on comparison of the predicted probability of smoking cessation (derived from the model-based logit) with the population prevalence of cessation.

Item nonresponse for the smoking, drinking and background variables was < 1% in most cases. The highest item nonresponse was for volume of ethanol intake during period of heaviest drinking (5%). Individuals with missing data on the drinking or smoking variables were removed from the bivariate analyses, and those with missing data on any variables, including background characteristics, were removed from the multivariate models.

RESULTS

Smoking and drinking on a lifetime basis

As shown in Table 1, half (49.9%) of U.S. adults 18 years of age and over were lifetime smokers. Among lifetime smokers, 92.7% were at some point daily smokers, and they smoked an average of approximately one pack of cigarettes per day. Almost half (44.1%) of all lifetime smokers were current nonsmokers who had stopped smoking for at least 3 months as of the time they were interviewed. The lifetime estimates of smoking and smoking cessation were strongly related to lifetime drinking history. The prevalence of lifetime smoking was lowest (28.2%) among lifetime abstainers from alcohol, more than twice as high (59.9%) among past-year drinkers and highest (63.3%) among former drinkers. Among individuals who were lifetime drinkers, the prevalence of lifetime smoking rose from 49.1% among those who were light drinkers during their period of heaviest consumption to 72.9% of those who were heavy drinkers. More than two thirds (68.0%) of individuals with lifetime alcohol abuse or dependence ever smoked, compared to less than half (45.8%) of those without these disorders.

Among lifetime smokers, those who were former drinkers and those who were heavy drinkers were the heaviest smokers, in terms of both quantity and frequency of smoking. Compared to individuals without a history of alcohol use disorders, those with lifetime alcohol abuse or dependence smoked more cigarettes per day, but their increased likelihood of daily smoking was only of marginal significance. Even when significant, differences in quantity and frequency of smoking among lifetime smokers were relatively small. Smoking cessation rates for lifetime smokers were highest (52.6%) among former drinkers, next highest (46.2%) among lifetime abstainers from alcohol and lowest (38.9%) among past-year drinkers. Smoking cessation

rates were lower for heavy drinkers (38.8%) than for those who drank at lower volumes or were lifetime abstainers (between 45.8 and 49.8%) and lower for those with lifetime alcohol abuse or dependence (33.8%) than for those without these disorders (47.4%).

Relationships among lifetime drinking measures

As indicated in Table 2, lifetime drinking status and volume of ethanol intake during period of heaviest consumption were strongly associated with the preva-

Table 1. Lifetime smoking characteristics of U.S. adults 18 years of age and older, according to history of alcohol use disorders and alcohol intake during period of heaviest consumption[a]

	N	% Of adults who were lifetime smokers	Characteristics of lifetime smokers		
			% Who were daily smokers	Mean cigarettes smoked per day	% Current non-smokers for at least 3 months
Total	42 862[b]	49.9 (0.3)	92.7 (0.2)	18.9 (0.1)	44.1 (0.4)
Lifetime drinking status:					
Past-year drinker	18 352	59.9 (0.4)	92.1 (0.3)	18.7 (0.2)	38.9 (0.6)
Former drinker	9264	63.3 (0.6)	94.5 (0.3)	21.1 (0.2)	52.6 (0.7)
Lifetime abstainer	15 246	28.2 (0.5)	91.7 (0.5)	16.2 (0.2)	46.2 (0.9)
t-test statistics for pairwise differences:					
Past-year drinker vs. former drinker		−4.8, $P<0.001$	−5.4, $P<0.001$	−7.8, $P<0.001$	−15.3, $P<0.001$
Past-year drinker vs. lifetime abstainer		51.4, $P<0.001$	0.6, $P=0.529$	8.1, $P<0.001$	−7.3, $P<0.001$
Former drinker vs. lifetime abstainer		46.5, $P<0.001$	4.7, $P<0.001$	14.8, $P<0.001$	5.6, $P<0.001$
Alcohol intake during period of heaviest consumption:[c]					
Heavy drinker	8983	72.9 (0.5)	94.7 (0.3)	21.9 (0.3)	38.8 (0.7)
Moderate drinker	9014	58.9 (0.6)	91.8 (0.4)	17.9 (0.2)	45.8 (0.8)
Light drinker	7457	49.1 (0.7)	91.1 (0.5)	17.4 (0.3)	49.8 (1.0)
Lifetime abstainer	15 246	28.2 (0.5)	91.7 (0.5)	16.2 (0.2)	46.2 (0.9)
t-test statistics for pairwise differences:					
Heavy drinker vs. moderate drinker		16.8, $P<0.001$	5.3, $P<0.001$	12.4, $P<0.001$	−6.7, $P<0.001$
Heavy drinker vs. light drinker		28.3, $P<0.001$	5.6, $P<0.001$	12.5, $P<0.001$	−9.3, $P<0.001$
Heavy drinker vs. lifetime abstainer		60.1, $P<0.001$	4.9, $P<0.001$	15.7, $P<0.001$	−6.8, $P<0.001$
Moderate drinker vs. light drinker		10.5, $P<0.001$	1.0, $P=0.310$	1.2, $P=0.217$	−3.2, $P<0.002$
Moderate drinker vs. lifetime abstainer		41.1, $P<0.001$	0.2, $P=0.851$	5.4, $P<0.001$	−0.4, $P=0.707$
Light drinker vs. lifetime abstainer		25.6, $P<0.001$	−0.8, $P=0.420$	3.5, $P=0.001$	2.8, $P=0.007$
History of lifetime DSM-IV alcohol use disorder:					
Lifetime abuse or dependence	7359	68.0 (0.7)	93.5 (0.4)	20.7 (0.3)	33.8 (0.8)
No lifetime abuse or dependence	35 503	45.8 (0.4)	92.4 (0.3)	18.3 (0.1)	47.4 (0.5)
t-test statistics for pairwise differences:					
Lifetime abuse/dependence vs. none		29.9, $P<0.001$	2.2, $P=0.029$	7.4, $P<0.001$	−15.0, $P<0.001$

[a] Note: figures in parentheses are standard errors of estimates.

[b] Total *N* includes individuals with unknown levels of alcohol intake during period of heaviest consumption.

[c] Heavy drinkers had an average daily ethanol intake of more than 1.0 oz., moderate drinkers of 0.23–1.0 oz., light drinkers of <0.23 oz., lifetime abstainers had less than 12 drinks in any year.

Table 2. Percentage of U.S. adults 18 years of age and older with lifetime DSM-IV alcohol abuse or dependence, according to lifetime drinking status and alcohol intake during period of heaviest consumption[a]

	N	% With lifetime abuse or dependence
Total	42 862[b]	18.2 (0.3)
Lifetime drinking status:		
Past-year drinker	18 352	29.8 (0.5)
Former drinker	9264	22.9 (0.6)
Lifetime abstainer	15 246	0.0 (0.0)
t-test statistics for pairwise differences:		
Past-year drinker vs. former drinker		9.1, $P<0.001$
Past-year drinker vs. lifetime abstainer		65.2, $P<0.001$
Former drinker vs. lifetime abstainer		38.6, $P<0.001$
Alcohol intake during period of heaviest consumption:[c]		
Heavy drinker	8983	53.5 (0.6)
Moderate drinker	9014	21.1 (0.6)
Light drinker	7457	6.1 (0.3)
Lifetime abstainer	15 246	0.0 (0.0)
t-test statistics for pairwise differences:		
Heavy drinker vs. moderate drinker		41.8, $P<0.001$
Heavy drinker vs. light drinker		70.8, $P<0.001$
Heavy drinker vs. lifetime abstainer		91.1, $P<0.001$
Moderate drinker vs. light drinker		23.4, $P<0.001$
Moderate drinker vs. lifetime abstainer		38.1, $P<0.001$
Light drinker vs. lifetime abstainer		17.8, $P<0.001$

[a] Note: figures in parentheses are standard errors of estimates.
[b] Total *N* includes individuals with unknown levels of alcohol intake during period of heaviest consumption.
[c] Heavy drinkers had an average daily ethanol intake of more than 1.0 oz., moderate drinkers of 0.23–1.0 oz., light drinkers of <0.23 oz., and lifetime abstainers had less than 12 drinks in any year.

lence of lifetime alcohol abuse and dependence. The proportion of individuals with an alcohol use disorder was 29.8% for past-year drinkers, compared to 22.9% for former drinkers. (Lifetime abstainers were not asked the questions used in the classification of these disorders and were assumed not to have satisfied the criteria for abuse or dependence.) Likewise, the prevalence of lifetime alcohol abuse or dependence rose from 6.1% among light drinkers to 21.1% of moderate and 53% of heavy drinkers.

Smoking and drinking in the past year

Less than one third (29.7%) of U.S. adults were past-year smokers (Table 3). Among these, 87.5% smoked on a daily basis during the past year, and the average number of cigarettes smoked per day was 18.1 or nearly one pack. The proportion of past-year smokers who were current nonsmokers as of the date when they were interviewed and who reported an interval of 3+ months since their last cigarette was 6.4%.

The prevalence of past-year smoking declined from 53.0% of heavy drinkers (based on past-year volumes of ethanol intake) to 39.7% of moderate drinkers, 30.7% of light drinkers and 22.5% of lifetime abstainers. The prevalence of past-year smoking among former drinkers (32.5%) was similar to that for light drinkers. Individuals with past-year alcohol abuse or dependence were twice as likely to have smoked in the past year as those without abuse or dependence (55.5 vs. 27.6%). Frequency of heavy drinking was even more strongly associated with the prevalence of past-year smoking, which rose from 23.8% of individuals who never consumed 5+ drinks in the past year to 61.8% of those who did so at least once a week, i.e. 52+ times during the year.

Past-year smokers with intermediate volumes of alcohol intake and intermediate frequencies of heavy drinking tended to be the least heavy smokers, both in terms of number of cigarettes smoked per day and prevalence of daily smoking, but many of these differences were of marginal statistical significance in addition to being of small magnitude. Individuals with past-year alcohol abuse or dependence were less likely to be past-year daily smokers than were those without these disorders (82.4 vs. 88.3%), but the quantity of cigarettes smoked per day did not vary according to the presence or absence of a past-year alcohol use disorder. The proportion of past-year smokers who had stopped smoking was inversely related to both volume of ethanol intake and frequency of heavy drinking, but only a few of the pairwise differences were statistically significant, e.g. the difference in cessation between heavy drinkers and former drinkers (4.6 vs. 7.8%) and the difference between those who drank 5+ drinks 52+ times and those who never drank 5+ drinks in the past year (3.4 vs. 7.3%). Rates of smoking cessation were lower for persons with past-year alcohol abuse or dependence (4.4%) than for those with neither of these disorders (6.7%).

Relationships among past-year drinking measures

As was true for the lifetime drinking measures, the past-year drinking measures—volume of intake, fre-

Table 3. Past-year smoking characteristics of U.S. adults 18 years of age and older, according to past-year alcohol use disorders, alcohol intake and frequency of drinking 5+ drinks[a]

	N	% Of adults who were past-year smokers	Characteristics of past-year smokers		
			% Who were daily smokers	Mean cigarettes smoked per day	% Current non-smokers for at least 3 months
Total	42 862[b]	29.7 (0.3)	87.5 (0.4)	18.1 (0.2)	6.4 (0.3)
Level of intake during past year:[c]					
Heavy drinker	3463	53.0 (0.3)	87.6 (1.1)	19.7 (0.5)	4.6 (0.5)
Moderate drinker	6956	39.7 (0.7)	82.1 (0.9)	16.7 (0.3)	5.6 (0.5)
Light drinker	7595	30.7 (0.6)	86.0 (0.8)	17.7 (0.4)	6.9 (0.6)
Former drinker	9264	32.5 (0.6)	91.5 (0.6)	20.4 (0.3)	7.8 (0.7)
Lifetime abstainer	15246	22.5 (0.3)	89.8 (0.7)	16.2 (0.3)	6.5 (0.7)
t-tests for pairwise differences:					
Heavy drinker vs. moderate drinker		10.5, $P<0.001$	3.8, $P<0.001$	5.8, $P<0.001$	−1.4, $P=0.153$
Heavy drinker vs. light drinker		19.5, $P<0.001$	1.2, $P=0.244$	3.7, $P<0.001$	−2.9, $P=0.005$
Heavy drinker vs. former drinker		18.3, $P<0.001$	−3.2, $P=0.002$	−1.2, $P=0.222$	−3.7, $P<0.001$
Heavy drinker vs. lifetime abstainer		34.7, $P<0.001$	−1.7, $P=0.096$	6.4, $P<0.001$	−2.3, $P=0.025$
Moderate drinker vs. light drinker		10.1, $P<0.001$	−3.2, $P<0.001$	−2.4, $P=0.021$	−1.7, $P=0.085$
Moderate drinker vs. former drinker		8.2, $P<0.001$	−8.6, $P<0.001$	−9.8, $P<0.001$	−2.7, $P=0.009$
Moderate drinker vs. lifetime abstainer		30.1, $P<0.001$	−6.4, $P<0.001$	1.3, $P=0.204$	−1.1, $P=0.283$
Light drinker vs. former drinker		−2.1, $P=0.036$	−5.5, $P<0.001$	−5.8, $P<0.001$	−1.1, $P=0.293$
Light drinker vs. lifetime abstainer		21.2, $P<0.001$	−3.6, $P=0.001$	3.2, $P=0.002$	0.5, $P=0.630$
Former drinker vs. lifetime abstainer		24.1, $P<0.001$	2.0, $P=0.052$	11.3, $P<0.001$	1.7, $P=0.102$
Frequency of drinking 5+ drinks during past year:					
52+ times	2118	61.8 (1.2)	87.1 (1.2)	19.8 (0.5)	3.4 (0.5)
12–51 times	2298	50.3 (1.2)	83.4 (1.4)	17.5 (0.5)	4.0 (0.6)
1–11 times	5291	40.6 (0.8)	83.7 (0.9)	17.5 (0.3)	6.5 (0.6)
Never	33 022	23.8 (0.3)	89.3 (0.4)	18.1 (0.2)	7.3 (0.4)
t-tests for pairwise differences:					
52+ times vs. 12–51 times		6.6, $P<0.001$	2.1, $P=0.039$	3.7, $P<0.001$	−0.8, $P=0.417$
52+ times vs. 1–11 times		14.5, $P<0.001$	2.2, $P=0.030$	4.0, $P<0.001$	−4.0, $P<0.001$
52+ times vs. never		31.7, $P<0.001$	1.7, $P=0.103$	3.4, $P=0.001$	−5.9, $P<0.001$
12–51 times vs. 1–11 times		6.4, $P<0.001$	−0.2, $P=0.828$	−0.1, $P=0.956$	−3.0, $P=0.004$
12–51 times vs. never		21.0, $P<0.001$	−4.1, $P<0.001$	−1.1, $P=0.282$	−4.6, $P<0.001$
1–11 times vs. never		19.8, $P<0.001$	−5.6, $P<0.001$	−1.3, $P=0.185$	−1.2, $P=0.253$
Past-year DSM-IV alcohol use disorder:					
Past-year abuse or dependence	2910	55.5 (1.1)	82.4 (1.2)	18.2 (0.5)	4.4 (0.5)
No past-year abuse or dependence	39 952	27.6 (0.5)	88.3 (0.4)	18.1 (0.2)	6.7 (0.3)
t-tests for pairwise differences:					
Past-year abuse/dependence vs. none		24.3, $P<0.001$	−4.7, $P<0.001$	0.1, $P=0.957$	−3.7, $P<0.001$

[a] Note: figures in parentheses are standard errors of estimates.

[b] Total *N* includes individuals with unknown levels of alcohol intake and/or frequency of drinking 5+ drinks during past year.

[c] Heavy drinkers had an average daily ethanol intake of more than 1.0 oz., moderate drinkers of 0.23–1.0 oz., light drinkers of <0.23 oz., and lifetime abstainers had less than 12 drinks in any year.

quency of heavy drinking and alcohol use disorders—were positively associated (Table 4). The proportion of past-year drinkers who met the criteria for past-year alcohol abuse or dependence rose from 4.5% of those who were light drinkers to 41.3% of those who were heavy drinkers and from 0.6% of those who never drank 5+ drinks to 54.3% of those who drank 5+ drinks once a week or more often (52+ times). Likewise, the proportion who drank 5+ drinks at least once a month (12+ times) increased from 5.4% of those with light volumes of intake to 63.8% of those with heavy volumes of intake. These strong patterns of association reinforce the need for multivariate analytic techniques to distinguish the independent contributions of each of the past-year drinking variables to the odds of smoking cessation.

Multivariate analysis of past-year smoking cessation

The results of the model containing only sociodemographic variables indicated that the odds of smoking cessation among past-year smokers were increased at ages 50 and over (OR = 1.42) among college graduates (OR = 1.86), among married individuals (OR = 1.38) and among persons who had been hospitalized for physical illness during the preceding year (OR = 1.81). The odds of smoking cessation also increased with rising volumes of total body water. The odds of

Table 4. Percentage of U.S. adults 18 years of age and older with past-year DSM–IV alcohol abuse or dependence and percentage who drank 5+ drinks 12+ times in the past year, by alcohol intake during past year and frequency of drinking 5+ drinks in past year[a]

	N	% With past-abuse or dependence	% Who drank 5+ drinks 12+ times in past year
Total	42 862[b]	7.4 (0.2)	11.4 (0.3)
Level of intake during past year:[c]			
Heavy drinker	3463	41.3 (1.1)	63.8 (1.0)
Moderate drinker	6956	17.1 (1.1)	28.0 (0.7)
Light drinker	7595	4.5 (0.3)	5.4 (0.3)
Former drinker	9264	0.0 (0.0)	0.0 (0.0)
Lifetime abstainer	15 246	0.0 (0.0)	0.0 (0.0)
t-tests for pairwise differences:			
Heavy drinker vs. moderate drinker		21.3, $P<0.001$	31.2 $P<0.001$
Heavy drinker vs. light drinker		33.8, $P<0.001$	56.3 $P<0.001$
Heavy drinker vs. former drinker		38.6, $P<0.001$	62.4, $P<0.001$
Heavy drinker vs. lifetime abstainer		38.6, $P<0.001$	62.4, $P<0.001$
Moderate drinker vs. light drinker		19.5, $P<0.001$	30.4, $P<0.001$
Moderate drinker vs. former drinker		28.4 $P<0.001$	38.4, $P<0.001$
Moderate drinker vs. lifetime abstainer		28.4, $P<0.001$	38.4, $P<0.001$
Light drinker vs. former drinker		15.9, $P<0.001$	17.4, $P<0.001$
Light drinker vs. lifetime abstainer		15.9, $P<0.001$	17.4, $P=0.001$
Former drinker vs. lifetime abstainer		NA	NA
Frequency of drinking 5+ drinks during past year:			
52+ times	2118	54.3 (1.3)	NA
12–51 times	2298	32.2 (1.2)	NA
1–11 times	5291	16.0 (0.6)	NA
Never	33 022	0.6 (0.1)	NA
t-tests for pairwise differences:			
52+ times vs. 12–51 times		13.2, $P<0.001$	NA
52+ times vs. 1–11 times		27.5, $P<0.001$	NA
52+ times vs. never		41.5, $P<0.001$	NA
12–51 times vs. 1–11 times		12.7, $P<0.001$	NA
12–51 times vs. never		26.8, $P<0.001$	NA
1–11 times vs. never		25.8, $P<0.001$	NA

[a] Note: figures in parentheses are standard errors of estimates.

[b] Total *N* includes individuals with unknown levels of alcohol intake and/or frequency of drinking 5+ drinks during past year.

[c] Heavy drinkers had an average daily ethanol intake of more than 1.0 oz., moderate drinkers of 0.23–1.0 oz., light drinkers of <0.23 oz., lifetime abstainers had less than 12 drinks in any year.

Table 5. Reduced logistic regression models predicting the odds of having stopped smoking for the past 3 months or longer among adults 18 years of age and over who smoked at some point during the past year

	Model containing sociodemographic variables only			Model adding smoking variables			Model adding drinking variables			Model adding drinking variables plus interactions		
	β	SE	*P*	β	SE	*P*	β	SE	*P*	β	SE	*P*
Main effects												
Intercept	−4.221	0.345	0.000	−4.494	0.390	0.000	−4.296	0.391	0.000	−4.396	0.395	0.000
Age 30–49	−0.171	0.102	0.099	−0.080	0.158	0.614	−0.101	0.157	0.523	0.047	0.164	0.774
Age 50+	0.354	0.120	0.004	−0.085	0.262	0.746	−0.137	0.262	0.603	0.010	0.264	0.969
Male	−0.503	0.157	0.002	−0.805	0.154	0.000	−0.704	0.154	0.000	−0.705	0.155	0.000
Black	−0.560	0.151	0.000	−0.637	0.151	0.000	−0.674	0.154	0.000	−0.669	0.155	0.000
Total body water	0.038	0.010	0.000	0.051	0.010	0.000	0.050	0.010	0.000	0.050	0.010	0.000
College graduate	0.618	0.108	0.000	0.586	0.111	0.000	0.547	0.112	0.000	0.549	0.112	0.000
Married	0.325	0.086	0.000	0.415	0.088	0.000	0.359	0.088	0.000	0.369	0.089	0.000
Employed	0.053	0.112	0.639	0.332	0.118	0.006	0.340	0.118	0.005	0.338	0.118	0.006
Major depression	−0.940	0.251	0.000	−0.957	0.271	0.001	−0.925	0.273	0.001	−0.950	0.273	0.001
Hospitalized for physical illness	0.595	0.136	0.000	0.587	0.140	0.000	0.580	0.141	0.000	0.584	0.141	0.000
Daily smoker				−0.346	0.151	0.024	−0.373	0.154	0.018	−0.370	0.155	0.019
Smoked <10 cigarettes/day				0.411	0.138	0.004	0.405	0.141	0.005	0.401	0.142	0.006
Smoked 40+ cigarettes/day				0.487	0.131	0.000	0.488	0.132	0.000	0.500	0.132	0.000
Duration of smoking				−0.042	0.015	0.007	−0.044	0.015	0.005	−0.044	0.015	0.006
Duration of smoking squared				0.001	<0.001	0.000	0.001	<0.001	0.000	0.001	<0.001	0.000
Average daily ethanol intake							−0.001	0.061	0.993	0.016	0.061	0.798
Frequent heavy drinker							−0.549	0.159	0.001	−0.583	0.164	0.001
Former drinker							0.152	0.106	0.158	0.147	0.107	0.173
Alcohol dependence/abuse							−0.023	0.170	0.892	0.447	0.216	0.043
Interaction terms												
Alcohol dependence/abuse × age 30–49										−1.043	0.324	0.002
Alcohol dependence/abuse × age 50+										−2.020	0.830	0.018
Goodness of fit statistics												
Sattersthwaite adjusted *F* (excluding intercept)[a]	12.9, df = 9.1, *P* = 0.00			16.9, df = 13.7, *P* = 0.00			15.3, df = 16.9, *P* = 0.00			15.3, df = 19.1, *P* = 0.00		
% of cases predicted concordantly	62.4			67.3			67.6			68.2		
Likelihood R^2	0.029			0.053			0.058			0.061		
Lack of fit chi square	11.98, df = 8, *P* = 0.15			7.73, df = 8, *P* = 0.46			8.93, df = 8, *P* = 0.35			3.70, df = 8, *P* = 0.88		

[a] Denominator degrees of freedom for all models = 68.

smoking cessation were reduced among men (OR = 0.60), among persons of black race (OR = 0.57) and among persons with one or more episodes of DSM–IV major depression in the past year (OR = 0.39). Neither being in the 30–49 year age group nor being employed was associated with the odds of smoking cessation in this model, but these variables did demonstrate significant associations in some of the subsequent models. Hispanic origin, income, presence of children, family history of alcoholism, past-year drug use, hospitalization related to pregnancy and smoking-related illness were unrelated to the odds of smoking cessation in this and all later models. Accordingly, these variables were omitted from presentation in Table 5.

The addition of the smoking variables in the second model considerably improved the fit of the model and changed the estimated effects of several of the background variables. There was a loss of statistical significance for the age group variables; being employed took on a significant positive effect (OR = 1.39); and the negative effect of male gender was almost doubled (OR = 0.45). In terms of the smoking variables themselves, daily smoking reduced the odds of smoking cessation among past-year smokers by almost 30% (OR = 0.71). Smoking less than 10 cigarettes per day (less than half a pack) or 40 or more cigarettes per day (two packs or more) both increased the odds of smoking cessation (OR = 1.51 and 1.63, respectively, compared to smoking 10–39 cigarettes per day). The two significant effects for duration of smoking (a negative effect for the linear measure and a positive effect for the quadratic measure) revealed that the odds of smoking cessation initially decreased but ultimately reversed to increase with duration. This pattern is revealed by the following pattern of odds ratios: 0.95 for a duration of 1 year, 0.73 for 10 years, 0.64 for 20 years, 0.69 for 30 years, 0.92 for 40 years, and 1.49 for 50 years.

The addition of the drinking variables in the third model did not cause significant changes in the values for any of the sociodemographic or smoking history variables. Compared to the addition of the smoking variables, the improvement in model fit attributable to adding the drinking variables was small. As was suggested by the earlier bivariate analyses, frequent heavy drinking demonstrated a negative effect on the odds of smoking cessation (OR = .58) among past-year smokers; however, after accounting for the frequency of heavy drinking, neither average daily ethanol intake, former drinker status nor the presence of an alcohol use disorder was significantly associated with the odds of smoking cessation among past-year smokers.

In the fourth-stage model, which tested for significant interactions between the alcohol and other variables, only two interaction terms were statistically significant. These were negative interactions between past-year alcohol abuse or dependence and the age groups 30–49 years and 50+ years. (Although neither of these individual terms was significant at the $P < 0.001$ level, their *combined* effect, representing the overall interaction between age and dependence, did attain that level of significance.) With the addition of these terms, the underlying effect of abuse or dependence, i.e. its effect at ages 18–29, was positive, although of marginal significance ($P = 0.043$). Thus, at ages 18–29, individuals with an alcohol use disorder were more likely than those with no disorder to have stopped smoking for at least 3 months (OR = 1.56, 95% CI = 1.03–2.38). At subsequent ages, the effects of alcohol use disorders were increasingly negative, with an odds ratio of 0.55 (95% CI = 0.30–0.93) at ages 30–49 and an odds ratio of 0.21 (95% CI = 0.05–0.98) at ages 50 or older.

DISCUSSION

This study confirmed, in a large sample of the general adult population, that lifetime and past-year smoking had positive bivariate associations with drinking status, volume of alcohol intake, frequency of heavy drinking and alcohol abuse and dependence. Multivariate analyses revealed that the effects of alcohol consumption and alcohol problems on smoking cessation were more complex. Among past-year smokers, drinkers who did not engage in frequent heavy drinking during the past year and who did not have a past-year alcohol use disorder were no more or less likely than nondrinkers to have stopped smoking. Those who did engage in frequent heavy drinking had a reduced probability of smoking cessation. The effect of alcohol abuse or dependence varied with age, ranging from a marginally significant positive effect at ages 18–29 to an increasingly stronger negative effect at older ages.

The strong effect of heavy drinking coupled with the lack of effect of lower levels of alcohol intake supported several other studies which found that ethanol-potentiated smoking was more likely to occur in heavier drinkers (see review in Zachny, 1990), as well as one laboratory study that reported increased puff volume following 0.7 g/kg of ethanol but not following lower doses of ethanol (Nil et al., 1984). These findings offer support for two of the mechanisms that have been proposed as explanatory factors in the association between smoking and drinking—the disinhibi-

tion theory and the theory of countervailing aversive effects. That is, smoking relapse might be especially likely to occur under the disinhibiting conditions of frequent heavy-drinking episodes, or, alternatively, heavy drinkers might continue smoking because they felt that smoking improved their performance (e.g. made them more alert or sociable) when intoxicated.

The negative effect of frequent heavy drinking also is consistent with the notion of cross tolerance between ethanol and nicotine, if one assumes that nicotine tolerance would reduce the probability of being able to stop smoking and that frequent heavy drinking may be an indicator of tolerance to the effects of alcohol. Cross tolerance based on a shared genetic susceptibility to tobacco and alcohol dependence (Swan et al., 1990) was not supported by this study, in that there was no significant effect of family history of alcoholism before or after accounting for the respondents' alcohol use disorders, but further research involving a more sophisticated measure of familial alcoholism is warranted before this possibility can be rejected.

The increasingly negative effect of alcohol abuse and dependence with advancing age that was found in this study probably reflects the fact that persons who remain dependent/abusers at later ages represent an increasingly select group with respect to failure to overcome their alcohol problems. That is, at each increasing year of age, individuals with alcohol use disorders become more heavily comprised of those who have tried and failed to recover from these disorders. It is not surprising that these individuals also would have a poorer chance of stopping smoking. The mechanisms underlying this increasing selectivity cannot be determined with certainty from these data but could include severity of alcohol problems (not likely as both the number of past-year symptoms and the prevalence of past-year tolerance declined rather than increased with age, data not shown), a generalized "addictive personality" characterized by lack of self control or disregard for risk and social disapproval, lack of access to treatment programs, poorer social support systems or some combination of factors such as these. In a related finding, the bivariate analyses indicated that individuals who had *stopped* drinking, nearly one quarter (22.9%) of whom had overcome prior alcohol use disorders, had the *highest* rates of smoking cessation. Former drinker status was not a significant predictor of smoking cessation in the multivariate models ($P = 0.173$), but it came closer to being of significance ($P = 0.072$) when the subgroup of former drinkers who had been dependent or abusers were distinguished from other former drinkers (data not shown).

The puzzling positive association between alcohol abuse and dependence and the odds of smoking cessation at the young adult ages of 18–29 led to exploration of the differences between individuals with and without alcohol use disorders in this age group. One of the most striking differences was in the percentage of individuals who reported children living in the home, ~50% for those without alcohol abuse or dependence and only 25% for those with an alcohol use disorder. Although the overall effect of children in the home had been found to be nonsignificant, subsequent modeling (data not shown) revealed a significant effect of children within the 18–29 year age group only. Contrary to the expectation that having young children might lead people to stop smoking because of concerns about second-hand smoke, the presence of children *reduced* the odds of smoking cessation (OR = 0.69) among individuals 18–29 years of age—possibly because nicotine was perceived as helping to alleviate the stresses associated with child rearing (a purely speculative interpretation). Moreover, when this factor was included in the model, the effect of alcohol abuse and/or dependence within the 18–29 year age group was no longer statistically significant. The negative effects of abuse and dependence at older ages did remain significant and of the same magnitude as already discussed.

As was expected, this study found that past-year major depression was associated with a large reduction in the odds of smoking cessation. This supports the argument that nicotine may be used as a form of self-medication for depressive symptoms and confirms findings of other studies that have demonstrated comorbidity between depression and nicotine dependence (Fergusson et al., 1996) and an adverse effect of major depression on attempts to stop smoking (Covey et al., 1993). Unlike the study by Covey and colleagues (1993), this study did not find an interaction between depression and alcohol dependence. When interactions with the drinking variables were tested, the interaction between alcohol abuse or dependence and major depression was indeed negative and of large magnitude, as was the case in their study, but it was not statistically significant ($P = 0.224$). It seemed likely that the present study's inclusion of frequency of heavy drinking in addition to alcohol use disorders might account for this disparity. That is, if depressive alcoholics had a lower probability of smoking cessation than other alcoholics because of being more frequent heavy drinkers, then adjusting for this frequency would remove this source of interaction between depression and alcohol use disorders. However, subsequent testing revealed that the frequency of heavy drinking among past-year smokers

with past-year alcohol abuse or dependence did not vary according to the presence or absence of major depression ($P = 0.486$, data not shown). Thus, sampling differences (e.g. the fact that the sample used by Covey et al. excluded individuals with either alcohol abuse or major depression in the 6 months preceding interview and was restricted to individuals with prior unsuccessful cessation attempts) may be most likely to account for the differences between the findings of the two studies.

This study was limited by lack of information on several potentially important variables, most notably nicotine dependence and its severity. Another important variable that could not be considered in this analysis is drinking context, most importantly whether drinking typically occurs in a smoking environment. Past research has indicated that the association of drinking with smoking relapse is especially strong in social situations involving other smokers (Baer and Lichtenstein, 1988). The measures of drinking context typically collected in large-scale alcohol epidemiological studies—where and with whom an individual usually drinks—are becoming increasingly inappropriate for assessing exposure to smoking as more and more public places restrict or prohibit smoking. Thus, the data needed to assess this element of the overall effect of drinking on smoking cessation are probably best measured in smaller-scale studies of specific relapse situations that can directly examine the timing of alcohol use relative to relapse.

This study examined the association between drinking patterns and the odds of having stopped smoking for at least 3 months. This outcome is the product of two elements, the odds of having attempted to stop smoking and the odds of that attempt having been successful. Future research with the NLAES data set will focus on distinguishing alcohol's effects on each of these elements. Based on the existing literature, drinking patterns have been more clearly linked with the success of attempts than with the probability of making the attempt to stop smoking. The relative impact of drinking patterns and alcohol use disorders on these two separate components of the smoking cessation process may help to clarify further the mechanisms that underlie its negative association with drinking.

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Age at Smoking Onset and Its Association With Alcohol Consumption and DSM-IV Alcohol Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant

The major purpose of this study was to examine the relationship of early onset smoking with lifetime drinking and the subsequent development of DSM-IV alcohol abuse and dependence using a large representative sample of the U.S. general population. Prevalences of lifetime drinking, alcohol abuse and dependence, and their associated severity were compared among smoking groups defined by age at onset of smoking and among nonsmokers. Linear logistic regression analyses were conducted to assess the relationship between age at smoking onset and lifetime drinking, alcohol abuse and dependence, controlling for important covariates. Early onset smoking was a significant predictor of lifetime drinking and the subsequent development of lifetime alcohol abuse and dependence, a relationship that generally remained consistent for males, females, whites and blacks. Early onset smoking was significantly associated with more excessive alcohol consumption and more severe alcohol use disorders relative to late onset smokers and nonsmokers. Early onset smoking was also significantly associated with heavier and longer smoking careers compared to late onset smokers. Implications of these findings are discussed in terms of prevention of adolescent smoking and the need for further research on understanding the mechanisms underlying the associations between early onset smoking and lifetime drinking, alcohol abuse and dependence.

INTRODUCTION

To date, a large body of literature has examined the relationship between tobacco use and alcohol consumption among general population samples and among alcoholics (Bien and Burge, 1990; Istvan and Matarazzo, 1984). The finding that there is a strong relationship between smoking and drinking is consistent across demographic subgroups of the population and generalizes to adult and adolescent populations alike. Smokers are more likely to drink than nonsmokers and drinkers are more likely to smoke than alcohol abstainers. The relationship has also been found to be dose-related in that heavy smokers drink more alcohol than light smokers and heavy drinkers smoke more than light drinkers (Johnson and Jennison, 1992).

Recent studies have also demonstrated that early onset tobacco use increases the risk of the subsequent development of tobacco dependence (Breslau et al., 1993) and that early onset of alcohol use is a powerful predictor of the subsequent development of alcohol abuse and dependence (Grant & Dawson, 1997). Given that the initiation of smoking and drinking occurs largely during the adolescent years, in addition to the abundance of correlational evidence that smoking and drinking are linked, it is very surprising that so little attention has been given to the role of early onset smoking in the development of subsequent alcohol abuse and dependence.

The need to study early onset smoking in relation to the subsequent development of alcohol abuse and dependence is urgent in view of recent national statistics that have shown minimal declines in adolescent smoking since 1985 and that current smoking prevalence among male and female adolescents became essentially equal by the early 1990s (Nelson, Giovino, Shopland, Mowery, Mills, and Eriksen, 1995; U.S. Department of Health and Human Services, 1997). Prior to 1985, smoking rates among adolescents had generally been on a decline and male rates exceeded those of females. Recent evidence has further suggested that while the separate adverse health and societal effects of smoking and drinking are both serious and legion, the combined use of tobacco and alcohol

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is associated with increased risks of mouth, throat and lung cancer, cardiovascular disease, hypertension and sudden death (Bien & Burge, 1990; Istvan & Matarazzo, 1984).

The major purpose of this study was to examine the relationship between age of onset of smoking and the prevalence of lifetime drinking, alcohol abuse and dependence. To our knowledge, no other study has determined the likelihood of drinking, alcohol abuse and dependence as a function of age of onset of smoking. Moreover, this study provides for the first time, an opportunity to examine this association in a large representative sample of the U.S. population using the most recent psychiatric classifications of alcohol abuse and dependence. An additional purpose was to examine whether early onset smoking was associated with more severe episodes of alcohol abuse and dependence and heavier alcohol consumption relative to later onset smoking and nonsmoking. Similarly, this study also examined whether a relationship existed between early onset smoking and a higher consumption and duration of smoking in adulthood compared to later onset smoking and nonsmoking.

METHOD

Study sample

This study was based on the National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national probability sample sponsored by National Institute on Alcohol Abuse and Alcoholism (NIAAA). Field work for the study was conducted by the United States Bureau of the Census in 1992. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. Approximately 91.9% of the selected households participated in this survey, and 97.4% of the randomly selected respondents in these households participated in this survey.

The NLAES utilized a complex multistage design which featured sampling of primary sampling units with probability proportional to size and oversampling of the black and young adult (18 to 29 years) populations. The NLAES design has been described in detail elsewhere (Grant et al., 1994; Massey et al. 1989).

Diagnostic assessment

Diagnoses of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV: American Psychiatric Association, 1994) alcohol use disorders were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained interviewers who were not clinicians (Grant & Hasin, 1992). The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV criteria for alcohol abuse and dependence.

The AUDADIS diagnoses of alcohol abuse and dependence satisfied the clustering and duration criteria of the DSM-IV definitions. The criteria of the DSM-IV included the requirement for a clustering of symptoms within any 1-year period, in addition to associating duration qualifiers with certain abuse and dependence criteria. The duration qualifiers are defined as the repetitiveness with which symptoms must occur to be counted as positive toward a diagnosis. They are represented by the terms 'recurrent,' 'often' and 'persistent' appearing in the description of the diagnostic criteria. Not only were the clustering criteria represented in past year AUDADIS diagnoses of abuse and dependence, but the corresponding past diagnoses (before the past year) also were measured as syndromes, or the clustering of the required number of symptoms necessary to achieve a diagnosis: (1) at the same time; (2) continuously for at least 1 month; or (3) repeatedly for at least 1 month. For the purposes of the present study, respondents were classified with a lifetime alcohol disorder if they had experienced an episode of abuse or dependence in the past year and/or before the past year. The DSM-IV abuse and dependence diagnostic groups were mutually exclusive. Respondents classified as lifetime alcohol abusers did not meet criteria for lifetime dependence. Respondents classified with lifetime dependence included those with and without abuse diagnoses. Reliabilities of past year and prior to past year alcohol use disorders were 0.76 and 0.73 as determined in an independent test-retest study conducted in the general population prior to fielding the survey proper. (Grant et al., 1995)

The severity of alcohol use disorders was measured by the number of lifetime DSM-IV abuse and dependence symptom items that operationalized the seven DSM-IV dependence criteria and four DSM-IV abuse criteria (range, 1–31) and the longest duration of an episode of alcohol abuse and dependence. Severity of alcohol use disorders was also measured as ever having sought treatment for a problem related to drinking. Respondents were specifically instructed to include any help they had received for their drinking, including help for combined alcohol and drug use if alcohol was the major problem for which they sought help.

Respondents were also asked to indicate whether they ever sought help from 23 specific types of treatment sources including: inpatient alcohol and/or drug rehabilitation programs and inpatient wards of general or psychiatric hospitals; outpatient clinics and alcohol and/or drug detoxification units; and 12-step groups (e.g. Alcoholics Anonymous). Receiving help from any inpatient, outpatient or 12-step program on a lifetime basis constituted the three alcohol treatment positive measures examined in this study. Each of these measures was associated with reliabilities of 0.72 (Grant et al., 1993).

Alcohol-related measures

Age of drinking onset was ascertained by asking respondents how old they were when they first started drinking, not counting small tastes or sips of alcohol. Other measures selected as control variables for multivariate analyses were demographic and alcohol-related items that have been shown to affect the risk of drinking, alcohol abuse and/or dependence. These included race (black vs. white), gender, age (18–29 years; 30–44 years; 45–64 years; 65+ years) and socioeconomic status scores (range: 0 = lowest, 100 = highest). Socioeconomic status scores were based on occupation, income and education according to the procedures outlined by Terrie and Nam (1994). Duration of drinking was estimated by subtracting the age at onset of drinking from either the age at last drink (for former drinkers) or age at interview (for past year or current drinkers). When age at drinking onset equaled age at last drink, the duration of drinking variable was set at 0.5 years. The test-retest reliability of the drinking onset variable was 0.72 (Grant et al., 1995).

Family history of alcoholism was ascertained through a series of questions that asked about each type of first-degree biological relative. For each type of relative, the respondent was asked how many relatives of that type lived to be at least ten years old and how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM-IV criteria for alcohol use disorders: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover." In a test-retest study conducted in conjunction with the pretest for the NLAES, the family history items generally showed good to excellent reliability, with kappas of 0.70 or higher for most types of first degree relatives (e.g., 0.72 for fathers, 1.00 for mothers, 0.90 for brothers, 0.73 for sisters) (Grant et al., 1993). Slightly lower kappa values were obtained for sons and daughters (0.65 for each). The family history measure in this study was considered as positive if any first-degree relative was reported as having been an alcoholic or problem drinker.

Smoking groups were compared with respect to their average daily ethanol intake during two time frames: (1) during the period of heaviest drinking during their lifetimes; and (2) during the past year for those respondents classified as current drinkers. Average daily ethanol intake for beer, wine and liquor for both periods was based on patterns of usual and heaviest consumption. The measures obtained for each type of beverage included frequency of drinking (converted to number of drinking days/year), typical number of drinks consumed/drinking day and typical size of drink (ounces of beer, wine or liquor). Ounces of beverage were converted to ounces of ethanol using the following conversion factors: 0.045 for beer, 0.121 for wine and 0.409 for liquor (Distilled Spirits Council of the United States, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams, Clem, & Dufour, 1993). To estimate average daily ethanol intake based on usual and heaviest consumption during the respondent's period of heaviest drinking, the volume of ethanol for each beverage type was calculated as follows: oz. $\text{Ethanol}_{\text{beverage}}$ = (total minus heavy drinking $\text{days/year}_{\text{beverage}}$ x number of drinks/usual drinking $\text{day}_{\text{beverage}}$ x ounces of beverage in a typical drink consumed on usual drinking $\text{day}_{\text{beverage}}$ x ethanol conversion $\text{factor}_{\text{beverage}}$) + (heavy drinking $\text{days/year}_{\text{beverage}}$ x number of drinks/heavy drinking x ounces of beverage in a typical drink consumed on heavy drinking $\text{days}_{\text{beverage}}$ x ethanol conversion $\text{factor}_{\text{beverage}}$). These volumes were then summed over the three beverage types and the resulting volume was divided by 365. Results of the independent test-retest study conducted prior to fielding the NLAES showed these two consumption variables to be highly reliable (kappas = 0.73 and 0.72) (Grant et al., 1995).

Smoking-related measures

In this study, respondents were classified as lifetime smokers if they had ever smoked at least 100 cigarettes, 50 cigars, or a pipe at least 50 times. Lifetime smokers were asked about the quantity and duration of smoking which was measured as the current monthly volume of smoking among current smokers or the volume of smoking at the time of quitting among ex-

smokers. For lifetime daily smokers, similar measures of volume and duration of smoking daily were constructed, with the exception that volume of smoking was measured on a daily basis. A measure of time to smoking daily was also constructed for this study and was expressed as the age at first daily smoking minus the age at onset of smoking. Reliabilities associated with onset of smoking (0.70), lifetime smoking (0.75), current smoking (0.88) and volume of smoking (0.60 – 0.80) were good to excellent (Grant et al., 1993).

Statistical analyses

Two sets of analyses were conducted. In the first set, the analyses were bivariate. Overall chi-square and *F* statistics were computed for categorical and continuous measures, respectively. If the overall X^2 or *F* statistic was significant (< 0.01), separate pairwise comparisons were conducted between the groups using partitioned X^2 analysis or *t* tests, respectively. To protect against type 1 error, due to multiple comparisons, any pairwise comparison was required to yield a test statistic at the level of < 0.0001 to be declared statistically significant.

The second set of analyses employed multivariate tests. These included three linear logistic regression analyses. These analyses were used to assess the relationship between age at smoking onset and the odds of: (1) lifetime drinking; (2) lifetime alcohol abuse; and (3) lifetime alcohol dependence, controlling for the effects of gender, race, age, and family history of alcoholism. Additional control variables for the analyses in which alcohol abuse and dependence served as the outcome measures included age of onset of drinking and the duration of drinking. In all three regression analyses, age at onset of smoking served as the exposure variable. Because of the importance of gender and race in the onset of both smoking and drinking, second order interactions (e.g., age of onset of smoking ≤ 13 x gender) between age at onset of smoking and these sociodemographic variables were examined in all three regression analyses.

The linear regression analysis in which lifetime drinking served as the outcome measure included the entire NLAES sample (*n* = 42,862) while similar analyses related to the outcome measures of alcohol abuse and dependence included all smokers and nonsmokers who were also lifetime drinkers (*n* = 27,616). All statistical analyses were conducted using SUDAAN, a software package that uses Taylor series linearization to adjust for the complex sample design of the NLAES (Research Triangle Institute, 1996).

RESULTS

Fifty-one percent (*n* = 21,807) of the NLAES sample was composed of current (*n* = 13,084) and former (*n* = 8,723) smokers. Less than 0.3% (*n* = 131) of the sample were excluded from the analyses presented here due to missing values for the age of onset at smoking measure. The percent of lifetime smokers who initiated smoking at ages 13 and younger, between the ages of 14 and 15 and at ages 17 and older were 22.6% (*n* = 4,930), 34.2% (*n* = 7,449), and 43.2% (*n* = 9,428), respectively. Approximately 66% (*n* = 27,616) of the total sample were classified as lifetime drinkers while 4.9% (*n* = 1,947) and 13.3% (*n* = 5,412) were classified with lifetime diagnoses of alcohol abuse and dependence, respectively.

Table 1 shows the demographic characteristics of respondents according to age at smoking onset among lifetime smokers and among lifetime nonsmokers. Smokers initiating onset of smoking at ages 13 and younger were significantly more likely to be male, married and have less than a high school education and less likely to be black compared to both smokers who initiated smoking after the age of 14 and nonsmokers.

Table 1. Sociodemographic Characteristics by Age at Smoking Onset Among Lifetime Smokers and Among Lifetime Nonsmokers

Sociodemographic Characteristic	Age at Onset of Smoking (in years)			Lifetime Nonsmokers	Significant Comparisons
	≤ 13 (N = 4,930) (1)	14–16 (n = 7,449) (2)	≥ 17 (n = 9,428) (3)	(n = 20,924) (4)	(X^2 or t, p < 0.0001)
% Male	66.7 (0.72)[a]	55.6 (0.63)	50.3 (0.62)	39.4 (0.45)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
% Black	6.4 (0.43)	8.0 (0.42)	12.7 (0.52)	13.3 (0.47)	1 vs. (2) (3) (4); 2 vs. (3) (4)
Current age (in years)	42.1 (0.27)	43.1 (0.26)	49.6 (0.22)	41.8 (0.21)	1 vs. (2) (3); 2 vs. (3) (4); (3) vs. (4)
% Married or living with someone if married	68.0 (0.77)	66.6 (0.71)	65.6 (0.57)	59.5 (0.51)	1 vs. (2) (3) (4); 2 vs. (4); 3 vs. (4)
% With less than a high school education	25.3 (0.83)	20.9 (0.59)	19.2 (0.51)	17.8 (0.39)	1 vs. (2) (3) (4); 2 vs. (3)(4)
Socioeconomic Status (x)	48.4 (0.46)	50.4 (0.36)	52.5 (0.33)	50.2 (0.28)	1 vs. (2) (3) (4)

Notes. Percentages expressed as weighted figures, n's expressed as unweighted figures.
a. Standard errors appear in parentheses.

Respondents in the youngest smoking onset category (i.e., < 13 years) had significantly lower socioeconomic status scores relative to the older onset smokers and nonsmokers and were also significantly younger at the time of the interview than older onset smokers, but not nonsmokers.

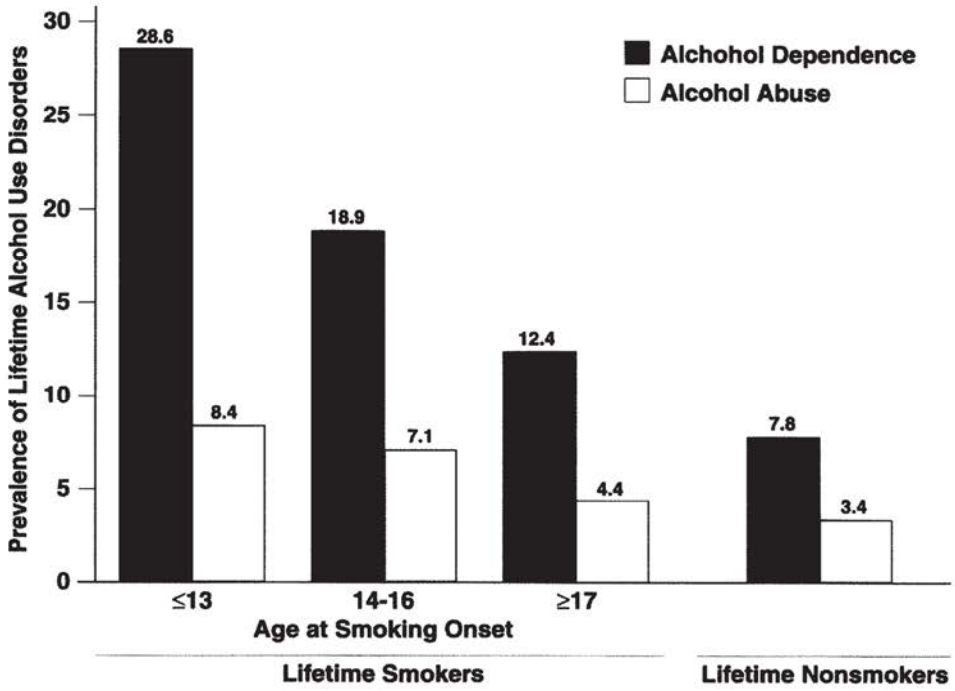
Compared to respondents who initiated smoking at ages 17 and older and nonsmokers, respondents who began to smoke between the ages of 14 and 16 were significantly more likely to be male and less likely to be black. Respondents initiating smoking between the ages of 14 and 16 were also more likely to be married and have less than a high school education compared to nonsmokers, but not compared to respondents who initiated smoking at ages 17 and older. There were no differences observed in socioeconomic status between nonsmokers and those respondents initiating smoking between the ages of 14 and 16 and at ages 17 and

Table 2. Alcohol-Related Characteristics by Age at Smoking Onset Among Lifetime Smokers and Among Lifetime Nonsmokers

Alcohol-Related Characteristic	Age at of Smoking Onset (in years) ≤ 13 (1)	14–16 (2)	≥ 17 (3)	Lifetime Nonsmokers (4)	Significant Comparisons (X^2 or t, $p < 0.0001$)
% Lifetime alcohol dependence	28.6 (0.79)[a]	18.9 (0.55)	12.4 (0.40)	7.8 (0.26)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
% Lifetime alcohol abuse	8.4 (0.47)	7.1 (0.36)	4.4 (0.24)	3.4 (0.15)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
Age at onset of drinking (x)	17.2 (0.08)	18.3 (0.06)	20.3 (0.07)	19.4 (0.06)	1 vs. (2) (3) (4); 2 vs. (3) (4)
Duration of longest episode of alcohol abuse in months (x)	56.7 (2.37)	44.3 (2.06)	47.2 (2.91)	32.6 (1.76)	1 vs. (2) (3) (4); 2 vs. (4); 3 vs. (4)
Duration of longest episode of alcohol dependence in months (x)	47.2 (2.27)	36.1 (1.75)	38.3 (2.22)	24.1 (1.49)	1 vs. (2) (3) (4); 2 vs. (4); 3 vs. (4)
Average daily ethanol consumption during period of heaviest lifetime drinking in ounces (x)	3.7 (0.13)	2.0 (0.07)	1.5 (0.05)	1.0 (0.03)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
Past year average daily ethanol consumption in ounces (x)	1.2 (0.06)	0.8 (0.03)	0.7 (0.02)	0.5 (0.01)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
Number of lifetime alcohol symptoms (x)	12.5 (0.18)	10.6 (0.14)	10.2 (0.18)	8.9 (0.12)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
% Lifetime alcohol inpatient treatment	7.5 (0.43)	3.6 (0.26)	1.9 (0.17)	0.4 (0.05)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
% Lifetime alcohol outpatient treatment	8.9 (0.46)	4.4 (0.29)	2.5 (0.19)	0.9 (0.07)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
% Lifetime alcohol 12-step program	10.7 (0.50)	5.0 (0.32)	3.0 (0.20)	0.9 (0.08)	1 vs. (2) (3) (4); 2 vs. (3) (4); 3 vs. (4)
% Any first-degree relative alcoholic	67.6 (80.8)	58.8 (0.74)	50.2 (0.63)	48.2 (0.45)	1.vs. (2) (3) (4); 2 vs. (3) (4)

Notes. Percentages expressed as weighted figures, n s expressed as unweighted figures;
a. Standard errors appear in parentheses.

Figure 1. Prevalence of Lifetime Alcohol Abuse and Dependence by Age at Smoking Onset Among Smokers and among Nonsmokers



older. Smokers initiating smoking at age 17 or older were more likely to be male and married and were older than nonsmokers.

Table 2 shows the prevalences of alcohol use disorders (Figure 1) and their associated severity among smokers classified by age of onset of smoking and among nonsmokers. The likelihood of developing lifetime alcohol abuse and dependence significantly decreased as a function of increasing age at onset of smoking, and was lowest among nonsmokers. Respondents initiating smoking at ages 13 and younger were approximately twice as likely to develop alcohol abuse and dependence as respondents initiating smoking at ages 17 and older. Nonsmokers were significantly less likely to develop alcohol abuse and dependence at some time during their lives relative to all smokers, regardless of age at smoking onset. Respondents initiating smoking prior to age 17 were also younger at the age of onset of drinking compared to those initiating smoking after age 17 and nonsmokers.

In general the severity of alcohol use disorders increased significantly as a function of decreasing age at onset of smoking, and lifetime nonsmokers were observed to have less severe alcohol use disorders than smokers regardless of age at onset of smoking. The earliest onset smokers (i.e., ≤ 13 years) had significantly more lifetime alcohol symptoms and were more likely to have received alcohol inpatient and outpatient treatment and to have participated in a 12-step programs than respondents classified with smoking onsets at ages 14 to 16 and 17 and older, and nonsmokers. Similarly, current and heaviest average daily ethanol consumption significantly decreased as a function of increasing age at smoking onset and was significantly greater among smokers than nonsmokers. The same relationship pertained to the duration of longest episode of alcohol abuse and dependence, with the exception that there were no significant differences observed in these measures between respondents initiating smoking at ages 14 to 16 and at ages 17 and older. The earliest onset smokers were also significantly more likely to have a family history of alcoholism compared to the older onset smoking groups and nonsmokers as were respondents who began to smoke between the ages of 14 and 16. Respondents who initiated smoking at ages 17 and older were no more likely to have a family history of alcoholism than were nonsmokers.

As can be seen in Table 3, early onset smoking was also related to subsequent heavier tobacco consumption. With the exception of the percentage of current (or most recent) smoking, the youngest smoking onset group smoked significantly more and longer compared to both the later smoking onset groups. Interestingly, the time to smoking daily was significantly greater among the youngest smoking onset group relative to the two older onset groups.

Three separate multivariate linear logistic regression analyses were conducted to assess the contribution of age at onset of smoking to the odds of lifetime drinking, alcohol abuse and alcohol dependence, controlling for the effects of gender, race, age and family history of alcoholism. For the two regression models associated with the outcome measures of alcohol abuse and dependence, age at drinking onset and duration of drinking were also included as control variables. The interaction of age at onset of smoking and both gender and race were examined in each of the three logistic analyses.

The results of the final logistic model (i.e., the model displaying only significant interaction terms) for lifetime drinking are presented in Table 4. After adjusting for all of the control variables, age at onset of smoking remained a major and significant contributor to determining lifetime drinking status. The only significant modifier of the smoking onset—drinking association was race. Specifically, the odds of lifetime drinking was approximately five times greater (adjusted odds ratio (AOR) = $e^{1.55} = 4.7$) for the youngest smok-

Table 3. Smoking-Related Characteristics of Lifetime Smokers by Age at Smoking Onset

Smoking Characteristic	Age at Smoking Onset			Significant Comparisons (X^2 or t, $p < 0.0001$)
	≤ 13 (n = 4,930) (1)	14–16 (n = 7,449) (2)	≥ 17 (n = 20,924) (3)	
% Current smokers	64.2 (0.87)[a]	62.5 (0.68)	56.3 (0.61)	1 vs. (2); 2 vs. (3)
Monthly volume of smoking (per month)	663.3 (4.14)	574.2 (6.21)	477.5 (5.14)	1 vs. (2)(3); 2 vs. (3)
Duration of smoking (in months)	227.8 (4.19)	198.2 (2.98)	196.0 (2.42)	1 vs. (2)(3)
Volume of smoking at time when smoked daily	24.3 (0.28)	20.9 (0.20)	18.3 (0.17)	1 vs. (2)(3); 2 vs. (3)
Duration of daily smoking (in months)	200.9 (3.03)	188.2 (2.58)	195.4 (2.27)	1 vs. (2)(3); 2 vs. (3)
Time to smoking daily (in years)	4.3 (0.08)	2.3 (0.04)	1.7 (0.05)	1 vs. (2)(3); 2 vs. (3)

Notes. Percentages expressed as weighted figures; n s expressed as unweighted figures.
a. Standard errors appear in parentheses.

ing onset group relative to the reference category of nonsmokers and approximately four times greater (AOR = 4.3) for respondents who initiated smoking between the ages of 14 and 16 compared to nonsmokers. For those respondents who began smoking at ages 17 and older, blacks were nearly five times more likely (AOR = 4.8) and whites were over three times more likely (AOR = 3.3) to be lifetime drinkers compared to nonsmokers.

The final regression models examining the association between smoking onset and alcohol abuse and dependence are shown in Table 5. Similar to the results for lifetime drinking, age at onset of smoking remained a significant contributor to the development of alcohol dependence among the sample of lifetime drinkers once other factors impacting on the relationship were controlled. The odds of dependence in the earliest smoking onset group were approximately two times greater (AOR = 2.1) than nonsmokers, while the odds of developing dependence among those initiating smoking between the ages of 14 to 16 (AOR = 1.6) and at the age of 17 years and older (AOR = 1.5) were approximately one and one half times greater than nonsmokers. Although the odds of abuse were one and one fourth times greater for respondents initiating smoking at ages 13 and younger (AOR = 1.2) and between the ages of 14 and 16 (AOR = 1.2) relative to nonsmokers, the odds of developing alcohol abuse at some time in one's lifetime were no greater for those initiating smoking at ages 17 and older than for nonsmokers.

DISCUSSION

This study was the first to show that early onset smoking was a significant predictor of lifetime drinking and the subsequent development of alcohol abuse and de-

Table 4. Final Linear Logistic Regression Model for Lifetime Drinking

Characteristic	*Beta*	*SE (Beta)*	*p-Value*
Age of smoking onset ≤ 13 years[1]	1.55	0.06	< 0.0000
Age of smoking onset 14–16 years[1]	1.47	0.04	< 0.000
Age of smoking onset ≥ 17 years[1]	1.21	0.04	< 0.0000
Current age 30-to-44 years[2]	0.08	0.03	< 0.01
Current age 45-to-64 years[2]	–0.46	0.04	< 0.0000
Current age ≥ 65 years[2]	–1.10	0.04	< 0.0000
Gender (effect of male)	0.95	0.03	< 0.0000
Race (effect of black)	–0.81	0.05	< 0.0000
Positive family history of alcoholism	0.48	0.03	< 0.0000
Race x Age at smoking onset ≥ 17 years	0.36	0.09	< 0.0003

Notes. Goodness of fit for overall model: Satterthwaite's F (9,68) = 468, p < 0.000.
1. Effect of each age category relative to 18-to-29 year olds.
2. Effect of each smoking onset age category relative to lifetime nonsmokers.

Table 5. Final Linear Logistic Regression Model for DSM-IV Alcohol Abuse and Dependence

Characteristic	*Alcohol Abuse*			*Alcohol Dependence*		
	Beta	*SE (Beta)*	*p-value*	*Beta*	*S E (Beta)*	*p-value*
Age at smoking onset ≤ 13 years[1]	0.19	0.08	< 0.01	0.75	0.06	< 0.0000
Age at smoking onset 14-16 years[1]	0.22	0.07	< 0.002	0.46	0.05	< 0.0000
Age at smoking onset ≥ 17 years[1]	0.05	0.01	N.S.[3]	0.39	0.05	< 0.0000
Current age 30-to-44 years[2]	–0.13	0.07	< 0.01	–0.06	0.05	N.S.
Current age 45-64 years[2]	–0.57	0.15	< 0.0003	–0.02	0.09	N.S.
Current age ≥ 65 years[2]	–1.33	0.23	< 0.0000	–0.24	0.13	N.S.
Gender (effect of male)	0.51	0.06	< 0.0000	0.53	0.03	< 0.0000
Race (effect of Black)	–0.58	0.13	< 0.0001	–0.22	0.07	< 0.006
Age at onset of drinking	–0.06	0.01	< 0.0000	–0.12	0.01	< 0.0000
Duration of drinking	0.01	0.001	N.S.	–0.03	0.001	< 0.0000
Positive family history of alcoholism	0.17	0.05	< 0.005	0.79	0.04	< 0.0000

Notes. Goodness of fit for overall model: Abuse, Satterthwaite's F (10,68) = 10.6; p < 0.0000; Dependence, F (10,68)=15, p < 0.0000
1. Effect of each age category relative to 18-to-29 year olds.
2. Effect of each smking onset age category relative to lifetime nonsmokers.
3. NS=not significant

pendence, particularly among smokers initiating tobacco use prior to the age of 17. With the exception of lifetime drinking among late onset smokers (≥ 17 years), these relationships between early smoking onset and lifetime drinking, alcohol abuse and alcohol dependence were observed to hold for males, females, blacks and whites. Moreover, the results of this study showed that early onset smoking (i.e., ≤ 13 years) was significantly associated with more excessive alcohol consumption and more severe alcohol use disorders relative to late onset smokers (i.e., ≥ 14 years) and nonsmokers. Early onset smokers were also significantly more likely to have smoked more heavily and for longer periods of time compared to late onset smokers.

A significant contribution of this study to the growing literature on the relationship between smoking, drinking and alcoholism is the focus it provides on the importance of epidemiologic and etiologic study findings in guiding prevention efforts. However, many questions remain unanswered that would form the basis of successful prevention strategies. Foremost is our lack of complete understanding of why early onset smoking is related to the development of alcohol consumption, abuse and dependence. Is it the delay in the onset of smoking itself, or a combination of other factors, that reduces the risk for the drinking and the subsequent development of alcohol abuse and dependence? That is, the relationship between early onset smoking and alcohol abuse and dependence may be due to a common vulnerability model, that is a model that posits that genetic or environmental factors predispose an individual to smoking and alcohol abuse and dependence. Alternatively, it may be possible that the abuse of one substance predisposes an individual to abuse of other substances as the result of any number of biobehavioral processes including cross-tolerance, synergistic physiological effects and cueing effects. High risk, multiwave prospective studies are needed to evaluate and test both common vulnerability models and unidirectional and reciprocal models as they relate to early onset smoking and alcohol abuse and dependence.

Our lack of complete understanding of the relationship between early onset smoking and drinking, alcohol abuse and alcohol dependence does not minimize the importance of the detrimental effects of smoking and drinking. The adverse health effects of excessive drinking accompanying alcohol abuse and dependence are serious and numerous, encompassing cardiovascular problems, birth defects, brain dysfunction, damage to the liver and pancreas, suicide, depression, nutritional deficiencies, and family violence (U.S. Department of Health and Human Services, 1997). Tobacco use has been referred to as the greatest single cause of preventable death (Pollin, 1977) and has been implicated in a variety of diseases, while smoking and drinking in combination has been linked to increased risks of mouth, throat and lung cancer, cardiovascular disease, hypertension and sudden death. The findings of this study serve to highlight the importance of early onset smoking in increasing one's risk of alcohol abuse and dependence, and the associated detrimental health risks related to smoking and drinking both singly and in combination.

In conclusion, the findings of this study, taken together with our knowledge of the health risks of smoking and drinking, suggest that preventive efforts would best be targeted among all adolescent smokers, especially those who initiate smoking prior to the age of 17. In this context, it is important to note that smoking initiation at the ages 13 and younger was associated with a significantly greater lag time (4.3 years) to daily smoking than smoking initiation between the ages of 14 and 16 (2.3 years) and after the age of 17 (1.7 years). This finding is most likely due to the lack of a supportive or permissive social environment for smoking among younger adolescents. The progression to daily smoking might well require circumstances that do not become available prior to the age of 17, such as driving, earning money and establishing greater independence from adult supervision. The finding that daily smoking will on average be delayed for four years among those initiating smoking at ages 13 and younger additionally supports the targeting of smoking intervention efforts at adolescents under the age of 17, prior to the development of daily smoking. The greater delay to daily smoking could serve as a second window of opportunity in which to focus on smoking cessation intervention among the earliest onset smokers for whom primary prevention was not successful.

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CHAPTER 7
DRUG DEPENDENCE AND ABUSE

Prevalence and Correlates of Drug Use and DSM–IV Drug Dependence in the United States: Results of the National Longitudinal Alcohol Epidemiologic Survey

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This study presents updated estimates of the prevalence and examines the correlates of drug use and dependence in a representative sample of the U.S. population. The prevalence of lifetime drug use was 15.6%, with 4.9% of the respondents reporting drug use during the past 12 months. Lifetime and 12-month prevalence of drug dependence were estimated at 2.9% and 0.8%, respectively. Men were significantly more likely to use drugs than women, and drug use and dependence were much more common among cohorts born after World War II. The data indicated that rates of dependence among women were quickly approaching the rates among men in the younger cohorts. Members of the youngest cohort, between the ages of 18 and 24 years at the time of the interview, were more likely to use drugs, to become dependent, and to persist in dependence compared to the older cohorts, including Cohort 2 who experienced adolescence at the height of widespread introduction of illicit drugs among youth in the mid-1960s. The demographic correlates of first use, onset of dependence, and persistence of dependence varied as a function of the stage of progression. Implications of these findings are discussed in terms of changes over time in drugs of choice and dependence liability and vulnerability among recent drug users.

Since the early 1970s, the National Institute on Drug Abuse and, more recently, the Substance Abuse and Mental Health Services Administration have conducted surveys to monitor drug use prevalence and trends at the national level. Foremost among these are the High School Senior Survey (Johnston et al., 1993), which tracks drug use levels among junior high, high school, college, and young adult subpopulations, and the National Household Survey on Drug Abuse (NHSDA; U.S. Department of Health and Human Services, 1992), which tracks the prevalence of drug use among the U.S. civilian noninstitutionalized population. Although these national surveys are useful for a number of purposes, they can be criticized because of their failure to produce estimates of drug abuse and dependence.

To date, only two surveys have reported national estimates of the prevalence of drug abuse and dependence. The first was the Epidemiologic Catchment Area (ECA) Survey (Robins, Locke, & Regier, 1990), in which 18,571 respondents, aged 18 years and older, were interviewed in a series of five community-based epidemiologic studies in the early 1980s. The second was the National Comorbidity Survey (NCS), a national probability sample of 8,098 respondents aged to 15 to 54, conducted in 1991 (Kessler et al., 1994). Unfortunately, there were several methodological limitations of these surveys. The five community-based samples underlying the ECA were not nationally representative of the U.S. adult population. Moreover, the ECA estimates are now over a decade old. Similarly, the representativeness and precision of the NCS can be called into question due to its exclusion of adults 55 years and older and its relatively small sample size, respectively. Estimates of the prevalence of drug abuse and dependence in both surveys also used historic diagnostic classifications no longer in use, namely the *Diagnostic and Statistical Manual of Mental Disorders-Third Edition* (DSM–III; American Psychiatric Association, 1980) used in the ECA and the DSM–III–Revised (DSM–III–R; American Psychiatric Association, 1987) used in the NCS.

The purpose of this study is to present updated estimates of the prevalence of drug use and dependence and to examine the demographic correlates of drug

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use and dependence in a representative sample of the U.S. population based on the National Institute on Alcohol Abuse and Alcoholism's (NIAAA) National Longitudinal Alcohol Epidemiologic Survey (NLAES; Grant, Peterson, Dawson, & Chou, 1994; Massey, Moore, Parsons, & Tadros, 1989). The NLAES was designed to overcome many of the methodological problems inherent in the previous population surveys, that is, the ECA and NCS. First, the diagnoses were based on the most current psychiatric classification, the DSM–IV (American Psychiatric Association, 1994). Second, unlike the ECA, the NLAES utilized a nationally representative sample of the United States adult population, and unlike the NCS, it included respondents aged 55 years and older. Compared to the NCS, the larger sample size of the NLAES, 42,862 respondents, also enabled both detailed and precise estimation of the prevalence of drug use and dependence within important sociodemographic subgroups of the population.

One of the major advances introduced with the NLAES was the more accurate measurement of drug dependence appearing on the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS; Grant & Hasin, 1992). The Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981) used in the ECA and the University of Michigan-Composite International Diagnostic Interview (UM–CIDI; Wittchen & Kessler, 1991) used in the NCS both failed to measure drug dependence disorders as syndromes (i.e., as the clustering of the required number of symptoms in time) as required by the DSM–III and DSM–III–R. Moreover, the DIS failed to represent the DSM–III dependence duration criterion (i.e., the duration of the disorder must persist at least 1 month), and the UM–CIDI did not represent the duration criterion of the DSM–III–R (i.e., some symptoms of the disturbance occurred continuously for at least 1 month or repeatedly over a longer period of time). In contrast, the AUDADIS diagnoses of 12-month and lifetime dependence satisfied both the clustering and duration criteria of the DSM–IV classification. As a result of these measurement weaknesses, the DIS and UM–CIDI were precluded from measuring the onset and recency of drug dependence as the onset and recency of episodes of the disorder, as was done in the AUDADIS. Rather, the DIS and UM–CIDI were relegated to measuring the onset and recency of a symptom of dependence, measures that often bear little or no relationship to the onset and recency of an episode of drug dependence as defined in both the DSM–III and DSM–III–R.

METHODS

Study sample

Prevalence estimates of DSM–IV drug use disorders were based on the 1992 NLAES, a national probability sample sponsored by the NIAAA. Field work for the study was conducted by the United States Bureau of the Census. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. The household response rate for this representative sample of the United States population was 91.9%, and the sample person response rate was 97.4%.

The NLAES consisted of a complex multistage design that featured sampling of primary sampling units with probability proportional to size and oversampling of the black and young adult (18–29 years) populations. The NLAES design has been described in detail elsewhere (Grant et al., 1994; Massey et al., 1989).

Drug use and drug dependence assessment

The NLAES measures of illicit or nonmedical drug use were defined as use (a) without a physician's prescription, (b) in greater amounts than prescribed, (c) more often than prescribed, (d) longer than prescribed, or (e) for a reason other than prescribed. Information on nonmedical drug use was ascertained separately for sedatives, tranquilizers, opioids other than heroin, amphetamines, cocaine (and crack cocaine), cannabis (and THC and hashish), heroin, methadone, hallucinogens, and other drugs of abuse (e. g., inhalants, solvents, and antidepressants) before being combined into the aggregate drug use measures.

Lifetime drug use, for the purpose of this study, was defined as ever having tried any of the drugs in any of the above drug classes at least 12 times. Twelve-month drug use was defined as the nonmedical use of any drug at least once during the year preceding the interview among the respondents who were classified as lifetime users. Only lifetime drug users were asked the survey questions concerning drug dependence.

In the AUDADIS, DSM–IV diagnoses of drug dependence were initially derived separately for each illicit drug and then combined into the aggregate measure of any drug dependence used in this study. Consistent with DSM–IV, AUDADIS diagnoses of drug dependence required that a person meet at least three of the following seven criteria defined for dependence in any 1 year: (a) tolerance; (b) withdrawal or

avoidance of withdrawal; (c) a drug is often taken in larger amounts or over a longer period than intended; (d) persistent desire or unsuccessful attempts to cut down or stop using; (e) spending much time obtaining a drug, using it, or recovering from its effects; (f) giving up or reducing occupational, social, or recreational activities in favor of drug use; or (g) continuing to use despite a physical or psychological problem caused or exacerbated by drug use.

The AUDADIS diagnoses of 12-month drug dependence satisfied both the clustering and duration criteria of the DSM–IV classification. Twelve-month diagnoses of drug dependence required the occurrence of at least three dependence criteria in the year preceding the interview, and the duration qualifiers associated with dependence criteria must have been satisfied. The duration qualifiers are defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms *recurrent, often,* and *persistent* appearing in the description of the diagnostic criteria. Prior to the past year, diagnoses were also measured as syndromes, or the clustering of three or more dependence criteria in the past that occurred (a) most days for at least 1 month, (b) repeatedly for a few months or longer, or (c) around the same time. Respondents classified with a lifetime diagnosis of dependence encompassed all those who had ever experienced an episode of dependence in the past 12 months and/or prior to the past year rather than those who demonstrated the required number of symptoms of these disorders over the life course as is done in the DIS and UM–CIDI.

In a separate test-retest study conducted in the general population, reliability coefficients (kappas) associated with any drug dependence were .79 for the past year and .73 for lifetime. Kappas for age of onset and use variables used in this study ranged from .70 to .84. (Grant, Harford, Dawson, Chou, & Pickering, 1995).

Statistical analysis

The analyses presented in this article included the estimation of 12-month and lifetime prevalences of drug use and dependence, cohort-specific curves for cumulative conditional probabilities of use and dependence, sociodemographic correlates of use and dependence, and conditional probabilities of use and dependence. Prevalence estimates were stratified by gender based on the importance of gender differences in drug use and drug use disorders documented in the preponderance of past research (Anthony & Helzer, 1991; Kessler et al., 1994; U.S. Department of Health and Human Services, 1992; Warner, Kessler, Hughes, Anthony, & Nelson, 1995). Because of the multiple comparisons conducted in this study, the significance level for all tests was set at $p < .001$. To take into account the NLAES sample design, all standard errors of the prevalence estimates presented here were generated using SUDAAN (Research Triangle Institute, 1995), a software program that uses Taylor series linearization to adjust for sample design characteristics.

Cumulative age-at-onset curves for drug use and drug dependence were generated using survival analysis methods (Lee, 1980) with individuals withdrawn from the risk of dependence at the time of their last reported nonmedical use of drugs. Prevalences were estimated within five cohorts for 5-year age intervals (e.g., cumulative conditional probabilities of use by the ages 4, 9, 14, 19, and 24 years among respondents aged 18–24 years at the time of the interview). Cumulative probabilities for ages that exceeded the current ages of some cohort members (e.g., cumulative prevalence by age 24 years in the cohort currently between 18 and 24 years) were based on the at-risk subsample of the cohort at the beginning of the 5-year interval. Because the formula used to calculate the standard errors of these cumulative probabilities assumed a simple random sample, the standard errors in these analyses were adjusted using SUDAAN estimates of the design effects on the simple probability of the outcome variable (i.e., use or dependence).

Associations between drug use and drug dependence and sociodemographic correlates were expressed in terms of odds ratios (OR). Odds ratios and their 95% confidence intervals were derived from separate logistic regression analyses using the SUDAAN LOGISTIC program that also adjusted for the complex sampling design of the NLAES. In each logistic regression, a single demographic variable was treated as a predictor, with drug use or drug dependence serving as the outcome variable.

RESULTS

Prevalence of lifetime and 12-month use

Table 1 presents the lifetime and 12-month prevalence of drug use by age and gender. The overall prevalence of lifetime use of an illegal or nonmedical drug was 15.62%, with 4.91% reporting drug use during the last 12 months. Men were significantly more likely to have used drugs than women both on a lifetime basis (19.53% vs. 12.02%, respectively), t=17.80, $p < .0001$, and a 12-month basis (6.37% vs. 3.56%, respectively),

t=10.80, $p < .0001$. There was also a strong inverse relationship between 12-month use and age, regardless of gender (t tests between 3.3 and 23.9 for all pairwise comparisons, all of them significant at $p < .001$).

For lifetime use, age, years at risk, and cohort were confounded, necessitating the disaggregation of age and cohort effects. In Figure 1, the respondent's age at first drug use is presented in curves for the age-specific cumulative conditional probabilities of lifetime use separately for each of the five birth cohorts in the NLAES. Cohort 1 (born between 1968 and 1974) represents the youngest NLAES respondents, followed by the older respondents in Cohort 2 (born between 1958 and 1967), Cohort 3 (born between 1948 and 1957), Cohort 4 (born between 1938 and 1947), and Cohort 5 (born between 1894 and 1937). It is useful while examining Figure 1 to recognize that respondents in Cohorts 4 and 5, born before or shortly after World War II, would have completed high school by the mid-1960s, that is, before the widespread introduction of illicit drugs into the youth subculture. Cohort 3 went through adolescence in the early years of the youth drug subculture, Cohort 2 at its height, and Cohort 1 after the decline in drug use over the past decade.

The age of onset curve for Cohort 5 was relatively stable, demonstrating low cumulative prevalences of drug use, rising slowly until reaching ages 54 to 98, at the end of which only 1.7% of the cohort had used drugs. For Cohort 4, the cumulative prevalence of drug use did not begin to rise until middle adolescence (15– to 19–year age range), at the end of which time only about 1.8% of the cohort had used drugs. Cumulative prevalence increased to 4.3% by late adolescence (20– to 24–year age range) and continued to rise to 8.1% to the present (50– to 54–year age range).

The curves in the three most recent cohorts consistently showed a dramatic increase in first use between early adolescence (10– to 14–year age range) and late adolescence (20– to 24–year age range), at which time 20.7% of Cohort 3, 26.4% of Cohort 2, and 24.0% of Cohort 1 had experiences with using drugs illicitly or nonmedically. The slopes of the onset curves in Cohorts 1 through 4 become flatter after late adolescence.

The age of onset curve for Cohort 2, who experienced adolescence during the height of the youth drug subculture, was consistently higher than that of its younger Cohort 1 or its older Cohort 3 beginning in the 15– to 19–year–old age range. However, significance tests for intercohort differences in the cumulative probability of use showed that the probability of use was not statistically different between Cohorts 1 and 2 in early, middle, or late adolescence. Contrasts between Cohorts 1 and 3 were found to be statistically significant (z tests between 3.3 and 8.7 for pairwise comparisons, all of them significant at $p < .001$). All other pairwise contrasts between cohorts for each adolescent and adult age range showed significant differences in the cumulative probability of use (z tests between 3.3 and 42.7 for pairwise comparisons, all them significant at $p < .001$), with the exception of early adolescence for Cohort 4 and Cohort 5.

With few exceptions, gender-specific cohort differences mirrored those observed in the total sample. However, among men, there was no significant differences in the cumulative probability of use in late adolescence between Cohorts 1 and 3 and between Cohorts 2 and 3. For women, there were no differences in the cumulative probability of use between Cohorts 3 and 4 and between Cohorts 3 and 5 in early adolescence. In general, the cumulative probability of use for each age range was consistently greater for men than women.

Prevalence of lifetime dependence

The results presented in Table 2 show that 2.91% of the respondents had a lifetime history of drug dependence. Men were significantly more likely than women to have a lifetime history of dependence (3.67% vs. 2.21%, respectively), t =8.10, $p <$. 001, but were no

Table 1. Lifetime and 12-Month Drug Use by Gender and Age

	Men		Women		Total	
Age	Lifetime	12-Month	Lifetime	12-Month	Lifetime	12-Month
Total	19.53 (0.37)	6.37 (0.23)	12.02 (0.25)	3.56 (0.14)	15.62 (0.24)	4.91 (0.14)
18–24	24.23 (1.03)	15.10 (0.99)	16.90 (0.75)	9.09 (0.59)	20.57 (0.65)	12.10 (0.59)
25–34	31.25 (0.83)	10.02 (0.50)	23.23 (0.60)	6.14 (0.36)	27.21 (0.53)	8.07 (0.32)
35–44	29.54 (0.86)	6.33 (0.45)	16.95 (0.60)	3.27 (0.28)	23.17 (0.52)	4.78 (0.26)
45–54	10.39 (0.66)	1.77 (0.29)	4.86 (0.43)	1.40 (0.23)	7.57 (0.39)	1.58 (0.19)
55 +	2.12 (0.23)	0.51 (0.11)	1.27 (0.16)	0.32 (0.09)	1.64 (0.14)	0.41 (0.07)

Note. Standard errors appear in parentheses.

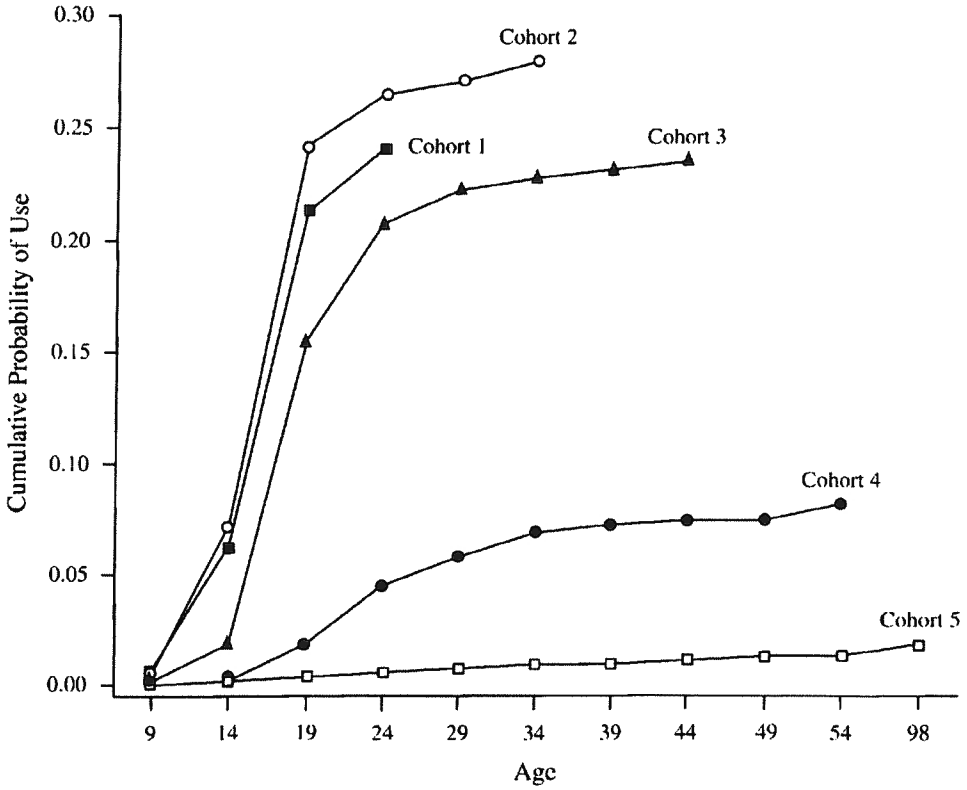
more likely than women to be dependent in the subsample of lifetime users. Thus, the gender differences observed in lifetime dependence were almost entirely due to the increased likelihood among men to have used drugs.

Table 2 also shows an age-cohort effect. In the total sample, respondents in Cohort 4 and Cohort 5 were significantly less likely to report a history of drug dependence than those in the three most recent cohorts (t tests between 3.4 and 15.8 for pairwise comparisons, all of them significant at $p < .001$). Among men and women, respondents in Cohort 4 were significantly less likely than the younger cohorts to have a lifetime history of dependence but were no less likely than the younger cohorts to be dependent in the subsample of lifetime users. In contrast, respondents in Cohort 5 were less likely to have a lifetime history of dependence than the younger cohorts, both in the total sample and in the subsample of lifetime users.

A disaggregation of age and cohort effects observed in Table 2 is shown in Figure 2, where age of onset of drug dependence is presented in curves for age-specific cumulative conditional probabilities of lifetime dependence among users. There was a consistent trend for each successive cohort to have higher prevalences of dependence among users. Only 1.5% and 6.2% of the respondents in Cohort 5 and Cohort 4, respectively, who reported using drugs had a lifetime history of dependence at age 24 compared to 11.7%, 20.0%, and 26.9%, among respondents in the three consecutively younger cohorts (z tests between 3.1 and 12.9 for pairwise comparisons, all of them significant at $p < .001$). Interestingly, the cumulative probability of lifetime dependence among users was greater in the most recent cohort (Cohort 1) than the second most recent cohort (Cohort 2; 26.9% vs. 20.0%, respectively, $z = 2.90$, $p < .001$).

A disaggregation of these cohort differences by gender showed a consistent trend for the probability of lifetime dependence to be generally greater for men than women for all cohorts, with an important exception. This trend was reversed at the latest ages for which data were available for Cohort 4 (at ages 50–54 years), Cohort 3 (at ages 40–44 years), and Cohort 2 (at ages 25–34 years). At these latest ages for which data were available, lifetime dependence was greater among women than men in Cohort 4 (34.9% vs. 21.0%, respectively), Cohort 3 (25.3% vs. 21.1%), and

Figure 1. Cumulative Probability of Lifetime Drug Use, by Birth Cohort



Cohort 2 (32.8% vs. 24.3%). There is further evidence that this trend may be continuing for Cohort 1 in which lifetime dependence among women is approaching that of males in the 20– to 24–year–old age range (25.6% vs. 27.7%, respectively). Among men, the majority of dependence generally occurred by the age of 24 years among Cohorts 1 through 4.

The prevalence of 12-month dependence

The prevalence estimate of 12-month drug dependence was 0.48% in the NLAES sample (Table 3). This represents 9.71% of 12-month users and 8.60% of respondents with a history of dependence prior to the past year. The overall percentage of respondents with 12-month dependence was significantly greater for men than women (0.61% vs. 0.35%, respectively), t= 3.30, p< .001.This is because men are more likely than women to use drugs at some time in their lives, but they are no more likely than women to become dependent once they have used them nor are they more likely than women to have their dependence persist once developed. That is, there were no gender differences in 12-month dependence among the subsample of 12-month users or among those respondents who had dependence in the past.

Table 3 shows that 12-month dependence was significantly more prevalent among respondents in the 18– to 24–year age range (1.47%) than among those who were 25 to 34 years old (0.74%, t = 3.70, p < .0001), 35 to 44 years old (0.36%, t = 5.80, p < .0001), 45 to 54 years old (0.15%, t = 7.00, p < .0001), and those 55 years and older (0.00%, t = 8.20, p < .0001). As shown in the last two columns of Table 3, this association results from the younger respondents being more likely to be recent drug users, more likely to become dependent, and more likely to persist in dependence once it has developed. That is, there were significant differences in 12-month dependence between the youngest and older cohorts in the subsample of 12-month users (t tests between 3.3 and 7.0 for pairwise comparisons, all of them significant at p < .001) and those respondents who had a history of dependence prior to the past 12 months (t tests 3.0 to 4.1 for pairwise comparisons, all of them significant at p < .001).

Other sociodemographic correlates

In Table 4, a larger set of sociodemographic correlates are considered relative to drug use and dependence. The contrasts presented in Table 4 focus on three stages of progression: (a) predictors of first use (lifetime use in the total sample), (b) predictors of first onset of dependence (lifetime dependence in the subsample of lifetime users), and (c) persistence with dependence (12-month dependence in the subsample of persons with prior to the past year dependence).

As can be seen in Table 4, whites were significantly more likely than blacks or hispanics to use drugs at some time in their lives. Although there was no significant difference between race and dependence in the subsample of lifetime users, both blacks and hispanics were significantly more likely to have 12-month dependence given that they had dependence in the past.

Respondents with 12 or fewer years of education were significantly less likely to have used drugs than the most highly educated respondents (16 or more years). Respondents with some college education (13–15 years) were more likely than the most highly educated respondents to have used drugs at some time in their lives. This pattern reversed, however, in predicting progression to dependence, with the least educated respondents (12 or fewer years) more likely to become dependent compared to the most highly educated. The odds for persistence of dependence was about two times (OR = 1.81) as great among the least educated (less than 12 years) compared to the most highly educated respondents.

Table 2. Lifetime DSM–IV Drug Dependence in the Total Sample and Among Lifetime Users, by Gender and Age

	Men		Women		Total	
Age	Total Sample	Lifetime Users	Total Sample	Lifetime Users	Total Sample	Lifetime Users
Total	3.67 (0.16)	18.79 (0.72)	2.21 (0.11)	18.42 (0.79)	2.91 (0.10)	18.64 (0.55)
18–24	4.91 (0.49)	20.24 (1.90)	3.31 (0.40)	19.59 (2.16)	4.11 (0.32)	19.97 (1.48)
25–34	6.46 (0.40)	20.68 (1.15)	5.12 (0.32)	22.05 (1.26)	5.79 (0.26)	21.27 (0.86)
35–44	5.18 (0.42)	17.52 (1.27)	2.47 (0.24)	14.60 (1.30)	3.81 (0.24)	16.44 (0.95)
45–54	1.66 (0.25)	15.93 (2.33)	0.66 (0.14)	13.50 (2.76)	1.15 (0.14)	15.13 (1.79)
55 +	0.13 (0.06)	6.21 (2.73)	0.08 (0.04)	6.08 (2.71)	0.10 (0.03)	6.15 (1.95)

Note. Standard errors appear in parentheses.

The odds of lifetime drug use and persistence of dependence were about two times greater (OR = 1.63) and three times greater (OR = 2.97), respectively, among never married respondents relative to married respondents. Respondents who were separated, divorced, or widowed were also at increased risk of dependence (OR = 1.29).

Respondents with household incomes of less than approximately $36,000.00 per year were significantly less likely to use drugs at some time in their lives relative to the highest income group. However, respondents in the three lower income groups were all more likely (ORs =1.51 to 2.15) to become dependent than the highest income group.

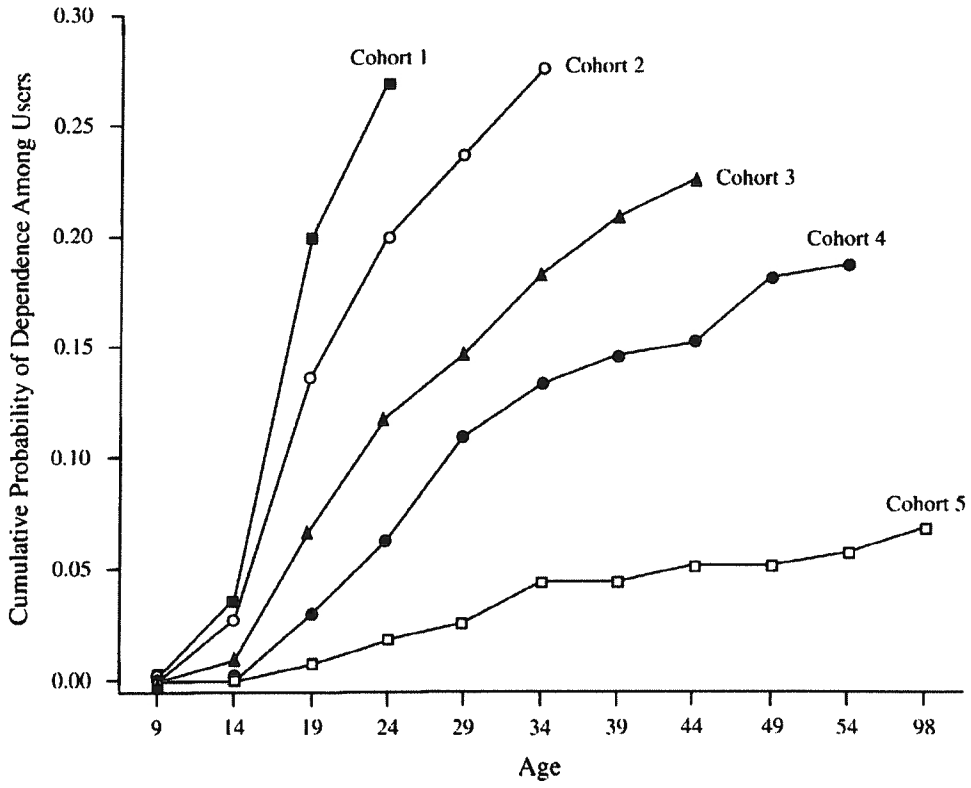
Lifetime probability of drug use was significantly higher in the west and midwest than in the south. Given use, respondents in the west and northeast had a higher odds of dependence, but there were no significant regional differences in the persistence of dependence. Urban respondents had greater odds of lifetime use compared to their rural counterparts, whereas there were no significant associations between urbanicity and the onset or persistence of dependence.

