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Editors’ Note

Measuring the Burden
Alcohol’s Evolving Impact on Individuals, Families, and Society

Jürgen Rehm, Ph.D., and Ralph Hingson, Sc.D.

Alcohol use is associated with tremendous costs to the drinker, those around him or her, and society as a whole. These costs result from the increased health risks (both physical and mental) associated with alcohol consumption as well as from the social harms caused by alcohol. This issue of Alcohol Research: Current Reviews examines the public health impact of alcohol consumption, looking at the full burden of disease that can be attributed to drinking.

The attempt to measure the impact of alcohol use on various disease categories is relatively new to the alcohol research field. In fact, much of our understanding of how alcohol affects health and disease in society is rooted in work from the 1980s and 1990s. This research reflects a truly international perspective. A group of Australian authors, led by Dr. Dallas English, were some of the first to look at the issues involved in attributing mortality and morbidity to substance abuse (English et al. 1995). Dr. James Shultz and his colleagues conducted other seminal research for the Centers for Disease Control and Prevention. They developed the Alcohol-Related Disease Impact (ARDI) software to allow States to calculate mortality, years of potential life lost (YPLL), direct health care costs, indirect morbidity and mortality costs, and non–health sector costs associated with alcohol use (Shultz et al. 1991). Canadian researchers, under the direction of Dr. Eric Single, developed the Canadian version of the alcohol-attributable fractions in the mid-1990s (Single 1999). Also at the forefront of research in this field are Dr. Robin Room and this issue’s Scientific Review Editor, Dr. Jürgen Rehm, both of whom have made significant contributions to our overall understanding of the field.

The study of the burden of disease on a truly global scale began with the World Health Organization’s (WHO’s) Global Burden of Disease Study (Murray and Lopez 1996). Thanks to the WHO efforts, we now have a worldwide view of the far-reaching consequences of alcohol use and misuse.

Although the field has made much progress and in a short time, there are research gaps that still remain. For example, more research is needed on the relationship between diseases and detailed drinking patterns; more needs to be known about the burden of alcohol-related mental disorders (e.g., depression); future research needs to disentangle the effect of comorbid conditions when assessing the burden of disease attributable to alcohol; the estimates of how alcohol contributes to infectious diseases like HIV need to be further refined; and the alcohol-attributable fractions need to be updated more frequently in response to new developments in science and as the population’s health status and behaviors change.

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Finally, the impact of alcohol on social harm, including harm to people other than the drinker, still is terra incognita in many areas (Gmel and Rehm 2003).

Over the last two decades, the United States has made substantial progress in improving public health. Still, alcohol remains an important risk factor for disease burden and social harm, not only in the United States but also globally (Murray et al. 2013). Additional research in this area will increase our understanding of alcohol’s role in creating disease burden and social harm and aid in the development of stronger, more effective measures to prevent these devastating effects.

References


MEASURING THE BURDEN: ALCOHOL’S EVOLVING IMPACT

Alcohol’s impact stretches far beyond the drinker, affecting his or her family as well as the community at large. This widespread impact makes alcohol a major public health concern. This introductory article by Drs. Ralph Hingson and Jürgen Rehm provides an overview of the history of alcohol’s burden of disease. (pp. 122–127)

USING SURVEYS TO CALCULATE DISABILITY-ADJUSTED LIFE-YEARS

To quantify the total burden of morbidity attributable to alcohol use, so-called disability weights (DWs) must be generated. General-population surveys can be used to derive DWs from health valuation tasks. This article by Drs. Wolfgang Weidermann and Ulrich Frick discusses the application of three psychometric methods—pairwise comparisons, ranking tasks, and visual analog scales—in general-population surveys and outlines their strengths and weaknesses. In addition, the article discusses a recently proposed health valuation framework, which highlights the underlying cognitive processes from a social judgment perspective. Furthermore, it presents a structured data-collection procedure that seems promising in deriving DWs from general-population surveys. (pp. 128–134)