DISCUSSION

Prevalence of drug use and dependence

Although it is difficult to directly compare the NLAES estimates of drug use with those of previous surveys, the comparison is of sufficient interest to researchers to be presented here. The NLAES drug use prevalence and estimates were lower than those reported in the NHSDA, NCS, and ECA, primarily because the NLAES excluded nonmedical drug users who had not used any drug in any of the relevant drug classes less than 12 times in their lives. The NHSDA and NCS included use of any drug at least once, whereas the ECA defined lifetime users as respondents who had used any drug at least five times in their lives. The NCS definition of lifetime drug use was also unique in including respondents who were taking a drug according to their physician's instructions but nonetheless felt dependent. The NLAES estimate for lifetime use (15.6%) was about half that reported in NHSDA (36.2%) and the ECA (30.5%) and about one third of that reported in the NCS (51.0%). Comparisons of 12-month use between NLAES and the two surveys for which data are avail-

Figure 2. Cumulative Probability of Lifetime Drug Dependence, Given Use, by Birth Cohort



able followed a similar pattern: 4.9% in NLAES; 11.1% in NHSDA; and 15.4% in NCS (Anthony & Helzer, 1991; U.S. Department of Health and Human Services, 1992; Warner et al., 1995).

It is also difficult to make comparisons to assess the consistency of NLAES estimates of the prevalence of drug dependence with those of the earlier ECA and NCS surveys because of differences between the surveys in diagnostic criteria and the psychiatric interviews used to operationalize them. However, the prevalence of 12-month drug dependence in NLAES (0.5%) was similar to that reported in the ECA (0.8%), but somewhat lower than that reported in the NCS (1.8%). The prevalence of lifetime dependence was also similar between the NLAES and ECA (2.9% vs. 3.5%, respectively), but again somewhat lower than that reported in the NCS (7.3%).

Gender, age, and cohort effects

The NLAES finding that men were more likely than women to use drugs is consistent with the majority of the previous empirical literature (Anthony & Helzer, 1991; Johnston et al., 1993; U.S. Department of Health and Human Services, 1992; Warner et al., 1995). The finding that men who used drugs were indistinguishable from women who used drugs with regard to becoming dependent is consistent with the ECA finding that lifetime drug use disorders were equally prevalent among men and women classified as lifetime users (Anthony & Helzer, 1991) but at odds with NCS results (Warner et al., 1995). Additionally, the NLAES found no gender difference in the persistence of dependence, that is, in the prevalence of 12-month dependence among the prior to the past year dependent subsample. Although the NCS found a similar result, persistence of dependence in that survey was defined incorrectly as the prevalence of 12-month dependence among the lifetime dependent subsample when in fact it should have been defined as the prevalence of 12-month dependence among the prior to the past year dependent subsample. Because the prior to the past year prevalence of drug dependence was not defined or measured in the NCS, the corresponding appropriate subsample could not be constructed to assess the persistence of dependence.

The inverse relationship between age and use found in this study was consistent with previous research (Robins & Przybeck, 1985; U.S. Department of Health and Human Services, 1992; Warner et al., 1995) as was the finding that use and dependence were both much more common among cohorts born after World World II (Anthony, Warner, & Kessler, 1994; Burke, Burke, Rae, & Regier, 1991; Johnston, 1985; U.S. Department of Health and Human Services, 1992; Warner et al., 1995). However, this lower prevalence of lifetime dependence in Cohort 4 resulted entirely from respondents in Cohort 4 being

Table 3. Twelve-Month DSM-IV Drug Dependence in the Total Sample, Among 12-Month Users, and Among Respondents With Prior to the Past Year Dependence

Gender/Age	Total Sample	12-Month User Subsample	Prior to the Past Year Dependent Subsample
Men	0.61 (0.06)	9.58 (0.94)	8.90 (1.22)
18–24	2.00 (0.30)	13.24 (1.91)	23.43 (4.67)
25–34	0.80 (0.14)	7.98 (1.33)	6.35 (1.46)
35–44	0.47 (0.12)	7.44 (1.79)	5.31 (1.73)
45–54	0.15 (0.06)	8.28 (3.40)	6.88 (3.26)
55 +	0.01 (0.01)	2.17 (1.03)	8.44 (6.89)
Women	0.35 (0.04)	9.93 (1.15)	8.15 (1.43)
18–24	0.95 (0.21)	10.43 (2.21)	16.30 (5.10)
25–34	0.67 (0.11)	10.97 (1.73)	6.69 (1.55)
35–44	0.26 (0.07)	7.99 (2.15)	5.00 (1.95)
45–54	0.15 (0.07)	11.05 (4.67)	10.20 (5.09)
55 +	0.00 (0.00)	0.00 (0.00)	0.00 (0.00)
Total	0.48 (0.04)	9.71 (0.71)	8.60 (0.92)
18–24	1.47 (0.18)	12.18 (1.43)	20.39 (3.45)
25–34	0.74 (0.01)	9.13 (1.03)	6.50 (1.05)
35–44	0.36 (0.07)	7.63 (1.38)	5.21 (1.33)
45–54	0.15 (0.04)	9.53 (2.77)	7.76 (2.75)
55 +	0.00 (0.00)	1.21 (0.98)	4.83 (3.26)

much less likely than the younger respondents to have ever used drugs, a result consistent with the NCS (Warner et al., 1995). However, in the NCS, this decreased prevalence in Cohort 4 was the result of decreased likehood of using drugs among women, but among men it was the result of a decreased likelihood of both using drugs and becoming dependent. The NLAES analyses found no such gender difference in Cohort 4. In contrast, the lower prevalence of lifetime dependence in Cohort 5 was due to the decreased likelihood of using drugs and becoming dependent relative to the younger cohorts.

Women were significantly more likely than men to have lifetime dependence in the oldest age groups of Cohort 4 (50– to 54–year–olds), Cohort 3 (40– to 44–year–olds), and Cohort 2 (25– to 34–year–olds), whereas men had greater prevalences of lifetime dependence than women in the younger age groups. A slightly weaker form of this interaction was found in the ECA and NCS (Anthony & Helzer, 1991; Warner et al., 1995). The fact that there is a dramatic increase in the prevalence of lifetime drug dependence that accompanies each of those older age groups, in which rates among women exceed those of men, is of some

Table 4. Demographic Correlates of Lifetime Use in the Total Sample, Lifetime Dependence Among Lifetime Users, and 12-Month Dependence Among Respondents With Prior to the Past Year Dependence

Demographic Characteristic	Lifetime Use (Total Sample)		Lifetime Dependence (User Subsample)		12-month Dependence (Prior to the Past Year Dependence Subsample)	
	OR	95% CI	OR	95% CI	OR	95% CI
Ethnicity						
White	1.00	—	1.00	—	1.00	—
Black	0.72[a]	(0.64, 0.81)	0.97	(0.74, 1.26)	2.33[a]	(1.23, 4.44)
Hispanic	0.59[a]	(0.51, 0.69)	0.86	(0.61, 1.19)	3.50[a]	(1.77, 6.92)
Education (Years)						
Less than 12 years	0.55[a]	(0.49, 0.61)	1.54[a]	(1.26, 1.91)	1.81[a]	(1.05, 3.10)
12 years	0.84[a]	(0.77, 0.92)	1.31[a]	(1.14, 1.52)	1.07	(0.67, 1.71)
13-15 Years	1.17[a]	(1.07, 1.27)	0.92	(0.79, 1.07)	0.80	(0.48, 1.32)
16+ years	1.00	—	1.00	—	1.00	—
Marital Status						
Married	1.00	—	1.00	—	1.00	—
Separated/Divorced/ Widowed	0.88[a]	(0.81, 0.96)	1.29[a]	(1.08, 1.54)	0.88	(0.52, 1.47)
Never Married	1.63[a]	(1.51, 1.74)	1.03	(0.89, 1.19)	2.97[†]	(1.86, 4.75)
Family Income						
Less than $20,400.00	0.79[a]	(0.71, 0.88)	2.15[a]	(1.62, 2.86)	2.05	(0.72, 5.88)
$20.401.00 - $35,988.00	0.92[a]	(0.83, 0.92)	1.89[a]	(1.41, 2.53)	1.03	(0.34, 3.13)
$35,989.00 - $71,988.00	1.04	(0.93, 1.16)	1.51[a]	(1.13, 2.02)	0.68	(0.21, 2.17)
$71,989.00 +	1.00	—	1.00	—	1.00	—
Region						
Northeast	1.09	(0.98, 1.21)	0.72[a]	(0.60, 0.86)	1.01	(0.54, 1.88)
Midwest	1.25[a]	(1.14, 1.38)	0.92	(0.77, 1.09)	0.78	(0.43, 1.41)
South	1.00	—	1.00	—	1.00	—
West	1.79[a]	(1.62, 1.97)	1.31[a]	(1.13, 1.53)	0.98	(0.61, 1.57)
Urbanicity						
Rural	1.00	—	1.00	—	1.00	—
Urban	1.39[a]	(1.28, 1.49)	0.93	(0.77, 1.12)	0.97	(0.54, 1.75)

Note. OR indicates odds ratio; CI indicates confidence interval.
[a] An odds ratio with a confidence interval that does not include 1.0 is statistically significant.

significance. This result suggests that women in Cohort 3 and Cohort 4, who were not exposed to the widespread introduction of illicit drug use in the mid-1960s throughout their entire adolescence, may have been predominately dependent on psychotherapeutic medicines at older ages. The majority of men in each of these cohorts had become dependent by the age range of 30 to 34 years, suggesting further that their dependence may relate to dependence on other illicit drugs.

In contrast, women experiencing adolescence at the height of the introduction of widespread illicit drug use among youth had surpassed the men in the probability of dependence among the younger age ranges (25– to 29–year age range).

There is also an indication that the same phenomenon may be happening among the youngest cohort, in which the probability of lifetime dependence is essentially equivalent among men and women in the 20– to 24–year age ranges. Whether women in Cohorts 1 and 2 sustain their dominance in dependence over men as they grow older or develop two periods of increased risk for dependence awaits the passage of time. Meanwhile, planned drug-specific analyses may help to clarify the gender-by-cohort interaction in dependence among users found in this study.

This study replicated the finding in the NCS that the conditional probability of dependence among users was greater among the youngest Cohort 1, currently aged 18 to 24 years, compared with Cohort 2, despite the finding that the probability of lifetime use was lower in Cohort 1 compared to Cohort 2 (Warner et al., 1995). This result may be due to differential recall bias or reporting. It may also be the case that respondents in Cohort 1, who are selected into drug use at a time when it is becoming less prevalent, may be more vulnerable to dependence as the result of being more likely to have a history of other psychopathology (Warner et al., 1995; Weiss, Mirin, Griffin, & Michael, 1988). Alternatively, there may be an increased dependence liability associated with currently used illicit drugs of choice (e.g., crack cocaine), along with associated reductions in cost and increased opportunities for use and relative drug availability.

A related finding was that there was a significantly greater prevalence of 12-month dependence in Cohort 1 compared to the older cohorts, which was due to younger people being more likely to be recent users, become more dependent, and to persist in dependence. This finding was at odds with the result from the NCS that the greater prevalence in 12-month dependence among Cohort 1 compared to the older cohorts was the result of younger people being more likely to be recent users (Warner et al., 1995). An age difference in drug of choice could help explain this result, particularly if the more recently used drugs of choice have a potentially greater dependence liability than those used in the past. Again, drug-specific analyses may help to elucidate this finding further.

Other sociodemographic correlates

Consistent with previous research, drug use was found to be positively correlated with being white rather than nonwhite, more highly educated than less educated, never married than married, an urban rather than a rural resident, and a resident of the midwest or west rather than the south or northeast (Anthony et al., 1994; U.S. Department of Health and Human Services, 1992).

The relationships between drug use and the various sociodemographic correlates varied as a function of the stage of the disorder. For example, whites were more likely to use drugs than blacks and hispanics, were indistinguishable from blacks and hispanics in their probability of dependence, but were significantly less likely than blacks or hispanics to persist in dependence. Being more poorly educated or of lower income was not predictive of lifetime drug use but was related to increased odds of dependence relative to the more highly educated and higher income respondents, respectively.

Implications and limitations

The findings of this study underscore the importance of collecting data on drug dependence as well as drug use. If data exclusively on drug use were collected in the NLAES, inaccurate conclusions would be drawn concerning the magnitude of the drug problem in this country. For example, the conclusion would have been drawn that drug use had become less prevalent in the youngest cohort (aged 18 to 24 years) compared to their predecessors who had experienced adolescence during a period of widespread illicit drug use. However, the observation that the conditional probability of dependence was greater among respondents in Cohort 1 compared to Cohort 2 would have been lost. As a result of this, public policy decisions and prevention efforts might have been misdirected and, at least, less than entirely informed. In this time of increasing concerns about government expenditures for health care, the distinction between drug use and drug dependence demands further consideration.

The NLAES has provided data on the prevalence and location of drug use and dependence in the United States and the transition from drug use to dependence, information of particular public health import. However, similar to most, if not all, national surveys of drug use and dependence, the NLAES was a household population survey that excluded the homeless and those

residing in most institutional settings (e.g., jails, prisons, group homes). As a result of this, estimates of drug use and dependence are likely to be conservative and to underestimate true prevalence. Although the AUDADIS was designed with the goal of achieving more reliable and accurate measures of drug use, drug dependence, and their associated onsets, the self-report nature of the measures is always subject to some degree of recall bias. Finally, this study uses a descriptive epidemiologic approach focusing on the bivariate relationships between individual sociodemographic correlates and drug use and dependence. Future analyses conducted within a multivariate environment should elucidate the important interrelationships between correlates of drug use and dependence for each progressive stage of the disorder.

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Age of Onset of Drug Use and Its Association With DSM–IV Drug Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant and Deborah A. Dawson

The purpose of this study was to examine the relationship between early onset drug use and the development of lifetime DSM-IV drug abuse and dependence using a representative sample of the U.S. population. Prevalences of lifetime drug abuse and dependence were estimated for each year of age of onset of drug use from ages 13 and younger to 21 and older for the overall sample of drug users by race and gender. Linear logistic analyses were conducted to assess the relationship between age of drug use onset and lifetime drug use disorders controlling for important covariates. The major finding of this study was that early onset drug use is a significant predictor of the subsequent development of drug abuse over the life course. Early onset drug use was also a significant predictor of the subsequent development of lifetime alcohol dependence among males, females, and nonblacks, but not among blacks. After adjusting for important model covariates, the likelihood of lifetime drug abuse and dependence among the total sample of lifetime drug users was reduced by 4% and 5% with each year drug use onset was delayed. Implications of these findings are discussed in terms of the importance of collecting national data on drug use, abuse and dependence and the need for further research and its integration with prevention efforts.

Although much has been learned over the past two decades about the onset of drug use among adolescents and the subsequent development of adverse consequences of drug involvement in young adulthood, a major gap remains in our understanding of the impact of age of onset and the development of serious long-term consequences, such as drug abuse and dependence. Early onset drug use has a major impact on adolescent morbidity, mortality and performance of age-related social roles, particularly work and family. Among adolescents and young adults, early onset drug use has been associated with motor vehicle fatalities and injuries (U.S. Congress, 1991), infrequent condom use and teen pregnancy (DiClemente, 1992; Epstein and Tamar, 1984), sexually-transmitted diseases (Shafer and Boyd, 1991), depression (Burke, Burke, and Rae, 1994; Deykin et al., 1987) and marital and work instability and delinquency (Bachman, O'Malley, and Johnson, 1985; Clayton, 1982; Kandel, Davis, Karus, and Yamaguchi, 1986).

Our current lack of understanding about the influence of age of onset of drug use and the development of the long-term consequences of drug abuse and dependence can be largely attributed to one factor. Major national surveys that have monitored drug use and trends since the early 1970s among adolescents, young adults and the U.S. civilian noninstitutionalized population, such as the Monitoring the Future Survey (Johnson, O'Malley, and Bachman, 1993) and the National Household Survey on Drug Abuse (NHSDA: Substance Abuse and Mental Health Services Administration, 1992), have measured drug use to the exclusion of drug abuse and dependence.

While the failure to differentiate drug use, abuse and dependence may be justified because of the illegal status of drugs and the consistent research finding that most drug use among adolescents is transitory, recent results of a national survey of adults have underscored the critical nature of these distinctions. Data from the National Institute on Alcohol Abuse and Alcoholism-sponsored National Longitudinal Alcohol Epidemiologic Survey (NLAES: Grant, Peterson, Dawson, and Chou, 1994; Massey, Moore, Parsons, and Tadros, 1989) have shown that 15.6% of the U.S. adult population (approximately 29 million

Reprinted from Journal of Substance Abuse, *Volume 10, Number 2, pp. 163–173, 1998, with permission from Elsevier Science.*

Americans) will engage in nonmedical or illicit drug use at some time in their lives, with 76% of the onset of such use occurring under the age of 19 years (Grant, 1996). Of those who have ever used drugs, approximately 20% (5.8 million Americans) will go on to develop drug abuse and 19% (5.4 million Americans) will go on to develop drug dependence at some time in their lives. Clearly, the finding that 40% of all persons who use drugs will go on to develop drug abuse and/or dependence is significant enough to distinguish drug use from drug abuse and dependence. Moreover, drug abuse and dependence are very serious conditions with their own associated physical, social and psychological consequences for the individual. In addition abuse and dependence have far reaching effects on society, including crime, hospitalization and economic costs associated with lost productivity.

Although the age at first alcohol use has recently been shown to be a powerful predictor of lifetime alcohol abuse and dependence (Grant and Dawson, 1997), very few attempts have been made to examine the relationship of early onset drug use in relation to the subsequent development of long-term consequences, including drug abuse and dependence. In one study, 15 year-olds were followed for 13 years and the onset of drug use was not found to be predictive of the development of drug-related adverse consequences at age 30 (Labouvie, Base, and Pandina, 1997). Implications of this finding remain limited for several reasons, including the exclusion of females from the sample, the measurement of adverse drug-related consequences for one point in a person's lifetime (i.e., during the three years prior to age 30), and the use of an aggregate measure of drug-related consequences that cannot directly be related to clinically significant definitions of drug abuse and dependence.

In another study, conducted in the general population, it was found that the earlier drug use begins, the greater the risk of drug abuse and dependence (Robins and Przybeck, 1985), a finding supporting an earlier study of young black men (Robins and Murphy, 1967). However, the sample in this study was restricted to 18-to-35 year-olds in St. Louis, thereby raising questions about the generalizability of the findings. In addition, the diagnostic criteria used to classify persons as abusers or dependent are now historic (i.e., the *Diagnostic and Statistical Manual of Mental Disorders,* 3rd ed.) and, more importantly, bear little resemblance to modern definitions of drug use disorders (Hasin, McCloud, and Endicott, 1996). Last, the definitions of drug abuse and dependence used in this study were made on the basis of drug-related symptoms occurring over the life course, with no requirement that the symptoms be long-lasting or cluster in time, thereby raising questions about the clinical significance and relevance of the drug use disorders as measured.

STUDY

Aim

The purpose of the present study was to directly examine the relationship between the age of onset of drug use and the prevalence of lifetime drug abuse and dependence. To our knowledge, no other study has determined the likelihood of drug abuse and dependence as a function of age of onset of drug use in a large representative sample of the U.S. population, using reliable measures based on the most current psychiatric classification, the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV: American Psychiatric Association, 1992).

METHOD

Study sample

This study was based on the National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). Fieldwork for the study was conducted by the United States Bureau of the Census in 1992. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. Approximately 92% of the selected households participated in this survey, and 97% of the randomly selected respondents in these households participated in this survey.

The NLAES utilized a complex multistage design which featured sampling of primary sampling units with probability proportional to size and oversampling of the black and young adult (18-to-29 years) populations. The NLAES design has been described in detail elsewhere (Grant et al., 1994; Massey et al., 1989).

Drug use, abuse and dependence assessment

The survey instrument used in the NLAES was the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS: Grant and Hasin, 1992). In the AUDADIS, illicit or nonmedical drug

use was defined as use: (1) without a physician's prescription; (2) in greater amounts than prescribed; (3) more often than prescribed; (4) longer than prescribed; or (5) for a reason other than prescribed. Information on nonmedical drug use was ascertained separately for sedatives, tranquilizers, opioids other than heroin, amphetamines, cocaine (and crack cocaine), cannabis (and THC and hashish), heroin, methadone, hallucinogens and other drugs of abuse (e.g., inhalants, solvents). Lifetime drug use, for the purpose of this study, was defined as ever having tried any of the drugs in any of the above drug classes at least twelve times.

In the AUDADIS, DSM-IV lifetime diagnoses of drug abuse and dependence were initially derived separately for each illicit drug and then combined into aggregate measures of any drug abuse and any drug dependence used in this study. An AUDADIS diagnosis of DSM-IV abuse required that a person meet at least one abuse criterion in any one year of their lives including: (1) continued use despite a social or interpersonal problem caused or exacerbated by the effects of use; (2) recurrent use in situations in which drug use is physically hazardous; (3) recurrent use resulting in a failure to fulfill major role obligations; or (4) recurrent drug-related legal problems. Consistent with DSM-IV, AUDADIS diagnoses of drug dependence required that a person meet at least three of the seven criteria defined for dependence in any one year including: (1) tolerance; (2) withdrawal or avoidance of withdrawal; (3) using a drug in larger amounts or over a longer period than intended; (4) persistent desire or unsuccessful attempts to cut down or stop using; (5) spending much time obtaining a drug, using it, or recovering from its effects; (6) giving up or reducing occupational, social or recreational activities in favor of drug use; or (7) continuing to use despite a physical or psychological problem caused or exacerbated by drug use.

The AUDADIS diagnoses of 12-month drug abuse and dependence satisfied both the clustering and duration criteria of the DSM-IV classification. The clustering criterion requires the clustering of symptoms within the year preceding the interview. The duration criterion is defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often," and "persistent," appearing in the description of the diagnostic criteria. Prior to the past year diagnoses were also measured as syndromes, or the clustering of the required number of symptom criteria for abuse or dependence in the past that occurred: (1) most days for at least one month; (2) repeatedly for a few months or longer; or (3) around the same time. Respondents classified with a lifetime diagnosis encompassed all those who had ever experienced an episode of either abuse or dependence in the past year and/or before the past year. The DSM-IV abuse and dependence groups were mutually exclusive. Respondents classified as lifetime drug abusers did not meet criteria for lifetime dependence, whereas respondents classified with lifetime dependence included those with or without abuse diagnoses at some time in their lives.

In a separate test-retest study conducted in the general population, reliability coefficients (kappas) associated with any drug abuse or dependence were 0.79 for the past year and 0.73 for lifetime. Kappas for age of onset of drug use used in this study were 0.68 and 0.69 for heroin and sedatives, 0.77 for cocaine, and exceeded 0.92 for tranquilizers, amphetamines, opioids (other than heroin) and cannabis (Grant et al., 1995).

In this study, family history of a drug use disorder was not measured for first-degree relatives. However, data on the family history of alcoholism among first-degree relatives were ascertained in this study and used as a control variable in the logistic regression analyses assessing the impact of age of onset of drug use on the odds of lifetime drug abuse and dependence. Support for the decision to include family history of alcoholism in these analyses is based on evidence that suggests that family history of alcohol abuse and dependence is a risk factor for drug abuse and dependence (Eisen, Grob, and Dill, 1987).

Family history of alcoholism was ascertained through a series of questions that asked about each type of first-degree biological relative. For each type of relative, the respondent was asked how many relatives of that type lived to be at least ten years old and how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent in a manner consistent with the DSM-IV criteria for alcohol use disorders: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover." In a test-retest study conducted in conjunction with the pretest for the NLAES, the family history items generally showed good to excellent reliability, with kappas of 0.70 or higher for most types of first-degree relatives (e.g., 0.72 for fathers, 1.00 for mothers, 0.90 for brothers, 0.73 for sisters). Slightly

lower kappa values were obtained for sons and daughters (0.65 for each). The family history measure in this study was considered positive if any first-degree relative was reported as having been alcoholic or problem drinker at some time in their lives.

Statistical analysis

The analysis consisted of two parts. First, prevalences of lifetime drug abuse and dependence were estimated for each year of age at onset of drug use from ages 13 and younger to 21 and older for the overall sample and separately by gender (male and female) and race (nonblack and black). Sample sizes were not sufficient to examine gender by race subgroups of the population. Second, linear logistic regression analyses were used to assess the relationship between age at onset of drug use and the odds of lifetime drug abuse or dependence, controlling for the effects of sex, race, age, duration of drug use, family history of alcoholism and drug use status, that is, current (past year) drug user versus former drug user (used a drug or drugs in the past, but not in the past year). All analyses were conducted using SUDAAN, a software package that uses Taylor series linearization to adjust for the complex design of the NLAES (Research Triangle Institute, 1997).

RESULTS

Approximately 16% *(n* = 6,447) of the NLAES sample were classified as lifetime drug users (Table 1). Males and nonblacks were more likely to have used drugs on a lifetime basis than their female and black counterparts, respectively. Among lifetime drug users, about 20% (*n* = 1,273) went on to develop drug abuse and 19% *(n* = 1,273) went on to develop drug dependence at some time in their lives. Although males and nonblacks were more likely to develop drug abuse than either females and blacks, respectively, lifetime drug dependence was equally prevalent among males and females and among blacks and nonblacks.

Table 2 and Figure 1 show the prevalences of lifetime drug abuse for each year of age at onset of drug use from ages 13 and younger to ages 21 and older. Overall, the prevalence of lifetime drug abuse declined as a function of increasing age at onset of drug use. In the total sample of drug users, almost 30% of the respondents who initiated drug use before 13 years of age were classified with drug abuse at some time in their lives. The corresponding prevalence among those who started using drugs at age 17 was 20.3%, decreasing steadily to 11.1% among those who started using drugs at ages 21 and older. The downward trend in the prevalence of abuse as a function of increasing age of onset of drug use observed in the total sample of drug users was similar to that observed within each gender and racial subgroup. At most ages of onset of drug use, males were more likely to develop abuse than females and nonblacks were more likely to develop abuse than blacks.

Table 3 and Figure 2 present the prevalence of lifetime drug dependence as a function of age at initiation of drug use. Similar to the finding for drug abuse, the prevalence of lifetime dependence decreased steeply with increasing age of onset of drug use. Overall, the prevalence of lifetime dependence among those who started using drugs under the age of 14 years was about 34%, dropping sharply to 15.1% for those initiating use at age 17 years to about 14% among these initiating use at ages 21 years and older. The downward trend of alcohol dependence as a function of increasing age of onset was remarkably consistent for males, females and nonblacks. Although the lifetime prevalence of dependence also decreased between ages 13 and younger (28.9%) to ages 21 and older (17.8%) among blacks, age of onset of drug use was not uniform across these years.

Multivariate linear logistic analyses were conducted to assess the contribution of age at onset of drug use to the odds of lifetime drug abuse and dependence, controlling for the effects of gender, race, age, duration of drinking, drug use status, and family history of alcoholism (Table 4). Age at first drug use was entered into this analysis as a continuous measure. After adjusting for these other factors, age at onset of drug use remained a major and significant contributor to the development of drug abuse and dependence among the total sample of drug users. Specifically the odds of lifetime drug dependence were reduced by 4% with each additional year that drug use onset was delayed. Similarly, the odds of lifetime drug abuse were reduced 5% with each additional year that drug use onset was delayed.

When multivariate analyses were conducted among each gender and race subgroup, slight differences were found in the contribution of age at onset of drug use to the odds of lifetime drug abuse and dependence. Among males, females and nonblacks the odds of lifetime alcohol abuse were reduced 4%, 6%, and 5%, respectively, with each additional year that drug use was delayed, while the corresponding reduction among blacks was somewhat lower (2%). The odds of lifetime dependence were reduced 5%, 2% and 4% among males, females, and nonblacks respectively, but among blacks, age of onset did not significantly impact on the development of subsequent lifetime dependence.

DISCUSSION

Age at first drug use is a powerful predictor of lifetime drug abuse among males, females, blacks and nonblacks. Age of onset of drug use was also a powerful predictor of lifetime drug dependence among males, females, nonblacks, but not among blacks. After using multivariate techniques to adjust for other important factors, the odds of lifetime drug abuse and dependence in the total sample of drug users were reduced by 5% and 4%, respectively, for each additional year that drug use onset was delayed.

Although there was a consistent decline in the prevalence of drug abuse and drug dependence across gender and race subgroups of drug users, the prevalence of lifetime abuse, but not lifetime dependence, was greater among males compared to females and greater among nonblacks compared to blacks at each year of drug use onset. Although the interpretation of this finding is as yet unclear, this result highlights the need for research on gender and racial minorities and other groups whose unique cultural traditions, drug use patterns, and life experiences may impact on the relationship between drug use onset and the development of drug abuse.

The decline in the prevalence of drug dependence with each increasing year of age of onset of drug use was not uniform among blacks. For blacks, the age of onset of drug use was not significantly related to lifetime drug dependence. The smaller number of black drug users relative to the number of drug users among other subgroups examined in this study may, in part, be responsible for this result. This finding may also suggest that the age of onset of drug use among blacks may relate to specific drugs used that may differ in dependence liability. Although the present study cannot conclusively confirm this interpretation, examination of drug-specific onset data deserves further study.

A significant contribution of this study is the focus it provides on the important role of epidemiological and other etiological research in guiding prevention efforts. Many research questions remain unanswered that would form the basis of successful prevention strategies. Foremost is our lack of complete understanding of why early onset drug use is related to the development of drug abuse and dependence. Is it the delay in

Table 1. Prevalence[1] of Lifetime Drug Use in the Total Sample and Lifetime DSM-IV Drug Abuse and Dependence Among Lifetime Drug Users by Gender and Race

Sociodemographic Characteristic	*Lifetime Drug Use in Total Sample*	*Drug Abuse Among Lifetime Drug Users*	*Drug Dependence Among Lifetime Drug Users*
Total	15.6 (0.2)	20.1 (0.6)	18.6 (0.6)
Male	19.5 (0.4)	22.6 (0.9)	18.8 (0.7)
Female	12.0 (0.3)	16.3 (0.8)	18.4 (0.8)
Black	12.3 (0.6)	13.9 (1.7)	18.1 (1.9)
Nonblack	16.0 (0.3)	20.7 (0.7)	18.7 (0.6)

Notes. Standard errors shown in parentheses.
1. Prevalences expressed as weighted figures.

Table 2. Age at First Drug Use and the Prevalence[1] of Lifetime DSM-IV Drug Abuse by Gender and Race

Age at First Drug Use (In Years)	*Male (n = 783)*	*Female (n = 490)*	*Black (n = 97)*	*Nonblack (n = 1,176)*	*Total (n = 1,273)*
13 and younger	30.8 (2.6)	25.5 (2.7)	16.8 (5.9)	29.8 (2.1)	28.9 (1.9)
14	26.7 (3.5)	15.5 (2.3)	30.3 (10.3)	20.7 (2.2)	21.5 (2.2)
15	26.8 (2.7)	20.8 (2.8)	11.7 (4.8)	25.3 (2.1)	24.3 (1.9)
16	28.8 (2.5)	19.5 (2.3)	12.9 (4.2)	26.0 (1.8)	25.1 (1.7)
17	23.4 (2.4)	16.0 (2.1)	16.9 (4.8)	20.7 (1.8)	20.3 (1.7)
18	19.3 (2.1)	15.1 (2.0)	10.9 (4.7)	18.5 (1.5)	17.7 (1.5)
19	19.2 (3.4)	15.6 (2.7)	11.6 (4.1)	18.8 (2.6)	18.0 (2.4)
20	17.9 (2.9)	6.0 (1.8)	10.4 (5.0)	13.9 (2.1)	13.6 (2.0)
21 and older	13.9 (1.6)	11.7 (1.5)	12.6 (3.2)	13.0 (1.2)	11.1 (0.9)

Notes. Standard errors appear in parentheses.
1. Prevalences expressed as a weighted figures, ns presented as unweighted figures.

the onset of drug use itself, or more likely a combination of additional factors, that reduces the risk for the subsequent development of drug use disorders? Is early onset use a critical and potentially modifiable risk factor in development of drug use disorders, or alternatively, a marker or early indicator of the inevitable, perhaps unmodifiable development of the disorders? Will the delay of drug use onset have any unintended or adverse effect? Both the goals and strategies of drug use prevention and intervention efforts are likely to be very different depending on the answers to these questions.

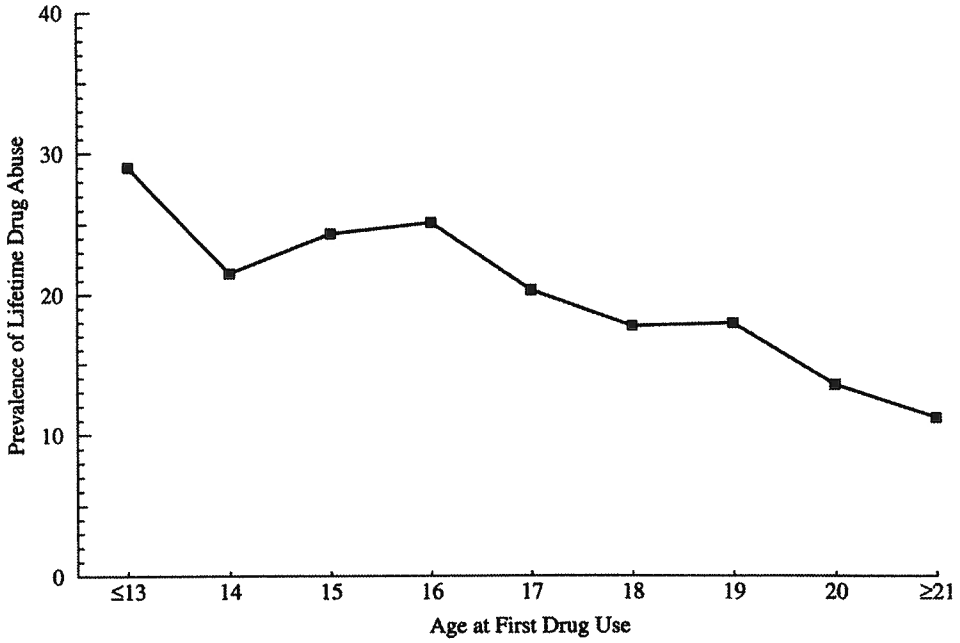
Another important contribution of this study is its role in highlighting the need for collecting data on drug abuse and dependence as well as drug use. Clearly, if 40% of all drug users go on to develop the serious disorders of drug abuse and dependence, there is an urgent need to identify risk factors associated with drug users who go on to develop serious problems with drugs and those factors that protect and insulate those who do not. One question raised from this study suggests further that these risk factors may indeed be different among important sociodemographic subgroups of the population. Understanding and integrating the complex of social, psychological, genetic, biological and environmental risk factors is paramount to the development of successful drug prevention and

Table 3. Age at First Drug Use and the Prevalence[1] of Lifetime DSM-IV Drug Dependence by Gender and Race

Age at First Drug Use (In Years)	Male (n = 663)	Female (n = 569)	Black (n = 121)	Nonblack (n = 1,111)	Total (n = 1,232)
13 or younger	33.8 (2.6)	33.2 (2.9)	28.9 (7.6)	33.9 (2.0)	33.6 (1.9)
14	28.4 (3.5)	25.1 (3.1)	14.8 (5.6)	27.9 (2.5)	26.8 (2.4)
15	24.9 (2.5)	26.1 (2.9)	29.7 (5.8)	25.0 (1.9)	25.4 (1.9)
16	15.9 (1.7)	20.4 (2.1)	18.7 (5.9)	17.6 (1.3)	17.7 (1.3)
17	15.7 (2.1)	14.3 (1.9)	14.0 (4.4)	15.2 (1.5)	15.1 (1.4)
18	15.1 (1.9)	14.8 (1.9)	21.6 (5.1)	14.3 (1.5)	15.0 (1.5)
19	17.1 (2.6)	6.1 (1.6)	7.3 (4.7)	14.2 (2.0)	13.4 (1.9)
20	11.7 (2.3)	10.9 (3.0)	11.3 (5.4)	11.4 (1.9)	11.4 (1.8)
21 and older	13.2 (1.6)	14.1 (1.6)	17.8 (3.7)	13.1 (1.2)	13.6 (1.2)

Notes. Standard errors are in parentheses.
1. Prevalences expressed as weighted figures, ns presented as unweighted figures.

Figure 1. Prevalence of Lifetime Drug Abuse by Age at First Drug Use



intervention programs. The achievement of this goal rests heavily on support for prospective longitudinal studies beginning in childhood and continuing beyond young adulthood.

Our lack of complete understanding of the relationship between early onset drug use and the subsequent development of drug abuse and dependence, does not minimize the importance of the adverse short-term consequences of adolescent drug use, including death and injury resulting from motor vehicle crashes, risky sexual behavior and associated teen pregnancy and exposure to sexually-transmitted diseases, depression, delinquency, violence, suicide, criminal involvement, accidental overdose, and marital and work instability. The major findings of this study showed that early onset drug use is a powerful predictor of the very serious long-term adverse consequences of drug abuse and dependence. A continuing program of research on the long-term, as well as the short-term, consequences of early onset drug use and continuous efforts to get these findings to the public is critical.

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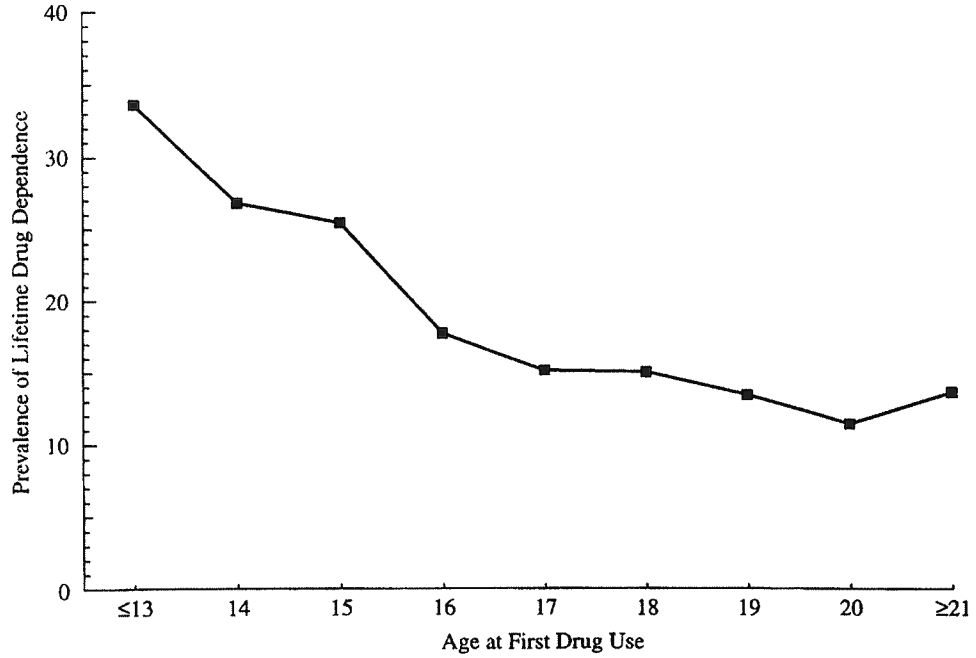
Table 4. Logistic Regression Results for DSM-IV Drug Abuse and Dependence

Characteristic	Drug Abuse			Drug Dependence		
	Beta	SE (Beta)	p-value	Beta	SE (Beta)	p-value
Age at onset of drug use	–0.05	0.01	< .00003	–0.04	0.01	< .01
Age 30 to 44 years [1]	0.02	0.09	NS	–0.51	0.09	< .00001
Age 45-to-64 years[1]	–0.28	0.21	NS	–0.77	0.23	< .002
Age 65 years and older[1]	–0.54	0.69	NS	–4.10	1.31	< .0005
Gender (effect of male)	0.39	0.08	< .00001	0.03	0.72	NS
Race (effect of black)	–0.55	0.15	< .0006	–0.02	0.14	NS
Positive family history of alcoholism	0.26	0.07	< .0009	0.71	0.08	< .00001
Duration of drug use	0.01	0.01	< .043	0.05	0.02	< .02
Drug use status (effect of former user)	–0.59	0.09	< .00001	0.18	0.09	< .05

Notes. 1. Effect of each age relative to 18-to-29 year olds.
2. NS = not significant.

Figure 2. Prevalence of Lifetime Drug Dependence by Age at First Drug Use



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The Relationship Between Cannabis Use and DSM–IV Cannabis Abuse and Dependence: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant and Roger Pickering

The purpose of this study was to determine the risk of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition *(DSM-IV) cannabis abuse and dependence at different levels of cannabis use in a nationally representative sample of the U.S. general population. Two separate logistic regression analyses were conducted to determine the association between cannabis use, and abuse and dependence. The risk of cannabis abuse and dependence was found to increase with the frequency of smoking occasions and slightly decreased with age. More severe comorbidity was associated with dependence compared to abuse, suggesting that cannabis might be used to self-medicate major depression. The strength of the association between cannabis use and abuse was also increased as a function of the number of joints smoked among females, but not males. These results were discussed in terms of differential societal reactions, the self-medication hypothesis, and gender biases in diagnosing cannabis abuse.*

INTRODUCTION

According to the National Institute on Drug Abuse's, and more recently the Substance Abuse and Mental Health Services Administration's National Household Survey on Drug Abuse, the peak year for current cannabis use was 1979, when there were an estimated 25 million users, representing 13.7% of the U.S. population (U.S. Department of Health and Human Services, 1995). By 1992, the rate of use declined steadily to 5.5%. Trends for the prevalence of cannabis use in the U.S. population as a whole parallel those of both high school and college students. From 1980 to 1992, rates of cannabis use declined from 48.8% to 21.9% among high school seniors, and from 51% to 27% among college students (Johnson et al., 1995). Although these national surveys on cannabis use, among other illicit drug use, may be useful for a number of purposes, they can be criticized for their failure to produce estimates of cannabis abuse and dependence. Estimates of cannabis use are not helpful in identifying the more severely affected population that abuses or is dependent on cannabis for which treatment might prove beneficial. Moreover, estimates of cannabis use alone can provide little insight into the transitions from use to abuse and dependence, and the sociodemographic profiles related to that transition.

To date, only three surveys have reported national estimates of the prevalence of cannabis abuse and dependence. The first was the Epidemiologic Catchment Area (ECA) Survey, in which 18,571 respondents, aged 18 years and older, were interviewed in a series of five community-based epidemiologic studies in the early 1980s (Robins et al., 1991). The second was the National Comorbidity Survey (NCS), a national probability sample of 8,098 respondents aged 15 to 54, conducted in 1991 (Kessler et al., 1994). The third was the National Longitudinal Alcohol Epidemiologic Survey (NLAES), consisting of a nationally representative sample of the U.S. population in which 42,862 respondents were interviewed (Grant et al., 1994).

In both of the earlier studies, the ECA and NCS, the relationship between cannabis use, abuse and dependence was presented as simple cross-tabulations or expressed as first-order correlations or odds ratios, ignoring the influence of multiple putative risk factors on the association (Anthony et al., 1994; Warner et al., 1995). Attempts were not made to examine the risk of cannabis abuse or dependence at various levels

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of cannabis use, nor was the potential for interaction and confounding in the data examined.

The purpose of the present study was to determine more precisely the risk of cannabis abuse and dependence at different levels of cannabis use using data from the NLAES. This survey also encompassed a broad array of putative risk factors among which to study interaction and confounding, including comorbid major depression and drug-use disorders, early parental loss, and recent death of a close relative or significant other.

METHODS

Study sample

The data for this study were based on the 1992 NLAES, a national probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism. Fieldwork for the study was conducted by the United States Bureau of the Census. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. The household response rate for this representative sample of the U.S. population was 91.9%, and the sample person response rate was 97.4%.

The NLAES consisted of a complex multistage design that featured sampling of primary sampling units with probability proportional to size and oversampling of the African American and young adult (18 to 29 years) populations. The NLAES design has been described in detail elsewhere (Massey et al., 1989).

Diagnostic measures

The diagnostic assessment instrument used in NLAES was the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS: Grant and Hasin, 1992). The AUDADIS diagnoses of 12-month cannabis dependence satisfied both the clustering and duration criteria of the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV; American Psychiatric Association, 1994) classification. Twelve-month diagnoses of cannabis dependence required the occurrence of at least three dependence criteria in the year preceding the interview, and the duration qualifiers associated with some dependence criteria must have been satisfied. The duration qualifiers are defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often," and "persistent" appearing in the description of the diagnostic criteria. Similarly, to qualify for a diagnosis of cannabis abuse, at least one abuse criteria needed to be satisfied during the past year and the duration qualifiers associated with positive criteria needed to be satisfied. In a separate test–retest study conducted in the general population, reliability coefficients (kappas) associated with past year cannabis abuse and dependence were 0.74 and 0.71, respectively (Grant et al., 1995).

Measures of DSM-IV alcohol and other drug-use disorders (excluding cannabis abuse and dependence) were constructed in the same way, as were cannabis abuse and dependence diagnoses. Consistent with the DSM-IV, the AUDADIS diagnoses of major depression required the presence of at least five depressive symptoms (inclusive of depressed mood or loss of pleasure and interest) nearly every day for most of the day for at least the same 2-week period. Social dysfunction, occupational dysfunction, or both must also have been present during the disturbance and episodes of depression exclusively due to bereavement or physical illness were ruled out.

In a separate test–retest conducted in the general population, reliability (kappa) coefficients for past-year alcohol and other drug-use disorders ranged from 0.70 to 0.84 (Grant et al., 1995). The corresponding coefficient for major depression was 0.65.

Cannabis and other drug use

Two dimensions of past year cannabis use were measured in this study. The first was the frequency of cannabis use defined as the number of days during the year preceding the interview that cannabis was used. The second measure was the usual number of joints smoked per smoking occasion. For the purposes of this study, other drug use in the past year was defined as the illicit use of any drug other than cannabis at least 12 times on a lifetime basis and at least once during the past year. The kappa coefficients associated with past year cannabis and other drug use were 0.77 and 0.73 as ascertained in an independent test–retest study conducted in a general population sample (Grant et al., 1995).

Sociodemographic and background measures

Sociodemographic factors empirically shown to influence cannabis abuse and dependence, included the following: sex; age at first illicit drug use; ethnicity (black vs. nonblack); education (less than high school graduate vs. high school graduate and beyond); current marital status (married or living with someone as if married vs. separated, divorced, never married or widowed);

current employment status (employed vs. unemployed during the past year); and urbanicity (urban vs. rural).

The income variable consisted of a continuous measure represented by the midpoints of 23 income categories ranging from less than $7,000 to $156,000 or more. The socioeconomic status measure was based on 1990 Nam-Powers-Terrie occupational status scores (Terrie and Nam, 1994). Background variables included children under 14 years living at home, death of a parent prior to age 16 years, and experiencing a death of a close relative or close non-relative during the past year.

Statistical analysis

Two separate linear logistic regression analyses were conducted to determine the association between

Table 1. Percentage Distribution of Respondents Classified with and without DSM-IV Cannabis Dependence and Abuse: Predictor Variables

	Dependence		*Abuse*	
Variable	*Positive*	*Negative*	*Positive*	*Negative*
Frequency of Cannabis Use (Days in Past Year)				
≤2	0.0 (0.00)	11.8 (0.94)*	1.4 (0.63)	14.0 (1.13)*
3–10	12.8 (4.10)	23.0 (1.22)	16.4 (2.27)	24.2 (1.41)
11–30	22.9 (4.67)	23.1 (1.23)	28.0 (2.70)	21.6 (1.34)
40–100	17.6 (3.95)	15.1 (1.11)	21.0 (3.17)	13.5 (1.09)
≥101	46.7 (5.39)	27.0 (1.31)	33.2 (3.05)	26.6 (1.40)
Quantity of Cannabis Use Per Occasion (Number of Joints)				
≤1	41.2 (5.20)	69.7 (1.35)*	59.3 (3.19)	70.6 (1.42)*
2	24.0 (4.68)	19.0 (1.12)	24.5 (2.57)	17.7 (1.22)
3–4	15.9 (3.87)	8.9 (0.96)	13.0 (2.21)	8.1 (0.94)
≥5	18.9 (4.42)	2.4 (0.45)	3.2 (1.14)	3.6 (0.57)
% Male	70.8 (5.01)	66.3 (1.33)	75.9 (2.68)	63.7 (1.49)*
Age (in Years)				
18–29	78.8 (4.04)	57.4 (1.78)*	67.9 (3.21)	55.9 (1.80)*
30–39	19.1 (3.87)	38.6 (1.67)	30.2 (3.09)	39.7 (1.75)
40–49	2.1 (1.33)	3.7 (0.57)	1.9 (1.04)	4.1 (0.61)
≥50	0.0 (0.00)	0.3 (0.15)	0.0 (0.00)	0.3 (0.19)
% Black	9.9 (3.10)	11.2 (0.98)	7.2 (1.43)	12.3 (1.12)
% Less than high school	20.9 (4.19)	16.5 (1.11)	16.4 (2.03)	16.9 (1.23)
% Married	40.2 (5.47)	38.2 (1.49)	36.3 (2.95)	39.0 (1.72)
% Employed in past year	88.1 (2.99)	92.9 (0.70)	96.8 (0.82)	91.3 (0.84)*
% Urban	79.9 (4.97)	80.7 (1.51)	72.4 (4.09)	83.3 (1.30)
Socioeconomic Score				
1–24	31.8 (5.15)	27.9 (1.48)	34.7 (3.31)	26.1 (1.48)
25–49	34.5 (5.05)	33.7 (1.41)	34.5 (3.02)	33.5 (1.51)
50–74	26.8 (4.77)	20.4 (1.17)	17.8 (2.19)	21.7 (1.28)
75–100	6.9 (2.82)	18.0 (1.13)	13.0 (1.94)	18.7 (1.26)
Household Income				
<$20,401	62.6 (5.32)	46.8 (1.77)	46.7 (3.35)	48.1 (1.79)
$20,401–$35,988	15.7 (3.76)	22.8 (1.46)	25.9 (3.03)	21.2 (1.38)
$35,989–$71,988	17.5 (4.15)	22.4 (1.33)	21.8 (2.53)	22.2 (1.39)
≥$71,989	4.2 (1.94)	8.0 (0.85)	5.6 (1.33)	8.5 (0.95)
% Age at first drug use prior to age 17 years	68.1 (4.90)	57.2 (1.53)	64.3 (2.88)	55.9 (1.68)
% Major depression in past year	29.1 (4.74)	9.5 (0.84)*	14.2 (1.86)	9.6 (0.91)
% Other drug use disorder in past year	28.1 (4.93)	7.1 (0.77)	14.0 (2.18)	6.6 (0.82)
% Other drug use in past year	23.1 (4.46)	19.3 (1.15)	20.4 (2.51)	19.3 (1.31)
% Alcohol dependence in past year	62.9 (5.39)	27.6 (1.40)*	41.2 (3.14)	26.2 (1.57)*
% Alcohol abuse in past year	9.3 (2.92)	17.0 (1.14)	23.8 (3.06)	14.3 (1.16)
% Child(ren) under 13 years living at home	21.3 (4.30)	25.8 (1.33)	20.7 (2.37)	26.9 (1.52)
% Death of parent(s) prior to age 16 years	3.3 (1.62)	4.8 (0.59)	3.8 (0.95)	4.9 (0.68)
% Experiencing death of close relative in past year	26.0 (5.33)	24.0 (1.41)	25.4 (2.85)	23.7 (1.41)
% Experiencing death of close non-relative in past year	13.8 (3.53)	16.2 (1.07)	16.8 (2.26)	15.8 (1.12)

Note. Numbers in parentheses are standard errors.
*$p < 0.001$.

cannabis use, and cannabis abuse and dependence (the dependent or outcome variables). Cannabis use expressed as frequency of use and quantity smoked per occasion served as the two major exposure variables of interest. All other sociodemographic, background, and diagnostic variables were treated as potential confounders or modifiers of the use-abuse or use-dependence associations. All statistical analyses presented were conducted with SUDAAN, a statistical program that accounts for the design effects of complex sample surveys like the NLAES (Research Triangle Institute, 1996). All analyses were also conducted among the subsample of past year cannabis users.

The data analytic strategy consisted of two stages. The first stage entailed the identification of important modifiers by allowing for interaction effects up to second-degree product terms involving the two exposure variables (e.g., frequency of cannabis use times sex). Using a backward elimination process, all nonsignificant cross-product terms ($p > 0.01$) were eliminated from the model while at the same time all main effect terms were retained. Only second-degree interaction terms were included in the model because cross-product terms involving higher levels of interaction are often questionable due to induced multicollinearity.

Stage 2 entailed the identification of confounders, or alternatively, the deletion of nonconfounders from the reduced model resulting from the first stage (i.e., a model containing all main effects and significant cross-product terms). In the case of no interaction, the elimination of nonconfounders consists of removing all main effects (i.e., predictors) to produce an even more reduced model. If the main effect coefficients of both exposure variables do not materially change when a predictor main effect is removed, the use of the reduced model without that main effect term can lead to a gain in precision. On the other hand, if the exposure-involved coefficients do show substantial change upon refitting, the main effect term should be retained in the model as a confounder. In the presence of interaction, main effect terms involved in the model as modifiers are not candidates for deletion. Each of these main effect terms must be retained to obtain precise estimates of the magnitude of the associated interaction effect. Under these conditions, the main effect exposure coefficient and all exposure-related cross-product coefficients must

Table 2. Results of Final Logistic Models for DSM-IV Cannabis Dependence and Abuse

	Dependence			*Abuse*		
Variable	*Beta*	*S.E. (Beta)*	*p*	*Beta*	*S.E. (Beta)*	*p*
Intercept	−3.71	0.64	<0.0001	−1.88	0.35	<0.0000
Frequency of cannabis use	0.27	0.08	<0.0007	0.22	0.04	<0.0000
Quantity of cannabis use	0.78	0.20	<0.0003	0.62	0.24	<0.0108
Sex (effect of male)				0.77	0.20	<0.0003
Age	−0.04	0.02	<0.0296	−0.02	0.01	<0.0109
Ethnicity (effect of Black)						
Education (effect of less than high school)						
Marital status (effect of married)						
Employment (effect of employed)						
Urbanicity (effect of urban)				−0.62	0.22	<0.0057
Socioeconomic status						
Household income						
Age at first drug use						
Major depression in past year	0.95	0.30	<0.0025			
Other drug use disorder in past year	0.79	0.33	<0.0202			
Other drug use in past year						
Alcohol dependence in past year	1.01	0.26	<0.0002	0.88	0.16	<0.0000
Alcohol abuse in past year				0.90	0.21	<0.0000
Child(ren) less than 13 years living at home						
Death of parent(s) prior to age 17 years						
Death of close relative in past year						
Death of close nonrelative in past year						
Quantity of cannabis use × sex				−0.74	0.28	<0.0097

Note. Goodness of fit of overall model: DSM-IV dependence — Satterthwaite's F (6,68) = 19.67, $p < 0.0000$; DSM-IV abuse — F (7,68) = 11.8, $p < 0.0000$.

Table 3. Estimated Adjusted Odds Ratios and 95% Confidence Intervals for the Cannabis Use–DSM-IV Abuse Association by Sex

Number of Joints Smoked Per Occasion	Male	Female
0.5	1.00	1.00
2	0.84[a] (0.56, 1.27)	2.35 (1.24, 4.45)
4	0.78 (0.42, 1.45)	3.60 (1.38, 9.39)
6	0.74 (0.35, 1.55)	4.63 (1.47, 14.56)
8	0.71 (0.31, 1.62)	5.52 (2.54, 12.82)

Note. Odds of abuse at selected quantities of cannabis consumed per occasion relative to the odds of abuse for smoking 0.50 joints per occasion.

be monitored for change upon refitting to determine if actual confounders should be retained in the model.

RESULTS

The prevalences of past year cannabis abuse and dependence among past year cannabis users were 23.1% and 6.3%, respectively. The univariate associations between each predictor variable and among those respondents classified with and without cannabis abuse and dependence are shown in Table 1. Significance levels were set at $p < 0.001$ due to the multiple tests considered. The results of these analyses showed that the relationships between cannabis abuse and dependence and each of the predictor variables were quite similar. Compared to respondents who were classified with no cannabis diagnosis, respondents with cannabis abuse and dependence were heavier users of cannabis in terms of both quantity and frequency, were older and significantly more likely to have had alcohol dependence during the past year. Respondents with diagnoses of cannabis dependence, but not abuse, were also significantly more likely to have had a major depressive episode during the past year.

The results of the final two logistic regression analyses are shown in Table 2. The odds of both cannabis abuse and dependence were slightly decreased by age but increased by quantity and frequency of cannabis use. The strength of the independent associations between abuse and dependence and frequency and quantity of cannabis use were similar, but greater for quantity (OR = $e^{0.78} = 2.2$ and OR = $e^{0.62} = 1.8$) than for frequency of use (OR = $e^{0.27} = 1.3$ and OR = $e^{0.22} = 1.2$).

Despite similarities between the results, some differences did emerge from the multivariate analyses of cannabis abuse and dependence. With respect to cannabis abuse, the odds for abuse were approximately two times greater (OR = $e^{0.77} = 2.2$) among males than females and the odds for abuse were lower among rural compared to urban respondents (OR = $e^{-0.62} = 0.53$).

The impact of comorbid disorders on the cannabis use–abuse and use–dependence associations also differed. The odds of dependence were 2.6 times greater among those respondents with comorbid major depression, 2.2 times greater among respondents with a comorbid drug-use disorder and 2.7 times greater among respondents with comorbid alcohol dependence compared to those not so classified. In contrast, the odds of cannabis dependence were increased 41% and 45% among respondents with comorbid alcohol abuse and dependence relative to those respondents with no alcohol use disorders.

Although there were no discernible modifiers for the use–dependence association, sex was found to modify the use–abuse relationship. Specifically, the number of joints smoked per smoking occasion increased the risk for abuse, but only among females (Table 3). The odds of abuse were 2.4 times greater among females who smoked on average two joints per occasion compared to those who smoked 0.50 joints on a typical occasion. For females who smoked on average eight joints per occasion, the odds of cannabis abuse were 5.5 times greater relative to the odds of smoking 0.50 joints per occasion.

DISCUSSION

Consistent with previous research, the risk of cannabis abuse and dependence increased with the frequency and quantity of use on smoking occasions and slightly decreased with age. Despite these general similarities among the multivariate relationships, the impact of various predictors on the cannabis abuse and dependence outcomes differed. Respondents living in urban areas demonstrated a lower risk of abuse, but not dependence, compared to respondents in rural areas. This finding suggests that abuse is more likely in regions of the country where cannabis might be more available and more easily cultivated for recreational use.

The most striking differences between the abuse and dependence results was the more severe psychopathology associated with dependence as opposed to abuse. Respondents with comorbid major depression, other drug-use disorders, and alcohol dependence had a greater risk of cannabis dependence relative to those without comorbid disorders. The risk of cannabis abuse was only increased among respondents with comorbid alcohol abuse and dependence compared to those with no alcohol use disorders.

The increased risk of cannabis dependence in the presence of comorbid alcohol, other drug use, and major depressive disorders suggests that a variety of substances might be being utilized to self-medicate depression. Although cannabis is not usually associated with the self-medication paradigm as it relates to major depression, chronic high dose use of cannabis may be accompanied by the development of amotivational syndrome. This syndrome is characterized by anhedonia, chronic apathy, difficulty concentrating, and social withdrawal symptoms, strikingly similar to those in major depression. In addition to this differential diagnostic issue, the results of this study also highlight the difficulty in extracting clear relationships between drugs of choice predicted by the self-medication hypothesis as the result of the common phenomena of poly-substance abuse.

The strength of the association between cannabis use and abuse was increased as a function of the number of joints smoked per smoking occasion among females, but not males. One reason for this observed risk differential may relate directly to the definition of cannabis abuse. The DSM-IV defines cannabis abuse, separately from cannabis dependence, as social, occupational, legal, and interpersonal consequences arising from the direct effects of cannabis. Indicators of patterns of compulsive use of cannabis (e.g., impaired control over use) and tolerance and withdrawal symptomatology were relegated to the DSM-IV dependence category. Unlike indicators of dependence, the DSM-IV abuse criteria reflect societal reactions to cannabis use behavior. As a socially subordinate subgroup of the population, women's cannabis use behavior may be more heavily sanctioned than that of men (Park, 1983; Makela, 1987), thereby increasing their vulnerability to social sanctions and the probability that they would be diagnosed with cannabis abuse. Moreover, the findings of this study strongly suggest that it is the quantity of cannabis use and its associated adverse consequences resulting from intoxication, and not the frequency of cannabis use, that increases women's vulnerability to cannabis abuse.

This study has served to close the gap in our understanding about the relationship between cannabis use and cannabis abuse and dependence. Further, the finding that there was increased risk of cannabis dependence associated with other psychopathology allows us to predict a more severe illness course and adverse impact on treatment outcome among individuals dependent on cannabis with accompanying comorbidity. As a population-based epidemiological study, further analyses of the NLAES data will have the goal of identifying risk factors associated with a variety of specific drug-use disorders with a view toward the prevention and intervention of co-occurring drug-use disorders and other comorbid psychiatric disorders. This epidemiological research should complement existing studies conducted in clinical samples that focus on the impact of comorbidity on drug-use disorders in relation to treatment outcome and illness course.

Finally, findings of this study highlighted the importance of collecting data on use, abuse, and dependence for the purpose of understanding the relationships between them. The NLAES has also provided, for the first time, reliable national estimates of cannabis use and dependence based on the newest DSM-IV diagnostic criteria. However, like other national surveys of drug use and drug-use disorders, the homeless and those residing in most institutional settings, such as jails and group homes, were not included in the NLAES sample. As a result of this, estimates of cannabis use, abuse, and dependence and their relation to one another in this study are likely to be underestimated.

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Parallels to Early Onset Alcohol Use in the Relationship of Early Onset Smoking With Drug Use and DSM–IV Drug and Depressive Disorders: Findings From the National Longitudinal Epidemiologic Survey

Eleanor Z. Hanna and Bridget F. Grant

This paper endeavored to determine (1) if early onset of regular tobacco use is as predictive of drug use and depressive disorders as it is of alcohol use disorders; and (2) if a biological commonality, as measured by a family history of alcoholism and both early onset and severity of disease, among all three disorders can be evidenced in a large nationally representative sample. Prevalences of lifetime drug use, drug abuse and dependence, and major depressive disorders, as well as indices of their severity, were compared among smoking groups defined by age at onset of regular tobacco use and among nonsmokers. Linear logistic regression analyses, controlling for important covariates, including a family history positive for alcoholism, were conducted to assess the relationship between age at smoking onset and drug use, abuse and dependence, as well as depressive disorders. Both objectives were met. Moreover, results suggest that smoking may play an equally, if not even more, insidious role than drinking in the use and development of dependence on illicit substances and depression.

Despite the longstanding tradition of treating mental, alcohol, and drug-related conditions as separate entities, many studies have addressed their interrelationships (Giovino et al., 1995; Bien and Burge, 1990; Istvan et al., 1984; Craig and Van Natta, 1977; Henningfield et al., 1990; Grant and Harford, 1995; Glassman, 1993; Breslau et al., 1991; Anda et al., 1990; Regier et al., 1990). Interest in this area has increased steadily since the Epidemiologic Catchment Area (ECA) findings (Regier et al., 1990) that established the national comorbidity rate of mental disorders with alcohol disorders at 37% and with drug disorders at 53%. The comorbidity rate of alcohol and drug disorders in that study was 47.3%. The National Comorbidity Study (Kessler et al., 1994) found that substance use, depressive disorders, and anxiety disorders were among the most commonly occurring conditions, and also noted an increase in tobacco use and dependence among younger people. In view of these continuing high rates of comorbidity, or at least overlap, it may prove fruitless to pursue each of these disorders as a separate entity when studying etiology, strategizing prevention, or planning treatment.

In addition to addressing these disorders comorbidly, it would be useful to study the stages of acquisition in the use of alcohol, cigarettes, and other drugs (Werch and Anzalone, 1995; Botvin and Botvin, 1992; Fleming et al., 1989; Single et al., 1974), as well as whether psychiatric conditions precede addictive disorders or vice versa (Masse and Tremblay, 1997; Deykin et al., 1987). Many studies have tried to determine which drugs and in which sequence should be targeted in these efforts (Werch and Anzalone, 1995; Botvin and Botvin, 1992; Fleming et al., 1989; Single et al., 1974; Masse and Tremblay, 1997; Deykin et al., 1987; Dupre et al., 1995; Yamaguchi and Kandel, 1984; Kandel and Faust, 1975; Yu and Williford, 1992). Their results, while generally suggesting that legal drug use precedes illegal drug use, have been equivocal about whether it is first use of cigarettes or alcohol or both that leads to the full development of becoming an illicit substance user/abuser. Some even debate whether wine or beer use precedes

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liquor use. Many of these studies focus on high school students. For example, Yu and Williford (1992) extended Kandel's (Yamaguchi and Kandel, 1984; Kandel and Faust, 1975) research and stake claim to a causal pattern of early onset of alcohol use, particularly between the ages of 13 and 16. They suggested that earlier alcohol use, by influencing current alcohol use, which in turn intensified the use of cigarettes and the likelihood of multiple drug use, was the precipitant leading to an increased likelihood of using cigarettes and marijuana.

In their excellent review, Shiffman and Balabanis (1995) noted that much support exists for the notion that alcohol influences smoking, and that drinking and smoking together are correlated with the use of other drugs at all stages of drug use. They also noted contrary evidence suggesting that teens smoke prior to drinking. In fact, teens were more likely to drink only after their first few tobacco exposures; then, only after smoking for 2 years did two-thirds of a these teens smoke while drinking. Additionally, Single et al. (1974) found that students in grade 12 who had never smoked were less likely to report any use of beer, liquor, stimulants, or depressants, compared with others in this age group who had smoked. Experimental evidence supports this latter view. Porthorf et al. (1983) discovered that rats implanted with slow-release nicotine pellets, compared with controls, doubled their alcohol intake and decreased their water intake. Other drugs did not lead to increased ethanol intake. They took this as support for the self-medication hypothesis, postulating that chronic administration of nicotine increased hyperactivity, which the rats learned, could be counteracted by alcohol.

Population studies, similar to those reported by Yamaguchi and Kandel (1984), Kandel and Faust (1975), and Yu and Williford (1992), but improved in that they were prospective, indicate some support for tobacco use leading to alcohol and other drug use. Fleming et al. (1989) studied middle school-aged children with the intent of collecting prospective information about initiation of first use of a variety of legal and illegal drugs. He also followed 12th grade students through two additional postgraduate years. His stated goal was to demonstrate that the use of cigarettes would form a scale similar to those found for other substances, so that when a cumulative pattern of drug use was noted, either cigarettes or alcohol would be the initial drug used. He did this for *any use,* as well as *daily use* of each substance. Additionally, he obtained data on temporal ordering. Cigarettes formed the floor for *daily use* of other substances; beer was the floor for *any use* of other substances. However, when they tracked usage among twelfth graders over an additional 2 years, he found a pattern of nonuse to new use, suggesting that those who were first-time users of cigarettes at grade 12 (time 1) were more likely to use marijuana. Those who used cigarettes or marijuana at time 1 were at least monthly beer drinkers at time 2. Students using all three at time 1 were monthly liquor drinkers at time 2, and those using all four at time 1 used stimulants and depressants at time 2. In short, he concluded that those who had never smoked either by the 12th grade or in their first two post-high school years were significantly less likely to report any use of beer, liquor, stimulants, or depressants.

Sher et al. (1996), focusing not on substance use but on the diagnosis of an alcohol or tobacco use disorder, found that either—if diagnosed earlier in time—increased the chances of developing the other later in time. When dealing with regular tobacco use, however, Grant (1998) recently demonstrated that, like early onset drinking (Grant and Dawson, 1997), early onset smoking was a predictor of lifetime drinking and the development of DSM-IV alcohol abuse and dependence. Additionally, she found that those who smoked prior to age 13 were also more likely to consume excessive amounts of alcohol and have more severe alcohol use disorders relative to late onset smokers and nonsmokers. This is in keeping with the order of use hypothesis presented by Single et al. (1974), Fleming et al. (1989), and Porthorf et al. (1983). Both Sher et al. (1996) and Grant (1998) found that a family history positive for alcoholism was associated with a diagnosis of either disorder.

Furthermore, Grant (1998) cited prevalence rates by age of smoking onset for alcohol dependence and abuse that suggest early onset smokers have rates of alcohol abuse and dependence far in excess of the ECA rates. In the ECA study Regier et al. (1990) reported prevalence rates for alcohol dependence and abuse of 7.9 and 5.6, respectively. Grant (1998) reported similar national rates for alcohol dependence and abuse for nonsmokers, but higher rates among smokers. The highest prevalence rates were found among early onset smokers. The prevalence rate for alcohol dependence was 7.8 among lifetime nonsmokers, as opposed to rates ranging from 28.6 among those who began smoking before age 13 to 12.4 among those who began smoking at 17 or older. For alcohol abuse, the national prevalence rate reported by Grant was 3.4 among nonsmokers in contrast to rates ranging from 8.4 among those who began smoking before age 13 to 4.4 among those who began smoking at age 17 or older.

In terms of comorbidity with depression, the ECA (Regier et al., 1990) reported a prevalence rate for any affective disorder of 13.4 among respondents with alcohol use disorders in contrast with a 7.5 rate among those without an alcohol use disorder. These same rates doubled for drug use and affective disorders. This is consistent with a number of studies that indicate a link between alcohol abuse and major depressive disorder (Deykin et al., 1987; Pitts and Winokur, 1996; Kendler et al., 1993; Penick et al., 1987), as well as alcohol and tobacco use and depression (Aneshensel and Huba, 1983) or depressive disorders comorbid with nicotine disorders (Breslau et al., 1992, 1993; Kendler et al., 1993; Glassman et al., 1990; Hall et al., 1993). It is unlikely that the convergence of results from these studies is either coincidental or serendipitous. One possible explanation may be the presence of a familial factor that predisposes the affected individuals to each condition. This was in fact hypothesized as an explanation for surprising findings of no gender differences among a comorbid group of concurrent depressives in a recent study of comorbid depression and alcohol use disorders (Hanna and Grant, 1997). DeWit et al. (1997) placed the risk for initiation and regular use of drugs, including alcohol, between the ages of 12 and 22, noting that it is over by age 22. The many studies cited herein indicated higher rates of comorbidity and thus severity for respondents who initiate substance use at the youngest ages. Perhaps, then, this younger group is made more vulnerable by a family history positive for alcoholism and is thus most at risk for developing all of these problems. If so, establishing this would be useful not only to the etiology and prevention, but also for the treatment of all these conditions. For example, consistent with studies of smoking cessation that link alcohol dependence to failure (Hymowitz et al., 1991; Dawson, 2000), the interrelationships of alcohol, tobacco, and depression were studied in a clinical trial of alcohol reduction among nonalcohol-dependent medical patients. Results indicated that, whereas all three were associated at baseline, at time 2, smoking change had an association only with the Alcohol Dependence Scale score, primarily an indicator of biological involvement, in common with changes in alcohol and depression (Hanna et al., 1994).

Thus, this paper attempts to extend findings from these earlier studies by examining the effects of early onset tobacco use on both illicit drug abuse and dependence, as well as on major depressive disorder. We would expect then, as with alcohol abuse and dependence, that early onset smokers relative to late onset smokers and nonsmokers will have higher rates and more severe conditions of depression, drug use, abuse, and dependence. Also, these should occur at a younger age and be associated with a family history of alcoholism. If results prove similar to those obtained for alcohol abuse and dependence, and if a family history positive for alcoholism is found in each condition, they may provide the basis for cross-fertilization of existing and development of new effective prevention and treatment techniques comprehensive to all three disorders.

METHODS

Sample

Data from 42,862 respondents aged 18 years and older collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES) were analyzed for this study. A complex multistage design was used to ensure a nationally representative sample of the United States population. The household response rate was 91.9%, and the sample person response rate was 97.4%. Oversampling of the black population to secure adequate numbers for analytic purposes and of the young adult population aged 18 to 29 to include greater representation of this heavy substance-using segment of the population was accomplished at the segment and household stage of selection, respectively. This survey is described in further detail elsewhere (Massey et al., 1989; Grant, 1995).

Diagnostic assessment

Diagnoses of drug use disorder and major depressive disorder, as defined in the *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition* (American Psychiatric Association, 1994), were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS) (Grant, 1995). As with alcohol abuse and dependence, the AUDADIS also included an extensive list of symptom questions to operationalize the DSM-IV criteria for depression, drug abuse, and dependence. For a symptom to be counted, it had to have occurred within any 1-year period and meet duration qualifications. Duration qualifiers associated with certain symptoms were defined as the repetitiveness of their occurrence and represented by the terms “recurrent,” “often,” and “persistent.” The corresponding past diagnoses were measured as syndromes, or the clustering at the same time and either continuously or repeatedly for at least 1 month of the required number of symptoms necessary to achieve a diagnosis. Reliabilities for drug use disorders were 0.66 through 0.91 and for depressive disorders, 0.60

and 0.65 as ascertained from an independent test-retest study conducted in the general population (Grant et al., 1995).

Measurement did not permit ascertainment of DSM-IV tobacco use disorders. Because it was the purpose of this study to search for common links among these three related disorders or behaviors and early onset smoking, not nicotine dependence, we felt justified in using daily smoking, duration, and volume of daily smoking, as well as time to daily smoking to approximate the possibility of tobacco dependence and thus its possible severity. Reliabilities associated with smoking dimensions were good to excellent, 0.70 to 0.88 (Grant et al., 1993).

Severity of drug use disorders was measured by the number of lifetime symptoms characterizing each disorder, duration of the disorder, and having received any form of treatment (e.g., inpatient, outpatient, 12-step, etc.) specific to the disorder. Severity of depressive disorders was measured similarly. The number of depressive episodes, the longest duration of the episode, the number of symptoms during the worst episode, and having received treatment specific to the disorder were obtained.

Age at first onset was ascertained for each disorder. In the case of drug use, we also collected information on age at first regular use.

Related Measures

Lifetime smokers, anyone who had ever smoked 100 cigarettes, 50 cigars, or a pipe at least 50 times were asked about the quantity and duration of smoking, measured as the current monthly volume of smoking among current smokers, or the volume of smoking prior to quitting among ex-smokers. Current smokers are those who have smoked at least 100 cigarettes, 50 cigars, or a pipe at least 50 times in the 12-month period immediately preceding the interview. Similarly, measures of volume and duration of daily smoking were constructed for lifetime daily smokers. A measure of time to smoking daily was also constructed for this study by subtracting the age at onset of regular smoking from the age at onset of daily smoking. We did not construct a regression model using daily smoking as a surrogate for tobacco dependence because they are not equivalent; to do so would lead to inflated estimates. Thus, the analytic focus for these variables is limited only to descriptive analysis.

Family history of alcoholism was ascertained by asking a series of questions, in a manner consistent with the DSM-IV criteria for alcohol use disorders, about 18 different types of first- and second-degree relatives. For each type of relative, the Respondent was first asked how many relatives of that type lived to be 10 years old and how many were ever alcoholics or problem drinkers. An alcoholic or problem drinker was defined for the respondent. Respondents were told by the interviewer that an "... alcoholic or problem drinker is ... a person who has physical or emotional problems because of drinking; problems with a spouse, family or friends because of drinking; problems at work because of drinking; problems with the police because of drinking—like drunk driving; or a person who seems to spend a lot of time drinking or being hungover." In the present study, *only biological, first-degree relatives* are considered. In the descriptive models, family history is scored separately for each subgroup of biological, first-degree relatives (e.g., mother, father, and grandparent). For purposes of the multiple linear regression analyses, family history was considered positive if any biological, first-degree relative was reported as either being an alcoholic or having an alcohol problem. In no case is this variable ever greater than 1. Reliabilities were high for most sub classifications of first-degree relatives (0.72 to 1.00) (Grant et al., 1993).

Demographic factors that have been shown to affect risk levels of drinking, alcohol abuse, and/or dependence, as well as other behavioral conditions, were also measured. These included gender, race (black vs. white), level of educational attainment (less than high school vs. high school or further), social class (Nam-Powers-Terrie), marital status (married or living with as married vs. all others), and current age.

Statistical Analyses

Descriptive statistics were computed for all categorical and continuous measures. If the overall X^2 or F statistic was significant (<0.01), separate pairwise comparisons, using partitioned X^2's analyses or t tests, were conducted between the smoking groups. To protect against type 1 error arising from multiple comparisons in this subset of analyses only a test statistic of <0.0001 was considered significant. The numbers for each age group of smoking onset presented in Table 1 are based on the 42,731 Respondents for whom age of smoking onset data was available.

The second part of the analysis consisted of four separate linear logistic regression equations to assess the relationship between ages of smoking onset, our exposure variable, and the odds of lifetime drug use, lifetime drug abuse, lifetime drug dependence, and lifetime diagnosis of major depressive disorder. It should be noted that the percentages of Respondents

in each of the age of smoking categories who used and/or had a drug use disorder in each of the seven drug categories measured are reported in the descriptive statistics, whereas the regression models focus on use of, abuse of, or dependence on *any* drug. Control variables in all models were gender, race, age at interview (18 to 29 years; 30 to 44 years; 45 to 64 years; 65+ years), age of onset for the disorder, and having a family history of alcoholism (any biological first-degree relative who qualified for inclusion as an alcoholic or problem drinker). Because of the importance of gender and race in substance use and depressive disorders, second-order interactions between age of onset at smoking and these sociodemographic variables were included in these models.

The linear regression analyses in which lifetime drug use and depression served as the outcome measures included the entire NLAES sample (n = 42,862). The analysis for depression used smokers and nonsmokers who had ever qualified for a depressive disorder (yes = 4,333, no = 38,529). The analysis for drug use used smokers and nonsmokers who had ever used drugs on their own (yes = 6,530, no = 36,322). Similar analyses relating to outcomes of drug abuse and dependence included only smokers and nonsmokers who had ever used drugs (n = 6,538) (abuse only: yes = 1,266, no = 5,092; dependence only: yes 1,217, no = 5,141).

RESULTS

Fifteen percent of this NLAES sample was lifetime drug users (n = 6,530) and slightly under 6% met criteria for either lifetime abuse or dependence (n = 1,266 and 1,217, respectively). Those qualifying for a lifetime depressive disorder (n = 4,333), on the other hand, comprised 10% of the sample. Approximately 1% of the drug user sample, but none of the depressive sample lacked age of onset information and were thus eliminated from the analysis presented herein.

Demographic characteristics

The demographic profile of this NLAES sample is presented in Table 1. Smoking begun before age 13 rather than at ages 14 to 16 or 17 to 20 occurs more frequently among respondents who are male (66.72%), non-black (93.66%), younger (42.05), married (68.01%), of a lower socioeconomic status (45.38), and have less than a high school education (25.32). Characteristics of lifetime nonsmokers differ significantly from those of all smoking groups. Nonsmokers are more likely to be women (60.61%) and black (13.32%). Although nonsmokers (41.81) are as young as early smokers, fewer of them have less than a high school education (17.85%) and are married (59.54%).

Genetic predisposition

Table 1 also demonstrates that subjects who began smoking at or before age 13 are significantly different from all other groups in that they are more likely to have a family history positive for alcoholism (67.57%). Their proportions with biological fathers (29.46%), mothers (10.28%), and grandparents (33.68%) were significantly different from those who began smoking at ages 14 to 16. Those subjects who began smoking at ages 14 to 16 were themselves significantly more likely to report a family history positive for alcoholism than were later smokers (age 17 and up) and lifetime nonsmokers.

Table 1. Sociodemographic and Family History Descriptors Among Lifetime Smokers by Age of Smoking Onset and Among Lifetime Nonsmokers

	Age at smoking onset (in years)				Significant comparisons between age at smoking onset groups[a] χ^2 or t, $p < 001$
	<13 (n = 4,930)	14–16 (n = 7,449)	≥17–20 (n = 9,428)	Nonsmokers (n = 20,924)	
Sociodemographic characteristics					
% Male	66.71 (0.72)	55.62 (0.63)	50.30 (0.62)	39.39 (0.45)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
% Black	6.44 (0.43)	8.02 (0.42)	12.73 (0.52)	13.32 (0.47)	(1) v (3) (4), (2) v (3) (4)
Current age (in years)	42.05 (0.27)	43.12 (0.26)	49.65 (0.22)	41.81 (0.21)	(1) v (3), (2) v (3)
% Married or living with someone as married	68.01 (0.77)	66.64 (0.71)	65.64 (0.54)	59.54 (0.51)	(1) v (9), (2) v (4), (3) v (4)
% With less than high school education	25.32 (0.83)	20.90 (0.59)	19.20 (0.51)	17.85 (0.39)	(1) v (2) (3) (4), (2) v (4), (3) v (4)
Socioeconomic status[b]	45.38 (0.53)	47.03 (0.47)	49.55 (0.41)	50.23 (0.28)	(1) v (2) (3) (4), (2) v (3), (3) v (4)
Family history					
% Biological father	29.46 (0.72)	23.70 (0.61)	17.62 (0.45)	16.10 (0.30)	(1) v (2) (3) (4), (2) v (3) (4)
% Biological mother	10.28 (0.54)	7.30 (0.37)	4.52 (0.26)	4.03 (0.17)	(1) v (2) (3) (4), (2) v (3) (4)
% Biological grandparent	33.68 (0.78)	27.10 (0.63)	19.86 (0.48)	18.03 (0.32)	(1) v (2) (3) (4), (2) v (3) (4)
% Family history positive	67.57 (0.80)	58.83 (0.74)	50.16 (0.63)	48.23 (0.45)	(1) v (2) (3) (4), (2) v (3) (4)

Note: Percentages expressed as weighted figures; n's expressed as unweighted figures. Standard errors are in parentheses.

[a] (1) = <13, (2) = 14–16, (3) = 17–20, (4) = nonsmokers.

[b] Socioeconomic status is based on the 1990 Nam-Powers-Terrie occupational index.

Smoking Characteristics

Smoking characteristics of the sample are presented in Table 2. Subjects who began smoking before age 17 were more likely to be current smokers than were those who started at or after age 17 (94.0% before age 13, 93.8% before age 17 vs. 88.3% at and above age 17). Although early onset smokers took significantly longer to begin smoking daily in comparison with the other two groups (4.33 years vs. 2.25 and 1.71 years) and they smoked more per day when smoking daily than did those in the older age groups (24.33 vs. 20.97 and 18.35 per day), their duration of daily smoking was not significantly different (16.7 years vs. 15.7 and 16.3 years, for the 14 to 16 and over 17 groups, respectively).

Depression-Related Characteristics

Table 3 demonstrates that respondents who were early onset smokers were significantly younger at age of onset for any lifetime major depressive disorder than were those in either of other smoking groups (21.7 vs. 24.2 and 26.2, respectively). However, there was no significant difference in age of onset for depression between this group of early onset smokers and respondents who were lifetime nonsmokers. Lifetime nonsmokers were themselves not significantly different from those who began smoking between the ages of 14 and 16 in their age of onset for depression.

Diagnoses of any lifetime depressive disorder were significantly greater among early onset smokers than among those in the other two age groups, as well as among lifetime nonsmokers (15.81 vs. 11.35, 8.61, and 8.36, respectively). Smokers who began between the ages of 14 and 16 were also significantly more likely to have diagnoses of major depressive disorder than were late onset (after age 17) smokers and lifetime nonsmokers.

In terms of severity of depressive disorder, early onset smokers had significantly more depressive episodes than did either late onset smokers or lifetime nonsmokers (7.34 vs. 5.71 and 5.06, for early onset smokers, late onset smokers, and lifetime nonsmokers, respectively). Additionally, at the time of their worst depressive episode, early onset smokers had significantly more symptoms than did both late onset smokers and lifetime nonsmokers (11.35 vs. 10.35 and 10.14, respectively). However, only late onset smokers had more treatment for depressive disorders than did lifetime nonsmokers (54.2% vs. 47.3%).

Drug-Related Characteristics

Drug-related characteristics are detailed in Table 4. Respondents who reported first smoking regularly before age 13 were significantly more likely to qualify for DSM-IV diagnoses of drug dependence (9.30%) and abuse (8.07%) than were those respondents who either initiated smoking later (4.82% and 4.94%) or were lifetime nonsmokers (1.99% and 1.44%), respectively for drug dependence and abuse. It is of note that even those who began smoking at or older than age 17 were twice as likely as nonsmokers to receive these diagnoses.

Not surprisingly, those who began smoking before age 13 also began using drugs at a significantly younger age (16.45 vs. 17.9 to 20.7) and received their first diagnoses of drug use disorder at an earlier age than all others (18.7 vs. 19.9 to 22.5). However, both smokers who began at age 14 to 16 years and lifetime nonsmokers were significantly younger than those who began after age 17 when given their drug use disorder diagnosis (20.1 vs. 22.5).

In terms of severity of drug use disorder, those who began smoking before age 13 had significantly more symptoms than did all others (13.4 vs. 8.9 to 11.2). However, those who began at ages 14 to 16 had significantly more symptoms than only the nonsmokers. In addition, the earlier age smokers also had more lifetime participation in all three types of drug treatment than did the other groups.

As seen in Table 4, the use of any type drug is associated with age of smoking onset. Those beginning be-

Table 2. Smoking-Related Characteristics Among Lifetime Smokers by Age of Smoking Onset

Smoking characteristics	Age at smoking onset (in years) <13 (1)	14–16 (2)	≥17 (3)	Significant Comparisons χ^2 or t, $p < 0001$
% Current smokers	94.07	93.80	88.33 (0.39)	(1) v (3), (2) v (3)
Monthly volume of smoking	663.26 (8.80)	574.19 (6.21)	477.50 (5.14)	(1) v (2) (3), (2) v (3)
Duration of smoking (in months)	227.77 (4.19)	198.19 (2.98)	196.04 (2.04)	(1) v (2) v (3)
Volume smoking when smoked daily	24.33 (0.28)	20.97 (4.19)	18.35 (0.17)	(1) v (2) (3), (2) v (3)
Duration of daily smoking (in months)	200.98 (3.03)	188.20 (2.58)	195.37 (2.27)	NS
Time to smoking daily (in years)	4.33 (0.08)	2.25 (0.04)	1.71 (0.05)	(1) v (2) (3) (2) v (3)

Note: Percentages expressed as weighted figures; *n*'s expressed as unweighted figures. Standard errors are in parentheses. NS, not significant.

fore age 13 are more likely to use each of the specific drugs and to develop a drug use disorder than are those in all other groups. Those beginning at ages 14 to 16 followed the same pattern relative to the older smokers and nonsmokers. Additionally, even the older smokers had rates of specific drug use and disorder that were significantly greater than those of the nonsmokers.

The results of the logistic linear multiple regression analysis are presented in Tables 5 and 6. They suggest some modification of these descriptive findings in examining reported lifetime drug use, abuse, and dependence.

Lifetime drug use

Age of smoking onset was significantly and positively associated with lifetime drug use. The earlier one began smoking regularly, the greater his or her chances of reporting lifetime drug use. Relative to lifetime nonsmokers, the odds of lifetime drug use were 7 times (AOR = $e^{1.99}$ = 7.3) as great for those starting before age 13, 5 times as great for those starting between ages 14 and 16, and 2.5 times as great for those initiating regular smoking at age 17 or older.

Respondents between the ages of 45 and 64, as well as those 65 and older at the time of the survey, were 87% and 97% less likely to have used drugs, compared with those under 30. It is of note that all these subjects would have been born before 1945. Another finding of note is that those respondents with a family history positive for alcoholism were twice as likely as all others to report lifetime drug use.

Whereas males were almost twice as likely as females and non-blacks were 60% more likely than blacks to have reported lifetime drug use, the interaction of both sex and race with age of smoking onset presents quite a different picture. Black women in each of the two early smoking (before age 13 and between ages 14 through 16) onset groups were more likely to be drug users than were black men and non-blacks of both sexes. A black woman's risk for drug use was 11.57 [confidence interval (CI) = 4.38, 8.25] when age of smoking onset was under age 13 and 8.21 (CI = 6.02, 11.19) when age of smoking onset was 14 to 16. When smoking onset occurred before age 17, the odds of drug use among non-black women were greater than those of non-black men (7.31, CI = 6.30, 8.49, vs. 4.91, CI = 4.31, 5.60) and approached those of black men (7.78, CI = 5.43, 11.15). When smoking onset occurred later (i.e., at or older than age 17, regardless of sex), Blacks were at greater risk than were non-blacks for using drugs (4.00, CI = 3.02, 5.29 vs. 2.53, CI = 2.28, 2.81).

DSM-IV drug abuse

In contrast to drug use, age of smoking onset was not significantly related to having a DSM-IV diagnosis of drug abuse. Nor did significant interactions of either race or gender modify this association.

Older respondents were less likely to qualify for a diagnosis of drug abuse. Respondents between the ages of 30 and 45, as well as those aged 45 and 64 at the time of the survey, were 24% and 56% less likely to have a DSM-IV diagnosis of drug abuse than were those under age 30. However, duration of drug use was significantly associated with increased risk for diagnosis of drug abuse, 4% for each yearly increment in use.

Men who smoked were 48% more likely than women were, and blacks were 41% less likely than were non-blacks to meet standards for a diagnosis of drug abuse. Respondents with a family history positive for alcoholism were significantly more likely, albeit at only 26% increased risk, to have a drug abuse diagnosis.

DSM-IV drug dependence

Respondents who began smoking before age 13 were twice as likely, and those beginning between ages 14 and 16 were 58% more likely to be diagnosed with drug dependence, as were their nonsmoking counterparts.

Age was also significantly associated with the probability of having a DSM-IV diagnosis of drug depen-

Table 3. Depression-Related Characteristics Among Lifetime Smokers by Age of Onset and Among Lifetime Nonsmokers

Depression-related characteristics	≤13 (1)	14–16 (2)	≥17 (3)	Lifetime nonsmokers (4)	Significant comparisons χ^2 or t, $p < 0001$
Age of onset for major depression	21.70 (0.39)	24.19 (0.47)	26.16 (0.41)	23.16 (0.29)	(1) v (2), (3) v (4)
No. of episodes of depression	7.34 (0.46)	6.03 (0.46)	5.71 (0.41)	5.06 (0.24)	(1) v (4), (3) v (4)
Longest duration of depressive episode	11.44 (1.05)	10.80 (1.54)	10.32 (1.11)	8.06 (0.75)	NS
No. of symptoms during worst episode	11.33 (0.13)	10.69 (0.15)	10.35 (0.12)	10.14 (0.08)	(1) v (3) (4)
% Positive for depressed mood	95.86 (0.71)	94.12 (1.91)	94.80 (0.99)	95.84 (0.57)	NS
% Positive for diminished interest	89.99 (1.36)	89.93 (1.27)	88.95 (1.31)	87.76 (0.87)	NS
% Positive for lifetime major depressive disorder	15.81 (0.58)	11.35 (0.45)	8.61 (0.35)	8.36 (0.22)	(1) v (2) (3), (2) v (3) (4)
% Treated for major depression	45.65 (2.12)	48.80 (2.08)	54.19 (2.10)	47.27 (1.39)	(3) v (4)

Note: Percentages expressed as weighted figures; *n*'s expressed as unweighted figures. Standard errors are in parentheses. NS, not significant.

dence. Relative to those who were 18 to 29 years of age at the time of the survey, respondents aged 30 through 44 at the time of survey 35% were less likely; those aged 45 through 64, 52% less likely; and those over 65, 97% less likely to be diagnosed with drug dependence.

Similarly, as with drug abuse, duration of drug use was significantly associated with increased risk for diagnosis of drug dependence, 4% for each yearly increment in use. However, age of first regular drug use was not a significant factor in accounting for drug dependence. Neither were there significant interactive effects found for race and gender.

DSM-IV Major Depressive Disorder

Early onset of regular tobacco use was significantly and positively associated with having a lifetime diagnosis of major depressive disorder. Relative to lifetime nonsmokers, the odds of ever having been diagnosed with major depression were almost twice as great for Respondents who began smoking before age 13 (1.84, CI = 1.64, 2.06), but were only 33% and 26% greater when regular smoking was begun at ages 14 to 16 and at age 17 and older, respectively.

Respondents who were under 30 years of age at the time of interview were more likely to have had a lifetime diagnosis of major depressive disorder. Compared with this group, Respondents who were 30 to 44, 45 to 64, and 65 or more years of age were 26%, 54%, and 89%, respectively, less likely to have been diagnosed with major depression.