ASSESSING THE IMPACT OF ALCOHOL USE ON COMMUNITIES

Community indicators are used to assess the impact of alcohol on communities. For communities, indicator data can be used to inform priority-setting agendas by identifying specific concerns within a community, guide policy and education initiatives, monitor community status on a particular measure over time or in comparison with other communities, and evaluate programs or policies. This article by Drs. Andrea Flynn and Samantha Wells reviews the main data sources for community indicators, discusses their strengths and limitations, and discusses indicators used in reference to four main topics relating to alcohol use and problems at the community level: alcohol use, patterns, and problems; alcohol availability; alcohol-related health outcomes/trauma; and alcohol-related crime and enforcement. (pp. 135–149)

FOCUS ON: CHRONIC DISEASES AND CONDITIONS RELATED TO ALCOHOL USE

Numerous chronic diseases and conditions are entirely or partially attributable to heavy alcohol use; for other conditions, however, alcohol can have a beneficial effect. Various factors, such as the average amount and pattern of alcohol consumption, have been found to influence alcohol’s impact on the mortality and morbidity related to chronic diseases and conditions. However, as Mr. Kevin D. Shield and Drs. Charles Parry and Jürgen Rehm report, although alcohol consumption indisputably contributes to the burden of chronic diseases and conditions, the methods currently used to calculate the relative risks and alcohol-attributable fractions have several limitations. Moreover, new studies and confounders may help further refine our knowledge of which chronic diseases and conditions are causally linked to alcohol consumption. (pp. 155–173)

FOCUS ON: ALCOHOL AND MORTALITY

Alcohol consumption has long been recognized as a risk factor for mortality. By combining data on alcohol per capita consumption, alcohol-drinking status and alcohol-drinking patterns, risk relationships, and mortality, the Comparative Risk Assessment Study estimated alcohol-attributable mortality for 1990 and 2010. In this article, Drs. Jürgen Rehm and Kevin D. Shield discuss alcohol’s role in the global burden of mortality. (pp. 174–183)

FOCUS ON CHILDREN AND PREADOLESCENTS

Because there are few surveillance studies of alcohol use and alcohol-related problems among children and preadolescents, estimating the alcohol burden in this population is especially difficult. This article by Dr. John E. Donovan summarizes information from U.S. national and Statewide surveys on the prevalence of alcohol use among children in grades 6 and lower. Although the rates of alcohol use are relatively low in this population, substantial numbers of children do in fact have experience with alcohol. Limited available data highlight the need for better ongoing surveillance of this population. Although alcohol burden in children appears relatively low, it is increased through the alcohol use and abuse of their parents, and through the increased likelihood among early drinkers of alcohol problems and other negative outcomes in adolescence and young adulthood. (pp. 186–192)

PREVALENCE AND PREDICTORS OF ADOLESCENT ALCOHOL USE AND BINGE DRINKING IN THE UNITED STATES

The Monitoring the Future study is an annual survey among 8th-,
10th-, and 12th-grade students assessing their alcohol use, thereby allowing researchers to better understand adolescents’ consumption patterns during this vulnerable developmental period. These surveys have found high prevalence of drinking and binge drinking by the time students leave high school, report Drs. Megan E. Patrick and John E. Schulenberg. The authors also discuss the factors that influence adolescent alcohol use, such as parent and peer relationships, school and work, behavioral and drug-use problems, or personality characteristics, as well as review potential long-term effects of adolescent alcohol consumption. (pp. 193–200)

EXCESSIVE ALCOHOL CONSUMPTION AND RELATED CONSEQUENCES AMONG COLLEGE STUDENTS

Research shows that multiple factors influence college drinking, from an individual’s genetic susceptibility to the positive and negative effects of alcohol, alcohol use during high school, campus norms related to drinking, expectations regarding the benefits and detrimental effects of drinking, penalties for underage drinking, parental attitudes about drinking while at college, whether one is member of a Greek organization or involved in athletics, and conditions within the larger community that determine how accessible and affordable alcohol is. This article by Drs. Aaron White and Ralph Hingson examines recent findings about the causes and consequences of excessive drinking among college students relative to their noncollege peers and many of the strategies used to collect and analyze relevant data, as well as the inherent hurdles and limitations of such strategies. (pp. 201–218)

FOCUS ON: WOMEN AND THE COSTS OF ALCOHOL USE

Although there are proven beneficial effects associated with light-to-moderate drinking, such levels of drinking also are associated with increased risks of breast cancer and liver problems, and heavy drinking increases risks of hypertension and bone fractures and injuries. In addition, women’s heavy-drinking patterns and alcohol use disorders are associated with increased likelihood of many psychiatric problems, including depression, posttraumatic stress disorder, eating disorders, and suicidality, as well as increased risks of intimate partner violence and sexual assault. In this article, Drs. Sharon C. Wilsnack and Richard W. Wilsnack and Ms. Lori Wolfgang Kantor discuss drinking patterns among women during midlife and the risks of heavy drinking in this population. (pp. 219–228)

FOCUS ON: ETHNICITY AND THE SOCIAL AND HEALTH HARMS FROM DRINKING

Native Americans, Hispanics, and Blacks are at higher risk of experiencing alcohol-attributable harms than Whites or Asians. This article by Drs. Karen G. Chartier, Patrice A.C. Vaeth, and Raul Caetano examines this health disparity with regard to alcohol and unintentional injuries, intentional injuries, fetal alcohol syndrome, gastrointestinal diseases, cardiovascular diseases, cancers, diabetes, and infectious diseases. (pp. 229–237)

GAPS IN CLINICAL PREVENTION AND TREATMENT FOR ALCOHOL USE DISORDERS

Numerous prevention and treatment approaches have been developed to ameliorate the morbidity, premature mortality, and other social and economic burdens on society caused by heavy drinking. Those measures can be targeted at different groups of drinkers, including nondependent heavy drinkers, those with functional dependence, and those with alcohol use disorders. However, as Dr. Mark L. Willenbring explains, the impact of current selective prevention and treatment strategies on public health is unclear. For example, existing screening and brief interventions for nondependent drinkers may potentially have a large public health impact, whereas more effective ways are needed to reduce the public health burden associated with functional dependence and alcohol use disorders. (pp. 238–243)

THE WORLD HEALTH ORGANIZATION’S GLOBAL MONITORING SYSTEM ON ALCOHOL AND HEALTH

With growing awareness of the impact of alcohol consumption on global health, the demand for global information on alcohol consumption and alcohol-attributable and alcohol-related harm as well as related policy responses has increased significantly. This article by Drs. Vladimir Poznyak and Alexandra Fleischmann, Mr. Dag Rekve, Ms. Margaret Rylett, and Drs. Jürgen Rehm and Gerhard Gmel examines the increasing demand from World Health Organization (WHO) Member States and the latest policies set forth by the WHO in response to alcohol’s growing burden of disease worldwide. (pp. 244–249)

RESULTS FROM THE NIAAA EXPERT PANEL ON ALCOHOL AND CHRONIC DISEASE EPIDEMIOLOGY

NIAAA Expert Panel on Alcohol and Chronic Disease Epidemiology workshop provided an excellent forum for summarizing the current state of the field of alcohol research and for identifying future research opportunities. This article by Drs. Rosalind A. Breslow and Kenneth J. Mukamal reviews the outcomes of the workshop and the ideas that highlight areas in need of additional study and offer a roadmap for moving forward across a variety of methodological approaches and content areas. (pp. 250–259)
Measuring the Burden: Alcohol’s Evolving Impact

Ralph Hingson, Sc.D., and Jürgen Rehm, Ph.D.