Neither race nor sex interacted significantly with age of smoking onset. The model indicates that women who smoke are 70% more likely than nonsmoking women, and all men and non-blacks who smoke are 60% more likely than all blacks and nonsmokers of other racial groups to have a lifetime diagnosis of major depression. However, respondents with a family history positive for alcoholism were slightly more than twice as likely to have had a diagnosis of major depression.

DISCUSSION

Results of these analyses support the initial study hypotheses that early onset of regular tobacco use is as predictive of drug use and depressive disorders as it is of alcohol use disorders. Additionally, these early onset smokers also evidenced an independent association between family history positive for alcoholism and diagnoses of depression and drug use disorders. This, plus earlier age of onset and more indicators of severity for each condition, suggests that similarities, perhaps of a

Table 4. Drug-Related Characteristics of Lifetime Smokers by Age of Smoking Onset and Lifetime Nonsmokers

Drug-related characteristics	Age at smoking onset (in years) ≤13 (1)	14–16 (2)	≥17 (3)	Lifetime nonsmokers (4)	Significant comparisons χ^2 or t, $p < 0001$
% Lifetime drug dependence	9.30 (0.46)	4.82 (0.28)	1.99 (0.17)	1.00 (0.08)	(1) v (2) (3) (4) (2) v (3) (4)
% Lifetime drug abuse	8.07 (0.45)	4.94 (0.31)	2.70 (0.21)	1.44 (0.09)	(1) v (2) (4), (2) v (3) (4)
Age at first drug use	16.45 (0.16)	17.90 (0.13)	20.15 (0.19)	18.76 (0.18)	(1) v (2) (3), (2) v (3)
Age at onset of drug use disorder	18.66 (0.24)	19.88 (0.22)	22.53 (0.35)	20.14 (0.32)	(1) v (2), (3) (4), (2) v (3), (3) v (4)
Lifetime drug symptoms	13.40 (0.29)	11.20 (0.32)	9.84 (0.38)	8.96 (0.33)	(1) v (2) (3) (4), (2) v (4)
% Lifetime drug inpatient treatment	3.21 (0.30)	1.16 (0.15)	0.44 (0.07)	0.20 (0.04)	(1) v (2) (3) (4), (2) v (3), (4)
% Lifetime drug outpatient treatment	3.56 (0.30)	1.22 (0.15)	0.56 (0.08)	0.27 (0.04)	(1) v (2) (3) (4), (2) v (3) (4)
% Lifetime drug 12-step program	3.70 (0.32)	1.59 (0.18)	0.44 (0.07)	0.21 (0.04)	(1) v (2) (3) (4), (2) v (3) (4)
Lifetime use on own					
Any drug	34.41 (0.79)	25.10 (0.60)	13.97 (0.44)	8.19 (0.24)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Prescription drugs	18.02 (0.63)	9.97 (0.44)	4.67 (0.26)	2.46 (0.14)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Sedatives	7.56 (0.40)	3.73 (0.27)	1.69 (0.15)	0.60 (0.06)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Tranquilizers	8.45 (0.46)	4.00 (0.27)	1.75 (0.15)	0.67 (0.07)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Amphetamines	13.68 (0.59)	6.81 (0.37)	2.86 (0.19)	1.34 (0.10)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Cannabis	31.36 (0.78)	22.54 (0.58)	12.41 (0.41)	7.03 (0.22)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Cocaine	11.39 (0.52)	6.13 (0.31)	2.88 (0.20)	1.32 (0.09)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Hallucinogens	7.29 (0.45)	3.22 (0.25)	1.39 (0.14)	0.50 (0.05)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Lifetime drug use disorder					
Any drug	17.37 (0.62)	9.77 (0.42)	4.69 (0.27)	2.44 (0.13)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Prescription drugs	7.11 (0.43)	2.99 (0.24)	1.34 (0.14)	0.65 (0.06)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Sedatives	2.57 (0.26)	1.01 (0.13)	0.39 (0.07)	0.12 (0.03)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Tranquilizers	0.87 (0.12)	2.99 (0.24)	0.34 (0.07)	0.11 (0.02)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Amphetamines	5.36 (0.38)	2.30 (0.22)	0.88 (0.11)	0.44 (0.05)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Cannabis	13.35 (0.58)	7.35 (0.39)	3.48 (0.23)	1.95 (0.11)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Cocaine	5.83 (0.36)	2.92 (0.23)	0.99 (0.11)	0.44 (0.05)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)
Hallucinogens	2.56 (0.26)	0.90 (0.12)	0.27 (0.06)	0.12 (0.03)	(1) v (2) (3) (4), (2) v (3) (4), (3) v (4)

Note: Percentages expressed as weighted figures; *n*'s expressed as unweighted figures. Standard errors are in parentheses.

Table 5. Logistic Regression Results for Drug Use

Characteristic	Drug Use β	Drug Use SE (B)	Drug Use p value
Age of smoking onset[a]			
Age ≤13	1.99	0.08	<000000
Age 14–16	1.53	0.06	<000000
Age 17+	0.93	0.05	<000000
Current age[b]			
Age 30–44	−0.07	0.04	NS
Age 45–64	−2.04	0.06	<000000
Age 65+	−3.70	0.16	<000000
Gender (effect of male)[c]	0.65	0.05	<000000
Race (effect of black)[c]	−0.55	0.10	<000001
Positive family history of alcoholism	0.69	0.04	<000000

	Onset with gender and race	
	AOR	95% CI
Age of smoking onset ≤13		
Black		
Men	7.78	(5.43, 11.15)
Women	11.57	(8.04, 16.66)
Non-black		
Men	4.91	(4.31, 5.60)
Women	7.31	(6.30, 8.49)
Age of smoking onset 14–16		
Black		
Men	6.01	(4.38, 8.25)
Women	8.21	(6.02, 11.19)
Non-black		
Men	3.37	(2.98, 3.82)
Women	4.61	(4.06, 5.23)
Age of onset 17+		
Black	4.00	(3.02, 5.29)
Nonblack	2.53	(2.28, 2.81)

SE, standard error; AOR, adjusted odds ratio; NS, not significant.
[a] Effect of each age relative to lifetime nonsmokers.
[b] Effect of each age group relative to 18- to 29-year olds.
[c] Odds ratios, 95% CIs for significant interactions of age of smoking.

biological nature, in substance use and depressive disorders may exist. This is especially relevant as knowledge has grown about how these conditions may share a common locus or loci in direct areas of the brain, or alternatively how they stimulate actions and reactions at the cellular level that cause neurochemical alterations common to learning the behaviors associated with these conditions. Indeed, it may be that laboratory findings will outstrip behavioral research (Pomerleau, 1995; Jarvick and Schneider, 1992; Zacny, 1990; Collins, 1990). However, even if a biological or chemical solution becomes available to interrupt the maintenance phase of substance abuse, as it has for depression, findings from this study, if further tested, will be helpful in the remaining need to develop and focus preventive efforts for behaviors antecedent to this phase.

Although the retrospective nature of our data does not permit an adequate assessment of the sequencing effects of early tobacco use and early alcohol use, results do suggest support for hypotheses implicating early, regular tobacco use as a precipitant in alcohol, drug use, and depressive disorders. At the very least, this study suggests, as did Fleming et al. (1989), that smoking itself may play an equally, if not more, insidious role as drinking in the use and development of dependence on illicit substances. Although the nature of our data cannot justify suggesting that tobacco, not alcohol, is the "gateway drug," it does support this possibility, and as such can be addressed in a longitudinal study that expands on the NLAES. At any rate, it highlights arguments by Gordis (1997) that suggest the labeling of alcohol, as a "gateway" [to what is considered the real drug problem (i.e., illicit drug use and depen-

Table 6. Logistic Regression Results for DSM-IV Diagnoses of Drug Abuse, Drug Dependence, and Major Depressive Disorder

	Drug abuse			Drug dependence			Depression		
Characteristic	B	SE (B)	p value	B	SE (B)	p value	B	SE (B)	p value
Age of smoking onset[a]									
Age ≤13	0.12	0.10	NS	0.73	0.11	<000000	0.73	0.07	<000000
Age 14–16	0.05	0.10	NS	0.45	0.11	<0001	0.30	0.06	<00001
Age 17+	0.17	0.11	NS	0.23	0.13	NS	0.24	0.06	<0001
Current age[b]									
Age 30–44	−0.28	0.09	<002	−0.43	0.09	<000004	−0.30	0.05	<000000
Age 45–64	−0.81	0.20	<0001	−0.73	0.21	<001	−0.77	0.06	<000000
Age 65+	−1.53	0.73	NS	−3.62	1.04	<001	−2.22	0.10	<000000
Gender (effect of male)	0.39	0.08	<00001	0.03	0.07	NS	−0.36	0.04	<000000
Race (effect of black)	−0.53	0.15	<001	0.05	0.14	NS	−0.53	0.07	<000000
Positive family history of alcoholism	0.23	0.07	<003	0.67	0.08	<000000	0.78	0.04	<000000
Age of first drug use	−0.03	0.01	<006	−0.03	0.01	NS	—	—	—
Duration of drug use	0.04	0.01	<000000	0.04	0.01	<000000	—	—	—

NS, not significant; SE, standard error.
[a] Effect of each age relative to lifetime nonsmokers.
[b] Effect of each age group relative to 18- to 29-year olds.

dence)] is counterproductive. Even if smoking proves to be the main precipitant of alcohol and drug use disorders, as well as depressive disorders, nicotine addiction itself, like alcoholism, is a serious disease with deadly consequences and its importance would be minimized as is that of any disorder when it is seen merely as preliminary to something more lethal. However, we cannot overlook the fact that like the prevalence of alcohol abuse and dependence, prevalence rates among the early onset smokers for drug abuse, drug dependence, and major depressive orders were far in excess of the comorbidity rates found in the ECA. Whereas the ECA cited prevalence rates of 2.6 and 3.5 for drug abuse and dependence, respectively, we noted rates among early onset smokers of 8.3 for abuse and 9.3 for dependence. With respect to depression, the overall rate of major depressive disorder found in the ECA was 5.9; our nonsmokers had a rate of 8.4 consistent with the rise in depressive illness in the last half of this century. Early onset smokers, however, had nearly double the rate of major depressive disorder: 15.8. It is this young group of smokers that appears to be most at risk for and most likely evidences some common origin for all these conditions. Perhaps this early pattern of smoking should be viewed as a signal of a more complex disorder. Respondents who begin smoking before age 13 are more likely to have a family history positive for alcoholism, be current smokers, and smoke more per day; they are also more likely to receive diagnoses of depression at a younger age and to have more episodes and symptoms of depression. They did not, however, have more treatment for depression relative to later onset smokers. One possible explanation for this is that they were receiving treatment for drug or alcohol conditions. In fact, these early onset smokers also used drugs at younger ages, were diagnosed earlier with drug use disorders of greater severity, and received more drug-specific treatments.

Regardless of age at first regular use, smokers were more likely than nonsmokers to use illicit drugs, as well as to have had a lifetime diagnosis of major depressive disorder. Results indicate that the effects of smoking may be more insidious than alcohol for women and blacks in terms of drug use and dependence. Whereas men were more likely than women to have a diagnosis of drug abuse, and women were more likely than men to have one of depression, there were no significant gender differences for drug use and dependence. Drug use and dependence diagnoses follow a different course.

Risk for drug dependence is greatest for those, regardless of gender and race, who begin smoking before age 16, thus indicating that early use of any drug can be considered a significant factor in later dependence. When smoking regularly begins after age 17, the main effects of race take precedence in explaining the cigarette-drug link. Blacks who begin smoking regularly at later ages are at a 4-fold risk for using drugs, whereas the risk for non-blacks is but twice that of lifetime nonsmokers. This may suggest that drug use is more acceptable in black culture than is alcohol. Or, alternatively, just as black men, like both white and black women, have too low a tolerance level for or a greater vulnerability to alcohol and cannot use enough of it to suit their recreational or medicinal purposes without risking adverse health effects (Hanna et al., 1997a); perhaps the same vulnerability holds true for smoking.

Race and gender modify the relationship of age at onset of regular tobacco use to indicate that relative to nonsmokers, black women who initiate regular smoking before age 13 are at the greatest risk for using drugs, a risk greater than that of black men, white women, and white men, respectively. This same order of risk, albeit of a lesser magnitude, obtains when regular smoking is begun at ages 14 through 16. It is possible that nicotine, for women, may be a more acceptable drug of choice than is alcohol, certainly one with far fewer visible effects. It may also be a more effective antidepressant for women. Additionally, this interaction effect is similar to one found between race and depression in earlier studies of the substance use behaviors of pregnant women (Hanna et al., 1994b). Black women who were depressed made very little change in their use of all substances, even after they learned of an impending pregnancy. They subsequently accounted for more fetal and infant deaths or live births that required extensive hospitalization (Hanna et al., 1997b). It may also serve to strengthen support for studies indicating women have greater difficulty than do men in quitting smoking.

In conclusion, this study—based on data from a large national survey that collected information sufficient to make DSM-IV diagnoses of depression, alcohol, and drug abuse and dependence—lends further support to the possibility of a common factor involved in addictive behaviors and depression. It also makes clear that no substance used early in life should be considered merely a precursor to a "more serious problem." First, use of any substance has its own hazards. Second, if smoking and alcohol can produce the same effects, how do we know for certain that substituting any risky behavior won't do the same? We look forward to addressing these questions in a future study based on longitudinal data that will permit full examination of sequencing, as well as a broader range of diagnoses to model. However, like Clark et al. (1998), who

came to similar conclusions in a small clinical study, we do feel there is sufficient groundwork presented herein to suggest that prevention activities, where successful with any one of these conditions, be developed and tested for applicability to the other problems.

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CHAPTER 8
OTHER ALCOHOL-RELATED PROBLEMS

Alternative Definitions of High Risk for Impaired Driving: The Overlap of High Volume, Frequent Heavy Drinking and Alcohol Dependence

Deborah A. Dawson

This paper examines the distributions of past-year volume of ethanol intake, frequency of drinking 5+ drinks and alcohol dependence in a representative sample of 18,352 U.S. current drinkers aged 18 years or over. Within categories defined by these three partially overlapping domains, it presents rates of self-perceived impaired driving, i.e., driving after having had too much to drink, in the year preceding interview. High volume drinkers, those with an average daily ethanol intake of 1 ounce or more, composed 19.7% of current drinkers and accounted for 66.5% of all reported ethanol consumption, 72.6% of all heavy drinking days, 49.2% of all alcohol dependence and 62.8% of all impaired driving incidents. Frequent heavy drinkers, those who drank 5+ drinks at least once a week, composed 12.3% of current drinkers and accounted for 42.9% of all reported ethanol consumption, 81.9% of all heavy drinking days, 40% of all alcohol dependence and 57% of all impaired driving incidents. Drinkers with DSM-IV alcohol dependence composed 9.9% of current drinkers and accounted for 28.9% of all reported ethanol consumption, 37% of all heavy drinking days and 56.9% of all impaired driving incidents. The overlap of these three high-risk groups, each of which had a probability of at least one impaired driving incident per year, was far from complete. Of individuals who met any of these criteria for high-risk drinking (i.e., high volume, frequent heavy drinking or dependence), more than half met only one criterion and only one in seven met all three. The group that did meet all three criteria had such a high rate of impaired driving incidents, an average of 5.14/year, that it accounted for 36.4% of all such incidents despite making up only 3.8% of all current drinkers. The results are discussed in terms of their implications for targeting prevention and intervention efforts, e.g., whether targeting one problematic aspect of drinking behavior will reach drinkers with other types of problem behaviors as well.

INTRODUCTION

The selection of an appropriate target population represents a significant challenge in the design of prevention and intervention efforts aimed at reducing alcohol related harm. Should specific categories of drinkers or specific drinking behaviors be targeted? If the former, should the focus be on the bulk of drinkers whose low individual risks of adverse outcomes are counterbalanced by their large numbers or on the few drinkers (e.g., alcoholics) who are at highest risk? If the latter, should the focus be on the total volume of consumption or on some appropriate definition of hazardous consumption?

Underlying these choices is the skewed distribution of alcohol consumption in the United States. A large proportion of U.S. adults either never have consumed or no longer consume any alcohol (Dawson et al., 1995), and even among drinkers a small proportion accounts for most of the consumption. In two recent surveys based on nationally representative samples of the U.S. adult population, the proportions of all reported ethanol intake consumed by the top 10% of drinkers in terms of volume were 56.5% and 61.1%, respectively (Greenfield and Rogers, in press). Earlier studies have reported similar findings, with the degree of skew a function of the population used for study. Those based on populations of fairly frequent heavy drinkers, e.g., individuals who had consumed alcohol in the week preceding interview (Klein and Pittman, 1994), have reported a less skewed distribution than those who defined current drinkers more broadly or who included non-drinkers (Malin et al., 1982; Moore and Gerstein 1981).

In the United States the distribution of alcohol *problems* is similarly skewed. Data from the 1984 National Alcohol Study showed that the proportions of current drinkers reporting various levels of depen-

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dence symptoms decreased from 80% reporting no symptoms to 2% reporting six or more symptoms. For social consequences, the proportions declined from 81% reporting no consequences to 1% reporting 16 or more consequences (Hilton, 1991). Thus the vast majority of alcohol related problems were concentrated among a very small percentage of drinkers. More recently, when the current DSM-IV diagnostic criteria (American Psychiatric Association, 1994) for alcohol use disorders were considered, individuals who met the criteria for alcohol dependence made up only 4.38% of the total adult population in the United States (Grant et al., 1994a).

It is this skewness that underlies the 'preventive paradox,' the term applied to the phenomenon described by Kreitman (1986) in which the majority of alcohol related problems were found among low and moderate risk drinkers, when risk was defined in terms of volume of ethanol intake. His widely cited paper is credited for raising awareness that the prevalence of alcohol related problems is a function of both the risks associated with different consumption levels and the distribution of those consumption levels in the general population. However, the paradoxical nature of his findings has been debated by Stockwell et al. (1996), whose analyses indicated that "episodic heavy consumption by people whose average alcohol intake can be classified as 'low' or 'medium' risk contributes to the bulk of such experiences of harm."

Kreitman's paper was frequently cited as an argument for the benefits of the low-risk as opposed to the high-risk approach to reducing alcohol problems, even prompting the recommendation that resources targeted for alcohol treatment programs be diverted into primary prevention efforts (Saunders, 1989). In this ongoing debate, there has been little evidence of the clear superiority of either the high-risk or low-risk approach, based on analyses that have simulated the effects of both approaches in reducing a variety of health and social harms (Norström, 1995; Dawson et al., 1996). However, as these comparisons have essentially rested on manipulating the overall volume of consumption, it can be argued that they have not adequately tested the high-risk approach advocated by Stockwell et al. (1996) i.e., one that is aimed at reducing hazardous consumption. Stockwell and his colleagues defined hazardous consumption as ethanol intake in excess of 60 g on any drinking day, a definition that is arguably more strongly aimed at reducing the acute, short-term consequences of excessive alcohol intake (e.g., violence, accidents, etc.) than at reducing chronic alcohol related morbidity (e.g., liver cirrhosis).

In the debate surrounding the most effective approach to reducing alcohol related harm, there have been few papers examining the overlap of risk groups or comparing attributable risks under different definitions of risk drinking. Even laudable efforts such as the paper previously cited by Stockwell et al. (1996) looked at only two dimensions of risk, volume and episodic heavy drinking, while ignoring alcohol dependence. This is an unfortunate omission, in that a better understanding of the degree of overlap among alcoholics, high volume drinkers and frequent heavy drinkers (and the extent to which these groups individually and jointly contribute to alcohol related problems) could help to inform decisions regarding the allocation of resources among prevention and intervention efforts.

This analysis utilizes a large (n = 18,352) representative sample of U.S. drinkers 18 years of age and over to examine the intersection of groups defined by average daily volume of ethanol intake, frequency of drinking 5+ drinks and DSM-IV alcohol dependence (American Psychiatric Association, 1994). Within categories of each of these three dimensions, the paper presents the mean volume of intake and frequency of heavy drinking, the prevalence of dependence, and the proportions of total intake, heavy drinking days and cases of dependence attributed to the category. For the outcome of self-perceived impaired driving, it also presents each category's mean prevalence of any impaired driving, mean number of past-year impaired driving incidents and the proportions of impaired drivers and impaired driving incidents attributed to the category. Defining as high-risk those categories with an annual rate of at least one impaired driving incident per person, the paper describes the overlap of the high-risk groups for each of the domains. Finally, the limitations and policy implications of the study results are discussed.

METHODS

SAMPLE

This analysis is based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism. This survey gathered information on alcohol consumption and alcohol related problems from 42,862 U.S. adults 18 years of age and over, one of whom was selected at random from each household in a sample that was representative of the non-institutionalized population of the coterminous United States. The household- and

sample-person response rates were 92% and 97%, respectively, with non-response representing refusal to participate and inability to locate the sample housing unit or to find anyone at home. Data were collected in personal interviews conducted in respondents' homes by U.S. Census Bureau interviewers. The average length of interview was 68 min. The complex, multistage sample design included the selection of primary sampling units with probability proportional to size and oversampling of individuals who were of black race or between the ages of 18 and 29 years (Grant et al., 1994b). This analysis was restricted to the 18,352 individuals identified as past-year drinkers, defined as individuals who in response to screening questions reported having consumed at least 12 alcoholic drinks in the year preceding interview.

Measures

For each type of beverage that NLAES respondents reported drinking, they were asked the overall frequency of drinking that beverage during the past year, usual quantity of drinks consumed per drinking day, usual size of drink, heaviest quantity of drinks consumed per drinking day, frequency of drinking the heaviest quantity and the size of drink associated with the heaviest quantity. All frequencies were converted to days/year using the midpoints of categorical response categories (e.g., 2–3 days a month = 30 days/year). Using ethanol conversion factors of 0.045 for beer, 0.121 for wine and 0.409 for liquor (DISCUS, 1985; Kling, 1989; Turner, 1990; Modern Brewery Age, 1992; Williams et al., 1993), the annual volume of ethanol intake was calculated as the sum of the beverage-specific volumes: [(total frequency$_{bev}$ – frequency of drinking heaviest quantity$_{bev}$) x usual quantity$_{bev}$ x usual size$_{bev}$ x ethanol conversion factor$_{bev}$] + [frequency of drinking heaviest quantity$_{bev}$ x heaviest quantity$_{bev}$ x size of heaviest quantity$_{bev}$ x ethanol conversion factor $_{bev}$]. Average daily ethanol intake was calculated by dividing the annual volume by 365. Frequency of drinking 5+ drinks was asked directly of the respondents, and categorical response categories were transformed into days per year using the midpoints of the categories as described above.

Past-year alcohol dependence was classified in accordance with the most recent DSM-IV criteria for these disorders (American Psychiatric Association, 1994) and was measured using the AUDADIS (Grant and Hasin, 1992), a structured interview schedule designed for administration by lay interviewers and embedded in the NLAES questionnaire. To be classified with alcohol dependence, an individual had to meet three or more of the seven DSM-IV criteria for dependence: (1) tolerance; (2) withdrawal (including relief or avoidance of withdrawal); (3) persistent desire or unsuccessful attempts to cut down on or stop drinking; (4) much time spent drinking, obtaining alcohol or recovering from its effects; (5) reduction or cessation of important activities in favor of drinking; (6) impaired control over drinking; and (7) continued use despite physical or psychological problems caused by drinking. Criteria not associated with duration qualifiers were satisfied if an individual reported one or more positive symptoms of the criterion during the past year. Criteria with duration qualifiers were satisfied if a person reported two or more symptoms during the past year or one symptom that occurred at least two times during the past year. To be consistent with the syndromal definition of the withdrawal criterion, two or more positive symptoms were required in addition to satisfaction of the duration qualifier.

Estimates of impaired driving were based on questions that asked whether and how often the respondent had within the year preceding interview driven a car or another motorized vehicle such as a boat or motorcycle after having had too much to drink. The categorical response categories were converted to number of impaired driving incidents per year as follows: 1 time = 1, 2 times = 2, 3–4 times = 3.5, 5–6 times = 5.5, 7–8 times = 7.5, 9–12 times = 10.5, 13–19 times = 16, and 20 or more times = 24.

In a test-retest survey conducted in a representative community sample (Grant et al., 1995), the classification of past-year alcohol use disorders (no disorder, abuse or dependence) demonstrated a reliability coefficient of 0.76, the number of symptoms of past-year dependence demonstrated a reliability coefficient of 0.75, and average daily ethanol intake demonstrated a reliability coefficient of 0.73—all in the excellent or near excellent range (Fleiss, 1981). The reliability of the frequency of drinking 5+ drinks was not estimated, but overall frequency of drinking had a reliability coefficient of 0.76. Likewise, the reliability of the measure of impaired driving was not estimated, but the reliability of the number of symptoms of past-year alcohol abuse, of which impaired driving was the most frequently endorsed, had a reliability of 0.73.

Analysis

All tables present percentages and means based on weighted data, accompanied by the unweighted numbers of cases (*n*'s) upon which they are based. The standard errors of the percentages of current drinkers in various categories and of mean volumes of ethanol

intake, frequencies of heavy drinking and frequencies of impaired driving were obtained directly from statistical runs performed in SUDAAN (Shah et al., 1995), a software package that utilizes Taylor series linearization to account for the effect of complex sample design characteristics on variance estimates. In order to obtain the standard errors for the proportions of volume, heavy drinking days, and impaired driving incidents accounted for by various drinking categories, the variances of the respective numerators and denominators were taken directly from SUDAAN runs, and the delta method (Stuart and Ord, 1987) was then used to estimate the variance of the numerator divided by the denominator.

RESULTS

Table 1 presents the distribution of current drinkers 18 years of age and over in terms of three dimensions: average daily volume of ethanol intake, frequency of heavy drinking and presence or absence of past-year alcohol dependence. Less than one-fifth (19.7%) of current drinkers had high volumes of intake, i.e., an average daily ethanol intake of one or more ounces, but these high volume drinkers accounted for two-thirds (66.5%) of all reported ethanol consumed. (The top 5% of drinkers in terms of volume, those whose average intake was 2.5 ounces or more, drank more than one-third of all reported ethanol intake.) High volume drinkers also accounted for nearly three-quarters (72.6%) of all heavy drinking days and for nearly half (49.2%) of all cases of alcohol dependence.

Nearly half (44.4%) of current drinkers reported no days of heavy drinking, i.e., never having consumed 5+ drinks in the past year. One-quarter (25.8%) reported drinking 5+ drinks at least once a month, and about half of these (12.3% of all current drinkers) reported heavy drinking on a weekly or more frequent basis. This latter group of frequent (weekly) heavy drinkers also accounted for 42.9% of the total volume of ethanol intake and 40% of all cases of alcohol dependence. In contrast, individuals with no heavy drinking days accounted for 19.3% of the total volume of intake and 5.7% of all cases of alcohol dependence.

Only one-tenth (9.9%) of current drinkers satisfied the criteria for past-year DSM-IV alcohol dependence, but this group accounted for 28.9% of all reported ethanol consumption and 37% of all heavy drinking days. Notably, the dependent drinkers had average daily ethanol intakes that were lower than those of frequent heavy drinkers (2.13 vs. 2.54 ounces) as well as having had fewer heavy drinking days in the past year (an average of 83.2 vs. 147.6).

Table 2 shows how these dimensions of drinking behavior and problems were related to impaired driving, that is, driving after having had too much to drink. Overall, 11.8% of current drinkers reported one or more incidents of impaired driving in the past year, and the mean annual number of impaired driving incidents was 0.54. The prevalence of any impaired driving

Table 1. Past-year characteristics of current drinkers 18 years of age and over within categories of average daily volume of ethanol intake, frequency of drinking 5+ drinks and DSM-IV alcohol dependence

	n	% Of all currrent drinkers in category	Average daily ethanol intake (mean)	% Of total ethanol intake consumed ny category	Frequency of drinking 5+ drinks (mean)	% Of all 5+ drinking days accounted for by category	Prevalence of DSM-IV alcohol dependence (%)	% Of all dependent drinkers in category
All current drinkers[a]	18 352	100.0 (0.0)	0.72 (0.1)	100.0 (0.0)	22.3 (0.6)	100.0 (0.0)	16.7 (0.4)	100.0 (0.0)
Past-year average daily ethanol intake (ounces)								
<0.10	4192	22.6 (0.4)	0.05 (<0.01)	1.7 (0.1)	1.2 (0.1)	1.3 (0.2)	2.2 (0.3)	3.1 (0.4)
0.10–0.24	3861	21.5 (0.4)	0.17 (<0.01)	5.0 (0.2)	3.4 (0.2)	3.4 (0.2)	7.3 (0.5)	9.4 (0.6)
0.25–0.49	3251	18.1 (0.3)	0.36 (<0.01)	9.0 (0.3)	8.2 (0.4)	6.8 (0.4)	12.7 (0.8)	13.8 (0.8)
0.50–0.74	2012	11.2 (0.3)	0.62 (<0.01)	9.6 (0.4)	17.6 (0.8)	9.0 (0.6)	19.9 (2.2)	13.4 (0.7)
0.75–0.99	1235	6.9 (0.2)	0.87 (<0.01)	8.2 (0.4)	22.4 (1.1)	7.0 (0.5)	26.9 (1.4)	11.1 (0.7)
1.00–2.49	2577	14.5 (0.3)	1.53 (0.01)	31.0 (1.1)	45.5 (1.6)	30.2 (1.6)	34.4 (1.2)	30.1 (1.0)
≥2.50	886	5.2 (0.2)	4.89 (0.17)	35.5 (2.1)	177.2 (5.5)	42.4 (2.7)	60.5 (2.0)	19.1 (1.0)
Frequency of drinking 5+drinks in past year								
Never	8389	44.4 (0.5)	0.31 (0.01)	19.3 (0.7)	0.0 (0.0)	0.0 (0.0)	2.1 (0.2)	5.7 (0.5)
1–2 times	2664	15.1 (0.3)	0.40 (0.01)	8.3 (0.4)	1.5 (<0.1)	1.0 (<0.1)	8.6 (0.6)	7.8 (0.5)
3–11 times	2554	14.7 (0.3)	0.64 (0.01)	13.1 (0.6)	6.5 (<0.1)	4.3 (0.2)	23.5 (1.0)	20.6 (0.9)
1–3 times/ month	2263	13.5 (0.3)	0.88 (0.02)	16.5 (0.8)	21.3 (0.2)	12.9 (0.6)	32.2 (1.2)	25.9 (0.9)
≥1/week	2049	12.3 (0.3)	2.54 (0.09)	42.9 (2.3)	147.6 (2.8)	81.9 (3.9)	54.3 (1.3)	40.0 (1.1)
Past-year DSM-IV alcohol dependence								
No dependence	16 338	90.1 (0.3)	0.57 (0.01)	71.1 (2.3)	15.6 (0.5)	63.0 (3.0)	0.0 (0.0)	0.0 (0.0)
Dependence	1676	9.9 (0.3)	2.13 (0.10)	28.9 (0.7)	83.2 (2.9)	37.0 (2.2)	100.0 (0.0)	100.0 (0.0)

[a] Including drinkers with unknown volume of intake and/or frequency of heavy drinking.

rose from 2.5% of the lowest volume drinkers to 39.4% of the highest volume drinkers and from 1.4% of individuals with no heavy drinking days to 37.2% of those who drank 5+ drinks once a week or more often. The mean number of impaired driving incidents showed an even steeper increase with volume and frequency of heavy drinking. Dependent drinkers were six times as likely to report any impaired driving as were those without alcohol dependence, 47.5 vs. 7.9%, and reported more than ten times as many impaired driving incidents, 3.10 vs. 0.26 on average.

The data in Table 2 present several examples of the preventive paradox, i.e., of cases where the drinking categories with the highest probabilities of impaired driving did not have the highest attributable risk of this outcome. For example, individuals with average daily ethanol intakes of 2.50 ounces or more had a prevalence of any impaired driving that was almost twice as high as that for drinkers who consumed a daily average of 1.00–2.49 ounces, 39.4 vs. 22.8%, but they accounted for a much smaller proportion of all impaired drivers, 17.3 vs. 27.9%. Because the latter group (consuming 1.00–2.49 ounces) was so much larger than the former (consuming 2.50+ ounces), the two accounted for almost equal proportions of all impaired driving incidents, despite the incidence rate being three times higher among the highest volume drinkers.

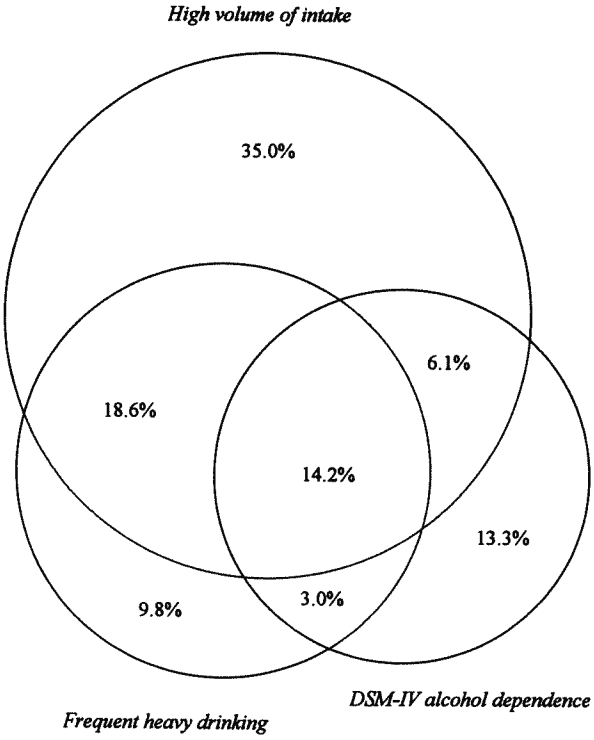
If we classify as high-risk drinkers those groups with mean numbers of impaired driving incidents that are greater than 1.00 (i.e., those who have an expected value of at least one such incident per year), the following groups of drinkers are designated as being at high risk: (1) high volume drinkers—those with average daily ethanol intakes of one or more ounces; (2) frequent heavy drinkers—those who drank 5+ drinks per day on a weekly or more frequent basis, and (3) dependent drinkers. Respectively speaking, these three groups accounted for 19.7%, 12.3% and 9.9% of all current drinkers, 45.2%, 38.6% and 39.5% of all impaired drivers and 62.8%, 57% and 56.9% of all impaired driving incidents. (Percentages may sum to more than 100% because of overlap among the three groups; i.e., an individual may be represented in more than one of the high-risk categories.) Taken as a whole, i.e., when the three high-risk categories are combined, high-risk drinkers made up 26.7% of all current drinkers (Table 3). They accounted for 71.7% of total ethanol intake, for 88.3% of all heavy drinking days and, by definition, for all cases of alcohol dependence.

Table 2. Level of past-year impaired driving among current drinkers 18 years of age and over within categories of average daily volume of ethanol intake, frequency of drinking 5+ drinks and DSM-IV alcohol dependence

	n	Prevalence of any impaired driving (%)	% Of all impaired drivers in category	Mean number of impaired driving incidents	% Of impaired driving incidents accounted for by category
All current drinkers[a]	18 352	11.8 (0.3)	100.0 (0.0)	0.54 (0.02)	100.0 (0.0)
Past-year average daily ethanol intake (ounces)					
<0.10	4192	2.5 (0.3)	4.7 (0.5)	0.06 (0.01)	2.4 (0.5)
0.10–0.24	3861	5.5 (0.4)	10.0 (0.7)	0.13 (0.02)	5.4 (0.7)
0.25–0.49	3251	10.1 (0.7)	15.3 (1.0)	0.29 (0.03)	9.8 (1.1)
0.50–0.74	2012	14.3 (0.9)	13.4 (0.8)	0.48 (0.05)	10.0 (1.1)
0.75–0.99	1235	19.7 (1.3)	11.4 (0.8)	0.75 (0.08)	9.6 (1.2)
1.00–2.49	2577	22.8 (1.0)	27.9 (1.2)	1.11 (0.08)	30.5 (2.8)
≥2.50	886	39.4 (2.0)	17.3 (1.1)	3.30 (0.28)	32.3 (3.5)
Frequency of drinking 5+drinks in past year					
Never	8389	1.4 (0.1)	5.4 (0.5)	0.03 (0.01)	2.8 (0.4)
1–2 times	2664	7.8 (0.6)	9.9 (0.7)	0.17 (0.02)	4.7 (0.6)
3–11 times	2554	16.6 (0.8)	20.5 (1.0)	0.55 (0.04)	14.9 (1.5)
1–3 times/month	2263	22.6 (1.0)	25.6 (1.1)	0.83 (0.07)	20.6 (2.0)
≥1/week	2049	37.2 (1.2)	38.6 (1.3)	2.51 (0.15)	57.0 (4.7)
Past-year DSM-IV alcohol dependence					
No dependence	16 338	7.9 (0.3)	60.5 (1.3)	0.26 (0.01)	43.1 (0.3)
Dependence	1676	47.5 (1.4)	39.5 (1.3)	3.10 (0.18)	56.9 (4.7)

[a] Including drinkers with unknown volumes of intake and/or frequency of heavy drinking.

Figure 1. Overlap of high risk drinkers defined by volume of ethanol intake, frequency of heavy drinking and DSM-IV alcohol dependence



From the perspective of targeting prevention and intervention efforts, a question of interest is the extent to which the three individual high-risk groups of drinkers overlap. As shown in Fig. 1 and Table 3, there was partial but by no means total overlap of high volume drinkers, frequent heavy drinkers and dependent drinkers. Only 14.2% of all high-risk drinkers fell into all three of the individual high-risk categories. More than half were characterized by only one of the risk patterns: 35% with high volume only, 9.8% with frequent heavy drinking only and 13.3% with alcohol dependence only. Nearly half of all high volume drinkers exhibited neither frequent heavy drinking nor dependence. In contrast, only 21.5% of frequent heavy drinkers exhibited neither high volume drinking nor dependence, and only 36.3% of dependent drinkers were neither high volume nor frequent heavy drinkers.

As might be expected, the prevalence of impaired driving and mean number of impaired driving incidents were greatest among individuals with multiple high-risk drinking patterns (Table 4). Those with high volume, frequent heavy drinking *and* alcohol dependence reported driving after having had too much to drink an average of 5.14 times in the year preceding interview, about twice as often as those with dependence coupled with *either* frequent heavy drinking or high volume (but not both) and almost four times as often as those with dependence only or with a combination of high volume and frequent heavy drinking. Thus the group with all three high-risk patterns accounted for 36.4% of all impaired driving incidents, although comprising only 3.8% of all current drinkers, 14.2% of the high-risk group and 18.8% of all impaired drivers.

DISCUSSION

This analysis revealed that although there is substantial overlap among high volume drinkers, frequent heavy drinkers and individuals with alcohol dependence, the overlap is by no means complete. In fact, more than half of the drinkers identified as being at high risk of impaired driving met the criterion for high risk in only one of these three domains. Only one in seven of the high-risk drinkers fell into all three risk domains.

The degree of overlap found in this study was necessarily a function of how the high-risk groups were defined—which was by means of thresholds that identified categories of individuals with an expected frequency of at least one impaired driving incident in the past year. Other thresholds for high risk would have yielded different results. For example, had the high-risk threshold for volume of intake been raised to 2.50 or more ounces of ethanol/day (which would have resulted in greater similarity in the rates of impaired driving across the three high-risk groups), the reduction in size of the high volume risk group would have resulted in its having a substantially greater overlap with the other high-risk groups. The proportion of high-risk drinkers with high volume as their sole risk criterion would have declined from 35% to 5.5%.

Changing the measures of volume, heavy drinking or alcohol use disorders also would have resulted in different results. Had the volume measure been adjusted for total body water (e.g., if the high-risk threshold had been set at a daily average of 50+ mg/dl total body water), then the proportion of high-risk drinkers with high volume as their sole risk criterion would have increased slightly, from 35% to 42.6%. Had individuals with *either* abuse or dependence been considered as at high risk, then the proportion of high-risk drinkers defined solely by the presence of an alcohol use disorder would have risen from 13.3% to 23.7%. (The reason that abusers were not considered as a risk group in this analysis is because impaired driving is one of the symptom item indicators by which abuse is defined.)

As the examples above indicate, redefining the measures or thresholds for risk does change the overlap of the high-risk groups. However, it does not change the basic finding of this study. Regardless of how the criteria for high risk are manipulated, the overlap among the risk groups defined by volume, heavy drinking and alcohol use disorders is far from complete.

It is difficult to predict how this study's estimates of the overlap of risk drinking domains and of attributable fractions of risk may have been affected by under-reporting of consumption. Studies based on retrospective recall of alcohol consumption typically account for only about half of the level of consumption estimated from sales data (Midanik, 1982, 1994). Some of this undercount is undoubtedly a function of the limited nature of household samples, which exclude the homeless, institutionalized and military populations. Rehm (1998) has argued that the consumption levels of these groups are such that we should expect coverage rates of no more than 70% for household surveys. A study of the non-household population conducted in one California county (Weisner et al., 1995) found rates of problem drinking that were more than quadruple those of the general population. Regarding that portion of under-reported consumption that is tied to incomplete recall, there is evidence that under-reporting of heavy drinking occasions or amounts exceeds that of lighter occasions or amounts (Hilton, 1991; Perrine et al., 1995). Accordingly, this analysis may underestimate the proportions of high-risk drinkers in the categories that include frequent heavy drinking and possibly also the proportion of impaired driving attributable to frequent heavy drinking. However, this conclusion is speculative, and no firm conclusions can be drawn as to the effect of under-reporting of consumption.

It should be noted that this study's estimates of the proportion of total ethanol intake consumed by high volume drinkers are remarkably consistent with those from two other recent surveys based on household samples (Greenfield and Rogers, in press), after accounting for the different populations of drinkers upon which the distributions were based. (Greenfield and Rogers considered all drinkers, whereas the NLAES definition of a current drinker stipulated a minimum consumption of at least 12 drinks in the past year. Other studies that have included abstainers in their distributions have of course found a far more skewed distribution.)

Another important question is the extent to which tolerance to the effects of alcohol may have affected the perception of impaired driving among alcoholics relative to non-alcoholics. Almost two-thirds (64%) of all individuals classified with past-year alcohol dependence reported tolerance symptoms, compared to only 11% of high volume drinkers and 7% of frequent heavy drinkers. Several multivariate models that were tested to examine this issue (data not shown) revealed that at comparable levels of volume, drinkers with tolerance were only about half as likely to report impaired driving as were those without tolerance. Interestingly, at comparable numbers of heavy drinking days, drinkers with and without tolerance were equally likely to report impaired driving; however, it is possible that drinkers with tolerance drank a greater *quantity* of drinks on those heavy drinking days than did drinkers without tolerance. In sum, one reason why such a small proportion of impaired driving incidents can be attributed to dependent individuals may be that their tolerance levels make them less likely to report this problem. What cannot be determined is whether their increased tolerance actually reduces the level of psychomotor impairment associated with a given blood

Table 3. Past-year characteristics of current drinkers 18 years of age and over within categories of high-risk drinking

	n	% Of all high risk drinkers in category	% Of all current drinkers in category	% Of total ethanol intake consumed by category	% Of all 5+ drinking days accounted for by category	% Of all drinkers with dependence in category
No high-risk drinking	13282	0.0 (0.0)	73.3 (0.4)	28.3 (0.8)	11.7 (0.1)	0.0 (0.0)
Any high risk drinking:	4637	100.0 (0.0)	26.7 (0.4)	71.7 (2.8)	88.3 (4.1)	100.0 (0.0)
High volume[a] only	1686	35.0 (0.9)	9.4 (0.2)	22.0 (0.9)	3.8 (0.2)	0.0 (0.0)
Frequent heavy drinking[b] only	453	9.8 (0.5)	2.6 (0.1)	2.3 (0.1)	10.7 (0.7)	0.0 (0.0)
Dependence only	602	13.3 (0.6)	3.5 (0.2)	2.2 (0.1)	1.8 (0.1)	36.3 (1.4)
High volume[a] and frequent heavy drinking[b]	829	18.6 (0.7)	5.0 (0.2)	18.5 (1.2)	37.0 (2.4)	0.0 (0.0)
High volume[a] and dependence	300	6.1 (0.4)	1.6 (0.1)	4.6 (0.4)	1.2 (0.1)	16.8 (1.0)
Frequent heavy drinking[b] and dependence	139	3.0 (0.3)	0.8 (0.1)	0.8 (0.1)	3.2 (0.3)	8.2 (0.7)
High volume[a], frequent heavy drinking[b] and dependence	628	14.2 (0.6)	3.8 (0.2)	21.3 (1.7)	30.6 (2.0)	38.7 (1.4)

[a] Average daily ethanol intake of 1 ounce or more.
[b] Drank 5+drinks once a week or more often.

alcohol level, or whether they are merely less likely to *perceive* it as being impaired.

In assessing the impact of these findings on policies for reducing alcohol related harm, it is important to recognize the potential for spillover in the aspects of risk drinking that are affected by different types of alcohol policies. For example, policies aimed at reducing overall volume of consumption may indirectly influence the prevalence or frequency of heavy drinking if, for example, heavy drinking groups such as young drinkers are particularly sensitive to cost increases (Grossman et al., 1987; Coate and Grossman, 1988). Similarly, any reduction of heavy drinking also will reduce the overall volume of alcohol consumed (except in the unlikely event that drinkers compensate for their reduced per occasion intake by engaging in more moderate drinking occasions); however, the reduction will be among heavy drinkers only and not among all drinkers. Policies aimed at getting alcoholics into treatment should result in a reduction in both heavy drinking and volume of consumption, but only among those drinkers who enter treatment and who succeed in reducing their consumption. (Treatment outcomes that fall short of a goal of lifelong abstinence may still result in a reduction in both volume of intake and frequency of heavy drinking.)

At the same time, there are limits to the extent to which effects can spill over from one domain of risk drinking to another. Preventive policies aimed at drinking behavior may have little effect on individuals with alcohol dependence if their impaired control or symptoms of physiological dependence make them less sensitive to cost increases than other drinkers. (To the extent that the heaviest drinkers spend more of their disposable income on alcohol, they would appear to be especially susceptible to price increases, especially if disproportionately represented among the poor; however, those with a true physiological addiction to ethanol may be willing to spend a greater share of their income on alcohol or, alternatively, to substitute lower for higher cost brands or private for public drinking milieus.) Additionally, policies that focus specifically on the treatment of alcohol use disorders are not likely to yield reductions in consumption outside of the treated population and thus may not lead to the drier drinking environment that might help to reduce consumption among non-dependent drinkers in the far right tail of the consumption distribution (Skog, 1985).

For the specific outcome of impaired driving there are, of course, many policies that do not inherently target any drinking behavior or type of drinker. These include aspects of prevention and enforcement such as random road testing, sentencing levels and license suspension. Applying the findings of this study beyond impaired driving to a wider range of alcohol related consequences, the incomplete overlap of high-risk drinking domains and limits of treatment and intervention to address all of these domains simultaneously suggest that resources should continue to be funneled into a combination of strategies—both those that pre-

Table 4. Level of past-year impaired driving among current drinkers 18 years of age and over within categories of high-risk drinking

	n	Prevalence of any impaired driving (%)	% Of all impaired drivers in category	Mean number of impaired driving incidents	% Of impaired driving incidents accounted for by category
No high-risk drinking	13 282	5.8 (0.3)	35.7 (1.3)	0.15 (0.01)	20.5 (1.7)
Any high-risk drinking:	4637	28.7 (0.8)	64.3 (1.3)	1.59 (0.08)	79.5 (5.7)
High volume[a] only	1686	11.3 (0.9)	8.8 (0.7)	0.39 (0.05)	6.8 (1.0)
Frequent heavy drinking[b] only	453	20.2 (2.1)	4.4 (0.5)	0.79 (0.13)	3.8 (0.7)
Dependence only	602	37.0 (2.1)	11.0 (0.7)	1.31 (0.15)	8.7 (1.2)
High volume[a] and frequent heavy[b] drinking	829	28.1 (1.9)	11.7 (0.9)	1.32 (0.13)	12.2 (1.5)
High volume[a] and dependence	300	43.5 (3.3)	6.0 (0.6)	2.40 (0.30)	7.4 (1.1)
Frequent heavy drinking[b]and dependence	139	53.5 (4.8)	3.6 (0.5)	2.78 (0.50)	4.2 (0.8)
High volume[a], frequent heavydrinking[b] and dependence	628	58.9 (2.3)	18.8 (2.3)	5.14 (0.38)	36.4 (3.8)

[a] Average daily ethanol intake of 1 ounce or more.
[b] Drank 5+drinks once a week or more often.

vent hazardous consumption among all drinkers, including those with low to moderate volumes of intake, and those that offer intervention in the form of treatment to individuals with alcohol dependence. In the absence of the latter, the substantial proportions of total consumption and heavy drinking occasions contributed by individuals with alcohol dependence may be only marginally affected by prevention efforts more suitable for the general population.

ACKNOWLEDGMENTS

I am grateful for the many helpful suggestions made by Tom Greenfield on early drafts and in the conceptualization of this paper.

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Suicidal Ideation Among the United States Drinking Population: Results From the National Longitudinal Alcohol Epidemiologic Survey

Bridget F. Grant and Deborah S. Hasin

Objective: *Data from a national representative sample of adults was used to identify major risk factors of suicidal ideation among the U.S. drinking population*. Method: *Data from a sample of 18,352 current drinkers, 18 years of age and older, were analyzed by means of multiple logistic regression analysis. In these analyses, multivariate associations were examined between risk factors for suicidal ideation and the occurrence of suicidal ideation*. Results: *For men and women, past year major depression and alcohol dependence were identified as risk factors of suicidal ideation, with major depression having the more sizable impact. Suicidal ideation was increased among men with a past alcohol use disorder, and elevated among women who had used drugs nonmedically and developed a drug use disorder during the past year. The occurrence of a recent physical illness and lifetime treatment for major depression among men and women increased the risk of suicidal ideation, while marriage was protective against ideation for both sexes. Unemployment and having a family history of alcoholism increased the risk of suicidal ideation among men, but not women*. Conclusions: *Major findings are discussed in terms of the impact of severity versus chronicity of psychopathology on suicidal ideation, gender roles and differential engagement in suicidal ideation, and the recognition and treatment of major depression as the single most important intervention in reducing suicidal behavior.*

Many investigators have attempted to characterize persons who commit suicide or have attempted suicide. Data on risk factors for suicide and attempted suicide have been obtained from psychological autopsy studies of suicide victims, interviews with selected subsamples of the general population (usually adolescents) and, primarily, from clinical interviews or follow-up studies of alcoholics. Risk factors consistently identified for suicide completion among alcoholics include being male and older than approximately 50 years of age (Roy, 1993; Roy et al., 1990), histories of major depression and serious medical problems, living alone, unemployment, having little or no social support (Murphy et al., 1992), and experiencing recent adverse life events (Duberstein et al., 1993; Heikkinen et al., 1994). In addition to most of these risk factors, studies of suicide attempters among alcoholics have identified other predictors, including histories of substance abuse and antisocial personality disorder (Hesselbrock et al., 1988; Roy et al., 1990; Windle, 1994), family histories of alcoholism (Schuckit, 1986), early onset of drinking or alcoholism (Hesselbrock et al., 1988; Roy, 1993), and previous use of drugs and prior alcohol treatment (Black et al., 1986; Schuckit, 1986). Similar risk factors have been identified among psychiatric patients (Hirshfeld and Davidson, 1988), suicide victims and attempters (Robins et al., 1959; Shafi et al., 1995) and adolescents (Bukstein et al., 1993).

Moreover, most of our knowledge of all suicidal behaviors, including suicidal ideation, is derived from psychological autopsies of suicide victims and interviews with selected community samples and alcoholics. Both types of data are difficult to relate to the general population. For example, studies of alcoholics in treatment are not representative of all alcoholics. Results from the 1992 National Longitudinal Alcohol Epidemiologic Survey indicate that of the population classified with a past year alcohol abuse and/or dependence diagnosis, only 9.9% had obtained alcohol treatment during that time (Grant, 1997). Similarly, studies of adolescents and psychological autopsies are not generalizable to the entire general population.

Also, very little is known about the role of alcohol in suicidal behaviors. Alcohol can relate to suicidal be-

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haviors in two ways. The disinhibitory effects of alcohol on behavior can be involved in suicidal behaviors, usually as a precipitant, or alcohol dependence itself can be considered a risk factor. Prior research focusing on the role of alcohol consumption exclusively among alcoholics has ignored the effect of alcohol consumption on suicidal behaviors among the broader population of alcohol users. Similarly, studies of untreated populations have focused on alcohol consumption at the exclusion of alcohol dependence.

Another limitation of existing research examining suicidal behaviors is the failure to equate measures of alcoholism, psychopathology and suicidal behaviors in terms of a single time frame. In most of these studies, relationships have been examined between lifetime measures of alcoholism and other psychiatric disorders and/or responses to questions of ever attempting suicide (Hesselbrock et al., 1988; Roy et al., 1990; Whitters et al., 1985; Windle, 1994). The use of lifetime measures makes it difficult to assess the impact of comorbidity between alcohol use disorders and other psychopathology on ideation. Other weaknesses in this literature is the exclusion of males (Gomberg, 1989) or females (Black et al., 1986; Burch, 1994; Schuckit, 1986) from the study samples, absence of analyses by gender (Whitters et al., 1985; Windle, 1994), and the restricted range of predictors for suicidal behaviors.

The present study overcomes many of the limitations of the previous research and fills a gap in the literature on suicidal behaviors. First, this study is based on a large representative sample of the United States drinking population with the major objective of identifying risk factors for suicidal ideation. The relationship between predictors and suicidal ideation among the general drinking population will help to determine the role of both alcohol consumption and alcohol dependence on suicidal ideation. Second, this study examines a broader set of putative risk factors for suicidal ideation than assessed in prior research on suicidal behaviors, importantly measuring alcohol and drug use and disorders, associated psychopathology and suicidal ideation within the same past year time frame. Third, the risk for suicidal ideation is estimated for respondents classified directly in terms of comorbidity, that is, among individuals with diagnoses of alcohol dependence without major depression, diagnoses of major depression without alcohol dependence, and among those respondents with comorbid alcohol dependence and major depression. Fourth, all psychiatric diagnoses were made using the most recent psychiatric classification of mental disorders, the Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition (DSM-IV; American Psychiatric Association, 1994), and analyses were conducted separately for each gender.

METHOD

Sample

This study is based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism (Grant et al., 1994b). Data were collected in personal interviews conducted in respondents' homes by interviewers of the U.S. Bureau of the Census. The NLAES sample consisted of U.S. adults 18 years of age or older who were selected at random from a nationally representative sample of households. The multistage sampling design featured selection of primary sampling units with probability proportional to size, oversampling of segments with high proportions of black residents and oversampling of young adults between the ages of 18 and 29 years, at the household level. The NLAES obtained a household response rate of 92% and a sample-person response rate of 97%, yielding a total sample size of 42,862. This analysis was restricted to 18,352 individuals defined as current drinkers; that is, respondents who reported having consumed at least 12 alcoholic drinks in the year preceding the interview.

Suicidal ideation

Respondents were asked if during the last 12 months they had (1) thought about suicide and (2) felt like they wanted to die. A positive response to either question defined the suicidal ideation positive group. Because suicidal behaviors constitute one diagnostic criterion for major depressive disorder, diagnoses of major depression were derived in this study by excluding the suicidal behavior criterion.

Alcohol and drug use measures

Three measures of alcohol consumption were used in the analyses. Average daily ethanol intake during the past year was included as a measure of the overall volume of consumption. The proportion of drinking occasions resulting in intoxication during the past year (i.e., the relative frequency of intoxication) was included as an indicator of heavy drinking. The NLAES questionnaire asked about the usual and heaviest amounts of beer, wine and distilled spirits consumed in the preceding year. Frequency, quantity and size of drink for each type of beverage were used to estimate the past year volume of ethanol intake, using ethanol conversion fac-

tors of .045 for beer, .121 for wine and .409 for spirits (Modern Brewery Age, 1992; Distilled Spirits Industry, 1985; Kling, 1989; Turner, 1990; Williams et al., 1993). Average daily intake was derived by dividing the past year volume by 365. Number of drinking days per year was estimated by taking the average of the sum of the beverage-specific frequencies and the largest individual beverage-specific frequency. The absolute frequency with which respondents became intoxicated was asked directly, and the proportion of drinking days resulting in intoxication was derived by dividing the absolute frequency of intoxication by the total number of drinking days. The third consumption measure represented average daily ethanol intake during the respondent's period of heaviest drinking in his or her lifetime. For the multivariate analyses, natural log transformations were applied to both past year consumption measures and a cube root transformation was applied to the heaviest drinking in lifetime measure in order to better satisfy the assumption of linearity required in the models.

Use of any drug on one's own, other than alcohol, on at least one occasion during the past year was measured as a dichotomous predictor variable. Use on one's own was defined as use of opioids (other than heroin), amphetamines, cocaine (and crack cocaine), cannabis (and THC and hashish), heroin, methadone and/or hallucinogens, either: (1) without a prescription, (2) in greater amounts than prescribed, (3) more often than prescribed or (4) for a reason other than prescribed. Any drug use on one's own was also measured for the prior to the past year time frame.

Diagnostic assessment

Diagnoses of DSM-IV alcohol and drug use disorders and major depression were derived from the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS; Grant and Hasin, 1992). The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV criteria for these disorders. Although the DSM-IV was not published until 1994, the specific diagnostic criteria of interest were known prior to the conduct of NLAES (American Psychiatric Association, 1991) and therefore incorporated in their entirety within the AUDADIS.

DSM-IV diagnoses of alcohol and drug dependence were constructed separately for two time frames, past year and prior to the past year. Drug-specific diagnoses of dependence were first derived separately for sedatives, tranquilizers, opioids, amphetamines, cocaine, cannabis, heroin, methadone, hallucinogens and other drugs before being combined into an aggregate measure of drug dependence on any substance. All alcohol and drug use disorder diagnoses satisfy both the clustering and duration criteria of the DSM-IV and specific algorithms underlying these diagnoses have been described in detail elsewhere (Grant, 1995; Grant et al., 1994a).

For the purpose of the analyses presented here, to be classified as positive for past year or prior to the past year drug use, the respondent could not have been classified also with a past year or prior to the past year drug use disorder, respectively. That is, for each time frame, any drug use (with no drug use disorder) and any drug use disorder categories were mutually exclusive.

Episodes of DSM-IV major depressive disorder were also constructed for past year and prior to the past year time frames. Consistent with the DSM-IV, the AUDADIS diagnoses of major depression required the presence of at least five depressive symptoms (inclusive of depressed mood or loss of pleasure or interest) nearly every day, most of the day, for the same 2-week period. Episodes exclusively due to bereavement and physical illness were ruled out. DSM-IV diagnoses of major depression additionally required that social and/or occupational dysfunction be present during the disturbance for a diagnosis of major depression to be positive. As previously noted, diagnoses of major depression did not include the suicidal behavior criterion.

Because suicidal behaviors are among the rarest depression symptoms, removing the suicidal behaviors criterion from consideration in the diagnosis produced only a negligible decrease in prevalence of the diagnosis. For example, the prevalence of past year and prior to the past year depression dropped from 3.3% to 3.1% and from 7.7% to 7.4%, respectively, in this general population sample as the result of removing the suicidal behaviors criterion for the depression diagnosis.

Past year alcohol dependence and major depression diagnoses were combined and entered into the analyses as three separate variables—(1) alcohol dependence with no major depression, (2) major depression with no alcohol dependence and (3) both alcohol dependence and major depression—in order to examine the effect of the comorbidity of these two disorders on the outcome variable, suicidal ideation.

In a separate test-retest study conducted in the general population, reliability coefficients (kappas) associated with alcohol and drug use and alcohol and drug use disorders exceeded 0.73 (Grant et al., 1995). Kappa values for past year and prior to the past year major depression diagnoses were 0.65 and 0.60, respectively.

Sociodemographic and other measures

Based on previous research, several sociodemographic variables were identified as potential predictors of sui-

cidal ideation. Age was measured in single years. Race, marital status and education were dichotomized as black versus nonblack, currently married versus all others, and less than a high school education versus all others, respectively. The past year household income variable, coded as the midpoints of 23 categories, was treated as a continuous variable once it was determined that it satisfied the linearity assumption. Other sociodemographic variables included employed in the past year versus not employed in the past year, urban versus rural residency, and a child or children under 14 years of age living at home. Measures on interpersonal loss included death of one or both parents prior to the age of 17 years, and loss of a close relative or close nonrelative through death during the past year.

Alcohol-related predictors included age at first drink and whether the respondent's current partner or spouse was an alcoholic. Respondents with any biological first- and/or second-degree relative reported to have been an alcoholic or problem drinker constituted the family history positive group examined in this study. An alcoholic or problem drinker was specifically defined for respondents as a person who has physical or emotional problems because of drinking; problems with a spouse, family or friends because of drinking; problems at work because of drinking; problems with the police because of drinking (e.g., drunk driving); or a person who seems to spend a lot of time drinking or being hungover.

Alcohol treatment resources were defined broadly and respondents were asked to indicate separately whether they sought help from 23 different treatment sources, including various inpatient and outpatient facilities and/or services and 12-step groups. A separate series of corresponding questions were asked regarding drug treatment from which the drug treatment variable in the study was constructed. Respondents who had ever sought treatment from a physician for depression, taken prescription drugs for an episode of major depression, or had been hospitalized for an episode of major depression were classified as having ever been treated for a major depression. Respondents were also classified as having a physical illness in the past year if they were hospitalized for: (1) the treatment of a physical condition, accident or injury; (2) an operation or surgical procedure; or (3) diagnostic tests for a physical condition.

Statistical analysis

In the first stage of the analysis, univariate associations between each predictor variable and suicidal ideation were estimated for each sex separately. To protect against Type 1 error due to multiple comparisons, a comparison needed to yield a chi-square test statistic value that could be declared statistically significant at a probability level of <.005. In the second stage of the analysis, multivariate associations were examined by conducting two linear logistic regression analyses, one for each sex, in which suicidal ideation served as the outcome measure. To take into account the NLAES sample design, all standard errors were generated and all statistical analyses were conducted using SUDAAN (Research Triangle Institute, 1997), a software program that uses Taylor series linearization to adjust for sample design characteristics of complex sample surveys.

RESULTS

There were 18,352 current drinkers in this general population sample (9,955 men and 8,397 women). The overall prevalence of suicidal ideation was 6.0% (n = 1,093), with 5.2% (n = 517) of the men and 6.9% (n = 576) of the women reporting ideation during the past year.

The univariate associations between each predictor variable and suicidal ideation estimated for each gender appear in Table 1. The univariate relationships for each sex were remarkably similar. Men and women who experienced suicidal ideation were younger, drank more, had lower incomes, and were significantly less likely to be married and more likely to have used drugs and to have developed a drug use disorder during the past year and prior to that time compared to respondents who did not report suicidal ideation. For each gender, ideators were younger at the time of their first drink and were significantly more likely to have a family history of alcoholism and to have histories of treatment for alcohol, drugs, major depression and a physical illness or injury relative to nonideators. Compared to respondents not experiencing suicidal ideation during the past year, suicidal ideators were significantly more likely to have alcohol use disorders, drug use disorders and major depression both in the past year and prior to the past year time frames. The only unique finding for men was that ideators were significantly less likely than nonideators to have children under 14 years of age living at home.

The results of the final logistic models for suicidal ideation are shown in Table 2 for each gender. Data are only shown for significant variables. Unlike the results of the univariate analyses, the multivariate risk profiles for men and women differed. Unlike women, age and employment status were negatively correlated with ideation among men. That is, employment was protective against ideation and the log odds of ideation

decreased approximately 2% for each 1-year increase in age. The odds of ideation was 1.53 times greater for men having a family history of alcoholism relative to respondents with no such family history. The odds of suicidal ideation were also elevated 59% (AOR = 1.53) among men with an alcohol use disorder occurring prior to the past year compared to men without an alcohol use disorder during that time. Neither of these effects was significant for women. In contrast, the odds for suicidal ideation were 2.62 and 1.95 times greater among women, but not men, who had used drugs nonmedically during the past year and had a drug use disorder during that time, respectively.

The risk for suicidal ideation was elevated approximately twofold among men (AOR = 1.93) and women (AOR = 1.77) who were treated for a physical illness

Table 1. Percentage distribution (mean ± SE) of men and women classified with and without suicidal ideation in the past year: Predictor variables

Variable	Men		Women	
	With suicide ideation	Without suicide ideation	With suicide ideation	Without suicide ideation
%Black	9.7 ± 1.58	8.8 ± 0.44	8.2 ± 1.29	7.6 ± 0.35
Age (years)				
18-29	50.5 ± 2.56	28.9 ± 0.70*	42.2 ± 2.38	29.5 ± 0.75*
30-39	32.6 ± 2.27	37.7 ± 0.63	39.0 ± 2.42	38.0 ± 0.67
40-49	13.0 ± 1.51	24.0 ± 0.60	14.5 ± 1.70	23.5 ± 0.60
≥50	3.9 ± 0.84	9.4 ± 0.34	4.3 ± 0.88	9.0 ± 0.39
% Less than high school	18.4 ± 1.99	13.7 ± 0.47	14.2 ± 1.82	9.7 ± 0.43
% Married	36.3 ± 2.49	65.5 ± 0.68*	42.5 ± 2.36	61.7 ± 0.74*
% Employed in past year	86.8 ± 1.63	88.5 ± 0.38	81.0 ± 1.90	79.6 ± 0.55
% Urban	76.9 ± 2.38	75.7 ± 0.85	82.5 ± 1.99	78.4 ± 0.75
Household income				
≤$20,400	47.9 ± 2.65	28.9 ± 0.65*	48.0 ± 2.40	32.9 ± 0.67*
$20,401-$35,988	22.0 ± 1.99	23.6 ± 0.52	22.9 ± 2.12	24.7 ± 0.53
$35,989-$71,988	19.3 ± 2.02	32.2 ± 0.65	20.7 ± 1.98	27.5 ± 0.59
≥$71,989	10.8 ± 1.61	15.4 ± 0.47	8.4 ± 1.38	15.3 ± 0.44
Average daily ethanol (oz/day) in past year				
≤0.49	44.0 ± 2.51	56.0 ± 0.65*	65.7 ± 2.26	72.9 ± 0.61*
0.50-0.99	17.0 ± 1.98	20.0 ± 0.48	17.5 ± 1.88	15.2 ± 0.46
1.00-1.99	16.7 ± 2.03	14.4 ± 0.43	9.3 ± 1.28	8.7 ± 0.39
≥2.00	22.3 ± 2.11	9.6 ± 0.39	7.5 ± 1.29	3.2 ± 0.24
Proportion of drinking occasions resulting in intoxication in past year				
0.00	27.5 ± 2.20	51.6 ± 0.69*	37.0 ± 2.24	60.6 ± 0.73*
0.01-0.12	5.3 ± 1.15	6.8 ± 0.30	5.1 ± 0.97	5.3 ± 0.31
0.13-0.60	19.5 ± 2.05	17.9 ± 0.45	20.7 ± 1.91	14.3 ± 0.44
>0.60	47.7 ± 2.65	23.7 ± 0.65	37.2 ± 2.31	19.8 ± 0.66
Average daily ethanol (oz/day) heaviest drinking period in lifetime				
≤0.49	21.1 ± 1.97	34.5 ± 0.62*	43.6 ± 2.21	56.8 ± 0.70*
0.50-0.99	16.1 ± 2.06	19.3 ± 0.48	18.1 ± 1.94	18.2 ± 0.51
1.00-1.99	17.1 ± 1.89	18.9 ± 0.49	14.8 ± 1.69	14.1 ± 0.49
≥2.00	45.7 ± 2.62	27.3 ± 0.58	23.5 ± 1.99	10.9 ± 0.44*
% Age at first drink prior to 17 years	47.4 ± 1.69	28.7 ± 0.38	38.6 ± 1.78	21.4 ± 0.38*
% Current spouse/partner alcoholic	1.7 ± 0.59	1.2 ± 0.14	7.3 ± 1.18	4.9 ± 0.28
% Family history of alcoholism	73.8 ± 2.16	52.4 ± 0.65*	77.5 ± 2.02	61.2 ± 0.63*
% Alcohol dependence/no major depression in past year	24.9 ± 2.24	9.4 ± 0.39*	12.6 ± 1.62	6.3 ± 0.35*
% Major depression/no alcohol dependence in past year	17.1 ± 1.77	1.22 ± 0.12*	24.1 ± 2.01	2.1 ± 0.18*
% Alcohol dependence and major depression in past year	12.2 ± 1.75	0.63 ± 0.10*	7.3 ± 1.12	0.5 ± 0.09*
% Alcohol use disorder prior to past year	54.3 ± 2.40	23.8 ± 0.55*	31.9 ± 2.10	15.2 ± 0.53*
% Major depression prior to past year	18.7 ± 2.11	6.2 ± 0.31*	9.7 ± 0.38	18.9 ± 1.82*
% Drug use disorder prior to past year	10.7 ± 1.56	4.4 ± 0.27*	7.5 ± 1.34	3.7 ± 0.24*
% Drug use disorder in past year	15.3 ± 1.67	3.1 ± 0.26*	9.9 ± 1.40	1.6 ± 0.16*
% Drug use in past year	16.1 ± 1.81	6.3 ± 0.28*	14.3 ± 1.72	5.3 ± 0.29*
% Drug use prior to past year	8.1 ± 1.31	10.8 ± 0.37	11.2 ± 1.49	10.0 ± 0.39*
% Ever alcohol treatment	22.8 ± 2.20	7.2 ± 0.31*	10.6 ± 1.35	2.7 ± 0.22*
% Ever drug treatment	9.2 ± 1.49	1.6 ± 0.18*	5.6 ± 1.23	0.9 ± 0.10*
% Ever treatment for major depression	32.3 ± 2.62	6.4 ± 0.27*	45.7 ± 2.33	15.6 ± 0.49*
% Treated for physical illness/injury in the past year	10.8 ± 4.63	5.6 ± 0.26	10.1 ± 1.45	5.9 ± 0.30*
% Child(ren) under 13 years living at home	18.5 ± 2.07	28.5 ± 0.60	33.0 ± 2.17	34.0 ± 0.66
% Death of parent(s) prior to age 17 years	5.6 ± 1.17	6.6 ± 0.27	7.3 ± 1.27	6.8 ± 0.33
% Experiencing death of close relative in past year	24.7 ± 2.27	21.6 ± 0.49	26.8 ± 2.22	22.3 ± 0.55
% Experiencing death of close nonrelative in past year	15.9 ± 1.89	13.7 ± 0.39	17.1 ± 1.72	16.0 ± 0.48

*χ^2 $p < .005$.

or injury during the past year, and approximately threefold among men (AOR = 3.49) and women (AOR = 3.10) who had lifetime treatment histories of depression. Suicidal ideation was also less likely among respondents of both sexes who were married compared to those who were not. Another similarity among the sexes was the increased risk of suicidal ideation among respondents with past year alcohol dependence, a past year depression, and those comorbid for both of these disorders in the past year. The magnitude of the risk for ideation associated with each of these two disorders and their combination was similar between the sexes. Among men and women, major depression without alcohol dependence was the strongest predictor (AOR = 13.58 and 10.91, respectively) and alcohol dependence without major depression was the weakest predictor (AOR = 2.20 and 1.59, respectively), while the risk for ideation did not increase among those with comorbid alcohol dependence and major depression (AOR 9.87 and 9.04, respectively) over and beyond the risk associated with major depression without alcohol dependence.

DISCUSSION

The data indicated that suicidal ideators could be identified by predictors that have been found to characterize alcoholic and nonalcoholic suicide attempters and completers. The strongest predictor of ideation for both men and women was the presence of major depression during the past year. Although the presence of alcohol dependence without major depression elevated the risk of ideation, the magnitude of that risk was approximately twofold compared to the approximate twelvefold risk associated with major depression in the absence of alcohol dependence. Interestingly, the approximate tenfold risk of ideation associated with comorbid major depression and alcohol dependence was not increased above the risk associated with major depression in the absence of alcohol dependence. These results, in combination, suggest that major depression and alcohol dependence are independent risk factors for suicidal ideation, with major depression having the more sizable impact. The risk for suicidal ideation was also greater for respondents who had re-

Table 2. Results of final logistic refression models for suicidal ideation for each gender

	Men					Women				
Variable	Beta	SE (Beta)	*p*	AOR[a]	95% CI	Beta	SE (Beta)	*p*	AOR[a]	95% CI
Intercept										
Ethnicity (effect of black)										
Age	−0.02	0.01	<.0101	0.98	0.97-0.99					
Education (effect of less than high school)										
Marital status (effect of married)	−0.52	0.17	<.0027	0.59	0.42-0.83	−0.33	0.12	.0089	0.71	0.56-0.92
Urbanicity (effect of urban)										
Employment (effect of employed in last year)	−0.70	0.21	.0016	0.49	0.32-0.76					
Household income										
Average daily ethanol intake in past year										
Proportion of drinking occasions resulting in intoxication										
Average daily ethanol during period of heaviest drinking										
Age at first drink										
Current spouse/partner alcoholic										
Family history of alcoholism	0.42	0.14	.0032	1.53	1.16-2.00					
Major depression/no alcohol dependence in past year	2.61	0.20	<.0000	13.58	9.10-20.20	2.39	0.18	<.0000	10.91	7.63-15.61
Major depression and alcohol dependence in past year	2.29	0.30	<.0000	9.87	5.41-17.99	2.20	0.31	<.0000	9.04	4.86-16.82
Alcohol dependence/no major depression in past year	0.79	0.18	<.0000	2.20	1.53-3.16	0.46	0.22	.0100	1.59	1.02-2.47
Alcohol use disorder prior to past year	0.44	0.16	<.0078	1.59	1.13-2.13					
Major depression prior to past year										
Drug use disorder prior to past year										
Drug use disorder in past year						0.96	0.29	.0014	2.62	1.47-4.68
Drug use in the past year						0.66	0.20	.0017	1.95	1.29-2.92
Drug use prior to past year										
Ever alcohol treatment										
Ever drug treatment										
Ever treatment for major depression	1.25	0.17	<.0000	3.49	2.49-4.92	1.13	0.13	.0000	3.10	2.36-4.08
Treated for physical illness/injury in past year	0.66	0.24	.0079	1.93	1.19-3.11	0.57	0.23	.0090	1.77	1.14-2.77
Child(ren) under 13 years living at home										
Death of parent(s) prior to age 17 years										
Death of close relative in past year										
Death of close nonrelative in past year										

Note: Goodness of fit for overall model: men: Satterthwaite's $F = 72$, 24/68 df, $p < .0000$; women: $F = 73$, 28/68 df, $p < .0000$.
[a]AOR = adjusted odds ratio.

ceived treatment for a major depressive episode, but there was no increase in risk for those who had prior to the past year episodes; results indicating that it is the severity, and not the chronicity, of major depression that may play a more important role in producing ideation.

Marriage was one factor identified among men and women that heightened the barrier to suicidal ideation. The protective effect of marriage relative to being divorced, separated, widowed or never married points to the importance of marriage in social integration for men and women. Respondents who are married and more socially integrated are also more strongly controlled by their environment, and their risk *for* ideation may be reduced because there is more to prevent it. This explanation could be considered a variant of Durkheim's (1966) concept of anomy.

The unique risk factors characterizing men and women suggest that gender roles within our society may affect men's and women's engagement in suicidal behaviors differently. Suicidal ideation was increased among men with a past alcohol use disorder, indicating that the chronicity of this disorder may contribute to ideation. In contrast, risk was elevated among women who had used drugs nonmedically and who had developed an associated drug use disorder during the past year. Jack (1992) has argued that the female role allows for a helpless attrition style that renders women indecisive, less able to cope with adversity and more likely to attempt to solve their problems with psychotropic drugs. Since this strategy often proves inadequate, helplessness may lead to hopelessness and, subsequently, to suicidal ideation. Hopelessness has been shown to be highly predictive of suicidal ideation (Beck et al., 1993). Moreover, the availability and use of drugs can serve as a vehicle for subsequent self-poisoning and overdosing. In contrast, the male role does not allow for a helpless lifestyle, but it does permit heavy alcohol consumption to a larger degree than does the female role. Females, as a socially subordinate subgroup of the population, are more heavily sanctioned for their drinking behavior than are their male counterparts (Mäkelä 1987; Park, 1983).

That a more chronic alcohol use disorder plays an important role in suicidal ideation among men might explain why the risk for ideation among men, but not women, was increased by having a family history of alcoholism and decreased by being employed. Family history of alcoholism has often been associated with the more severe problem course of alcohol use disorders and it seems evident that alcohol use disorders can have quite destructive effects on family and work relationships.

Unemployment among males and a recent physical illness among men and women increased the risk for suicidal ideation. Recent physical illness, unemployment and associated financial problems, representing life crises and stress, may increase one's propensity toward suicidal ideation. However, while these traumatic events are likely to increase stress, continual marital social bonds may serve to protect against suicidal ideation, as observed in this study.

The risk for suicidal ideation among men decreased slightly with age, a finding inconsistent, with few exceptions (Hesselbrock et al., 1988), with the existing clinical literature (Roy, 1993). It should be noted, however, that the risk for ideation attributable to age among men was very small, representing an approximate 2% decrease in risk for each 1-year increase in age. The reduction in risk associated with increased age may also represent the impact of ideators who have gone on to complete suicide. The age effect found in this study might also have served as a proxy for physical illness that grows more prevalent with age.

One of the most surprising findings in this study is that none of the three consumption variables was significantly associated with suicidal ideation. This finding was inconsistent with research conducted in treated and untreated populations on suicide attempts and completions in which recent heavy drinking was identified as a major risk factor for these suicidal behaviors (Garrison et al., 1993; Murphy et al., 1992). However, in most studies conducted in untreated populations (e.g., among adolescents), alcohol consumption was measured, but alcohol use disorders were not. Moreover, in studies of suicidal behaviors among alcoholics, only consumption could be measured. It is possible that alcohol consumption relates to suicidal ideation and other suicidal behaviors via intoxication; that is, as a precipitant proximal to suicide attempts and completions. If this were the case we should not have expected the two measures of average daily consumption to relate to suicidal ideation nor to the mechanism underlying intoxication. On the other hand, it is difficult to understand why no elevation in risk for suicidal ideation was observed among men and women as a function of increasing drinking occasions resulting in intoxication, as represented by the third consumption variable examined in this study. In general, these results suggest that it may not necessarily be the psychopathology of alcoholism that impacts on suicidal ideation, but rather it may be that alcohol dependent persons in this sample dominated high frequency levels of intoxication, thus obscuring the alcohol intake and ideation relationship. Alternatively,

measures of consumption used in this study might have failed to identify different types of drinking or different groups of drinkers that might relate more strongly to suicidal ideation (e.g., other measures of patterns of drinking). On a similar note, it is also possible that our measures of ideation could have been more detailed. For example, the one aspect of our definition of ideation, operationalized as thoughts about suicide, could have included general thoughts about suicide as well as thoughts about one's own suicide.

To date, it has proved extremely difficult to unravel the relationships among the suicidal behaviors of ideation, attempts and completions. Until such time as these relationships are fully elucidated, there is no basis for determining which ideators will go on to attempt or complete suicide and which ideators will not. However, what is certain is that suicide attempters and completers do constitute a subgroup of those individuals who experience suicidal ideation. In view of the state of the art, the results of this study strongly suggest that the most important single intervention that will reduce suicides overall is the recognition and vigorous treatment of major depression and, to a lesser, but not insignificant degree, of alcohol and drug use disorders, among persons experiencing suicidal ideation. Recognition and treatment of major depression would be particularly important in persons with certain other risk factors, including unemployment, being unmarried and having recent treatment for a somatic illness. Equally important is the management and alleviation of suicidal ideation that constitutes a pathological state of mind in its own right. In view of the results of this study that point to the importance of external stressors on suicidal ideation, intervention models such as stress management, social skills training and relapse prevention should supplement traditional clinical paradigms focusing on pharmacological and psychodynamic intervention among persons experiencing suicidal ideation.

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Alcohol, Drugs, Fighting and Suicide Attempt/Ideation

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In a representative U.S. sample of 18,352 current drinkers 18 years of age and over, past-year alcohol- or other drug-related fighting and suicide attempt/ideation both showed strong positive bivariate associations with volume of alcohol intake, proportion of drinking days resulting in intoxication (the intoxication index) and past-year drug use, especially multiple drug use. After adjusting for potential confounders in a series of multiple logistic regression models, average daily ethanol intake retained a significant positive association with the odds of alcohol- and other drug-related fighting, as did the intoxication index—except among drinkers who used marijuana only (i.e., no other drugs). The odds of this outcome also were increased by use of stimulants or cocaine only, use of multiple drugs and use of marijuana—the latter primarily among women. The odds of past-year suicide attempt/ideation were positively associated with the intoxication index but were not significantly associated with average daily ethanol intake. Suicide attempt/ideation also was positively associated with the use of marijuana only, sedatives/tranquilizers only, cocaine/stimulants only and multiple drugs. Because drug use was positively associated with alcohol use, models restricted to only alcohol or only drug use measures overestimated some of their associations with the two outcome measures. Simultaneous use of alcohol and drugs was not significantly associated with the odds of either of the outcomes considered in this analysis, but the data were suggestive of a positive effect of simultaneous use on alcohol- and drug-related fighting.

INTRODUCTION

There is an extensive literature linking alcohol and drug use to aggression and violence. Animal studies generally have found that defensive, offensive and maternal aggression increased in response to low acute doses of ethanol and decreased following doses heavy enough to produce a sedative effect; however, some studies found that the dose-response effect was dependent upon the underlying level of aggression or upon the presence of circulating androgens (see reviews in Archer, 1988; Miczek and Thompson, 1983; and Wagner *et al.,* 1993). Animal studies involving other drugs generally have found that opiates and THC (tetrahydrocannabinol, the major psychoactive ingredient in marijuana) suppressed or decreased aggressive behavior, that amphetamines may have increased aggressive or defensive behaviors at low acute doses but tended to decrease attack frequencies at higher doses and resulted in social withdrawal and victimization when chronically administered, and that both benzodiazapenes and alprazolam increased aggression at low acute doses as well as augmenting the proaggressive effects of alcohol (Bond and Silveira, 1993; Miczek and Thompson, 1983; Miczek and O'Donnell, 1980; Miczek *et al.,* 1993a).

Human studies employing a variety of experimental paradigms have yielded inconsistent results, with most but not all studies showing an alcohol-induced increase in aggressive behavior (as indicated by willingness to administer shocks of varying intensities). Findings have indicated that the main factor contributing to a positive effect of alcohol on aggression was the presence of provocation, threat or frustration; alcohol was least likely to be associated with aggression in situations where a nonaggressive alternative was offered, where opponents explicitly indicated lack of threat, or where subjects heard behavioral norms described or had a chance to reflect on the consequences of their actions (see reviews in Graham *et al.,* 1995; Gustafson, 1993; Gustafson, 1994; and Taylor and Chermack, 1993). Gustafson (1993) has argued that these findings contradict hypotheses that attempt to explain the proagressive qualities of alcohol in terms of its disinhibition, arousal or expectancy effects; rather, they are more supportive of the attentional hypothesis that the relative salience of instigatory or inhibitory cues determine whether or not alcohol increases aggressive behavior.

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Few human experimental studies have examined substances other than alcohol, but a series of studies conducted at Kent State University (reviewed in Taylor and Chermack, 1993) did find that high-dose THC reduced aggression relative to low-dose THC, that amphetamines had no effect on aggression, and that diazepam had a proaggressive effect that did not extend to all benzodiazapenes nor to secobarbital or pentobarbital.

Much of the strength of the association between alcohol and/or drug use and violence has been inferred from studies of criminal offenses or incarcerated populations (see reviews in Miczek *et al.,* 1993b; Valdez *et al.,* 1995; Walfish and Blount, 1989). Miczek and colleagues (1993b) have argued that alcohol is more often associated with violent and aggressive behavior than all other drugs combined, and several studies of criminal offenders support this. For example, Flanagan and Jamieson (1988) found that of state prison inmates incarcerated for violent crimes, 13 percent had used drugs only prior to committing the crime, as compared to 20 percent who had used alcohol only and 20 percent who had used both alcohol and drugs. Likewise, a study of female Florida state prison inmates found that approximately 60 percent of those under the influence of alcohol at the time of the offense for which they were incarcerated had committed violent crimes, as opposed to 33 percent of those who were under the influence of drugs (Blount *et al.,* 1988). In a sample of inmates incarcerated in New York state correctional facilities for homicides committed in 1984, 32 percent reported having been intoxicated at the time of the homicide, compared to 18 percent and 14 percent, respectively, who reported having been high on marijuana and cocaine. The proportion of inmates who attributed their offense to having been under the influence of a psychoactive substance also was higher for those who reportedly were under the influence of alcohol than for those under the influence of any other drug (Spunt *et al.,* 1994; Spunt *et al.,* 1995). In a study of male jail detainees, Teplin (1994) found that those with alcohol abuse or dependence were comparable to those with no disorder in terms of the violence and seriousness (felony vs. misdemeanor) of their offense, whereas those with drug abuse or dependence were less likely than those with no disorder to have committed a violent offense.

Studies based on drug and alcohol treatment samples and school-based samples of adolescents also have supported a link between substance use—especially polysubstance use—and violence. For example, Schuckit and Morrissey (1978) found that of adolescents referred by the court system for alcohol counseling, those who had used phencyclidine (PCP) alone or with propoxyphene had a greater history of fights, arrests and weapon use than those who had used neither drug. In a sample of inpatient alcoholics, Schuckit and Bogard (1986) found that those who were primary drug users with secondary alcoholism or who were primary alcoholics with a history of intravenous drug use were more likely to have hurt peers or used weapons than those primary alcoholics who had never used intravenous drugs. McCormick and Smith (1995) reported that of 3,367 substance abusers seeking treatment, polysubstance users had higher measures of hostility and aggression than did single drug users, regardless of whether the polysubstance use included cocaine. In a study of adolescents in the general Canadian population, Boyle and Offord (1991) determined that the odds of both conduct and affective disorders were positively associated with regular alcohol use and hard drug use, with the latter showing the stronger association. Marijuana use increased the odds of conduct disorders but not affective disorders. Valois and colleagues (1995), studying a sample of South Carolina high school students, found that binge drinking was predictive of fighting for males but not females and that illegal drug use was predictive of fighting for white but not black students, with the effect of drug use stronger among white females than white males.

Suicide is arguably a special class of violence, the result of aggression resulting in violence against oneself rather than against another individual. The factors that potentiate in favor of suicide as opposed to other-directed violence are quite distinct, with depression more strongly associated with the former and impulsivity more strongly associated with the latter (Plutchik and Van Praag, 1994). Like other acts of violence, though, suicide, suicide attempt, and suicidal ideation have been strongly linked with alcohol and drug use. For example, Kinkel and colleagues (1989) found that past-year suicide attempts in a youth sample were significantly associated with use of alcohol and various drugs, with the highest odds ratios for drugs other than marijuana and a significant effect of marijuana use only for females. Garrison *et al.* (1993), using a sample of high school students, determined that the odds of a suicide attempt were increased by all classes of drug use as well as by frequency of drinking, binge drinking, and a measure of behavioral aggression. All of these factors except marijuana and cocaine use also increased the odds of suicidal ideation.

Despite the numerous studies that have examined associations between alcohol and drug use and violence, our ability to extend these relationships to the

general population is quite limited. The comorbid occurrence of alcohol use, drug use and other psychiatric disorders has been established in several population-based studies (Grant, 1995; Grant and Harford, 1995; Grant and Pickering, 1996; Lavelle *et al.,* 1993; MacDonald *et al.,* 1992) and can be inferred from comparing the prevalences of these disorders in the general population with their prevalences in incarcerated or treatment samples (Teplin *et al.,* 1996). Because individuals with one disorder are at increased risk of a second disorder, samples selected on the basis of psychiatric disorders or where such disorders are overrepresented will almost certainly overstate the associations among disorders (Berkson, 1946) and are likely to overstate the associations between manifestations or indicators of these disorders, e.g., between alcohol or other drug use and acts of violence.

Because most of the population or community samples that have been used to study the association between substance use and violence have been restricted to adolescents, there is little information on the magnitude of this association within the adult population. This study attempts to remedy that lack, using data from a national sample of U.S. adults 18 years of age and over to examine the individual and conjoint associations of alcohol and other drug use with alcohol- or other drug-related fighting and suicide attempt or ideation after adjusting for the effects of demographic and socioeconomic characteristics. It also examines the extent to which the correlation between alcohol and other drug use may lead to overestimation of their individual effects in the absence of information on both types of substance.

METHODS

Sample

This analysis is based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism. Data were collected in personal interviews conducted in respondents' homes by interviewers trained and employed by the U.S. Bureau of the Census. The NLAES sample consisted of U.S. adults 18 years of age or older who were selected at random from a nationally representative sample of households. The multistage sampling design featured selection of primary sampling units with probability proportional to size, oversampling of segments (secondary sampling units) with high proportions of black residents and oversampling of young adults between the ages of 18 and 29 (among whom heavy consumption and alcohol problems are disproportionately represented) at the household level. The NLAES obtained a household response rate of 92 percent and a sample-person response rate of 97 percent, yielding a total sample size of 42,862. This analysis was restricted to 18,352 individuals defined as current drinkers, that is, who reported having consumed at least 12 alcoholic drinks in the year preceding interview.

Measures

Two measures of alcohol consumption were used in this analysis. Average daily ethanol intake was included as a measure of the overall volume of consumption, and the intoxication index (that is, the proportion of drinking occasions resulting in intoxication) was included as an indicator of a heavy or binge drinking pattern. The NLAES questionnaire asked about the usual and heaviest amounts of beer, wine and liquor consumed in the preceding year. Frequency, quantity and size of drink for each type of beverage were used to estimate the past-year volume of ethanol intake, assuming ethanol conversion factors of .045 for beer, .121 for wine and .409 for liquor (DISCUS, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams *et al.,* 1993). Average daily intake was derived by dividing the past-year volume by 365. All frequency questions utilized response categories (e.g., "once or twice a month," "three or four times a week") that were converted to days per year based on the midpoint of the category. Number of drinking days per year was estimated by taking the average of the sum of the beverage-specific frequencies (with the result not allowed to exceed 365) and the largest individual beverage-specific frequency. The absolute frequency with which respondents became intoxicated was asked directly, and the intoxication index was derived by dividing the absolute frequency of intoxication by the total number of drinking days. Total body water, which affects the blood alcohol level resulting from a given level of ethanol intake (Goist and Sutker, 1985), was estimated on the basis of each respondent's age, gender, height and weight (Moore *et al.,* 1963).

The drug use variables were taken from a series of questions that began by asking whether respondents had ever used any of the following types of drugs or medicines at least 12 times *on their own* (defined as either without a doctor's prescription or for longer or in greater amounts than was prescribed): sedatives, tranquilizers, painkillers, stimulants, marijuana (including hashish and THC), cocaine (including crack cocaine), heroin, methadone, and other drugs (e.g., hallucino-

gens, inhalants and antipsychotics or antidepressants). Respondents were handed flashcards that gave examples of individual drugs within each broad category, and drugs erroneously reported as "other" drugs were moved into their correct categories during data cleaning. Past-year drug users comprised that subset of ever users who reported that they had used drugs on their own during the year preceding the NLAES interview. For each type of drug that past-year drug users reported ever having used, they were asked whether and how often the drug had been used in the past year and how often it was used simultaneously with alcohol ("at the same time or within a couple hours of drinking beer, wine or liquor").

Alcohol- and other drug-related fighting during the past year was ascertained by positive responses to both of the following questions: "In your entire life, did you EVER get into a physical fight while drinking or right after drinking?" and "Did this happen in the last 12 months?" or by positive responses to both of the following questions: "In your entire life, did you EVER get into a physical fight while under the influence of a medicine or drug?" and "Did this happen in the last 12 months?" Past-year suicide attempt/ideation was ascertained by means of a positive response to either of the following two questions: "At ANY time in the last 12 months, did you attempt suicide?" or "At ANY time in the past 12 months, did you think about suicide from time to time?"

Individuals were defined as having a positive family history of alcoholism if they reported that any of 18 different types of first- and second-degree biological relatives had ever been alcoholics or problem drinkers. An alcoholic or problem drinker was defined as "a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hungover."