Measuring the impact of alcohol consumption on morbidity and mortality depends on the accurate measurement of alcohol exposure, risk relationships, and outcomes. A variety of complicating factors make it difficult to measure these elements. This article reviews these factors and provides an overview of the articles that make up this special issue on current research examining alcohol’s role in the burden of disease. These topics include estimating alcohol consumption as well as alcohol-related morbidity and mortality in various demographic groups, and the burden of alcohol use disorders.

**Key words:** Alcohol use consumption; alcohol use frequency; alcohol use pattern; alcohol burden; public health impact; burden of disease; morbidity; mortality; disease; measurement; outcomes; specificity of measurement; sensitivity of measurement

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This issue of *Alcohol Research: Current Reviews* examines the public health impact of alcohol consumption beyond the role of alcohol use disorders alone (Room et al. 2005)—that is, it looks at the burden of disease. Determining impact hinges on accurate and consistent “measurements.” As demonstrated in the articles in this issue, impact typically is estimated based on three elements (Rehm et al. 2010b; Walter 1976):

- The measurement of exposure (i.e., the relevant dimension of alcohol use) causing the burden (Kehoe et al. 2012; Rehm et al. 2010b);
- The measurement of the risk relations (i.e., what level/pattern of consumption is linked to what outcome) (Rehm et al. 2010a); and
- The measurement of outcomes.

These different measurements are central to this issue of *Alcohol Research: Current Reviews*.

**Measurement Challenges**

Numerous challenges exist when measuring the extent and predictors of alcohol-related mortality and morbidity. Those challenges also affect our ability to evaluate the effectiveness of interventions to reduce alcohol-related morbidity and mortality. Different challenges exist for acute alcohol-related mortality and chronic disease mortality and morbidity, although some of the same challenges confront measurement in both areas. The following list of challenges is illustrative but not exhaustive.

**Acute Mortality and Morbidity (Injuries and Poisonings)**

Postmortem alcohol test data are not consistently available for many types of acute injury or poisoning deaths. The best available U.S. estimates indicate alcohol-attributable acute deaths outnumber chronic disease deaths 44,000 to 35,000 (Centers for Disease Control and Prevention [CDC] 2013). Traffic crashes have been the leading category of alcohol-attributable injury or death over the past 30 years. During that time period, the majority of drivers in fatal crashes (both fatally injured and, to a lesser extent, surviving drivers) have been tested for alcohol. This permits researchers to make accurate estimates of the number of drivers, passengers, and others who die in fatal crashes in which a driver was known to have been drinking.
In addition, by examining the characteristics of crashes and drivers in fatal crashes where alcohol is present, the National Highway Traffic Safety Administration (NHTSA) (Klein 1986; NHTSA 2002) has developed an imputational approach to estimate the proportion of fatal crashes involving alcohol even when the driver is not tested. This approach was verified using data from States with high percentages of drivers involved in fatal crashes that were tested for alcohol use. Researchers used the NHTSA model to predict the percentage of fatal crashes that involved alcohol and then compared those findings with the actual alcohol test results. NHTSA found they could estimate with great accuracy not only the proportion of fatal crashes involving alcohol-positive drivers but also the blood alcohol level (BAL) of the driver at the time of the crash.

Using this approach, researchers have been able to determine annual State and national estimates of alcohol involvement in fatal traffic crashes since 1982. Furthermore, having those accurate direct-test results and imputed results has permitted researchers to make epidemiologic estimates of the increased odds of fatal crashes and other crash involvement at various BALs (Voas et al. 2012). Drivers who were stopped at random in roadside surveys were compared with drivers who were fatally injured in single-vehicle crashes and who were driving in the same States on the same types of roads on the same days of the week and times of day.

Because these data are available monthly on a national, State, and community level, researchers also have been able to monitor trends in fatal crashes involving alcohol relative to fatal crashes where alcohol is not involved over time. In addition, researchers are able to use quasi-experimental and other research designs to evaluate whether State-level traffic safety legislation and community-level education and law enforcement and treatment programs are effective in reducing alcohol-related traffic deaths (Ferguson 2012; Hingson and White 2013).

Such studies have guided policymakers to select and implement effective programs and policies. Since the early 1980s, alcohol-related traffic death rates per 100,000 have been reduced more than 50 percent versus the decline in traffic crash death rates where alcohol is not involved (figure 1). It has been estimated that as many as 300,000 deaths have been prevented as a result of reduced incidences of drinking and driving, which is greater than those attributed to increased use of airbags, seat belts, and motorcycle and bicycle

Figure 1  Alcohol-related versus non–alcohol-related traffic fatalities, rate per 100,000, all ages, United States, 1982–2010

helmets combined (Cummings et al. 2006; Fell and Voas 2006).

Unfortunately, unlike traffic deaths, postmortem alcohol testing is not nearly as complete for other types of unintentional or intentional poisoning and injury deaths. In 18 States (figure 2), a violent-death registry is in place where 80 percent or more of all homicides and suicides are tested. However, testing levels of alcohol for other types of injuries or deaths is not routine. As a consequence, imputations for alcohol involvement in other types of injuries or deaths have not been developed, and studies of laws and programs to reduce those types of injuries and deaths do not have the same precision as evaluations of efforts to prevent alcohol-related traffic deaths.

Second, research is emerging indicating that alcohol may interact with and pharmacologically potentiate the effects of other drugs, thereby increasing risks of motor-vehicle crashes (Asbridge et al. 2012; Li et al. 2012) and poisoning/overdose deaths (White et al. 2011). People may be involved in traffic crashes or poisoning deaths at lower BALs if other drugs are present, and this may modify BAL levels used in establishing attributable fractions for motor-vehicle and poisoning deaths. Most national surveys and many research projects inquire about alcohol and drug consumption separately not simultaneously. If alcohol and drugs pharmacologically interact, simultaneous-use questions should be considered.

Third, it is important to calculate the secondhand harm alcohol misuse poses. Just as awareness of the secondhand negative consequences of passive smoke inhalation has heightened the public health resolve to curb smoking, learning about the secondhand effects of alcohol misuse may heighten the resolve to study and implement effective interventions to reduce alcohol misuse. For example, 40 percent of people who die in traffic crashes involving drinking drivers in the United States are not driving. Half of the deaths in crashes involving drinking drivers under the age of 25 are those other than the driver. This has incited citizen activists and policymakers to pass more than 2,000 laws at the State and Federal levels to reduce alcohol-impaired driving (Hingson et al. 2003).

Fourth, many prevention activities are implemented at the community level, and community-level data are needed to stimulate the planning and evaluation of those interventions (Hingson and White 2012). Yet most surveillance data-monitoring systems measure behavior and consequences at the State and Federal levels. Strategies are needed to either facilitate more community-level data collection or to offer technical assistance to concerned communities and researchers so that they can collect their local data using standardized questions and sampling procedures for comparison with other communities, their State, and the Nation.

Figure 2 States participating in National Violent Death Registry (18 States)

Chronic Conditions

When examining either acute-disease and chronic-disease mortality and morbidity, a variety of measurement challenges may produce underreporting. First, drinking levels reported in surveys account for only 40 to 60 percent of alcohol sales (Midanik 1982; World Health Organization [WHO] 2011). Underreporting may lead to underestimates of alcohol's contribution to chronic disease (Meier et al. 2013). Second, survey respondents often underestimate alcohol serving sizes, particularly when consumed in containers that vary from accepted standard drink sizes. Memory may become an issue after respondents have consumed so many drinks so rapidly that they incur partial memory lapses or total blackouts. Also, the duration of time that respondents are asked to recall consumption can vary in different studies. In general, shorter time periods of recall (e.g., days and weeks) produce higher consumption, estimates than requests for monthly, yearly, or lifetime consumption. On the other hand, drinking patterns may vary over time and even in the same year, prompting recommendations to use yearly recall periods. Method or mode of data collection (i.e., face-to-face, telephone, mail, or Web based) also can influence drinking reports as well as response rates and biases in survey samples. Household-based surveys may not include groups with high levels of alcohol consumption, such as students, the homeless, or people in institutions or in inpatient alcohol treatment facilities (Meier et al. 2013; Stockwell et al. 2004). Also, unrecorded alcohol, use of alcohol in food, spillage, waste, and consumption by children and tourists may not be considered in surveys (Meier et al. 2013).