Analysis

First, the percentage distributions of current drinkers within categories of average daily ethanol intake, proportion of drinking occasions resulting in intoxication and type of drug use were estimated, and the prevalences of alcohol- and drug-related fighting and suicide attempt/ideation were compared across these categories. On the basis of the literature demonstrating increased risks of violence among users of certain types of drugs, the following six categories of past-year drug use were distinguished: 1) no drug use, 2) use of painkillers only, 3) use of marijuana only, 4) use of sedatives or tranquilizers only, 5) use of cocaine or stimulants only, and 6) use of multiple drugs. Stimulants were combined with cocaine and sedatives were combined with tranquilizers because of their similar modes of action and because there were too few cases to isolate each type of drug. Individuals who used a single drug not included in this categorical scheme (e.g., heroin, methadone, hallucinogens, etc.) were excluded from the drug-specific analysis because of their small numbers (most individuals using these drugs were multiple drug users) and the inadvisability of combining such pharmacologically diverse drugs. These individuals were retained in comparisons of drug vs. non-drug users and single- vs. multiple-drug users.

Second, individuals in the six drug use categories were compared in terms of background and alcohol use characteristics to ascertain whether their estimated effects might be confounded by their associations with these other factors. Third, a pair of multiple logistic regression models was constructed to estimate the odds of alcohol- and other drug-related fighting and suicide attempt/ideation among current drinkers and to isolate the associations of the alcohol and drug use variables with the odds of these outcomes. In addition to testing for the main effects of alcohol intake and drug use, the models also tested for all possible two-way interactions between the two alcohol use measures, the five dummy variables representing drug use and male gender. Gender was included as a potential effect modifier because of the literature linking the proaggressive effects of alcohol to the presence of testosterone (DeBold and Miczek, 1985; Winslow and Miczek, 1988) and findings of gender differences in the effects of alcohol and some drugs (Boyle and Offord, 1991; Kinkel *et al.,* 1989; Lavelle *et al.,* 1993; Valois *et al.,* 1995). In these and subsequent multiple logistic regression models, a cube-root transform was applied to the intoxication index to better satisfy the assumption of linearity with the log odds of the outcome measures.

Next, these models were refitted omitting first the drug use and then the alcohol use variables, in order to see how these omissions would affect the remaining parameters. Finally, a separate set of models was constructed to estimate the odds of past-year alcohol-related and other drug-related fighting and suicide attempt/ideation among current drinkers who used one or more types of drugs, in order to determine whether the simultaneous use of alcohol and drugs was associated with these outcomes independent of the number of drugs used. This contrast could not be tested in the initial models because some of the drug use categories were too small to split into subgroups of individuals who did and did not use the drug simultaneously with alcohol.

RESULTS

Approximately one sixth of all current drinkers, 17.2 percent, had an average daily ethanol intake level that was the equivalent of less than 1 drink per week (Table I). Nearly half drank the equivalent of 1–6 drinks per week, and more than one third drank the equivalent of at least 1 drink per day. The proportion of drinkers who reported alcohol-related or other drug-related fighting increased sharply with average daily ethanol intake, from 0.1 percent of those who drank less than 1 drink per week to 11.9 percent of those who drank the equivalent of 4 or more drinks per day. The prevalence of suicide attempt/ideation rose less sharply across these categories of intake, from 2.5 percent to 8.7 percent.

More than half of all current drinkers, 53.8 percent, did not report any episodes of intoxication during the past year, and an additional 28.6 percent reported that some but less than 10 percent of their past-year drinking days resulted in intoxication. Fewer than 10 percent of current drinkers reported becoming intoxicated on 25 percent or more of their drinking days, and fewer then 5 percent reported becoming intoxicated at least half of the time, that is, on 50 percent or more of their drinking days. The prevalence of alcohol- or other drug-related fighting rose from 0.4 percent of drinkers who never became intoxicated to 16.3 percent of those who became intoxicated on half or more of their drinking days, and the prevalence of suicide attempt/ideation rose from 2.1 percent to 13.9 percent across these same categories.

Considering all types of medicines and drugs, less than one tenth of all current drinkers (9.1 percent) admitted to any type of drug use; 90.9 percent reported

Table I. Number and percentage of current drinkers 18 years of age and over and prevalence of past-year alcohol- or other drug-related fighting and suicide attempt/ideation, by level of ethanol intake and intoxication and type of illicit drug use

	N		*Prevalence of past-year:*	
		Percentage of current drinkers	*Alcohol- or other drug-related fighting*	*Suicide attempt/ ideation*
All current drinkers[a]	18,352	100.0 (0.0)	2.7 (0.2)	3.9 (0.2)
Average daily ethanol intake:				
<0.075 oz. (<1 drink/week)	3,201	17.2 (0.3)	0.1 (0.05)	2.5 (0.3)
0.075–0.24 oz. (1–3 drinks/week)	4,852	26.9 (0.4)	0.6 (0.08)	3.7 (0.3)
0.25–0.49 oz. (4–6 drinks/week)	3,251	18.1 (0.3)	1.9 (0.4)	3.6 (0.4)
0.50–0.99 oz. (1–2 drinks/day)	3,247	18.0 (0.3)	3.1 (0.4)	3.6 (0.4)
1.00–1.99 oz. (2–3 drinks/day)	2,182	12.2 (0.3)	5.3 (0.5)	4.4 (0.5)
2.00 oz. or more (4+ drinks/day)	1,281	7.6 (0.2)	11.9 (1.2)	8.7 (0.9)
Intoxication index (proportion of drinking occasions resulting in intoxication):				
.000	10,022	53.8 (0.5)	0.4 (0.08)	2.1 (0.2)
.001–.100	5,113	28.6 (0.4)	3.0 (0.3)	4.3 (0.3)
.100–.249	1,503	9.0 (0.3)	6.0 (0.8)	6.5 (0.7)
.250–.499	720	4.2 (0.2)	9.0 (1.0)	8.9 (1.2)
.500 or more	718	4.3 (0.2)	16.3 (1.7)	13.9 (1.4)
Type of illicit drug use:				
No drug use	16,709	90.9 (0.3)	1.7 (0.1)	3.0 (0.1)
Any drug use[b]	1,643	9.1 (0.3)	12.1 (1.0)	13.7 (0.9)
Single drug only[c]	1,185	6.7 (0.2)	8.9 (1.1)	9.9 (0.9)
Painkillers only	63	0.3 (0.05)	4.7 (3.0)	8.5 (3.5)
Marijuana only	978	5.6 (0.2)	8.9 (1.2)	8.7 (1.0)
Sedatives/tranquilizers only	68	0.4 (0.05)	5.3 (3.1)	17.9 (6.1)
Cocaine/stimulants only	64	0.4 (0.05)	18.7 (6.3)	20.2 (5.1)
Multiple drugs	458	2.5 (0.1)	21.1 (2.2)	24.0 (2.2)

Note: Figures in parentheses are standard errors of percentages.
[a]Including individuals unknown as to alcohol intake or type of drug use.
[b]Including sedatives (0.4%), tranquilizers (0.9%), painkillers (1.0%), stimulants (0.8%), marijuana/hashish/THC (7.8 %), cocain/crack (1.2 %), heroin (0.1%), methadone (<0.1%) and other drugs such as hallucinogens, inhalants, antipsychotics and so forth (0.7%).
[c]Including individuals who used a single type of drug other than those presented in this table, e.g. heroin, methadone, hallucinogens, inhalants, etc.

not using any drugs. The prevalence of individual drug use varied from 7.8 percent for marijuana (including hashish and THC) to 0.1 percent for heroin and methadone. Among current drinkers, 6.7 percent used a single drug only—a painkiller (0.3 percent), marijuana (5.6 percent), a sedative or tranquilizer (0.4 percent), or a stimulant or cocaine (0.4 percent). Another 2.5 percent, more than one quarter of all drug users, used more than one type of drug. The prevalence of alcohol- or other drug-related fighting was seven times as high among drug users as among non-drug users (12.1 percent vs. 1.7 percent) and was more than twice as high among multiple-drug users as among single-drug users (21.1 percent vs. 8.9 percent). Among single-drug users, though, those who used a stimulant or cocaine were about as likely to have engaged in alcohol- or other drug-related fighting (18.7 percent) as were multiple drug users. Drug use was associated with the risk of suicide as well, with a more than fourfold increase in suicide attempt/ideation among drug users relative to non-drug users (13.7 percent vs. 3.0 percent) and a more than twofold increase among multiple- as opposed to single-drug users (24.0 percent vs. 9.9 percent).

As shown in Table II, drinkers within the different categories of drug use differed in terms of background and alcohol-related characteristics. Drug users were younger, more likely to be male and to be employed, less likely to be married and to have attended college, and had lower family incomes than drinkers who had not used drugs in the past year. In addition, drug users were far more likely to have a positive family history of alcoholism, started drinking at younger ages, had a heavier volume of alcohol intake and had larger intoxication indices than non-drug users.

Single- and multiple-drug users did not differ in terms of most demographic characteristics (except that the former were more likely to have attended college and had higher family incomes), but they did differ in terms of their alcohol histories and consumption levels. Compared to single-drug users, those who used multiple drugs were more likely to have a positive family history of alcoholism, started drinking at a slightly earlier age, and had higher levels of average alcohol intake and intoxication. They also were more likely to have used drugs simultaneously with alcohol and first used drugs at an earlier age. Although most differences between individual types of drug use were not statistically significant, individuals who used only a stimulant or cocaine did have consumption levels more suggestive of binge drinking than did users of other drugs, and individuals

Table II. Selected characteristics of current drinkers 18 years of age and over, by type of past-year illicit drug use

	No drug use	*Used any drug*	*Used single drug only*	*Used painkillers only*	*Used marijuana only*	*Used sedatives/ tranquilizers only*	*Used cocaine/ stimulants only*	*Used multiple drugs*
Sociodemographic characteristics:								
Mean age	40.9 (0.2)	29.2 (0.3)	29.6 (0.3)	34.6 (1.6)	28.7 (0.4)	38.4 (1.7)	29.0 (1.2)	28.1 (0.4)
% male	59.7 (0.5)	66.5 (1.3)	66.9 (1.5)	52.3 (6.7)	69.2 (1.5)	38.8 (6.5)	72.2 (6.1)	65.2 (2.5)
% Black	8.3 (0.3)	9.5 (0.9)	9.6 (1.0)	8.2 (3.2)	9.6 (1.1)	3.3 (1.9)	18.1 (5.5)	9.4 (1.7)
% Hispanic	6.9 (0.3)	5.3 (0.6)	4.7 (0.7)	6.6 (3.5)	4.7 (0.8)	2.2 (1.6)	2.3 (1.8)	6.7 (1.2)
% married	64.9 (0.6)	39.8 (1.5)	40.5 (1.8)	57.1 (6.5)	37.8 (2.0)	62.4 (6.4)	50.4 (8.8)	37.9 (2.8)
% employed	84.1 (0.4)	93.1 (0.6)	93.3 (6.2)	88.7 (3.6)	94.1 (0.8)	87.6 (4.3)	92.6 (3.0)	92.6 (1.2)
% attended college	59.4 (0.6)	53.6 (1.4)	57.7 (1.7)	64.8 (6.7)	58.8 (1.8)	50.8 (6.9)	41.6 (8.0)	42.8 (2.6)
% urban	75.0 (0.7)	78.1 (1.4)	77.1 (1.8)	79.3 (5.5)	77.3 (2.1)	76.8 (6.6)	71.7 (9.5)	80.7 (2.3)
Mean family income ($000s)	49.3 (0.7)	35.0 (1.6)	37.1 (1.9)	48.5 (8.0)	37.1 (2.2)	36.8 (4.3)	29.5 (3.3)	29.4 (2.1)
Alcohol measures:								
% with FHP[a]	55.3 (0.5)	73.3 (1.3)	70.4 (1.6)	64.1 (6.8)	70.4 (1.8)	83.5 (5.0)	62.6 (7.1)	81.2 (2.1)
Mean age at 1st drink	18.6 (0.03)	16.6 (0.07)	16.8 (0.09)	17.9 (0.5)	16.7 (0.09)	17.7 (0.8)	16.4 (0.4)	16.0 (0.1)
Mean dally ethanol intake	0.6 (0.01)	1.5 (0.06)	1.3 (0.07)	0.8 (0.1)	1.3 (0.07)	0.8 (0.2)	2.1 (0.7)	2.1 (0.2)
Mean percentage of drinking occasions resulting in intoxication[b]	6.3 (0.2)	18.1 (0.8)	16.2 (0.9)	9.0 (2.4)	16.3 (1.0)	10.9 (2.5)	24.6 (4.9)	23.3 (1.5)
Drug use measures:								
% who used drugs simultaneously with alcohol	NA	83.7 (1.1)	80.9 (1.3)	46.3 (6.8)	87.2 (1.3)	42.4 (6.9)	66.5 (6.8)	91.1 (1.6)
Mean age at first drug use	NA	17.2 (0.2)	17.7 (0.2)	22.6 (1.5)	16.9 (0.2)	26.1 (1.9)	17.5 (0.8)	15.6 (0.2)
Mean number of drugs used	NA	1.4 (0.03)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	2.6 (0.06)

Note: Figures in parentheses are standard errors of estimates.
[a]Positive family history of alcoholism
[b]Intoxication index × 100

who used only painkillers, sedatives or tranquilizers began drug use at an older age than did those who used only 'street drugs' such as marijuana or cocaine.

The patterns of association demonstrated by Table II underscore the need for using multivariate techniques to isolate the independent associations of the sociodemographic, alcohol and drug measures with the risks of alcohol-related and other drug-related fighting and of suicide attempt/ideation. Table III shows the two final logistic regression models predicting the odds of these outcomes, excluding any nonsignificant interaction terms but retaining all main effects. It also shows the parameters estimated for the alcohol use variables when the drug use variables were excluded from the models and the parameters estimated for the drug use variables when the alcohol use measures were excluded. Table IV presents the odds ratios associated with the alcohol and drug use variables on the basis of the models that included both.

The associations between alcohol- and other drug-related fighting and the sociodemographic variables were in keeping with findings of prior studies that have examined this problem domain (Dawson *et al.,* 1995; Dawson, in press). The odds of fighting were inversely related to age and income, were positively related to male gender and were negatively associated with being married, being employed and having attended college. Most of the alcohol and drug use measures retained a significant association with the odds of past-year alcohol- or other drug-related fighting after adjusting for these sociodemographic measures. The odds of such fighting were almost doubled by a positive family history of alcoholism (OR = $e^{0.640}$ = 1.90) and were higher among individuals who started drinking at earlier ages. Using an average daily intake level of 0.015 ounces (approximately one drink per month) as a reference level, the odds of alcohol- or other drug-related fighting rose from 1.01 at an intake level of .075 ounces per day (approximately 1 drink per week) to 1.67 at an intake level of 2.50 ounces (about 5 drinks per day). For the majority of current drinkers, the intoxication index was positively associated with the odds of alcohol- or other drug-related fighting, with odds ratios increasing from 2.40 among those who became intoxicated on 10 percent of their drinking occasions to 4.46 among those who became intoxicated half of the time when they drank. For one subgroup of current drinkers, those who used marijuana but no other drug during the past year, the association between fighting and the intoxication index was not statistically significant.

Use of painkillers only was not associated with a significant increase in the odds of alcohol- or other drug-related fighting. Use of marijuana only was associated with increased odds of this outcome among women, but the odds ratios decreased as the intoxication index increased. Among women with no reported episodes of intoxication in the past year, the odds of alcohol- or other drug-related fighting were almost quintupled by the use of marijuana only; among women who became intoxicated on half of their drinking days, the odds were doubled. Among men who used marijuana only, increased odds of alcohol- or drug-related fighting were observed only for the 17 percent who never became intoxicated (OR = 2.11). Use of sedatives or tranquilizers only was not associated with the odds of alcohol- or drug-related fighting, but using either stimulants or cocaine only or multiple drugs significantly increased the odds of this outcome (OR = 3.17 and 2.60, respectively).

Past-year suicide attempt/ideation was uncorrelated with most of the sociodemographic variables examined in this analysis, although male gender, being married and being employed each exerted a protective effect. As with fighting, suicide attempt/ideation was positively associated with family history of alcoholism (OR = $e^{0.565}$ = 1.76) and negatively associated with age at first drink. The association between average daily ethanol and suicide attempt/ideation fell short of statistical significance (b = 0.069, p = .084), and the effect of the intoxication index, while positive, was weaker than its effect on fighting. Among individuals who drank to the point of intoxication on half of their drinking days, the odds of suicide attempt/ideation were slightly more than twice as high as among those who never became intoxicated (OR = 2.36).

Current drinkers who used painkillers only did not experience any higher odds of suicide attempt/ideation than those who did not use drugs at all. Use of marijuana only increased the odds of suicide attempt/ideation by a factor of 1.71, and use of sedatives or tranquilizers only, use of cocaine/stimulants only and multiple drug use were each associated with a four- to five-fold increase in the odds of this outcome (OR = 4.97, 3.93, and 4.32, respectively).

As shown in the bottom panels of Table III, exclusion of the drug use variables had virtually no effect on the estimated associations between each of the alcohol use measures and alcohol- or other drug-related fighting. In contrast, exclusion of the alcohol use variables led to an increase of 39 percent in the beta parameter for use of cocaine/stimulants and an increase of 55 percent in the beta parameter for multiple drug use. In the models pre-

dicting past-year suicide attempt/ideation, exclusion of the drug use variables resulted in a statistically significant association between average daily ethanol intake and this outcome. In the absence of the alcohol use variables, the beta parameters for all of the drug use variables were slightly inflated, but not to so great an extent as in the model predicting alcohol- or other drug-related fighting.

The analysis designed to identify whether the simultaneous use of alcohol and drugs contributed to the risk of violent behavior yielded results that were suggestive of such an effect for the outcome of alcohol- or other drug-related fighting but were not conclusive (Table V). Within the subsample of current drinkers who used drugs during the past year, and controlling for the number of drugs used, the beta parameter for the simultaneous use of alcohol and drugs indicated a positive effect that fell just short of statistical significance (b = .336, p = .087). For the outcome of suicide attempt/ideation, there was no evidence of any effect of simultaneous drug and alcohol use (b = 0.220, p = .243).

Table III. Multiple logistic regression models predicting odds of past-year alcohol- or other drug-related fighting and suicide attempt/ideation among drinkers 18 years of age and over

	Past-year alcohol- or other drug-related fighting			*Past-year suicide attempt/ideation*		
	Beta	*SE*	*p*	*Beta*	*SE*	*p*
PARAMETERS FROM FULL MODELS:						
Intercept	–1.031	0.756	.177	–1.844	0.555	.001
Age	–0.097	0.011	<.001	–0.008	0.004	.066
Male	0.484	0.226	.036	–0.369	0.183	.047
Black	–0.049	0.208	.814	–0.167	0.155	.287
Hispanic	0.048	0.232	.837	0.102	0.185	.584
Married	–0.457	0.167	.008	–0.637	0.093	<.001
Employed	–0.616	0.205	.004	–0.371	0.146	.013
Attended college	–0.504	0.185	.008	–0.153	0.102	.137
Family income ($000s)	–0.006	0.002	.005	–0.001	0.001	.163
Urban	–0.249	0.163	.130	0.076	0.138	.584
Total body water	0.034	0.015	.026	0.002	0.011	.881
FHP[a]	0.640	0.189	.001	0.565	0.107	<.001
Age at first drink	–0.084	0.027	.002	–0.060	0.017	.001
Average daily ethanol intake[b]	0.205	0.030	<.001	.069	0.040	.084
Intoxication index[c]	1.882	0.244	<.001	1.083	0.180	<.001
Used painkillers only	0.621	0.638	.334	0.212	0.526	.687
Used marijuana only	1.600	0.374	<.001	0.539	0.142	<.001
Used sedatives/tranquilizers only	0.928	0.670	.171	1.603	0.429	<.001
Used cocaine/stimulants only	1.154	0.406	.006	1.369	0.390	<.001
Used multiple drugs	0.954	0.182	<.001	1.465	0.167	<.001
Used marijuana only × male	–0.854	0.349	.017	—	—	—
Used marijuana only × intox. index[c]	–1.072	0.483	.030	—	—	—
Model goodnesss of fit F	F = 29.1; df = 15.8, 68; p < .001			F = 30.6; df = 17.3, 68; p < .001		
Hosmer-Lemeshow lack of fit X^2	X^2 = 6.5; df = 8; p = .587			X^2 = 8.5; df = 8; p = .383		
ALCOHOL USE PARAMETERS FROM MODELS EXCLUDING DRUG USE VARIABLES:						
Average daily ethanol intake[b]	0.227	0.032	<.001	0.097	0.037	.011
Intoxication index[c]	1.814	0.229	<.001	1.249	0.171	<.001
DRUG USE PARAMETERS FROM MODELS EXCLUDING ALCOHOL USE VARIABLES:						
Used painkillers only	0.587	0.709	.410	0.681	0.512	.188
Used marijuana only	1.123	0.288	<.001	0.641	0.141	<.001
Used sedatives/tranquilizers only	0.961	0.651	.144	1.692	0.440	<.001
Used cocaine/stimulants only	1.609	0.429	<.001	1.680	0.356	<.001
Used multiple drugs	1.474	0.171	<.001	1.682	0.149	<.001
Used marijuana only × male	–0.768	0.351	.032	—	—	—

[a]Positive family history of alcoholism
[b]Ounces of ethanol
[c]Proportion of drinking occasions resulting in intoxication, on a cube-root scale

DISCUSSION

This study found that level and pattern of alcohol intake and illicit use of drugs were highly correlated among current drinkers. Accordingly, multivariate analysis of the associations between alcohol and other drug use and violence revealed adjusted effects for the alcohol and drug use variables that were weaker than their unadjusted observed effects; however, the proportion of drinking occasions resulting in intoxication, the use of cocaine/stimulants only and multiple drug use all retained significant positive associations with both alcohol- and other drug-related fighting and suicide attempt or ideation even after adjusting for their correlation and the effects of potentially confounding background variables. Average daily ethanol intake was positively associated with the former but not the latter of these two outcomes, and use of sedatives/tranquilizers only was positively associated solely with the latter. Although marijuana use was positively associated with both outcomes, its association with alcohol- or other drug-related fighting was restricted to women and the small proportion of men who never became intoxicated.

Neither outcome was significantly associated with the use of painkillers, which mirrors findings from human experimental studies (Taylor and Chermack, 1993). Contrary to experimental studies that have found proaggressive effects of certain benzodiazapenes (Taylor and Chermack, 1993), the effect of sedative/tranquilizer use on alcohol- or other drug-

Table IV. Odds ratios and 95% confidence intervals for odds of alcohol- or other drug-related fighting and suicide attempt/ideation among current drinkers 18 years of age and over, according to level of alcohol intake and illicit drug use

	Past-year alcohol- or other drug-related fighting		*Past-year suicide attempt/ideation*	
	OR	*95% CI*	*OR*	*95% CI*
Average daily ethanol intake:				
0.015 oz. (= 1 drink/month)	1.00	(1.00–1.00)	1.00	(1.00–1.00)
0.075 oz. (= 1 drink/week)	1.01	(1.01–1.02)	NS	(1.00–1.01)
0.50 oz. (= 1 drink/day)	1.10	(1.07–1.14)	NS	(1.00–1.07)
1.00 oz. (= 2 drinks/day)	1.22	(1.15–1.30)	NS	(0.99–1.16)
2.50 oz. (= 5 drinks/day)	1.67	(1.44–1.93)	NS	(0.98–1.44)
Intoxication index:[a]				
Users of marijuana only				
.000	1.00	(1.00–1.00)	1.00	(1.00–1.00)
.100	NS	(0.93–2.30)	1.65	(1.40–1.95)
.250	NS	(0.90–3.09)	1.98	(1.58–2.47)
.500	NS	(0.87–4.13)	2.36	(1.78–3.13)
All other current drinkers				
.000	1.00	(1.00–1.00)	1.00	(1.00–1.00)
.100	2.40	(1.92–3.00)	1.65	(1.40–1.95)
.250	3.27	(2.42–4.42)	1.98	(1.58–2.47)
.500	4.46	(3.05–6.52)	2.36	(1.78–3.13)
Type of illicit drug use:				
None	1.00	(1.00–1.00)	1.00	(1.00–1.00)
Painkillers only	NS	(0.53–6.50)	NS	(0.44–3.46)
Marijuana only:				
Male				
Intoxication index[a] = .000	2.11	(1.06–4.22)	1.71	(1.30–2.26)
Intoxication index[a] = .100	NS	(0.82–2.01)	1.71	(1.30–2.26)
Intoxication index[a] = .250	NS	(0.69–1.67)	1.71	(1.30–2.26)
Intoxication index[a] = .500	NS	(0.55–1.47)	1.71	(1.30–2.26)
Female				
Intoxication index[a] = .000	4.96	(2.38–10.33)	1.71	(1.30–2.26)
Intoxication index[a] = .100	3.01	(1.74–5.22)	1.71	(1.30–2.26)
Intoxication index[a] = .250	2.52	(1.44–4.41)	1.71	(1.30–2.26)
Intoxication index[a] = .500	2.12	(1.15–3.90)	1.71	(1.30–2.26)
Sedatives/tranquilizers only	NS	(0.68–9.40)	4.97	(2.14–11.52)
Cocaine/stimulants only	3.17	(1.43–7.03)	3.93	(1.83–8.44)
Multiple drugs	2.60	(1.82–3.71)	4.32	(3.12–6.00)

[a]Proportion of drinking occasions resulting in intoxication.

related fighting fell short of statistical significance; neither did this study find support for a positive interaction between sedative/tranquilizer use and alcohol use, as might have been expected on the basis of animal studies (Miczek *et al.,* 1993a; Miczek and O'Donnell, 1980). This may reflect inclusion of sedatives and tranquilizers other than benzodiazapenes in this category.

In interpreting the effects for all of the different prescription-type drugs considered in this study, it is important to bear in mind that only the illicit use of these drugs, i.e., use without or beyond the limits of a doctor's prescription, was considered. Although it could be argued that the direct pharmacological effects of prescription drug use occur without regard to whether they are licitly or illicitly used, it seems reasonable to expect that aggressive, violent or suicidal outbursts rarely would result from medically prescribed dosages. The effects that this study found for prescription drugs probably reflect specific aspects of illicit use—e.g., excessive dosage, use without regard for adverse pharmacological interactions, and settings that may promote fighting and/or suicide attempt/ideation—that would not generalize to the use of these drugs under medical supervision.

The finding that marijuana use was associated with alcohol- and other drug-related fighting among women but not among most men bears some similarities to the findings of Valois *et al.* (1995), who reported that the effect of drug use on fighting was stronger for white females than white males. The NLAES data indicated that male and female marijuana users did not exhibit meaningful differences in the frequency of their marijuana use, the number of joints typically smoked, duration of use, underlying prevalence of use or the frequency with which they used marijuana and alcohol simultaneously. Thus, gender differences in the characteristics of use do not appear to account for this finding. While it is possible that males and females differ in their ability to biosynthesize THC, another possible explanation is suggested by animal studies which found that THC increased the level of attacks provoked and injuries received by subordinate rats that were threatened. Perhaps the characteristics of marijuana stupor leave women, whose social status is generally subordinate to that of men, particularly vulnerable to attack when marijuana-induced paranoia increases their perceived level of threat.

Also puzzling was the negative interaction between marijuana use and the intoxication index for the outcome of alcohol- or other drug-related fighting. The insignificant effect of the intoxication index among in-

Table V. Multiple logistic regression models predicting odds of past-year alcohol- or other drug-related fighting and suicide attempt/ideation among current drinkers 18 years of age and over who used illicit drugs in past year

	Past-year alcohol- or other drug-related fighting			*Past-year suicide attempt/ideation*		
	Beta	*SE*	*p*	*Beta*	*SE*	*p*
Intercept	–0.891	0.762	.246	–1.804	0.558	.002
Age	–0.094	0.011	<.001	–0.008	0.004	.088
Male	0.336	0.221	.134	–0.356	0.182	.055
Black	0.007	0.202	.973	–0.133	0.158	.401
Hispanic	0.032	0.236	.890	0.050	0.189	.793
Married	–0.469	0.169	.007	–0.634	0.094	<.001
Employed	–0.614	0.204	.004	–0.350	0.146	.019
Attended college	–0.531	0.186	.006	–0.160	0.103	.127
Family income ($000s)	–0.006	0.002	.004	–0.001	0.001	.147
Urban	–0.236	0.162	.150	0.072	0.138	.602
Total body water	0.032	0.015	.035	<0.001	0.011	.999
FHP[a]	0.621	0.186	.001	0.565	0.107	<.001
Age at first drink	–0.081	0.027	.003	–0.061	0.017	<.001
Average daily ethanol intake[b]	0.194	0.028	<.001	0.066	0.039	.097
Intoxication index[c]	1.711	0.236	<.001	1.098	0.179	<.001
Number of drugs used	0.262	0.083	.002	0.445	0.083	<.001
Ever used any drug simultaneously with alcohol	0.336	0.194	.087	0.220	0.187	.243
Model goodnesss of fit F	F = 38.0; df = 12.3, 68; p < .001			F = 35.2; df = 14.8, 68; p < .001		
Hosmer-Lemeshow lack of fit X^2	X^2 = 4.9; df = 8; p = .767			X^2 = 11.1; df = 8; p = .199		

[a]Positive family history of alcoholism.
[b]Ounces of ethanol.
[c]Proportion of drinking occasions resulting in intoxication, on a cube-root scale.

dividuals who used marijuana only suggests that THC might counter the proaggressive effects of an acute dose of ethanol. This interpretation at first seems inconsistent with the *increased* risk of alcohol- or other drug-related fighting that was observed among female marijuana users who *never* became intoxicated, a finding that suggests a proaggressive rather than a counteraggressive effect of THC. However, if women's marijuana-related fighting is largely defensive in nature (in response to attacks provoked by their vulnerability), then increased assertiveness or aggression associated with intoxication might reduce such fighting and thus be consistent with the pattern of results observed in this study. Alternatively, occasions of intoxication may overlap with marijuana use more than with the use of other types of drugs. If this were the case, then the negative interaction between marijuana use and the intoxication index might merely indicate that marijuana use contributes less to the already increased odds of fighting when intoxicated than it does to the odds of fighting when sober and, conversely, that intoxication has a diminished effect on occasions when the odds of fighting have already been increased by marijuana use.

In order to ascertain that the alcohol and other drug use effects identified in this analysis were not merely indirect measures of an underlying psychopathology associated with the use of these substances, the model for past-year suicide attempt/ ideation was recalculated with a dichotomous measure of DSM-IV (American Psychiatric Association, 1994) major depression included among the predictor variables. Suicide attempt and ideation were removed from the symptom items upon which this measure of major depression was based, in order to avoid inflating its estimated effect. When major depression was included in the model, the effect of average daily ethanol intake remained insignificant and the beta parameter for the intoxication index was reduced by 19 percent. The effects of using marijuana only and of using cocaine/stimulants only were no longer statistically significant after adjusting for major depression, and the effect of using painkillers only remained insignificant. Using sedatives or tranquilizers only retained a significant association with the odds of suicide attempt/ideation, but the magnitude of the beta parameter was reduced by one third. Multiple drug use retained a significant effect, with less than a five percent reduction in magnitude. In summary, the underlying disorder of major depression accounted for some but by no means all of the observed associations between suicide attempt/ideation and alcohol and drug use. No such manipulation was performed for the model estimating alcohol- or other drug-related fighting, because the NLAES did not measure antisocial personality, the disorder that would be most likely to be associated with this outcome.

This analysis demonstrated that failure to account for both alcohol use and other drug use may lead to overestimation of associations of each individual substance with fighting and suicide attempt/ideation. This reflects the frequent overlap in the use of alcohol and other drugs, which must be considered both in understanding their respective roles in the etiology of various social problems and in tailoring treatment and prevention efforts to reduce the prevalence and impact of alcohol- and other drug-related violence. In comparing the magnitudes of the effects of the alcohol and drug use variables on the two different outcomes, it is important to bear in mind that the associations of these variables with fighting are inherently exaggerated relative to those with suicide attempt/ideation because the former included only fights related to alcohol or other drug use whereas the latter was unrestricted. Given this bias favoring larger odds ratios for the outcome of fighting, it is notable that the use of multiple drugs exerted so much stronger an effect on suicide attempt/ideation—even after adjusting for major depression—and marks this group of polysubstance users as those for whom treatment and prevention efforts are strongly needed.

This study did not touch upon many of the forms of aggression and violence that may be associated with the use of alcohol and drugs, for example, sexual abuse, spousal or child abuse, verbal aggression, aggressive driving, and violence associated with criminal activities or law enforcement. Although these types of violent actions are not good candidates for inclusion in large, general population studies (they are either too rare and too unlikely to be reported), they could be examined in smaller studies of appropriate populations that could help to clarify the mechanisms of alcohol and drug effects (e.g., disinhibition versus arousal or expectancy) and the moderating effects of both individual characteristics such as comorbid psychiatric disorders and the settings in which alcohol or drug use takes place and in which violence occurs. Additional clarification of these issues may help to understand why only a small minority of drug users and drinkers, even heavy drinkers, commit various types of violent acts, whereas the majority do not engage in violent behavior despite their alcohol and drug use.

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CHAPTER 9
TREATMENT AND ALCOHOL DEPENDENCE

Toward an Alcohol Treatment Model: A Comparison of Treated and Untreated Respondents With DSM–IV Alcohol Use Disorders in the General Population

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The purpose of this study was to compare characteristics of persons with alcohol use disorders who sought alcohol treatment with those who did not using data from a nationally representative sample of the United States. Applying an organizing framework from the larger literature on service utilization, a logistic regression analysis was conducted to examine the interaction among factors influencing treatment. The results identified unemployment status and lower educational level as barriers to alcohol treatment, but the impact of these factors differed depending on whether the respondent had previous experience with alcohol treatment. The major findings of this study are discussed in terms of consumer satisfaction, minimizing barriers to alcohol treatment services, and the need to examine individual determinants of alcohol treatment within the larger context of organizational and sociopolitical factors.

The majority of the literature on factors influencing alcohol treatment utilization consists of studies examining the characteristics of people in treated samples without comparison groups.[1,2] These studies are uninformative with regard to the population of greatest clinical and policy relevance (i.e., those persons with an alcohol use disorder missing from the treatment population). The few studies that have contrasted general population problem drinkers with and without treatment, or general population problem drinkers with persons in treatment have been limited by small or unrepresentative samples, ill-defined measures of alcohol problems, and the small number of factors influencing alcohol treatment examined.[3–5] The restriction in the nature and number of variables included in these studies have hindered both the development of models to organize factors influencing treatment utilization on the basis of the function they play in the help-seeking process and the means by which to examine important interactions between variable domains.

The purpose of this study was to compare characteristics of persons with past year alcohol use disorders (i.e., alcohol abuse and/or dependence) who sought treatment during the same time period and those who did not using data from a nationally representative sample of the United States population. In this study, a framework from larger literature on service utilization in the general medical care was adapted to organize factors influencing alcohol treatment utilization.

Health care researchers have generally partitioned variables that play a role in the help-seeking process into predisposing, enabling, and illness level or need variables.[6–8] Predisposing variables such as sociodemographic characteristics define a propensity for an individual to seek treatment that exists before the onset of a specific episode of an alcohol use disorder. Experiences with past alcohol use disorders and associated treatment are included in this domain, because there is considerable evidence that people with past disorders and treatment are more likely to seek treatment in the future than those without such experiences.[2] Enabling factors are available individual and community resources that impact on the use of services such as income, health insurance coverage, and urban-rural composition. Illness level or need factors are those related to the severity of the alcohol use disorder or factors contributing to the severity of an alcohol use disorder, including comorbidities and the quantity and frequency of alcohol intake.

In addition to the organizing framework adapted from the general health services utilization literature, this study

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was premised on the public policy concept of equitable distribution (i.e., alcohol treatment services should be distributed solely on the basis of alcohol treatment need). The goal of the concept of equitable distribution is that factors influencing treatment utilization, other than those directly related to illness level or need, such as predisposing or enabling factors, should be identified and further minimized if amenable to change. Of particular interest in this study was the identification of factors that modified the relationship between need and treatment utilization.

METHODS

Sample

The analysis of factors influencing alcohol treatment utilization were based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism. Fieldwork for the study was conducted by the U.S. Bureau of the Census. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. The household response rate was 91.9%, and the sample person response rate was 97.4%.

The NLAES featured a complex multistage design.[9,10] Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probability proportional to size. From a sampling frame of ~2,000 PSUs, 198 were selected for inclusion in the 1992 NLAES sample, including 52 that were self-representing (i.e., selected with certainty). Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically for sample. Oversampling of the black population was accomplished at this stage of sample selection to secure adequate numbers for analytic purposes. Segments then were divided into clusters of ~4–8 housing units, and all occupied housing units were included in the NLAES. Within each household, one randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18–29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavier drinking subgroup. This subgroup of young adults was randomly sampled at a ratio of 2.25:1.00.

Alcohol treatment variable

Respondents in this survey were asked if during the past year they had gone anywhere or seen anyone for problems related to their drinking. To capture more completely the entire alcohol help-seeking population, respondents were specifically instructed to include any help they had received for their drinking, including help for combined alcohol and drug use *if* alcohol was the major problem for which they sought help. Alcohol treatment sources were defined broadly, and respondents were asked to indicate separately whether they sought help from 23 different treatment sources: inpatient alcohol and/or drug rehabilitation programs and inpatient wards of general or psychiatric hospitals; outpatient clinics and alcohol and/or drug detoxification units; 12-step groups, including Alcoholics Anonymous, Narcotics or Cocaine Anonymous, or Al-anon; social services; and various health professionals, such as psychiatrists, psychologists, social workers, and the clergy. Respondents receiving help from any of these sources during the past year constituted the treatment group examined in this study. Low prevalences precluded separate analyses based on treatment type.

Diagnostic assessment of alcohol use disorders

Diagnoses of alcohol abuse and dependence were derived from the NLAES survey instrument, the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS).[11] The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV criteria for alcohol use disorders.[12] These questions are described in detailed elsewhere.[13] Although the DSM-IV was not published until 1994, the specific diagnostic criteria of interest were known before the conduct of the NLAES[14] and, therefore, incorporated in their entirety within the AUDADIS. In an independent test-retest study conducted in the general population, AUDADIS diagnoses of alcohol use disorders were shown to be highly reliable achieving reliability (kappa) coefficients of 0.76 and 0.73 for the past year and before the past year time frames.[15]

Consistent with DSM-IV, an AUDADIS diagnosis of alcohol abuse required that a person exhibit a maladaptive pattern of alcohol use, leading to clinically significant impairment or distress, as demonstrated by at least one of the following in any 1 year: (1) continuing to drink despite a social or interpersonal problem caused or exacerbated by the effects of drinking; (2) recurrent drinking in situations in which alcohol use is physically hazardous; (3) recurrent drinking resulting in a failure to fulfill major role obligations; or (4) recurrent alcohol-related legal problems. An AUDADIS diagnosis of alcohol dependence required that a person meet at least 3 of 7 criteria defined for dependence in

any 1 year including: (1) tolerance; (2) withdrawal or relief or avoidance of withdrawal; (3) persistent desire or unsuccessful attempts to cut down or stop drinking; (4) spending much time drinking or recovering from its effects; (5) giving up or reducing occupational, social, or recreational activities in favor of drinking; (6) impaired control over drinking; and (7) continuing to drink despite a physical or psychological problem caused or exacerbated by drinking.

Past year diagnoses of alcohol abuse and dependence also satisfied the clustering or duration criteria of the DSM-IV definitions. The duration criteria of the DSM-IV included the requirement for a clustering of symptoms within any 1-year period, in addition to associating duration qualifiers with certain abuse and dependence symptoms. The duration qualifiers are defined as the repetitiveness with which symptoms must occur to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often," and "persistent" appearing in the diagnostic criteria.

Predisposing variables

Predisposing factors, shown in past research[2] to influence alcohol treatment utilization, included the following sociodemographic variables: ethnicity (black versus nonblack), education (less than high school graduate versus high school graduate and beyond), and current marital status (married or living with someone as if married versus separated, divorced, never married, or widowed).

A family history of alcoholism variable was constructed on the premise that respondents with family histories of alcohol use disorders are likely to be more knowledgeable of the nature of the disorders and their adverse consequences than those with no family histories and therefore more likely to recognize their own alcohol problem and to enter treatment. Respondents with any biological or nonbiological first- and second-degree relative reported to have been an alcoholic or problem drinker constituted the family history-positive group examined in this study. Family history of alcoholism was determined by eliciting separate responses for each biological and nonbiological first- and second-degree relative to the question: "Has your/how many of your (*relative type*) are now, or were in the past, alcoholics or problem drinkers?" An alcoholic or problem drinker was specifically defined for respondents as a person who has physical or emotional problems because of drinking; problems with a spouse, family, or friends because of drinking; problems at work because of drinking; problems with the police because of drinking (e.g., drunk driving); or a person who seems to spend a lot of time drinking or being hungover.

Measures of prior to the past year DSM-IV alcohol use disorders and prior to the past year alcohol treatment were also examined as predisposing variables. Diagnoses of past DSM-IV alcohol abuse and/or dependence were also measured as syndromes on the AUDADIS, or as episodes defined as the repetitive clustering of the required number of symptoms necessary to achieve a diagnosis prior to the past year. The prior to the past year alcohol treatment measure was derived using the same detailed list of treatment sources described for constructing the current treatment variable.

Enabling variables

Enabling variables consisted of a continuous measure of monthly family income represented by the midpoints of 23 income categories, ranging from less than $555 to $13,000. The income categories in this study were selected to be consistent with the Bureau of the Census's Supplementary Income Survey of Program Participation. Other enabling variables included current health insurance coverage status (health insurance versus no health insurance), employment status in the past year (employed versus not employed), and urban versus rural locale, which served as an indicator of the relative availability of community resources for alcohol treatment services. Other enabling variables that may impact on treatment utilization or serve as barriers to treatment included the presence of a current alcoholic spouse or partner in the household and the presence of children under the age of 14 years in the household that may impact specifically on women's entry to treatment.

Illness level or need variables

The severity of alcohol use disorders, measured as the number of alcohol symptoms representing the seven DSM-IV dependence and four DSM-IV abuse criteria (ranging from 1 to 31) present during the past year episode, served as the major exposure variable of interest in this study. Other need variables contributing to the severity of the alcohol use disorders included early onset (< 25 years) versus late onset (25 years and older) of first episode of an alcohol use disorder, comorbid major depression and drug disorders, and any use of an illicit drug occurring during the past year. Consistent with the DSM-IV, the AUDADIS diagnosis of major depression required the presence of at least five depressive symptoms (inclusive of depressed mood or loss of pleasure and interest) nearly every day for most of the day for at least the same 2-week period. Social and/or occupational dysfunction must also have been present during the disturbance, and episodes of

major depression exclusively caused by bereavement or physical illness were ruled out. Past year DSM-IV drug-specific diagnoses of abuse and dependence were first derived separately for sedatives, tranquilizers, opioids (other than heroin), amphetamines, cocaine (and crack cocaine), cannabis (and tetrahydrocannabinol and hashish), heroin, methadone, and hallucinogens. Then, a composite measure of any of these drug use disorders was constructed. Reliability coefficients (kappas) associated with past year DSM-IV major depressive disorder and any drug use disorder were 0.65 and 0.80, as determined from an independent test-retest study conducted in a general population sample.[15]

Average daily ethanol intake for beer, wine, and liquor was based on patterns of usual and heaviest consumption during the past year. The measures obtained for each type of beverage included frequency of drinking (converted to number of drinking days per year), typical number of drinks consumed per drinking day, and typical size of drink (ounces of beer, wine, or liquor). Ounces of beverage were converted to ounces of ethanol using the following conversion factors: 0.045 for beer, 0.121 for wine, and 0.409 for liquor.[16–20] To estimate average daily ethanol intake based on usual and heaviest past-year consumption, the annual volume of ethanol for each beverage type was calculated as follows: oz $\text{ethanol}_{\text{beverage}}$ (total minus heavy drinking $\text{days/year}_{\text{beverage}}$ x number of drinks/usual drinking $\text{day}_{\text{beverage}}$ x oz of beverage in typical drink consumed on usual drinking $\text{day}_{\text{beverage}}$ x ethanol conversion $\text{factor}_{\text{beverage}}$) + (heavy drinking $\text{days/year}_{\text{beverage}}$ x number of drinks/heavy drinking $\text{day}_{\text{beverage}}$ x oz of beverage in typical drink consumed on heavy drinking $\text{day}_{\text{beverage}}$ x ethanol conversion $\text{factor}_{\text{beverage}}$). These volumes were then summed over the three beverage types, and the resulting volume was divided by 365.

Statistical analyses

Because of the complex survey design of the NLAES, variance estimation procedures that assume a simple random sample cannot be used. Research has shown that clustering and stratification of the NLAES sample may result in standard errors somewhat larger than those that would be obtained with a simple random sample of equal size. To take into account the NLAES sample design, all standard errors and the results of the linear logistic regression analysis were generated using SUDAAN,[21] a software program that uses Taylor series linearization to adjust for sample design characteristics.

A linear logistic regression analysis was conducted to determine the association between the need for alcohol treatment services and treatment (i.e., the dependent or outcome variable) as premised on the public policy concept of equitable distribution. In this model, need or illness severity, expressed as the number of positive DSM-IV alcohol symptoms, served as the major exposure variable of interest. Consistent with the concept of equitable distribution of alcohol treatment services, all of those factors not related to treatment need were specified as potential confounders or modifiers of the need-treatment association, including all predisposing, enabling, and illness level factors contributing to the severity of the alcohol use disorders. Operationally defined, a variable is a confounder if its adjustment in the data results in a substantial change (e.g., >0.1) in the estimate of the effect of severity on the outcome measure and treatment, conditional upon all other potential confounders. A statistically significant cross-product term is indicative of nonuniformity across strata of another risk factor, or what is usually referred to as effect modification.

Data analytic strategy consisted of two stages. The first stage entailed the identification of important modifiers by allowing for interaction effects up to second-degree product terms involving the exposure or severity or need variable (e.g., severity x sex). Using a backward elimination process, all nonsignificant cross-product terms ($p > 0.01$) were eliminated from the model, while at the same time all main effect terms were retained. We included only second-degree interaction terms because cross-product terms involving higher levels of interaction are often questionable because of induced multicollinearity.

Stage 2 entailed the identification of confounders, or alternatively, the deletion of nonconfounders from the reduced model resulting from the first stage (i.e., a model containing all main effects and significant cross-product terms). In the case of no interaction, the elimination of nonconfounders consists of removing all main effects (i.e., predictors) to produce an even more reduced model. If the main effect coefficient of the exposure variable, severity of the alcohol use disorder, does not materially change, the use of the reduced model without the main effect terms can lead to a gain in precision. On the other hand, if the exposure-involved coefficient does show substantial change upon refitting, the main effect terms should be retained in the model as confounders. In the presence of interaction, main effect terms involved in the model as modifiers are not candidates for deletion. Each of these main effect terms must be retained to obtain precise estimates of the magnitude of the associated interaction effect. Under these conditions, the main effect ex-

posure coefficient and all exposure-related cross-product coefficients must be monitored for change upon refitting to determine if actual confounders should be retained in the model.

RESULTS

The 1-year prevalence of DSM-IV alcohol abuse and/or dependence in this general population sample was 7.4% (n = 2,910). Among those with a current alcohol use disorder, 9.9% (n = 2,625) had obtained alcohol treatment during the past year, whereas 90.1% (n = 285) had not obtained treatment. Compared with respondents who had not obtained alcohol treatment, respondents who had obtained treatment were younger, slightly less educated, and had more experiences with family histories of alcohol use disorders, past alcohol use disorders, and past alcohol treatment (Table 1). With regard to enabling factors, respondents who entered treatment had lower family incomes, were less likely to have health insurance coverage or be employed, and were more likely to have a spouse or partner who was an alcoholic or problem drinker during the past year than those who did not enter treatment (Table 2). Compared with those who had not obtained alcohol treatment during the past year, respondents who did obtain treatment had a greater number of alcohol symptoms, consumed more alcohol, and were more likely to have a comorbid major depressive or drug use disorders over the course of the past year (Table 3).

The results of the logistic regression analysis of past year treatment is shown for the full and reduced models in Table 4. None of the predisposing or enabling factors included in the model was independently associated with the odds of obtaining treatment. Among the illness level factors that might contribute to the severity of an alcohol use disorder, both average daily ethanol intake and comorbid major depression were identified as significant predictors of obtaining treatment. The presence of a comorbid major depression and greater levels of consumption were both associated with an increased probability of obtaining alcohol treatment.

A history of past treatment for alcohol use disorder, employment status, and education were found to modify the need-treatment association. Table 5 presents the adjusted odds ratios and 95% confidence intervals for the need-treatment association at various levels of severity, education, employment, and past treatment status. In general, the probability of alcohol treatment increased with severity of the alcohol use disorders, but this association was not statistically significant in all population subgroups. Among respondents with prior treatment for a past alcohol use

Table 1. Percentage Distribution of Respondents Entering Treatment and Not Entering Treatment for an Alcohol Use Disorder: Predisposing Factors

Factor	Entering treatment	Not entering treatment
Sex		
Male	76.9 (2.7)	70.6 (1.0)
Female	23.1 (2.7)	29.4 (1.0)
Age		
18–29 years	19.4 (2.0)	22.8 (0.4)
30–44 years	50.2 (2.6)	33.2 (0.3)
45–64 years	26.0 (2.3)	26.6 (0.3)
65+ years	4.4 (1.0)	17.4 (0.3)
Ethnicity		
Black	9.6 (1.8)	8.0 (0.7)
Non-Black	90.4 (1.8)	92.0 (0.7)
Education		
Less than high school	22.5 (2.7)	15.4 (1.0)
High school and beyond	77.5 (2.7)	84.6 (1.0)
Marital status		
Married/living with someone	40.7 (3.5)	43.3 (1.3)
Separated/divorced/widowed/ never married	59.3 (3.5)	56.7 (1.3)
Family history of an alcohol use disorder		
Yes	86.6 (2.5)	71.3 (1.1)
No	13.4 (2.5)	28.7 (1.1)
Past alcohol use disorder		
Yes	77.0 (2.8)	48.9 (1.2)
No	23.0 (2.8)	51.1 (1.2)
Treatment for past alcohol use disorder		
Yes	57.9 (3.4)	10.0 (0.7)
No	42.1 (3.4)	90.0 (0.7)

Note: Standard errors are in parentheses.

Table 2. Percentage Distribution of Respondents Entering Treatment and Not Entering Treatment for an Alcohol Use Disorder: Enabling Factors

Factor	Entering treatment	Not entering treatment
Urbanicity		
Urban	76.4 (3.1)	76.4 (1.2)
Rural	23.6 (3.1)	23.6 (1.2)
Health insurance		
Yes	62.0 (3.2)	75.6 (1.1)
No	38.0 (3.2)	24.4 (1.1)
Income		
≤$1,100	38.3 (3.5)	26.1 (1.6)
$1,101–$1,999	19.1 (2.7)	20.2 (0.9)
$2,000–$3,999	23.6 (2.9)	32.6 (1.1)
≥$4,000	19.0 (2.9)	21.1 (1.1)
Employment in past year		
Yes	85.9 (2.3)	92.8 (0.6)
No	14.1 (2.3)	7.2 (0.6)
Children under 14 years living at home		
Yes	21.6 (2.7)	24.2 (1.0)
No	78.4 (2.7)	75.8 (1.0)
Current spouse/partner with an alcohol use disorder		
Yes	6.8 (1.6)	3.6 (0.5)
No	93.2 (1.6)	96.4 (0.5)

Note: Standard errors are in parentheses.

disorder, the probability of obtaining alcohol treatment for a current disorder was significantly increased as a function of severity only for those who were employed and who had graduated high school. For those respondents without a prior history of alcohol treatment, the probability of treatment significantly increased with severity for each level of education and employment status, except among those who were unemployed and who had less than a high school education.

DISCUSSION

The major results of this study indicate that unemployment status and less than a high school education serve as barriers to alcohol treatment. Both factors were shown to reduce the positive association between severity of alcohol problems and the probability of obtaining treatment. However, the impact of these factors differs in important ways between individuals who have and have not had experiences with prior alcohol treatment. For those respondents with no prior alcohol treatment, unemployment status serves as a barrier to treatment entry only among those having lower educational levels. Among respondents with prior alcohol treatment, unemployment was also identified as a barrier to care, regardless of educational level, whereas lower educational level serves as a barrier to care, regardless of unemployment status. The finding that lower levels of education serve to impede treatment entry among the employed with prior alcohol treatment experiences, but not among respondents with no prior alcohol treatment suggests that consumer satisfaction may play an important role in influencing alcohol treatment seeking. Respondents of lower education may not have the requisite skills, knowledge, or expertise to perform efficiently in various types of alcohol treatment settings or to understand information about dealing with their alcohol use disorder—both of which can lead to dissatisfaction with the care received. This finding underscores the need for future studies examining factors influencing alcohol treatment to collect data on consumer satisfaction and to differentiate clearly between those factors that may impact differentially on the two major components of service utilization (i.e., treatment initiation and treatment continuation).

Table 3. Percentage Distribution of Respondents Entering Treatment and Not Entering Treatment for an Alcohol Use Disorder: Illness Severity Factors

Factor	Entering treatment	Not entering treatment
Severity of alcohol use disorders		
No. of alcohol symptoms		
1–3 symptoms	4.6 (1.3)	15.9 (0.9)
4–6 symptoms	18.3 (2.8)	37.2 (1.1)
7–13 symptoms	37.2 (3.2)	39.1 (1.2)
≥14 symptoms	39.9 (3.3)	7.8 (0.6)
Factors contributing to severity of alcohol use disorders		
Onset of alcohol use disorder		
≤25 years	69.2 (3.0)	72.1 (1.2)
>25 years	30.8 (3.0)	27.9 (1.2)
Average daily ethanol intake		
≤0.49 oz	15.8 (2.6)	27.4 (1.0)
0.5–0.99 oz	13.4 (2.4)	25.7 (1.0)
1.0–1.99 oz	23.1 (2.7)	23.9 (1.0)
≥2.00 oz	47.7 (3.6)	23.0 (1.1)
Comorbid major depression		
Yes	21.5 (3.2)	8.3 (0.6)
No	78.5 (3.2)	91.7 (0.6)
Comorbid drug use disorder		
Yes	25.5 (2.8)	11.8 (0.8)
No	74.5 (2.8)	88.2 (0.8)
Illicit drug use in past year		
Yes	14.1 (2.4)	15.6 (0.8)
No	85.9 (2.4)	84.4 (0.8)

Note: Standard errors are in parentheses.

Unemployment status directly impacted on the relationship between alcohol treatment need (i.e., severity of the alcohol use disorder) and alcohol treatment, particularly among respondents with less than a high school education. That factors other than illness severity or need, such as an individual's level of education, should serve as barriers to treatment is not compatible with the concept of equitable distribution. In general, the goal of equitable distribution is to determine how individual characteristics identified as barriers to alcohol treatment can be minimized. In view of this study's findings, it would seem more realistic to consider possible changes to the enabling variable, unemployment, rather than the predisposing education variable. In this regard, it is interesting that the other enabling factors examined in this study representing individual and family financial resources, namely family income and health insurance coverage, were not shown to influence treatment utilization. These findings, in combination, suggest that current federal programs designed to equalize access to medical care through reducing the proportion of individual or family economic resources spent on care (e.g., Medicare and Medicaid) may not be addressing the needs of those with relatively little or no financial resources, such as the unemployed. In addition, the finding that severity was only associated with treatment among the more highly educated employed who had prior treatment experience suggests that repeated alcohol treatment may be beyond the scope of those who do not have private health insurance coverage (i.e., those with lower educations). In contrast, predisposing factors, such as educational level, cannot generally be mini-

mized to influence their adverse impact on obtaining alcohol treatment. However, given the effect of educational level on the need-treatment association, the apparent inequity in seeking treatment may be minimized by tailoring alcohol services to meet the special needs of those with lower educational levels.

What is possibly the most interesting aspect of this study was the failure to identify certain predisposing factors (e.g., age, ethnicity, and sex) and enabling vari-

Table 4. Results of Logistic Regression Analysis

Variable	All potential confounders included			All actual confounders included		
	β	SE(β)	p	β	SE(β)	p
Intercept	−3.96	0.81	<0.000	−3.69	0.48	<0.000
Predisposing factors						
Sex (effect of male)	−0.03	0.20	0.869			
Age	0.01	0.01	0.639			
Ethnicity (effect of Black)	−0.11	0.29	0.705			
Education (effect of less than high school)	0.90	0.39	0.024	0.96	0.41	0.021
Marital status (effect of married/living with someone)	0.07	0.21	0.751			
Family history of alcohol use disorder	0.39	0.24	0.110			
Past alcohol disorder	0.16	0.20	0.441			
Treatment for past alcohol use disorder	2.88	0.38	<0.000	3.09	0.36	<0.0005
Enabling factors						
Urbanicity (effect of urban)	0.25	0.21	0.252			
Health insurance	−0.35	0.19	0.064			
Employment in past year	−1.09	0.52	0.039	−1.31	0.48	<0.009
Income	0.01	0.01	0.889			
Children under 14 living at home	−0.06	0.24	0.808			
Current spouse/partner with alcohol use disorder	0.20	0.41	0.624			
Illness-level or need factors						
Severity of alcohol use disorder						
No. of alcohol symptoms	0.10	0.04	0.014	0.10	0.04	<0.006
Factors contributing to severity of alcohol use disorder						
Onset of alcohol use disorder (effect of <25 years)	−0.34	0.23	0.168			
Average daily ethanol intake*	0.13	0.09	0.145	0.14	0.09	0.101
Comorbid major depression	0.50	0.26	0.058	0.52	0.24	0.034
Comorbid drug use disorder	−0.07	0.24	0.760			
Illicit drug use in past year	−0.31	0.26	0.255			
Interactions						
Severity × past treatment for alcohol use disorder	−0.07	0.03	0.003	−0.08	0.03	0.003
Severity × employment in past year	0.09	0.36	0.001	0.10	0.04	0.006
Severity × education	−0.08	0.03	0.007	−0.08	0.03	0.006

Note: Goodness-of-fit for final reduced model: Satterthwaites, $F(10,66)$ $p < 0.00001$.
* Ethanol intake in ounces on a log scale.

Table 5. Estimated Adjusted Odds Ratios and 95% Confidence Intervals for the Need-Treatment Association at Various Levels of Illness Severity, Education and Employment, and Past Treatment Status

Past treatment for an alcohol use disorder/No. of alcohol symptoms	Employment in past year		No employment in past year	
	Less than high school	High school and beyond	Less than high school	High school and beyond
Prior treatment for an alcohol use disorder				
3 symptoms	1.12 (0.92, 1.35)	1.44 (1.25, 1.66)	0.82 (0.65, 1.04)	1.06 (0.86, 1.31)
5 symptoms	1.20 (0.87, 1.65)	1.84 (1.45, 2.33)	0.72 (0.48, 1.06)	1.10 (0.78, 1.56)
7 symptoms	1.30 (0.83, 2.02)	2.35 (1.69, 3.27)	0.63 (0.36, 1.09)	1.15 (0.71, 1.86)
9 symptoms	1.40 (0.78, 2.48)	3.00 (1.96, 4.59)	0.55 (0.28, 1.11)	1.19 (0.64, 2.22)
11 symptoms	1.50 (0.75, 3.03)	3.83 (2.28, 6.43)	0.49 (0.21, 1.41)	1.24 (0.58, 2.65)
13 symptoms	1.62 (0.70, 3.70)	4.89 (2.65, 9.03)	0.43 (0.16, 1.17)	1.29 (0.54, 3.17)
15 symptoms	1.74 (0.67, 4.53)	6.24 (3.08, 12.67)	0.37 (0.11, 1.19)	1.34 (0.48, 3.78)
No prior treatment for an alcohol use disorder				
3 symptoms	1.44 (1.20, 1.73)	1.86 (1.61, 2.15)	1.06 (0.84, 1.34)	1.37 (1.10, 1.70)
5 symptoms	1.84 (1.36, 2.49)	2.82 (2.22, 3.57)	1.10 (0.75, 1.63)	1.69 (1.17, 2.43)
7 symptoms	2.35 (1.54, 3.59)	4.27 (3.05, 5.95)	1.15 (0.66, 1.98)	2.08 (1.25, 3.46)
9 symptoms	3.01 (1.74, 5.19)	6.46 (4.21, 9.91)	1.19 (0.59, 2.41)	2.57 (1.34, 4.93)
11 symptoms	3.84 (2.01, 7.48)	9.77 (5.80, 16.50)	1.24 (0.53, 2.93)	3.16 (1.42, 7.02)
13 symptoms	4.90 (2.23, 10.78)	14.80 (8.00, 27.00)	1.29 (0.47, 3.56)	3.90 (1.52, 10.01)
15 symptoms	6.26 (2.52, 15.53)	22.41 (10.01, 32.56)	1.34 (0.42, 4.32)	4.81 (2.01, 14.28)

ables (e.g., urbanicity and income) as important confounders or mediators of the need-treatment entry association. In this respect, the results of this study are at variance with most of the literature on alcohol treatment utilization to date.[22–25] The reasons for these discrepancies are largely the result of differences in the research questions addressed and the populations of interest. The preponderance of the literature on alcohol treatment utilization asks why people enter alcohol treatment and consists primarily of studies investigating characteristics of individuals in treated populations. The question addressed in the present study was why more people in need of treatment do not obtain treatment with a focus on the population of persons with alcohol use disorders missing from the treated population. As Weisner[2] appropriately points out, much of the alcohol treatment literature focusing on treated populations is misdirected, "the literature often forgets the intrinsic differences between the groups and makes assumptions about the missing alcoholic population—about who they are, when they came, and the characteristics that helped them avoid treatment earlier, such as denial and recalcitrance—on the basis of their counterparts, those in treatment."

The major strengths of this study include the direct comparison of persons with alcohol use disorders who do and do not seek alcohol treatment in a general population sample, the examination of numerous factors and their interactions posited to influence alcohol treatment utilization, adoption and expansion of general medical care service utilization models to variables of specific relevance to the alcohol treatment-seeking equation, and the use of the public policy concept of equitable distribution that served to guide statistical modeling methodology and aid in the identification of barriers to care.

On the other hand, an examination of the general health care utilization literature also underscores the limitations inherent in the present study. Factors identified in the literature that may importantly predispose individuals to seek help for an alcohol use disorder—such as health beliefs and attitudes, the availability of social networks, interpersonal crises and legal problems—were not fully measured in this survey. Factors that are commonly cited as reasons for failure or delay in going to treatment (i.e., denial and stigmatization) were also not directly measured in this study independent of the treatment-seeking variable. This study also focused on predisposing, enabling, and need components as individual determinants of alcohol treatment utilization. However, there is a rapidly growing awareness in general medical service and alcohol service utilization literatures that these individual level determinants need to be examined within the larger context of both organizational and environmental factors and sociopolitical and health policy factors. Organizational factors include culturally determined attitudes about seeking help, client selection practices, consumer satisfaction with the quantity and quality of care received, and the actual characteristics of the alcohol treatment service delivery system (e.g., type, site, purpose, intensity, and continuity of services) and the structure in which alcohol treatment services are provided (e.g., combined alcohol and drug treatment services and linkages between alcohol treatment and other health and social services systems). Sociopolitical factors refer to various aspects of health care policy, including mode of financing, education, manpower, shifting public opinion concerning the way alcohol use disorders are viewed, and the impact of factors related to health care trends, such as managed health care and mandated treatment. Although many of these organizational and sociopolitical factors can be studied as characteristics of the population at risk, other factors can only be examined using the health care delivery system as the unit of analysis. This observation suggests that future research in alcohol service utilization should benefit immensely from not only the study of interrelationships between the domains of individual, organizational, and sociopolitical factors, but also from diversification in unit of analysis to encompass representative and comparable samples in the general population and in treatment.

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Correlates of Past-Year Status Among Treated and Untreated Persons With Former Alcohol Dependence: United States, 1992

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Past-year status was investigated in a sample of 4,585 adults with prior DSM-IV alcohol dependence. Those who had and had not received treatment for alcohol problems were compared in terms of past-year status and its correlates, to see if the experience of treatment samples would be reflective of the course of alcoholism in the general population. In the past year, 27.8% of the total sample met the criteria for alcohol abuse or dependence, 22.3% were abstinent, and 49.9% were drinkers who did not satisfy the criteria for either abuse or dependence. Persons who had been treated for alcohol problems were about twice as likely to be abstainers as those who had not been treated (38.8% vs. 16.4%), but only about half as likely to fall into the past-year category of drinking without abuse or dependence (28.0% vs. 57.8%). These differentials were of constant magnitude, regardless of the interval since the onset of dependence. For the sample as a whole, persons who had received treatment were slightly more likely than their untreated counterparts to have had alcohol abuse or dependence in the past year (33.2% vs. 25.8%), and this differential increased with the interval since the onset of dependence. The odds of both past-year abstinence and drinking without abuse or dependence were decreased by male gender, Black race, rapidity of the onset of dependence and ethanol intake per drinking day, and were increased by ever having been married and by later ages at onset of dependence. The odds of drinking without abuse or dependence relative to abstinence were increased by college attendance and reduced by the number of dependence symptoms, and having been a daily drinker was associated with increased odds of past-year abstinence. Treatment history modified the associations between past-year status and race, marital and educational status, number of past alcohol problems, and rapidity of onset of dependence and age at onset. These results suggest that treatment studies may not be generalizable to alcoholics who do not seek treatment.

Almost all of our knowledge concerning the course of alcoholism has come from studies of treatment samples in which the effects of either long-term treatment or brief interventions were evaluated. Treatment outcomes, often measured in terms of the proportion of patients remaining abstinent or the percentage reduction in intake or heavy drinking occasions, have varied markedly with factors, such as type, intensity and duration of treatment, availability of aftercare, and patient treatment matching.[1] Since the 1950s, a number of studies have documented varying levels of resumed nonproblem drinking in follow-up studies of treated alcoholics.[2–9] More recently, a few studies have evaluated treatment outcome within programs specifically designed to evaluate a return to moderate drinking as an alternative goal to that of abstinence.[10–15] Notwithstanding the controversy surrounding the issue of "controlled" drinking (usually understood to mean the return to moderate asymptomatic drinking by persons with former alcohol use disorders, a phenomenon that critics consider incompatible with the concept of alcoholism as an irreversible disease), our ability to draw conclusions from this body of work has been hampered by broad differences in the periods of follow-up and definitions of outcome status used in these studies.[2]

Despite the fact that many individuals with alcohol problems never enter the treatment system,[16] few studies have examined the course of alcohol dependence in the general population. Fillmore and Midanik[17] used follow-up data from both a community and a national sample to determine that chronicity of alcohol problems was greater among men who were older at the time of initial contact. Temple and Fillmore[18] examined changes over a 10-year period in the proportion of adolescents and young men who engaged in prob-

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lem drinking and found little continuity over time. A 40-year follow-up study of inner city youth conducted by Vaillant and Milofsky[19] found that of 110 identified as ever having shown signs of alcohol abuse, 38 were abstinent and 5 had resumed nonproblem drinking when last contacted. Öjesjö[20] used data from the prospective Norwegian Lundy study to follow the outcome of 96 men classified as alcoholics in 1957. After 15 years, 30% were classified as either abstinent or drinking without problems.

None of these general population studies examined the course of alcohol dependence per se. Identification of subjects for follow-up was based on individual problem indicators, counts of problems, and brief screening instruments. One reason why population studies have yielded so few data on the natural history of alcoholism is that measuring alcohol abuse and dependence in large-scale surveys is time consuming and expensive. Numerous symptom item indicators are required to operationalize the definitions of alcohol use disorders, and additional questions are necessary to identify age at onset of dependence.

Recently, a large-scale national survey of drinking practices and problems collected the data required to classify DSM–IV alcohol use disorders[21] within the past year (i.e., the year immediately before interview) and the period before the past year. The survey also collected information on the history of treatment for alcohol problems, thus permitting investigation of whether the course of alcoholism is the same for persons who do and do not enter the alcohol treatment system. Using these data, this analysis compared the distribution of past-year status—(1) abuse or dependence, (2) abstinence, or (3) drinking without abuse or dependence—for former alcoholics who did and did not receive treatment, taking into account duration since the onset of dependence. The characteristics of persons in the three categories of past-year status were compared for the total sample and among those who did and did not receive treatment, and multivariate models were used to identify correlates of past-year status. The models also assessed the extent to which these correlates were modified by receipt of treatment to see whether factors associated with outcome in treatment samples can be generalized to alcoholics who do not enter the treatment system.

METHODS

Sample

This study is based on data collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and conducted by the U.S. Bureau of the Census. This study gathered information on alcohol consumption and alcohol-related problems, depression and drug use, family history of alcoholism, history of alcohol and drug treatment, and assorted sociodemographic background variables. Data were obtained in personal interviews conducted in respondents' homes, and proxy respondents were not permitted. The NLAES sample was representative of U.S. adults 18 years of age and over, residing in households in the civilian, noninstitutionalized population. The household and sample person response rates were 92% and 97%, respectively, yielding a total sample size of 42,862. This analysis was based on a subsample of 4,585 individuals with prior-to-past-year alcohol dependence.

The complex, multistage NLAES sample design[22] featured selection of primary sampling units with probability proportional to size and oversampling of Blacks and young adults between the ages of 18 and 29. Because of these design features, standard errors associated with NLAES estimates are ~20% greater than those that would be obtained with a simple random sample of equal size. To incorporate these design effects adequately into variance estimation procedures, the estimates presented herein were generated by SUDAAN,[23] a software package that accommodates the design features of complex sample designs.

Measures

Prior-to-Past-Year Dependence. To be included in this analysis, an individual had to have satisfied the criteria for prior-to-past-year DSM–IV alcohol dependence by meeting at least 3 of the 7 DSM–IV criteria for dependence: tolerance; withdrawal (including relief or avoidance of withdrawal); persistent desire or unsuccessful attempts to cut down on or stop drinking; much time spent drinking, obtaining alcohol or recovering from its effects; reduction or cessation of important activities in favor of drinking; impaired control over drinking; and continued use despite physical or psychological problems caused by drinking. These criteria were operationalized by means of 25 symptom item indicators.[24] A criterion was satisfied if a person reported one or more positive symptoms of that criterion previous to the past year. To meet the syndromal definition of the withdrawal criterion, two or more positive symptoms were required. In addition, to satisfy the duration qualifiers for the period previous to the past year, the respondents had to report (in response to direct questions) that some of these symptoms occurred "at around the same time" or "at around the same time,

on and off for a few months or longer" or "at around the same time, most days for at least a month." In addition to establishing the duration criteria, a positive response to one of these questions ensured that the symptoms upon which the dependence classification was based were clustered at a period in time rather than occurring at different times over the life span. Age at onset of dependence was defined as the age when symptoms first began to co-occur as previously defined.

Past-Year Status. Individuals classified as having had an alcohol use disorder in the past year had to meet the criteria for either alcohol abuse or dependence during that period. A diagnosis of past-year dependence required satisfying at least three of the previously listed DSM–IV dependence criteria. A diagnosis of past-year abuse required satisfying one or more of the following criteria: continued use despite social or interpersonal consequences; hazardous use; alcohol-related legal problems; and neglect of role responsibilities in favor of drinking. Abuse and dependence criteria not associated with duration qualifiers were satisfied if a person reported one or more positive symptoms of the criterion during the past year, and criteria with duration qualifiers were satisfied if a person reported two or more symptoms of the criterion or one symptom that occurred at least two times during the past year. Individuals classified as abstinent in the past year reported an interval of 12 months or longer since their last drink. They were not asked the symptom item indicators for the past year. Individuals whose past-year status was classified as drinking without abuse or dependence reported an interval of <12 months since their last drink and failed to meet the criteria previously listed for either past-year abuse or dependence. Approximately one-fourth of the drinkers in this category reported drinking <12 drinks in the past year. Like the total abstainers, these individuals were not asked the symptom item indicators for past-year alcohol use disorders (which were asked only of respondents who consumed at least 12 drinks), but were assumed to have experienced no drinking-related problems.

Background and Drinking History Measures. Individuals were classified as having a positive family history of alcoholism if they reported that any of 18 different types of first- and second-degree biological relatives was an alcoholic or problem drinker, defined as "a person who has physical or emotional problems because of drinking; problems with a spouse, family or friends because of drinking; problems at work because of drinking; problems with the police because of drinking—like drunk driving; or a person who seems to spend a lot of time drinking or being hungover." To be classified as having had a history of prior-to-past-year DSM–IV major depression, an individual had to have reported five or more depressive symptoms during that period, inclusive of low mood or lack of interest, that occurred nearly every day for at least the same 2-week period, that co-occurred with social and/or occupational dysfunction, and that were not attributed to physical illness or bereavement.

Severity of dependence was estimated by a count of the number of DSM–IV dependence symptoms reported for the period previous to the past year. At least three were required for the diagnosis, and the maximum number was 25. Based on their importance in the treatment outcome literature, two specific problem indicators were included as well: history of withdrawal symptoms and impaired control, the latter defined as desire or unsuccessful attempts to stop drinking or drinking for longer or in greater quantity than intended.

Alcohol consumption measures were estimated for both the period of heaviest drinking and the past year. For each beverage type, ethanol intake per drinking day was estimated as the product of usual number of drinks per drinking day, usual drink size in ounces, and an ethanol conversion factor (0.045, 0.121, and 0.409, respectively, for beer, wine, and liquor).[25–29] Average daily intake was estimated by multiplying intake per drinking day times number of drinking days per year for each type of beverage, summing across beverages and dividing by 365. Frequency of drinking was based on categorical responses that were converted to number of days per year. For the period of heaviest drinking, this measure was dichotomized to daily versus nondaily drinking. Total body water, which mediates the blood alcohol level produced by a dose of ethanol, was estimated on the basis of sex, age, and self-reported height and weight.[30]

Individuals were counted as having received alcohol treatment if they reported ever having gone to any of 24 different treatment sources (including one open-ended) for problems relating to their own drinking. These sources included 12-step programs and others ranging from inpatient wards in general or psychiatric hospitals to half-way houses, employee assistance programs, and various types of doctors and health providers.

To improve goodness of fit and to satisfy the assumption of linearity, log transforms were applied to several of the continuous variables used in the multivariate models. These measures were number of symptoms, interval from first drink to onset of dependence, age at onset of dependence, interval since onset of dependence, and ounces of ethanol consumed per drinking day. Age at interview was not included as a model

covariate, because its value was measured implicitly by the combination of age at onset of dependence and the length of the interval since onset.

RESULTS

Among United States adults who had formerly met the DSM–IV criteria for alcohol dependence, 27.8% met the criteria for alcohol abuse or dependence in the past year, 22.3% were abstinent, and 49.9% no longer met the criteria for alcohol abuse or dependence despite having had one or more drinks. The distribution by past-year status varied according to the length of the interval since onset of dependence. The proportion of the formerly dependent with abuse or dependence in the past year declined steadily from 57.1% of those with an interval of <5 years since the onset of dependence to 12.4% of those with an interval of 20 years or more, and the proportion who were abstainers rose steadily from 6.6% to 35.5%. The proportion of the formerly dependent who were drinking without abuse or dependence in the past year was 36.3% among those whose dependence had begun in the 5 years preceding interview and slightly >50% among those with longer intervals since the onset of dependence.

Regardless of interval since the onset of dependence, past-year abstinence was about twice as common among persons who had been treated for alcohol problems, whereas drinking without abuse or dependence was approximately twice as common among the untreated. Those who had received treatment were somewhat more likely than the untreated to have had an alcohol use disorder in the past year, and this differential increased with interval since the onset of dependence.

Comparing individuals in the three endpoint categories, persons with past-year alcohol abuse or dependence were the youngest, the most likely to be male, the least likely ever to have been married, and had the shortest interval since the onset of dependence (Table 1). Past-year abstainers were the oldest and the most likely ever to have been married, to have a positive family history of alcoholism, and to have experienced an episode of DSM–IV major depression. Abstainers also reported the greatest number of alcohol-related problems, the oldest age at onset of dependence, the longest interval from first drink to onset of dependence, and the longest interval since the onset of dependence. Abstainers were twice as likely as persons in the other two categories to have been daily drinkers and were the most likely to have received all forms of alcohol treatment. Persons who drank without abuse or dependence in the past year were the least likely to be male or to be Black, were the most likely to have attended college, reported the fewest alcohol-related problems and the lowest level of ethanol intake during their period of heaviest consumption, and were the least likely to have received alcohol treatment.

Table 1. Percentage Distribution of Adults 18 Years of Age and Over with Prior-to-Past Year DSM–IV Alcohol Dependence by Past Year Status, According to Interval Since the Onset of Dependence and Whether Ever Received Treatment for Alcohol Problems

Interval since onset of dependence	*n*	Past-year status: Alcohol abuse or dependence	Past-year status: Abstinent	Past-year status: Drinking without abuse or dependence
Total sample				
<5 years	634	57.1 (2.4)	6.6 (1.1)	36.3 (2.2)
5–<10 years	1,060	34.5 (2.9)	15.0 (1.3)	50.5 (1.9)
10–<20 years	1,664	21.5 (1.2)	24.3 (1.2)	54.2 (1.4)
20 or more years	1,137	12.4 (1.2)	35.5 (1.7)	52.1 (1.7)
Total (all intervals)	4,585	27.8 (0.8)	22.3 (0.7)	49.9 (0.8)
Ever received treatment				
<5 years	139	69.9 (4.4)	11.1 (2.8)	19.0 (3.6)
5–<10 years	296	38.2 (3.3)	29.6 (3.1)	32.2 (3.2)
10–<20 years	489	25.6 (2.2)	44.4 (2.5)	30.0 (2.4)
20 or more years	284	20.4 (2.8)	55.4 (3.2)	24.2 (2.9)
Total (all intervals)	1,233	33.2 (1.6)	38.8 (1.6)	28.0 (1.5)
Never received treatment				
<5 years	439	53.3 (2.8)	5.3 (1.1)	41.4 (2.7)
5–<10 years	757	32.8 (2.3)	9.4 (1.3)	57.8 (2.2)
10–<20 years	1,160	19.8 (1.4)	15.9 (1.3)	64.3 (1.7)
20 or more years	840	9.9 (1.3)	29.9 (1.8)	60.2 (1.9)
Total (all intervals)	3,309	25.8 (1.0)	16.4 (0.8)	57.8 (1.0)

Note: Numbers in parentheses are standard errors of estimates. Totals for all intervals include persons with unknown length of interval since the onset of dependence. Total sample figures include persons unknown as to whether ever received treatment.

Past-year consumption levels were consistent with diagnostic status. Persons with past-year alcohol abuse or dependence had by far the highest levels of consumption: an average daily ethanol intake of 2.3 oz, 86.5 days of drinking 5+ drinks, and 41.4 occasions of intoxication. For drinkers without abuse or dependence, the past-year consumption estimates were considerably lower (average daily intake of 0.5 oz, 14.4 days of drinking 5+ drinks, and 4.0 occasions of intoxication) and were almost identical to those for persons who had never been diagnosed with alcohol dependence [average daily intake of 0.6 oz of ethanol, 14.7 days of drinking 5+ drinks, and 3.7 occasions of intoxication (data not shown)]. Data in Table 2 for past-year drinkers with neither abuse nor dependence were based on persons who drank at least 12 drinks in the 12 months preceding interview, because the NLAES

questionnaire did not ask lighter drinkers about their past-year consumption. Imputing values reflecting six drinks per year and no occasions of heavy drinking or intoxication for these light drinkers would result in the following values for the category as a whole: an average daily intake of 0.4 oz of ethanol, 10.7 days of drinking 5+ drinks, and 3.0 occasions of intoxication.

As shown in Table 3, the characteristics of persons who had received treatment for alcohol problems differed somewhat from the characteristics of those who had not received treatment, both in terms of their absolute levels and in terms of how they varied with respect to past-year status. For example, regardless of past-year status, persons who had received treatment were more likely to be male, to have had a positive family history of alcoholism, and to have had an episode of major depression than persons who had not received treatment. The treated also reported more alcohol-related problems, greater quantities of ethanol intake, and were more likely to have been daily drinkers.

The associations of past-year status with race, education, and history of major depression differed for the treated and untreated. Among those who had received treatment, the proportion of Blacks was lowest among abstainers; among those who had not received treatment the opposite was true: the proportion of Blacks was highest among abstainers. Among the treated, the percentage of persons who had attended college was highest among abstainers and lowest among those with a past-year alcohol use disorder. In contrast, among the untreated, the percentage who had attended college was highest for drinkers without disorder and lowest for abstainers. Within the treated population, the proportion of persons with a history of major depression was greatest among past-year abstainers; within the untreated population, history of depression did not have a statistically significant association with past-year status.

To adjust for possible interrelationships among the correlates of past-year status and to test for the statistical significance of treatment as an effect modifier, multiple logistic regression models were estimated to contrast three pairs of past-year status categories: (1) abstinence versus abuse or dependence (excluding persons who drank without abuse or dependence), (2) drinking without abuse or dependence versus having had abuse or dependence (excluding abstainers), and (3) drinking without abuse or dependence versus abstinence (excluding persons with abuse or dependence). The results of these models are shown in Table 4, and

Table 2. Selected Characteristics of Adults 18 Years of Age and Over with Prior-to-Past-Year DSM–IV Alcohol Dependence, According to Past-Year Status

Characteristic	Past-year status		
	Alcohol abuse or dependence	Abstinent	Drinking without abuse or dependence
Mean age at interview	31.5 (0.4)	44.9 (0.5)	36.9 (0.3)
% male	75.6 (1.5)	69.6 (1.5)	62.4 (1.1)
% Black	7.7 (0.9)	8.8 (0.9)	5.2 (0.6)
% ever married	56.5 (1.9)	88.8 (1.0)	78.9 (1.0)
% who attended college	50.6 (1.8)	43.5 (1.7)	62.9 (1.1)
% with family history of alcoholism	75.8 (1.6)	82.6 (1.3)	74.1 (1.0)
% with history of depression	36.2 (1.6)	43.3 (1.8)	38.3 (1.2)
Mean number of alcohol-related problems	9.6 (0.1)	12.8 (0.2)	8.2 (0.1)
% reporting withdrawal symptoms	90.3 (1.0)	93.9 (1.0)	90.4 (0.7)
% reporting impaired control	98.0 (0.5)	98.5 (0.4)	99.4 (0.2)
Mean years from first drink to onset of dependence	5.4 (0.2)	8.1 (0.3)	4.8 (0.1)
Mean age at onset of dependence	21.8 (0.2)	25.8 (0.3)	22.1 (0.2)
Mean years since the onset of dependence	9.6 (0.3)	18.9 (0.4)	14.6 (0.2)
Consumption during period of heaviest drinking			
Mean ounces of ethanol consumed per drinking occasion	7.9 (0.5)	8.0 (0.3)	5.5 (0.1)
% who were daily drinkers	27.8 (1.6)	52.7 (1.9)	25.0 (0.9)
Consumption during past year			
Mean daily ethanol intake (oz)	2.3 (0.1)	0.0 (0.0)	0.5 (0.02)
Mean days when consumed 5+ drinks	86.5 (3.5)	0.0 (0.0)	14.4 (1.2)
Mean days when intoxicated	41.4 (2.3)	0.0 (0.0)	4.0 (0.5)
% who ever received treatment			
Of any type	31.8 (1.6)	46.0 (1.8)	14.9 (0.9)
12-step program	23.7 (1.4)	39.7 (1.8)	9.8 (0.7)
Other treatment (inpatient, outpatient, etc.)	24.2 (1.4)	38.3 (1.8)	10.3 (0.7)
Starting 0–5 years after onset of dependence	21.2 (1.4)	23.3 (1.5)	9.3 (0.7)
Starting 6+ years after onset of dependence	9.9 (0.9)	21.6 (1.5)	4.5 (0.5)

their associated odds ratios are presented in Table 5. The purpose of this analysis was to determine whether factors associated with the course of alcoholism differed among treated and untreated individuals and was not to evaluate the effect of treatment. Accordingly Table 5 does not show odds ratios for treatment per se (although they can be estimated from Table 4), but rather presents odds ratios for the other correlates among the treated and the untreated. For those variables that demonstrated a significant interaction with treatment history, the odds ratios differ for the treated and the untreated; identical odds ratios indicate lack of interaction.

The odds of abstinence as opposed to abuse or dependence were reduced by male gender, increased with age at onset of dependence, decreased with quantity of ethanol intake, and were increased by having been a daily drinker. Among the untreated only, the odds of abstinence versus abuse or dependence were increased by having been married, were reduced by having attended college, and were inversely related to the length of the interval from first drink to onset of dependence. Among the treated only, severity (number of past symptoms) was positively associated with this outcome.

The odds of drinking without abuse or dependence as opposed to having experienced abuse or dependence in the past year were reduced by male gender, were increased by having been married, having attended college, having a history of major depression and a history

Table 3. Selected Characteristics of Adults 18 Years of Age and Over with Prior-to-Past-Year DSM–IV Alcohol Dependence, According to Past-Year Status and Whether Ever Treated for Alcohol Problems, Standardized for Interval Since the Onset of Dependence

Characteristic	Past-year status		
	Alcohol abuse or dependence	Abstinent	Drinking without abuse or dependence
Ever treated for alcohol problems			
Mean age at interview	36.0 (0.5)	40.8 (0.6)	37.4 (0.6)
% male	82.2 (2.5)	71.9 (2.7)	71.6 (2.8)
% Black	8.1 (1.6)	4.8 (1.0)	7.2 (1.6)
% ever married	67.8 (1.9)	81.6 (2.4)	77.9 (2.3)
% who attended college	39.9 (3.0)	53.5 (3.0)	48.6 (3.0)
% with family history of alcoholism	83.2 (2.3)	85.7 (2.4)	82.3 (2.7)
% with history of depression	40.1 (3.1)	54.4 (3.0)	45.0 (3.2)
Mean number of alcohol-related problems	12.2 (0.3)	16.4 (0.3)	11.5 (0.3)
% reporting withdrawal symptoms	90.9 (1.7)	96.3 (1.7)	90.0 (1.9)
% reporting impaired control	96.6 (0.9)	98.8 (0.6)	99.3 (0.5)
Mean years from first drink to onset of dependence	6.5 (0.3)	10.2 (0.5)	6.7 (0.4)
Mean age at onset of dependence	22.3 (0.4)	26.8 (0.6)	23.2 (0.5)
Consumption during period of heaviest drinking			
Mean ounces of ethanol consumed per drinking occasion	9.8 (1.0)	9.7 (0.6)	7.4 (0.4)
% who were daily drinkers	43.1 (3.0)	66.6 (2.7)	43.3 (3.0)
Consumption during past year			
Mean daily ethanol intake (oz)	4.1 (0.6)	0.0 (0.0)	0.7 (0.1)
Mean days when consumed 5+ drinks	133.3 (8.2)	0.0 (0.0)	24.6 (4.5)
Mean days when intoxicated	71.8 (6.4)	0.0 (0.0)	8.1 (1.7)
Never treated for alcohol problems			
Mean age at interview	35.0 (0.5)	40.1 (0.6)	36.1 (0.2)
% male	74.5 (2.0)	61.8 (2.9)	60.1 (1.2)
% Black	8.0 (1.2)	9.6 (1.3)	8.0 (1.2)
% ever married	65.7 (1.8)	87.1 (2.1)	77.0 (1.0)
% who attended college	55.0 (2.8)	39.8 (2.9)	64.8 (1.3)
% with family history of alcoholism	73.2 (2.3)	76.9 (2.5)	73.0 (1.1)
% with history of depression	32.7 (2.1)	37.7 (2.9)	37.7 (1.3)
Mean number of alcohol-related problems	8.8 (0.2)	9.4 (0.3)	7.6 (0.1)
% reporting withdrawal symptoms	91.9 (1.2)	89.2 (2.1)	90.4 (0.8)
% reporting impaired control	98.5 (0.8)	98.3 (0.7)	99.4 (0.2)
Mean years from first drink to onset of dependence	5.4 (0.2)	8.1 (0.3)	4.8 (0.1)
Mean age at onset of dependence	21.4 (0.3)	24.9 (0.6)	22.0 (0.2)
Consumption during period of heaviest drinking			
Mean ounces of ethanol consumed per drinking occasion	7.7 (0.7)	5.8 (0.5)	5.0 (0.2)
% who were daily drinkers	25.4 (2.3)	33.6 (2.8)	21.4 (1.0)
Consumption during past year			
Mean daily ethanol intake (oz)	1.8 (0.1)	0.0 (0.0)	0.5 (0.02)
Mean days when consumed 5+ drinks	64.4 (4.3)	0.0 (0.0)	12.5 (1.1)
Mean days when intoxicated	71.8 (6.4)	0.0 (0.0)	8.1 (1.7)

of impaired control, were inversely related to interval from first drink to dependence and quantity of consumption, and were positively related to age at onset of dependence. Among the untreated only, the odds of this outcome decreased with severity (i.e., number of symptoms) of past dependence.

The odds of drinking without abuse or dependence relative to abstinence were increased by a history of impaired control, were inversely related to age at onset of dependence, and were reduced by having been a daily drinker. Among the untreated only, the odds of this outcome relative to abstinence were reduced by Black race and having been married and were increased by having attended college. Severity was inversely related to the odds of drinking without abuse or dependence versus abstinence for both treated and untreated individuals, but the reduction in odds was consistently greater (i.e., the odds ratios were lower) among the former.

DISCUSSION

This study of the adult population of the United States found that both the natural history of alcoholism and its correlates varied for persons who did and did not receive treatment for alcohol-related problems. These results suggest that studies of treatment outcome based on clinical samples may not accurately reflect the course of alcohol dependence among persons who do not elect to enter treatment. Although prospective studies are needed to clarify the patterns of onset, chronicity, and remission of alcohol dependence in the general population, the findings of this study may provide some clues as to the progression of this disorder once the biases associated with its design are taken into account.

One source of bias reflects the varying lengths of observation associated with retrospective, cross-sectional data. In this analysis, the interval between the past-year endpoint and onset of dependence was less than a year for some respondents; for others, it exceeded 20 years. Although data appear to simulate the progression through time of a single cohort, they differ from longitudinal data in that they are based on a sample of survivors and thus do not represent former alcoholics who had died or entered institutions. The proportion of persons with past-year abuse or dependence may have been increasingly underrepresented over increasing intervals since the onset of dependence, if individuals with continued alcohol problems were the most likely to have died or become institutionalized.

Table 4. Multiple Logistic Regression Models Predicting Odds of Selected Categories of Past-Year Status for Adults 18 Years of Age and Over with Prior-to-Past-Year DSM–IV Alcohol Dependence

	Abstinence vs. alcohol abuse or dependence			Drinking without abuse or dependence vs. alcohol abuse or dependence			Drinking without abuse or dependence vs. abstinence		
	β	SE	p	β	SE	p	β	SE	p
Intercept	−8.029	(1.403)	<0.001	−2.329	(1.008)	0.024	5.939	(1.175)	<0.001
Main effects									
Male	−0.605	(0.244)	<0.001	−0.762	(0.188)	<0.001	−0.357	(0.191)	0.066
Black	−0.024	(0.193)	0.899	−0.399	(0.204)	0.055	−0.887	(0.208)	<0.001
Ever married	1.189	(0.207)	<0.001	0.685	(0.111)	<0.001	−0.462	(0.188)	0.017
Attended college	−0.594	(0.172)	0.001	0.222	(0.105)	0.038	0.968	(0.127)	<0.001
Positive family history of alcoholism	−0.021	(0.172)	0.904	−0.056	(0.123)	0.650	−0.164	(0.130)	0.211
Prior-to-past-year DSM-IV major depression	0.224	(0.141)	0.116	0.250	(0.097)	0.013	0.068	(0.117)	0.560
No. of prior-to-past-year dependence symptoms (log)	0.122	(0.189)	0.520	−0.988	(0.152)	<0.001	−0.958	(0.163)	<0.001
Ever experienced withdrawal symptoms	−0.072	(0.264)	0.786	0.168	(0.168)	0.321	0.270	(0.233)	0.250
Ever experienced impaired control	−0.151	(0.638)	0.813	1.095	(0.462)	0.021	0.946	(0.464)	0.045
Years from first drink to onset of dependence (log)	−0.216	(0.091)	0.019	−0.171	(0.063)	0.008	0.113	(0.066)	0.091
Age at onset of dependence (log)	1.499	(0.314)	<0.001	0.715	(0.264)	0.008	−0.993	(0.262)	<0.001
Years since the onset of dependence (log)	1.174	(0.097)	<0.001	0.706	(0.063)	<0.001	−0.555	(0.084)	<0.001
Total body water	0.011	(0.014)	0.417	0.017	(0.011)	0.133	0.018	(0.011)	0.118
Ounces of ethanol intake per drinking occasion (log)	−0.336	(0.084)	<0.001	−0.282	(0.067)	<0.001	0.127	(0.070)	0.072
Daily drinker	0.401	(0.149)	0.009	0.052	(0.121)	0.672	−0.301	(0.115)	0.011
Ever treated for alcohol problems	−5.023	(0.828)	<0.001	−1.862	(0.607)	0.003	2.695	(0.710)	<0.001
Interaction terms									
Ever treated × Black	—	—	—	—	—	—	1.245	(0.446)	0.007
Ever treated × ever married	−0.854	(0.313)	0.008	—	—	—	0.715	(0.294)	0.018
Ever treated × attended college	0.982	(0.281)	<0.001	—	—	—	−0.960	(0.222)	<0.001
Ever treated × number of prior-to-past-year dependence symptoms (log)	2.091	(0.115)	0.013	0.593	(0.263)	0.027	−1.374	(0.259)	<0.001
Ever treated × years from first drink to onset of dependence (log)	0.293	(0.115)	0.013	—	—	—	−0.266	(0.094)	0.007
Goodness of fit									
Satterthswaite adjusted *F*	18.3; *df* = 18,68; *p* < .001			25.5; *df* = 16,68; *p* < .001			24.2; *df* = 20,68; *p* < .001		

Retrospective data also are subject to recall bias and deliberate misreporting. Arguably, both the extent to which prior alcohol dependence is reported at all and the accuracy with which its onset is recalled may diminish over time. If reporting errors were independent of past-year status, they would not bias the estimates of its distribution; however, there are many ways in which the two could be associated. For example, previous alcohol problems might be more salient to those who were still dependent or had stopped drinking. If persons who had progressed to drinking without abuse or dependence were less likely to recall the full extent of their past problems, they might have failed to meet the criteria for prior-to-past-year dependence (false-negatives), resulting in their exclusion from the analysis and in underestimation of the prevalence of past-year drinking without abuse or dependence. Alternatively, the magnitude of this category could have been overestimated if recall bias led to false coalescence of prior-to-past-year symptoms (false-positives), resulting in the analysis having included persons who never truly met the criteria for dependence.