Second, especially with chronic disease, in etiology studies the time proximity of drinking data collection to disease outcome must be carefully evaluated. Although some chronic diseases may take years to develop, cessation or reduction of drinking may stop the process and reduce morbidity or mortality consequences almost immediately. This can be seen in the immediate gains in mortality and life expectancy in Russia following the Gorbachev reforms that led to a reduction of drinking (Leon et al. 1997). However, these immediate gains could be found for some chronic diseases, but not for others, such as cancer. In cohort studies, drinking patterns can vary in the same individual over time, and etiology studies vary in how often someone drank over time and/or the intervals over time when drinking was measured.

Third, maintaining high response rates in surveys and longitudinal studies has become increasingly difficult over time, particularly using telephone methods, as the percentage of the population who uses mobile phones increases. If nonresponse becomes high and disproportionately involves people with characteristics and behaviors (involving but not limited to alcohol use that influence disease and injury etiology), that may cloud our understanding of alcohol's role in the development and progression of disease. It also can limit the ability of researchers to monitor disease and death-rate trends over time.

Fourth, both in estimates of acute and chronic conditions, attributable fractions from meta-analyses of epidemiologic studies are used to estimate alcohol's contribution to mortality and disability. Yet, these attributable fractions may change over time. For example, the percentage of factual traffic-crash deaths that involve alcohol have dropped from 60 percent to just under 40 percent in the past 30 years (NHTSA 2012). If the most current epidemiologic studies are not used in alcohol-attributable fraction estimates, the proportion of acute and chronic disease mortality and morbidity attributed to alcohol may be inaccurate.

Fifth, when chronic disease morbidity and mortality attributions are made, the range of diseases considered may vary. Current U.S. estimates may not fully consider alcohol's role in chronic diseases such as HIV.

Overview of Measuring the Burden: Alcohol's Evolving Impact

This issue of ARCR examines the methodology involved in measuring the burden of alcohol use in greater detail. Drs. Flynn and Wells (2013) provide an overview on consumption indicators; environmental background indicators such as availability information; alcohol-attributable problems; indicators for alcohol-attributable health outcomes (both chronic and acute); and, last but not least, law enforcement indicators. They also make a case for triangulating different data sources in order to come to valid conclusions as well as outline methods and statistical techniques to technically integrate these data.

Dr. Cheryl Cherpitel focuses more in depth on one of these data sources for the community in her examination of hospital emergency departments (Cherpitel 2013). She describes not only the methodologies to make use of these data, such as case-control or case-crossover designs and their potential biases, but also the use of such data to derive alcohol-attributable fractions, which is a research topic in its own right (Shield et al. 2012a).

The next two chapters deal with two other outcomes of alcohol use. Dr. Shield and colleagues (2013b) summarize findings on the impact of alcohol and chronic disease (i.e., cancer, neuropsychiatric conditions, cardiovascular, and digestive diseases). Methodologically, they discuss limitations of current techniques used to derive risk relations and consequently, attributable fractions.