Although retrospective data are especially vulnerable to biases such as these, even survival analyses of prospective data make the assumption that survival is independent of outcome, and no survey that relies on self-report is immune from the effects of recall bias. The diagnostic and consumption measures used in this analysis were evaluated in a test-retest study conducted in a large community sample.[31] Both the diagnostic and consumption measures showed high levels of reliability, with kappas and intraclass correlation coefficients that were generally in the range of 0.70 to 0.80. Thus, whereas the limitations associated with this study design need to be considered in interpreting its findings, they should not be overestimated and would not have been likely to result in gross distortion of the distribution of past-year status.

This study found that half of the individuals who had formerly met the DSM–IV criteria for alcohol dependence were drinking without abuse or dependence in the past year. There are several reasons why the level of past-year drinking without disorder exceeded the

Table 5. Odds Ratios for Selected Characteristics and Categories of Past-Year Status, for Treated and Untreated Adults 18 Years of Age and Over with Prior-to-Past-Year DSM–IV Alcohol Dependence

	Abstinence vs. alcohol abuse or dependence		Drinking without abuse or dependence vs. alcohol abuse or dependence		Drinking without abuse or dependence vs. abstinence	
	Treated	Untreated	Treated	Untreated	Treated	Untreated
Male	0.55	0.55	0.47	0.47	NS	NS
Black	NS	NS	NS	NS	NS	0.41
Ever married	NS	3.28	1.98	1.98	NS	0.63
Attended college	NS	0.55	1.25	1.25	NS	2.63
Positive family history of alcoholism	NS	NS	NS	NS	NS	NS
Prior-to-past-year DSM-IV major depression	NS	NS	1.28	1.28	NS	NS
No. of prior-to-past-year dependence symptoms*						
5	3.10	NS	NS	0.60	0.30	0.61
10	14.36	NS	NS	0.30	0.06	0.32
15	35.23	NS	NS	0.20	0.02	0.21
Ever experienced withdrawal symptoms	NS	NS	NS	NS	NS	NS
Ever experienced impaired control	NS	NS	2.99	2.99	2.58	2.58
Years from first drink to onset of dependence†						
2	NS	0.74	0.79	0.79	NS	NS
5	NS	0.61	0.67	0.67	NS	NS
10	NS	0.52	0.60	0.60	NS	NS
Age at onset of dependence‡						
18	1.31	1.31	1.14	1.14	0.83	0.83
21	1.66	1.66	1.27	1.27	0.72	0.72
25	2.15	2.15	1.44	1.44	0.60	0.60
Ounces of ethanol intake per drinking occasion§						
2	0.79	0.79	0.82	0.82	NS	NS
4	0.63	0.63	0.68	0.68	NS	NS
6	0.55	0.55	0.60	0.60	NS	NS
Daily drinker	1.49	1.49	NS	NS	0.74	0.74

NS, not significant.

* Reference group consists of persons with three symptoms, the minimum required for prior-to-past-year dependence.

† Reference group consists of persons with <1 year from first drink to onset of dependence.

‡ Reference group consists of persons with onset of dependence at age 15.

§ Reference group consists of persons who consumed an average of 1.0 oz of ethanol (2 drinks) per drinking occasion.

level of controlled drinking reported in previous studies. Most importantly, the category of drinking without abuse or dependence was less conservative than most definitions of controlled drinking, which usually exclude individuals on the basis of heavy drinking, intoxication, and/or alcohol-related problems. Use of the outcome categories recommended by Heather and Tebbutt[3] would redistribute the category of past-year drinkers without abuse or dependence in the following manner: 13% of the sample (8% of those who had received treatment and 14% of those who had not received treatment) would fall into the category of partial abstainers (i.e., persons who had consumed <1 drink per month). Eleven percent (4% of the treated and 13% of the untreated) would fall into the category of nonproblem controlled drinkers, comprised of individuals with no symptoms of drinking problems over the past year and no instances of intoxication. This figure lies within the range reported as the prevalence of controlled drinking by the studies cited previously. Seven percent of this sample (5% of the treated and 7% of the untreated) would be classified as nonproblem, mostly controlled drinkers (asymptomatic), but with 1 to 11 instances of intoxication, and less than one-half of 1% of both the treated and the untreated would be classified as nonproblem, uncontrolled drinkers with no symptoms, but 12 or more instances of intoxication. Finally, 20% (11% of the treated and 23% of the untreated) would fall into the category of problem drinkers, much improved. This would include individuals with one or more symptoms of alcohol-related problems, but an insufficient number of symptoms to meet the criteria for abuse or dependence.

Two other factors also help to account for the unusually high prevalence of drinking without abuse or dependence. First, this study was based on the general population, comprised of individuals whose alcohol problems were less severe and thus more amenable to continued drinking than those in treatment samples. Second, age at onset of dependence was very young within this general population sample. Whereas the mean ages at onset varied from the early to mid-twenties, depending on past-year status, nearly half of the sample reported onset of dependence before age 21. Thus, the findings of this study may be viewed as consistent with longitudinal studies by Fillmore and Midanik[17] and by Temple and Fillmore,[18] which found a lack of continuity in young men's drinking problems and a high level of return to nonproblematic drinking, and with the argument that youthful drinking problems may resolve themselves with increasing age and maturity.[32]

This study supported previous research findings indicating that attendance at Alcoholics Anonymous was strongly predictive of abstinence.[8,11] It also supported earlier studies which found that successful resumed drinking was more common among former alcoholics and problem drinkers with lower levels of severity at outset (including fewer physiological symptoms and less loss of control), those with lower levels of intake, and those who perceived their problems to be of relatively short duration.[6,33] In this analysis, though, it must be recognized that neither the consumption measures nor the number of symptoms was measured at outset (i.e., as of the onset of dependence). Three quarters of the respondents reported that their period of heaviest drinking began at an age that preceded or equaled age at the onset of dependence; the remainder reported drinking most heavily after becoming dependent. Thus, individuals whose dependence continued unabated for a relatively long period had more time in which to drink heavily and in which to develop alcohol-related problems, clouding the relationship of these variables with past-year status.

This study's finding that daily drinking was positively associated with the outcome of abstinence differed from a previous study by Elal-Lawrence et al.[11] In that study, which was based on a follow-up of patients offered an option of treatment goals, continuous drinking was associated with the outcome of controlled drinking, whereas binge drinking was associated with the outcome of abstinence. However, the authors stated that, after adjusting for the different male/female compositions of their outcome groups, the differences with respect to daily drinking were not statistically significant. The present study did not adjust directly for age, because it was indirectly measured through length of interval since the onset of dependence. Because daily drinking is more typical of older than younger drinkers, additional studies that directly address the issues of age and/or cohort effects would help to clarify the association between frequency of drinking and the course of alcohol dependence.

Although some previous studies have found that younger age was positively associated with controlled drinking,[6] other sociodemographic factors generally have not been correlated with outcome. This study found that male gender was negatively associated with both past-year abstinence and drinking without disorder, even after adjusting for past level of consumption, number of alcohol problems, and history of depression. This finding could reflect greater sensitivity to and/or reporting of symptoms among women, which would result in an inability to adjust adequately for

severity even through use of multivariate techniques. Also, adjustment for history of depression would not necessarily control for differences in treatment for depression. If women with a history of major depression were more likely than men to have been treated for that disorder, this could have resulted in a greater reduction in secondary alcohol dependence among women.

One drawback of this analysis was the lack of variables measuring prior-to-past-year social status and functioning (e.g., employment status and income). The NLAES included measures of variables such as these, but only as of the time of interview. Although college education and ever having been married were included in this analysis, even these may be mistimed with respect to the period of risk (i.e., they may reflect effects of abstinence or other categories of past-year status rather than predicting them). Any true understanding of how the factors such as the presence of or separation from a spouse, level of social interaction, and occupational status may contribute to or impede the course of recovery from alcohol dependence requires dynamic measures of both social and economic characteristics and alcohol problems.

In summary, the results of this study underscore the need for additional research examining the natural history of alcohol dependence among persons who never enter or delay entering the alcohol treatment system. This is a dynamic process that can only be roughly approximated by data from two points in time. Vaillant's recent follow-up of his original community sample[34] provides valuable insights into changes in drinking patterns over the recovery period, leading him to propose definitions of abstinence and asymptomatic drinking that incorporate the element of continuity. Additional longitudinal studies based on general population samples could help to clarify further the behavioral changes that enable some formerly dependent drinkers to reduce their alcohol consumption without total abstinence to a level that is relatively free of problems, could document how sustainable such changes are, and could explore factors in the social environment that provide an alternative structure of support and incentive to that usually obtained through clinical and maintenance programs.

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Gender Differences in the Probability of Alcohol Treatment

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Interview data from 7,359 adults 18 years of age and over who met the DSM–IV criteria for alcohol abuse or dependence at some point during their lives revealed that 23.0% of the men and 15.1% of the women ever received treatment for alcohol problems. The median interval from onset of disorder to first treatment was between 2 and 3 years longer for men than women. This difference did not result from women being more likely than men to initiate treatment shortly after onset of an alcohol use disorder, but rather from men being more likely than women to initiate treatment in the period well after onset. Excluding treatment initiated prior to the clinical onset of the disorders or after cessation of drinking, men's and women's cumulative conditional probabilities of having initiated treatment by 30 years after onset of alcohol abuse or dependence were .424 and .356, respectively. Within the first 8 years after onset of abuse or dependence, men's and women's probabilities of initiating treatment were about the same, but men were 13% to 20% more likely to initiate treatment in the period from 8 to 25 years after onset. Use of proportional hazards models to adjust for factors including sociodemographic characteristics, prior consumption, severity of disorder, and comorbid drug use and depression revealed that men's and women's likelihoods of ever having received treatment did not differ for the most severely affected, those with 20 or more symptoms of abuse or dependence. Among those less severely affected, the male-to-female ratio in the likelihood of treatment declined with severity from 1.75 (1 symptom) to 1.24 (15 symptoms).

Most studies of gender differences in alcohol treatment utilization fall into two categories, those based on clinical or treatment samples and those based on samples of the general population or that incorporate a general population comparison group. Studies of the former type have provided comparative data that underscore many of the differences between men and women in treatment. For example, they have revealed that women generally enter treatment sooner after the onset of drinking or of alcohol-related problems than do men. Despite this and despite women's lower self-reported levels of alcohol intake, their problem symptoms upon presentation are equally as severe as those of men, leading to the hypothesis of "telescoping" or the more rapid development of alcohol use disorders in women (Hasin, Grant, & Weinflash, 1988; Lisansky, 1957; Piazza, Vrbka, & Yeager, 1989; Ross, 1989). Other differences between men and women in treatment include different factors influencing entry into treatment, with social, job, and legal consequences less important among women (Thom, 1987; Weisner, 1990), more attendant marital disruption and more alcoholic partners among women (Blume, 1986; Glatt, 1961; Gomberg, 1991; Hesselbrock et al., 1984; Rathod & Thomson, 1971), a higher level of emotional distress and comorbid psychiatric symptoms among women (Blume, 1986; Ojehagen, Berglund, Appel, Nilsson, & Skjaerris, 1990; Wallen, 1992), and a greater level of victimization (violence and sexual abuse) among women (Thom, 1987; Wallen, 1992). Data such as these are valuable in structuring treatment programs to meet the possibly disparate needs of male and female clients and in providing a basis for understanding gender differences in treatment outcome. However, because clinical samples contain no information on persons *not* entering treatment, they cannot be used to compare men's and women's rates of entry into treatment or to identify factors associated with differential utilization of treatment services.

Gender differences in treatment utilization have been inferred primarily from differences between the gender ratios of the prevalence of alcohol use disorders in the general population and the gender ratios of treatment populations. The most recent nationally representative prevalence data indicated that the combined rates of past-year DSM–IV alcohol abuse and dependence (American Psychiatric Association, 1994) were 11.0% for men and 4.1% for women (Grant,

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Harford et al., 1994), yielding a male-to-female ratio of 2.7 to 1.0. This is similar to the male-to-female ratio of individuals receiving treatment for alcohol problems in alcohol or drug treatment and prevention centers. In 1991, the proportions of male and female clients receiving treatment for alcohol problems alone were 76.7% and 23.3%, respectively, a ratio of 3.3 to 1.0. The proportions receiving treatment for combined alcohol and drug problems were 72.5% and 27.5%, respectively, a ratio of 2.6 to 1.0. The overall proportion of women in treatment for either drug or alcohol problems rose from 27.5% in 1991 to 28.9% in 1992, and presumably the proportion of women among alcohol treatment clients rose as well (U.S. Department of Health and Human Services, 1993, 1995). These figures suggest that men and women with alcohol use disorders are about equally likely to receive treatment.

A limited number of studies have directly examined gender differences in treatment utilization within the general population. In a population-based study of individuals who met the criteria for past-year DSM–IV alcohol use disorders, Grant (1996) did not find any significant effect of gender on the odds of having been treated for alcohol problems in the past year. However, that study controlled for prior utilization of alcohol treatment services, which may have subsumed any underlying gender differences in ever having initiated treatment. Weisner, Greenfield, and Room (1995), using a combined sample of data from national surveys conducted in 1979, 1984, and 1990, found that the odds of having ever received help for alcohol problems were 4.17 times higher for men than women after adjusting for survey year and sociodemographic characteristics. Controlling for dependence symptoms and social consequences reduced this odds ratio to 2.01. In a study based on samples of both the general population and clinic patients in a Northern California county who were interviewed between 1988 and 1989, Weisner and Schmidt (1992) found a male-to-female ratio of 3.3 to 1.0 in the lifetime prevalence of treatment for alcohol problems in the general population. However, this study did not adjust for gender differences in the prevalence of alcohol use disorders (52% of the men but only 43% of the women reported two or more dependence symptoms). The male-to-female treatment ratios reported for samples of patients in mental and primary health care settings were lower than that for the general population, leading the authors to conclude that women may seek alcohol treatment in a wider range of settings than men. In a separate analysis of data from the same community sample, Weisner (1993) found that the factors that discriminated between the treated and untreated groups differed for men and women, suggesting that the underlying effect of gender as a predictor of treatment utilization may be modified by other factors including sociodemographic characteristics, social consequences, and history of treatment for other types of problems.

Ideally, studies that compare men and women in terms of alcohol treatment utilization should control for differences in the need for treatment, for example, differences in severity or duration of alcohol-related problems. Restricting the analysis to persons with alcohol use disorders or controlling for symptoms of those disorders partially ensures comparability, but these measures do not account for possible gender differences in having stopped drinking or otherwise having experienced remission from the disorders in question, events which may be considered as obviating the need for initiation of treatment. In a study of past-year status among adults who had ever met the DSM–IV criteria for alcohol dependence, Dawson (1996) found that only 28% were still classified with either dependence or abuse in the year preceding interview. Male gender significantly reduced the odds of having neither abuse nor dependence in the past year. Notably, only a small minority of the persons who had achieved remission reported having received treatment.

This study used survival techniques to ensure that treatment rates were estimated for men and women who were comparable in terms of their need for treatment. The analysis was based on a national sample of U.S. adults identified as ever having met the DSM–IV criteria for alcohol abuse or dependence. Survival from onset of the disorder to initiation of treatment was compared for men and women, and individuals were withdrawn from the risk of initiating treatment either at the time of remission (when they stopped drinking or no longer met the criteria for abuse or dependence despite continued drinking) or as of the date of interview (beyond which data on initiation of treatment were not available). Proportional hazards models were used to compare men's and women's likelihoods of initiating alcohol treatment after adjusting for sociodemographic characteristics, severity and timing of disorder, consumption level, and comorbid conditions that might have affected entry into the treatment system.

METHOD

SAMPLE

The data used in this analysis were drawn from the 1992 National Longitudinal Alcohol Epidemiologic

Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism. The NLAES sample consisted of 42,862 adults 18 years of age and over who were selected at random from a nationally representative sample of households. This analysis was based on a subsample of 7,359 respondents who met the criteria for lifetime DSM–IV alcohol abuse and/or dependence. Interviewers hired and trained by the U.S. Bureau of the Census collected the NLAES data in personal interviews conducted in respondents' homes. Proxy respondents were not permitted. The household and sample person response rates were 92% and 97%, respectively.

The NLAES sample was similar in design to the 1985 redesign of the National Health Interview Study (Massey, Moore, Parsons, & Tadros, 1989), featuring first-stage sampling of primary sampling units (PSUs) with probability proportional to size and oversampling of blacks. Unlike the NHIS, the NLAES also oversampled young adults between the ages of 18 and 29 at the household level (Grant, Peterson, Dawson, & Chou, 1994) in order to ensure an adequate sample size for this subset of the population which is most at risk of heavy drinking and alcohol use disorders. Because of the complex, multistage sampling design of the NLAES, estimates derived from NLAES data have larger standard errors than estimates that would be derived from a simple random sample of equal size. Accordingly, all estimates presented in this article were produced using SUDAAN (Research Triangle Institute, 1995), a software package that uses first-order Taylor series linearization to accurately estimate variance under the conditions of a complex sampling design.

MEASURES

Alcohol use disorders

The measure of lifetime DSM–IV alcohol abuse and/or dependence, which has been fully described elsewhere (Grant, Hasin, & Dawson, 1996), was derived from separate estimates of these disorders for two distinct time periods, the past year and prior to the past year. Within each period, a person was classified as (a) dependent if he or she met at least three of the seven criteria for dependence (tolerance, withdrawal, desire or attempts to cut down on or stop drinking, much time spent on alcohol-related activities, reduction/cessation of important activities in favor of drinking, impaired control, and continued use despite physical or psychological problems) and (b) an abuser if he or she met one or more of the four criteria for abuse (continued use despite social or interpersonal consequences, hazardous use, alcohol-related legal problems, and neglect of role responsibilities in favor of drinking). For the past year period, criteria not associated with duration qualifiers were satisfied if a person reported one or more positive symptoms. To satisfy a criterion associated with a duration qualifier, a person had to report two or more symptoms of the criterion or one symptom that occurred two or more times. To satisfy the DSM–IV definition of withdrawal as a syndrome, two or more symptoms were required in addition to satisfaction of the duration qualifier. For the period prior to the past year, clustering of symptoms was established by determining that the number of symptoms required to achieve a diagnosis occurred (a) at the same time, (b) repeatedly for a few months or longer, or (c) most days for at least 1 month.

For individuals who had an alcohol use disorder in the past year but not in the period prior to the past year, age at onset of abuse or dependence was set to the age at interview. For persons with an alcohol use disorder in the period prior to the past year, age at onset was determined by asking the age at which the clustering of the requisite number of symptoms first occurred. Individuals classified with abuse or dependence in the period prior to the past year were considered to have achieved remission from alcohol use disorders if they did not meet the criteria for either abuse or dependence during the year preceding interview. Age at remission was defined as the earlier of the ages at which the respondent stopped drinking (based on a question that asked for the interval since last drink) or reported last experiencing the clustering and duration of symptoms required for the diagnosis of an alcohol use disorder.

Alcohol treatment

Receipt of alcohol treatment was established by a positive response to the question "Have you ever gone anywhere or seen anyone for a reason that was related to your drinking—a physician, counselor, Alcoholics Anonymous, or any other community agency or professional? Include help for combined alcohol and other drug use if alcohol was the major problem for which you sought help." Persons responding affirmatively to this question were asked about their utilization of 23 specific sources of treatment, ranging from Alcoholics Anonymous to different types of health professionals, employee assistance programs, and so forth. For each type of treatment reported by the respondent, the age at first use was established by asking "How old were you when you *first* went there?" Interval from onset of abuse or dependence to treatment was derived by sub-

tracting age at onset of disorder from the earliest age reported for any form of treatment.

Period of exposure

The period of exposure to the risk of initiating alcohol treatment—the interval which forms the basis for the survival analyses used in this study—was measured in single years by subtracting age at onset of alcohol use disorder from the earliest of the following ages: age at treatment (if treatment was received), age at remission of alcohol use disorder (if remission occurred), and age at interview. When age at treatment equaled or preceded the ages at remission and at interview, this period of exposure represented the interval to treatment. When age at remission or interview preceded age at treatment (primarily cases where no treatment was received), this period of exposure represented the interval to removal from risk. For some individuals, the interval from onset of alcohol use disorder to onset of treatment was negative; that is, they reported receiving treatment before their symptoms were sufficient to satisfy the DSM–IV criteria for alcohol abuse or dependence. These individuals were removed from this analysis. The magnitude and effect of this removal will be discussed subsequently.

Model covariates

Measurement of basic demographic characteristics (age in single years, male/female, black/nonblack, Hispanic/non-Hispanic) was straightforward. Education was summarized with three dummy variables (high school graduate, attended college, college graduate) that used not having completed high school as the reference category. Marital status consisted of two dummy variables (ever married but never divorced/separated, ever divorced/separated) that used never married as a reference category. The measure of whether or not the respondent had children under 18 years of age in the home during the period of exposure was derived by comparing the ages of the oldest and youngest children to the ages that defined the start and end of the period of exposure. Family history of alcoholism was ascertained through a series of questions that asked about 18 different types of first- and second-degree biological relatives. For each type of relative, the respondent was asked "In your judgment, has (your/any of your) _____been an alcoholic or problem drinker at ANY time in his/her life?" An alcoholic or problem drinker was defined for the respondent in the following manner: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking, problems with a spouse, family or friends because of drinking, problems at work because of drinking, problems with the police because of drinking—like drunk driving—or a person who seems to spend a lot of time drinking or being hung over." Number of symptoms referred to how many of the 31 symptoms used in the NLAES to operationalize DSM–IV alcohol abuse and dependence were reported by the respondent as ever having occurred.

Consumption measures referred to the period identified by the respondent as his or her period of heaviest consumption. Average ounces of ethanol consumed per drinking day was calculated on the basis of questions that asked for the usual and largest numbers and sizes of drinks of beer, wine, and liquor that the respondent drank on a drinking day and overall frequency of drinking as well as the frequency with which the largest quantities were consumed (Dawson, Grant, Chou, & Pickering, 1995). Ounces of beverage were converted to ethanol using the following conversion factors: .045 for beer, .121 for wine, and .409 for liquor (DISCUS, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams, Clem, & Dufour, 1993). In order to account for the differing effects of ethanol intake on individuals with different levels of total body water (Goist & Sutker, 1985), the measure of ounces of ethanol was converted to a measure of milligrams of ethanol per liter of total body water (Moore et al., 1963).

This analysis also incorporated measures of comorbid DSM–IV drug use disorders and treatment and comorbid episodes of DSM-IV major depression. The derivation of these diagnoses has been explained fully elsewhere (Grant, 1995). The criteria for drug use disorders were comparable to those for alcohol use disorders and referred to use of illegal drugs, such as marijuana, cocaine, and heroin, as well as misuse of psychoactive prescription drugs such as sedatives and tranquilizers. Two dummy variables were used for drug use disorders, one that indicated treatment for a drug use disorder during the period of exposure and another that indicated the presence of an untreated disorder during the period of exposure. DSM–IV major depression, which was dichotomized as present or absent during the period of exposure, required that a respondent report five or more depressive symptoms (inclusive of low mood or lack of interest) that occurred nearly every day for at least the same 2-week period, that co-occurred with social and/or occupational dysfunction, and that were not attributed to physical illness or bereavement. Dating of all of these measures with respect to the period of exposure was

accomplished by comparing the ages at which the disorders or treatment began with the ages defining the start and end of the period of exposure, as defined previously.

Analysis

There were three stages to this analysis. The first stage consisted of estimating simple descriptive statistics on the prevalence and timing of treatment for men and women. In the second stage, life table techniques were used to estimate men's and women's cumulative conditional probabilities of having received treatment by the start of selected intervals since onset of disorder. The cumulative conditional probability of having initiated treatment by the start of an interval is the complement of the survival function ($1\text{-}S_i$) for that interval. The survival function (S_i) is the product of the conditional probabilities of having "survived" the interval *without* initiating treatment (conditional upon being still at risk of initiating treatment at the start of the interval) for all intervals up to but not including the one in question, for example, $P_1P_2 \ldots P_{i-1}$ (Lee, 1980). Because the formula used to calculate the standard errors of these conditional probabilities (Lee, 1980) assumed a simple random sample, the standard errors were adjusted by SUDAAN estimates of the design effects on the simple proportions of men and women who ever received treatment during the period of exposure. Male-to-female ratios were estimated by dividing the male by the female probabilities, and the variances of these ratios were derived by the delta method (Stuart & Ord, 1987).

The third stage of the analysis consisted of fitting a discrete-time proportional hazards model to estimate the effects of gender and the other model covariates on the hazard of initiating alcohol treatment. The hazard function (h_i) for a specified interval is the number of persons initiating treatment per year during the interval divided by the number of persons still exposed to the risk of initiating treatment at the midpoint of the interval. Within any interval, the hazard function, coded 1 if the interval terminated in an event (treatment) or 0 if it terminated in withdrawal, is expressed by the formula $h_i(z) = 1 - (1 - h_i)^{\exp(\beta' z)}$, where z is a vector of the model covariates, β is the corresponding vector of regression coefficients, and h_i is the underlying hazard when $z = 0$ (Shah et al., 1993). Proportional hazards models assume that the effect of each covariate on the hazard function remains constant over time, that is, across all intervals. There is no intercept term in proportional hazards models, because the underlying hazard function—the analog of the intercept term in regular multiple regression models—varies across intervals. As in logistic regression models, the beta coefficients for the model covariates can be exponentiated to yield an estimate of the hazard ratio, for example, the hazard function for men relative to that of women.

The dependent variable used in fitting the proportional hazards model was the length of period of exposure, which was coupled with an indicator of whether this period terminated with treatment or removal from risk of treatment to determine the underlying hazard function for each interval. Because age at onset of disorder and current age can be combined to exactly predict the length of this period for all individuals who still had an alcohol use disorder in the year preceding interview, these two variables cannot both be used in continuous form as independent variables without overspecifying the model. To avoid this problem, age at onset of abuse or dependence was categorized into ages 18–20 (34% of the sample), ages 21–24 (20%), and ages 25 and over (26%), using onset below age 18 (20%) as the reference category.

The model-fitting procedure entailed testing for first-order interactions between gender and all of the other model covariates. After removal of nonsignificant interaction terms, $p > .05$, main effects were again tested for statistical significance. Nonsignificant covariates were retained in the model only if their presence was required for interpretation of significant interaction terms or if their removal materially affected the remaining parameters.

RESULTS

Twenty-three percent of the men and 15% of the women with lifetime alcohol use disorders had received treatment for alcohol problems at some point during their lives by the age at interview (Table 1), yielding a male-to-female ratio of 1.52 to 1.0. One eighth of the treated men (2.9% of all those with alcohol use disorders) first received treatment prior to onset of the disorder, that is, prior to the point when they reported enough symptoms to satisfy the diagnostic criteria for abuse or dependence, as did one sixth of the treated women (2.6% of all those with disorders). Another one eighth of both the treated men and women first began treatment after remission of the disorder, presumably as a means of maintaining abstinence or moderate drinking patterns. The median interval from onset of disorder to initiation of treatment was 5.0 years for men and 2.1 years for women (4.7 and 2.3 years, respectively, if based solely on indi-

viduals who received treatment during the period of exposure, that is, between the onset and remission of the alcohol use disorder). There were no significant differences in the proportions of men and women who stopped drinking (16.7% of men and 15.3% of women) or who otherwise achieved remission from alcohol use disorders (31.8% of men and 32.6% of women) prior to the year preceding interview. However, the median intervals to the two types of remission were 2 to 3 years shorter for women than men, as was the interval from onset of disorder to time of interview. Thus, men remained at risk of initiating treatment for a slightly longer period than did women, which may have contributed to their excess probability of ever having been treated.

Table 2 compares men's and women's cumulative conditional probabilities of having initiated treatment by the start of selected intervals since onset of alcohol use disorder. The 95% confidence intervals for the male-to-female ratios indicate that men's and women's probabilities of ever having received treatment were not significantly different during many of the intervals that were examined. The only significant differences (as indicated by confidence intervals that did not encompass the value of 1.0) occurred during the intervals from 8 to 25 years after onset of disorder, during which time the conditional probability of having initiated treatment was 13% to 20% higher for men than women. The male-to-female ratios did not demonstrate any significant variation across intervals, indicating that the proportionality assumption used in subsequent multivariate modeling was not violated.

Table 3 presents the proportional hazards models predicting the likelihood of initiating treatment for alcohol problems, before and after removal of nonsignificant covariates. The only variable that formed a significant interaction with gender was number of symptoms of abuse and dependence. The negative sign of this interaction indicates that the underlying positive effect of male gender on the likelihood of treatment decreased as the severity of the disorder increased. Other factors that were associated with an increased likelihood of having received treatment include ever having been divorced or separated, ever having been employed, a positive family history of alcoholism, having been a daily drinker, increasing age at onset of alcohol use disorder, and having received treatment for a drug use disorder during the period of exposure for alcohol treatment. Factors that were inversely associated with the likelihood of alcohol treatment were black race, having graduated from college, having had children under the age of 18 during the period of exposure, and age at interview. (The increasing probability of an individual having received treatment over time, i.e., with advancing age, was captured by the unmeasured intercept term of the proportional hazards model. The negative parameter for age at interview reflects the cohort aspect of age, indicating that for any given interval since onset of disorder, members of older cohorts were less likely to report having received treatment than members of younger cohorts.)

Table 1. Selected Treatment-Related Characteristics of Adults 18 Years of Age and Over With Lifetime Alcohol Use Disorders, by Gender

	Male	Female
Percentage who ever received treatment:		
At any time**	23.0 (0.7)	15.1 (0.8)
Before meeting criteria for disorder	2.9 (0.3)	2.6 (0.3)
Between onset and remission**	17.5 (0.7)	10.7 (0.7)
After remission**	2.7 (0.3)	1.8 (0.3)
Percentage who stopped drinking (no drinks in year preceding interview)	16.7 (0.7)	15.3 (0.8)
Percentage who experienced remission despite continued drinking	31.8 (0.8)	32.6 (1.0)
Median interval (years) from onset to:		
Initiation of treatment**	5.0 (0.3)	2.1 (0.3)
Interview**	12.7 (0.3)	9.5 (0.2)
Last drink**	11.3 (0.2)	8.6 (0.2)
Remission despite continued drinking**	5.4 (0.2)	3.1 (0.2)

Note. Figures in parentheses are standard errors.
** Gender differences significant at the $p < .05$ level.
[a] Interval shown is for all persons ever treated. For persons treated between onset and remission of alcohol use disorder, the mean intervals to treatment were 4.7 years for men and 2.3 years for women.

Table 4 summarizes the effects of gender on the likelihood of having received treatment for alcohol problems, incorporating the interaction of gender with severity as indicated by number of symptoms of abuse and/or dependence. Among persons with 20 or more symptoms, there was no gender difference in the hazard function for initiation of alcohol treatment. At lower levels of severity, the male-to-female hazard ratio declined from 1.75 among persons with 1 symptom (the minimum number required for a diagnosis of abuse) and 1.67 among persons with 3 symptoms (the minimum required for dependence) to 1.26 among those with 15 symptoms.

DISCUSSION

This analysis found that in general, men with alcohol use disorders were more likely than their female counterparts to ever have received treatment for alcohol-related problems. The only exceptions occurred among the most severely affected men and women, those with 20 or more symptoms of abuse and/or dependence, who were equally likely to have initiated treatment.

However, this level of severity was reported by only 11% of the men and 8% of the women with alcohol use disorders. Thus, gender differentials in treatment-seeking behavior affected the great majority of men and women with alcohol-related problems. At the most common levels of severity, between 5 and 10 symptoms, men were about 50% more likely than women to have received treatment.

The nature of the interaction between gender and severity leads to over-representation of the most severely affected women in the treatment group, supporting previous findings which have indicated that women have more severe problems than men upon presentation for treatment (Farid & Clarke, 1992; Hasin et al., 1988). Coupled with women's shorter median interval to treatment, these data also support the hypothesis of telescoping (i.e., the more rapid progression from initiation of drinking to development of alcohol-related problems and entry into treatment) that has been reported in prior studies (Piazza et al., 1989). It is important to note that the shorter median interval to treatment among women did not result from women being more likely than men to initiate treatment shortly after onset of disorders; rather it resulted from men being more likely than women to initiate treatment at most of the later intervals. This may reflect the phenomenon of women's drinking being more "hidden," that is, more strongly comprised of solitary drinking and more likely to occur in the home, especially as it progresses to the stage of heavy or problem drinking (Blume, 1986; Hanna, 1991). This pattern of drinking may be more sustainable—that is, less likely to result in the sort of incident provoking entry into treatment—than the more public drinking styles of men. Alternatively, given the importance of social network influence (Kandel, 1983), this pattern could reflect the fact that over time, women are more likely than men to have lived with a heavy-drinking partner whose behavior would tend to validate their own drinking patterns and increase denial of their need for treatment.

As was noted earlier, the survival analyses utilized in this analysis excluded cases of treatment received before the criteria for an alcohol use disorder were met and treated cases where the treatment was initiated after remission of the disorder as having been withdrawn from the risk pool at the time remission occurred. In order to determine whether the exclusion of treatment received before or after the period of exposure had a strong impact on the gender differential, the multivariate analysis was repeated using a simple logistic regression model predicting the odds of *ever* having received treatment (data not shown). The results of this model were almost identical to those obtained from the proportional hazards model, with a positive underlying effect of male gender ($\beta = 0.789$) and a negative interaction between gender and severity ($\beta = -0.023$). Thus, exclusion of treatment received at the preclinical stage does not appear to have resulted in overestimation of men's excess likelihood of treatment.

This study did not find many of the interactions between gender and other correlates of treatment that might have been expected on the basis of past research. For example, there was no interaction between gender and experience of DSM–IV major depression, despite evidence that women enter alcohol treatment via the mental health treatment system more often than men (Weisner & Schmidt, 1992). Likewise, despite the fact that women more often cite child care

Table 2. Life Table Estimates of Cumulative Conditional Probability of Having Initiated Alcohol Treatment by Start of Selected Intervals Since Onset of Alcohol Use Disorder, by Gender

Interval Since Onset of Alcohol Use Disorder (in years)	Male		Female		Male-to-Female Ratio and 95% CI
	Probability	*SE*	Probability	*SE*	
1–<2	.026	(.003)	.023	(.002)	*ns* (0.82–1.39)
2–<3	.042	(.004)	.045	(.002)	*ns* (0.75–1.11)
3–<4	.063	(.005)	.065	(.003)	*ns* (0.81–1.13)
4–<6	.079	(.005)	.083	(.003)	*ns* (0.81–1.09)
6–<8	.121	(.007)	.111	(.003)	*ns* (0.96–1.23)
8–<10	.164	(.008)	.142	(.004)	1.15 (1.03–1.28)
10–<15	.196	(.009)	.173	(.004)	1.13 (1.01–1.25)
15–<20	.271	(.012)	.227	(.004)	1.20 (1.09–1.31)
20–<25	.335	(.014)	.286	(.005)	1.17 (1.07–1.28)
25–<30	.381	(.017)	.347	(.032)	*ns* (0.88–1.31)
30 or more	.424	(.020)	.356	(.034)	*ns* (0.94–1.44)

problems as barriers to treatment (Dawson, 1994; Grant, in press) and that the presence of children under 18 was indeed negatively associated with the likelihood of treatment, the effect of this factor was not significantly different for men and women. Nor was the positive effect of ever having been divorced or separated modified by gender, despite studies that have shown greater marital instability among female than male alcoholics (Glatt, 1961; Rathod & Thomson, 1971). These discrepancies underscore the fact that gender differences in various characteristics within treatment samples cannot be construed as factors that modify gender differences in the likelihood of entering treatment. At the same time, the lack of a statistically significant interaction between gender and some other correlate of treatment does not preclude gender differences in the perceived *priority* of that correlate as an impetus or barrier to entering treatment.

Table 3. Full and Reduced Proportional Hazards Models Predicting Hazard of Ever Having Initiated Treatment for Alcohol Use Disorder

	Full Model			Reduced Model		
	β	*SE*	*p*	β	*SE*	*p*
Main Effects:						
Male	0.553	0.199	.007	0.584	0.200	.005
Age	−0.078	0.006	<.001	−0.077	0.006	<.001
Black	−0.331	0.160	.042	−0.314	0.147	.036
Hispanic	−0.163	0.192	.399	—	—	—
College graduate	−0.315	0.140	.028	−0.361	0.097	<.001
Attended college	0.062	0.127	.637	—	—	—
High school graduate	0.109	0.125	.386	—	—	—
Ever married, never divorced/ separated	0.024	0.094	.801	—	—	—
Ever divorced/separated	0.201	0.127	.118	0.225	0.103	.032
Ever employed	0.282	0.126	.028	0.335	0.110	.003
Had children >18 years of age[a]	−0.726	0.089	<.001	−0.726	0.074	<.001
Positive family history of alcoholism	0.320	0.126	.013	0.370	0.121	.003
Ever had alcoholic partner	0.001	0.099	.993	—	—	—
Daily drinker during period of heaviest consumption	0.254	0.085	.004	0.285	0.081	<.001
Amount of ethanol consumed per drinking day during period of heaviest consumption[b]	<0.001	<0.001	.245	—	—	—
Alcohol abuse only	−0.177	0.126	.163	—	—	—
N of symptoms of abuse/dependence	0.129	0.010	<.001	0.128	0.010	<.001
Onset of alcohol use disorder at ages 18–20	0.349	0.111	.002	0.364	0.104	<.001
Onset of alcohol use disorder at ages 21–24	0.719	0.129	<.001	0.790	0.120	<.001
Onset of alcohol use disorder at age 25+	1.817	0.133	<.001	1.847	0.128	<.001
Experienced untreated drug use disorder[a]	−0.163	0.099	.104	—	—	—
Received treatment for drug use disorder[a]	0.574	0.139	<.001	0.589	0.115	<.001
Experienced period of major depression[a]	−0.130	0.089	.149	—	—	—
Interaction Terms:						
Male × *N* of symptoms of abuse/ dependence	−0.024	0.011	.026	−0.024	.011	.033
Goodness of Fit:						
Overall model	$F(24, 68) = 32.26$ $p < .001$			$F(15, 68) = 62.32$, $p < .001$		

[a] During period of exposure.
[b] Milligrams of ethanol per deciliter of total body water.

Table 4. Male-to-Female Hazard Ratios for Ever Having Initiated Treatment for Alcohol Use Disorders, According to Number of Symptoms of Alcohol Abuse and/or Dependence

N Symptoms	Hazard Ratio	95% CI
1[a]	1.75	(1.20–2.54)
3[b]	1.67	(1.19–2.34)
5	1.59	(1.18–2.15)
10	1.42	(1.14–1.78)
15	1.26	(1.05–1.51)
20	*ns*	(0.93–1.36)
25	*ns*	(0.77–1.27)
30	*ns*	(0.63–1.23)

[a] Minimum number of symptoms required for abuse diagnosis.
[b] Minimum number of symptoms required for dependence diagnosis.

Many studies of entry into alcohol treatment have adapted Aday and Anderson's (1974) framework of considering predisposing, enabling, and need variables. The multivariate models used in this analysis omitted a number of important enabling variables, for example, place of residence, income, and health insurance, because of lack of data on these variables for periods other than the past year. A number of the other variables thought to be associated with the likelihood of initiating treatment were measured in terms of ever or never present during the period of exposure, rather than with the precision ideally suited to a survival analysis. Had we been able to identify and control for the exact timing of changes in measures such as marital status, presence of children in the household, drug treatment utilization, comorbid depression and associated treatment, employment, place of residence and associated access to treatment options, and health care coverage, this analysis might have uncovered more factors modifying gender differences in the likelihood of receiving alcohol treatment.

This analysis utilized a single count of all abuse and dependence symptom items as a measure of overall severity of alcohol-related problems. Further research could take advantage of the criterion-specific data collected in the NLAES to clarify the relative importance of different types of symptoms (e.g., legal, social, and so forth) with regard to men's and women's respective probabilities of initiating treatment. The NLAES data also permit gender comparisons of different types of treatment—inpatient, outpatient, 12-step, and so forth. Examination of the different treatment modalities employed by men and women, and the order in which they are utilized, may provide additional insight as to why men's rates of utilization exceed those of women. Finally, another interesting area for additional research is the association between interval to treatment and treatment outcome, and whether and how this association differs for men and women.

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Symptoms and Characteristics of Individuals With Different Types of Recovery From DSM–IV Alcohol Dependence

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Symptoms and criteria for DSM-IV alcohol dependence and demographic and drinking characteristics are compared for three groups. Group I (N = 1,044) consists of persons who formerly met the criteria for dependence but were abstinent in the past year, Group II (N = 2,325) consists of persons who were formerly dependent but did not meet the criteria for abuse or dependence in the past year despite drinking, and Group III (N = 22,204) consists of persons who never met the criteria for dependence. Members of Group II lay between members of Groups I and III in terms of the numbers of prior dependence symptoms and criteria, past level of intake, degree of familial alcoholism and history of alcohol treatment. Both groups of former alcoholics were equally likely to have experienced withdrawal, drinking more/longer than intended and tolerance. The criteria that most strongly distinguished the groups were continued use despite physical or psychological consequences, time spent drinking and activities given up, all of which were far more common in Group I than in Group II. Members of Group II had earlier onsets of heavy drinking and dependence than members of Group I, supporting the existence of a developmentally limited subtype of alcoholism that is subject to spontaneous remission in early adulthood without treatment. The paper compares three options for tightening the criteria for dependence, all of which remove more members from Group II (28% to 41% depending on option) than from Group I (12% to 19%).

The alcohol literature contains a number of studies that have found varying levels of a symptomatic or relatively problem-free drinking among individuals formerly classified as alcoholics or problem drinkers, both in clinical samples (Gottheil, Thornton, Skoloda, and Alterman, 1982; Heather and Tebbutt, 1989; Helzer et al., 1985; McCabe, 1986; Miller, 1983; Nordstrom and Berglund, 1987; Pettinati, Sugarman, DiDonato, and Mauren, 1982: Polich, Armor, and Braiker, 1980) and in tests of alternative treatment goals (Booth, Dale, Slade, and Dewey, 1992; Elal-Lawrence et al., 1986; Miller, Leckman, Delaney, and Tinkcom, 1992; Orford and Keddie, 1986; Rychtarik, Foy, Scott, Lokey, and Prue, 1987; Sanchez-Craig and Lei, 1986). Several general population studies (Fillmore and Midanik, 1984; Ösjesjö, 1981; Sobell, Cunningham, and Sobell, 1996; Temple and Fillmore, 1985-86; Vaillant, 1982, 1995) have reported similar findings, but none of these defined alcohol problems in a manner that was consistent with the current diagnostic classifications of abuse and dependence. For example, problem drinkers were identified on the basis of a scale score that included items such as intake and binge drinking in the study by Fillmore and Midanik and on the basis of frequency of drinking to get high in the study by Temple and Fillmore. In the study by Sobell et al., individuals with former alcohol problems were those who reported problems that affected one or more of the following areas: work or studies, family or home life, physical health, friendships, social life, or finances. Ösjesjö's categories of abusers, addicts and chronics crossed the current boundaries separating abuse and dependence, and Vaillant used a combination of the now outdated DSM-III (American Psychiatric Association, 1980) criteria for dependence and the Problem Drinking Scale (Vaillant, 1983). One recent study of the general population (Dawson, 1996a), that did define alcohol dependence in accordance with the current DSM-IV criteria (American Psychiatric Association, 1994), found that half of all adults who satisfied the criteria for alcohol dependence at some point prior to the past year were classified as drinkers who no longer met the criteria for either abuse or dependence during the year preceding interview. When stratified by interval since onset of depen-

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dence, more than half of all individuals with intervals of five years or longer fell into this category. These individuals reported fewer alcohol-related problems than those who were abstinent, still dependent or abusers in the year preceding interview, and they also reported far lower rates of treatment, suggesting that studies of treatment samples may underestimate the probability of this type of recovery.

Findings such as these highlight the disparity between the current psychiatric definition of alcohol dependence and the disease concept of alcoholism. The DSM-IV and ICD-10 criteria currently used to define alcohol dependence (American Psychiatric Association. 1994; World Health Organization, 1992) are strongly derivative of the alcohol dependence syndrome (Edwards and Gross, 1976), conceptualized as a cohesive cluster of interrelated symptoms centered around impaired control over alcohol and characterized by varying levels of severity and assorted symptom arrays. Both the DSM-IV and ICD-10 classification systems dichotomize alcohol dependence as present or absent based on arbitrary cutpoints on this scale of severity. Although Meyer (1994) has argued that these definitions of dependence identify any explicit disease that may lie within the broad spectrum of alcohol-related problems, alcohol dependence and the dependence syndrome differ from a strict interpretation of the disease concept of alcoholism (Jellinek, 1960) in several important ways (Schuckit, 1995; Edwards, 1992; Flavin and Morse, 1991) that include the prognosis for recovery. The disease model treats alcoholism as a lifelong, progressive and incurable condition whose symptoms can be controlled only by abstinence. In contrast, the DSM-IV criteria for partial and full remission from alcohol dependence are independent of ethanol consumption. They do not exclude the possibility that individuals formerly diagnosed with alcohol dependence may achieve a full or partial recovery while continuing to drink.

The goal of this study is to investigate the patterns of alcohol-related problems that underlie the disparity between dependence and the disease concept by comparing two groups of U.S. adults who satisfied the DSM-IV criteria for alcohol dependence during some period prior to the past year but who were *not* classified as either dependent or abusers during the past year (i.e., the year immediately preceding the interview). The first group (Group I) is comprised of individuals who were totally abstinent in the past year, and the second group (Group II) is comprised of individuals who were neither dependent nor abusers during the past year despite having consumed one or more alcoholic drinks during that period. In so far as this second group's experience runs contrary to the central tenets of the disease model, i.e., the irreversible course of alcoholism and the control of its symptoms exclusively by means of abstinence, the individuals in Group II might be considered as false positives with respect to the disease concept of alcoholism. A third group (Group III), occasionally used for purposes of comparison, is comprised of lifetime drinkers who had *never* met the DSM-IV criteria for alcohol dependence. This analysis compares levels and patterns of dependence symptoms and criteria within and among these groups and discusses these differences with respect not only to the disease concept of alcoholism versus dependence but also with respect to typologies for alcohol dependence.

METHOD

Sample

This study is based on data collected in the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and conducted by the U.S. Bureau of the Census. The NLAES sample was representative of U.S. adults 18 years of age and over, residing in households in the civilian, noninstitutionalized population. Data were obtained in personal interviews conducted in respondents' homes, and proxy respondents were not permitted. The household and sample person response rates were 92% and 97%, respectively, yielding a total sample size of 42,862. The sample used for this analysis consisted of 1,044 individuals in Group I, 2,325 individuals in Group II and 22,204 individuals in Group III.

The complex, multistage NLAES sample design (Grant, Peterson, Dawson, and Chou, 1994a) featured selection of primary sampling units with probability proportional to size and oversampling of blacks and young adults between the ages of 18 and 29. To adequately incorporate these design effects into variance estimation procedures, the estimates presented in this paper were generated by SUDAAN (Shah, Barnwell, and Bieler, 1996), a software package that accommodates the design features of complex sample designs. Because of the multiple comparisons employed in this analysis, a significance level of $p < .002$ was required before between-group differences were cited as statistically significant.

Measures

To be classified with prior-to-past-year DSM-IV alcohol dependence (that is, dependence within any one-year period other than that immediately preceding the

NLAES interview), an individual had to meet at least three of the seven DSM-IV criteria for dependence: (1) tolerance; (2) withdrawal (including relief or avoidance of withdrawal); (3) persistent desire or unsuccessful attempts to cut down on or stop drinking; (4) much time spent drinking, obtaining alcohol or recovering from its effects; (5) reduction or cessation of important activities in favor of drinking; (6) impaired control over drinking; and (7) continued use despite physical or psychological problems caused by drinking. These criteria were operationalized by means of 25 symptom item indicators (Grant et al., 1994b) that were asked of all lifetime drinkers, that is, persons who had consumed 12 or more drinks during at least one year of their lives. A criterion was satisfied if a person reported that one or more positive symptoms of that criterion occurred during the period prior to the past year. To be consistent with the syndromal definition of the withdrawal criterion, two or more positive symptoms were required from a list of eight discrete withdrawal symptoms. In order to satisfy the duration qualifiers for the period prior to the past year, respondents additionally had to report that some of the symptoms of dependence occurred "at around the same time" *or* "at around the same time, on and off for a few months or longer" *or* "at around the same time, most days for at least a month." In addition to establishing the duration criteria, a positive response to one of these questions ensured that the symptoms were clustered at a period in time rather than occurring at different times over the life span. Age at onset of dependence was defined as the age when symptoms first began to co-occur as defined above.

Past-year alcohol dependence required satisfying at least three of the previously listed DSM-IV dependence criteria during the one-year period immediately preceding the NLAES interview. Past-year alcohol abuse required satisfying one or more of the following criteria: continued use despite social or interpersonal consequences; hazardous use; alcohol-related legal problems; and neglect of role responsibilities in favor of drinking. The abuse criteria were operationalized by means of six symptom item indicators. Abuse and dependence criteria not associated with duration qualifiers were satisfied if an individual reported one or more positive symptoms of the criterion during the past year, and criteria with duration qualifiers were satisfied if a person reported two or more symptoms of the criterion during the past year or one symptom that occurred at least two times during the past year.

Individuals classified as members of Group I were those who: (a) satisfied the criteria for prior-to-past-year dependence, *and* (b) who did *not* meet the criteria for either past-year dependence or past-year abuse, *and* (c) who were past-year abstainers, i.e., who reported an interval of at least 12 months since their last drink. Individuals classified as members of Group II were those who: (a) satisfied the criteria for prior-to-past-year dependence, *and* (b) who did *not* meet the criteria for either past-year dependence or past-year abuse, *and* (c) who reported having consumed one or more drinks in the past year. (Approximately one-fourth of the drinkers in this category reported drinking less than 12 drinks in the past year. These individuals, like the total abstainers, were not asked the symptom item indicators for past-year alcohol use disorders but were assumed to have experienced no drinking-related problems during that period.) Individuals who were classified as members of Group III were those (a) who did not satisfy the criteria for prior-to-past-year alcohol dependence, *and* (b) who did not satisfy the criteria for past-year alcohol dependence. Throughout this study, the term 'formerly dependent' will be used to denote individuals with prior-to-past-year alcohol dependence who did not meet the criteria for either dependence or abuse in the past year, and the term 'recovery' will be applied to this process of remission from DSM-IV alcohol dependence.

Family history of alcoholism was derived from respondents' reports of alcoholism in any of 18 different types of first- and second-degree relatives, based on the definition of an alcoholic as a person with drinking-related physical or emotional problems, problems with a spouse, family or friends, problems at work, problems with the police—like drunk driving—or as a person who spends a lot of time drinking or being hungover. The proportion of alcoholic first- and second-degree relatives was obtained by dividing the number of alcoholic relatives by the total number of relatives who lived to be at least ten years of age.

Consumption measures were defined for the period identified by each respondent as his or her period of heaviest drinking. Average daily ethanol intake was derived by dividing the annualized volume by 365, where annualized volume was the sum over beer, wine and liquor of the following: [(number of drinking days per $\text{year}_{\text{usual-heavy}}$)(number of drinks per drinking $\text{day}_{\text{usual}}$)(size of drink in $\text{ounces}_{\text{usual}}$)(ethanol conversion factor) + (number of drinking days per $\text{year}_{\text{heavy}}$) (number of drinks per drinking $\text{day}_{\text{heavy}}$)(size of drink in $\text{ounces}_{\text{heavy}}$)(ethanol conversion factor)]. Ethanol conversion factors were defined as .045 for beer, .121 for wine and .409 for liquor (DISCUS, 1985; Kling, 1989; *Modern Brewery Age,* 1992; Turner, 1990;

Williams, Clem, and Dufour, 1993). Average ethanol intake per drinking day was derived by dividing the annualized volume by the total number of drinking days per year, estimated as the mean of the largest beverage-specific frequency and the sum of the beverage-specific frequencies (the latter not to exceed 365).

Treatment history was based on questions about 23 different sources of treatment, including 12-step programs such as Alcoholics Anonymous. An open-ended question on "other" sources of treatment was included as well. All questions stipulated that the individual must have gone to that source of treatment primarily for problems relating to his or her own drinking.

The items utilized in this analysis generally showed good to excellent levels of reliability in a test-retest study conducted in a representative community sample (Grant, Harford, Dawson, Chou, and Pickering, 1995) and in a local area pretest. Selected reliability coefficients include 0.75 and 0.63, respectively, for past year and prior to past year dependence, 0.85 for age at first drink, 0.65 for average daily ethanol consumption during period of heaviest drinking, and 0.70 for alcohol treatment. For the family history variables, the reliability coefficients were 0.72 and 1.00 for alcohol problems in the father and mother, 0.90 and 0.73 for brothers and sisters, 0.65 for both sons and daughters and between 0.64 and 1.00 for different types of grandparents.

RESULTS

Table 1 reveals many differences in demographic and drinking history characteristics among the individuals in Groups I–III. Most measures—i.e., the proportion of males, proportion with positive family histories, level of family history saturation, all measures of alcohol consumption during period of heaviest consumption and the proportion ever treated for alcohol problems—declined steadily across the three groups. Thus, the formerly dependent who had progressed to drinking without abuse or dependence (Group II) lay between those who had progressed to abstinence (Group I) and those who had never been dependent (Group III). There were a few exceptions to this pattern. For example, individuals in Group II were younger on average and less likely to be of black race than those in both Groups I and III, and both of the formerly dependent Groups I and II reported an earlier average age at first drink than did the members of Group III, i.e., those who never were dependent. Among the formerly dependent, members of Group II reported earlier ages at onset of both heaviest drinking and dependence than did members of Group I.

As indicated in Table 2, both the number of prior-to-past-year symptoms of dependence and the number of positive criteria decreased steadily from Group I to

Table 1. Demographic, Family History, Consumption and Treatment Characteristics of Selected Categories of Current and Former Drinkers 18 Years of Age and Over, According to History of DSM-IV Alcohol Dependence

	Formerly Dependent		
	Past-year Abstainers (Group I)	*Past-year Drinkers with Neither Abuse nor Dependence (Group II)*	*Never Met Criteria for Dependence (Group III)*
Mean age	44.9 (0.5)[a]	36.9 (0.3)[b]	43.3 (0.2)
% male	69.6 (1.6)[a,b]	62.4 (1.1)[b]	54.4 (0.4)
% black	8.8 (1.0)[a]	5.2 (0.6)[b]	9.3 (0.3)
% with positive family history of alcoholism	82.6 (1.3)[a,b]	74.1 (1.0)[b]	54.0 (0.4)
Mean % of alcoholic 1st and 2nd degree relatives	18.8 (0.6)[a,b]	14.4 (0.4)[b]	7.8 (0.1)
Mean age at first drink	17.4 (0.2)[b]	17.3 (0.1)[b]	19.5 (<0.1)
Mean age at onset of heaviest drinking	24.5 (0.3)[a,b]	21.6 (0.1)[b]	22.7 (0.1)
Mean age at onset of dependence	25.8 (0.3)[a]	22.1 (0.2)	NA
% with onset of dependence before age 21	40.6 (1.8)[a]	54.2 (1.3)	NA
Consumption during period of heaviest drinking:			
Mean daily ethanol intake (oz.)	6.6 (0.3)[a,b]	3.5 (0.1)[b]	1.1 (<0.1)
Mean ethanol intake per drinking day (oz.)	8.0 (0.3)[a,b]	5.5 (0.1)[b]	2.9 (<0.1)
Mean number of drinking days per year	269.0 (4.3)[a,b]	207.8 (2.7)[b]	119.6 (1.0)
% who drank daily or nearly daily	65.1 (1.6)[a,b]	39.9 (1.1)[b]	16.7 (0.3)
% ever treated for alcohol problems	46.0 (1.9)[a,b]	14.9 (0.9)[b]	3.1 (0.2)

Notes. a. Estimates are significantly different from those for Group II ($p < .002$)
b. Estimates are significantly different from Group III ($p < .002$)

Group III, the former from 12.8 to 2.0 and the latter from 5.2 to 1.1. (Notably, only 45% of the individuals in Group III were negative for all seven of the prior-to-past-year dependence criteria. Twelve percent reported three or more positive criteria but failed to meet the duration and clustering of symptoms required for a positive classification for dependence.) The prevalence of the individual criteria also tended to decline from Group I to Group III, but for three of the four most common criteria—withdrawal, drinking more/longer than intended and tolerance—the differences between Groups I and II were not statistically significant at the $p < .002$ level. That is, these criteria were equally prevalent within both groups of the formerly dependent, regardless of their type of recovery. The criteria for both withdrawal and drinking more/longer than intended were each satisfied by approximately 30% of never-dependent individuals in addition to by more than 90% of the formerly dependent. Desire/attempts to cut down/stop drinking and tolerance were both reported by 21% of the never dependent as well as by more than three quarters of the formerly dependent. Thus these criteria were three to five times as prevalent among persons with prior-to-past-year dependence as among those who never were dependent. In contrast, the less commonly endorsed criteria—continued use despite physical or psychological difficulties, much time spent drinking, and important activities given up—were more than ten times as common in Group II as in Group III, and roughly 30 times as common in Group I.

Among individuals who satisfied the withdrawal criterion in a period prior to the past year, the number of symptoms of withdrawal ranged from 3.9 in Group I to 2.4 in Group III. Between 83% and 88% of the individuals in all three groups reported bad headaches and between 76% and 83% reported nausea or vomiting. Notably, these symptoms were least prevalent among the members of Group I. For the other, less commonly reported symptoms of withdrawal, the preva-

Table 2. Prior-to-Past Year Alcohol Problems for Selected Categories of Current and Former Drinkers 18 Years of Age and Over, According to History of DSM-IV Alcohol Dependence

	Formerly Dependent		
	Past-year Abstainers (Group I)	*Past-year Drinkers with Neither Abuse nor Dependence (Group II)*	*Never Met Criteria for Dependence (Group III)*
Mean number of symptoms of DSM-IV alcohol dependence	12.8 (0.2)[a,b]	8.2 (0.1)[b]	2.0 (<0.1)
Mean number of positive criteria for DSM-IV alcohol dependence	5.2 (0.1)[a,b]	4.0 (<0.1)[b]	1.1 (<0.1)
% positive for:			
Withdrawal	93.9 (1.0)[a,b]	90.5 (0.7)[b]	29.0 (0.5)
Drank more/longer than intended	93.1 (0.9)[b]	92.4 (0.6)[b]	30.5 (0.5)
Desire/attempts to cut down/stop drinking	88.5 (1.3)[a,b]	76.3 (1.0)[b]	20.8 (0.4)
Tolerance	80.0 (1.4)[b]	79.2 (0.9)[b]	20.5 (0.4)
Continued use despite physical/psychological problems	61.8 (1.7)[a,b]	27.2 (1.1)[b]	2.0 (0.1)
Much time spent drinking	57.6 (1.8)[a,b]	26.1 (1.0)[b]	1.8 (0.1)
Important activities given up	45.8 (1.8)[a,b]	12.4 (0.8)[b]	0.8 (0.1)
Mean number of withdrawal symptoms among those positive for withdrawal	3.9 (0.1)[a,b]	3.0 (<0.1)[b]	2.4 (<0.1)
Of those positive for withdrawal, % reporting:			
Bad headaches	82.6 (1.5)	87.3 (0.8)	87.6 (0.5)
Nausea or vomiting	76.3 (1.7)[a,b]	83.4 (0.9)	82.6 (0.6)
Depression, irritability, nervousness	70.4 (1.7)[a,b]	45.6 (1.2)[b]	19.6 (0.6)
Sweating or heart palpitations	48.6 (1.8)[a,b]	28.1 (1.1)[b]	16.2 (0.5)
Shaking or tremor	46.4 (1.8)[a,b]	22.1 (1.0)[b]	10.7 (0.5)
Problems sleeping	37.7 (1.8)[a,b]	28.2 (1.1)[b]	19.9 (0.6)
Hallucinations	25.0 (1.8)[a,b]	7.0 (0.7)[b]	3.4 (0.3)
Fits or seizures	7.7 (1.1)[a,b]	0.7 (0.2)	0.5 (0.1)

Notes. a. Estimates are significantly different from those for Group II ($p < .002$)
b. Estimates are significantly different from those for Group III ($p < .002$)

Table 3. Percentage of Current and Former Drinkers 18 Years of Age and Over Who Met Selected Criteria for Prior-to-Past-Year DSM-IV Alcohol Dependence, Conditional upon Presence of other Criteria, According to History of DSM-IV Alcohol Use Disorders

	Percentage Who Also Were Positive for:						
Of Individuals Positive for Criteria Listed Below:	*Withdrawal*	*Drank More/ Longer Than Intended*	*Desire/ Attempts Cut Down/Stop*	*Tolerance*	*Continued Use*[a]	*Much Time Spent Drinking*	*Important Activities Given Up*
Past-year abstainers (Group I):							
Withdrawal	100.0 (0.0)	92.7 (0.9)[b]	88.1 (1.3)[a,b]	79.6 (1.4)[b]	63.6 (1.8)[a,b]	59.2 (1.8)[a,b]	47.3 (1.8)[a,b]
Drank more/longer than intended		100.0 (0.0)	89.3 (1.3)[a,b]	80.3 (1.4)[b]	62.8 (1.8)[a,b]	59.0 (1.8)[a,b]	46.4 (1.8)[a,b]
Desire/attempts to stop/cut down			100.0 (0.0)	80.3 (1.4)[b]	62.7 (1.8)[a,b]	58.8 (1.9)[a,b]	47.4 (1.9)[a,b]
Tolerance				100.0 (0.0)	65.1 (2.0)[a,b]	62.7 (1.9)[a,b]	50.7 (2.1)[a,b]
Continued use[a]					100.0 (0.0)	75.4 (2.1)[a,b]	62.8 (2.2)[a,b]
Much time spent drinking						100.0 (0.0)	72.0 (2.1)[a,b]
Important activities given up							100.0 (0.0)
Past-year drinkers with neither abuse nor dependence (Group II):							
Withdrawal	100.0 (0.0)	92.3 (0.7)[b]	74.5 (1.0)[b]	77.8 (1.0)[b]	27.6 (1.1)[b]	27.0 (1.1)[b]	13.0 (0.8)[b]
Drank more/longer than intended		100.0 (0.0)	75.0 (1.0)[b]	78.5 (1.0)[b]	27.4 (1.1)[b]	26.4 (1.1)[b]	12.8 (0.8)[b]
Desire/attempts to stop/cut down			100.0 (0.0)	76.3 (1.1)[b]	29.0 (1.2)[b]	26.7 (1.1)[b]	12.9 (0.9)[b]
Tolerance				100.0 (0.0)	27.7 (1.2)[b]	26.3 (1.2)[b]	12.6 (0.8)[b]
Continued use					100.0 (0.0)	53.6 (2.3)[b]	31.0 (2.0)[b]
Much time spent drinking						100.0 (0.0)	35.6 (2.2)[b]
Important activities given up							100.0 (0.0)
Never met criteria for dependence (Group III):							
Withdrawal	100.0 (0.0)	56.0 (0.7)	31.5 (0.7)	32.9 (0.8)	5.2 (0.3)	4.9 (0.3)	2.2 (0.2)
Drank more/longer than intended		100.0 (0.0)	34.2 (0.7)	34.1 (0.8)	4.9 (0.3)	4.3 (0.3)	2.1 (0.2)
Desire/attempts to stop/cut down			100.0 (0.0)	40.1 (0.9)	6.3 (0.4)	5.5 (0.4)	2.8 (0.3)
Tolerance				100.0 (0.0)	6.0 (0.4)	5.3 (0.4)	2.9 (0.3)
Continued use					100.0 (0.0)	31.6 (2.5)	20.7 (2.5)
Much time spent drinking						100.0 (0.0)	25.1 (2.6)
Important activities given up							100.0 (0.0)

Notes. a. Estimates are significantly different from those for Group II ($p < .002$)
b. Estimates are significantly different from those for Group III ($p < .002$)

lences decreased from Group I to Group III. Members of Group I who were positive for withdrawal syndrome reported sleep problems less frequently than they reported shaking, whereas in Groups II and III sleep problems were the more common of these two symptoms.

Table 3 examines patterns of association among the criteria for dependence. Among individuals positive for any given criterion during the period prior to the past year, with these index criteria ranked in order of decreasing prevalence, it shows the conditional probabilities of being positive for each of the less commonly endorsed criteria. It is important to note that these conditional probabilities do not reflect chronological staging. As the work by Langenbucher and Chung (1995) has demonstrated, symptoms or criteria that are less prevalent within a population do not always occur at a later stage in the development of alcoholism. Rather, this table serves to further illustrate the differences in arrays or combinations of positive criteria that distinguish Groups I, II and III.

The data in this table reveal the interchangeability of the four most common criteria for dependence with respect to predicting the conditional probabilities of additional positive criteria. Irrespective of group, the probability of having any second positive criterion was about the same regardless of which of the first four criteria—withdrawal, drinking more/longer than intended, desire/attempts to cut down/stop drinking, or tolerance—was used as the index. The one exception to this general rule occurred among the never dependent (i.e., members of Group III), for whom the index criterion of desire/attempts to stop/cut down on drinking was more predictive of tolerance than were either withdrawal or drinking more/longer than intended. Indexing on the presence of either continued use or time spent drinking as opposed to one of the four most commonly endorsed criteria substantially increased the conditional probabilities of satisfying the other less prevalent criteria. Comparing the three analytic subgroups, it is evident that Group I is distinguished from Group II by higher conditional probabilities of the less common criteria among the former. In contrast, Group II is differentiated from Group III by the former's higher conditional probabilities of *all* the criteria.

As Tables 1 through 3 have indicated, symptoms of the alcohol dependence syndrome form a continuum, and the cutoff for establishing a diagnosis of dependence is necessarily an arbitrary one. Moreover, the association between the patterns in array of dependence

Table 4. Percentage of Formerly Dependent Individuals 18 Years of Age and Over with Selected Problem Arrays and Who Would be Reclassified Under Selected Options for Revising DSM-IV Dependence Criteria, According to Type of Recovery

	Formerly Dependent	
	Past-year Abstainers (Group I)	*Past-year Drinkers with Neither Abuse nor Dependence (Group II)*
% satisfying only three prior-to-past year dependence criteria	18.8 (1.4)[a]	41.4 (1.1)
% satisfying only one other prior-to-past-year criterion in addition to withdrawal and drank more/longer than intended	11.6 (1.1)[a]	28.3 (1.1)
Of those positive for prior-to-past-year withdrawal:		
% with only two symptoms	23.4 (1.5)[a]	36.1 (1.2)
% with only bad headaches and nausea/vomiting	13.4 (1.3)[a]	24.7 (1.0)
% who would be reclassified as no longer meeting criteria for prior-to-past-year dependence under:		
Option 1[b]	11.6 (1.1)[a]	28.3 (1.1)
Option 2[c]	12.6 (1.2)[a]	22.4 (0.9)
Option 3[d]	19.4 (1.4)[a]	40.7 (1.1)

Notes. a. Estimates are significantly different from those for Group II ($p < .002$)

b. Option 1 would require at least three positive criteria for dependence, including at least two in addition to withdrawal and drinking more/longer than intended.

c. Option 2 would require at least two positive symptoms for withdrawal, including at least one in addition to bad headaches and nausea/vomiting.

d. Option 3 would combine the modifications of both Options 1 and 2, i.e., it would require at least two positive criteria in addition to withdrawal and drinking more/longer than intended and at least one positive symptom in addition to bad headaches and nausea/vomiting.

criteria and past-year status suggests that the cutoff used for determining dependence may be associated with the prognosis for different types of recovery. As shown in Table 4, 18.8% of the individuals in Group I and 41.4% of those in Group II satisfied just the minimum of three criteria required for alcohol dependence during the period prior to the past year. For 11.6% of the former and 28.3% of the latter, the diagnosis was based on just one positive criterion in addition to withdrawal and drinking more/longer than intended—criteria that were so lacking in specificity as to be satisfied by almost one third of never-dependent individuals. Of individuals positive for withdrawal syndrome, 23.4% of Group I and 36.1% of Group II satisfied just the minimum requirement of two symptoms, and for 13.4% and 24.7%, respectively, the only two positive symptoms were bad headaches and nausea/vomiting.

Table 4 also compares the effects of three options for tightening the criteria for alcohol dependence. These are geared towards eliminating positive diagnoses based on combinations of symptoms and criteria that are fairly common among all drinkers, including those not meeting the existing DSM-IV criteria for dependence. Under Option 1, the requirement of three or more positive criteria would be modified to stipulate that at least two of those criteria be in addition to withdrawal and drinking more/longer than intended. Thus, individuals meeting those two criteria and just one other would no longer meet the requirements for dependence. Under Option 2, the requirement of two or more positive symptoms for withdrawal would be augmented to include the requirement that at least one of those symptoms be in addition to bad headaches and nausea/vomiting. Thus, individuals with headaches and nausea/vomiting as their only two withdrawal symptoms would no longer be positive for withdrawal. Option 3 combines the requirements of both Options 1 and 2.

If the criteria for alcohol dependence were modified as per Option 1, 11.6% of the individuals in Group I and 28.3% of those in Group II would no longer be classified as having been dependent in the period prior to the past year. Option 2 would result in 12.6% of Group I and 22.4% of Group II being reclassified as not having been dependent, and Option 3 would reclassify 19.4% of Group I and 40.7% of Group II. Thus, nearly half of the formerly dependent individuals who achieved recovery from alcohol use disorders despite continuing to drink—those who might be considered as false positives relative to the disease model—would no longer be considered as dependent with these modifications to the diagnostic criteria.

DISCUSSION

This study corroborates earlier findings that severity of dependence affects not only the prognosis for recovery (Babor, Cooney, and Lauerman, 1987; Langenbucher, Sulesand, Chung, and Morgenstern, 1996) but also the type of recovery, e.g., the probability of a return to moderate drinking (Polich, Armar, and Braiker, 1980). It indicated that desire/attempts to cut down/stop drinking were slightly more prevalent among past-year abstainers than among individuals who were drinking without abuse or dependence but that the criteria which most strongly distinguished these two categories were: (a) continued use of alcohol despite physiological or psychological problems, (b) time spent drinking and (c) important activities given up in favor of drinking. The proportions of individuals who satisfied these three criteria were considerably lower than has been reported in recent clinical studies (Langenbucher and Chung, 1995; Morgenstern, Langenbucher, and Labouvie, 1994) in which they were endorsed by more than two thirds of the respondents, a contrast that again reiterates the importance of these specific criteria as indicators of severity and as predictors of entry into treatment.

The physiological criteria of withdrawal and tolerance, in addition to drinking more/longer than intended, did not discriminate at all between the members of Groups I and II. Thus within this general population sample, the distinction between physiological and non-physiological dependence that was reintroduced in the DSM-IV criteria appears to be unrelated to the path by which remission was achieved. This finding is consistent with results of an analysis based on a clinical sample (Langenbucher et al., 1997), in which short-term relapse was not associated with the distinction between physiological and non-physiological dependence. The authors of that study concluded that "this failure was due to operational problems of physiological dependence in DSM-IV, rather than to lack of conceptual merit for physiological dependence, per se, as a course modifier."

The options for tightening the diagnostic criteria for dependence that were presented in this paper merit consideration, but they were intended primarily to illustrate how the existing criteria contribute to the seemingly anomalous finding that many individuals formerly classified with alcohol dependence appear to return to drinking without symptoms of abuse or dependence. Other ways in which the criteria might be modified to address the situation include applying a more stringent definition of the frequency with which problems, particularly withdrawal symptoms, must oc-

cur (Caetano, 1996) or in tying the withdrawal symptoms more explicitly to times when the respondent attempted to reduce or was deprived of his usual alcohol intake.

One of the more striking findings of this study was that the members of Group II, i.e., the individuals who appear to have effected a return to moderate drinking, had a younger average age at onset of dependence than those in Group I who had achieved recovery via abstinence. Typologies of alcohol dependence, for example the Type I vs. Type II distinction (Cloninger, 1987), Type A vs. Type B distinction (Babor et al., 1992) and early- vs. late-onset distinction (Buydens-Branchey et al., 1989), have consistently demonstrated that alcoholic subtypes with early onsets of alcoholism tend to be those with higher concentrations of familial alcoholism, more severe dependence symptoms and poorer prognoses for recovery. In contrast, this study found that the recovery group with the youngest ages at onset of dependence had the lowest concentrations of familial alcoholism and the fewest popular dependence symptoms. This finding is consistent with an earlier analysis of the NLAES data, which found that age at onset of dependence was inversely associated with the odds of recovery through abstinence as opposed to drinking without abuse or dependence, after controlling for sociodemographic characteristics, drinking history and duration, and severity of dependence (Dawson, 1996a). The finding that early onset was overrepresented among those with a return to moderate drinking suggests several possible interpretations.

First, although inconsistent with most alcohol typologies, these data are consistent with Zucker's formulation of four types of alcoholism (Zucker, 1987), including one which is developmentally limited. According to Zucker, this developmentally limited type of alcoholism is somewhat more common among males than females, is an extension of adolescent problem drinking that resolves itself in young adulthood with the successful assumption of adult role responsibilities (spontaneous remission), and is associated with infrequent treatment entry. Because such alcoholics are infrequently treated, their prevalence may be far greater than could be estimated on the basis of clinical samples and could account for the both the high proportion of formerly dependent individuals who had returned to moderate drinking and the low age at onset of dependence in this group. Some of the studies cited earlier, particularly those that examined the continuity of drinking problems from adolescence to adulthood (Fillmore and Midanik, 1984; Temple and Fillmore, 1985-86) have already provided support for the existence of such an alcoholic subtype. The NLAES did not collect data on antisocial personality disorder, but if such data were available, they might prove useful in distinguishing those early onset cases that are developmentally limited from those that correspond to the classic Type II/Type B profile.

At the same time, the earlier onset of dependence in Group II brings to mind the concern (Room, 1977) that many of the symptoms of alcohol dependence could easily be confused with the physiological responses of young, inexperienced drinkers to acute episodes of heavy drinking and to the observation that the symptoms of withdrawal syndrome overlap to some extent with those of a simple hangover (Cameron, 1996). Headaches and nausea/vomiting, in particular, are characteristic of hangovers and thus may be associated with the type of binge drinking that is more common in younger than older drinkers. The importance of these issues with respect to age at onset of dependence was underscored by age patterns in the declassification for prior-to-past-year dependence that occurred under the options for tightening the dependence criteria that were examined in this analysis. For example, when satisfying the withdrawal criterion was revised to require more than just headaches and nausea/vomiting as symptoms, the individuals who were declassified for prior dependence had an average onset of dependence that was more than two years younger than those who retained their dependence classification (data not shown).

The widespread occurrence of such ambiguous symptoms among young drinkers may be one factor in understanding why the average age at onset of dependence in this population sample (Dawson, 1996b) was so much younger than is commonly seen in clinical samples and why the sample contained so high a proportion of dependent cases that would be considered as false positives in comparison to the disease concept of alcoholism and its tenet of irreversibility. Whether some of these cases would also be considered as false positives relative to a clinical assessment of dependence is another important question that could not be addressed by this analysis but which would be a worthwhile area for additional research. The challenge of such a study would of course be to find a general population sample with a sufficient number of dependent cases to ensure a diverse array of dependence symptoms.

In interpreting the results of this study, the issue of retrospective recall must be addressed, particularly in regard to the age difference between the members of Groups I and II. To make certain that the differences between the groups were not solely due to age differ-

ences, which might represent differential accuracy of recall or genuine cohort effects, the data presented in Tables 1 and 2 were rerun after standardizing for age, sex and race (data not shown). This procedure had little effect on most of the variables. For example, the proportion of individuals with a positive family history was 82.4% in Group I and 73.6% in Group II after standardization, as opposed to 82.6% and 74.1% before standardization. All the pairwise differences between Groups I and II retained their level of significance except two, the mean age at onset of heaviest drinking and the percentage with onset of dependence before age 21.

To the extent that respondents between the ages of 18 and 21 at the time of interview could *only* have been formerly dependent if they had experienced dependence before age 21, these 'age at onset' variables are necessarily sensitive to the age distribution of respondents. However, this sensitivity calls attention to the fact that differences attributed to age at onset of dependence might also reflect cohort effects or the fact that some of the younger members of Group II might prove unable to sustain nonproblematic drinking by the time they reach the ages of the members of Group I. Obviously, recovery from alcohol dependence is an ongoing process. Individuals drinking without abuse or dependence at one point in time may slip back into abuse or dependence at a subsequent point and may ultimately choose the recovery strategy of abstinence. Ideally, a study of factors discriminating between types of recovery should be based on longitudinal data, which not only could control for the interval since onset of dependence and recovery but could determine whether the important discriminating factors varied over these intervals.

Despite the limitations of this study that reflect its cross-sectional design, the size, reliability and statistical power of the data base employed in this analysis permitted an examination of dependence symptom patterns that would not have been possible with a smaller sample—an examination that elucidated some of the specifics of the association between severity of symptoms and prognosis for recovery from DSM–IV alcohol dependence. These data strongly supported the concept of a developmentally limited form of alcohol dependence that may resolve itself in adulthood without need for treatment. While the characteristics of this type of recovery may not be generalizable to the prognosis for recovery among individuals entering treatment, these findings suggest that persons with relatively low levels of dependence severity, particularly those who have not yet experienced continued use despite physical/psychological problems, much time spent drinking or giving up important activities in favor of drinking, may be suitable candidates for treatment options that include alternatives to lifelong abstinence.

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The Influence of Comorbid Major Depression and Substance Use Disorders on Alcohol and Drug Treatment: Results of a National Survey

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INTRODUCTION

The co-occurrence of alcohol use disorders, drug use disorders, and major depression has frequently been reported in alcoholic, drug abuse, and psychiatric patient samples (Allen and Francis 1986; Demilio 1989; El-Guebaly 1990; Ross et al. 1988; Rounsaville et al. 1982). Significant associations between substance use disorders and major depression have also been found in general population surveys (Regier et al. 1990; Robins et al. 1988; Weissman and Meyers 1980), but the magnitude is much lower than that reported in clinical samples. This suggests that people with comorbid substance use disorders and major depression may be more likely to seek alcohol or drug treatment than those without such comorbidities. However, to date, no studies have examined the impact of comorbidity on alcohol or drug treatment in the population of greatest clinical and policy relevance, that is, among those persons with an alcohol use disorder or drug use disorder not found in the treated population.

The purpose of this study was to separately compare the comorbidity status of persons with alcohol and drug use disorders who did or did not seek alcohol or drug treatment, respectively. Separate comparisons were also examined for major types of treatment facilities, including 12-step group programs and inpatient and outpatient facilities.

METHODS

Sample

The study was based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), a nationwide household survey sponsored by the National Institute on Alcohol Abuse and Alcoholism (Grant et al. 1992). Field work for the study was conducted by the Bureau of the Census. During the survey, direct face-to-face interviews were conducted with 42,862 respondents, 18 years of age and older, in the contiguous United States and the District of Columbia. The household response rate for the NLAES was 91.9 percent, and the person response rate was 97.4 percent.

The NLAES featured a complex multistage design (Massey et al. 1989). Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probabilities proportional to size. Approximately 2,000 PSUs comprised the 1992 NLAES sample, 52 of which were self-representing—that is, selected with certainty. Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically for each sample. Oversampling of the black population was accomplished at this stage of sampling in order to have adequate numbers for analytic purposes.

Segments were then divided into clusters of approximately four to eight housing units, and all occupied housing units were included in the NLAES. Within each household, one randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18 to 29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this heavier substance-abusing population subgroup. This subgroup of young adults was sampled at a ratio of 2.25 percent to 1.00.

Diagnostic Assessment

The survey questionnaire, the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS) (Grant and Hasin 1992), included an extensive list of symptom questions that operationalized

Reprinted from Treatment of Drug-Dependent Individuals with Comorbid Mental Disorders, *L.S. Onken, J.D. Blaine, and A.M. Horten, Jr., Editors, National Institute on Drug Abuse Research Monograph No. 172, pp. 4–15, 1997, NIH Pub. No. 97–4172.*

the *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed. (DSM-IV) (American Psychiatric Association 1994) criteria for alcohol/drug use disorders and major depression. These questions are described in detail elsewhere (Grant et al. 1994). Past year DSM-IV drug-specific diagnoses of abuse and dependence were first derived separately for alcohol, sedatives, tranquilizers, opioids (other than heroin), amphetamines, cocaine (and crack cocaine), cannabis (and THC and hashish), heroin, methadone, and hallucinogens. A composite measure of any of these drug use disorders (except alcohol) was then constructed.

Consistent with the DSM-IV, an AUDADIS diagnosis of alcohol or drug abuse required that a person exhibit a maladaptive pattern of substance use leading to clinically significant impairment or distress, as demonstrated by at least one of the following in any 1 year: (1) continuing to use despite a social or interpersonal problem caused or exacerbated by the effects of use, (2) recurrent use in situations in which substance use is physically hazardous, (3) recurrent use resulting in a failure to fulfill major role obligations, or (4) recurrent substance-related legal problems. An AUDADIS diagnosis of substance dependence required that a person meet at least three of seven criteria defined for dependence in any 1 year, including: (1) tolerance; (2) avoidance of withdrawal; (3) persistent desire or unsuccessful attempts to cut down or stop using; (4) spending much time obtaining a drug, using it, or recovering from its effects; (5) giving up or reducing occupational, social, or recreational activities in favor of use; (6) impaired control over use; and (7) continuing to use despite a physical or psychological problem caused or exacerbated by use.

Diagnoses of alcohol and drug abuse and dependence also satisfied the clustering or duration criteria of the DSM-IV. The duration criteria of the DSM-IV include the requirement for a clustering of symptoms within any 1-year period, in addition to associating duration qualifiers with certain abuse and dependence symptoms. The duration qualifiers are defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often," and "persistent" appearing in the description of the diagnostic criteria.

Consistent with the DSM-IV, the AUDADIS diagnosis of major depression required the presence of at least five depressive symptoms (inclusive of depressed mood or loss of pleasure and interest) nearly every day for most of the day during any 2-week period. Social and/or occupational dysfunction must also have been present during the disturbance, and episodes of major depression exclusively due to bereavement or physical illness were ruled out. The reliabilities of the diagnoses of DSM-IV alcohol and drug use disorders and major depression were 0.73, 0.80, and 0.65, as determined from an independent test-retest study conducted in a general population sample (Grant et al. 1995).

Alcohol and drug treatment

Respondents in the survey were asked if, during the past year, they had gone anywhere or seen anyone for problems related to their drinking. To more completely capture the entire alcohol help-seeking population, respondents were specifically instructed to indicate any help they had received for their drinking, including help for combined alcohol and drug use if alcohol was the major problem for which they sought help. Alcohol treatment sources were defined broadly and respondents were asked to indicate separately whether they sought help from 23 different treatment sources: inpatient alcohol and/or rehabilitation programs and inpatient wards of general or psychiatric hospitals; outpatient clinics and alcohol and/or drug detoxification units; 12-step groups including Alcoholics Anonymous, Narcotics or Cocaine Anonymous, or Alanon; social services; and various health professionals such as psychiatrists, psychologists, social workers, and the clergy. Respondents receiving help from any of these sources during the past year constituted the alcohol treatment group examined in this study. The drug treatment measure included the same range of treatment sources as described for alcohol, but information was solicited from respondents regarding help they had received for a drug problem, including help for combined drug and alcohol use if use of a drug or drugs was the major problem for which they sought help.

RESULTS

Tables 1 and 2 separately present the population estimates and prevalence of individuals with past year alcohol and drug use disorders by comorbidity and treatment status. The most striking finding in these tables is the extremely low prevalence of alcohol and drug treatment among those classified with an alcohol or drug use disorder, respectively. Only 1,365,111 (9.9 percent) of the 13,759,846 Americans with alcohol abuse or dependence in the past year sought treatment. Among the 2,855,751 Americans with a past-year drug use disorder, 8.9 percent (N = 253,611) sought treatment for a drug problem.

As shown in table 1, the percentage of respondents with alcohol use disorders seeking alcohol treatment

approximately doubled when a comorbid drug use disorder (from 7.8 to 14.9 percent) or a comorbid major depression (from 7.8 to 16.9 percent) was present. The corresponding percentage was four times as great (35.3 percent) when both a comorbid drug use disorder and major depression were present compared to when they were absent (7.8 percent). The percentage of respondents seeking drug treatment with no comorbid disorder (8.6 percent) was greater than those with an additional alcohol use disorder (5.7 percent). However, the presence of a comorbid major depression with (15.8 percent) or without (18.8 percent) a comorbid alcohol use disorder nearly doubled the percentage of respondents with drug use disorders seeking

Table 1. Number and percentage of respondents with alcohol use disorder by comorbidity and treatment status: United States, 1992

Comorbidity status	No alcohol treatment	Alcohol treatment		
	N	N	Percent	(SE)
Any alcohol dx (no MDD/no drug dx)	10,141,815	857,915	7.80	(0.68)
Alcohol abuse (no MDD/no drug dx)	4,573,922	199,168	4.17	(0.73)
Alcohol dependence (no MDD/no drug dx)	5,567,893	658,747	10.58	(1.03)
Any alcohol dx (no MDD/any drug dx)	1,223,770	213,373	14.85	(2.24)
Alcohol abuse (no MDD/any drug dx)	445,763	18,829	4.05	(2.13)
Alcohol dependence (no MDD/any drug dx)	778,007	194,544	20.00	(3.10)
Any alcohol dx (MDD/no drug dx)	782,098	159,099	16.90	(2.85)
Alcohol abuse (MDD/no drug dx)	281,788	29,245	9.40	(3.71)
Alcohol dependence (MDD/no drug dx)	500,310	129,854	20.61	(3.65)
Any alcohol dx (MDD/any drug dx)	247,052	134,724	35.29	(5.99)
Alcohol abuse (MDD/any drug dx)	79,547	0	0.00	(0.00)
Any alcohol dependence (MDD/any drug dx)	167,505	134,724	44.58	(7.09)
Totals	12,394,735	1,365,111	9.92	-0.59

KEY: MDD = Major depressive disorder.

Table 2. Number and percentage of respondents with a drug use disorder by comorbidity and treatment status: United States, 1992

Comorbidity status	No drug treatment	Drug treatment		
	N	N	Percent	(SE)
Any drug dx (no MDD/no alcohol dx)	807,981	76,448	8.64	(1.88)
Any drug abuse (no MDD/no alcohol dx)	616,522	36,230	5.55	(1.98)
Any drug dependence (no MDD/no alcohol dx)	191,459	40,218	17.36	(4.25)
Any drug dx (no MDD/any alcohol dx)	1,355,859	81,284	5.66	(1.31)
Any drug abuse (no MDD/any alcohol dx)	1,010,822	11,854	1.16	(0.77)
Any drug dependence (no MDD/any alcohol dx)	345,037	69,430	20.00	(3.85)
Any drug dx (MDD/no alcohol dx)	128,345	24,058	16.90	(5.29)
Any drug abuse (MDD/no alcohol dx)	73,315	6,580	9.40	(3.98)
Any drug dependence (MDD/no alcohol dx)	55,030	17,478	20.61	(5.09)
Any drug dx (MDD/any alcohol dx)	309,955	71,821	35.29	(6.24)
Any drug abuse (MDD/any alcohol dx)	188,530	25,826	0.00	(5.10)
Any drug dependence (MDD/any alcohol dx.)	121,425	45,995	44.58	(7.68)
Totals	2,602,140	253,611	8.88	(1.32)

KEY: MDD = Major depressive disorder.

drug treatment compared to those with no comorbidity. Not surprisingly, the percentage of respondents seeking treatment for an alcohol use disorder was greater when the comorbid drug use disorder was abuse than when it was dependence. A similar trend was observed for comorbid alcohol use disorders among respondents classified with a drug use disorder who sought treatment during the past year.

Tables 3 and 4 present the past-year prevalence of individuals with past-year alcohol and drug use disorders by comorbidity status and specific type of treatment facility. Although the percentage of respondents seeking help from 12-step group programs and inpatient and outpatient facilities increased as a function of comorbidity status, help seeking for an alcohol use disorder in each type of facility increased twofold in the

Table 3. Percentage of respondents in alcohol treatment by comorbidity and treatment status: United States, 1992

Comorbidity status	12-step group	Inpatient	Outpatient	Any treatment
Any alcohol dx (no MDD/no drug dx)	5.4	2.7	4.6	7.8
Alcohol abuse (no MDD/no drug dx)	2.3	1.0	1.9	4.2
Alcohol dependence (no MDD/no drug dx)	7.8	3.9	6.7	10.6
Any alcohol dx (no MDD/any drug dx)	10.8	6.5	9.9	14.9
Alcohol abuse (no MDD/any drug dx)	1.7	1.1	1.2	4.1
Alcohol dependence (no MDD/any drug dx)	15.1	9.0	14.1	20.0
Any alcohol dx (MDD/no drug dx)	10.7	9.1	12.2	16.9
Alcohol abuse (MDD/no drug dx)	7.7	1.4	3.1	9.4
Alcohol dependence (MDD/no drug dx)	12.2	12.9	16.6	20.6
Any alcohol dx (MDD/any drug dx)	20.8	16.8	27.4	35.3
Alcohol abuse (MDD/any drug dx)	0.0	0.0	0.0	0.0
Alcohol dependence (MDD/any drug dx)	26.3	21.2	34.7	44.6

KEY: MDD = Major depressive disorder.

Table 4. Percentage of respondents in drug treatment by comorbidity and treatment status: United States, 1992

Comorbidity status	12-step group	Inpatient	Outpatient	Any treatment
Any drug dx (no MDD/no alcohol dx)	6.0	3.3	7.2	8.6
Any drug abuse (no MDD/no alcohol dx)	3.0	2.2	4.6	5.6
Any drug dependence (no MDD/no alcohol dx)	14.5	6.5	14.5	17.4
Any drug dx (no MDD/any alcohol dx)	4.6	4.4	3.9	5.7
Any drug abuse (no MDD/any alcohol dx)	0.5	0.9	0.0	1.2
Any drug dependence (no MDD/any alcohol dx)	14.9	12.9	13.6	16.8
Any drug dx (MDD/no alcohol dx)	8.4	3.0	10.4	15.8
Any drug abuse (MDD/no alcohol dx)	7.5	0.0	0.7	8.2
Any drug dependence (MDD/no alcohol dx)	9.4	6.3	21.1	24.1
Any drug dx(MDD/any alcohol dx)	12.3	6.0	10.4	18.8
Any drug abuse (MDD/any alcohol dx)	9.6	2.5	2.5	12.1
Any drug dependence (MDD/any alcohol dx)	15.8	10.6	20.6	27.5

KEY: MDD = Major depressive disorder.

presence of a drug use disorder, threefold in the presence of a comorbid major depressive disorder, and fivefold in the presence of both comorbidities. In contrast, help seeking for a drug use disorder decreased in the presence of an additional comorbid alcohol use disorder, but increased 30 percent or remained unchanged in the presence of a comorbid major depressive disorder, and increased 30 to 51 percent in the presence of both comorbid conditions.

Among respondents with alcohol use disorders and comorbid drug use disorders, help was sought more often from 12-step group programs, while outpatient services were more often sought when a comorbid major depressive disorder was involved. For respondents with drug use disorders, help seeking from 12-step group programs and outpatient services were equally likely regardless of comorbidity status.

DISCUSSION

The major findings of this study show that comorbid substance use disorders and major depressive disorder have a major impact on obtaining treatment for an alcohol or drug use disorder regardless of type of treatment facility. In general, respondents with past-year alcohol use disorders were twice as likely to seek help for their alcohol problems in the presence of either a comorbid drug use disorder or a major depression, and five times more likely to seek help when both comorbidities were present. In contrast, a comorbid alcohol use disorder alone did not increase help seeking among respondents with past-year drug use disorders, while help seeking increased twofold for these respondents when a major depressive disorder was present with or without a comorbid alcohol use disorder. These results, in combination, suggest that the severity of an alcohol or drug use disorder may be greater in the presence of a comorbid major depression, thereby increasing help-seeking behaviors. Moreover, the results indicate that the magnitude of the association often cited between substance use disorders and major depression in treated samples may be artificially inflated. That is, this association may represent the greater propensity of respondents with comorbid major depression to seek treatment for a substance use disorder compared to those individuals with no comorbid major depression.

Perhaps one of the most interesting results of this study is the sheer number of respondents with alcohol and drug use disorders missing from the treated population. Only 9.9 percent and 8.8 percent of the respondents classified with past-year alcohol or drug use disorders, respectively, sought treatment. The percentages of respondents not seeking treatment are much lower than the corresponding percentage reported in other general population surveys. For example, in the Epidemiologic Catchment Area (ECA) survey, 21 percent of the respondents with an alcohol use disorder sought treatment while 28 percent of the respondents with a drug use disorder did so during the year preceding the interview (Narrow et al. 1993). The examination of the reasons the majority of individuals with substance use disorders do not seek treatment, regardless of comorbidity status, would require a more in-depth analysis of factors impacting on help seeking than is possible here. Future studies using the present survey data will address this important unexplored issue.

This study helped to answer the fundamental question of whether the association between substance use disorders and major depression observed in clinical settings is artifactual, that is, a function of increased treatment seeking. The findings suggest that the magnitude of the association between substance use disorders and major depression seen in clinical samples is, in part, due to increased treatment-seeking behavior in comorbid individuals. However, it remains unclear whether increased treatment seeking among comorbid individuals is the result of the increased severity of the substance use disorder due to comorbid major depression or of other factors not examined here. Future analyses of the survey data will explore the numerous factors influencing treatment entry, including a full array of sociodemographic variables, enabling variables (e.g., income, availability of health insurance coverage), and need factors that impact on the severity level of both comorbid disorders.

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Barriers to Alcoholism Treatment: Reasons for Not Seeking Treatment in a General Population Sample

Bridget F. Grant

Objective: *The present study reports responses to numerous direct questions related to reasons for not seeking alcoholism treatment given the perceived need for treatment among respondents classified with an alcohol use disorder (*N = *964, 69.8% male, 93.5% nonblack) in a large representative sample of the United States population.* Method: *Data were derived from the 1992 National Longitudinal Alcohol Epidemiologic Survey, a national probability sample of 42,862 respondents, aged 18 years and older, from the noninstitutionalized population of the contiguous states.* Results: *Lack of confidence in the alcoholism treatment system and its effectiveness, stigmatization and denial were identified as significant barriers to alcoholism treatment at the aggregate level. In general, enabling factors such as lack of financial resources or facilities for child care were much less important barriers to care than were individual predisposing factors including attitudes toward alcoholism treatment.* Conclusions: *Important sociodemographic differences in identified barriers to care are discussed in terms of their minimization through proposed changes in education, screening, outreach, detection, and referral patterns in alcoholism treatment delivery systems.*

The majority of individuals with alcohol use disorders do not enter treatment. Given that the estimated ratio of untreated individuals needing treatment to treated individuals ranges from 3:1 to 13:1 (Sobell et al., 1992), there exists a great need to understand what people perceive as the barriers to seeking help. Despite this need, the alcohol literature that examines barriers to care consists mostly of studies conducted in clinical samples (Miller et al., 1988; Thom, 1986) that inappropriately generalize their findings to the untreated or those missing from the alcoholism treatment population. With few exceptions (Wells et. al., 1989), those general population surveys that have focused on barriers to alcoholism treatment have been limited as the result of small sample sizes and number of reasons examined, lack of standardized assessments of alcohol use disorders and failure to present variations in reasons for not seeking treatment by important sociodemographic variables (Cunningham et al., 1993; Hingson et al., 1982).

The present study reports responses to numerous direct questions relating to reasons for not seeking alcoholism treatment once the need for treatment was perceived among respondents with alcohol use disorders in a large representative sample of the United States population. In this study, alcohol use disorders (i.e., abuse and dependence) were classified according to the most recent psychiatric classification, the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) (American Psychiatric Association, 1994), and variations in barriers to alcoholism treatment were examined across sociodemographic subgroups of the population defined by sex, ethnicity and age.

METHOD

Sample

The present study was based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous states, including the District of Columbia. The household response rate was 91.9%, and the sample person response rate was 97.4%, for a total response rate of 90%.

The NLAES consisted of a multistage design (Grant et al., 1994; Massey et al., 1989). Primary sampling units (PSUs) were stratified according to sociodemographic criteria and were selected with probability proportional to size. From a sampling frame of

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approximately 2,000 PSUs, 198 were selected for inclusion in the 1992 NLAES sample, including 52 that were self-representing (i.e., selected with certainty due to their size). Within PSUs, geographically defined secondary sampling units, referred to as segments, were selected systematically for the sample. Oversampling of the black population was accomplished at this stage of sample selection to secure adequate numbers for analytic purposes. Segments were then divided into clusters of approximately four to eight housing units, and all occupied housing units were included in NLAES. Within each household, one randomly selected respondent, 18 years of age or older, was selected to participate in the survey. Oversampling of young adults, 18–29 years of age, was accomplished at this stage of the sample selection to include a greater representation of this subgroup at higher risk for alcohol-related problems. This subgroup of young adults was randomly sampled at a ratio of 2.25 to 1.00.

Diagnostic assessment of alcohol use disorders

Diagnoses of lifetime alcohol abuse and dependence were derived from the NLAES survey instrument, the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS) (Grant and Hasin, 1992). The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV criteria for alcohol abuse and dependence. These questions are described in detail elsewhere (Grant et al., 1995). Although the DSM-IV was not published until 1994, the specific component criteria of the diagnoses of interest were known prior to the conduct of the NLAES (American Psychiatric Association, 1991) and were, therefore, incorporated in their entirety within the AUDADIS. The AUDADIS computer algorithms were used to create the final DSM-IV diagnoses for alcohol abuse and dependence. In an independent test-retest study conducted in the general population, AUDADIS combined diagnoses of lifetime alcohol abuse and dependence were shown to be highly reliable, achieving a reliability (kappa) coefficient of 0.75 (Grant et al., 1995).

Table 1. Percentage (±SE) of respondents reporting reasons for not seeking treatment once the need was perceived, by sex: United States, 1992

Reason	Male (N = 621)	Female (N = 343)	Total (N = 964)
Didn't think anyone could help	9.0 ± 1.2	7.1 ± 1.4	8.4 ± 0.9
Didn't know any place to go for help	3.8 ± 0.8	8.3 ± 1.7*	5.2 ± 0.8
Couldn't afford to pay the bill	10.4 ± 1.4	13.4 ± 2.2	11.3 ± 1.2
Didn't have any way to get there	0.9 ± 0.4	1.0 ± 0.5	0.9 ± 0.3
Didn't have time	8.1 ± 1.6	4.8 ± 1.4	7.1 ± 1.2
Thought the problem would get better by itself	19.2 ± 2.1	22.2 ± 2.7	20.1 ± 1.7
Were too embarrassed to discuss it with anyone	9.9 ± 1.4	14.3 ± 2.1	11.2 ± 1.2
Were afraid of what your boss, friends, family or others would think	6.7 ± 1.2	10.1 ± 1.8	7.7 ± 1.0
Thought it was something you should be strong enough to handle	30.1 ± 2.1	26.1 ± 2.9	28.9 ± 1.8
Were afraid they would put you into the hospital	4.1 ± 0.9	3.6 ± 1.1	3.9 ± 0.7
Were afraid of the treatment they would give you	4.0 ± 0.8	5.8 ± 1.3	4.5 ± 0.7
Hated answering personal questions	4.2 ± 0.9	4.3 ± 1.1	4.2 ± 0.7
The hours were inconvenient	1.1 ± 0.4	1.0 ± 0.5	1.1 ± 0.3
A member of your family objected	0.3 ± 0.1	1.1 ± 0.7	0.5 ± 0.2
Your family thought you should go but you didn't think it was necessary	4.6 ± 1.0	3.6 ± 1.1	4.3 ± 0.8
Can't speak English very well	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
Were afraid you would lose your job	1.9 ± 0.5	2.6 ± 0.9	2.1 ± 0.5
Couldn't arrange for child care	0.0 ± 0.0	1.6 ± 0.6*	0.5 ± 0.1
Had to wait too long to get into program	0.6 ± 0.2	0.9 ± 0.5	0.7 ± 0.2
Wanted to keep drinking or got drunk	13.5 ± 1.7	10.6 ± 1.8	12.6 ± 1.3
Didn't think drinking problem was serious enough	25.1 ± 2.0	19.3 ± 2.5	23.4 ± 1.6
Didn't want to go	5.5 ± 1.0	3.9 ± 1.1	5.0 ± 0.8
Denial	5.2 ± 1.0	2.8 ± 0.8	4.4 ± 0.7
Stopped drinking on own	3.2 ± 0.8	5.2 ± 1.3	3.7 ± 0.7
Drinking a symptom of other problem/situation	0.6 ± 0.3	2.7 ± 1.0*	1.2 ± 0.4
Family/friends helped me to stop drinking	0.7 ± 0.3	0.6 ± 0.4	0.7 ± 0.3

Note: N's are unweighted figures; percentages are weighted figures. *Significant odds ratios ($p < .01$) indicating sex differences. Female sex served as the reference group in logistic regressions.

Consistent with the DSM-IV, an AUDADIS diagnosis of alcohol abuse required that a person exhibit a maladaptive pattern of alcohol use, leading to clinically significant impairment or distress, as demonstrated by at least one of the following in any 1 year: (1) continuing to drink despite a social or interpersonal problem caused or exacerbated by the effects of drinking; (2) recurrent drinking in situations in which alcohol use was physically hazardous; (3) recurrent drinking resulting in a failure to fulfill major role obligations; or (4) recurrent alcohol-related legal problems. An AUDADIS diagnosis of alcohol dependence required that a person meet at least three of seven criteria defined for dependence in any one year: (1) tolerance; (2) withdrawal or avoidance of withdrawal; (3) persistent desire or unsuccessful attempts to cut down or stop drinking; (4) spending much time drinking, obtaining alcohol, or recovering from its effects; (5) giving up or reducing occupational, social or recreational activities in favor of drinking; (6) impaired control over drinking; and (7) continuing to drink despite a physical or psychological problem caused or exacerbated by drinking.

Past year diagnoses of alcohol abuse and dependence also satisfied the clustering and duration criteria of the DSM-IV definitions. These DSM-IV criteria included the requirement for the clustering of symptoms within any 1-year period, in addition to associating duration qualifiers with certain abuse and dependence symptoms. The duration qualifiers are defined as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms "recurrent," "often" and "persistent" appearing in the diagnostic criteria. In addition to requiring the clustering criteria for past year diagnoses of abuse and dependence, prior to the past year diagnoses were also measured as syndromes, or the clustering of the required number of symptoms at the same time that is necessary to achieve a diagnosis. In this study, respondents classified with a lifetime diagnosis encompassed all those individuals who had ever experienced an episode of abuse or dependence, rather than those demonstrating the required number of symptoms of these disorders over the life course.

Reasons for not seeking treatment

During the course of the interview, respondents were asked whether there was ever a time when they thought they should see a doctor, counselor or any

Table 2. Percentage (±SE) of respondents reporting reasons for not seeking treatment once the need was perceived, by ethnicity: United States, 1992

Reason	Black (N = 101)	Nonblack (N = 863)
Didn't think anyone could help	10.2 ± 3.3	8.3 ± 1.0
Didn't know any place to go for help	8.2 ± 3.1	4.9 ± 0.8
Couldn't afford to pay the bill	13.4 ± 3.9	11.1 ± 1.2
Didn't have any way to get there	2.6 ± 1.5	0.8 ± 0.3
Didn't have time	7.4 ± 3.3	7.1 ± 1.2
Thought the problem would get better by itself	12.3 ± 3.4	20.9 ± 1.8
Were too embarrassed to discuss it with anyone	6.1 ± 2.4	11.7 ± 1.2
Were afraid of what your boss, friends, family or others would think	0.3 ± 0.1	8.4 ± 1.1*
Thought it was something you should be strong enough to handle	20.1 ± 4.8	29.7 ± 1.9
Were afraid they would put you into the hospital	5.5 ± 2.8	3.8 ± 0.7
Were afraid of the treatment they would give you	5.4 ± 2.8	4.4 ± 0.7
Hated answering personal questions	3.4 ± 2.1	4.3 ± 0.7
The hours were inconvenient	0.0 ± 0.0	1.2 ± 0.4
A member of your family objected	0.0 ± 0.0	0.6 ± 0.3
Your family thought you should go but you didn't think it was necessary	3.9 ± 2.9	4.4 ± 0.8
Can't speak English very well	0.0 ± 0.0	0.0 ± 0.0
Were afraid you would lose your job	0.4 ± 0.3	2.3 ± 0.5
Couldn't arrange for child care	2.1 ± 1.2	0.3 ± 0.1*
Had to wait too long to get into program	3.2 ± 1.9	0.4 ± 0.2*
Wanted to keep drinking or got drunk	14.9 ± 4.0	12.4 ± 1.4
Didn't think drinking problem was serious enough	27.4 ± 6.3	22.9 ± 1.6
Didn't want to go	8.2 ± 3.1	4.7 ± 0.8*
Denial	0.9 ± 0.5	4.8 ± 0.7*
Stopped drinking on own	2.5 ± 1.5	3.8 ± 0.7
Drinking a symptom of another problem/situation	2.3 ± 1.7	1.2 ± 0.4
Family/friends helped me to stop drinking	0.0 ± 0.0	0.7 ± 0.3

Note: N's are unweighted figures; percentages are weighted figures. *Significant odds ratios ($p < .01$) indicating sex differences. Nonblack served as reference group in logistic regressions.

other health professional for any reason that was related to their drinking, even though they failed to go. This question could have been answered positively both by people who had received help on other occasions and by those who had never sought treatment. Those who answered positively were separately asked whether 21 reasons for not seeking treatment pertained to them. The particular reasons for not seeking treatment in this study were selected from the preexisting literature. Respondents were additionally asked to volunteer their own reasons for not seeking treatment. These responses were coded into 5 categories: denial, stopped drinking on their own, didn't want to go, viewed drinking as a symptom of another problem or situation, and family or friends had helped them stop drinking.

Statistical analyses

The particular subgroup of the NLAES sample of interest consisted of all respondents who were classified with a lifetime diagnosis of DSM-IV alcohol abuse and/or dependence who additionally reported having perceived the need for alcohol treatment at some time in their lives but failed to seek treatment at that time. Because of small sample sizes and the lack of adequate precision, the analyses could not be disaggregated by those who did or did not obtain alcohol treatment at some other time in their lives. Because the NLAES was a complex sample survey consisting of both stratification and clustering design aspects, variance estimation procedures that assume a simple random sample could not be employed. In order to take into account the NLAES sampling design, all standard errors of the estimates of the percentage of respondents endorsing or reporting specific barriers to alcohol treatment presented here were generated using SUDAAN (Research Triangle Institute, 1994), a software program that uses Taylor series linearization to adjust for these sample design characteristics. For the purposes of this study, all percentages presented are weighted figures while the sample sizes shown are unweighted figures.

To test differences across sociodemographic subgroups of the population in reasons endorsed or volunteered for failing to seek treatment, univariate logistic regression analyses were conducted in which a single sociodemographic variable was treated as a predictor of the dichotomous outcome measures of reasons for not

Table 3. Percentage (±SE) of respondents reporting reasons for not seeking treatment once the need was perceived, by age: United States, 1992

Reason	18-29 years (N = 269)	30-44 years (N = 430)	45-64 years (N= 234)	65+ years (N = 31)
Didn't think anyone could help	9.8 ± 2.2	6.8 ± 1.2*	8.8 ± 1.9	19.5 ± 7.2
Didn't know any place to go for help	5.6 ± 1.4	4.3 ± 1.1	5.9 ± 1.8	8.9 ± 5.9
Couldn't afford to pay the bill	11.7 ± 2.2	12.9 ± 1.9	8.6 ± 2.0	2.4 ± 1.6
Didn't have any way to get there	0.6 ± 0.4*	0.8 ± 0.3*	0.4 ± 0.3*	15.4 ± 8.0
Didn't have time	9.4 ± 2.4*	8.1 ± 1.9*	3.1 ± 1.3	1.8 ± 1.3
Thought the problem would get better by itself	19.3 ± 2.8	20.7 ± 2.9	21.2 ± 3.1	9.4 ± 4.5
Were too embarrassed to discuss it with anyone	11.4 ± 2.2	10.4 ± 1.6	12.8 ± 2.6	9.4 ± 5.9
Were afraid of what your boss, friends, family or others would think	11.5 ± 2.3	6.3 ± 1.4	6.1 ± 1.7	5.7 ± 4.2
Thought it was something you should be strong enough to handle	28.2 ± 3.1	31.2 ± 2.8	26.1 ± 3.3	20.8 ± 7.5
Were afraid they would put you into the hospital	3.9 ± 1.4	3.3 ± 0.9	5.4 ± 1.6	1.9 ± 1.2
Were afraid of the treatment they would give you	4.3 ± 1.3	4.4 ± 1.0	5.0 ± 1.7	5.5 ± 3.9
Hated answering personal questions	3.3 ± 1.0	3.2 ± 0.9	7.1 ± 1.8	6.3 ± 4.4
The hours were inconvenient	2.5 ± 1.0	0.4 ± 0.2	0.7 ± 0.5	0.0 ± 0.0
A member of your family objected	0.3 ± 0.2	0.3 ± 0.2	1.2 ± 0.8	0.0 ± 0.0
Your family thought you should go but you didn't think it was necessary	4.1 ± 1.2	2.9 ± 0.9	7.5 ± 2.2	3.7 ± 2.9
Can't speak English very well	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
Were afraid you would lose your job	3.5 ± 1.1*	1.1 ± 0.4*	2.5 ± 1.0*	0.0 ± 0.0
Couldn't arrange for child care	0.9 ± 0.5	0.4 ± 0.2	0.0 ± 0.0	0.0 ± 0.0
Had to wait too long to get into program	0.8 ± 0.4	0.7 ± 0.3	0.5 ± 0.3	0.0 ± 0.0
Wanted to keep drinking or get drunk	11.7 ± 2.2	12.6 ± 2.0	13.9 ± 2.6	10.9 ± 5.5
Didn't think drinking problem was serious enough	24.9 ± 3.1	21.0 ± 2.1	26.1 ± 3.2	23.2 ± 8.1
Didn't want to go	6.8 ± 1.7*	3.4 ± 0.9*	6.5 ± 1.8*	0.0 ± 0.0
Denial	2.8 ± 1.2*	3.8 ± 0.9*	8.3 ± 2.1*	0.0 ± 0.0
Stopped drinking on own	6.0 ± 1.6	3.1 ± 1.0	2.2 ± 0.9	4.6 ± 3.9
Drinking a symptom of another problem/situation	0.7 ± 0.5	1.8 ± 0.8	0.4 ± 0.3	3.9 ± 2.8
Family/friends helped me to stop drinking	1.3 ± 0.6	0.5 ± 0.3	0.4 ± 0.3	0.0 ± 0.0

Note: N's are unweighted figures; percentages are weighted figures. *Significant odds ratios ($p < .01$) in identified age group relative to reference age group of 65 years and older.

seeking treatment. In these analyses, female, nonblacks (including Asians and Pacific Islanders) and respondents in the oldest age group (i.e., aged 65 years and older) served as the reference groups. All logistic regressions were conducted using the SUDAAN LOGISTIC program that also adjusted for the complex sampling design aspects of the NLAES. As the result of multiple comparisons, the significance levels for these analyses were adjusted upward to $p < .01$.

RESULTS

The lifetime prevalence of DSM-IV alcohol abuse and/or dependence in the total sample was 18.2% (n = 7,359). Among those respondents classified with a lifetime DSM-IV alcohol use disorder, only 12.7% (n = 964) responded that they had perceived a need for alcohol treatment at some time in their lives and failed to seek treatment on an occasion. Among the 12.7% of respondents in this study who were classified with a lifetime alcohol use disorder and who identified barriers to alcohol treatment, 69.8% were male and 93.5% were nonblack. Age categories were: 28.2%, 18–29 years; 45.7%, 30–44 years: 23.6%, 45–64 years; 2.5%, 65 years or older.

Table 1 presents the percentages of respondents with lifetime alcohol use disorders who endorsed or volunteered each reason for not seeking treatment, by sex. For both sexes combined, the reasons most frequently endorsed were thought it was something you should be strong enough to handle (28.9%), didn't think their drinking problem was serious enough (23.4%) and thought the problem would get better by itself (20.1%). Approximately 8% to 12% of the respondents endorsed couldn't afford to pay the bill, were too embarrassed to discuss it with anyone, wanted to keep drinking or get drunk, didn't think anyone could help, and were afraid of what your boss, friends, family or others would think. Although very few sex differences in perceived barriers to treatment were observed, women were significantly more likely than men to endorse that they didn't know any place to go for help and couldn't arrange for child care, and to volunteer that drinking was a symptom of another problem or situation.

With regard to ethnicity, nonblacks were more likely than blacks to endorse that they did not seek alcohol treatment when they perceived a need because they were afraid of what their boss, friends, family or others would think (8.4% vs 0.3%), while blacks were more likely than nonblacks to endorse couldn't arrange for child care (2.1% vs 0.3%) and had to wait too long to get into a treatment program (3.2% vs 0.4%) as reasons for not seeking treatment (Table 2). Blacks were significantly more likely than nonblacks to cite that they did not want to go to treatment as a perceived barrier (8.2% vs 4.7%) while nonblacks were significantly more likely than blacks to volunteer that denial prevented them from seeking treatment (4.8% vs 0.9%).

Table 3 presents the percentages of respondents endorsing or volunteering each reason for not seeking treatment given a perceived need to do so by age group. It should be noted that all of the age contrasts presented in Table 3 that included a zero prevalence of endorsement in the reference group (i.e., respondents 65 years and older) were statistically significant. However, in these situations the percentage of respondents in the three younger or nonreference age categories endorsing these reasons was so low as to render the statistical differences between the older age group and three younger age groups not substantially meaningful. This was not the case for volunteered reasons relating to didn't want to go to treatment and denial which demonstrated nonnegligible prevalence among the three younger age groups despite the zero prevalence of reporting in the oldest age group. In addition to being more likely to volunteer that they didn't want to go to treatment and denial as barriers to seeking treatment, the three youngest age groups were significantly less likely than the oldest age group to endorse not having a way to get to treatment as a reason for failing to obtain treatment. Respondents in the 18–29 years old and 30–44 years old age groups were also significantly more likely to endorse not having the time to seek treatment than were the 45–64 years old and 65 years and older age groups. The 30–44 years old group was less likely to endorse didn't think anyone could help as a perceived barrier to help seeking compared to the other three age groups, and respondents in the three youngest age groups more frequently endorsed being afraid they would lose their jobs.

DISCUSSION

This study directly asked respondents why they failed to seek alcoholism treatment when they perceived a need for it. Among the eight most frequently endorsed reasons for not seeking treatment among those respondents classified with an alcohol use disorder was a perception that individuals should be strong enough to handle their drinking problem by themselves, that their drinking problem would get better by itself, and that, despite the perceived need for help, their drinking problem was not serious enough to actually seek treatment or that no one could help. Consistent with

other studies (Cunningham et al., 1993; Hingson et al., 1982), many respondents who expressed the need for help most frequently explained their failure to seek help by the lack of conviction that treatment was really necessary or needed or would be effective. These findings, in combination, suggest that beliefs about the appropriateness of alcoholism treatment and confidence in the treatment establishment are important factors in impeding help-seeking. These results also strongly suggest that removing these barriers to care in the future will require educating the public about the warning signs of alcohol use disorders and what symptoms should be brought to the attention of professionals, what treatment to expect and the success of treatment once received. It is important also that the public become more knowledgeable about the critical role of the individual in achieving treatment success.

In the literature, the concept of denial, which is described as endemic to alcohol use disorders (Baekeland and Lundwall, 1977; Clancey, 1961; DiCicco et al., 1978), is the most commonly cited reason for failure to seek treatment. In this study, three of the most frequently endorsed reasons for failing to seek treatment, the view that an individual's drinking problem would get better itself, the drinking problem was not serious enough and wanting to keep drinking or get drunk, could all be considered differential expressions of denial. Although these barriers to treatment representing denial were not the most frequently endorsed reasons, it must be remembered that our analyses were conducted only among those individuals classified with a lifetime alcohol use disorder who perceived a need for treatment but failed to go on an occasion. This group of respondents comprised only 12.7% of all individuals with a lifetime alcohol use disorder. The remaining 87.3% of respondents with an alcohol use disorder never perceived an unmet need for treatment at all, and of these respondents only 14.1% ever received treatment with the remaining 85.9% never receiving treatment. This striking percentage of respondents with alcohol use disorders who failed to obtain treatment or perceive a need for treatment suggests that denial may serve not only to impede the treatment seeking process, but to impede the perception of a problem altogether.

Two frequently endorsed reasons for failing to seek treatment on an occasion were conceptually related to stigmatization associated with alcohol use disorders: being too embarrassed to discuss it with anyone and being afraid of what a boss, friends, family or others would think. Although stigma is often cited in the literature as an important impediment to seeking treatment (Hingson et al., 1980; Roizen, 1977), it is surprising that the promulgation of the disease concept of alcoholism does not seem to have served its purpose, which is to reduce the stigma associated with alcohol use disorders. It appears that the central message of the disease concept of alcoholism may have gone too far in relieving the individual's personal responsibility for his/her drinking problems. Perhaps there is a need for a new public education response to reducing stigma, one that emphasizes that the development of alcohol use disorders has many causes, some of which may not be directly under the individual's control, while at the same time emphasizing the need for personal responsibility in helping to overcome such disorders, regardless of their origin.

In addition to endorsing their beliefs about the alcoholism treatment establishment, denial and stigmatization, respondents frequently reported not being able to pay the bill as a factor in not seeking treatment despite the perceived need to do so. Unlike the other barriers to treatment identified in this study, not being able to afford alcoholism treatment could be considered an enabling factor, or a factor representing individual financial resources that impact on treatment seeking, rather than a predisposing factor reflecting the attitudes or beliefs of the individual relative to his/her disorder or treatment. Although these findings, taken together, suggest that predisposing factors may be much more important than enabling or structural factors in impeding the help-seeking process, there is reason to believe that efforts to minimize inequity in access to treatment due to lack of financial resources may be more realistic to achieve in the short-term than changes aimed at altering attitudes and beliefs, especially those related to stigma and denial.

Sociodemographic factors were relatively important in influencing failure to seek treatment when the need was perceived. With regard to sex differences in endorsed or volunteered barriers to care, women were significantly more likely than men to endorse that they didn't know any place to go for help. This finding implicated sex differences in referral patterns that hinder help seeking among women. Consistent with the treatment literature, women may be less likely than men to be referred through conventional routes such as physicians and employers (Duckert, 1987; Moore et al., 1989). If women are more likely to rely on referrals by family and friends or learn about treatment programs through advertisements or word of mouth (Beckman and Kocel, 1982), then the results of this study suggest a need to expand the more conventional referral routes for women. Efforts to overcome this barrier for women should focus on increasing awareness and knowledge of

women's drinking problems among physicians, community programs and employee assistance programs.

Women were also more likely than men to endorse not being able to arrange for child care as a reason for failing to seek treatment. Although the percentage of women endorsing this reason was relatively low, this finding underscores the need to minimize the gender-specific impediments to treatment, specifically increasing services for the care of children in women's entry to treatment. It is interesting that women were also more likely than men to volunteer that viewing drinking as a symptom of another problem or situation influenced their failure to seek help when the need was perceived. This result was consistent with clinical studies that have shown that generally women's perception of their drinking problem is different from men's perception of their problem. Women often do not believe that their drinking is a problem, but rather believe that drinking is a symptom of another problem or situation, or a coping response to a particular crisis, problem or social situation (Thom, 1986). Minimization of this barrier to women's help seeking would address reshaping alcohol education aimed toward women and increasing the availability of female-sensitive services that focus on gender-specific problems, social contexts and the roles of women in society. Female-sensitive services should include the components of screening and outreach in addition to treatment and follow-up programs.

With respect to ethnic differences in reported barriers to treatment, more nonblacks than blacks reported stigmatization (i.e., fear of what their boss, family, friends or others would think) and more frequently denial as influencing their failure to seek treatment. More blacks volunteered that their failure not to seek help, once a need was recognized, was the result of not wanting to go. Although the meaning of this volunteered barrier to care is unclear, it may reflect either a cultural expression of denial or a fear of, or lack of confidence in, alcohol treatment or its effectiveness. Unlike nonblacks who more frequently endorsed predisposing beliefs and attitudes as barriers to treatment, blacks were more likely to endorse enabling factors related to their failure to seek help, including having to wait too long to get into an alcohol treatment program and not being able to arrange for child care. Future efforts to minimize these aspects of the service system among blacks may be more feasible in the short term than altering nonblacks' perceptions and beliefs regarding denial and stigmatization of alcohol use disorders.

Age differences were somewhat influential in impeding the help-seeking process. Respondents in the three younger age groups were more likely than the oldest age group to identify denial, fear of losing their jobs, not wanting to go and not having the time as reasons for their failure to seek help given the perceived need to do so. The 30–44 years old age group was less likely than the younger or older age groups to believe that no one could help with their drinking problem, an encouraging finding for this age subgroup of the population most frequently found in the treatment setting. It is interesting to note that the oldest age group was significantly more likely than the younger age groups to endorse that they didn't have a way to get to alcoholism treatment. It seems clear that most age-specific barriers to care identified in this study relate to age-related roles and difficulties of the respondents. The lack of transportation that comes with increasing age and the time limitations of younger respondents are general circumstances that impact on numerous situations including failure to seek treatment for a perceived alcohol-related problem. Sensitivity to these general life circumstances may improve delivery of alcoholism treatment services.

This study was one of the few studies that directly asked respondents in the general population who were classified with an alcohol use disorder their reasons for failing to seek alcohol treatment once they perceived the need. The study identified an extremely large proportion of respondents who met criteria for an alcohol use disorder who never received treatment nor felt that they ought to seek it. Among those respondents who failed to seek treatment once a need was recognized, important aggregate and subgroup-specific barriers to care were identified and proposals for minimizing these barriers were presented. However, it should be noted that this study was based entirely on self-report as to the respondents' alcohol-related symptoms, their treatment history and their perceived need for treatment. In addition, the precision of the estimates in this survey was too low to replicate the analyses on subgroups of respondents who had ever received treatment and those who had not. This precluded an analysis of barriers to alcoholism treatment that may differentially effect delaying alcoholism treatment and failure to seek treatment. Future research aimed at understanding barriers to alcoholism treatment among individuals missing from the treatment population will be critical to our understanding of why more people do not seek or receive alcoholism treatment and to our efforts to minimize the impediments to the help-seeking and treatment processes.

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Variations in the Prevalence of Alcohol Use Disorders and Treatment by Insurance Status

Bridget Grant

INTRODUCTION

Coverage of alcohol treatment services currently varies widely under both public and private health payment plans. Equitable distribution of resources across these sectors has been a concern among policymakers involved in state health reform initiatives. Another major goal has been to quantify the proportion of persons utilizing alcohol treatment services who are not insured as a way to integrate treatment services for the uninsured into a more comprehensive and cost-effective coverage system. However, efforts to improve the provision of alcohol services have been hampered by lack of data on the prevalence of alcohol use disorders. This report provides these estimates using data collected in a recent national survey.

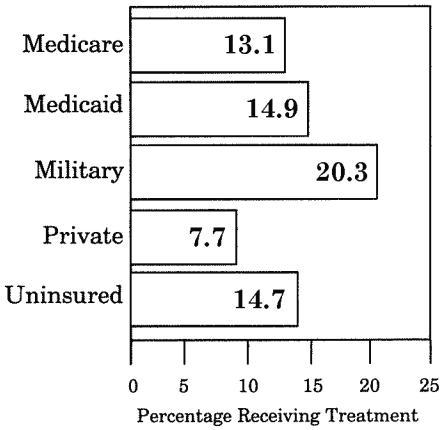
METHODS

In 1992, the National Institute on Alcohol Abuse and Alcoholism sponsored and designed a study of alcohol use and related disorders in the general population. The National Longitudinal Alcohol Epidemiologic Survey (NLAES) included detailed questions on • alcohol intake and alcohol use disorders, • drug use and drug use disorders, • depression, • family history of alcoholism, • physical morbidity associated with alcohol use, and • utilization of alcohol and drug treatment. A broad range of background measures included age, race, sex, education, ethnic origin, marital and fertility history, work history, sources of income and health insurance coverage.

The NLAES was unique in its inclusion of symptom item indicators and follow-up questions that permitted classification of alcohol and drug use disorders according to syndromal definitions appearing in the *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition* (DSM–IV). Respondents were classified into the categories of DSM–IV abuse and/or dependence for the past year. Similarly, health insurance and alcohol treatment measures were based on the past year time frame.

The NLAES sample consisted of 42,862 adults, 18 years of age and older. One adult was selected at random from each household in a representative sample of the civilian, noninstitutionalized population in the United States. Field work for the NLAES was carried out by the U.S. Bureau of the Census, and data were collected in personal interviews conducted in respondents' homes. The household and sample person response rates for the NLAES were 92 and 97 percent, respectively.

Table 1. Percentage of Adults with Alcohol Use Disorders Who Received Treatment, by Insurance Coverage[1]



Source: 1992 NLAES

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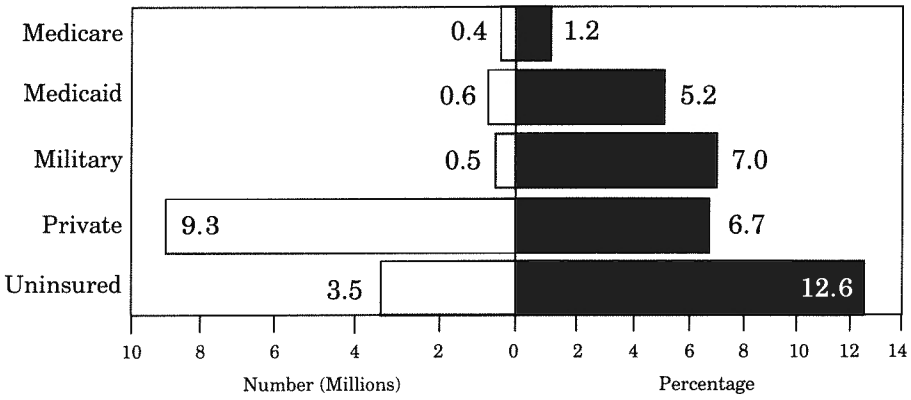
RESULTS

In 1992, there were 9.3 million adults covered by private health insurance who met the DSM–IV criteria for past year alcohol abuse and/or dependence, representing a prevalence rate of 6.7 percent among all persons with private health insurance. The prevalence of alcohol use disorders was almost twice as high among individuals with no health insurance (12.6 percent), but because there were fewer persons in the uninsured category, the total number of uninsured adults with past year alcohol abuse and/or dependence was only 3.5 million. Among persons receiving Medicare, Medicaid, and military health insurance, the numbers with alcohol use disorders were 0.4, 0.6 and 0.5 million, respectively, reflecting prevalences of 1.2 percent, 5.2 percent and 7.0 percent.

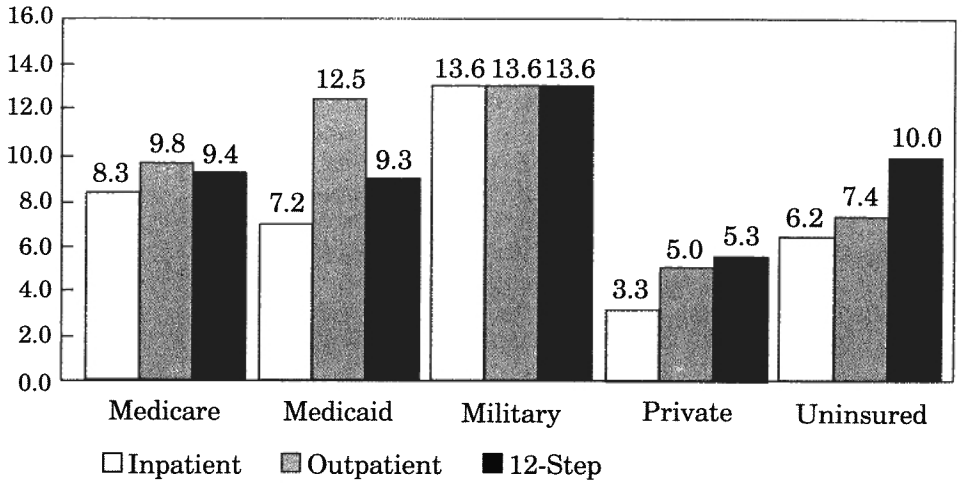
Among persons who met the criteria for past year alcohol abuse and/or dependence, the proportion who received alcohol treatment in that year varied from a low of 7.7 percent among those with private health insurance to a high of 20.3 percent among those in the military health care system. Persons with military health insurance were equally likely to have received treatment in outpatient, inpatient or 12-step programs, but persons with other or no health insurance were less likely to have utilized inpatient services. Individuals covered by Medicare and private health insurance were equally likely to have participated in outpatient or 12-step treatment programs. Among Medicaid recipients,

Table 2. Number and Percentage of Adults with Alcohol Use Disorders[2]



Source: 1992 NLAES

Table 3. Percentage of Adults with Alcohol Use Disorders Who Received Various Types of Treatment



Source: 1992 NLAES

outpatient programs were the most common form of treatment, whereas the uninsured relied most heavily on 12-step programs.

DISCUSSION

A total of 13.7 million adults met the criteria for current DSM–IV alcohol abuse and/or dependence, including 3.5 million with no health insurance.[1] Although the rates of alcohol abuse and dependence were greater among the uninsured than among those covered by private insurance, the largest number of cases requiring alcohol treatment services fell among the 9.3 million persons with private health insurance. The percentage of adults with alcohol use disorders who were in treatment was at least twice as large in the public sector and among the uninsured as for those covered by private health insurance plans. These results indicate a substantial need for treatment services across insurance coverage types, particularly among persons with private health insurance.

Variation in the utilization of inpatient, outpatient and 12-step programs was noted in the public and private sectors. More recipients receiving Medicaid utilized outpatient treatment, whereas inpatient, outpatient and 12-step programs were utilized equally by recipients with other public sector insurance coverage.

These results indicate that access to alcohol treatment should be more equitably distributed across the public and private sectors. However, these findings do not appear to support the growing perception in recent years that the availability of services and types of facilities differs markedly among the private sector, which treats mainly insured individuals, and the public sector, which serves the indigent, uninsured or underinsured.

REFERENCES

[1]The one-year prevalence of total alcohol abuse and dependence in the NLAES sample was 7.41 percent of U.S. adults, ages 18 and older.

[2]While Table 2 indicates that 14.3 million adults have alcohol use disorders, some of those adults have dual insurance coverage and thus were counted twice. The actual number of adults who met the criteria is 13.7 million.

CHAPTER 10
GUIDELINES FOR PUBLIC HEALTH

Reducing Alcohol Use Disorders via Decreased Consumption: A Comparison of Population and High-Risk Strategies

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This study compared three alternative scenarios for effecting a 25% reduction in U.S. alcohol consumption in terms of their respective impacts on the prevalence of alcohol abuse and dependence. The three approaches were (1) an overall 25% reduction in the volume of ethanol intake for all current drinkers, (2) an equivalent absolute reduction taken only among drinkers whose current intake ever exceeds moderate drinking guidelines, and (3) an equivalent reduction taken only among drinkers whose current intake usually exceeds moderate drinking guidelines. The per-occasion cutpoint for moderate consumption was set at the intake level demonstrated to produce psychomotor impairment, and was based on each individual's total body water level. The impact of reducing consumption on the prevalence of alcohol use disorders was estimated by means of a logistic regression model that adjusted for sociodemographic characteristics, family history of alcoholism, and age at first drink, and that took into account interactions between the consumption and other variables. Taking an overall 25% reduction in intake resulted in the same decrease in the prevalence of abuse and dependence (21.7%) as was achieved by taking an equal volume of reduction among only those drinkers whose consumption usually exceeded the moderate drinking cutpoint. Restricting the reduction in consumption to those drinkers whose consumption ever exceeded this cutpoint resulted in a slightly greater reduction in alcohol use disorders, 24.6%.

INTRODUCTION

In 1987, the United States Department of Health and Human Services, with the input of more than 300 national organizations, proposed a series of changes aimed at improving the nation's health by the year 2000 (U.S. Department of Health and Human Services, 1991). These changes encompassed 22 priority areas, including the use of alcohol and other drugs. One explicit goal was to reduce U.S. alcohol consumption to an annual level not to exceed two gallons of ethanol per person aged 14 and over by the year 2000. This represented a decrease of 21% in relation to the consumption level of 2.54 gallons reported for 1987 (Williams et al., 1994). In 1991, the European Member States of the World Health Organization proposed a 25% reduction in alcohol consumption by the year 2000 as a revised target in achieving its strategy for 'attaining good health for all' (World Health Organization, 1992). The justification for these goals was the association between alcohol consumption and health problems, some of which derive from the direct effects of ethanol on various body systems and some of which result from adverse social consequences such as drunken driving and alcohol-related accidents and intentional violence (U.S. Department of Health and Human Services, 1993). Symptoms of physiological, psychological and social consequences of excessive alcohol consumption also characterize the conditions of alcohol abuse and dependence, whose combined prevalence is estimated at 7.4% of the U.S. adult population (Grant et al., 1994a).

An ongoing debate centers on whether policies aimed at decreasing alcohol consumption and related problems should be aimed at the general population or at the group of high-risk drinkers among whom alcohol-related consequences are most prevalent. These alternative approaches are often termed the population strategy and the high-risk strategy, respectively (Rose, 1985). The logic of the latter approach, often advocated by the beverage industry (Maloy, 1984; Meister, 1992; Schneider, 1984; Task Force on Responsible Decisions About Alcohol, 1975), is self-evident based on the far higher rates of mortality, morbidity, dependence and so forth among high-risk drinkers. However, low-risk drinkers, despite their lower rates of

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problems, comprise a far greater proportion of the population. Thus, reductions in consumption among the overall population may be as successful as the high-risk approach in reducing the overall prevalence of alcohol-related problems (Rose, 1992).

In a recent analysis, Norström (1995) compared the impacts of the population and high-risk strategies in terms of the reduction in rates of mortality attributed to liver cirrhosis, accidents and suicide that would derive from a 25% reduction in alcohol consumption in Sweden. He found that the two strategies yielded similar results for deaths from accidents and suicide. While the high-risk strategy yielded better results with respect to deaths from alcohol cirrhosis, additional data suggested that this specific cause of death may have somewhat overestimated the relative potential of the high-risk strategy for mortality from other diseases in that most diseases are not as concentrated in the upper tail of the consumption distribution as is cirrhosis. Thus, his findings did not unequivocally support the superiority of either the population or high-risk strategy with respect to reducing all cause alcohol-related mortality.

This study compares the effects of the population and high-risk strategies on another outcome, the prevalence of alcohol abuse and/or dependence. The decrease in the prevalence of this outcome that would derive from a 25% reduction in alcohol consumption in the United States was estimated using three alternative approaches: (1) an across-the-board reduction of 25% among all current drinkers, (2) an equivalent absolute reduction taken only among drinkers whose current consumption occasionally or usually exceeds a moderate drinking cutpoint, and (3) an equivalent absolute reduction taken only among drinkers whose current consumption usually exceeds a moderate drinking cutpoint. The analysis is based on self-reported data on past-year alcohol consumption and alcohol-related problems collected from a nationally representative sample of U.S. adults who consumed at least 12 drinks in the year preceding interview.

METHODS

Sample

This analysis is based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism and conducted by the U.S. Bureau of the Census. The NLAES collected information on alcohol consumption and alcohol-related problems, drug use and problems, periods of low mood, alcohol and drug abuse treatment, and family history of alcoholism. The NLAES sample consisted of 42,862 U.S. adults 18 years of age and older, selected at random from a nationally representative sample of households. Interviews were conducted in respondents' homes, and proxies were not permitted. The household and sample person response rates for the NLAES were 92% and 97%, respectively.

The NLAES featured a complex, multistage sample design. Primary sampling units were chosen with probability proportional to size, and blacks and young adults 18–29 years of age were oversampled (Grant et al., 1994b). In order to account for the effects of the sample design on the precision of estimates, the results presented in this paper were produced using SUDAAN (Research Triangle Institute, 1993), a software package specifically designed to account for sample design characteristics by using Taylor series linearization techniques for variance estimation.

Abuse and Dependence Measures

The outcome of interest in this analysis is the prevalence of DSM-IV past-year alcohol abuse and/or dependence, which was determined using the Alcohol Use Disorder and Associated Disabilities Interview Schedule or AUDADIS (Grant and Hasin, 1992). The definitions of past-year abuse and dependence have been fully described elsewhere (Grant et al., 1994a). Briefly, a person was considered positive for alcohol abuse if he or she met one or more of the following four criteria for DSM-IV abuse: continued use of alcohol despite social or interpersonal consequences; hazardous use; alcohol-related legal problems; and neglect of role obligations. A person was considered as dependent if he or she met at least three of the seven criteria for DSM-IV dependence: tolerance; withdrawal (including relief or avoidance of withdrawal); persistent desire or unsuccessful attempts to cut down or stop drinking; much time spent drinking or recovering from its effects; important activities reduced or given up in favor of drinking; impaired control over drinking; and continued drinking despite a physical or psychological problem caused or exacerbated by drinking.

As specified by the DSM-IV guidelines, duration qualifiers were imposed on a criterion-specific basis. These qualifiers define the repetitiveness with which symptoms must occur to count as positive toward a diagnosis. Criteria not associated with duration qualifiers were satisfied if a person reported one or more positive symptoms of the criterion over the past year. In order to satisfy a criterion associated with a duration qualifier, a person had to report having experienced at least

one symptom two or more times over the past year or two or more symptoms of the criterion during the same time period. To meet the criterion for withdrawal, which is defined as a syndrome or cluster of symptoms, two or more positive symptoms were required in addition to satisfaction of the duration qualifier.

Alcohol consumption measures

The moderate drinking categories used in this analysis were based on findings indicating that individuals experienced significant levels of psychomotor impairment at ethanol doses of 0.75 g/kg of body weight (Hindmarch et al., 1991; Turner et al., 1981), which corresponds to about three to five standard drinks depending on the size of the drinker. This cutpoint was adjusted to account for the fact that persons with identical body weights have different levels of total body water, depending on their age and sex (Watson et al., 1980). Using a 21-year-old male weighing 160 pounds (72.6 kg) as the standard (since this closely corresponded to the sample from which the impairment data were collected, the cutpoint of 0.75 g of ethanol per kg of body weight was converted to a cutpoint of 0.0534 ounces (oz.) of ethanol per 1 of total body water.

The NLAES questionnaire asked current drinkers to report the usual and heaviest quantities and sizes (oz. of beverage) of beer, wine and alcohol that they had consumed during the past year. Using ethanol conversion factors of .045 for beer, .121 for wine and .409 for liquor (Distilled Spirits Council of the United States, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams et al., 1994), each person's usual and heaviest daily consumption of ethanol from each of those three beverage sources was calculated. Individuals were categorized as never exceeding the moderate drinking cutpoint if neither their usual nor heaviest amounts of ethanol consumed from any beverage source exceeded 0.0534 oz. of ethanol per 1 of total body water. They were categorized as occasionally exceeding the moderate drinking cutpoint if their heaviest intake of one or more of the beverage types exceeded this level, but none of their usual intakes were in excess of the cutpoint. They were categorized as usually exceeding the cutpoint if their usual intake of one or more of the beverage types exceeded the level of 0.0534 oz. of ethanol per 1 of total body water.

Average daily ethanol intake was based on annual intakes for the three beverage types, each of which was calculated as follows: oz. ethanol = (total – heavy drinking days per year x number of drinks per usual drinking day x oz. of beverage in typical drink consumed on usual drinking day x ethanol conversion factor) + (heavy drinking days per year x number of drinks per heavy drinking day x oz. of beverage in typical drink consumed on heavy drinking day x ethanol conversion factor). The beverage-specific volumes were summed over the three beverage types, and the resulting volume was divided by 365 to yield average daily intake.

Analysis

In the first stage of the analysis, moderate drinking cutpoints were estimated for all current drinkers. Individuals' past-year levels of consumption were compared with their cutpoints to estimate the proportions of current drinkers whose consumption never exceeded the moderate consumption cutpoint, whose consumption occasionally exceeded the cutpoint, and whose consumption usually exceeded the cutpoint. Individuals within these three categories were then compared in terms of their average and total consumption levels and their prevalence of alcohol abuse and/or dependence.

In the second stage of the analysis, a multiple linear logistic regression model was fitted to estimate the odds of abuse and/or dependence among current drinkers based on their average daily ethanol intake (using a natural log transformation to linearize the relationship between this measure and the log odds of the outcome) and their status with respect to the moderate drinking cutpoint (represented by two dummy variables for occasional and usual consumption in excess of the cutpoint). Other variables entered into the model on the basis of their known association with alcohol-use disorders were age, sex (effect of male vs. female), race (black vs. nonblack), Hispanic origin (Hispanic vs. non-Hispanic), marital status (currently (married vs. not married), education (college graduate vs. not college graduate), family income, employment status (currently employed vs. all others), family history of alcoholism (positive vs. negative), age at first drink (ages less than 12 recoded to 12 and ages greater than 21 recoded to 21 to reduce the influence of a few outliers) and total body water. Also tested for inclusion in the model were first-order interactions between average daily intake and the other predictor variables. In the first step of the model fitting, backwards stepwise regression techniques were used to eliminate all interactions that were not statistically significant at the $P < 0.05$ level. In the second step, main effects that were not significant were removed unless their inclusion was required to interpret statistically significant interaction terms or to avoid materially affecting the remaining model parameters.

In the third stage of the analysis, the model parameters were used to estimate the prevalence of alcohol abuse and dependence under three different scenarios for reducing per capita alcohol consumption by 25%, that is, by 14.9 million oz. of ethanol per day. The first option entailed reducing all current drinkers' average daily consumption levels by 25%. Under the second option, consumption levels among drinkers who never exceeded the moderate drinking cutpoint were left unchanged, and consumption levels among those who ever (occasionally or usually) exceeded the cutpoint were reduced by 28%. Under the third option, consumption levels were left unchanged among persons who never or only occasionally exceeded the moderate drinking cutpoint and reduced by 41% among persons whose consumption usually exceeded the cutpoint.

For this analysis, it was assumed that the reductions in intake under all three of the options were achieved by reducing the quantity of ethanol consumed on each drinking day (with equal levels of reduction across beverage type) rather than by reducing the number of drinking days per year. Thus, the reduced levels of intake could be used to reclassify drinkers as to whether their consumption never, occasionally or usually exceeded the moderate consumption cutpoints.

In order to estimate the effects of these three options on the prevalence of alcohol abuse and dependence, individuals' consumption levels were reduced as described above, their moderate drinking status was reassessed, and their log odds of abuse and/or dependence were calculated as $\text{logit}_i = \mathbf{x}_{i1}\beta_1 + x_{i2}\beta_{2\cdots} + x_{ik}\beta_k$, where k equals the number of parameters in the model, including the intercept. Individual log odds were converted to individual probabilities using the formula $p_i = 1/[1 + \exp(-\text{logit}_i)]$. The estimated population prevalence of abuse and dependence was equal to the mean of the individual probabilities, $\varepsilon(w_i p_i)/\varepsilon w_i$, where w_i equals the individual case weight adjusted for the slight differential in the prevalence of abuse and dependence among all drinkers (16.7%) compared to the prevalence among the individuals for whom there were no missing data and who were therefore used in the model for estimating the beta parameters (16.8%).

The variance of each individual's log odds was estimated using the standard formula for the variance of a linear combination (Stuart and Ord, 1987): $\text{Var}(\text{logit}_i) = x^2_{i1}\text{Var}(\beta_1)\ldots + x^2_{ik}\text{Var}(\beta_k) + 2x_1x_2\text{Cov}(\beta_1\beta_2)\ldots + 2x_1x_k\text{Cov}(\beta_1\beta_k\ldots + 2x_{k-1}x_k\text{Cov}(\beta_{k-1}\beta_k)$. The variance of each individual's probability was estimated using the delta method (Stuart and Ord, 1987): $\text{Var}(p_i) = \text{Var}(\text{logit}_i)[\exp(-\text{logit}_i)/(1 + \exp(-\text{logit}_i)^2]^2$. Since each case is independent of all others and there is no covariance among cases, the formula for the variance of the total population prevalence is simply $\varepsilon[w_i\text{Var}(p_i)]/\varepsilon w^2_i$, where w_i is once again the adjusted case weight.

RESULTS

Table 1 shows the moderate consumption cutpoints for men and women of different ages and body weights. Assuming an average of 0.5 oz. of ethanol in a standard drink, it can be seen that five or more drinks would exceed the moderate consumption cutpoint for most men and that four or more drinks would exceed the cutpoint for most women. As shown in Table 2, U.S. drinkers were equally distributed among three categories: those whose daily consumption of beer, wine or liquor never exceeded the moderate consumption cutpoint (32.9%), those whose consumption occasionally exceeded the cutpoint (33.3%), and those whose consumption usually exceeded the cutpoint (33.8%).

Table 1. Moderate consumption cutpoints[a] (oz. of ethanol per day) by sex, age and body weight

	Body weight				
	120 lb./54.4 kg.	140 lb./63.5 kg.	160 lb./72.6 kg.	180 lb./81.6 kg.	200 lb./90.7 kg.
Men					
Age 21	1.99	2.16	2.34	2.52	2.70
Age 30	1.94	2.11	2.29	2.46	2.64
Age 45	1.84	2.02	2.19	2.37	2.54
Age 65	1.71	1.89	2.07	2.24	2.42
Women					
All ages	1.51	1.64	1.76	1.89	2.01

[a] Not to exceed ethanol to total body water ratio of 0.0534 (equivalent to 0.75 g of ethanol per kg of body weight for a 21-year-old male weighing 160 lbs.)

Table 2. Percentage distribution of current drinkers 18 years of age and over by frequency with which daily consumption of beer, wine, or liquor exceeds moderate consumption cutpoint[a]

	Total
Consumption never exceeds moderate consumption cutpoint[a]	32.9 (0.5)
Consumption occasionally exceeds moderate consumption cutpoint[a]	33.3 (0.4)
Consumption usually exceeds moderate consumption cutpoint[a]	33.8 (0.5)

Figures in parentheses are standard errors of percentages.
[a]Ethanol to total body water ratio of 0.0534 (equivalent to 0.75 g of ethanol per kg of body weight for a 21-year-old male weighing 160 lbs).

Table 3 illustrates how divergent these three groups were in terms of their consumption levels and prevalence of alcohol abuse and/or dependence. Compared to drinkers whose consumption never exceeded the moderate drinking cutpoint, those whose consumption occasionally exceeded the cutpoint had an average daily ethanol intake over twice as high (0.57 oz. vs. 0.26 oz.), and those whose consumption usually exceeded the cutpoint had an average daily intake five times as high (1.31 oz.). Nine percent of the ethanol intake reported by occasionally immoderate drinkers was in excess of the moderate drinking cutpoint, compared to 45% of the intake of usually immoderate drinkers. While only 2% of persons whose intake never exceeded the moderate drinking cutpoint met the criteria for alcohol abuse and or dependence, the prevalences of these conditions were 15% and 33%, respectively, among those who occasionally or usually exceeded the cutpoint.

Table 4 presents the reduced logistic regression model used to estimate the association between alcohol consumption and the odds of alcohol abuse and or dependence. The underlying effects of consumption that occasionally or usually exceeded the moderate consumption cutpoints were positive, with odds ratios of 3.25 and 4.83, respectively. The excess odds associated with average daily ethanol intake were also positive and were increased among college graduates and persons who were usually immoderate drinkers as indicated by the two positive interactive terms

Table 5 illustrates the effects of the three approaches to reducing consumption that were derived from inputting reduced consumption values into the regression model. Under option 1, in which a 25% reduction was taken in the average daily intake of all current drinkers, the prevalence of abuse and or dependence was reduced from 16.7% to 13.0%, a 22% decrease in prevalence. Under option 2, in which a 28% reduction was taken in the average daily intakes of all drinkers whose consumption occasionally or usually exceeded the moderate drinking cutpoint, the estimated prevalence of abuse and/or dependence was 12.6%, a 25% decrease. Option 3, in which the consumption of usually immoderate drinkers was reduced by 41%, resulted in a 13.0% prevalence of abuse and or dependence, a decrease of 22%.

In order to test whether or not these results would be affected by the choice of the underlying regression model, the techniques for estimating the reduced prevalence of abuse and/or dependence were applied

Table 3. Selected characteristics of current drinkers 18 years of age and over, by frequency with which daily consumption of beer, wine or liquor exceeds moderate consumption cutpoint[a]

	Population size (000's)	Average daily ethanol intake (oz.)	Total daily ethanol intake (000's oz.)	Total daily intake in excess of cutoff (000's oz.)	Prevalence of abuse and/or dependence (%)
All current drinkers	82 464.1	0.72 (0.01)	59 500.3	18 109.0	16.7 (0.4)
Consumption never exceeds moderate consumption cutpoint[a]	27 133.8	0.26 (0.01)	7139.0	0.0	1.9 (0.2)
Consumption occasionally exceeds moderate consumption cutpoint[a]	27 449.5	0.57 (0.01)	15 701.7	1443.5	15.1 (0.6)
Consumption usually exceeds moderate consumption cutpoint[a]	27 880.8	1.31 (0.04)	36 659.6	16 665.5	32.7 (0.8)

Figures in parentheses are standard errors of means/percentages.
[a]Ethanol to total body water ratio of 0.0534 (equivalent to 0.75 g of ethanol per kg of body weight for a 21-year-old male weighing 160 lbs.)

to the parameters from two additional models, one that contained only the consumption variables and one that included additional sociodemographic variables: work status, presence of children in household, residential characteristics, Hispanic origin, and a finer breakdown by marital status that included widowed, divorced and separated. When only the consumption variables were used in the model, the estimated prevalences of abuse and or dependence were lower than those obtained from the model used in this paper, 12.0%, 11.3% and 11.8%, respectively, for reduction options 1 through 3; however, the relative ranking of the reduction options was not strongly affected. When the model containing additional predictor variables was used, the resulting prevalence estimates, 13.1%, 12.6% and 13.1%, were virtually identical to those derived from the model used in this analysis. Thus, conclusions as to the relative benefits of the population and high-risk approaches do not appear to be affected by model selection. As to the actual level of reduction, it should be more accurately predicted by the models that included sociodemographic covariates, which produced a much better fit than the model based on the consumption variables alone.

DISCUSSION

This analysis indicated that reductions in the estimated prevalence of alcohol abuse and/or dependence would be virtually identical regardless of whether a population or high-risk strategy was adopted for effecting the reduction. A marginally greater reduction was obtained by using what might be called a moderate-to-high-risk strategy, that is, by taking the reduction in consumption only among persons whose consumption ever exceeded the cutpoint for moderate drinking. However, in comparing the results of these three strategies, several assumptions of the analysis and limitations of the data must be considered.

One crucial assumption was that the reduction in consumption would be achieved by reducing the number of drinks of each beverage type consumed per drinking day rather than by reducing the number of drinking days or eliminating one or more types of beverage. Had alternative assumptions been made, individuals' reduced intakes might not have had as much impact on their moderate drinking classification. As a result, the reduction in the prevalence of abuse and/or dependence would not have been as great. Since the extent of this reclassification was greatest among drinkers who occasionally or usually exceeded their moderate drinking cutpoints, this assumption may have overstated the results of the high-risk and moderate-to-high-risk strategies relative to the population strategy.

A second critical assumption was that the association between the consumption measures and the outcome measure would remain the same under conditions of reduced consumption. For the symptoms of alcohol dependence that reflect physiological sequelae of ethanol consumption, e.g., tolerance and withdrawal syndrome, this assumption seems fully justified. However, the social and to some extent the psychological consequences that make up the other criteria for abuse and dependence may reflect relative as well as absolute drinking levels. That is, social and psychological reactions to an individual's drinking patterns may be based on how far those patterns deviate from group norms rather than solely on the resulting level of intoxication. If this were the case, then this analysis would be biased in the direction of overestimating the reduction in abuse and/or dependence that would be achieved under all of the alternative reduction strategies. Without knowing the exact changes that might occur in the risk curve, it is impossible to state with certainty what kind of differential effect this source of bias would have on the three reduction strategies.

Related to this issue is the question of how adequately what is essentially a prospective process, the reduction of consumption levels and associated problems, can be predicted on the basis of cross-sectional data. This analysis examines how alcohol problems at time t_1 would be changed if consumption levels at time t_1 were reduced in varying ways. It disregards the fact that reductions in consumption cannot be achieved instantaneously and that, in the course of time between t_1 and t_2, other changes in consumption may have occurred. For example, if individuals with alcohol abuse or dependence tended to increase their levels of consumption over time, the percentage reductions in intake specified in this analysis, which were taken relative to intake at t_1, would not take into account any additional reduction in the level of expected increase that would be required to achieve the reduction in problem levels that was predicted in this analysis.

The NLAES consumption data were collected separately for beer, wine and liquor. The moderate drinking classification used in this analysis was based on the frequency with which daily consumption of any one of these beverages exceeded individuals' moderate drinking cutpoints. Had information been obtained on overlapping beverage consumption (days when more than one type of beverage was consumed), the proportions of drinkers occasionally and usually exceeding the moderate drinking cutpoint would likely have been

higher. Accordingly, the 25% reduction in intake might have led to a greater reclassification of drinkers with respect to the moderate drinking guidelines and a greater relative decrease in prevalence under the high-risk and moderate-to-high-risk strategies. In other words, this limitation of the data may have overstated the relative impact of the population strategy. However, counteracting this possible bias is the fact that the consumption measures used in this analysis were based on intake per drinking day. These intake levels may have exceeded the level of intake per uninterrupted drinking occasion, which would have come closer to approximating the dose relationship used in establishing the moderate drinking cutpoint.

In summary, the assumptions used in this analysis, coupled with lack of information on overlapping beverage consumption, probably led to some overestimation of the reduction in the prevalence of alcohol-use disorders that would be achieved by a 25% reduction in alcohol intake. Since the resulting biases were in opposing direction with respect to which prevention strategy they favored, the relative strengths of the strategies probably were not affected strongly by these assumptions and limitations. This leaves us with the conclusion that neither the population nor the high-risk strategy would be strongly superior to the other in reducing the prevalence of alcohol abuse and dependence and that the choice of a prevention strategy might better be dictated by the relative feasibility of the options.

A 25% reduction in across-the-board ethanol consumption would not be inconsistent with recent U.S. trends in apparent per capita alcohol consumption of all beverages. From an immediate post-prohibition level of 0.97 gallons per capita in 1934, ethanol consumption more than doubled to 2.30 gallons by 1946. In 1947 consumption decreased by 13%, to about the 2-gallon level currently proposed by the U.S. Department of Health and Human Services, and remained at that level until the early 1960's. During the 1960's and 1970's consumption rose by nearly 40%, peaking at 2.76 gallons in 1980 and 1981. In the following decade, consumption decreased by 16% to the most recent estimates of 2.31 gallons for 1991 and 1992 (Williams et al., 1993). However, the fluctuations observed over time in U.S. alcohol consumption may be more strongly reflective of general demographic and economic shifts than of events that could be controlled as part of a prevention strategy.

Population-based strategies for lowering overall alcohol consumption include cost controls (e.g., increased taxes on consumers, retailers or producers), limits on the availability of alcohol (e.g., on the density of retail outlets or the legal availability of liquor by the drink), limits on alcohol advertising, minimum drinking age requirements, and broad-based prevention campaigns (e.g.. warning labels, public service announcements and school programs). Of these, it is primarily cost controls and minimum drinking age requirements that have been shown to have an impact on consumption level. There is little research to date on the impact of limits on availability, and preventive campaigns and warning labels appear to have had

Table 4. Reduced logistic regression model predicting odds of DSM-IV abuse and/or dependence

	Beta	SE	*P*
Intercept	0.6921	0.3563	.056
Main effects:			
Age	−0.0417	0.0029	<.001
Male	0.4170	0.1093	<.001
Black	−0.2955	0.1118	.010
Married	−0.4099	0.0582	<.001
College graduate	−0.0889	0.0713	.217
Total body water	−0.0036	0.0062	.569
Positive family history	0.6112	0.0611	<.001
Age at first drink	−0.0968	0.0138	<.001
Occasionally exceeds moderate consumption cutpoint[a]	1.1788	0.1195	<.001
Usually exceeds moderate consumption cutpoint[a]	1.5766	0.1380	<.001
Average daily ethanol intake[b]	0.6893	0.0505	<.001
Interactions:			
College graduate × average daily ethanol intake[b]	0.1892	0.0716	0.010
Usually exceeds moderate consumption cutpoint[a] × average daily ethanol intake[b]	0.1292	0.0600	0.035

Goodness of fit: Wald $F = 171.4$, df = 11,68, $P < 0.001$; Hosmer and Lemeshow lack of fit statistic 9.38, df = 8, $P = 0.312$.
[a] Ethanol to total body water ratio of 0.0534 (equivalent to 0.75 g of ethanol per kg of body weight for a 21-year-old male weighing 160 lbs.)
[b] Oz. of ethanol on a log scale.

greater cognitive than behavioral effects (see reviews in Institute of Medicine, 1989 and U.S. Department of Health and Human Services, 1994). In addition, while these population approaches are designed to reduce overall rather than high-risk consumption, whether or not they would actually result in an even level of reduction across consumption levels would depend on whether the price elasticity of the demand for alcohol was invariant with respect to intake or declined with consumption as Skog (1985) has argued. This would be a fruitful area for additional research.

The feasibility of the high-risk and moderate-to-high-risk approaches depends on changing the distribution and not just the absolute level of alcohol consumption. In the analysis of alcohol-related mortality cited previously, Norström (1995) found that the reduced dispersion of the alcohol consumption distribution corresponding to the high-risk reduction strategy was similar to that which actually occurred during a 35-year period of alcohol rationing in Sweden. However, rationing is not likely to be perceived as an acceptable or efficacious way of modifying the alcohol consumption distribution within the contemporary United States. Rather, prevention and treatment programs targeted toward reducing heavy overall and per-occasion consumption are the approaches more likely to represent viable alternatives to the population strategy. In a recent review of alcoholism treatment outcomes, Nathan (1986) cited one-year abstinence rates of 50% or higher for good treatment prospects, and evaluations of assorted brief interventions likewise suggest favorable results (see review in Bien et al., 1993).

The feasibility of a substantial reduction in consumption among high-risk drinkers also was demonstrated in a recent analysis of individuals who had met the criteria for DSM-IV alcohol dependence in a period prior to the past year (Dawson, 1996). Twenty-two percent of these formerly heavy drinkers had achieved total abstinence in the past year, and an additional 50% had reduced their consumption to a level matching that of persons who had never been classified as dependent. Most of this reduction occurred among individuals who had never participated in any type of alcohol treatment program. This indicates that even in the absence of widespread treatment efforts, individual heavy drinkers are likely to curtail their consumption over time. Reduction of the proportion of heavy drinkers in the population could therefore be achieved by preventing new individuals from filling the ranks of those dropping out of the heavy drinker category. This directly relates to the synergistic nature of the population and high-risk strategies for reducing alcohol consumption and related problems. As other researchers have pointed out (Norström, 1995; Rose, 1985; Skog, 1985), drinking is a collective behavior, and individual-level changes in the consumption habits of heavy drinkers can only be facilitated by changes that promote a 'drier' consumption environment in the general population. Thus, while the purpose of this paper has been to contrast the relative effectiveness of the population and high-risk strategies, there is in reality no reason to choose one approach or the other when optimum results are most likely to be achieved by permitting both of these strategies to complement each other.

A question remains as to whether these conclusions can be generalized to other cultures or other time periods. In all likelihood, they are fairly limited. First, the underlying associations between consumption, other variables and alcohol use disorders are somewhat culture specific. Thus, the risk of abuse or dependence as-

Table 5. Estimated prevalence and reduction in prevalence of DSM-IV alcohol abuse and dependence under different options for reducing alcohol consumption

	Prevalence	% Reduction in prevalence
Current level of consumption	16.7 (0.010)	NA
Consumption reduced by 25 percent:		
Option 1: Reduction equally distributed among all current drinkers[a]	13.0 (0.009)	21.7
Option 2: Reduction restricted to current drinkers who occasionally or usually exceed moderate consumption cutpoint[b]	12.6 (0.009)	24.6
Option 3: Reduction restricted to current drinkers who usually exceed moderate consumption cutpoint[c]	13.0 (0.009)	21.7

[a] Overall reduction of 14.9 million oz. of ethanol per day achieved by taking a 25% reduction in average daily intake of all current drinkers.
[b] Overall reduction of 14.9 million oz. of ethanol per day achieved by taking a 28% reduction in average daily intake of all current drinkers whose consumption occasionally or usually exceeds moderate consumption cutpoint of ethanol to total body water ratio of 0.0534 (equivalent to 0.75 g of ethanol per kg of body weight for a 21-year-old male weighing 160 lbs.)
[c] Overall reduction of 14.9 million oz. of ethanol per day achieved by taking a 41% reduction in average daily intake of all current drinkers whose consumption usually exceeds moderate consumption cutpoint of ethanol to total body water ratio of 0.0534 (equivalent to 0.75 g of ethanol per kg of body weight for a 21-year-old male weighing 160 lbs.)

sociated with a certain drinking volume and pattern in the contemporary United States may be quite different from the risk that would be observed in a setting with totally different norms surrounding intoxication and the role of alcohol in everyday life. This would result in different beta parameters that might more strongly favor one or the other of the reduction options. In addition, the feasibility of enacting the different reduction options is culture specific, reflecting both the strength and economic importance of the beverage industry and the relative importance of individual freedom and privacy as opposed to willingness to enforce public health goals through coercive measures. While the conclusions of this paper may not be universally generalizable, the techniques that were used are universally adaptable and can provide the basis for addressing the question of the population versus high-risk strategies in virtually any setting for which there are data linking consumption and the risk of alcohol use disorders.

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U.S. Low-Risk Drinking Guidelines: An Examination of Four Alternatives

Deborah A. Dawson

Background: *This study compared four sets of U.S. low-risk drinking guidelines (two interpretations of the U.S. Dietary Guidelines and two variations of the NIAAA physicians' guidelines) in terms of adherence and how well they predicted five different alcohol-related outcomes.* Methods: *Using data from a nationally representative sample of 17,542 U.S. adults 21 years of age and over who drank 12 or more drinks in the past year, this study assessed the sensitivity, specificity, overall accuracy, positive and negative predictive values, and odds ratios of the various drinking guidelines (specifically, of having exceeded them with different degrees of frequency) as predictors of alcohol dependence, impaired driving, liver disease, peptic ulcer, and hypertension.* Results: *The proportions of past-year regular drinkers exceeding the four sets of guidelines varied from 20.9%, whose average intake exceeded the weekly limits, to between 21.0% and 42.7% who exceeded the daily guidelines at least once a week, and to between 69.2% and 94.2% who ever exceeded the daily limits in the year preceding the interview. Sensitivity and odds ratios were highest for the ever exceeding the Dietary Guidelines daily limits, intermediate for ever exceeding the two variations based on the NIAAA physicians' guidelines, and lowest for exceeding the Dietary Guidelines interpreted as weekly limits. The opposite pattern was observed for specificity and overall predictive accuracy. When frequently exceeding the daily limits was considered, their sensitivity declined but their specificity and positive predictive value increased.* Conclusions: *If sensitivity and specificity are deemed equally important, the NIAAA physicians' guidelines incorporating both daily and weekly limits seem to do the best job of balancing these dimensions in the prediction of a variety of alcohol-related outcomes.*

In the past decade, a number of countries have formulated guidelines for moderate or low-risk drinking that attempt to specify the consumption levels at which any possible benefits (e.g., cardio-protective effects) of light-to-moderate drinking are outweighed by the risks of adverse alcohol-related outcomes, social and economic as well as physical. Although a recent summary indicated a dozen different countries with formal low-risk drinking guidelines (International Center for Alcohol Policies, 1996), English-speaking nations have been particularly active in this area. A recent review of Canadian guidelines (Walsh et al., 1998) cited 18 different examples, all but 2 of which dated from the 1990s. Another set of guidelines has been issued since that review was published (Bondy et al., 1999). The authors have included medical and research organizations in addition to local and national government agencies. Other countries with strong traditions of drinking guidelines include Australia. New Zealand, and the United Kingdom, where the 1995 *Sensible Drinking* recommendations (United Kingdom Department of Health, 1995*)* generated considerable debate among medical researchers, the alcohol industry, and the Ministry of Health (British Medical Association, 1995*;* Hawks, 1996; Marmot, 1995) as a result of the extent to which they liberalized previously issued guidelines.

Both within and among countries, drinking guidelines have varied substantially in a number of important ways that may reflect country and cultural differences as much as differing appraisals of scientific evidence. These include differences in the groups for whom the guidelines are considered inappropriate (e.g., underage drinkers, pregnant women, alcoholics, and so forth) and differences in the characteristics of the drinking limits themselves (Pols and Hawks, 1992). Originally, the majority of guidelines stated the limits for low-risk consumption in terms of average daily levels of intake, often stated in terms of their equivalent weekly limits, reflecting the fact that most of the epidemiologic research upon which they were based employed average daily volume measures of alcohol consumption. However, some of the more recent guidelines have added daily limits as well as or in

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place of weekly limits, reflecting growing awareness of the importance of drinking patterns as predictors of acute adverse consequences of drinking (Hingson et al., 1999; Midanik et al., 1996; Rehm et al., 1996) and of the potential difficulties in interpreting weekly limits (Roche, 1997). The guidelines also have varied in whether they impose different limits on consumption for males and females, with sex differences often varying in magnitude for weekly limits and daily limits. Some guidelines have specified optimal drinking levels, whereas the majority has specified upper limits only. Finally, the weekly and/or daily limits themselves have varied. A recent international summary of guidelines indicated a range of daily limits of actual alcohol intake from 13.5 to 70 g for males and from 10.8 to 50 g for females. When stated in terms of standard drinks, this variation was compounded by the fact that the size of a standard drink varied and contained from 8 to 19.75 g of ethanol per drink (International Center for Alcohol Policies, 1996, 1998).

In 1995 and again in 2000, the United States Department of Agriculture (USDA), in conjunction with the Department of Health and Human Services (DHHS), published *Nutrition and Your Health: Dietary Guidelines for Americans* (1995, 2000), an overall set of nutritional guidelines that included limits for low-risk alcohol consumption that were not to exceed two standard drinks per day for males and not to exceed one standard drink per day for females. A standard drink was defined as 12 ounces of beer, 5 ounces of wine, or 1 1/2 ounces of 80-proof distilled spirits, all yielding roughly 14 g of ethanol. Although these limits were based on research that consisted largely of studies that examined risks associated with the average daily volume of ethanol intake, the wording of the guidelines suggested that these were daily limits not to be exceeded on any drinking day. Had they been interpreted as limits on an average intake per day, they would have corresponded to weekly limits of up to 7 drinks for females and up to 14 drinks for males.

These were in fact the weekly limits (14 drinks for males and 7 for females) that were published by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) in its *Physicians' Guide to Helping Patients With Alcohol Problems* (1995). In recognition of the fact that these weekly limits might permit levels of consumption on any given day that would increase the risks of acute consequences, such as impaired driving (e.g., if a male drank 7 drinks each on Friday and Saturday nights to make up his weekly allotment), the physicians' guidelines also included daily limits on alcohol intake. These daily limits stipulated that consumption was not to exceed 4 drinks on any day for males and not to exceed 3 drinks on any day for females. Thus, to consume the maximum weekly limit of 14 drinks, a male would have to allocate this amount quite evenly over at least 4 separate days to avoid exceeding the daily limit of 4 drinks. Likewise, a female, to consume the maximum weekly limit of 7 drinks, would have to allocate this amount fairly evenly over at least 3 separate days to avoid exceeding the daily limit of 3 drinks.

Despite the attention that has been given to developing and promoting low-risk drinking guidelines as a tool for the primary prevention of alcohol problems, there have been few formal attempts to evaluate or compare any of the existing guidelines in terms of how many people exceed them, the degree of risk associated with exceeding them, or their effectiveness as predictors of alcohol-related harm. As Walsh et al. (1998) noted in their review of Canadian guidelines, most evaluations have been of individual sets of guidelines and based solely on content validity as determined by review of the pertinent literature linking intake and risks. A rare comparative evaluation performed by Room et al. (1995) contrasted two sets of Canadian guidelines and one set of British guidelines. They found that the prevalence of reporting two or more harmful consequences of drinking among drinkers classified as low-risk was minimized by using the most stringent set of guidelines. However, the difference in the absolute prevalence of harm between drinkers classified as low-risk and higher-risk was greatest in the case of the least stringent guidelines.

The purpose of this analysis is to compare four alternative sets of U.S. low-risk drinking guidelines. The first two are based on the Dietary Guidelines, i.e., the recommendations published in *Nutrition and Your Health* (United States Department of Agriculture, 1995, 2000). In the first set of guidelines, the recommendations are considered as daily limits not to be exceeded on any day. In the second set of guidelines, the recommendations are considered as limits on *average* intake per day and were multiplied times seven to yield weekly limits. The remaining two sets of guidelines are based on the NIAAA physicians' guidelines (United States Department of Health and Human Services, 1995). The third set reflects the NIAAA guidelines as currently written, with limits on both weekly and daily intake, and the fourth set drops the weekly limits altogether, to examine the effect of this approach to simplifying the guidelines. The specific limits associated with these four sets of guidelines are as follows:

(1) For males, intake not to exceed 2 standard drinks on any day; for females, intake not to exceed 1 standard drink on any day

(2) For males, intake not to exceed 14 standard drinks per week; for females, intake not to exceed 7 standard drinks per week

(3) For males, intake not to exceed 14 standard drinks per week AND not to exceed 4 standard drinks on any day; for females, intake not to exceed 7 standard drinks per week AND not to exceed 3 standard drinks on any day

(4) For males, intake not to exceed 4 standard drinks on any day; for females, intake not to exceed 3 standard drinks on any day

The comparison of the four sets of guidelines has two areas of focus. First, what proportion of drinkers exceeds the low-risk guidelines, and does this proportion vary among population subgroups? In the case of the daily guidelines, both the proportion exceeding the guidelines at least once in the year preceding the interview and those exceeding the guidelines once a week or more often are considered. Second, how effective are the guidelines as predictors of various adverse chronic and acute consequences of excessive alcohol consumption?

METHODS

Sample

This analysis was based on data from the 1992 National Longitudinal Alcohol Epidemiology Survey (NLAES), which was designed and sponsored by the National Institute on Alcohol Abuse and Alcoholism. This survey gathered information on alcohol consumption and alcohol-related problems from 42,862 U.S. adults 18 years of age and over, one of whom was selected at random from each household in a sample that was representative of the noninstitutionalized population of the coterminous United States. The household and sample-person response rates were 92% and 97%, respectively. Data were collected in personal interviews conducted in respondents' homes. The complex, multistage sample design included the selection of primary sampling units with probability proportional to size and oversampling of individuals who were African American or between the ages of 15 and 29 years (Grant et al., 1994). This analysis was restricted to the 17,542 individuals 21 years of age and older who were identified as past-year drinkers—individuals who in response to screening questions reported having consumed at least 12 alcoholic drinks in the year preceding interview. Although these will be referred to as past-year drinkers for the sake of simplicity, it is important to note that infrequent past-year drinkers were not included in this analysis, as they could not be distinguished from abstainers on the basis of the items contained in the NLAES.

Consumption measures

In assessing whether or not respondents exceeded the weekly limits for low-risk consumption, average weekly intake was determined on the basis of consumption during the year preceding the NLAES interview. In a separate series of questions for beer, wine, and distilled spirits, NLAES respondents were asked the overall frequency of consumption during the past year, usual and heaviest quantities of drinks consumed per drinking day, and the frequency and size of drink associated with the heaviest quantity. Frequencies were converted to days per year using the midpoints of the categorical response categories (e.g., 2 to 3 days a month = 30 days per year). Using ethanol conversion factors of 0.045 for beer, 0.121 for wine, and 0.409 for spirits (DISCUS, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams et al., 1993), the annual volume of ethanol intake was calculated as the sum of the beverage-specific volumes, each of which was calculated as [(total frequency minus frequency of drinking heaviest quantity) x usual quantity x usual size x ethanol conversion factor] + [frequency of drinking heaviest quantity x heaviest quantity x size of heaviest quantity x ethanol conversion factor]. Average weekly ethanol intake was then calculated by dividing the annual volume of intake by 52, and this was converted to average number of drinks per week by dividing the weekly volume by 0.6 ounces (approximately 14 g), the assumed ethanol content of a standard drink. (As was mentioned previously, the U.S. drinking guidelines define a standard drink in beverage-specific terms that result in a standard drink of beer containing 0.54 ounces of ethanol if an ethanol content of 0.045 is assumed. In contrast, wine and spirits are defined so as to contain approximately 0.60 ounces of ethanol. To facilitate computation of intake for all types of beverages combined and because many beers have an ethanol content higher than 0.045, this analysis used 0.6 ounces as the size of a standard drink.)

To assess whether respondents exceeded the daily limits for low-risk consumption, the usual and largest quantities of each beverage were converted to ounces of ethanol based on the reported size of drink and the ethanol conversion factors cited above. Number of ounces was then converted to number of standard

drinks, again assuming an ethanol content of 0.60 ounces. The resulting usual and largest numbers of standard drinks were then compared to the daily limits. To account for days when more than one type of beverage was consumed, a question on frequency of drinking five or more drinks of any type (i.e., including combinations of beverages) was also considered. The frequency of exceeding the daily guidelines was estimated as the largest of: (1) the overall frequency of consuming any beverage for which the usual quantity exceeded the daily limit, (2) the frequency of consuming the largest quantity of any beverage for which this largest quantity exceeded the daily limit, or (3) the frequency of drinking 5 or more drinks of any type. If the respondent never drank five or more drinks and reported usual and largest quantities of all individual beverages that lay within the daily guidelines, they were assumed never to have exceeded the guidelines.

Outcome measures

Past-year alcohol dependence was classified in accordance with the DSM–IV criteria (American Psychiatric Association, 1994) and was measured using the AUDADIS (Grant and Hasin, 1992), a structured interview schedule designed for administration by lay interviewers. To be classified with alcohol dependence, an individual had to meet three or more of the seven DSM–IV criteria for dependence: (1) tolerance; (2) withdrawal (including relief drinking or avoidance of withdrawal); (3) persistent desire or unsuccessful attempts to cut down on or stop drinking (4) much time spent drinking, obtaining alcohol, or recovering from its effects; (5) reduction or cessation of important activities in favor of drinking; (6) impaired control over drinking; and (7) continued use despite physical or psychological problems caused by drinking. Criteria *not* associated with duration qualifiers (i.e., qualifiers that stipulate the repetitiveness with which problems must occur) were considered to be satisfied if an individual reported one or more positive symptoms of the criterion during the past year. Criteria *with* duration qualifiers were considered to be satisfied if a person reported two or more symptoms during the past year or one symptom that occurred at least two times during the past year. To be consistent with the syndromal definition of the withdrawal criterion, two or more positive symptoms were required in addition to satisfaction of the duration qualifier.

Respondents were considered positive for impaired driving if they reported that in the past year they had driven a car, motorcycle, truck, boat, or any other vehicle "after having too much to drink." Liver disease, peptic ulcer, and hypertension were taken from a list of 24 medical conditions. Respondents were asked whether they had ever had any of the conditions, whether it had caused them problems during the last

Table 1. Percentage of Current Drinkers 21 Years of Age and Over With Consumption in Excess of Low-Risk Drinking Guidelines

	Dietary Guidelines interpreted as:			NIAAA Physicians' Guidelines			
	daily limits[a]		weekly limits[b]	weekly and daily limits[c]		daily limits only[d]	
	(1)[e]	(2)[f]		(1)[e]	(2)[f]	(1)[e]	(2)[f]
Total	94.2 (0.2)	42.7 (0.5)	20.9 (0.4)	71.0 (0.4)	28.1 (0.4)	69.2 (0.5)	21.0 (0.4)
Male	91.2 (0.4)	42.5 (0.6)	19.9 (0.5)	71.9 (0.6)	29.5 (0.6)	70.8 (0.6)	23.7 (0.5)
Female	98.7 (0.1)	42.9 (0.7)	22.3 (0.6)	69.6 (0.6)	25.9 (0.6)	66.9 (0.6)	16.9 (0.5)
Ages 18–29	98.3 (0.2)	45.6 (0.8)	22.4 (0.8)	87.4 (0.6)	32.8 (0.8)	87.3 (0.6)	28.8 (0.8)
Ages 30–44	95.7 (0.3)	40.8 (0.7)	18.0 (0.5)	73.7 (0.6)	25.4 (0.6)	73.1 (0.6)	20.1 (0.6)
Ages 45–64	92.2 (0.6)	43.6 (0.9)	23.1 (0.8)	60.5 (0.9)	28.4 (0.8)	57.5 (1.0)	18.2 (0.7)
Ages 65+	81.7 (1.0)	39.9 (1.2)	22.8 (1.0)	41.2 (1.3)	25.3 (1.0)	33.1 (1.2)	10.5 (0.8)
Non-Hispanic white	94.4 (0.2)	42.3 (0.5)	20.7 (0.4)	70.9 (0.5)	27.5 (0.4)	69.1 (0.5)	20.0 (0.4)
Non-Hispanic black	94.5 (0.7)	50.5 (1.6)	25.3 (1.4)	69.2 (1.4)	34.3 (1.5)	67.8 (1.4)	29.4 (1.4)
Other Non-Hispanic	87.9 (1.9)	33.0 (2.8)	15.9 (2.0)	66.4 (2.8)	20.4 (2.2)	65.3 (2.7)	15.3 (2.0)
Hispanic	94.1 (0.8)	41.8 (1.8)	19.9 (1.5)	76.1 (1.6)	31.2 (1.7)	74.8 (1.6)	25.7 (1.7)
Less than HS graduate	91.5 (0.7)	49.6 (1.4)	28.9 (1.4)	72.8 (1.2)	39.3 (1.4)	70.8 (1.2)	33.9 (1.4)
HS graduate	94.6 (0.4)	44.0 (0.8)	22.3 (0.7)	74.0 (0.7)	31.1 (0.8)	72.2 (0.8)	24.4 (0.7)
Some college	95.6 (0.3)	42.3 (0.9)	20.2 (0.6)	73.4 (0.8)	26.3 (0.7)	72.0 (0.8)	19.8 (0.7)
College graduate	93.5 (0.4)	39.1 (0.8)	17.2 (0.6)	65.5 (0.8)	22.5 (0.7)	63.5 (0.8)	13.9 (0.5)

Note: Figures in parentheses are standard errors of percentages.

[a] For males, not to exceed 2 standard drinks on any day; for females, not to exceed 1 standard drink on any day.

[b] For males not to exceed 14 standard drinks per week; for females, not to exceed 7 standard drinks per week.

[c] For males, not to exceed 14 standard drinks per week AND not to exceed 4 standard drinks on any day; for females, not to exceed 7 standard drinks per w
lD not to exceed 3 standard drinks on any day.

[d] For males, not to exceed 4 standard drinks on any day; for females, not to exceed 3 standard drinks on any day.

[e] Exceeded daily limits on at least 1 day in last year.

[f] Exceeded daily limits at least once a week in last year.

12 months, and whether a doctor or other health professional had diagnosed the condition. Conditions were counted as positive only if "yes" was answered to both questions: experienced in the past year and diagnosed by a health professional. A respondent was considered having liver disease if (s)he answered yes to any one of the following conditions: "enlarged liver," "yellow jaundice," "cirrhosis of the liver," "hepatitis," and "other liver disease." Peptic ulcer was referred to as "a stomach ulcer," and hypertension was referred to as "high blood pressure."

Analysis

In addition to presenting descriptive statistics on the proportion of drinkers exceeding the alternative low-risk drinking guidelines, this analysis used two techniques to evaluate and compare the guidelines. First, the sensitivity, specificity, overall proportion of cases predicted accurately, and positive and negative predictive values were assessed with respect to each of the five alcohol-related outcome measures. In accordance with standard practice, sensitivity was defined as the percentage of individuals scored positive for the outcome whose consumption exceeded the low-risk drinking limits, and specificity was defined as the percentage of those scored negative for the outcome whose consumption lay within (i.e., did not exceed) the low-risk limits. The overall percentage of cases correctly classified was defined as the percentage of all drinkers who *either* drank within the limits and did not experience the outcome *or* exceeded the limits and did experience the outcome. Positive predictive value (PPV) was defined as the prevalence of the outcome among individuals whose intake exceeded the limits of the drinking guideline, and negative predictive value (NPV) was defined as the proportion of respondents *without* the outcome (100% minus the prevalence) among those who did *not* exceed limits.

The relative risk of an outcome among persons who exceed the drinking-limits (compared to those who do not) is an intuitively appealing measure of how well the guidelines function, but one that is susceptible to confounding. That is, persons who do and do not exceed the drinking limit may differ in terms of characteristics (age, education, etc.) that independently affect the risk of the outcome. To avoid this possible source of bias, multivariate logistic regression models were used to estimate the excess odds of the outcomes among persons whose consumption exceeded low-risk limits relative to those whose consumption lay within the limits, controlling for the potentially confounding effects of age, sex (male versus female), race/ethnicity (Hispanic, black, and other versus white), and education (high school graduate, some college, and college graduate versus less than a high school graduate). The goodness of fit of each of the models was expressed in terms of its pseudo R^2 value, which approximates the proportion of variance in the outcome 'explained' by the model. The higher this value, the better the drinking limits predict the outcome in question. This R^2 value was calculated by dividing the reduction in the –2 log likelihood that resulted from adding the model covariates by the –2 log likelihood of the model containing the intercept only. Because of the complex sample design of the NLAES, all statistics and standard errors were estimated using SUDAAN (Research Triangle Institute, Research Triangle Park, NC), a software package that uses Taylor series linearization to adjust for the effects of clustering and stratification (Shah et al., 1997).

RESULTS

As shown in Table 1, when the Dietary Guidelines were interpreted as daily limits, i.e., maximum levels not to be exceeded on *any* day, nearly all U.S. drinkers 21 years of age and over (94.2%) were classified as having exceeded those limits on at least one day during the past year. However, less than half (42.7%) exceeded the Dietary Guidelines daily limits once a week or more often. When the Dietary Guidelines were interpreted as limits on *average* daily intake and expressed as weekly limits, only about one-fifth (20.9%) of all drinkers had average volumes of intake that exceeded those weekly limits. When compared with the NIAAA physicians' guidelines, using both the weekly and daily limits, 71.0% of adult drinkers had levels of alcohol intake in excess of the limits, i.e., either their average intake exceeded the weekly limits *or* they exceeded the daily limits at least once during the past year. The proportion whose average intake exceeded the weekly limits or whose intake exceeded the NIAAA daily limits once a week or more often was far lower, 28.1%. Similarly, when the weekly limits were dropped and only the NIAAA daily limits were considered, the proportion of drinkers who ever exceeded the guidelines in the year preceding the interview was 69.2%, compared with 21.0% who exceeded the daily limits weekly or more often.

As these figures show, most drinkers whose consumption was in excess of the low-risk levels had sporadic occasions of heavy drinking that exceeded the daily low-risk drinking limits, compared with an overall volume of intake that exceeded the weekly limits. The majority of those exceeding the daily limits did so less

than once a week. As can be inferred from the small impact of dropping the weekly limits from the NIAAA physicians' guidelines (the fourth set of guidelines compared to the third), there were very few high volume drinkers whose daily consumption levels *never* exceeded the low-risk daily limits. In fact, of those drinkers whose consumption exceeded the NIAAA weekly limits, 92.3% also exceeded the daily limits on at least one occasion, and more than half did so at least once a week (data not shown).