Drs. Rehm and Shield (2013) focus on mortality, more specifically on global estimates of alcohol-attributable mortality for the year 2010. They report the causes of death with comprise the overwhelming majority of all alcohol-attributable deaths: cancer, liver cirrhosis, and injury. Clearly, cancer reflects the mortality-related alcohol use 15 to 20 years ago, liver cirrhosis mainly current drinking but
also a bit of the history, and injury with the exception suicide mainly the level of current acute consumption (Holmes et al. 2012).

No overview on alcohol use and consequences would be complete without mentioning the efforts to enumerate alcohol-attributable economic costs. The sidebar focuses on the last attempt to estimate such costs for the United States, focusing on heavy drinking (Bouchery et al. 2010).

The first part of the volume is complimented by three sidebars. Dr. Poznyak and colleagues (2013) give insight into the WHO system to collect data on alcohol consumption, alcohol-attributable harm, and alcohol policy. Drs. Wiedermann and Frick (2013) describe the use of surveys to derive disability weights to calculate the disability-adjusted life-years. Dr. Hilton (2013) introduces an important national data bank—the NIAAA Alcohol Policy Information System—for alcohol research.

In the second part of the volume, the focus is on alcohol use and its consequences over the lifespan. It starts chronologically with use by children and adolescents (Donovan 2013; Patrick 2013). Another group, in part defined by age, is college students (White 2013). All three groups again are characterized by specific methodological problems. For example, regular quantity–frequency measures do not capture alcohol use best, as consumption tends to vary. Also, especially for high-school and younger students, a lot of research is based on cross-sectional studies, which makes causal conclusions impossible. More longitudinal studies are needed to start disentangling the web of the impact of alcohol starting from earliest consumption. Such studies may append the usual self-report measures with objective measures such as repeated BAL for college students (for example, see Thombs et al. 2009).

For children, adolescents, and college students, despite the problems with establishing causality, a number of alcohol-attributable consequences have been identified. Some of them may be further in the future, as there are links between age of onset of alcohol use and alcohol dependence or other consequences in later years in the United States (Donovan 2013; Grant and Dawson 1997). The most important consequence is alcohol-attributable death. Although this outcome is relatively infrequent, a comparison to all-cause deaths during this stage of life shows that alcohol is the most important risk factor for morality (and serious illness) (Rehm et al. 2006).

Other groups of concern highlighted in this issue include women (Wilsnack et al. 2013) and ethnic groups (Chartier et al. 2013). Although women in all countries drink less, have less heavy-drinking occasions, and experience less alcohol-attributable harm than men (WHO 2011), this gap seems to be closing in several countries including the United States (Shield et al. 2012b; Wilsnack et al. 2013). As for ethnicity and race, Native Americans, Hispanics, and Blacks experience higher rates of alcohol-attributable harm than Whites in the United States. This is of course in part linked to different drinking patterns (Chartier et al. 2013; Shield et al. 2013a); but it also may be worsened by an interaction of socioeconomic status and alcohol (Schmidt et al. 2010). Drs. Chartier and colleagues show that more detailed studies are needed, specifically on the mechanisms of alcohol’s impacts on health consequences in different ethnicities and races.

Alcohol use disorders (AUDs) are one of the most important consequences of alcohol use. Dr. Willenbring (2013) examines some of the issues related to measuring the public health impact of AUDs and treatment. Although heavy drinking is responsible for the majority of the alcohol-attributable burden of disease and mortality (for estimates, see Rehm et al. 2013), the public health impact of interventions—from screening and brief interventions to treatment of alcohol dependence—is not fully understood (for exceptions, see McQueen et al. 2011, who found that brief interventions in the hospital setting were associated with a reduction of mortality after one year of 40 percent, or Rehm et al. 2013, who found that increases of the treatment rate to 40 percent in Europe could help avoid more than 10 percent of alcohol-attributable mortality). Again, more research is needed to understand the long-term consequences of interventions.

The issue concludes with contributions on the new Diagnostic and Statistical Manual of Mental Disorders—IV (Grant 2013). Notes from a special NIAAA expert panel on alcohol and chronic diseases (Breslow and Mukamal 2013) also are included and outline future research opportunities for the field.