Given that the low-risk drinking limits had lower thresholds for females than males, one might expect a greater proportion of females to have exceeded the low-risk drinking guidelines. As Table 1 indicates, the gender differences in the proportions of drinkers exceeding the guidelines were generally small, and not always indicative of a greater excess among females. The disparity was largest when the Dietary Guidelines were interpreted as daily limits, resulting in 98.7% of female drinkers ever exceeding the limits compared with 91.2% of male drinkers. However, the proportions of males and females who *frequently* (once a week or more often) exceeded the Dietary Guidelines daily limits were virtually identical, 42.5% and 42.9%, respectively. When the Dietary Guidelines were interpreted as weekly limits, females were only slightly more likely than males to exceed the limits (22.3% vs. 19.9%), and when the NIAAA physicians' guidelines were considered, males were slightly more likely than females to have exceeded the low-risk guidelines (especially those based solely on the daily limits).

In terms of other characteristics, the proportion of drinkers exceeding the Dietary Guidelines interpreted as weekly limits was lowest among individuals aged 30–44 years, whereas the proportions exceeding the sets of guidelines that included daily limits (alone or in conjunction with weekly limits) were usually lowest among individuals 65 years of age or older and highest among those below the age of 30. Non-Hispanic black drinkers were the most likely to exceed the guidelines based solely on weekly limits and, along with Hispanics, to *frequently* exceed the daily limits (i.e., once a week or more often). Hispanic drinkers were the most likely to *ever* exceed the daily limits of the NIAAA guidelines. Non-Hispanic individuals of "other" races, most of whom were of Asian descent, were the least likely to exceed all four sets of drinking guidelines. Finally, the proportion of drinkers that exceeded the guidelines based on weekly limits alone decreased with increasing education, as did the proportion who *frequently* exceeded the daily drinking limits. There was little variation by education in the proportion of drinkers who *ever* exceeded the daily limits of the Dietary Guidelines, but drinkers with college educations were the least likely to have reported ever exceeding the less stringent NIAAA daily limits.

The power of the drinking guidelines to predict various types of adverse alcohol-related outcomes varied substantially according to which set of guidelines was considered and how often the daily limits were exceeded (Table 2). There was also considerable variation across outcome. Ever exceeding the Dietary Guidelines daily limits was the measure of risk drinking with the highest sensitivity, in excess of 95% for four of the five outcomes. For alcohol dependence and impaired driving, ever exceeding the daily limits of the NIAAA physicians' guidelines was almost as sensitive, >95% regardless of whether or not the weekly limits were considered. For the other outcomes, ever exceeding the NIAAA daily limits yielded sensitivity values in the range of approximately 60% to 80%. The sensitivity of all of the daily drinking limits was reduced if the risk measure was interpreted as *frequently* exceeding the limits rather than *ever* exceeding them. The sensitivity for exceeding the Dietary Guidelines daily drinking limits once a week or more often varied from 45.8% to 79.2%, depending on outcome The sensitivity for frequently exceeding the NIAAA daily drinking limits varied from 30.8% to 70.8% when in combination with the weekly limits and from 21.9% to 62.1% when the weekly limits were disregarded. These were in approximately the same range as the sensitivity for exceeding the Dietary Guidelines weekly limits, 24.8% to 58.4%.

The excessive drinking measures with the highest specificity were those with the lowest sensitivity, i.e., exceeding the Dietary Guidelines weekly limits and exceeding the NIAAA guidelines daily limits on a weekly or more frequent basis. These demonstrated specificity values of approximately 80%. Specificity was somewhat lower, 72.0% to 76.1%, when the NIAAA weekly limits were considered in addition to the daily limits. Frequently exceeding the Dietary Guidelines daily limits resulted in a lower level of specificity, approximately 60%. *Ever* exceeding the daily drinking limits yielded sensitivity levels of approximately 30% for the NIAAA daily limits and less than 10% for the Dietary Guidelines daily limits.

The results in terms of overall percentage of cases accurately predicted (in terms of presence or absence of the outcome variables) closely mirrored those for specificity. This reflects the fact that this percentage is a weight average of sensitivity and specificity, where the respective weights are the proportions of the population positive and negative for the outcomes in ques-

tion. As all of the outcomes considered in this analysis were fairly rare (ranging in prevalence from less than 1% for liver disease to 11% for impaired driving), the overall percentage of cases accurately predicted was far more heavily influenced by specificity than by sensitivity.

As is generally the case when infrequent outcomes are considered, PPVs were low for all of the drinking guidelines considered in this analysis. The only measures for which the PPV was greater than 20% were exceeding the Dietary Guidelines weekly limits or frequently exceeding the NIAAA daily limits, and then only for the outcomes of alcohol dependence and impaired driving. For the other outcomes, the PPVs were consistently less than 10% and usually less than 5%, regardless of which risk-drinking measure was considered. Conversely, the negative predictive values were consistently higher than 90% for all drinking measures and outcomes.

For the same set of five outcomes, Table 3 examines that odds of having experienced the outcome among individuals whose intake exceeded the low-risk drinking guidelines relative to those whose intake lay within the limits of the guidelines, after adjusting for age, sex, race/ethnicity and education in multiple logistic regression models. As might be expected on the basis of Table 2, there was substantial variation in the magnitude of the odds ratios both by measure of risk drinking and by outcome. For alcohol dependence and impaired driving, the odds ratios were ex-

Table 2. Sensitivity, Specificity, Positive and Negative Predictive Values, and Overall Percentage of Cases Predicted Accurately When Consumption in Excess of Low-Risk Drinking Guidelines Is Used to Predict Selected Past-Year Outcomes

	Dietary Guidelines interpreted as:			NIAAA Physicians' Guidelines			
	daily limits[a]		weekly limits[b]	weekly and daily limits[c]		daily limits only[d]	
	(1)[e]	(2)[f]		(1)[e]	(2)[f]	(1)[e]	(2)[f]
				Sensitivity			
Alcohol dependence	99.8 (0.2)	79.2 (1.3)	58.4 (1.5)	98.5 (0.3)	70.8 (1.4)	98.4 (0.3)	62.1 (1.5)
Impaired driving	99.9 (0.1)	71.3 (1.1)	44.4 (1.3)	97.9 (0.3)	58.6 (1.2)	97.7 (0.3)	51.4 (1.3)
Liver disease	95.4 (2.4)	52.7 (6.2)	40.1 (6.1)	81.2 (4.6)	45.5 (6.1)	81.2 (4.6)	37.9 (5.9)
Peptic ulcer	97.3 (0.7)	49.4 (2.5)	37.5 (2.4)	74.5 (2.4)	34.7 (2.6)	73.3 (2.4)	30.0 (2.6)
Hypertension	92.2 (1.0)	45.8 (2.0)	24.8 (1.6)	64.3 (1.9)	30.8 (1.8)	60.8 (2.0)	21.9 (1.7)
				Specificity			
Alcohol dependence	6.3 (0.2)	60.9 (0.5)	82.7 (0.3)	31.7 (0.5)	76.1 (0.4)	33.6 (0.5)	83.0 (0.4)
Impaired driving	6.5 (0.3)	61.0 (0.3)	82.1 (0.4)	32.5 (0.5)	75.9 (0.4)	34.5 (0.5)	82.9 (0.4)
Liver disease	5.8 (0.2)	57.4 (0.5)	79.2 (0.4)	29.1 (0.4)	72.0 (0.4)	30.8 (0.5)	79.1 (0.4)
Peptic ulcer	5.9 (0.5)	57.5 (0.5)	79.3 (0.4)	29.1 (0.5)	72.1 (0.4)	30.9 (0.5)	79.3 (0.4)
Hypertension	5.7 (0.2)	57.5 (0.5)	79.3 (0.4)	28.7 (0.5)	72.1 (0.4)	30.4 (0.5)	79.1 (0.4)
				Overall percentage correctly predicted			
Alcohol dependence	15.7 (0.4)	62.5 (0.5)	80.7 (0.3)	38.4 (0.5)	75.6 (0.3)	40.1 (0.5)	81.2 (0.3)
Impaired driving	18.2 (0.4)	62.2 (0.5)	78.1 (0.3)	40.7 (0.4)	73.9 (0.3)	42.4 (0.4)	79.3 (0.3)
Liver disease	7.4 (0.3)	57.4 (0.5)	79.4 (0.4)	30.2 (0.5)	71.9 (0.4)	32.0 (0.5)	78.9 (0.3)
Peptic ulcer	9.4 (0.3)	57.3 (0.5)	78.3 (0.4)	31.2 (0.5)	71.1 (0.4)	32.9 (0.5)	78.0 (0.3)
Hypertension	10.8 (0.3)	57.0 (0.5)	77.0 (0.4)	31.1 (0.5)	70.1 (0.4)	32.6 (0.5)	76.4 (0.4)
				Positive predictive value			
Alcohol dependence	9.4 (0.3)	16.5 (0.6)	24.5 (0.9)	12.4 (0.4)	22.4 (0.7)	12.6 (0.4)	26.3 (0.9)
Impaired driving	12.1 (0.3)	19.1 (0.6)	24.1 (0.9)	15.7 (0.4)	23.8 (0.8)	16.1 (0.4)	28.0 (0.9)
Liver disease	0.5 (0.1)	0.6 (0.1)	0.9 (0.2)	0.5 (0.1)	0.7 (0.1)	0.5 (0.1)	0.8 (0.1)
Peptic ulcer	2.7 (0.1)	3.0 (0.2)	3.4 (0.3)	2.8 (0.2)	3.3 (0.3)	2.8 (0.2)	3.8 (0.4)
Hypertension	4.6 (0.2)	5.0 (0.3)	5.6 (0.4)	4.3 (0.2)	5.1 (0.4)	4.1 (0.3)	4.9 (0.4)
				Negative predictive value			
Alcohol dependence	99.7 (0.2)	96.7 (0.2)	95.4 (0.2)	99.5 (0.1)	96.4 (0.2)	99.5 (0.1)	95.7 (0.2)
Impaired driving	99.8 (0.2)	94.3 (0.3)	92.0 (0.3)	99.2 (0.1)	93.4 (0.3)	99.2 (0.1)	93.0 (0.3)
Liver disease	99.6 (0.2)	99.6 (0.1)	99.7 (0.1)	99.7 (0.1)	99.7 (0.1)	99.7 (0.1)	99.6 (0.1)
Peptic ulcer	98.8 (0.3)	97.7 (0.2)	97.6 (0.2)	97.7 (0.3)	97.6 (0.2)	97.7 (0.2)	97.7 (0.1)
Hypertension	93.7 (0.8)	95.6 (0.2)	95.5 (0.2)	94.2 (0.4)	95.5 (0.2)	94.0 (0.4)	95.4 (0.2)

Note: Figures in parentheses are standard errors of percentages.

[a] For males, not to exceed 2 standard drinks on any day; for females, not to exceed 1 standard drink on any day.

[b] For males, not to exceed 14 standard drinks per week; for females, not to exceed 7 standard drinks per week.

[c] For males, not to exceed 14 standard drinks per week AND not to exceed 4 standard drinks on any day; for females, not to exceed 7 standard drinks per week AND not to exceed 3 standard drinks on any day.

[d] For males, not to exceed 4 standard drinks on any day; for females, not to exceed 3 standard drinks on any day.

[e] Exceeded daily limits on at least 1 day in last year.

[f] Exceeded daily limits at least once a week in last year.

tremely high (15.3 to 37.5) for ever exceeding the daily drinking limits of either the Dietary Guidelines or the NIAAA physicians' guidelines. For these same two outcomes, the odds ratios were more moderate (4.0 to 7.2) for *frequently* exceeding any of the daily drinking limits or for exceeding the Dietary Guidelines interpreted as weekly limits. For the other outcomes, the odds ratios were considerably lower (1.2 to 2.4) and often of marginal statistical significance. The odds ratios for these chronic conditions showed little if any variation according to which measure of risk drinking was considered.

As is evident from Table 3, the models resulting in the highest odds ratios were not the best fitting models. For the outcomes of alcohol dependence and impaired driving, the models that explained the highest proportion of variance were those based on the NIAAA weekly and daily limits, when the daily limits were exceeded once a week or more often. For liver disease, the Dietary Guidelines interpreted as weekly limits resulted in the best-fitting model. For peptic ulcer and hypertension, all of the sets of drinking guidelines demonstrated comparable goodness of fit. The extremely low proportions of variance explained by these models, especially those predicting liver disease, peptic ulcer, and hypertension, reflect the omission of important risk factors specific to the various outcomes, e.g., measures of diet, exercise, smoking, etc. The purpose of these models was not to fully investigate all potential risk factors for these outcomes but simply to illustrate the variation in explanatory power associated with the different drinking guidelines.

DISCUSSION

This evaluation of four different sets of low-risk drinking guidelines found striking differences in level of adherence and in relationship to adverse outcomes based on whether daily and/or weekly limits were considered and according to the stringency of those limits and the frequency with which they were exceeded. Interpreting the Dietary Guidelines as daily drinking limits—which is the interpretation suggested by the manner in which they are now written—resulted in almost all drinkers being classified as ever having exceeded the low-risk threshold. Accordingly, this set of guidelines was able to identify (i.e., categorize as being at risk) almost all of the individuals experiencing the five outcomes that were studied, resulting in extremely high levels of sensitivity. However, more than 90% of the drinkers who did *not* experience the outcomes also were classified as being at risk by virtue of their consumption levels, indicating that these guidelines lack both specificity and face validity as predictors of alcohol problems (or the lack thereof, to be more precise). This is not surprising, as it would be challenging to find any studies citing evidence of significant harm associated with the *occasional* consumption of, for example, three drinks for males or two drinks for females. Even when exceeding the Dietary Guidelines daily limits on a *frequent* basis (weekly or more often) was considered as a measure of risk drinking, this did not yield levels of specificity and PPV that were as high as when the less stringent daily limits of the NIAAA physicians' guidelines were considered.

Table 3. Odds Ratios[a] for Selected Past-Year Outcomes Among Current Drinkers 21 Years of Age and Over Whose Consumption Exceeded Low-Risk Drinking Guidelines, Relative to Those Whose Consumption Lay Within the Guidelines

	Dietary Guidelines interpreted as:			NIAAA Physicians' Guidelines			
	daily limits[b]		weekly limits[c]	weekly and daily limits[d]		daily limits only[e]	
	(1)[f]	(2)[g]		(1)[f]	(2)[g]	(1)[f]	(2)[g]
Alcohol dependence	23.4 (8.1–67.8) R^2 = 8.4	5.8 (4.9–6.8) R^2 = 15.6	7.2 (6.2–8.3) R^2 = 18.2	21.3 (13.7–33.3) R^2 = 13.0	7.2 (6.2–8.3) R^2 = 18.6	21.1 (13.8–32.5) R^2 = 13.2	6.5 (5.7–7.5) R^2 = 17.3
Impaired driving	37.5 (12.5–112.7) R^2 = 10.2	4.0 (3.5–4.5) R^2 = 14.7	4.3 (3.7–4.8) R^2 = 14.6	15.4 (11.4–20.9) R^2 = 14.5	4.4 (3.9–4.9) R^2 = 15.6	15.3 (11.4–20.6) R^2 = 14.7	4.4 (3.9–5.0) R^2 = 15.2
Liver disease	1.8 (0.6–5.5) R^2 = 2.3	1.4 (0.8–2.3) R^2 = 2.4	2.4 (1.4–4.1) R^2 = 3.5	2.0 (1.1–3.7) R^2 = 2.8	2.0 (1.2–3.4) R^2 = 3.0	2.3 (1.2–4.2) R^2 = 3.0	2.2 (1.3–3.7) R^2 = 3.1
Peptic ulcer	2.1 (1.2–3.8) R^2 = 1.9	1.3 (1.0–1.5) R^2 = 1.9	1.4 (1.1–1.7) R^2 = 1.9	1.2 (0.9–1.5) R^2 = 1.8	1.3 (1.0–1.6) R^2 = 1.9	1.2 (0.9–1.6) R^2 = 1.8	1.5 (1.2–1.9) R^2 = 2.1
Hypertension	1.4 (1.0–1.9) R^2 = 7.4	1.2 (1.0–1.4) R^2 = 7.4	1.2 (1.0–1.4) R^2 = 7.4	1.2 (1.0–1.4) R^2 = 7.4	1.2 (1.0–1.4) R^2 = 7.4	1.2 (1.0–1.5) R^2 = 7.4	1.3 (1.0–1.6) R^2 = 7.4

Note: Figures in parentheses are 95% confidence intervals of odds ratios.

[a] Adjusted for age, sex, race/ethnicity, and education.

[b] For males, not to exceed 2 standard drinks on any day; for females, not to exceed 1 standard drink on any day.

[c] For male, not to exceed 14 standard drinks per week; for females, not to exceed 7 standard drinks per week.

[d] For males, not to exceed 14 standard drinks per week AND not to exceed 4 standard drinks on any day; for females, not to exceed 7 standard drinks per week AND not to exceed 3 standard drinks on any day.

[e] For males, not to exceed 4 standard drinks on any day; for females, not to exceed 3 standard drinks on any day.

[f] Exceeded daily limits on at least one day in last year.

[g] Exceeded daily limits at least once a week in last year.

When the Dietary Guidelines were interpreted as limits on *average* daily intake and expressed as weekly limits, only about one-fifth of drinkers were classified as being at risk, an estimate that probably corresponds far more closely to public perceptions of the prevalence of problematic drinking and that still exceeds the prevalence of any of the alcohol-related problems examined in this analysis. These limits were among the best in terms of specificity (the ability to predict the *absence* of alcohol problems), overall proportion of cases accurately predicted, and PPV. However, they identified a less than desirable proportion of the individuals who actually did experience the alcohol-related outcomes under study in this analysis, e.g., less than 60% of alcoholics, only 40% of individuals with liver disease, and just 25% of those with hypertension. Moreover, the exclusion of any daily limits from these guidelines runs counter to the literature indicating that much of the alcohol-related harm experienced by low to moderate volume drinkers results from occasional episodes of heavy drinking (Stockwell et al., 1996). Thus, reinterpreting the Dietary Guidelines as weekly limits is not an optimal approach for providing safe and realistic limits for low-risk drinking.

The NIAAA physicians' guidelines including both weekly *and* daily drinking limits resulted in approximately 70% of drinkers being considered at risk, when the daily component of risk was defined as *ever* exceeding the daily limit This proportion fell to 28.1% when the daily component was defined as *frequently* exceeding the limits. Interpreted in this latter way, these guidelines demonstrated an improvement in sensitivity over the Dietary Guidelines weekly limits for three of the five outcomes, with no loss in PPV. Their specificity and overall accuracy was about five percentage points lower than for the Dietary Guideline weekly limits, but still in excess of 70% for all outcomes. When the weekly limits were removed from the NIAAA guidelines, the performance of frequently exceeding the daily limits was indistinguishable from the Dietary Guidelines weekly limits. The similarities with the values for the Dietary Guidelines weekly limits should come as no surprise, given the evidence presented earlier that most of the high-volume drinkers who exceeded the Dietary Guideline weekly limits did so by means of occasionally exceeding the NIAAA daily limits. That is, there was substantial overlap among the individuals who exceeded the Dietary Guidelines weekly limits and the NIAAA daily limits.

In deciding which guidelines yield the "best" combination of predictive values, one is forced to confront two difficult issues. The first is the question of weighing sensitivity versus specificity and PPV. Given that no set of guidelines can predict risk perfectly, is it worse to underestimate or overestimate the harm associated with exceeding proposed low-risk drinking limits? Is the virtue of setting drinking limits at a level where the majority of individuals can enjoy drinking without experiencing adverse consequences outweighed by the liability of providing a false sense of safety for the minority of individuals who *do* experience alcohol-related problems at those drinking levels?

There is a longstanding tradition that emphasizes a conservative approach to both the prevention and treatment of health problems. One implication of this tradition is that the consequences of setting the threshold for low-risk drinking so high that a substantial proportion of adverse outcomes occur at consumption levels below that threshold (low sensitivity) are more serious than the consequences of setting the threshold so low that the majority of people exceeding it do not experience any adverse consequences (low PPV). Accordingly, sensitivity is generally given at least as much emphasis as specificity and PPV, even though specificity contributes far more strongly to the overall accuracy of prediction. At the same time, there is widespread recognition that some minimal level of specificity and/or PPV must be maintained for a test, screener, or guideline to have enough face validity to ensure its acceptability and use. There are no agreed-upon standards for weighing these competing demands, but it is arguable that in this study, the NIAAA physician's guidelines incorporating both weekly and daily limits did the best job of *balancing* these concerns, that is, of simultaneously maximizing all of these dimensions.

The second difficult issue in evaluating the performance of the different guidelines has to do with whether and how weekly limits, which reflect an individual's overall level of consumption and aggregate exposure to risk, can be compared with daily limits, which reflect in-the-event risks that must be cumulated across an individual's drinking occasions to reflect an equivalent level of aggregate exposure to risk. The results of this study clearly indicated that daily limits did not match weekly limits in terms of their predictive ability until they were exceeded with some degree of frequency, e.g., once a week or more often. Is it fair to compare *ever* exceeding weekly drinking limits with *frequently* exceeding daily limits? Yes, such a comparison is perfectly appropriate, because both of these are summary descriptive measures that reflect an individual's overall drinking pattern and not just his behavior on a single drinking occasion. The more challenging question is whether it is appropriate and informative to

promote drinking guidelines in the form of daily limits, if the risks are minimal for infrequently exceeding these limits. I would argue that it is appropriate to do so. In fact, it is no different than advising the public to refrain from smoking or engaging in unsafe sex. However, the wording of the guidelines requires careful consideration to convey a message that is both scientifically accurate and easily understood, i.e., one that unambiguously promotes moderation without exaggerating risk.

As to *how* the weekly and daily limits can be compared, one way is to look at various frequencies for exceeding the daily limits and to evaluate the predictive measures of sensitivity, specificity, and so forth at each of these levels. Then one can select a frequency at which exceeding the daily limits yields a specificity equal to that yielded by the weekly limits and investigate whether, at that same frequency, the daily limits do a better or worse job than the weekly limits in terms of sensitivity. As an example from this analysis, the NIAAA daily limits, when exceeded at least once a week, predicted impaired driving with about the same specificity as the Dietary Guidelines weekly limits, 82.9% and 82.1%, respectively. However, the NIAAA daily limits (again when exceeded once a week or more often) demonstrated more sensitivity in predicting this outcome, 51.4% vs. 44.4%. Of course, such an approach is most useful when the individuals who exceed the daily limits do not comprise the same individuals who exceed the weekly limits, i.e., when there are sizeable numbers of drinkers who exceed only one or the other of the guidelines. In cases where the groups are largely overlapping, then measures that yield similar sensitivities will also yield similar specificities.

This study demonstrated that although odds ratios provide a convenient summary measure of the difference in risk between individuals who exceed and do not exceed the low-risk drinking guidelines, the highest odds ratios do not necessarily identify the guidelines that are the best predictors of alcohol-related harm. Rather, the magnitudes of the odds ratios are strongly influenced by the shape of the risk curve linking the consumption measure and outcome. If the association is represented by a linear, a logarithmic, or an exponential risk curve, the odds ratio will tend to increase the lower the threshold for low-risk drinking is set. However, the cost of increasing the odds ratio is a large proportion of false positives. In the case of a U-shaped curve, the odds ratio will not be far from unity unless both a lower and an upper limit for low-risk intake are specified. Thus, the one unqualified conclusion that can be drawn from odds ratios is that an odds ratio of significantly less than 1.0 is indicative of a poor guideline. Odds ratios should never be used as the sole basis for evaluating drinking guidelines.

The results of this study suggested that a combination of weekly and daily drinking limits did the best job of maximizing sensitivity to outcomes without reducing specificity to an unacceptable level. However, this does not address the question of whether the inclusion of both weekly and daily limits makes the guidelines so complex and difficult to remember as to preclude their having any positive influence on drinking behavior. Although Sellman and Ariell (1996) reported that not a single telephone respondent from a sample of 249 was able to correctly quote the daily and weekly limits of the New Zealand drinking guidelines, confusion may be equally likely to arise from different limits for males and females and/or from contradictory guidelines from different sources. Moreover, comprehension, recollection and even belief are no guarantees of adherence to guidelines. Kaskutas (2000), in a study of pregnant minority women, found that nonrisk drinkers were more likely than risk drinkers to report having been influenced by warning labels and public service announcements regarding the risks of drinking during pregnancy; however, they were *not* more likely to have believed these messages. Thus, belief did not *necessarily* lead to any change in drinking behavior in this population. Additional research is needed to identify factors that might prevent beliefs about drinking risks from influencing alcohol consumption, but the impaired control associated with alcohol dependence is certainly one possibility. This argues in favor of retaining the weekly drinking limits, given their contribution toward increased sensitivity in predicting alcohol dependence relative to daily limits alone. Finally, it should be noted that the inclusion of both weekly and daily drinking limits in low-risk drinking guidelines does not preclude targeted prevention messages that focus on one or the other of these components. That is, guidelines may legitimately contain both weekly limits that target the individual drinker (which might be publicized in settings such as doctors' offices) and daily limits that target the drinking occasion (which might be publicized in settings such as bars).

In interpreting the results of this paper, it is important to note that the NLAES definition of a current drinker as one who drank at least 12 drinks in the preceding year undoubtedly inflated the percentage of drinkers exceeding the low-risk guidelines relative to the estimates that would have been yielded by including infrequent drinkers. This affected all four sets of guidelines and thus probably did not have a large ef-

fect on the differences that were noted among them. The cross-sectional design of the NLAES also limited interpretation of the association between alcohol consumption and the chronic conditions that were considered among the outcomes used in this analysis. This may be one reason why the associations between the risk drinking measures and these outcomes were generally weak. In future analyses, it would be useful to replicate this analysis using a sample of *all* past-year drinkers and/or using a longitudinal study design. Other important areas for future research include extending this analysis to consider other thresholds for weekly and daily low-risk drinking and to determine whether the differences in the limits for males and females are justified. In addition, some of the approaches that have been used to evaluate the effect of warning labels on drinking behavior (Greenfield, 1997; Greenfield and Kaskutas, 1998, 2000; Kaskutas et al., 1998) could be extended to study whether knowledge of the drinking guidelines and/or belief that they represent reasonable assessments of drinking-related risks are associated with drinking behavior.

ACKNOWLEDGMENTS

I deeply appreciate the comments of the anonymous reviewers, whose contributions greatly strengthened this analysis.

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CHAPTER 11
MEASUREMENT AND METHODS

Volume of Ethanol Consumption: Effects of Different Approaches to Measurement

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Objective: *Eight different approaches to measuring alcohol consumption were compared in terms of average daily volume of ethanol intake, selected percentiles of the volume distribution, the proportion of drinkers exceeding a volume-based cutpoint for moderate drinking and the estimated association between volume of intake and alcohol use disorders.* Method: *Data were drawn from the 1988 National Health Interview Survey and the 1992 National Longitudinal Alcohol Epidemiologic Survey. The eight approaches compared overall and beverage-specific questions, reference periods of varying lengths, and measures based solely on usual intake with those that incorporated different aspects of atypical heavy drinking.* Results: *Average daily ethanol intake ranged from 0.43 oz based on two questions on current usual frequency and quantity of drinking (assuming 0.5 oz of ethanol per drink) to 0.72 oz based on 21 questions that included usual and heaviest consumption of beer, wine and distilled spirits in the year preceding interview.* Conclusions: *Estimated volume was highly sensitive to the number and types of questions upon which it was based, and changes in formulation that resulted in relatively small increases in mean volume often were associated with much larger increases in the proportion of drinkers exceeding some specified level of intake and in the estimated association between consumption and alcohol use disorders. These issues should be considered when deciding on the consumption items to be included in alcohol surveys.*

Although volume of ethanol intake has been faulted (Knupfer, 1984; Rehm et al., 1996; Room, 1977) for its inability to distinguish meaningful variations in drinking patterns (e.g., in the frequency of heavy episodic drinking), it remains an important measure in alcohol research. Volume alone is highly correlated with morbidity and mortality (English et al., 1995) and when combined with other indicators of consumption pattern is a useful predictor of various social and physical consequences of drinking (Dawson et al., 1995c; Harford et al., 1991). As fully summarized in Room (1990), volume of ethanol intake has been derived in diverse ways in the national alcohol studies that have been conducted in the United States during the past three decades, with few systematic studies comparing the effects of these different approaches to measurement. This study evaluates eight different ways to measure average daily ethanol intake and compares these approaches in terms of characteristics of the resulting consumption distributions, the estimated proportion of drinkers whose intake exceeds a volume-based cutpoint for moderate drinking and the relationship between volume of intake and alcohol use disorders.

METHOD

The eight volume measures considered in this analysis were derived from consumption data collected in the 1988 National Health Interview Survey (NHIS) and the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES) and are based on U.S. adults 18 years of age and older who in screening questions reported drinking at least 12 alcoholic drinks in the year preceding interview (n = 22,108 for the NHIS and n = 18,352 for the NLAES). In both surveys, one respondent was selected at random from a representative sample of U.S. households. Each sample is representative of the civilian, non-institutionalized adult population of the United States. All measures are based on self-reported level of intake as described in personal interviews conducted in respondents' homes.

The 1988 NHIS collected the following beverage-specific data for the 2-week period preceding interview (or the 2 weeks preceding the most recent drink, if alcohol was not consumed in the 2 weeks prior to interview): frequency of drinking, usual quantity of drinks per drinking day and usual size of drink. These data were supplemented by a general question on usual quantity and frequency of drinking (no reference period specified) and by questions on frequencies of con-

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suming five or more (5+) drinks and of consuming nine or more (9+) drinks in the year preceding interview. The 1992 NLAES collected the following beverage-specific information for the year preceding interview: whether or not the beverage was consumed, frequency of drinking, usual quantity of drinks per drinking day, usual size of drink, largest quantity of drinks per drinking day, size of drink corresponding to largest quantity, and frequency of consuming largest quantity. It also collected information on frequency of drinking 5+ drinks during the preceding year. Both the NHIS and the NLAES collected frequency of drinking within categories (e.g., 2 to 3 days a month) whose midpoints were used to estimate number of drinking days per year, and the resulting annual volume estimates were divided by 365 to yield average daily volume of ethanol intake. For estimates that incorporated respondents' reports of beverage-specific drink size, the following ethanol conversion factors were used to convert ounces of beverage to ounces of ethanol: .045 for beer, .121 for wine and .409 for spirits (DISCUS, 1985; Kling, 1989; Modern Brewery Age, 1992; Turner, 1990; Williams et al., 1995). The exact formulas used in calculating the eight volume measures are shown in the Appendix.

Measures 1 and 2, based on the 1988 NHIS, were based on the usual frequency of drinking any type of alcoholic beverage (no specific time period indicated) and usual number of drinks per drinking day of all types of beverage combined. Measure 1 assumed the "standard" 0.5 oz of ethanol per drink. Measure 2 assumed 0.55 oz of ethanol per drink based on information derived from the other NHIS questions that indicated the distribution of drinks by type of beverage and the median reported size of drink for different types of beverage (12 oz for beer, 4 oz for wine and 1.5 oz for spirits).

Measures 3–6, also based on the 1988 NHIS, were based on the 2-week period preceding interview or the most recent drink. Measure 3 was derived from beverage-specific frequencies and numbers of drinks per drinking day, using the median drink size (see above) for each type of beverage. Measures 4 through 6 used reported instead of median drink size. In Measure 5, volume derived from days of drinking 5+ drinks was incorporated into the overall estimated volume by replacing the average daily intake from Measure 4 with an average intake of six drinks or 3.0 oz of ethanol for the number of days when 5+ drinks were consumed and then recomputing the average daily volume. Measure 6 used information on the reported frequencies of drinking both 5+ and 9+ drinks in a similar manner, assuming average ethanol intakes of 3.0 oz (six drinks) on days of drinking 5+ drinks and an additional 2.0 oz (representing the increase from six to 10 drinks) on days of drinking 9+ drinks. The estimates of six and 10 drinks as midpoints for the open-ended categories of 5+ and 9+ drinks were derived from beverage-specific frequency distributions of quantity of drinks per drinking day. These represented the modal and median values within these ranges for both wine and spirits. (For beer, the modal values were six and 12, corresponding to six packs.)

Measures 7 and 8 were based on the 1992 NLAES, which asked about consumption during the year preceding the interview. Measure 7 is analogous to Measure 5 (based on beverage-specific frequency, quantity and reported drink size, and using volume from days when respondents drank 5+ drinks). Measure 8 was based on both the usual and largest quantities consumed of each type of beverage, as well as their associated frequencies and drink sizes.

Drinkers whose intake exceeded the cutpoint for moderate drinking were defined as men with an average daily ethanol intake of greater than 1.0 oz and women with an average daily ethanol intake of greater than 0.5 oz. These cutpoints have been cited in the U.S. dietary guidelines (Department of Agriculture/Department of Health and Human Services, 1995); however, the guidelines do not really clarify whether these levels refer to average daily intake or maximum intake per occasion. (Obviously, if the guidelines defined as immoderate drinkers men who on *any* drinking occasion consumed more than 1.0 oz of ethanol or women who on *any* occasion consumed more than 0.5 oz of ethanol, then the proportion of drinkers exceeding the moderate-drinking guidelines would be far greater than the proportion based on average daily intake; in fact, it would make more sense to refer to the frequency of immoderate drinking rather than the simple proportion of drinkers exceeding the guideline.) This article employed a volume-based cutpoint for moderate drinking to illustrate how the proportion of drinkers with levels of intake above or below a fixed point in the volume distribution could be affected by different consumption measures. Use of this cutpoint is not intended as an endorsement of a volume-based measure as the sole indicator upon which moderate-drinking guidelines should be based.

Alcohol abuse and dependence, combined into a single measure for this analysis, were defined in accordance with the DSM-IV criteria for alcohol use disorders (American Psychiatric Association, 1994) and were derived from lists of symptom item indicators that asked whether and how often current drinkers ex-

perienced various alcohol-related problems in the year preceding interview. The list of problems included in the NLAES was specifically designed to operationalize the DSM-IV criteria in their proposed form (these were known in advance to the principal investigator of the NLAES, who was a participant in their formulation). Although the symptom item questions contained in the NHIS were designed to produce the DSM-III-R diagnoses, they contained at least one indicator of each of the DSM-IV criteria for abuse and dependence. The diagnostic measures derived from the NLAES were based on 31 symptoms of abuse and dependence; those derived from the NHIS were based on 23 symptoms. An individual was classified as an abuser if he or she met at least one of the four abuse criteria (continued use despite social or interpersonal consequences, hazardous use, alcohol-related legal problems, and neglect of role responsibilities in favor of drinking) and as dependence if he or she met at least three of the seven DSM-IV criteria for dependence (tolerance, withdrawal, desire or attempts to cut down on or stop drinking, much time spent on alcohol-related activities, reduction or cessation of important activities in favor of drinking, impaired control, and continued use despite physical or psychological problems). Criteria not associated with duration qualifiers were satisfied if respondents reported one or more positive symptoms. Criteria associated with duration qualifiers were satisfied if respondents reported that one or more symptoms occurred at least two times or that two or more symptoms occurred at least once. To satisfy the DSM-IV definition of withdrawal as a syndrome, two or more symptoms were required in addition to satisfaction of the duration qualifiers.

In this analysis, the odds of past-year alcohol abuse or dependence were predicted by means of multiple logistic regression models that adjusted for age, sex, race, marital status, education and family history of alcoholism. To better satisfy the assumption of a linear relationship between the predictor variables and the log odds of the outcome, a natural log transform was applied to average daily ethanol intake. This compensated for the fact that an increase at the lower end of the consumption distribution (e.g., from 0.5 to 1.0 oz per day) was associated with a greater increase in the odds of abuse or dependence than an equivalent increase at the upper end of the consumption distribution (e.g., from 2.5 to 3.0 oz per day). To aid in interpretation of the beta parameters for each consumption measure, an odds ratio was calculated to represent the odds of abuse or dependence among individuals who consumed three drinks per day (an average daily ethanol intake of 1.5 oz) relative to the odds among those who consumed three drinks per week (an average daily intake of 0.21 oz). These odds ratios were estimated by exponentiating the product of

Table 1. Selected characteristics and correlates of the distribution of average daily ethanol intake, according to type of formulation used

	Mean volume	Selected percentiles of consumption distribution					% exceeding cutpoint	Models predicting odds of alcohol abuse or dependence		
		.01	.10	.50	.90	.99		% predicted concordantly	Beta	Odds ratio[a]
Measures based on the 1988 National Health Interview Survey										
1. $Q_{TOT} \times F_{TOT} \times E_{TOT}$ (E = .50 for all drinks)	0.43	0.01	0.02	0.21	1.00	3.00	12.2	82.0	0.719	4.1
2. $Q_{TOT} \times F_{TOT} \times E_{TOT}$ (E = .55 for all drinks)	0.47	0.01	0.03	0.23	1.10	3.30	18.1	82.0	0.719	4.1
3. $Q_{BEV} \times F_{BEV} \times E_{BEV}$ (E = .54 for beer, .48 for wine and .61 for spirits)	0.52	0.02	0.04	0.23	1.22	3.84	18.4	82.6	0.831	5.1
4. $Q_{BEV} \times F_{BEV} \times E_{BEV}$ (E based on reported drink size[b])	0.56	0.02	0.04	0.23	1.29	4.57	19.1	82.4	0.785	4.7
5. $Q_{BEV} \times F_{BEV} \times E_{BEV}$ (E based on reported drink size[b]; adjusted for F when drank 5+ drinks)	0.58	0.03	0.04	0.25	1.50	3.53	22.1	83.6	0.877	5.6
6. $Q_{BEV} \times F_{BEV} \times E_{BEV}$ (E based on reported drink size[b]; adjusted for F when drank 5+ and 9+ drinks)	0.61	0.03	0.04	0.26	1.56	4.58	22.7	83.8	0.877	5.6
Measures based on the 1992 National Longitudinal Alcohol Epidemiologic Survey										
7. $Q_{BEV} \times F_{BEV} \times E_{BEV}$ (E based on reported drink size[b]; adjusted for F when drank 5+ drinks)	0.64	0.01	0.04	0.31	1.67	3.40	26.7	85.4	1.051	7.9
8. $Q_{BEV} \times F_{BEV} \times E_{BEV}$ (E based on reported drink size[b]; adjusted for heaviest intake of each beverage)	0.72	0.02	0.05	0.31	1.64	5.77	25.9	84.9	0.987	7.0

[a]Odds of abuse or dependence among persons with an average daily ethanol intake of 1.5 oz (approx. three drinks per day) relative to odds among persons with an average daily intake of 0.21 oz (approx. three drinks per week) = exp (beta [log(1.5) − log(0.21)]).

[b]Based on ethanol conversion factors of .045 for beer, .121 for wine and .409 for spirits.

Note: Standard errors are .01 for all mean volumes; are <.001 for all $P_{.01}$; vary from <.001 to .003 for $P_{.10}$; vary from .001 to .005 for $P_{.50}$; vary from <.001 to .04 for $P_{.90}$; vary from .001 to 1.00 for $P_{.99}$; vary from 0.3 to 0.4 for percentages of drinkers exceeding cutpoint; and vary from .023 to .030 for beta coefficients.

the beta parameter times the log of 1.5 minus the log of 0.21, that is, $e^{beta\ (log\ 1.5–log\ 0.21)}$. These two consumption levels were selected to exemplify the contrast between an immoderate and a moderate level of average daily ethanol intake.

The NHIS and the NLAES both employed complex sample designs (Grant et al., 1994; Massey et al., 1989). Both surveys selected primary sampling units with probability proportional to size and oversampled blacks, and the NLAES also oversampled young adults ages 18–29 at the household level. Accordingly, standard errors of estimates were derived using SUDAAN (Shah et al., 1995), a software packages that uses Taylor series linearization to account for the characteristics of complex, multistage samples in its variance estimates.

RESULTS

Measure 1, based on usual quantity and frequency of all beverage types combined and an assumed ethanol content per drink of 0.5 oz, yielded the lowest average volume of any measure examined, 0.43 oz of ethanol per day (Table 1). On the basis of this measure, 12.2% of drinkers 18 years of age and over were classified as having exceeded the moderate-drinking cutpoint. The odds of past-year alcohol abuse or dependence were 4.1 times as high for an individual who consumed three drinks per day as for an individual who consumed three drinks per week. Increasing the assumed ethanol content per drink to 0.55 oz (Measure 2) yielded a 10% increase in mean daily volume (0.47 oz) and in each of the percentiles of the consumption distribution and almost a 50% increase in the proportion of drinkers exceeding the moderate-drinking cutpoint (18.1%). Since all individuals' intakes were increased by the same proportion, there was no change in the relationship of consumption and alcohol use disorders, and the beta coefficient and odds ratio for abuse and dependence were identical to those for Measure 1.

Accounting for individuals' beverage choices and changing the time reference period to the 2 weeks preceding interview or the most recent drink (Measure 3) yielded an additional increase in mean daily volume of intake (0.52 oz) as well as in both tails of the consumption distribution, but little additional increase in the proportion of drinkers exceeding the moderate-drinking cutpoint (18.4%). Using reported rather than median drink size (Measure 4) further increased both volume (0.56 oz) and the upper percentiles of the consumption distribution, but again had only a slight impact on the proportion of drinkers exceeding the moderate-drinking cutpoint (19.1%). Both Measures 3 and 4 resulted in odds ratios (5.1 and 4.7, respectively) that were higher than those for Measures 1 and 2.

Incorporating volume derived from days of drinking 5+ drinks (Measure 5) and days of drinking both 5+ and 9+ drinks (Measure 6) resulted in the highest volumes for the NHIS (0.58 oz and 0.61 oz, respectively). This reflected increases at most levels of the consumption distribution; however, accounting only for volume from days of drinking 5+ drinks (using the assumption of 3.0 oz of ethanol intake on those heavy drinking days) resulted in a reduction in the 99th percentile relative to Measures 3 and 4. Measures 5 and 6 yielded additional increases in the proportions of drinkers exceeding the moderate-drinking cutpoint (22.1% and 22.7%, respectively) and in the odds ratios for alcohol use disorders (coincidentally 5.6 for both measures).

Measures 6 and 7, based on the past-year reference period of the 1992 NLAES, resulted in the highest estimates of average daily ethanol intake (0.64 oz and 0.72 oz), of the proportion of drinkers exceeding the moderate-drinking cutpoint (26.7% and 25.9%) and of the odds ratios for alcohol use disorders (7.9 and 7.0). Use of the data on both usual and heaviest consumption of each type of beverage yielded considerably higher consumption values than were obtained by merely adjusting for frequency of drinking 5+ drinks, although this difference was evident only in the far right tail of the consumption distribution. Since the NLAES did not gather information on the frequency of drinking 9+ drinks, the adjustment for both 5+ and 9+ drinks could not be compared with usual and heaviest intake of each beverage.

Among the volume measures based on the NHIS (1-6), there was perfect correlation between Measures 1 and 2, which were constructed identically except with respect to the assumed ethanol content per drink. Measures 3–6, all based on beverage-specific data, were highly correlated among themselves (r = .85 to .99). Measures 1 and 2 were less strongly correlated with Measure 3 (r = .71) and with measures 4–6 (r = .58 to .63). The level of correlation between the two consumption measures based on the NLAES, Measures 7 and 8, was r = .72.

DISCUSSION

This comparison of volume measures found that estimated average daily intake was directly related to the number of questions upon which the estimate was based, with a range in mean volume from 0.43 oz based on two questions (usual frequency and quantity per drinking day) to 0.72 oz based on 21 questions

(ever drank beverage, overall frequency, usual quantity per drinking day, usual size of drink, heaviest quantity per drinking day, size of drink corresponding to largest quantity and frequency of drinking largest quantity, for each of the three types of beverage). These findings closely replicated results reported by Russell et al. (1991), who found that average daily ethanol intake in a sample of New York residents was 0.72 if based on beverage-specific questions and 0.49 if based on global questions. Replacing the nine questions on beverage-specific heaviest drinking with two general questions on frequency of drinking 5+ drinks and frequency of drinking 9+ drinks appeared to yield the next highest estimate of consumption, although this could not be verified on the basis of the NLAES, which asked only for the frequency of drinking 5+ drinks. The data from the NHIS demonstrated that using frequency of drinking 5+ drinks as the sole indicator of heavy drinking resulted in substantial foreshortening of the far right tail of the consumption distribution, because it capped the intake per heavy-drinking day to an assumed level (3.0 oz of ethanol) that was lower than that based on many of the heaviest drinkers' reported quantity and drink size.

Because so many current drinkers had not had any drinks in the 2 weeks preceding the NHIS interview (a reference period chosen for comparability with other questions in the NHIS), drinkers who had not consumed alcohol during that period were asked about their consumption during the 2 weeks immediately preceding their last drink. Although the date of last drink was asked, there were so many drinkers unable to recall this date that frequency of drinking could not be adjusted on the basis of interval since last drink. Thus, all estimates were based on a 2-week reference period that had to have contained at least one drink, essentially forcing all respondents to have a volume of intake based on at least 26 drinks per year. The effect of this can be seen in the lower tails of the consumption distribution for Measures 3–6, which are not as low as those based on reference periods that permitted drinkers to have as few as 12 drinks per year (the minimum quantity to be defined as a current drinker in both the NHIS and the NLAES). These findings indicate that, if interval since last drink must be incorporated into the estimation of frequency of drinking, it would be prudent to include a follow-up question utilizing broad response categories in order to obtain data from individuals unable to specify an exact interval length.

In comparing the effect of the 2-week reference period of the NHIS to the 1-year reference period of the NLAES, temporal shifts in alcohol consumption must be considered. Although sales data indicated that the apparent per capita level of annual ethanol consumption for the population 14 years of age and older decreased from 2.49 gallons in 1988 to 2.31 gallons in 1992 (Williams et al., 1995), this decrease was the result of a decline in the proportion of drinkers in the adult population from 51.6% to 44.4% (Dawson and Archer, 1992; Dawson et al., 1995b). Adjusting the per capita consumption figures by the proportion of current drinkers revealed that consumption among current drinkers actually increased by about 8% between 1988 and 1992. Thus, the 10% increase in volume based on Measure 7 compared to Measure 5 is just slightly more than would be expected solely on the basis of temporal changes in consumption, lending support to Room's statement (1990) that "there is little evidence that the time period specified makes much difference in the aggregate when the respondent is being asked about his or her customary behavior" (p. 73).

At the same time, any tendency for recall to have deteriorated over the NLAES 1-year reference period may have been overshadowed by other factors favoring larger consumption estimates in the NLAES, most notably the coding for the size of a drink of wine. If respondents did not provide a specific estimate of the number of ounces per glass of wine, the NLAES questionnaire contained response categories linking a "small" glass of wine to 6 oz, a "medium" glass of wine to 8 oz and a "large" glass of wine to 12 oz. In contrast, NHIS interviewers were instructed to code a "medium" glass of wine (exact size not specified) as 4 oz. Hence, the modal value for a glass of wine doubled between 1988 and 1992. In addition to its effect on aggregate volume estimates, a shift of this nature has implications for gender comparisons in consumption, since women obtain a far greater proportion of their ethanol from wine than do men (Dawson et al., 1995a).

This analysis indicated that the proportion of drinkers whose average daily ethanol intake exceeded a volume-based cutpoint for moderate drinking was more sensitive to changes in consumption measurement than was either mean volume or the beta coefficient representing the association between consumption and alcohol use disorders, suggesting that analyses based on absolute cutpoints may be particularly vulnerable to errors or assumptions employed in measurement. In general, the strongest associations between intake and alcohol use disorders (i.e., the largest estimated beta coefficients) were obtained when the reference period for the problem indicators matched the reference period for the measure of alcohol intake, as was the case with the measures based on the NLAES, and when the consumption measure in-

corporated aspects of drinking that are likely to result in problems, such as the frequency of heavy drinking. Because odds ratios are derived by exponentiating a multiplicative function of the beta coefficient, they are inherently more sensitive to changes in measurement than are the coefficients themselves, particularly if the odds ratios reflect a comparison of widely divergent consumption levels. For example, had the odds ratios presented in Table 1 compared a consumption level of three drinks per day with a level of one drink per month, the odds ratio for Measure 7 would have been approximately five times as high as that for Measure 1 (126.5 compared to 27.4).

A comparison of the model results across consumption measures illustrates the impact of making assumptions that ignore the association between intake and alcohol use disorders. In Measure 3, the ethanol content per drink was estimated on the basis of the median size of drink for beer, wine and spirits. Since the actual drink sizes reported by individuals with abuse or dependence exceeded those reported by individuals without alcohol use disorders, the use of the median size tended to foreshorten the consumption distribution of the former while extending that of the latter. As a result, the use of the median drink size increased the slope or beta coefficient for Measure 3 relative to that obtained by using reported drink size in Measure 4. Use of the median drink size did not adversely affect the predictive ability of the consumption measure, as the proportion of cases predicted concordantly was larger for Measure 3 than for Measure 4. In this analysis, the proportion of cases predicted concordantly increased directly with the magnitude of the beta coefficient—the larger the beta coefficient, the higher the predicted probability of abuse or dependence for each case and the more cases with predicted probabilities sufficiently high to be predicted as "positive" for abuse or dependence. Increasing the number of cases predicted as positive for an outcome measure would not necessarily result in a higher proportion of cases predicted concordantly; the opposite would be true if the increase resulted in more false positives than true positives. In this analysis, however, the measures with the strongest levels of association were consistently the best in terms of predictive power—probably because they had the greatest sensitivity in discerning alcohol use disorders among individuals whose relatively low levels of average daily intake disguised episodic bouts of heavy drinking.

The data presented in this report clearly illustrate that relatively small differences in aggregate volume of intake may be accompanied by far larger differences in specific aspects or correlates of intake (e.g., in the proportion of drinkers exceeding some specific level of intake or in the association between intake and alcohol-related problems). The sensitivity of such measures to shifts in the overall consumption distribution and the importance of these measures in both formulating and evaluating public health policy initiatives underscore the need for careful consideration of the most appropriate consumption measures for inclusion in alcohol surveys. Three general areas of additional research are suggested by these findings. First is the extension of this analysis to include differential effects of measurement on various subgroups of drinkers, including those defined by frequency or quantity of drinking. Second is exploration of the effect of varying the assumptions as to intake level on days of drinking 5+ or 9+ drinks. Third is assessment of the differential reliabilities of frequency, quantity, drink size, frequency of heavy drinking and summary measures based on different combinations of these elements.

APPENDIX

The exact formulas for the eight consumption measures analyzed in this report are presented below:

Measure 1: ([Usual quantity of drinks per drinking day] x [usual number of drinking days per year] x .50)/365; based on no specified reference period.

Measure 2: ([Usual quantity of drinks per drinking day] x [usual number of drinking days per year] x .55)/365; based on no specified reference period.

Measure 3: {([Usual quantity of beers per beer-drinking day] x [usual number of days per year when drank beer] x .54) + ([usual quantity of drinks of wine per wine-drinking day] x [usual number of days per year when drank wine] x .48) + ([usual quantity of drinks of spirits per spirits-drinking day] x [usual number of days per year when drank spirits] x .61)}/365; based on 2 weeks preceding interview or most recent drink.

Measure 4: {([Usual quantity of beers per beer-drinking day] x [usual number of days per year when drank beer] x [usual size of beer in oz] x .045) + ([usual quantity of drinks of wine per wine-drinking day] x [usual num-

ber of days per year when drank wine] x [usual size of drink of wine in oz] x .121) + ([usual quantity of drinks of spirits per spirits-drinking day] x [usual number of days per year when drank spirits] x [usual size of drink of spirits in oz[a]] x .409)}/365; based on 2 weeks preceding interview or most recent drink.

Measure 5: {([365 – number of days per year when drank 5+ drinks] x [Measure 4]) + ([frequency of drinking 5+ drinks] x 3.0)}/365; with Measure 4 based on 2 weeks preceding interview or most recent drink and frequency of drinking 5+ drinks reported for year preceding interview.

Measure 6: {([365 – number of days per year when drank 5+ drinks] x [Measure 4]) + ([number of days per year when drank 5+ drinks] x 3.0) + ([number of days per year when drank 9+ drinks] x 2.0)}/365; with Measure 4 based on 2 weeks preceding interview or most recent drink and frequencies of drinking 5+ and 9+ drinks reported for year preceding interview.

Measure 7: {([365 – number of days per year when drank 5+ drinks] x [average daily intake as measured in Measure 4, except with NLAES data]) + ([number of days per year when drank 5+ drinks] x 3.0)}/365; with Measure 4 and frequency of drinking 5+ drinks both reported for year preceding interview.

Measure 8: {([Usual quantity of beers per beer-drinking day] x [total number of days per year when drank beer – number of days when drank largest quantity of beer] x [usual size of beer in oz] x .045) + ([largest quantity of beers per beer-drinking day] x [number of days per year when drank largest quantity of beer] x [size of beer in oz when drinking largest quantity] x .045) + ([usual quantity of drinks of wine per wine-drinking a day] x [total number of days per year when drank wine – number of days when drank largest quantity of wine] x [usual size of drink of wine in oz] x [.121]) + ([largest quantity of drinks of wine per wine-drinking day] x [number of days per year when drank largest quantity of wine] x [size of drink of wine in oz when drinking largest quantity] x .121) + ([usual quantity of drinks of spirits per spirits-drinking day] x [total number of days per year when drank spirits – number of days when drank largest quantity of spirits] x [usual size of drink of spirits in oz[a]] x [.409]) + ([largest quantity of drinks of spirits per spirits-drinking day] x [number of days per year when drank largest quantity of spirits] x [size of drink of spirits in oz[a] when drinking largest quantity] x .409)}/365; based on year preceding interview.

[a]Excluding mixer, water, etc.

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Theoretical and Observed Subtypes of DSM–IV Alcohol Abuse and Dependence in a General Population Sample

Bridget F. Grant

The purpose of this study was to quantify the degree of heterogeneity of the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition *(DSM-IV) alcohol abuse and dependence categories by comparing the number of theoretically predicted subtypes of each category with those observed in a nationally representative sample of the U.S. general population. Among respondents classified with a past year diagnosis of abuse, only 11 (47.8%) of the 23 theoretically predicted subtypes of abuse were observed, while 53 (53.5%) of the 99 theoretically predicted subtypes of dependence were observed in this general population sample. Approximately 90% of the respondents classified with abuse could be represented by three subtypes of abuse and 70% of the respondents with current diagnoses of dependence could be characterized by six subtypes of dependence, indicating the relative homogeneity of both diagnostic categories. Sociodemographic differentials were also observed including the reduction in the number of observed subtypes of abuse and dependence with age as well as the larger numbers of subtypes associated with males and whites relative to females and blacks, respectively. Implications of these results are discussed in terms of increased physical morbidity and disruption of family life as persons with alcohol use disorders age, the potential role of physiological and impaired control over drinking indicators of dependence as critical features of the disorder, and the future need to examine the conceptual basis of the abuse category and to conduct longitudinal epidemiological research.*

INTRODUCTION

The heterogeneity of diagnostic categories appearing in psychiatric classification systems has been a matter of concern for many years (Wittenborn and Holzberg. 1951, 1953; Rotter, 1954). The definition of alcohol dependence appearing in the *Diagnostic and Statistical Manual of Mental Disorders-Third Edition-Revised* (DSM-III-R) (American Psychiatric Association, 1987) has been subject to similar criticism. The requirement that at least three of nine dependence criteria be met for a positive DSM-III-R diagnosis of dependence predicts the diversity of behaviors subsumed within its broad boundaries.

Despite the defined inclusiveness of the DSM-III-R dependence construct, only one empirical study has been conducted to assess its departure from homogeneity. In that study, the number of theoretical subtypes of dependence predicted from combinatorial theory was compared with the number of empirically observed subtypes of dependence (Grant et al., 1992). Forty-one percent (n = 189) of the theoretically predicted subtypes of alcohol dependence (n = 466) were observed in the general population sample, indicating that the category was indeed heterogeneous, but not as heterogeneous as predicted. Symptoms of alcohol dependence representing physiological dependence (i.e. tolerance and or withdrawal) and impaired control over drinking also appeared as criteria in over 80% of all empirical subtypes of dependence regardless of gender, race or age.

The purpose of this study is to utilize the methodology of Grant et al. (1992) to examine departures from homogeneity resulting from the broad and inclusive structure of the most current psychiatric classification of alcohol dependence appearing in the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV) (American Psychiatric Association. 1992) in a general population sample. Moving beyond the approach of Grant et al. (1992), the quantification of the degree of heterogeneity of the alcohol abuse category will also be examined by comparing the number of theoretically predicted subtypes of abuse to those empirically observed. Major empirical subtypes of alcohol abuse and dependence will be identified across important sociodemographic subgroups of the population with a view toward understanding the structure underlying these diagnostic categories. It will also be of interest in this study to quantify the potential reduction in heterogeneity of

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the dependence category resulting from the reduction of the total number of alcohol dependence criteria from nine in the DSM-III-R to seven in the DSM-IV.

METHODS

Sample

This study was based on the National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism. Fieldwork for the study was conducted by the United States Bureau of the Census in 1992. Direct face-to-face interviews were administered to 42,862 respondents, 18 years of age and older, residing in the noninstitutionalized population of the contiguous United States, including the District of Columbia. Approximately 92% of the selected households participated in this survey while 97.4% of the randomly selected respondents in these households participated in this survey, yielding an overall response rate of 90%.

The NLAES consisted of a complex multistage design which featured sampling of primary units with probability proportional to size and oversampling of the black and young adult (18–29 years) populations. The NLAES design has been described in detail elsewhere (Massey et al., 1989; Grant et al., 1994).

DSM-IV dependence and abuse measures

DSM-IV alcohol use disorders were derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained lay interviewers (Grant and Hasin, 1992). The AUDADIS included an extensive list of symptom items that operationalized the DSM-IV criteria for alcohol abuse and dependence.

Respondents were classified with past year dependence if they met at least three of the seven DSM-IV criteria for dependence within the 1-year period preceding the interview: tolerance; withdrawal or avoidance of withdrawal; desire/attempts to cut down or stop drinking; much time spent on drinking, obtaining alcohol or recovering from its effects; reduction/cessation of important activities in favor of drinking; impaired control; and continued drinking despite physical or psychological problems caused or exacerbated by drinking. Respondents were classified with past year alcohol abuse if they met at least one of the four DSM-IV criteria for abuse in the 1-year period preceding the interview: alcohol-related legal problems; continued drinking despite interpersonal problems; neglect of role responsibilities due to drinking; and drinking in hazardous situations.

The AUDADIS diagnoses of past year alcohol abuse and dependence also satisfied the clustering and duration criteria of the DSM-IV definitions. The criteria of the DSM-IV require the clustering of symptoms of each diagnosis within the 1-year period preceding the interview in addition to associating duration qualifiers with certain abuse and dependence criteria. The duration qualifiers are defined in the DSM-IV as the repetitiveness with which symptoms must occur in order to be counted as positive toward a diagnosis. They are represented by the terms 'recurrent' and 'persistent' appearing in the description of the diagnostic criteria. The reliability of past year DSM-IV alcohol abuse and dependence diagnoses was 0.76 as ascertained from an independent test-retest study conducted in a general population sample (Grant et al., 1995).

Analysis

All tables present percentages of empirically derived subtypes of alcohol abuse and dependence based on weighted data, accompanied by the unweighted number of subtypes upon which they are based. All analyses included respondents who were classified with current (i.e. past 12 month) diagnoses of alcohol abuse and or dependence. Consistent with the DSM-IV definitions, the alcohol dependence group included respondents with and without additional diagnoses of abuse, while those classified with abuse diagnoses did not have diagnoses of alcohol dependence. Thus, the two groups were mutually exclusive.

The total number and theoretical subtype of alcohol dependence was determined by the formula for combinatorials, $[n!/(n - r!)r!]$, where $n = 7$ (i.e. the total number of dependence criteria) and $r = 3 - 7$ (i.e. the number of criteria that must be present above the cutoff for a positive diagnosis). For example, the number of potential subtypes for three out of seven dependence criteria can be calculated using the aforementioned formula where $n! = 7 \times 6 \times 5 \times 4 \times 3 \times 2 \times 1$ and $r! = 3 \times 2 \times 1$. The same formulation was used to estimate the total number of theoretical subtypes of the alcohol abuse category, where $n = 4$ (i.e. the total number of abuse criteria) and $r = 1 - 4$ (i.e. the number of criteria that must be present to achieve a diagnosis of abuse).

RESULTS

The prevalences of past year DSM-IV alcohol abuse and dependence in this general population sample

Table 1. Theoretical and observed subtypes of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) alcohol abuse and dependence by number of criteria satisfied

No. criteria satisfied	No. potential subtypes	No. observed subtypes	Percentage of total number of observed subtypes
Abuse			
4	1	0	0.0
3	12	3	1.2
2	6	4	15.1
1	4	4	83.7
Total	23	11	100.0
Dependence			
7	1	1	4.7
6	7	3	6.0
5	21	7	14.0
4	35	14	24.7
3	35	28	50.6
Total	99	53	100.0

were 3.03% (n = 1,186) and 4.38% (n = 1,724), representing 5,628,000 and 8,132,000 Americans, respectively. As shown in Table 1, only 11 (47.8%) of the 23 predicted subtypes of abuse were observed in this general population sample. Of the 99 theoretically predicted subtypes of dependence, only 53 (53.5%) were empirically observed. The majority of subtypes of abuse (83.7%) contained only one positive criterion, while the majority of dependence subtypes consisted of three (50.6%) or four (24.7%) positive criteria.

The number and percentage of empirically observed subtypes of alcohol abuse and dependence among sociodemographic groups of the population are presented in Table 2. Paralleling the distribution of the prevalence of abuse and dependence in the general population, more empirical subtypes were observed for males than females, whites than blacks, while the number of subtypes declined as a function of age.

Table 3 focuses on the number and percentage of empirical subtypes of abuse and dependence containing each diagnostic criterion. The 'drinking larger amounts or over longer periods than intended' and 'time spent in obtaining alcohol, drinking or recovering from its effects' criteria were the two most prevalent single diagnostic criterion, appearing in 62.3% of all empirically observed subtypes of dependence. The 'drinking despite physical or psychological problems' criterion was the least prevalent among the subtypes observed in this general population sample (45.3%). Interestingly, the two physiological indicators of dependence (i.e. tolerance and withdrawal), appeared together or alone in 43 of the 53 or in 81.1% of all observed subtypes of dependence. 'Hazardous use' was the most prevalent abuse criterion appearing in 63.6% of all observed abuse subtypes, while 'legal problems' was the least prevalent criterion (36.4%).

While examining the percentage of subtypes containing each criterion is informative, it can tell us very little about the most prevalent subtypes observed in this general population sample. For example, a particular criterion can appear in a majority of observed subtypes, but most of those subtypes may be of extremely low prevalence. To address this issue, the most prevalent subtypes of abuse and dependence among diagnosed respondents were examined. Fig. 1 shows the three most prevalent subtypes of abuse that together classified approximately 90% of all respondents with abuse diagnoses. Overall, the most prevalent subtype of abuse consisted of one criterion, 'hazardous use', classifying 66.9% of all abusers, followed in magnitude by the 'neglect of role' subtype and the combined 'neglect of role/hazardous use' subtype, each characterizing 10.6% of all respondents classified with abuse. The remaining eight of the total 11 subtypes of abuse not shown in Fig. 1 were generally associated with prevalences of less than l% of the total number of observed subtypes. With the exception of the respondents 45 years of age and older, the subtypes of greatest prevalence noted for the total sample were consistent for males and females, blacks and whites and among respondents in the two youngest age groups. For respondents aged 45 years and older the 'drinking despite interpersonal problems' criterion replaced the 'neglect of role' criterion in the second and third most prevalent subtypes of abuse.

Fig. 2 shows the six most prevalent subtypes of alcohol dependence by gender, race and age. For the total sample, the three most prevalent subtypes each classified between 13 and 15% of all respondents with dependence diagnoses. The fourth and fifth most prevalent subtypes each accounted for approximately 9% of all respondents, while the sixth most prevalent

subtype described 6% of all respondents classified with dependence. With one exception, each of the six most prevalent subtypes of dependence contained at least one physiological indicator of dependence (i.e. tolerance and/or withdrawal) and at least one indicator of impaired control over drinking (i.e. drinking more or longer than intended and/or desire or unsuccessful attempts to cut down or stop drinking). The remaining subtypes of dependence (n = 47) not shown in Fig. 2 were generally each associated with prevalences of less than 1% of the total number of observed subtypes. Although the six most prevalent subtypes noted for the overall sample were consistent across sociodemographic subgroups of the population, there was a departure noted for the 65 years and older age group. In that age group, the most prevalent subtype of dependence (14.7%) consisted of the two impaired control indicators, but unlike the other groups. the 'tolerance' criterion was replaced with the 'drinking despite physical or psychological problems' criterion. Most interestingly, all 53 observed subtypes of dependence contained at least one indicator of physiological dependence or impaired control over drinking.

DISCUSSION

Forty-eight percent (11/23) of the theoretical subtypes of DSM-IV abuse and 54% (53/99) of the theoretical subtypes of DSM-IV dependence were observed in this general population sample. These findings indicate that both diagnostic categories are heterogeneous, but not as heterogeneous as theoretically predicted. In previous work of Grant et al. (1992) in this area, 41% (n = 189) of the theoretical subtypes of DSM-III-R alcohol dependence were observed in a similar general population sample. Recall that the DSM-III-R required at least three positive dependence criteria of nine in order to achieve a dependence diagnosis while the DSM-IV requires at least three positive criteria of seven. Taken together, these results indicate that a re-

Table 2. Number and percentage of empirical subtypes of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) alcohol abuse and dependence by gender, race and age

Diagnosis/characteristic	No.	Percentage of total number of observed subtypes
Abuse		
Total	11	100.00
Gender		
Male	10	90.9
Female	9	81.8
Race		
White	11	100.0
Black	8	72.7
Age (in years)		
18–29	11	100.0
30–44	9	81.8
45–64	7	63.6
65 and older	3	27.3
Dependence		
Total	53	100.0
Gender		
Male	52	98.1
Female	36	67.9
Race		
White	51	96.2
Black	29	54.7
Age (in years)		
18–29	40	75.5
30–44	41	77.4
45–64	31	58.5
65 and older	18	40.0

Table 3. Number and percentage of empirical subtypes of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) alcohol abuse and dependence containing each criterion[a]

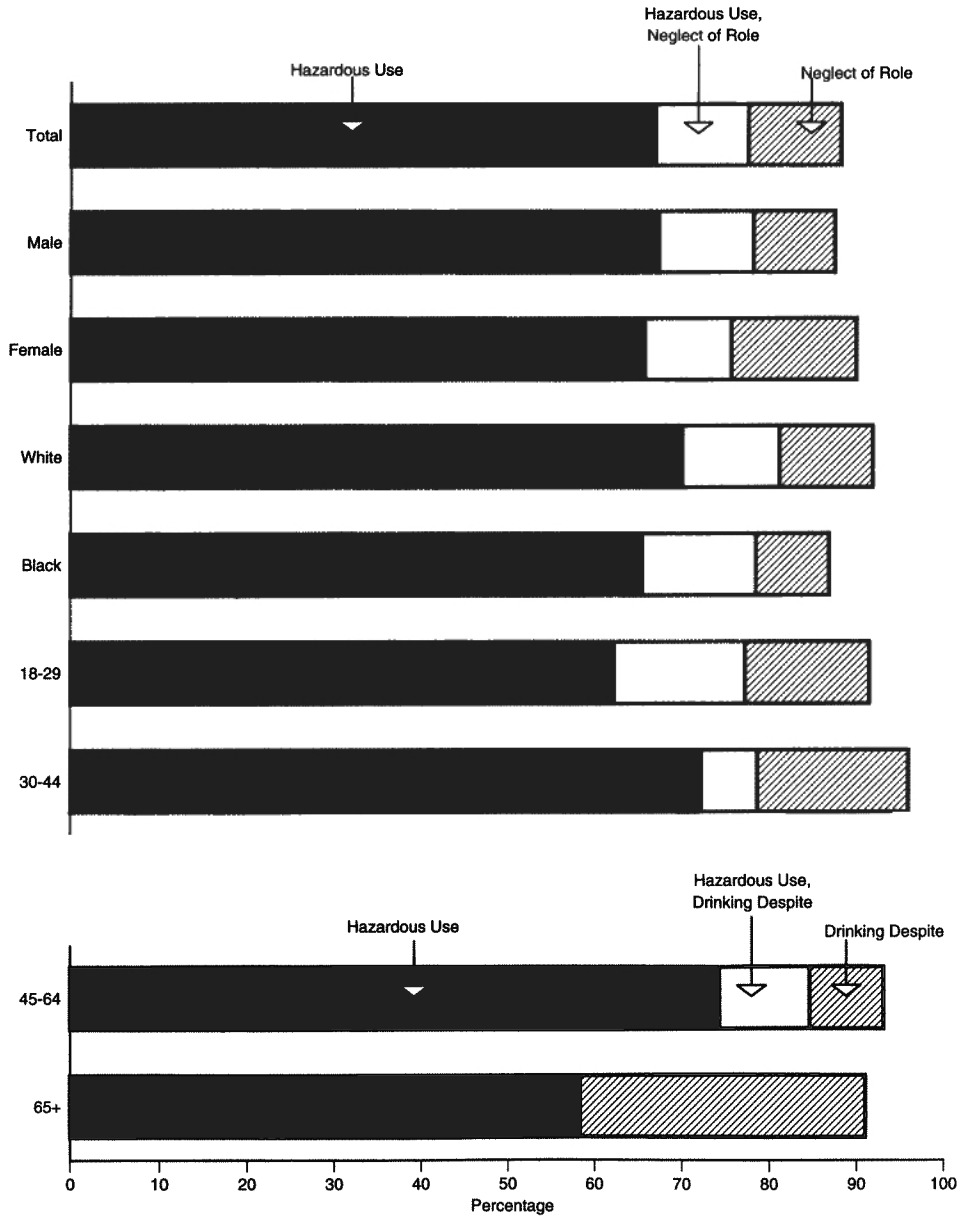
Criterion	No.	Percent of total number of observed subtypes
Abuse		
Total	11	100.0
Hazardous use	7	63.6
Neglect of role	5	45.5
Drinking despite interpersonal problems	5	45.5
Legal problems	4	36.4
Dependence		
Total	53	100.0
Physiological dependence	43	81.2
Tolerance	26	49.1
Withdrawal/relief withdrawal	28	52.8
Impaired control	42	79.3
Larger amounts/longer period	33	62.3
Desire/attempts to stop or cut down	26	49.1
Time spent	33	62.3
Important activities given up	30	56.6
Drinking despite physical/psychological problem	24	45.3

[a] Percentages based on weighted figures.

duction in the total number of dependence criteria from DSM-III-R to DSM-IV has not led to any significant reduction in heterogeneity of the alcohol dependence category.

The sociodemographic differential observed in the number of empirical subtypes of alcohol abuse and dependence shown in this sample may be, in part, attributed to death from competing causes among the elderly as the rate of common disabling medical problems, such as arthritis, cardiovascular disease, hypertension and stroke, and diabetes rise with age. Heavy drinking also increases the risk of numerous severe medical problems, most notably liver cirrhosis, cardiomyopathy, malnutrition and chronic pancreatitis. These explanations are supported by the finding that the number of empirical subtypes of abuse and dependence decreased with age. Unlike the other sociodemographic subgroups of the population, the most prevalent subtype of dependence among respondents 65 years and older contained the 'drinking despite

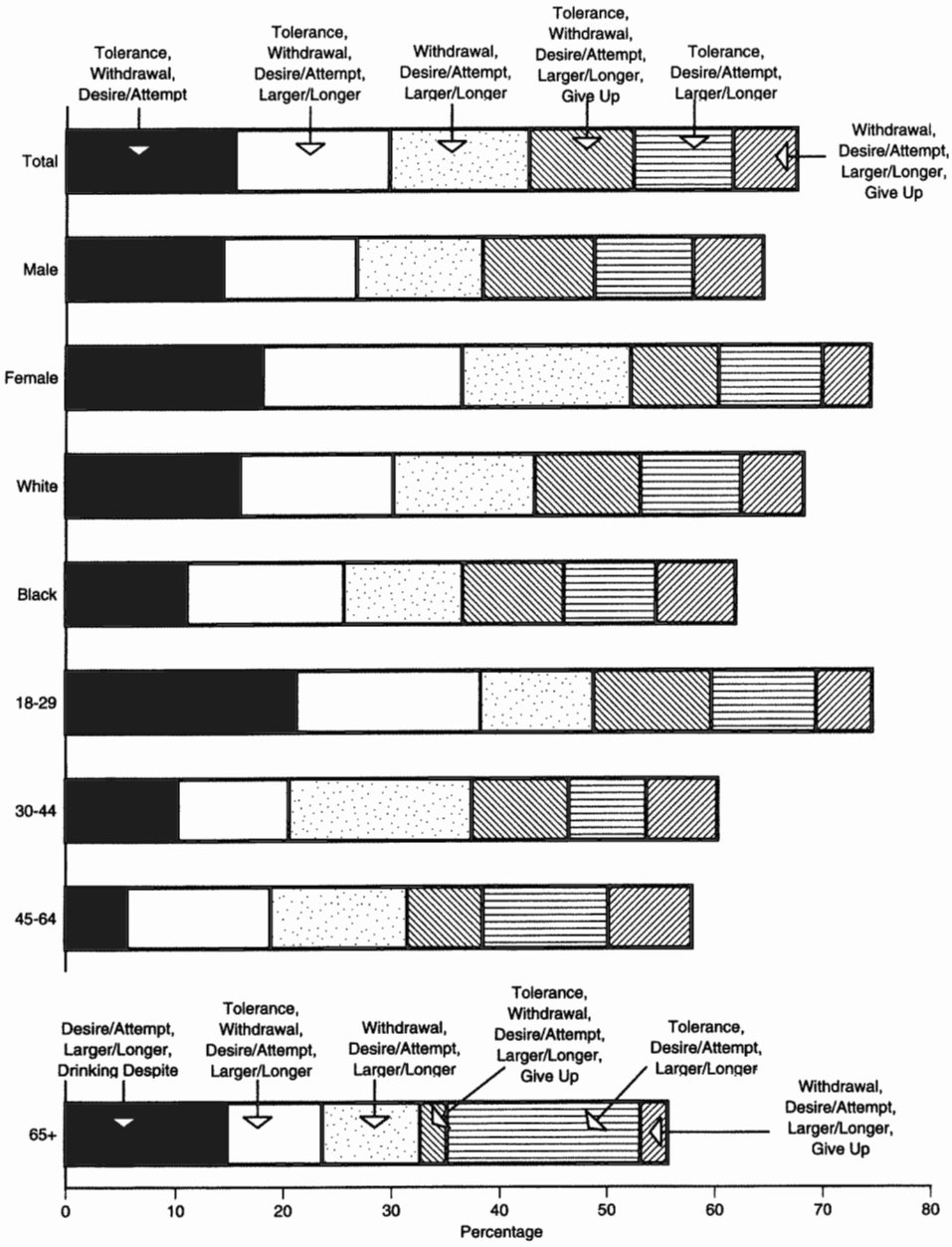
Figure 1. Three most prevalent subtypes of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) alcohol abuse as a function of gender, race and age



physical or psychological problems' criterion, again indicating that the physical morbidity from alcoholism may indeed be taking its toll as persons with alcohol use disorders age.

The results of this study also implicated reporting biases noted in other studies (Makela, 1978; Knupfer, 1982) (i.e. females and blacks may be less likely to report, but not to experience, alcohol abuse and dependence symptoms given the more severe societal constraints on excessive drinking in these subgroups of the population). Other findings suggest that, as people age, those with alcohol use disorders often have severe disruptions in family life. Unlike the other sociodemographic groups, the second and third most prevalent subtypes of alcohol abuse included the 'drinking despite interpersonal problems' criterion. This result may signal separation, divorce, or lack of contact with children frequently seen among older persons with alcohol use disorders. Since adult offspring are at greater risk themselves of abusing alcohol, they can also become

Figure 2. Six most prevalent subtypes of Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) alcohol dependence as a function of gender, race and age



supporters of continuing and excessive drinking patterns in aging parents, thereby reinforcing severe disruptions in interpersonal relationships.

Similar to the findings of Grant et al. (1992), criteria of alcohol dependence representing physiological dependence and impaired control over drinking were identified as playing a key role in the configuration of empirical subtypes, appearing in approximately 80% of all reported subtypes regardless of age, race or sex. These findings suggest that these criteria may be necessary, but not entirely sufficient, to identify individuals as belonging to the dependence category. Thus, these results support one aspect of the DSM-IV definition of alcohol dependence that provides for the subtyping of dependence as physiological and nonphysiological. It should be noted, however, that the observed subtypes of dependence for other drugs may differ from those of alcohol depending on their physiological addiction liability.

When viewed in terms of overall prevalence of each subtype, and not in terms of the total number of observed subtypes, the striking degree of homogeneity of both DSM-IV abuse and dependence categories becomes evident. Approximately 90% of the respondents classified with abuse could be characterized by three subtypes of dependence, while almost 70% of all respondents classified with alcohol dependence could be described by six subtypes of dependence. These are remarkable findings when viewed within the context of the sheer number of theoretically predicted subtypes of DSM-IV alcohol abuse and dependence, that is, 23 and 99, respectively. With respect to alcohol abuse, hazardous use was the single most prevalent subtype reported among abusers. This result suggests that, at the conceptual level, the abuse construct may indeed be measuring a dimension of hazardous drinking. Future research directed towards defining and examining the conceptual basis of the DSM-IV alcohol abuse category might help clarify this issue.

Considering that almost 70% of all respondents classified with a dependence diagnosis could be characterized by only six subtypes of dependence speaks not only to the homogeneity of the category but also helps define what might be considered the critical features of the disorder. Not only did the six most prevalent subtypes of alcohol dependence contain a physiological and impaired control over drinking criteria, but all 53 observed subtypes of dependence contained at least one of these criteria. Clearly, both the physiological indicators of dependence might be candidates for the defining features of dependence. Whether the definition of dependence should be altered to require tolerance and/or withdrawal should be an issue subject to further investigation.

Although this study has provided an increased understanding of the structure underlying the DSM-IV alcohol abuse and dependence categories and their degree of homogeneity, there are limits to the strength of conclusions drawn from cross-sectional data. Future research should include longitudinal epidemiological research to define the appearance of dependence indicators over time. Longitudinal research can also establish the validity of the DSM-IV categories by identifying those abuse and dependence indicators that are most predictive of etiology, future course and treatment outcome.

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The Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS): Reliability of Alcohol and Drug Modules in a General Population Sample

Bridget F. Grant, Thomas C. Harford, Deborah A. Dawson, S. Patricia Chou, and Roger P. Pickering

Using a representative sample of the general population, the test-retest reliability of the alcohol and drug modules of the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS) was examined. The AUDADIS showed good to excellent reliability for measures of alcohol consumption and use of sedatives, tranquilizers, amphetamines, opioids (other than heroin), cannabis and cocaine. Equally good reliability was demonstrated for diagnoses of alcohol, cannabis, cocaine and heroin abuse or harmful use and dependence defined in terms of the International Classification of Diseases-Tenth Revision (ICD–10) and the Diagnostic and Statistical Manual of Mental Disorders-Third Edition-Revised (DSM–III–R) and Fourth Edition (DSM–IV). Results are discussed in terms of the need for future research on the psychometric properties of the AUDADIS in clinical and general population samples.

INTRODUCTION

Purpose

Very little is known about the psychometric properties of substance use and substance use disorder (i.e., abuse and dependence) modules of psychiatric assessment instruments. In a recent paper by Wittchen (1994), reliability studies of two fully structured diagnostic interviews incorporating substance use disorder modules were reviewed. These commonly used interviews were the Diagnostic Interview Schedule (DIS) of the National Institute of Mental Health (NIMH) (Robins et al., 1981) and the Composite International Diagnostic Interview (CIDI) of the World Health Organization (World Health Organization, 1990). This review clearly identified the dearth of test-retest reliability studies in general population samples as the major gap in our knowledge about the measurement of alcohol and drug use, abuse and dependence. Of the ten test-retest reliability studies associated with the DIS and CIDI, only one based on the general population was conducted in Germany (Semler and Wittchen, 1983) using a small (n = 20) community sample.

It is remarkable that while both the DIS and CIDI were primarily designed for use in epidemiological surveys, relatively little, if anything, is known concerning the reliability of their alcohol and drug use and disorder modules in the target population. Moreover, while the test-retest reliability coefficients reported by Wittchen (1994) for alcohol and drug use disorders among patient or clinical samples were good to excellent ($\kappa > 0.70$), reliabilities associated with alcohol consumption and drug use remained either unanalyzed or unreported.

The present paper reports the results of a test-retest of the alcohol and drug modules of the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS) of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) (Grant and Hasin, 1990) conducted in a general population sample. The purpose of this study was to determine if the promising reliability figures presented for alcohol and drug modules of fully structured psychiatric interviews found in clinical samples held true in the general population. Further, test-retest reliabilities are presented for major alcohol and drug use measures in addition to alcohol and drug use disorder measures, because of the critical distinction that must be maintained between these three distinct concepts of use, abuse and dependence.

Overcoming sources of unreliability

Similar to all methodological work conducted in general population samples, test-retest studies are costly and time and labor intensive. Thus, as an important preliminary to the fielding of this study, an in-depth review and analysis of sources of unreliability associated with fully standardized diagnostic interviews in

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the field was conducted. It became the major task in the development of the AUDADIS to overcome these reliability problems found in currently existing fully structured diagnostic interviews, particularly those related to the treatment of alcohol and drug measures. As a fully structured diagnostic interview, the AUDADIS is entirely standardized, relying exclusively on respondent self-report. The high degree of standardization of the AUDADIS aimed to reduce four of six sources of variance often cited by researchers (Spitzer and Fleiss, 1974; Robins, 1989; Wittchen, 1994) as responsible for clinical diagnostic unreliability. These sources of variance are: (i) the questions asked to assess psychiatric symptoms; (ii) the symptom information provided by the respondent; (iii) the time criteria used to evaluate clinical significance; and (iv) the interpretation of the information provided by the respondent.

The fifth source of variation impeding the reliability of clinical diagnosis concerns the interpretation of the diagnostic criteria described in the major classification systems. Efforts to reduce this source of unreliability in the AUDADIS included the conduct of extensive focus group sessions aimed at the translation of complex diagnostic criteria or symptomology into terminology understandable to lay persons. Often this translation process resulted in the disaggregation of an individual diagnostic criterion into several short and simplified questions representing the complex symptomology expressed by many single diagnostic criteria. The sixth source of variation, interviewer error, was reduced, in part, by simplifying the structure and format of the interview. The development of an extensive standardized training procedure including 16 h of pretraining and one week of in-class training also aimed to reduce interviewer error. The AUDADIS training included a detailed interviewer's manual, didactic presentation, role playing, and specific modules focused on increasing interviewers' knowledge of psychopathology in general and specifically of alcohol and drug classes and effects.

In addition to the sources of unreliability associated with the clinical diagnostic process discussed above, there are many other sources of unreliability that derive from the formatting of fully structured diagnostic interviews used in the past. Unlike the DIS and CIDI, the AUDADIS eliminated the use of skip-outs or the strategy of not asking subsequent symptom questions once enough questions had been reported as positive to achieve an alcohol or drug diagnosis. In the AUDADIS, once a screen is responded to affirmatively (i.e., a respondent reports having drunk at least twelve drinks in any one year or having used a drug twelve or more times on his or her own), all alcohol or drug symptom questions are asked, respectively, regardless of whether diagnostic criteria have been met for a disorder. The elimination of skip-outs was important since it provides for a dimensional measure of psychopathology in addition to categorical measures or diagnoses.

Wittchen (1994) also noted another structural source of unreliability in both the DIS and CIDI, that is, the use of the probe flowchart. The probe flowchart consisted of a series of questions that followed a positive response to selected symptom items. These probe questions assessed the severity and clinical relevance of a symptom including whether the symptom caused significant impairment and help seeking or was always caused by medication, alcohol, drugs, or a physical illness or injury. In the AUDADIS, these important components of the severity and clinical relevance of each disorder are determined at the syndromal level as opposed to the symptom level, thereby helping to reduce yet another source of unreliability. It should also be noted that the CIDI and DIS probe flowchart questions did not appear in their associated alcohol and drug modules, thus leaving important aspects of substance use disorders unmeasured (e.g., social and occupational impairment, help seeking and their relationship to physical illness and injury). Moreover, the AUDADIS has incorporated a detailed module on alcohol and drug treatment utilization, a particularly important section for health services research and intervention and outcome studies.

Unlike the DIS and CIDI, the AUDADIS was the first instrument of its kind to include: (i) a clear distinction between current (i.e., past 12 months) and past (i.e., prior to the past 12 months) disorders for the purpose of determining lifetime diagnoses; (ii) measures of onset and recency of each disorder rather than the onset and recency of the first and last symptoms of the disorder; (iii) adequate measures of duration criteria (i.e., the repetitiveness of symptoms necessary to assess their clinical relevance); (iv) provisions for deriving hierarchical and non-hierarchical diagnoses; (v) comorbidity modules relating disorders; and (vi) measures of self-medication.

METHODS

Interview schedule

The AUDADIS is a fully structured diagnostic interview schedule developed for use in the National Longitudinal Alcohol Epidemiologic Survey (NLAES) of the NIAAA, a nation-wide survey on comorbidity of

substance use disorders and other psychiatric disorders initiated in 1992 (Grant and Hasin, 1992). The AUDADIS includes modules to measure major mood and anxiety disorders, antisocial personality disorder, substance-related medical conditions and family history of alcohol and drug use disorders (Grant and Towle, 1990). Alcohol and drug use diagnoses are derived according to three major classification systems: the Diagnostic and Statistical Manual of Mental Disorders-Third Edition-Revised (DSM–III–R; American Psychiatric Association, 1987); the Fourth Edition of the DSM (DSM–IV; American Psychiatric Association, 1994); and the International Classification of Diseases-Tenth Revision (ICD–10; World Health Organization, 1992). Even though the ICD–10 and DSM–IV diagnostic classifications of alcohol and drug use disorders were not finalized until 1992 and 1994, respectively, the diagnostic criteria that were to form the definitions were known in 1991 to close consultants and expert committee members that included the principal author. In this way, all newly developed DSM–IV and ICD–10 criteria were incorporated within the AUDADIS during the two years of its development, thus allowing for the test of these finalized diagnoses.

As a fully structured, standardized and precoded instrument, the AUDADIS can be administered by nonclinicians or clinicians, and the precoded response categories facilitate fully computerized diagnostic analysis. The AUDADIS assesses harmful use, abuse and dependence diagnoses for alcohol and eight classes of psychoactive drugs including tranquilizers, sedatives, opioids (other than heroin), amphetamines, cocaine (and crack-cocaine), cannabis (including hash and THC), heroin and methadone, and provides for diagnoses of 'other' drugs of abuse including hallucinogens, inhalants and solvents, antidepressants and antipsychotics. Generally, diagnostic questions associated with alcohol and drug use disorders are asked of all respondents drinking at least 12 drinks during any one-year period of their lives or using any drug on their own (i.e., without a prescription, or longer, more often or in greater amounts than prescribed) more than 12 times on a lifetime basis. The AUDADIS also contains extensive alcohol and drug use modules that measure frequency and quantity (if appropriate) of use. For alcohol, a comprehensive set of questions also measures beverage-specific quantity and frequency during the usual and heaviest drinking periods of the respondent.

Sample

The test-retest of the AUDADIS was conducted in a representative sample of Essex County, New Jersey. The sampling design was both stratified and clustered, and consisted of several stages. First, an initial screening sample was selected consisting of 5,987 persons in 4,040 households. In-person screening interviews were conducted with a contact person in each of these initial-screening households. The contact person was asked to provide information on the critical alcohol screening question (i.e., the largest number of drinks consumed on any one occasion during the past twelve months) for themselves and for each member of their household 18 years of age and older. Screening for consumption level at this stage in the sampling was conducted in order to increase the prevalence of alcohol and drug use disorder diagnoses available for analysis. From the initial sample of households, a sample of households eligible for the final sample was selected. Households eligible for final sample contained at least one individual who drank three or more drinks on any one occasion during the last 12 months. There were 1,652 persons in 1,142 households defined to be eligible for final sample. Use of the informant method of ascertaining drinking status for other family members most likely resulted in an underestimation of the number of households eligible for sample. However, it is important to remember that all screening techniques are, by definition, probabilistic and designed to yield higher prevalences of target disorders. As such, screening measures used in our study were successful.

From the list of eligible households, a final sample of 664 households (one individual per household) was selected for the reliability study using a two-step procedure. In the first step, a random sample from within each sampling stratum was selected using a procedure for oversampling females. The goal of this oversampling procedure was to select households in such a way as to have half females in each race-ethnic group-age cell in the final reliability sample. Without oversampling, the universe of heavy drinkers contains many more men than women. The second step was only enforced for each household selected for the final sample that contained more than one eligible individual. In these cases, one individual was randomly subsampled, again using a procedure for oversampling females that preserved the oversampling for females achieved at the household level in the first step (i.e., approximately 50% females in each race-ethnic group-age cell). If it was not possible for half of the final sample in a given race-ethnic group-age cell to be female, then females were sampled at the maximum possible rate from that cell.

Of the 664 persons contacted for the initial interview, 545 persons agreed to participate in the study and 473 persons completed both interviews. The re-

sponse rate was 82.1% after the first interview and 86.7% after the second interview, for a total response rate of 84.4%. The sample was 57.1% male and 42.9% female. Thirty-four percent were black with the remaining 66.0% non-black. The majority of the sample was aged 25–44 years (51.8%) and 45–64 years (30.4%), with 8.0% in the youngest age group (18–24 years) and 9.8% in the oldest age group (65 years and older). Sixty-four percent of the sample had less than a high school education and 36.0% had earned a high school or higher degree. Over half (52.1%) the sample was married or living with someone as if married, while 18.4% were divorced, separated or widowed and 29.5% were never married.

Test-retest design

Interviewer assignments during the initial test and subsequent retest were entirely randomized among the 33 interviewers, with the qualification that no one interviewer was allowed to interview the same respondent twice. The interviewers administering the retest interviews were also blind to the results of the initial interview. Any communication between the interviewers concerning information collected during the interview was strictly confidential in accordance with Bureau of the Census and NIAAA standards and requirements. The initial and retest interviews were conducted four to six weeks apart.

Statistical analysis

For dichotomous and categorical data elements, kappa was used as a measure of reliability and is defined as a measure of pairwise agreement corrected for chance (Schouten, 1980). For continuous measures, intraclass correlation coefficients are presented as measures of reliability. Since our reliability design assumed that interviewers were randomly drawn from a larger population of interviewers, we used a one-way random effects ANOVA model to derive intraclass correlation coefficients (Shrout and Fleiss, 1979). Kappa and intraclass correlation coefficients (when the number of interviewers equals 2) range from 1.00 (perfect agreement) to –1.00 (total disagreement). A kappa or intraclass correlation coefficient of zero indicates agreement no better than chance. Kappas of 0.75 and above indicate excellent agreement, from 0.40 to 0.74 indicate fair to good agreement, and below 0.39 indicate poor agreement (Fleiss, 1981).

For the purposes of present analyses, reliabilities were calculated for harmful use or abuse and dependence for all substances with a non-zero prevalence: alcohol, any drug use, cannabis, cocaine and heroin.

Table 1. Reliability of AUDADIS alcohol consumption measures

Consumption measure	Intraclass correlation coefficient
Days per year drank usual quantity of all beverage types combined (past year)	0.76 (0.719, 0.796)[a]
Days per year drank usual quantity of all beverage types combined (period of heaviest consumption)	0.70 (0.647, 0.741)
Quantity consumed per occasion when drinking usual quantity (past year) of:	
Beer	0.76 (0.713, 0.792)
Wine	0.75 (0.704, 0.785)
Liquor	0.87 (0.842, 0.887)
Quantity consumed per occasion when drinking heaviest quantity (past year)	
Beer	0.84 (0.792, 0.851)
Wine	0.88 (0.858, 0.899)
Liquor	0.59 (0.528, 0.647)
Typical size, in ounces, of beverage consumed when drinking usual quantity (past year)	
Beer	0.97 (0.968, 0.979)
Wine	0.99 (0.986, 0.991)
Liquor	0.62 (0.560, 0.672)
Typical size, in ounces, of beverage consumed when drinking heaviest quantity (past year)	
Beer	0.72 (0.671, 0.762)
Wine	0.94 (0.929, 0.951)
Liquor	0.77 (0.733, 0.807)
Average daily ethanol intake (oz) from all beverage types when drinking usual quantities (past year)[b]	0.73 (0.682, 0.768)
Average daily ethanol intake (oz) taking into account usual and heaviest quantities of all beverage types (past year)[b]	0.73 (0.689, 0.774)
Average daily ethanol intake (oz) from all beverage types when drinking usual quantities (period of heaviest consumption)[b]	0.72 (0.670, 0.759)
Average daily ethanol intake (oz) taking into account usual and heaviest quantities all beverage types (period of heaviest consumption)[b]	0.71 (0.662, 0.752)

[a]Numbers in parentheses are 95% confidence intervals.

[b]Classified in ounces as: 1, less than 0.10; 2, 0.10–0.24; 3, 0.25–0.49; 4, 0.50–0.74; 5, 0.75–0.99; 6, 1.00–1.49; 7, 1.50–1.99; 8, 2.00–2.49; 9, 2.50 or more.

Similarly, kappa and intraclass correlation coefficients were calculated for all those substance use variables with a non-zero prevalence: alcohol, sedatives, tranquilizers, amphetamines, opioids (other than heroin), cannabis and cocaine. All analyses were conducted on

Table 2. Reliability of AUDADIS drug use measures[a]

Drug	Kappa coefficient (S.E.) (prevalence, first interview/prevalence, second interview)	
	Ever used 12 + times in lifetime	Used in the past 12 months
Sedatives	0.46 (0.17) (1.48/1.48)	0.50 (0.17) (1.48/1.48)
Tranquilizers	0.51 (0.16) (2.32/2.11)	0.50 (0.14) (2.32/2.11)
Amphetamines	0.82 (0.10) (1.90/1.69)	0.82 (0.10) (1.90/1.69)
Opioids (other than heroin)	0.66 (0.18) (1.05/0.84)	0.66 (0.19) (1.05/0.84)
Heroin	0.82 (0.10) (2.11/1.48)	0.79 (0.11) (2.11/1.48)
Cannabis	0.78 (0.04) (19.45/16.70)	0.77 (0.04) (19.45/16.70)
Cocaine	0.83 (0.06) (5.28/5.28)	0.86 (0.05) (5.28/5.28)

Prevalence reported as weighted percentages.
[a]Measures coded: 0, no; 1, yes.

Table 3. Intraclass correlation coefficients for AUDADIS drug use measures

Drug	Age at first use	Number of times used prior to past year
Sedatives	0.69 (0.633, 0.729)	0.80 (0.765, 0.831)
Tranquilizers	0.92 (0.903, 0.931)	0.79 (0.740, 0.812)
Amphetamines	0.92 (0.907, 0.934)	0.77 (0.693, 0.821)
Opioids (other than heroin)	0.94 (0.931, 0.951)	0.93 (0.928, 0.949)
Heroin	0.68 (0.622, 0.721)	0.69 (0.640, 0.736)
Cannabis	0.93 (0.916, 0.948)	0.78 (0.739, 0.811)
Cocaine	0.77 (0.726, 0.801)	0.85 (0.817, 0.869)

Numbers in parentheses are 95% confidence limits.

the total sample ($n = 473$). Analyses by age, ethnicity and sex were precluded by low prevalence.

RESULTS

Intraclass correlation coefficients for major AUDADIS alcohol consumption measures are presented in Table 1. Average daily ethanol intake measures shown in Table 1 represent the product of frequency, quantity, drink size and an ethanol conversion factor (beer, 0.045; wine, 0.121; liquor, 0.409) summed over the three beverage types, with the resulting volume divided by 365.

Intraclass correlations for consumption measures were good to excellent, ranging from 0.70 to 0.99. Our volume measures of average daily consumption demonstrated good reliability (0.71–0.73) regardless of whether usual and/or heaviest quantities across beverage types were considered and regardless of time frame (i.e., past year, period of heaviest consumption). In general, the typical size of each beverage type demonstrated greater reliability than quantity or frequency of consumption of each beverage type.

Tables 2 and 3 show the reliability coefficients associated with major AUDADIS drug use measures. Kappa values for ever use of amphetamines, heroin, cannabis and cocaine at least twelve times in one's lifetime were excellent (> 0.78), while corresponding reliabilities for sedatives, tranquilizers and opioids were fair to moderate (0.46–0.66). A similar level of agreement by drug type was also found for the measures of use in the past twelve months. Intraclass correlation coefficients for age of first use measures were greatest for tranquilizers, amphetamines, opioids and cannabis (> 0.92) and somewhat lower, but fair, for sedatives (0.69), heroin (0.68) and cocaine (0.77). The reliability of the number of times used prior to the past year measures were somewhat lower than those associated with ages at first use but could all be considered fair to excellent. Not shown in Table 3 was the intraclass correlation coefficient associated with age of first use of alcohol, which was moderate (0.72).

Table 4 shows the kappa coefficients for all relevant alcohol and drug use disorders, for each diagnostic classification system and for both time frames. Agreement for diagnostic variables consisted of no diagnosis classification on test and retest, an abuse (or alternatively a harmful use) diagnosis on both occasions or a dependence diagnosis on both occasions. For past year diagnoses, reliabilities for any drug, cannabis and cocaine abuse and dependence were excellent, while alcohol and heroin abuse and dependence reliabilities were good despite their low prevalence in this general population sample. Reliabilities of all diagnoses associated with the prior to the past year time frame generally were lower than those demonstrated for the past year time frame, but were nonetheless quite good (0.50–0.80). As a general trend, DSM–III–R diagnoses had a slightly greater reliability (average $\kappa = 0.76$) in this sample than either DSM–IV (average $\kappa = 0.74$) or ICD–10 (average $\kappa = 0.72$) diagnoses. Regardless of time frame, an order effect was noted for

most alcohol and drug diagnoses in that the prevalence of most disorders was lower for the second interview as opposed to the first interview.

In addition to representing alcohol and drug symptoms as categorical diagnostic variables, the elimination of skip-outs in the AUDADIS also provided for the measurement of continuous or dimensional scales of symptom items. In Table 5, the intraclass correlation coefficients associated with alcohol and drug abuse and dependence scales are presented. Reliabilities associated with alcohol and drug use scales mirrored a pattern similar to their categorical counterparts. Intraclass correlation coefficients were greater for all drug classes compared to alcohol and greater for the past year time frame compared to prior to the past year time frame. However, unlike their corresponding categorical representations, reliabilities associated with DSM–IV harmful use and dependence scales were greater than either the DSM–III–R or ICD–10 scales.

DISCUSSION

The AUDADIS showed good to excellent reliability for major alcohol consumption measures and drug use measures for sedatives, tranquilizers, amphetamines, opioids, heroin, cannabis and cocaine. Equally good reliability was demonstrated for ICD–10, DSM–III–R and DSM–IV diagnoses of alcohol, cannabis, cocaine and heroin abuse or harmful use and dependence. While our concerns about poor reliability associated with low prevalence were not realized, our attempt to increase the prevalence of alcohol and drug use disorders by screening for high levels of alcohol consumption was not entirely successful. That is, at the present time, the reliability of AUDADIS methadone and "other" drug use (e.g., hallucinogens, inhalants and solvents) measures and prescription drug and methadone abuse and dependence measures is unknown in the general population. However, it was not surprising to find zero prevalence of prescription drug use, abuse and dependence in our sample since it encompassed Newark, New Jersey, a highly urban region characteristic of illicit use of cannabis, cocaine and heroin. Additional reliability studies are needed that target more suburban areas in which prescription drug use is more common.

It is noteworthy that the reliability of AUDADIS alcohol and drug use disorder measures in most cases equaled or exceeded those reported by Wittchen (1994) in patient samples for DSM–III–R alcohol (0.78) and drug use (0.73) disorders. Test-retest reliability should be greater in clinical compared to general population samples since more severe cases of substance use disorders are found in treatment while many more less severe, mild and borderline cases are found among community respondents. In view of this finding, it appears that the multiple efforts made during the AUDADIS development stage to reduce variation that often leads to diagnostic unreliability were somewhat successful. Specifically, the elimination of skip-outs, the assessment of help seeking and impairment and the attribution of medication, alcohol, drug and physical illness and injury at the syndromal level, and attention to simplifying operationalizations of complex

Table 4. Reliability of AUDADIS ICD-10, DSM-III-R, and DSM-IV alcohol and drug use disorder diagnoses[a]

Time frame/diagnosis	Kappa coefficients (S.E.) (prevalence, first interview/prevalence, second interview)		
	ICD-10	DSM-III-R	DSM-IV
Past year			
Alcohol abuse[b] and dependence	0.62 (0.09) (6.76/4.02)	0.70 (0.07) (10.36/7.19)	0.76 (0.05) (8.03/7.19)
Any drug abuse and dependence	0.80 (0.13) (1.27/0.84)	0.86 (0.05) (1.48/1.05)	0.79 (0.10) (2.11/1.47)
Cannabis abuse and dependence	0.82 (0.19) (0.42/0.21)	0.95 (0.07) (0.42/0.21)	0.78 (0.15) (0.84/0.63)
Cocaine abuse and dependence	0.93 (0.07) (1.05/.84)	0.94 (0.05) (1.27/1.05)	0.91 (0.06) (1.48/1.05)
Heroin abuse and dependence	0.67 (0.31) (0.42/0.21)	0.67 (0.31) (0.42/0.21)	0.66 (0.31) (0.42/0.21)
Prior to past year			
Alcohol abuse[b] and dependence	0.68 (0.11) (7.01/4.36)	0.69 (0.09) (9.72/8.45)	0.73 (0.06) (7.82/8.24)
Any drug abuse and dependence	0.56 (0.09) (5.28/4.65)	0.69 (0.07) (4.65/5.28)	0.66 (0.08) (5.70/4.65)
Cannabis abuse and dependence	0.69 (0.14) (1.48/0.84)	0.70 (0.14) (3.17/2.95)	0.71 (0.11) (3.17/3.17)
Cocaine abuse and dependence	0.60 (0.15) (1.69/1.69)	0.66 (0.12) (2.32/2.12)	0.68 (0.11) (2.54/2.12)
Heroin abuse and dependence	0.79 (0.13) (1.05/1.05)	0.80 (0.13) (1.05/1.05)	0.80 (0.13) (1.05/1.05)

Prevalence reported as weighted percentages.

[a]All diagnoses coded: 0, no diagnosis; 1, abuse (or harmful use) only; 2, dependence.

[b]For ICD-10, harmful use rather than abuse was examined.

diagnostic criteria should lead to future gains in the reliability of measures of psychopathology.

For most alcohol and drug use disorders, an order effect was observed, that is, the prevalence of these disorders generally decreased from first to second interview. This order effect may result from boredom or irritation on the part of respondents who participated twice in the same interview within a four- to six-week period or may, alternatively, reflect a change in psychopathological state. Although a conclusive explanation for the order effect cannot be offered from the present study, the decline in the prevalence of diagnoses from test to retest has been observed in a wide variety of other psychiatric assessment procedures (Helzer et al., 1981; Bromet et al., 1986). The decline in the prevalence of diagnoses in the second interview can be attributed to either a reduction in reporting symptoms or a failure to respond positively to the alcohol or drug use screening questions that are a prerequisite to being asked the subsequent alcohol or drug symptom items. Data from another test-retest study of the AUDADIS alcohol and drug use screening questions conducted with a subsample of 2,527 respondents participating in the NIAAA NLAES showed a gross error rate for alcohol and drug use screening measures of 2.0% and 0.8%, respectively (Grant et al., 1994). In view of this finding, it would appear that the order effect may be the result of a decline in symptom reporting rather than an increase in negative responses to the alcohol or drug use screens during the second interview. Future methodological research should focus on this important, yet not clearly understood, phenomenon.

The high degree of reliability found in this study for both alcohol and drug use, abuse and dependence measures suggests that the AUDADIS can be a useful diagnostic tool in research settings. More importantly, unlike other fully structured diagnostic interviews, the AUDADIS has demonstrated good to excellent reliability in a general population sample, the target sample for which it was designed.

Table 5. Intraclass correlation coefficients for AUDADIS alcohol and drug abuse and dependence items by classification system[a]

	Classification system		
	ICD-10	DSM-III-R	DSM-IV
Past year			
Alcohol			
Abuse	0.63 (0.569, 0.681)	0.74 (0.689, 0.774)	0.73 (0.683, 0.768)
Dependence	0.76 (0.722, 0.798)	0.77 (0.725, 0.801)	0.75 (0.706, 0.786)
Cannabis			
Abuse	0.92 (0.908, 0.935)	0.83 (0.796, 0.854)	0.86 (0.836, 0.883)
Dependence	0.95 (0.939, 0.957)	0.97 (0.965, 0.975)	0.94 (0.931, 0.952)
Cocaine			
Abuse	0.93 (0.908, 0.935)	0.81 (0.771, 0.835)	0.81 (0.779, 0.842)
Dependence	0.98 (0.982, 0.987)	0.97 (0.971, 0.979)	0.99 (0.989, 0.993)
Heroin			
Abuse	0.71 (0.657, 0.748)	0.80 (0.767, 0.832)	0.87 (0.841, 0.887)
Dependence	0.74 (0.698, 0.780)	0.74 (0.698, 0.780)	0.73 (0.686, 0.771)
Prior to past year			
Alcohol			
Abuse	0.66 (0.602, 0.706)	0.70 (0.644, 0.739)	0.73 (0.686, 0.771)
Dependence	0.64 (0.587, 0.694)	0.65 (0.597, 0.702)	0.63 (0.569, 0.680)
Cannabis			
Abuse	0.87 (0.845, 0.889)	0.68 (0.625, 0.724)	0.65 (0.595, 0.699)
Dependence	0.70 (0.649, 0.743)	0.73 (0.685, 0.771)	0.70 (0.647, 0.741)
Cocaine			
Abuse	0.64 (0.579, 0.688)	0.81 (0.774, 0.838)	0.86 (0.829, 0.878)
Dependence	0.89 (0.875, 0.911)	0.86 (0.831, 0.879)	0.89 (0.871, 0.908)
Heroin			
Abuse	0.77 (0.727, 0.802)	0.78 (0.750, 0.819)	0.83 (0.799, 0.856)
Dependence	0.81 (0.781, 0.843)	0.89 (0.877, 0.913)	0.81 (0.774, 0.837)

Numbers in parentheses are 95% confidence intervals.

[a]The number of items in abuse and dependence scales ranged from 5 to 13 and 19 to 30, respectively.

Future psychometric studies of the AUDADIS will be necessary in both clinical and general population samples to replicate the reliability findings of the present study. Currently, there are several research efforts aimed toward further understanding the psychometric properties of the AUDADIS. In 1995, a test-retest study of the alcohol and drug modules appearing in the AUDADIS is planned for primary care and psychiatric patient samples in Puerto Rico and New York. In mid-1995, the test-retest results of a modified version of the AUDADIS, the AUDADIS-Alcohol/ Drug–Revised (AUDADIS–A/D–R) prepared for the World Health Organization, will be available. These reliability studies were conducted in general population samples in India, Romania, Belgium and Australia. As part of this international project, the procedural validity of the AUDADIS–A/D–R was assessed using a clinical diagnostic interview referred to as the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) (World Health Organization, 1994) at two sites, the United States and Greece. Once the results of all of this psychometric research are available, the AUDADIS will have undergone the most extensive psychometric scrutiny of any existing psychiatric assessment procedure to date.

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DSM–IV, DSM–III–R, and ICD–10 Alcohol and Drug Abuse/Harmful Use and Dependence, United States, 1992: A Nosological Comparison

Bridget F. Grant

This study assessed agreement between DSM-IV, DSM-III-R, and ICD-10 diagnoses of alcohol and drug use disorders using data from a large representative sample of the United States population. Agreement between the three diagnostic systems for dependence was good to excellent for past year, prior to the past year, and lifetime diagnoses, for both genders, each ethnic group, and younger and older respondents. Cross-system comparisons between DSM-IV and DSM-III-R abuse were good to excellent, but concordance was consistently poor when ICD-10 harmful use diagnoses were compared with DSM-IV and DSM-III-R abuse diagnoses. Implications of these results are discussed in terms of the degree to which future research findings could be integrated with one another and the results from earlier studies using older versions of the DSM, to advance scientific knowledge in the drug and alcohol fields.

Beginning in the mid-1980s, the two major classifications of alcohol and drug use disorders, the Diagnostic and Statistical Manual of Mental Disorders-third edition-revised (DSM-III-R)[1] and the International Classification of Diseases-ninth revision (ICD-9)[2] were undergoing intensive reviews for the purpose of revision. As part of these revision processes, an international effort was undertaken by the World Health Organization, the American Psychiatric Association, the National Institute of Mental Health, the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and the National Institute on Drug Abuse to bring together these disparate classification systems. In June 1992, the World Health Organization published the final version of the ICD-10 classification subtitled "Clinical Descriptions and Diagnostic Guidelines" (ICD-10).[3] This version was developed primarily for general clinical, educational, and service-related purposes. Two years later, the final version of the DSM-fourth edition (DSM-IV) was published.[4]

The purpose of this study was to compare the final DSM-IV, DSM-III-R, and ICD-10 diagnostic categories of alcohol and drug abuse/harmful use and dependence using data from a large representative sample of the United States. The results of this nosological comparison have several important research implications. Because the DSM-IV and ICD-10 classifications are necessary tools for collecting and communicating accurate public health statistics, it is important to understand the concordance between the systems for comparative purposes, both nationally and internationally. The degree to which the DSM-IV and ICD-10 classifications agree will also represent an important gauge by which to judge the success of international efforts to integrate the diagnostic criteria for alcohol and drug use disorders. Most importantly, similarities and differences between each of the systems will determine the degree to which future research findings can be integrated with one another and with those results from earlier studies using older revisions of the DSM, to advance scientific knowledge in the alcohol and drug fields. Because of the increasing importance of sociodemographic characteristics in studies on health and illness in general, and in research on alcohol and drug use disorders specifically, the agreement between diagnoses was examined for subgroups defined by sex, ethnicity, and age.

METHODS

Study sample

This study was based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), a national probability sample sponsored by the

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NIAAA. The NLAES consisted of direct face-to-face interviews with 42,862 adults, 18 years of age and older, randomly selected from a nationally representative sample of households. Interviews were conducted in the respondents' homes and proxies were not permitted. The sampling design of the NLAES included stratification

Table 1. DSM-IV, DSM-III-R, and ICD-10 Diagnostic Criteria for Alcohol and Drug Dependence

	DSM-IV	DSM-III-R	ICD-10
Clustering criterion	A. A maladaptive pattern of substance use, leading to clinically significant impairment or distress as manifested by three or more of the following occurring at any time in the same 12-month period:	A. At least three of the following:	A. Three or more of the following have been experienced or exhibited at some time during the previous year:
Tolerance	(1) Need for markedly increased amounts of a substance to achieve intoxication or desired effect; or markedly diminished effect with continued use of the same amount of the substance	(1) Marked tolerance-need for markedly increased amounts of a substance (i.e., at least 50 percent increase) to achieve intoxication or desired effect, or markedly diminished effect with continue use of the same amount of a substance	(1) Evidence of tolerance, such that increased doses are required in order to achieve effects originally produced by lower doses
Withdrawal	(2) The characteristic withdrawal syndrome for a substance or use of a substance (or a closely related substance) to relieve or avoid withdrawal symptoms	(2) Characteristic withdrawal symptoms for substance (3) Substance often taken to relieve or avoid withdrawal symptoms	(2) A physiological withdrawal state when substance use has ceased or been reduced as evidenced by: the characteristic substance withdrawal syndrome, or use of substance (or a closely related sub of substance) to relieve or avoid withdrawal symptoms
Impaired control	(3) Persistent desire or one or more unsuccessfull efforts to cut down or control substance use (4) Substance use in larger amounts or over a longer period than the person intended	(4) Persistent desire or one or more unsuccessful efforts to cut down or control substance use (5) Substance use in larger amounts or over a longer period than the person intended	(3) Difficulties in controlling substance use in terms of onset, termination, or levels of use
Neglect of activities	(5) Important social, occupational, or recreational activities given up or reduced because of substance use	(6) Important social, occupational, or recreational activities given up or reduced because of substance use	(4) Progressive neglect of alternative pleasures or interests in favor of substance use; or
Time spent	(6) A great deal of time spent in activities necessary to obtain, to use, or to recover from the effects of substance use	(7) A great deal of time spent in activities necessary to obtain, to use, or to recover from the effects of substance use	A great deal of time spent in activities necessary to obtain, to use or to recover from the effects of substance use
Inability to fulfill roles	None	(8) Frequent intoxication or withdrawal symptoms when expected to fulfill major role obligations at work, school, or home; or	None
Hazardous use	None	Using a substance in a physically hazardous situation	None
Continued use despite problems	(7) Continued substance use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to be caused or exacerbated by use	(9) Continued substance use despite knowledge of having a persistent or recurring social, psychological, or physical problem that is caused or exacerbated by substance use	(5) Continued substance use despite clear evidence of overtly harmful physical or psychological consequences
Compulsive use	None	None	(6) A strong desire or sense of compulsion to use substance
Duration criterion	B. No duration criterion separately specified. However, several dependence criteria must occur repeatedly as specified by duration qualifiers associated with criteria (e.g., "often," "persistent," "continued")	B. Some symptoms of the disturbance have persisted for at least 1 month or have occurred repeatedly over a longer period of time	B. No duration criterion separately specified.
Criterion for subtyping dependence	With physiological dependence: Evidence of tolerance or withdrawal (i.e., any of items A(1) or A(2) above are present) Without physiological dependence: No evidence of tolerance or withdrawal (i.e., none of items A(1) or A(2) above are present)	None	None

Table 2. DSM–IV, DSM-III-R, and ICD-10 Diagnostic Criteria for Alcohol and Drug Abuse/Harmful Use of Alcohol

DSM-IV Abuse

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following occurring within a 12-month period:
   (1) recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home
   (2) recurrent substance use in situations in which use is physically hazardous
   (3) recurrent substance-related legal problems
   (4) continued substance use despite having persistent or recurrent social or interpersonal problems caused for exacerbated by the effects of alcohol
B. The symptoms have never met the criteria for substance dependence for the same class of substance.

DSM-III-R Abuse

A. A maladaptive pattern of substance use indicated by at least one of the following:
   (1) Continued use despite knowledge of having a persistent or recurrent social, occupational, psychological, or physical problem that is caused or exacerbated by use
   (2) Substance use in situations in which use is physically hazardous
B. Some symptoms of the disturbance have persisted for at least one month, or have occurred repeatedly over a longer period of time.
C. Never met the criteria for substance dependence for the same class of substance.

ICD-10 Harmful Use

A. A pattern of substance use that is causing damage to health. The damage may be physical or mental. The diagnosis requires that actual damage should have been caused to the mental or physical health of the user.
B. No concurrent diagnosis of the substance dependence syndrome for same class of substance.

and clustering and oversampling for blacks and young adults (aged 18–29 years) and is more fully described elsewhere.[5,6] The household and sample person response rates for the NLAES were 91.9 and 97.4%, respectively.

Diagnostic assessment and comparison

Diagnoses of DSM-IV, DSM-III-R, and ICD-10 alcohol and drug use disorders were derived from the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS), a fully structured psychiatric interview designed to be administered by trained interviewers who were not clinicians.[7] The AUDADIS included an extensive list of symptom questions that operationalized the DSM-IV, DSM-III-R, and ICD-10 criteria for alcohol and drug use disorders. Drug-specific diagnoses of abuse and dependence were derived separately for sedatives (e.g., Valium, Librium), opioids (other than heroin), amphetamines, cocaine (and crack cocaine), cannabis (and THC and hashish), heroin, methadone, and hallucinogens. A prescription drug use disorder measure was also constructed to represent abuse and/or dependence on sedatives, tranquilizers, opiates, and/or amphetamines, whereas an any drug use disorder measure represented abuse and/or dependence across all drug classes measured. Concordance statistics, however, are not shown separately for opiates, heroin, or methadone because of their negligible prevalence in this general population sample.

Comparisons between DSM-IV, DSM-III-R, and ICD-10 definitions of alcohol and drug dependence and alcohol and drug abuse/harmful use appear in Tables 1 and 2, respectively. As shown in Table 1, the diagnostic criteria for dependence across these diagnostic systems are similar. However, subtle but important differences exist between the systems. The ICD-10 aggregates four of the DSM-IV and DSM-III-R dependence criteria into two, combining the two impaired control criteria and the progressive neglect of interests in favor of drinking and the great deal of time spent drinking criteria. Both the DSM-IV and ICD-10 combine the withdrawal and avoidance of withdrawal criteria, whereas these criteria remain separate in the DSM-III-R. The inability to fulfill role obligations and hazardous use criteria appear in both abuse and dependence definitions of the DSM-III-R, are relegated to the abuse category of the DSM-IV, and do not appear in the ICD-10 classification. In contrast, the ICD-10 includes a compulsive use indicator not included in the DSM-IV or DSM-III-R concepts of alcohol or drug dependence. The continued use despite problems criteria of DSM-IV and ICD-10 dependence encompass physical and psychological problems, whereas the corresponding DSM-III-R criterion is more broad, including social problems as well.

Although all three systems require the occurrence of three or more dependence criteria within any 1-year period, differences exist in the nature of the associated duration criteria. In the DSM-III-R, the duration criterion is satisfied if some symptoms of dependence have persisted for at least 1 month or occurred repeatedly over a longer period of time. While the ICD-10 dependence diagnosis requires no duration criterion, the DSM-IV does not specify a separate duration criterion, but does define duration qualifiers associated with specific dependence criteria. These qualifiers specify the repetitiveness with which certain dependence criteria must occur to be counted as positive through the use of terms such as "recurrent," "persistent," "often," and "continued." Although the DSM-IV was not published until 1994, both the criteria sets and associated duration qualifiers were known before the fielding of the NLAES and thus incorporated in their entirety in the AUDADIS.[8] The DSM-IV also uniquely allows

for the subtyping of dependence diagnoses with and without physiological dependence.

In contrast to the dependence definitions, the criteria for ICD-10 harmful use of alcohol and DSM-IV and DSM-III-R alcohol abuse criteria differ vastly (Table 2). According to the ICD-10, a diagnosis of harmful use of alcohol requires a pattern of drinking that has caused physical or psychological harm to the user. To qualify for a diagnosis of DSM-IV or DSM-III-R abuse, the respondent must have reported a maladaptive pattern of use manifested by one of four or one of two diagnostic criteria, respectively, including continued use despite physical or psychological harm to the user. The ICD-10 does not specify duration criteria for its harmful use category, whereas the DSM-IV and DSM-III-R do. Structurally, the DSM-III-R is different from the DSM-IV and ICD-10. That is, the two DSM-III-R abuse criteria are also DSM-III-R dependence criteria, rendering the abuse category residual to the dependence category in this system. In contrast, both the dependence and harmful use/abuse categories of the DSM-IV and ICD-10 are independent of one another and have no overlapping criteria. All three classification systems preclude the diagnosis of harmful use or abuse in the presence of a related concurrent dependence diagnosis, while the DSM-III-R and DSM-IV take this concept one step further by not allowing a diagnosis of abuse if the individual has ever been classified as dependent on a lifetime basis.

Operationally, to qualify for a past year diagnosis of ICD-10 harmful use or DSM-IV or DSM-III-R abuse, at least one harmful use or abuse criterion must have occurred during the past year. In addition, at least one positive abuse symptom had to occur two or more times during the past year to meet the DSM-IV and DSM-III-R duration criteria for abuse. Similarly, positive diagnoses of ICD-10 dependence required the occurrence of three dependence criteria during the past year preceding the interview, whereas DSM-IV and DSM-III-R dependence diagnoses further required that at least three of the positive dependence criteria occurred at least two or more times during the past year.

The DSM-IV, DSM-III-R, and ICD-10 past year diagnoses of dependence were further qualified in two ways. Because the withdrawal criterion of alcohol dependence is defined as a withdrawal state or syndrome in each classification, at least two symptoms of withdrawal needed to occur at least twice over the course of the past year for the withdrawal criterion to be counted as positive. In addition, tolerance need only to have been reported once during the past year to satisfy the DSM-III-R and DSM-IV duration criteria for alcohol dependence. This decision was based on the perspective that the tolerance criterion is less episodic relative to other dependence criteria.

Corresponding prior to the past year diagnoses of DSM-IV, DSM-III-R, and ICD-10 alcohol and drug use disorders were also measured as syndromes or the clustering of the required number of symptoms at the same time, on most days for at least 1 month, or on and off for a few months or longer. Respondents classified with a lifetime alcohol or drug use disorder diagnosis for each classification system encompassed all those who had ever experienced an episode of abuse and/or dependence during the past year and/or prior to the past year.

In a separate test-retest study conducted in the general population, reliability coefficients (kappas) for past year and prior to the past year alcohol and drug use disorders ranged between 0.62 and 0.76 and between 0.60 and 0.95, respectively.[6]

Statistical analysis

Kappa coefficients were used to measure the degree of chance corrected agreement between DSM-IV, DSM-III-R, and ICD-10 diagnoses. The form of kappa used in this study was for fixed (specific) raters, because the central interest was in the specific diagnostic systems as "raters."[9] Perfect agreement or concordance between diagnoses would produce kappa coefficients of 1.0. Kappa values > 0.75 have traditionally been considered excellent, from 0.40 to 0.74 indicates fair to good agreement, and below 0.39 indicates poor agreement. A kappa of zero indicates agreement no better than chance.

RESULTS

The prevalences of DSM-IV, DSM-III-R, and ICD-10 alcohol and drug abuse/harmful use and dependence are shown in Table 3. Diagnoses are separated into three time frames: past year, prior to the past year, and lifetime. For all three time frames, the prevalences of any alcohol use disorder were similar for DSM-III-R and DSM-IV classifications, with ICD-10 rates being in all instances smaller. With regard to alcohol abuse diagnoses, the DSM-III-R produced the largest prevalences and the ICD-10 produced the lowest prevalences, with DSM-IV rates being intermediary. This trend was reversed for prevalences of dependence diagnoses, which were greatest for the DSM-III-R system, followed by DSM-IV and ICD-10 diagnoses. Similar relationships were observed for any drug use disorder and prescription, amphetamine, and cannabis use disorders. There was little variation in the prevalences of abuse/harmful use, dependence, and any drug diagnoses for sedatives,

Table 3. Prevalence of DSM-IV, DSM-III-R, and ICD-10 Diagnoses of Alcohol and Drug Use Disorders by Time Frame

Disorder	Past year			Prior to past year			Lifetime		
	DSM-III-R	DSM-IV	ICD-10	DSM-III-R	DSM-IV	ICD-10	DSM-III-R	DSM-IV	ICD-10
Alcohol abuse and/or dependence	7.60	7.40	4.61	15.83	14.59	10.38	19.18	18.17	12.31
Alcohol abuse only	1.74	3.03	0.46	2.45	3.39	0.57	3.11	4.88	0.69
Alcohol dependence	5.86	4.37	4.15	13.38	11.20	9.81	16.07	13.29	11.62
Any drug abuse and/or dependence	1.56	1.54	0.73	5.77	5.36	4.13	6.39	6.05	4.45
Any drug abuse only	0.84	1.06	0.14	2.31	2.70	1.28	2.56	3.14	1.28
Any drug dependence	0.72	0.48	0.59	3.46	2.66	2.85	3.83	2.91	3.17
Prescription drug abuse and/or dependence	0.25	0.28	0.14	2.55	1.85	1.39	2.64	2.01	1.48
Prescription drug abuse only	0.11	0.17	0.01	0.97	0.89	0.39	0.99	0.98	0.38
Prescription drug dependence	0.14	0.11	0.13	1.56	0.96	1.00	1.65	1.03	1.10
Sedative abuse and/or dependence	0.02	0.02	0.02	.65	0.63	0.46	0.66	0.64	0.46
Sedative abuse only	0.00	0.00	0.00	.21	0.30	0.11	0.22	0.30	0.11
Sedative dependence	0.02	0.02	0.02	.44	0.33	0.35	0.44	0.34	0.35
Tranquilizer abuse and/or dependence	0.09	0.09	0.04	0.62	0.59	0.43	0.66	0.63	0.45
Tranquilizer abuse only	0.04	0.05	0.00	0.22	0.29	0.12	0.24	0.31	0.12
Tranquilizer dependence	0.05	0.04	0.04	0.40	0.30	0.31	0.42	0.32	0.33
Amphetamine abuse and/or dependence	0.11	0.14	0.08	1.51	1.41	1.03	1.55	1.48	1.07
Amphetamine abuse only	0.04	0.09	0.01	0.58	0.72	0.30	0.59	0.76	0.30
Amphetamine dependence	0.07	0.05	0.07	0.93	0.69	0.73	0.96	0.72	0.77
Cannabis abuse and/or dependence	1.21	1.19	0.46	4.36	4.05	2.62	4.89	4.64	2.74
Cannabis abuse only	0.79	0.94	0.13	1.94	2.40	0.83	2.24	2.86	0.77
Cannabis dependence	0.43	0.25	0.33	2.42	1.65	1.79	2.65	1.78	1.97
Cocaine abuse and/or dependence	0.23	0.23	0.16	1.70	1.55	1.40	1.78	1.66	1.47
Cocaine abuse only	0.05	0.09	0.01	0.63	0.61	0.43	0.60	0.64	0.42
Cocaine dependence	0.18	0.14	0.15	1.07	0.94	0.97	1.18	1.02	1.05
Hallucinogen abuse and/or dependence	0.09	0.09	0.04	0.57	0.54	0.39	0.61	0.59	0.42
Hallucinogen abuse only	0.04	0.06	0.00	0.21	0.28	0.10	0.22	0.30	0.10
Hallucinogen dependence	0.05	0.03	0.04	0.36	0.26	0.29	0.39	0.29	0.32

Table 4. Agreement between DSM-IV, DSM-III-R, and ICD-10 Diagnoses of Alcohol and Drug Use Disorders

Disorder	Past year			Prior to past year			Lifetime		
	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10
Alcohol abuse and/or dependence	0.87	0.62	0.67	0.92	0.79	0.76	0.90	0.73	0.73
Alcohol abuse only	0.61	0.06	0.22	0.81	0.22	0.32	0.73	0.13	0.24
Alcohol dependence	0.85	0.80	0.75	0.90	0.90	0.83	0.89	0.72	0.80
Any drug abuse and/or dependence	0.89	0.53	0.60	0.88	0.72	0.80	0.88	0.70	0.77
Any drug abuse only	0.82	0.15	0.23	0.73	0.36	0.61	0.75	0.32	0.52
Any drug dependence	0.80	0.88	0.85	0.87	0.94	0.90	0.86	0.93	0.89
Prescription drug abuse and/or dependence	0.84	0.54	0.65	0.88	0.72	0.80	0.88	0.72	0.79
Prescription drug abuse only	0.73	0.05	0.18	0.71	0.33	0.58	0.71	0.30	0.54
Prescription drug dependence	0.84	0.82	0.87	0.86	0.95	0.88	0.86	0.95	0.88
Sedative abuse and/or dependence	0.97	0.95	0.92	0.89	0.74	0.79	0.89	0.74	0.79
Sedative abuse only	0.83	0.53	0.41	0.72	0.34	0.51	0.73	0.34	0.51
Sedative dependence	1.00	1.00	1.00	0.85	0.92	0.89	0.86	0.93	0.89
Tranquilizer abuse and/or dependence	0.97	0.63	0.66	0.89	0.74	0.79	0.90	0.73	0.78
Tranquilizer abuse only	0.93	0.12	0.14	0.74	0.38	0.56	0.76	0.34	0.50
Tranquilizer dependence	0.94	0.96	0.94	0.86	0.94	0.88	0.87	0.94	0.89
Amphetamine abuse and/or dependence	0.87	0.54	0.73	0.87	0.71	0.78	0.87	0.70	0.79
Amphetamine abuse only	0.53	0.05	0.26	0.71	0.31	0.59	0.70	0.30	0.57
Amphetamine dependence	0.78	0.80	0.87	0.85	0.96	0.88	0.85	0.95	0.88
Cannabis abuse and/or dependence	0.89	0.43	0.51	0.86	0.63	0.71	0.87	0.61	0.68
Cannabis abuse only	0.84	0.14	0.23	0.73	0.26	0.49	0.75	0.23	0.40
Cannabis dependence	0.73	0.85	0.80	0.81	0.92	0.84	0.80	0.91	0.84
Cocaine abuse and/or dependence	0.88	0.77	0.80	0.90	0.82	0.89	0.91	0.82	0.88
Cocaine abuse only	0.65	0.25	0.31	0.76	0.50	0.77	0.76	0.48	0.73
Cocaine dependence	0.87	0.92	0.88	0.93	0.97	0.95	0.93	0.97	0.94
Hallucinogen abuse and/or dependence	0.89	0.55	0.63	0.89	0.70	0.76	0.88	0.69	0.75
Hallucinogen abuse only	0.74	0.01	0.14	0.73	0.28	0.48	0.74	0.23	0.43
Hallucinogen dependence	0.76	0.86	0.80	0.85	0.93	0.86	0.85	0.92	0.86

tranquilizers, cocaine, and hallucinogens. In general, the prevalences of dependence were usually greater than the corresponding abuse or harmful use category across alcohol, each drug class, and time frame.

Agreement between the three systems for each of the three time frames is presented in Table 4. For past year diagnoses of any abuse/harmful use and/or dependence for all alcohol and drug categories shown in Table 4, the agreement between DSM-IV and DSM-III-R was excellent, whereas agreement between DSM-IV and ICD-10 and DSM-III-R and ICD-10 was somewhat lower, but nonetheless fair to excellent. Regardless of time frame, there was excellent agreement between each of the classification systems for de-

Table 5. Agreement between DSM-IV, DSM-III-R, and ICD-10 Diagnoses of Alcohol and Drug Use Disorders by Time Frame, Sex, Ethnicity, and Age

Time frame/sociodemographic characteristic	Harmful use/abuse			Dependence			Combined		
	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10
Past year									
Male	0.63	0.06	0.19	0.84	0.81	0.75	0.87	0.61	0.66
Female	0.58	0.04	0.29	0.85	0.80	0.75	0.86	0.63	0.69
Black	0.57	0.10	0.27	0.89	0.83	0.77	0.89	0.70	0.73
Nonblack	0.62	0.05	0.22	0.84	0.80	0.75	0.86	0.61	0.67
< 30 years	0.47	0.06	0.38	0.84	0.82	0.79	0.84	0.66	0.74
30 + years	0.37	0.11	0.31	0.83	0.88	0.78	0.84	0.72	0.75
Prior to past year									
Male	0.82	0.24	0.33	0.90	0.90	0.83	0.92	0.79	0.76
Female	0.78	0.18	0.29	0.90	0.89	0.81	0.91	0.77	0.75
Black	0.85	0.43	0.57	0.93	0.90	0.85	0.93	0.83	0.81
Nonblack	0.80	0.21	0.30	0.90	0.90	0.82	0.91	0.78	0.76
< 30 years	0.75	0.29	0.48	0.91	0.91	0.86	0.92	0.82	0.82
30 + years	0.82	0.30	0.52	0.90	0.91	0.81	0.92	0.80	0.76
Lifetime									
Male	0.73	0.14	0.23	0.88	0.71	0.79	0.90	0.72	0.71
Female	0.71	0.12	0.25	0.89	0.74	0.79	0.90	0.74	0.73
Black	0.71	0.23	0.39	0.92	0.76	0.81	0.92	0.77	0.77
Nonblack	0.73	0.13	0.23	0.89	0.72	0.80	0.90	0.73	0.72
< 30 years	0.65	0.18	0.41	0.90	0.76	0.84	0.90	0.78	0.79
30 + years	0.74	0.22	0.42	0.90	0.76	0.80	0.91	0.78	0.74

Table 6. Agreement between DSM-IV, DSM-III-R, and ICD-10 Diagnoses of Any Drug Use Disorder by Time Frame, Sex, Ethnicity, and Age

Time frame/sociodemographic characteristic	Harmful use/abuse			Dependence			Combined		
	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10
Past year									
Male	0.84	0.17	0.21	0.79	0.90	0.84	0.90	0.51	0.56
Female	0.74	0.12	0.29	0.81	0.84	0.86	0.86	0.59	0.69
Black	0.82	0.41	0.38	0.88	0.93	0.86	0.91	0.72	0.72
Nonblack	0.81	0.13	0.22	0.79	0.87	0.84	0.88	0.51	0.59
< 30 years	0.75	0.22	0.29	0.79	0.86	0.86	0.87	0.64	0.70
30 + years	0.87	0.46	0.32	0.94	0.79	0.73	0.92	0.61	0.61
Prior to past year									
Male	0.75	0.36	0.59	0.86	0.93	0.89	0.88	0.70	0.78
Female	0.69	0.37	0.64	0.88	0.94	0.89	0.87	0.74	0.82
Black	0.68	0.45	0.73	0.89	0.93	0.91	0.87	0.77	0.86
Nonblack	0.74	0.36	0.60	0.86	0.94	0.89	0.88	0.71	0.80
< 30 years	0.70	0.47	0.69	0.90	0.96	0.92	0.87	0.79	0.86
30 + years	0.78	0.43	0.65	0.83	0.93	0.86	0.89	0.71	0.77
Lifetime									
Male	0.78	0.31	0.50	0.86	0.93	0.89	0.89	0.68	0.75
Female	0.70	0.34	0.57	0.87	0.93	0.89	0.88	0.73	0.80
Black	0.71	0.40	0.64	0.90	0.93	0.91	0.88	0.76	0.84
Nonblack	0.76	0.31	0.51	0.86	0.93	0.88	0.88	0.70	0.76
< 30 years	0.71	0.41	0.62	0.90	0.94	0.91	0.89	0.78	0.84
30 + years	0.82	0.44	0.57	0.83	0.92	0.85	0.91	0.71	0.74

pendence diagnoses. Similarly, DSM-III-R and DSM-IV abuse diagnoses were shown to be highly concordant for alcohol and each drug, regardless of time frame. For all past year diagnoses, agreement between DSM-III-R and ICD-10 and DSM-IV and ICD-10 abuse/harmful use categories was poor with the exception of sedative abuse that showed fair agreement (– 0.53) between these systems. Although this result remained consistent for comparisons involving prior to the past year and lifetime DSM-IV versus ICD-10 diagnoses, comparisons between DSM-III-R and ICD-10 abuse and harmful use showed fair agreement for prior to the past year and lifetime diagnoses.

Concordance for alcohol dependence and any alcohol use disorder diagnoses among each of the three classification systems was excellent for past year, prior to the past year, and lifetime diagnoses for both genders, both ethnic groups, and for older and younger respondents (Table 5). Any differences between subgroups were negligible. Agreement between DSM-IV and DSM-III-R alcohol abuse was good to excellent, whereas agreement between DSM-III-R alcohol abuse and ICD-10 harmful use of alcohol was consistently poor across each time and each socioeconomic subgroup of the population. Although agreement remained poor between past year diagnoses of DSM-III-R alcohol abuse and ICD-10 harmful use of alcohol, kappa values observed for the corresponding prior to the past year and lifetime comparisons were slightly higher (kappa = 0.23–0.57), indicating poor to fair agreement. Identical patterns of agreement were observed for diagnoses of any drug use disorder (Table 6) and for each specific drug diagnosis of interest to this study as illustrated for cannabis use disorders in Table 7.

DISCUSSION

The prevalences of any alcohol or drug use disorder and dependence in DSM-IV were consistently lower than those in the DSM-III-R, but higher than those similarly classified in the ICD-10. These results are consistent with the findings of the only three studies examining similar nosological comparisons in smaller samples.[10-12] It appears that the observed prevalences are the result of the inclusiveness of dependence diagnoses across systems. The DSM-III-R dependence classification is most inclusive, requiring three of nine positive criteria for a diagnosis; the ICD-10 classification is most exclusive, requiring three of six positive criteria; and the DSM-IV lies somewhere in between, requiring three of seven positive criteria for a diagnosis.

The prevalences of alcohol and drug abuse diagnoses were consistently greater for the DSM-IV than the DSM-III-R, with ICD-10 yielding the lowest prevalences of harmful use diagnoses. These differences appear to relate to the divergence in the criteria themselves. Because the DSM-III-R abuse criteria are also DSM-III-R dependence criteria and the DSM-IV abuse and dependence categories do not share criteria, it is not surprising that the prevalences of DSM-IV alcohol and drug abuse exceeds the corresponding prevalences of

Table 7. Agreement between DSM-IV, DSM-III-R, and ICD-10 Diagnoses of Cannabis Use Disorders by Time Frame, Sex, Ethnicity, and Age

Time frame/sociodemographic characteristic	Harmful use/abuse			Dependence			Combined		
	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10	DSM-IV vs. DSM-III-R	DSM-IV vs. ICD-10	DSM-III-R vs. ICD-10
Past year									
Male	0.87	0.14	0.20	0.75	0.90	0.80	0.90	0.41	0.47
Female	0.74	0.13	0.33	0.68	0.75	0.81	0.83	0.46	0.61
Black	0.85	0.31	0.33	0.78	0.89	0.76	0.90	0.55	0.57
Nonblack	0.84	0.13	0.22	0.72	0.85	0.81	0.89	0.42	0.50
< 30 years	0.80	0.24	0.31	0.75	0.86	0.85	0.87	0.56	0.63
30 + years	1.00	0.15	0.15	1.00	0.89	0.82	1.00	0.54	0.61
Prior to past year									
Male	0.74	0.25	0.46	0.80	0.91	0.89	0.86	0.62	0.70
Female	0.70	0.28	0.54	0.82	0.92	0.89	0.85	0.64	0.73
Black	0.61	0.31	0.58	0.80	0.89	0.91	0.82	0.67	0.77
Nonblack	0.74	0.26	0.48	0.81	0.92	0.89	0.86	0.63	0.71
< 30 years	0.64	0.29	0.55	0.85	0.94	0.89	0.85	0.72	0.79
30 + years	0.74	0.34	0.56	0.67	0.92	0.70	0.85	0.59	0.64
Lifetime									
Male	0.77	0.21	0.37	0.80	0.92	0.84	0.87	0.59	0.66
Female	0.72	0.26	0.47	0.80	0.90	0.83	0.85	0.63	0.71
Black	0.67	0.28	0.49	0.80	0.89	0.82	0.84	0.64	0.73
Nonblack	0.76	0.23	0.40	0.80	0.91	0.84	0.87	0.60	0.68
< 30 years	0.67	0.24	0.46	0.84	0.93	0.89	0.86	0.69	0.77
30 + years	0.77	0.34	0.52	0.68	0.90	0.70	0.87	0.56	0.61

DSM-III-R abuse. Whenever a residual relationship between diagnostic categories pertains, as in the DSM-III-R, the boundaries of dependence will be broadened at the expense of narrowing the abuse category.

It is more than interesting that the prevalences of abuse and harmful use were consistently lower than their dependence counterparts in each of three systems. This finding stands in marked contrast to current clinical thinking in which abuse and harmful use in all three diagnostic approaches are viewed as less severe conditions, whereas dependence is viewed as the more severe and pervasive syndrome. However, if abuse is a milder, less severe condition compared with dependence, then it should be a more prevalent condition than dependence in the general population. This finding has never been the case in any previous epidemiological prevalence survey. Moreover, the negligible prevalences associated with ICD-10 harmful use would be more indicative of a severe or end-stage alcohol use disorder. Recall that ICD-10 harmful use is characterized as psychological or physical harm to the user, a condition that could easily be equated with an end-stage disease process associated with excessive alcohol or drug use.

With regard to abuse and harmful use, the results of this study are consistent with the recent studies of Hasin et al.,[10,11] but at variance with the findings of Schuckit and his colleagues,[12] who found the prevalences of ICD-10 harmful use exceeded those of DSM-III-R abuse for alcohol as well drugs. The discrepancies between these research findings are most probably due to the differences in the study's samples. Hasin et al. used a general population sample (n = 962), and Schuckit and his colleagues used probands (n = 259), relatives of probands (n = 1373), and a small outpatient-community (n = 290) sample drawn from the larger NIAAA Collaborative Study on the Genetics of Alcoholism (COGA).[13] However, the entire latter sample mirrored clinical samples of alcohol and drug-dependent persons with regard to the prevalences of alcohol use (83%), amphetamine use (14%), cocaine use (20%), cannabis use (31%), hallucinogen use (8%), and sedative use (9%). Not only was the Schuckit et al. sample not representative of all persons with alcohol and drug use disorders, but most persons in that sample were likely to report most, if not all, abuse and dependence symptoms. That is, this more-affected population was more likely to have severe alcohol-related conditions, accounting for the greater prevalence of ICD-10 harmful use diagnoses compared with DSM-III-R abuse diagnoses. The prevalence of DSM-III-R abuse diagnoses would be expected to be lower in a treated sample because the more severely affected individuals are likely to report enough symptoms to achieve a dependence diagnosis, hierarchically precluding a diagnosis of abuse.

Agreement among the three systems on alcohol and drug dependence diagnoses was good to excellent for past year, prior to the past year, and lifetime diagnoses, for both genders, each ethnic group, and younger and older respondents. This finding indicated that similarity in the content of dependence definitions across systems was important in producing high agreement, despite differences in the number of dependence criteria and the duration criteria. Corresponding any alcohol or drug use disorder diagnoses were also good to excellent, but somewhat lower than those for dependence diagnoses primarily because this subclassification additionally included abuse and harmful use diagnoses in contrast to those with no diagnosis and dependence. Unlike dependence, concordance between DSM-IV and ICD-10 and DSM-III-R and ICD-10 alcohol and drug abuse and harmful use categories was extremely poor. The only exception to this finding was that DSM-III-R and ICD-10 diagnoses achieved fair to good agreement across sociodemographic subgroups of the population for prior to the past year and lifetime diagnoses.

Interestingly, kappa values associated with DSM-IV and DSM-III-R abuse comparisons indicated good to excellent agreement. This result was consistent with the Hasin et al. studies for alcohol but at variance with the findings of Schuckit and his colleagues who found no better than chance agreement between the DSM-IV and DSM-III-R abuse categories across alcohol and drug diagnoses. The good to excellent agreement between the DSM-IV and DSM-III-R abuse classifications found in the Hasin et al. study and the present study is most probably due to the similarity in content of the two abuse categories, despite the fact that their structural relationship to dependence differs (i.e., the DSM-III-R abuse category is residual to its dependence category, whereas the DSM-IV abuse category is independent of its dependence category). Both abuse categories share the hazardous use criterion and both share substance-related psychological, interpersonal, social, and occupational consequences.

The chance level of agreement between DSM-IV and DSM-III-R abuse categories found by Schuckit and his colleagues may be the result of employing a diagnostic interview (i.e., the Semi-structured Assessment for the Genetics of Alcoholism: SSAGA),[14] which was developed specifically to select probands and assess their relatives using DSM-III-R criteria for alcohol and drug use disorders. That is, the SSAGA was not developed to directly measure DSM-IV alcohol and

drug use disorder criteria and such measures were developed by extracting items from the SSAGA that approximated the DSM-IV classification, but did not replicate the criteria completely. In contrast, both the Hasin et al. studies and the present study used different versions of the same interview, both of which were explicitly designed to measure DSM-IV alcohol and drug use disorders.

The results of this study have several implications regarding international efforts designed to integrate these classification systems. Specifically, efforts to find a common ground with regard to the definition of alcohol and drug dependence was achieved. However, the results document differences in the meaning of abuse and harmful use between the systems. The ICD-10 criteria for harmful use focus only on psychological and physical harm to the user, while the DSM-IV and DSM-III-R criteria for abuse include psychological, social, interpersonal and legal consequences of use. The inclusion of such psychosocial criteria in the DSM-IV and DSM-III-R abuse category was primarily responsible for their larger prevalences compared with the ICD-10 harmful use category.

The good to excellent agreement found among DSM-IV, DSM-III-R, and ICD-10 dependence also suggested that comparisons across studies using any of the three criteria sets may be made confidently for past year, prior to the past year, and lifetime diagnoses and within subgroups of the population defined in terms of important sociodemographic characteristics. The absence of between-system agreement for ICD-10 harmful use and DSM-IV and DSM-III-R abuse indicates that comparisons across studies for this diagnostic category would be contraindicated. The discrepancies between harmful use and abuse diagnoses did lower levels of agreement for the three category combined harmful use/abuse and dependence diagnoses. However, the concordance between the systems for these diagnoses remains good to the lower range of excellent, indicating that cross-study comparisons are possible, but should be made cautiously.

This study provided empirical evidence on the degree to which research using DSM-IV, DSM-III-R, and ICD-10 classification systems for alcohol and drug use disorders can be integrated to advance knowledge in the alcohol and drug fields. The results also confirm the success of the international effort to integrate the two new major classification systems with regard to diagnoses of dependence. In contrast, the ICD-10 harmful use category with its accompanying negligible prevalences and less than chance agreement with the DSM-IV and DSM-III-R abuse categories appears to be in need of both focused theoretical and empirical examination.

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The Validity of DSM–IV Alcohol Abuse: Drunk Drivers Versus All Others

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Objective: *Prior research in a community sample indicated that almost half the individuals receiving a diagnosis of DSM-IV alcohol abuse did so on the basis of only one symptom, driving after drinking too much. While this is certainly unwise behavior, it may not be a psychiatric disorder. Therefore, we investigated the differential validity of this subgroup of abuse cases by testing the association of a set of external validating criterion variables with three groups: those who met criteria for abuse just for drinking-driving, those who met criteria by other means, and those with no alcohol diagnosis. Present status of past cases of abuse was also investigated.* Method: *Subjects were 22,204 U.S. household residents (a subset of a national probability sample) interviewed in 1992 with the Alcohol Use Disorders and Associated Disabilities Interview Schedule. The generalized logit model was the principal means of analysis.* Results: *Subjects who met criteria for DSM-IV alcohol abuse just for drinking-driving differed from subjects with no diagnosis on about half the variables tested, while those who met criteria for abuse in other ways differed from subjects with no diagnosis on all variables tested. The two abuse groups differed from each other on some but not all variables. Past cases of abuse for drinking-driving and past cases of other abuse were equally likely to have remitted in the last 12 months, and slightly less likely to meet criteria for current dependence.* Conclusions: *Further conceptual and empirical work is needed to resolve the difficulties with the DSM-IV alcohol abuse category.*

Alcohol abuse was first formally differentiated from alcohol dependence with the publication of DSM-III in 1980 (American Psychiatric Association, 1980). At the time that DSM-III was published, the grounds for the abuse/dependence distinction were not clearly articulated. Other problems with the criteria for these two disorders were identified as well (Rounsaville, 1987; Rounsaville et al., 1986). Given these difficulties, the criteria for alcohol abuse and dependence were modified considerably in DSM-III-R (American Psychiatric Association, 1987).

The validity of the concept of dependence (Edwards and Gross, 1976) that serves as the basis for DSM-III-R, DSM-IV (American Psychiatric Association, 1994) and ICD-10 (World Health Organization, 1992) has been well-established (e.g., Edwards, 1986; Kosten et al., 1987; Rounsaville et al., 1987; Feingold and Rounsaville, 1994; Carroll et al., 1994; Hasin et al., 1997b,c,d). According to this conceptualization of alcohol use disorders, alcohol dependence constitutes one axis or dimension, while other types of alcohol problems lie on different dimensions (Edwards, 1986). This concept leaves room for the possibility of a second axis in the form of an abuse diagnosis, but does not specify its nature. In ICD-10 the abuse category consists entirely of "hazardous" use, including impaired judgment and dysfunctional behavior. In DSM-IV, four criteria are available to be used in making a diagnosis of alcohol abuse: hazardous use (e.g., driving after drinking), failure to fulfill major role obligations, continued use despite social or interpersonal problems, and legal problems. Unlike most other mental disorders which consist of a syndrome, DSM-IV alcohol abuse can be diagnosed when only one of the four criteria has been met. Thus, instances within a 1-year period of driving after drinking too much would be considered sufficient to receive a DSM-IV diagnosis of alcohol abuse, and hence be considered a mental disorder. In both ICD-10 and DSM-IV, a diagnosis of alcohol dependence precludes a diagnosis of abuse.

Using DSM-IV criteria, comparisons of subjects with dependence to subjects with abuse in the general population have shown marked cross-sectional differences (Hasin et al., 1997c). Using older as well as more up-to-date data and criteria, abuse and dependence were shown to have clear differences in longitudinal course (Ojesjo, 1983; Hasin et al., 1990, 1997d), with no evidence that abuse constituted

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merely a prodromal condition to dependence. Although factor analytic studies of abuse and dependence items in clinical samples appear to suggest that dependence and abuse form a single factor (Hasin et al., 1988; Feingold and Rounsaville, 1995), DSM-IV abuse is extremely rare in treatment settings (Hasin et al., 1996b; Hasin et al., 1997a) and, thus, these may not be the best settings in which to study DSM-IV abuse. Factor analytic studies using large nationally representative samples have indicated the existence of two factors rather than one for alcohol use disorders, with factors that generally correspond to dependence and abuse (Muthen et al., 1993a,b). All of this material supports the importance of not assuming that abuse is simply a mild, prodromal form of dependence and of keeping abuse cases distinct from those classified as dependent. However, when subjects in the general population with a diagnosis of DSM-IV alcohol abuse were compared to subjects with no alcohol diagnosis, the grounds for making a differentiation (and thus a diagnosis) were weaker (Hasin and Paykin, in press; Hasin et al., 1997c), using both community and national data. Nosological comparisons between diagnostic systems (a method of validation through multimethod convergence on an underlying concept) have shown much lower agreement for abuse/hazardous use than for dependence (Grant, 1996; Hasin et al., 1996b, 1997b). The reliability of DSM-IV abuse is also generally much lower than that of dependence (Hasin et al., 1996a, 1997a; Chatterji et al., 1997; Easton et al., 1997). Thus, earlier questions about the validity and utility of the abuse category (Helzer, 1994) remain unanswered. The problems with the diagnostic category of DSM-IV abuse require more knowledge and the testing of some potential solutions.

A study of DSM-IV alcohol abuse in a community sample (Hasin and Paykin, 1999) showed that a large proportion of the cases of DSM-IV alcohol abuse received the diagnosis only for one symptom, hazardous use. Furthermore, for current diagnoses of abuse, this hazardous use consisted mainly of driving after drinking too much (about 47% of all cases meeting criteria for DSM-IV alcohol abuse). Subjects meeting criteria for current DSM-IV alcohol abuse on this basis drove after drinking approximately four times within a 1-year period. The negative consequences of driving after drinking are obvious and do not require elaboration here. We hypothesized, however, that this behavior might not be sufficient grounds for making the diagnosis of a mental disorder. If this particular means of assigning the DSM-IV alcohol abuse diagnosis proved to be invalid, then perhaps its modification (e.g., through requirements for other or additional symptoms) would improve the validity of the alcohol abuse category.

To investigate this issue, we undertook a study of cases of DSM-IV alcohol abuse where criteria were met *only* for driving after drinking too much. We used data from a U.S. national survey because this offered a larger sample than the community sample in which we did prior work. We compared these cases of DSM-IV alcohol abuse to subjects with DSM-IV alcohol abuse made on other grounds, and to subjects with no alcohol diagnosis. The groups were compared on aspects of drinking history and on indicators of a disorder specifically connected to alcohol use. These included participation in alcoholism treatment, a subjective sense that alcoholism treatment was needed, input from someone else that alcoholism treatment was needed, age at onset of drinking and a family history of alcoholism. We also compared the groups on depressed mood and drug use, since these are both associated with alcohol use disorders in clinical as well as general population samples (Hasin and Nunes, 1998). In addition, we investigated the current statuses of past cases of DSM-IV alcohol abuse diagnosed just for driving after drinking and compared these to the current statuses of past cases of DSM-IV alcohol abuse diagnosed for other reasons.

In this article, all comparisons were based on the following reasoning: If the DSM-IV alcohol abuse cases diagnosed just for driving after drinking were similar to subjects with no diagnosis and different from the other DSM-IV alcohol abuse cases, the validity of the "drinker-driver" alcohol abuse group would be in question. Conversely, if the "drinker-driver" cases of DSM-IV alcohol abuse were similar to the other cases of DSM-IV alcohol abuse but differed from the no-diagnosis group, the validity of the diagnosis when made for driving after drinking would be supported.

METHOD

Sample and data

This study was based on data from the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES; Grant et al., 1994a). The data were derived from a national probability sample sponsored by the National Institute on Alcohol Abuse and Alcoholism. Field work for the NLAES was conducted by the U.S. Bureau of the Census. Direct face-to-face interviews were administered to 42,862 respondents, 18 years old or older, residing in the noninstitutionalized population of the contiguous United States, including the

District of Columbia. The household response rate (the response rate of the households selected) for this representative sample of the U.S. population was 91.9%, and the sample person response rate (the response rate for persons sampled within the households) was 97.4%. More detail on the complex multistage design of the NLAES full sample is provided elsewhere (Grant et al., 1994b).

SUBJECTS

From the full NLAES sample of 42,862, a total of 1,947 subjects met criteria for lifetime DSM-IV alcohol abuse (current, prior to the past year, or both). Of these, 867 were current (last 12 months) cases of DSM-IV alcohol abuse. Of all subjects with a lifetime DSM-IV alcohol abuse diagnosis, 848 (42.0% weighted) met criteria only for driving after drinking too much. Of the 867 current DSM-IV alcohol abuse cases, 429 (47.3% weighted) met criteria solely for driving after drinking too much. The group of current alcohol abusers excludes all subjects who met criteria for past alcohol dependence. According to DSM-IV, these subjects are in partial remission from dependence.

For the analyses, we constructed three groups of subjects for current diagnoses and three analogous groups for lifetime diagnoses. Group 1 in each timeframe consisted of subjects who received the DSM-IV alcohol abuse diagnosis *only* for driving after drinking. Group 2 in each timeframe consisted of all subjects who received the DSM-IV alcohol abuse lifetime diagnosis on some basis other than the sole criterion of driving after drinking too much. This basis of diagnosis could be one or more other symptoms of DSM-IV alcohol abuse, or other symptoms in addition to driving

Table 1. Characteristics (weighted proportions and means) of subjects with lifetime DSM-IV alcohol abuse due to drunk driving only (Group 1); lifetime DSM-IV alcohol abuse due to additional/other reasons (Group 2); and drinkers with no lifetime DSM-IV diagnosis (Group 3)

Characteristic	Group 1 (drinker-drivers) (n = 848)	Group 2 (other abuse cases) (n = 1,099)	Group 3 (no alcohol diagnosis) (n = 20,257)	Total (N = 22,204)	Bivariate test of group difference[a,b] (log-likelihood chi square)	Significant post hoc group comparisons[c,d] 1 vs 2	1 vs 3	2 vs 3
Sex[e]					140.53, 2 df‡	NS	.000	.000
Male	.66	.70	.53	.54				
Female	.34	.30	.47	.46				
Age (yrs)[e]					213.63, 6 df‡	.000	.000	.000
18-29	.28	.39	.22	.23				
30-39	.31	.28	.24	.25				
40-49	.23	.17	.20	.20				
50+	.19	.15	.34	.32				
Race[e]					25.79, 2 df‡	.014	.000	NS
White	.93	.89	.86	.87				
Nonwhite	.07	.11	.14	.13				
Marital[e]					53.48, 2 df‡	.001	NS	.000
Unmarried	.39	.47	.35	.36				
Ever used drugs[e]	.32	.42	.14	.16	462.26, 2 df‡	.000	.000	.000
Family history[e]	.38	.44	.31	.32	66.54, 2 df‡	.014	.001	.000
Alcohol treatment ever[e]	.04	.13	.02	.03	264.20, 2 df‡	.000	.014	.000
Was there ever a time you thought you should seek help?[e]	.01	.05	.01	.01	111.46, 2 df‡	.000	NS	.000
Was there ever a time someone else thought you should seek help?[e]	.03	.15	.02	.03	298.87, 2 df‡	.000	NS	.000
Education (yrs)[e]					21.39, 4 df‡	NS	.000	NS
0-11	.08	.13	.15	.15				
12	.30	.29	.31	.31				
13+	.62	.58	.54	.54				
Age first started drinking[f]	17.81 ± 0.13	17.10 ± 0.11	19.67 ± 0.04	19.46 ± 0.04	149.63‡[g]	.000	.000	.000

[a]Test of differences among the three groups are based on the test for no interaction in a log-linear model for proportions, or the Wald F statistic for means.
[b]‡$p < .005$.
[c]Test of pairwise differences in proportions are based on the test for no interaction in a log-linear model; test of differences in means based on t statistic.
[d]Using Bonferroni-corrected α to adjust for multiple tests required for three pairwise comparisons; a p value of less than .05/3 = 0.0167 is required for significance. Otherwise, a pairwise comparison is considered nonsignificant (NS).
[e]Proportion.
[f]Mean (±SE) years.
[g]Wald F statistic.

after drinking. Group 3 in each timeframe consisted of drinkers who received no diagnosis of a DSM-IV alcohol use disorder. On a lifetime basis, drinkers were defined in the interview as those who reported that they had ever had at least 12 drinks of alcohol within any 1-year period. On a current basis, drinkers were defined in the interview as those who reported that they had had at least 12 drinks of alcohol over the year prior to the day of the interview. Tables 1 and 2 show the char-

Table 2. Characteristics (weighted proportions and means) of subjects with current DSM-IV alcohol abuse due to drunk driving only (Group 1); DSM-IV alcohol abuse due to additional/other reasons (Group 2); and current drinkers with no DSM-IV diagnosis ever (Group 3)

Characteristic	Group 1 (drinker-drivers) (n = 397)	Group 2 (other abuse cases) (n = 470)	Group 3 (no alcohol diagnosis) (n = 13,054)	Total (N = 13,921)	Bivariate test of group difference[a,b] (log-likelihood) chi square)	Significant post hoc group comparisons[c,d] 1 vs 2	1 vs 3	2 vs 3
Sex[e]					69.37, 2 df‡	NS	.000	.000
Male	.73	.72	.56	.58				
Female	.27	.28	.44	.43				
Age (yrs)[e]					221.28, 6 df‡	.000	.000	.000
18-29	.40	.60	.25	.26				
30-39	.29	.22	.25	.25				
40-49	.20	.10	.21	.20				
50+	.11	.08	.30	.28				
Race[e]					8.26, 2 df*	NS	.005	NS
White	.93	.86	.87	.87				
Nonwhite	.07	.14	.13	.13				
Marital[e]					123.90, 2 df‡	.000	.000	.000
Unmarried	.51	.67	.37	.39				
Current drug user[e]	.14	.25	.04	.05	260.01, 2 df‡	.001	.000	.000
Unemployed[e]	.07	.06	.04	.04	6.63, 2 df*	NS	NS	NS
Drink during daytime (besides 6 pm-3 am daily and 3 am-6 am Friday/Saturday)[e]	.32	.32	.24	.24	19.13, 2 df‡	NS	.003	.001
Drink on weekdays[e]	.37	.40	.27	.28	37.89, 2 df‡	NS	.001	.000
Current alcohol treatment[e]	.02	.05	.01	.01	58.20, 2 df‡	NS	NS	.000
Depressed mood[e]	.10	.18	.08	.09	40.37, 2 df‡	.006	NS	.000
Education[e]					2.99, 4 df	NS	NS	NS
0-11	.08	.11	.12	.12				
12	.31	.30	.29	.29				
13+	.61	.59	.59	.59				
Frequency of 5+ drinks[e]					444.82, 8 df‡	NS	.000	.000
Once/month or less	.59	.55	.88	.86				
2-3 days/month	.16	.16	.05	.06				
1-2 days/week	.16	.22	.04	.05				
3-4 days/week	.03	.03	.01	.01				
Every day/ nearly every day	.06	.04	.01	.01				
Frequency of drinking to intoxication[e]					371.55, 6 df‡	.000	.000	.000
Once/month or less	.90	.76	.98	.97				
2-3 days/month	.06	.13	.01	.02				
1-2 days/week	.04	.11	.01	.01				
3-4 days/week or more	.00	.01	.00	.00				
Blackouts[e]	.14	.35	.03	.04	494.78, 2 df‡	.000	.000	.000
Average daily ethanol intake (in ounces)[f]	0.95 ± 0.08	1.10 ± 0.09	0.44 ± 0.01	0.48 ± 0.01	50.85‡[g]	NS	.000	.000

[a]Test of differences among the three groups are based on the test for no interaction in a log-linear model for proportions, or the Wald F statistic for means.
[b]*$p < .05$; ‡$p < .005$.
[c]Test of pairwise differences in proportions are based on the test for no interaction in a log-linear model; test of differences in means based on t statistic.
[d]Using Bonferroni-corrected α to adjust for multiple tests required for three pairwise comparisons; a p value of less than .05/3 = 0.0167 is required for significance.
[e]Proportion.
[f]Mean (±SE).
[g]Wald F statistic.

acteristics of the three groups used in the lifetime and current analysis, respectively.

Measures

The diagnosis of DSM-IV alcohol abuse was derived from the Alcohol Use Disorders and Associated Disabilities Interview Schedule (AUDADIS; Grant et al., 1995). The AUDADIS is a fully structured psychiatric interview designed to be administered by systematically trained interviewers who are not clinicians. The interview contains sections with detailed items on substance use disorders, including DSM-IV alcohol abuse. The AUDADIS also contains sections covering areas potentially related to alcohol use disorders. These sections were the source of the other variables used in the analyses described below. In addition to demographic characteristics, these variables included numerous drinking variables and variables potentially related to drinking: alcohol consumption, frequency of drinking five or more drinks per occasion during the past year ("5 + occasions"), frequency of drinking to intoxication during the past year (defined for the respondents as times when their speech was slurred, they felt unsteady on their feet, or they had blurred vision), blackouts (times when they could not remember some of the things they did while drinking), age at onset of drinking, utilization of treatment or a self-help group for alcohol-related problems (any one from an inclusive list of different types of treatment facilities), a subjective feeling that treatment was needed even if not sought, input from someone else that treatment was needed even if not sought, depressed mood, and a history of drug use (which summed a series of questions that covered drugs by specific categories). Most of these variables were subjected to a test-retest study in a general population sample of 473 subjects (Grant et al., 1995), and shown to have good to excellent test-retest reliability.

In this study, we used a measure of current ethanol consumption, usual daily ethanol consumption, that was similar to measures developed by specialists at the National Institute on Alcohol Abuse and Alcoholism (Dawson, 1998). Usual daily ethanol consumption, a volume measure, combines quantity and frequency of drinking, since quantity or frequency alone do not provide a good indication of usual drinking behavior. Usual daily ethanol consumption was calculated in several steps. First, volume of ethanol was computed separately for beer, wine and distilled spirits, based on items in the alcohol consumption section of the AUDADIS. For each of the three beverage types, volume (ounces of ethanol) was calculated as follows: the number of days per year when subjects drank their usual quantity of the beverage was multiplied by the number of ounces consumed per occasion, which was multiplied by a beverage-specific ethanol conversion factor. The ethanol conversion factor for each beverage converted the volume of alcohol into the volume of ethanol. The three conversion factors were: beer = .045; wine =.121; and distilled spirits = .409. The three beverage-specific volume indicators were then summed, and the total was divided by 365.

The family history of alcoholism variable combined AUDADIS information from a series of questions about relatives and whether they had ever had problems with alcohol. Only relatives over the age of 10 were covered, to ensure that they had entered the age at risk. Each category of relative was queried separately. Subjects were asked if the relatives had been alcoholics or problem drinkers; or had physical or emotional problems because of drinking; or problems with a spouse, family or friends, at work, with the police; or drunk driving; or had spent a lot of time drinking or being hung over. The variable was positive if subjects reported that their biological mother, father, or a sister or brother had a history of such alcohol-related problems. The family history items were included in the test-retest study cited above (Grant et al., 1995) and were shown to be very high for most relatives, including father, mother, brother and sister (k = .87, 1.00, .90 and .73, respectively; Grant and Dawson, 1997).

Analysis

Our primary aim was to compare the characteristics and experiences of Groups 1 and 2 to each other and to Group 3. We wished to do this within the two timeframes of interest, current (last 12 months) and lifetime. The principal method of analysis was multivariate because it is important to control for other factors when investigating the relationship between a trait and group membership, and it is also important not to place undue importance on scattered results from an excess of bivariate statistical tests. If we had only two groups (for example, if we were comparing any subjects with abuse to those with no diagnosis), we would have used logistic regression with a two-level dependent variable (abuse, yes or no) and the traits as predictor variables. However, with three groups (drivers, others and no diagnosis), we needed a dependent variable with three levels. Therefore, we used an analogous model that is now in common use, the generalized logit model, which simultaneously compares all groups. The three-level dependent variable for the logit model was defined by membership in one of the three groups. The characteristics tested (treatment sta-

tus, family history, etc.) were included in the models as predictor variables. Demographic characteristics (age, sex and race) were used as control variables. Odds ratios (ORs) and 95% confidence intervals (CIs) were obtained from the regression parameters of the generalized logit models. While it would have been possible to show many statistical terms from the generalized logit models, we included only the odds ratios and 95% confidence intervals for simplicity. Because of the complex survey design of the NLAES, variance estimation procedures assuming a simple random sample were not appropriate. To take into account the sample design of the NLAES, SUDAAN (Shah et al., 1996) was used. SUDAAN uses Taylor series linearization to adjust for sample design characteristics (e.g., probability of selection).

In working with the multivariate models for both lifetime and current cases of abuse, we found that age did not meet the assumption of linearity in the logit required by the generalized logistic regression model. Although age was not a main focus of the investigation, we did not wish to violate the linearity assumptions of the model. Therefore, in developing both the lifetime and current models, each of which has a three-category outcome, we performed three separate pairwise logistic models, using the no-diagnosis group as the baseline (drunk drivers versus no diagnosis, abuse for additional/other reasons versus no diagnosis, and drunk drivers versus abuse for additional/other reasons), to determine the relationship between the logit and the continuous variable age. Age was broken into ten equal-size groups and, for each group, the logit of the group mean was plotted against the group midpoint. In both the lifetime and current models, the resulting curve showed the relationship to be a quadratic one. This finding, in addition to a graphical investigation, indicated that the final models should be fit using both a linear term, age, and a quadratic term, age^2 (see Hosmer and Lemeshow, p. 90). This was done.

Methods to test the goodness-of-fit of generalized logit models have not yet been developed. Thus, to determine whether the fit of the models (current and lifetime) was adequate, the following method was used: Hosmer and Lemeshow goodness-of-fit tests were performed on each of the three pairwise logistic regression models that could be formed from the three-category outcome variable. Ordinarily, for each pairwise logistic regression model, p values larger (not smaller) than .05 indicate that the model is adequate. Although multiple tests were run, we chose to perform each of the goodness-of-fit tests using an α of .05, a conservative approach. All p values obtained from the pairwise logistic regressions indicated a good fit. Thus, the multinomial regression models that were used, which require even fewer parameter estimates than several pairwise logistic regressions, were determined to provide an adequate fit to the data.

Since the data were derived from a cross-sectional survey, information on history was obtained by comparing present status (considered the "outcome") in various groups of individuals defined retrospectively by their past status. Thus, the "outcome" for past (prior to the last 12 months) Group 1 and Group 2 cases of DSM-IV alcohol abuse was determined by the current status of these subjects. They could remain in the DSM-IV alcohol abuse category (chronic), they could be in remission from DSM-IV alcohol abuse (no abuse symptoms within the last year), or they could receive a current diagnosis of DSM-IV alcohol dependence at the time of their AUDADIS interview. A logistic regression model was used to test differences in outcome in current status between past Group 1 and Group 2 subjects when bivariate tests were significant. From the logistic regression model, an odds ratio with associated 95% confidence interval was derived.

For the interested reader, we also provided bivariate tests of group differences, along with post hoc tests of specific group comparisons. The overall test of a difference between the three groups was done using the test for no interaction based on the log odds ratio, a chi-square test appropriate for survey data (Shah, 1996). The continuous covariates, age of onset of drinking and average daily ethanol consumption, were tested using the Wald F statistic. Post hoc pairwise comparisons of the three groups were then performed using the same statistical method for categorical covariates, while the continuous covariate age of onset of drinking was tested on each pair of groups using the t statistic. A Bonferroni-corrected α for each family of tests (i.e., for each covariate) was used to adjust for the multiple tests performed. Because of the very large number of these bivariate tests, we consider them part of the descriptive material of the article and do not go into them in detail.

RESULTS

Lifetime diagnoses

Bivariate tests of independence were performed on all categorical covariates and age of onset of drinking to examine the relationship of each of the characteristics shown in Table 1 to group membership (lifetime). As shown in the table, all bivariate tests were significant with p values of $< .001$. The significance of each pair-

Table 3. Comparison by lifetime DSM-IV alcohol diagnostic status: Group 1 (drinker-drivers), Group 2 (other abuse), Group 3 (no diagnosis)

	Odds ratio (95% confidence interval)		
	Group 1 vs Group 3	Group 2 vs Group 3	Group 2 vs Group 1
Family history of alcohol problems	1.23 (1.04-1.46)*	1.52 (1.29-1.80)‡	1.24 (0.99-1.54)
Age first started drinking	0.92 (0.90-0.95)‡	0.90 (0.88-0.92)‡	0.97 (0.94-1.01)
Ever used drugs	1.99 (1.66-2.39)‡	2.60 (2.18-3.11)‡	1.31 (1.03-1.65)*
Ever in treatment for alcohol problems	1.26 (0.76-2.06)	4.08 (3.01-5.52)‡	3.25 (1.96-5.38)‡

*$p < .05$; ‡$p < .005$.

wise comparison is also noted in Table 1; 26 of the 33 of these tests showed significant group differences.

Table 3 shows the odds ratios and their significance levels derived from the generalized logit model comparing Group 1, Group 2 and Group 3 on family history of alcoholism, age the subject first started drinking, lifetime drug use, and lifetime treatment of self-help for alcohol-related problems. As noted above, the analysis controlled for age, sex, race, marital status and education. For family history of alcoholism, both diagnostic groups (Groups 1 and 2) were more likely to have a family history of alcoholism than Group 3 (the no-diagnosis group) and did not differ significantly from each other on family history. Age at onset of drinking was significantly lower in Groups 1 and 2 than in Group 3, and, again, the two diagnostic groups did not differ from each other. For a history of drug use, Groups 1 and 2 (the two abuse groups) differed significantly from the no-diagnosis group, but in this case, they also differed from each other, with Group 1 (the drinker-drivers) less likely to have used drugs than Group 2 (other). For a lifetime history of treatment, the pattern was different. Group 1 (drinker-drivers) did not differ from Group 3 (no-diagnosis group) in terms of a lifetime treatment history, but members of Group 2 were substantially more likely to have had treatment at some point in their lives than either Group 1 or Group 3. Including or removing age, sex, race, marital status or education from the model singly or in sets did not alter the effects for these variables, and hence the associations shown in Table 3 were not confounded by demographic characteristics.

To determine whether the fit of the above model was adequate, Hosmer-Lemeshow goodness-of-fit tests were performed on each of the three pairwise regression models which could be formed from the three-category outcome. Recall that p values greater than .05 indicate that the model is adequate. The test statistics and p values were as follows: 9.67, 8 df, p = .29 for the drunk-driving group vs the other abuse group; 11.63, 8 df, p = .17 for the drunk-driving vs no-diagnosis group; and 11.90, 8 df, p = .16 for the other abuse group vs the no-diagnosis group. These results indicated the aptness of the three pairwise models and thus supported the use of the even more efficient three-category multinomial logit model.

In order to investigate the relationship between group membership and the opinion (of the subject or someone else) that help seeking for an alcohol-related problem was needed although not obtained, an analysis was conducted with the subset of subjects who had never been in treatment. The small numbers of subjects with positive responses to the help-seeking questions called for a simple analysis of the difference in proportions of positive responses in the three groups. As shown in Table 4, Group 1 (drinker-drivers) did not differ from Group 3 (no-diagnosis group) for either a subjective feeling that treatment was needed or having someone else think that they needed treatment. In contrast, Group 2 (other abuse group) differed significantly from both the drinker-drivers abuse group as well as the no-diagnosis group for each of these variables, with a higher proportion of Group 2 members reporting that help seeking was believed by both others and themselves to be needed. As noted on Table 4, we adjusted for the use of multiple tests with a Bonferroni-corrected α of .05 ÷ 3 = .017. Thus a p value of .017 or smaller was required for a pairwise comparison to reach statistical significance. This occurred for both help-seeking covariates in the Group 2 versus Group 1 comparison, as well as in the Group 2 versus Group 3 comparison.

Current diagnoses

Bivariate tests of independence were performed on all categorical covariates and average daily ethanol intake (in ounces) to examine the relationship of each of the characteristics shown in Table 2 to group membership. The significance level of all tests was smaller than .005 except for two of the 15 variables tested (unemployment and education). When post hoc pairwise group comparisons were conducted, 29 showed significance at the corrected α of .017 (.05 ÷ 3 = .017). Table 5 shows the results of the main statistical analysis of the current status of subjects, the odds ratios and their significance levels derived from the generalized logit

model comparing Group 1, Group 2 and Group 3 on the covariates. These included average daily ethanol consumption, drinking during the week, drinking during the daytime, current drug use, blackouts, current treatment for alcohol-related problems (within the last 12 months) and current depressed mood (2 weeks or longer of continuous depressed mood at some point during the last 12 months). Age, race, sex, education, marital status and unemployment were included as control covariates. Total body water was also included as a control covariate when the alcohol consumption variable of interest was average daily ethanol consumption. As shown in Table 5, both Group 1 and Group 2 (current abuse groups) had a higher average ethanol consumption level over the last 12 months than Group 3 (no-diagnosis group), but did not differ significantly from each other. The same pattern was found for drinking during the daytime and current drug use. However, Group 1 (drinker-drivers) did *not* differ from Group 3 (no-diagnosis group) on drinking during the week, current alcohol treatment or depressed mood, while Group 2 (other abuse group) *did* differ from the no-diagnosis group on these three covariates. The only covariate on which the two abuse groups differed significantly from one another was blackouts, with the other abuse group significantly more likely to have blackouts than drinker-drivers and substantially more likely to have blackouts than the no-diagnosis group.

To determine whether or not the fit of the final model for current diagnoses was adequate, Hosmer-Lemeshow goodness-of-fit tests were again performed. The test statistics and p values were as follows: 8.45, 8 df, $p = .39$ for the drunk-driving group vs the other abuse group; 11.32, 8 df, $p = .18$ for the drunk-driving vs no-diagnosis group; and 12.23, 8 df, $p = .14$ for the other abuse group vs the no-diagnosis group. These results again indicated the aptness of the three pairwise models, and again supported the use of the more efficient three-category multinomial logit model.

To investigate further whether additional features of recent drinking history differentiated the groups, two additional current multivariate models were fit. In each of these models, an alcohol consumption variable was substituted for average daily ethanol consumption. These two other alcohol variables were: (1) frequency of five or more drinks during the past year (termed binge drinking by some investigators), and (2) frequency of drinking to intoxication during the past year. Each of these measures was coded as an ordinal variable. Frequency of 5+ drinks had five levels: once/month or less, 2–3 days/month, 1–2 days/week, 3–4 days/week, and every day/nearly every day. Frequency of intoxication was coded the same as the frequency of 5+ drinks covariate, except that the highest two levels were combined due to sparseness. The identical models were refit as the model described above for average daily ethanol consumption (with the exception of total body water, which was irrelevant as a control covariate in these models).

The results of these two models were similar to the results of the model shown in Table 5. The odds of being in either abuse group were significantly higher as the level (frequency) of either measure increased. The results of the model in which frequency of 5+ drinks was substituted for ethanol consumption showed that Group 1 differed significantly from Group 3 (adjusted odds ratio [AOR] 1.68, 95% CI 1.47–1.92, $p = .000$) for each unit increase in 5+ drinks per occasion. For the comparison of Group 2 to Group 3, the analogous AOR was 1.57 (95% CI 1.41–1.76, $p = .000$). Groups 1 and 2 did not differ significantly on frequency of 5+ drinks ($p = .413$). The model in which frequency of intoxication was substituted for ethanol consumption showed an AOR of being in Group 1 compared to

Table 4. Comparison of lifetime DSM-IV alcohol abuse diagnostic status by need for help seeking: Group 1 (drinker-drivers), Group 2 (other abuse), Group 3 (no diagnosis); weighted row proportions (n)

	Group 1 (n = 822)	Group 2 (n = 953)	Group 3 (n = 19,859)	Total (N = 21,634)	Significant post hoc group comparisons[a,b] 1 vs 2	1 vs 3	2 vs 3
Was there ever a time someone else thought you should seek help?	.02 (19)	.11 (100)	.01 (252)	.02 (371)	.000	NS	.000
Was there ever a time you thought you should seek help?	.01 (8)	.04 (34)	.00 (86)	.01 (128)	.002	NS	.000

Note: Subjects ever in treatment for alcohol-related problems are excluded.
[a]Based on the test for no interaction in a log-linear model.
[b]Using Bonferroni-corrected α to adjust for multiple tests required for three pairwise comparisons; a p value of less than .05/3 = 0.0167 is required for significance. Otherwise, a pairwise comparison is considered nonsignificant (NS).

Table 5. Comparison by current DSM-IV alcohol diagnostic status on need for help seeking: Group 1 (drinker-drivers), Group 2 (other abuse), Group 3 (no diagnosis)

	Odds ratio (95% confidence interval)		
	Group 1 vs Group 3	Group 2 vs Group 3	Group 2 vs Group 1
Average daily ethanol consumption	1.53 (1.32-1.77)‡	1.54 (1.32-1.79)‡	1.01 (0.92-1.11)
Drink during the week	1.24 (0.95-1.62)	1.41 (1.06-1.89)*	1.14 (0.79-1.64)
Drink during the daytime	1.41 (1.04-1.90)*	1.65 (1.26-2.16)‡	1.17 (0.81-1.70)
Current drug user	1.96 (1.36-2.81)‡	2.58 (1.85-3.60)‡	1.32 (0.84-2.07)
Blackouts	3.48 (2.33-5.21)‡	8.62 (6.19-12.00)‡	2.48 (1.60-3.85)‡
Currently in treatment for alcohol-related problems	1.71 (0.54-5.39)	4.42 (2.17-8.98)‡	2.59 (0.80-8.41)
Depressed mood	1.08 (0.71-1.63)	1.68 (1.21-2.32)‡	1.55 (0.96-2.51)

*$p < .05$; ‡$p < .005$.

Group 3 of 1.45 (95% CI 1.15–1.84, $p = .002$) for each unit increase in frequency of intoxication. The analogous AOR of being in Group 2 compared to Group 3 was 1.94 (95% CI 1.55–2.42, $p = .000$), and the AOR of being in the other abuse group vs the drinker-driver group was 1.33 (95% CI 1.04–1.71, $p = .024$).

As before, to determine whether or not the fit of the final models for these other consumption variables were adequate, Hosmer-Lemeshow goodness-of-fit tests were performed. The tests statistics and p values once again indicated the aptness of the three pairwise models and again supported the use of the more efficient three-category multinomial logit model.

Present status of subjects who met past criteria for abuse

In the NLAES data set, 1,380 subjects met criteria for past DSM-IV alcohol abuse (e.g., they met criteria at some point in their lives prior to the last 12 months). Of these, 38.7% (weighted) were of the Group 1 type (drinker-drivers) and 61.3% were of the Group 2 type (other abuse). We then determined if there was a difference in present status between subjects who had met criteria for abuse in the past just for drinking and driving compared to subjects who met criteria for abuse in other ways. We first examined present remission of abuse. We did this by determining the proportion of subjects in each group who had no symptoms of an alcohol use disorder in the last 12 months. Among the subjects who had met criteria for alcohol abuse in the past from Group 1 (drinker-drivers), 68.7% (weighted) had no current symptoms of an alcohol use disorder, compared to 65.5% of subjects who met criteria for past alcohol abuse for other reasons (Group 2 cases). These proportions did not differ significantly. We next examined the likelihood of developing current dependence. Among past cases of abuse from Group 1, 5% developed current DSM-IV alcohol dependence, compared to 10% of past cases of abuse from Group 2. In comparing the two groups, the crude OR of developing dependence was 2.05 (95% CI 1.24–3.40, $p = .00$). A logistic regression model was fit for this outcome to control for age, sex and race. After controlling for these covariates, Group 2 showed a greater risk at a marginal significance level for developing current DSM-IV alcohol dependence status (AOR 1.72, 95% CI 1.03–2.86, $p = .04$). The value of the Hosmer-Lemeshow goodness-of-fit statistic for this model was 5.09 ($p = .75$), indicating the model provided an adequate fit to the data.

DISCUSSION

In all cases, Group 2, the subjects who received a DSM-IV alcohol abuse diagnosis on some basis other than just drinking and driving, differed from Group 3, the subjects with no diagnosis. In many instances, the size of the effect was substantial. Thus, in the process of investigating the drinker-drivers (Group 1), support has been provided for the validity of the DSM-IV abuse diagnosis when made on some other basis. The validity results for the drinker-drivers were not so consistent.

For the lifetime diagnosis of DSM-IV alcohol abuse, two patterns of results emerged. For the three variables unrelated to treatment history, Group 1, the drinker-drivers, differed from the no-diagnosis group but did not differ from other subjects with DSM-IV alcohol abuse. These three variables were age at onset of drinking, drug use, and family history of alcoholism. We presented this pattern as one supporting the validity of the drinker-driver abuse category. For the three variables related to treatment, the results were different. For a history of treatment, for a subjective feeling that treatment was needed and for a recommendation from someone else that treatment was needed, the drinker-drivers did not differ from the subjects with no diagnosis. For two of these treatment-related variables, the two abuse groups did differ from each other, with Group 2 much more likely to have had treatment and to have had someone else suggest treatment than Group 1.

For the current diagnoses of DSM-IV alcohol abuse, we tested the relationships of six variables to the different groups. For three of these variables, Group 1

(drinker-drivers) differed from the no-diagnosis group but not from Group 2. For the other three variables, Group 1 did not differ from the no-diagnosis group and did differ from Group 2 on depressed mood. Thus, results were mixed for both lifetime and current diagnoses of DSM-IV alcohol abuse.

Finally, we examined the current status of subjects who had met criteria for abuse in the past. The two abuse groups did not differ in their likelihood of current remission. In terms of developing dependence, Group 2 was at higher risk. Thus, examining the history also produced mixed results.

This study offered a number of considerable methodological benefits and one shortcoming in the study of DSM-IV alcohol abuse. The benefits included the fact that the data were obtained from a nationally representative sample of the U.S. general population, providing generalizability. Also, the sample was large, allowing investigation of the two subsets of the DSM-IV abuse category for both current and lifetime diagnoses, an option that would not be possible in other existing data sets. The measures were derived from an interview that was tested for reliability in the general population and found to be reliable. The study (in conjunction with a series of other recent articles on nosology; for example, Hasin et al., 1997c,d; Hasin and Paykin, 1998, 1999, in press) also provided a model for investigating the validity of different definitions of alcohol use disorders other than one that permits a simple comparison of rates under various definitions.

A drawback of the study is the absence of prospectively collected longitudinal data on the two DSM-IV abuse groups. Prospective data are less vulnerable to bias than are retrospective data (e.g., memory problems, or differential loss from the underlying population of members of the two groups through mortality). If the results on retrospectively determined history had been consistent with each other but had contrasted with a set of opposite findings from the multivariate results, we would have given the multivariate results more weight, since they do not suffer from the same potential methodological problems. We did not have to make this choice, however, as both sets of results were mixed.

Analysis of the relationship of antisocial personality disorder to the groups studied above would have added interesting information to this study. However, this personality disorder was not included in the interview. Future similar nosologic studies might be improved with the addition of antisocial personality disorder.

In general, the argument for a change in a major nomenclature must be supported by clear empirical evidence. Numerous studies have indicated difficulties in the abuse category, suggesting that some type of change is needed. We investigated one possibility for change: elimination of a common but conceptually questionable way to receive a diagnosis of DSM-IV alcohol abuse, one based on driving after drinking too much when this occurred in isolation from other alcohol-related problems. The premise of the investigation was that a few instances of driving after drinking represent unwise behavior but not necessarily a psychiatric disorder. In contrast to the very consistent results differentiating subjects with no DSM-IV alcohol diagnosis from DSM-IV abuse based on other ways of getting the diagnosis, the validity results for the drinker-driver group were inconsistent. The large sample size eliminated poor statistical power as a cause of these findings. These results would probably not have been seen as strongly supporting the addition of the drinker-driver abuse category to DSM-IV if it had not already been there. Further conceptual and empirical work remains to be done, however, to resolve the question of how best to modify the substance abuse category for DSM-V.

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NIH Publication No. 02–4997
National Institutes of Health
National Institute on Alcohol Abuse and Alcoholism
Printed June 2002