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Mapping a certain disease into a system of disabling attributes allows researchers to compare diseases within a common framework. To quantify the total burden of morbidity (e.g., morbidity attributable to alcohol use), so-called disability weights (DWs) must be generated. General-population surveys can be used to derive DWs from health valuation tasks. This article describes the application of three psychometric methods (i.e., pairwise comparisons, ranking tasks, and visual analog scales) in general-population surveys and outlines their strengths and weaknesses. A recently proposed health valuation framework also is presented, which highlights the underlying cognitive processes from a social-judgment perspective and presents a structured data-collection procedure that seems promising in deriving DWs from general-population surveys.

To quantify the burden of a disease within a population, a health-gap measure is more useful than measures of health expectancy or quality-adjusted life-years (see Etches et al. 2006). Disability-adjusted life-years (DALYs), the most prominent of the health-gap measures, combine the burden attributable to early death and to morbidity into one single number. Alcohol affects a long list of diseases and disabilities in varying intensities, each of which can be described by a number of health-state attributes. Common measures of health outcomes include the EuroQol5D (EQ5D) (Brooks and EuroQol Group 1996), the Health Utilities Index III (HUI III) (Feeny et al. 2002), the Short-Form 36 Health Survey (SF36) (Ware and Sherbourne 1992), and the CLAssification and MEasurement System of Functional Health (CLAMES) (McIntosh et al. 2007).
gamble with one better and one worse outcome (e.g., full health or death). Respondents are asked what probability of the better outcome would make them indifferent to remaining in the described state (ill health) for certain or choosing the risky option. Therefore, if they are indifferent to the ill-health state and gamble with a 0.8 probability of the better outcome (but 0.2 probability of the worse outcome), 0.8 represents the utility of the ill health.

In a TTO task, respondents are asked to consider the relative amounts of time (e.g., number of life-years) they would be willing to sacrifice to avoid a certain poorer health state (e.g., frequent headaches). Assuming a scenario of 10 years with frequent headaches, the respondent may be indifferent to this state and a shorter lifetime of 7 years, resulting in an estimated utility for the frequent-headaches health state of 0.7 (7 years divided by 10 years).

A typical PTO elicitation asks respondents to choose between two equally expensive health care treatment programs that improve quality of life or save lives for two groups of patients. The decisionmaker must choose to fund one of the two mutually exclusive programs, one of which has a fixed number of patients. Respondents are asked how many patients would need to be treated to make them indifferent to the two programs. For example, program A might extend the life of 100 healthy individuals for 1 year, whereas program B might cure 100 individuals of a chronic health condition.

All three methods are time consuming, require highly motivated respondents, and are hardly feasible without a trained interviewer or computer program. Whereas TTO has been used in face-to-face interviews in the general population quite often (e.g., Badia et al. 2001; Chevalier and de Pouvourville 2011; Dolan 1997; Greiner et al. 2005; Jelsma et al. 2003; Jo et al. 2008; Lamers et al. 2006; Lee et al. 2009; Shaw et al. 2010; Tsuchiya et al. 2002; Wittrup-Jensen et al. 2009; Zarate et al. 2008), in mail surveys only two studies (Burström et al. 2006; Lundberg et al. 1999) used TTO to quantify respondents’ own health states. SG and PTO have rarely been used for eliciting health-state preferences in mailed surveys (i.e., they are usually used in face-to-face or phone interviews), and they have only been used among former patients (i.e., not the general public) (Hammerschmidt et al. 2004).

Readers are referred to Rehm and Frick (2010) for an overview on the methodological problems associated with econometric elicitation methods in this context. Recently, Wittenberg and Proszer (2011) described two additional sources of bias or mistaken responses in preference measurement in surveys: ordering errors (i.e., illogical responses, which violate a naturally given order, whereas inconsistent responses contradict each other within a person), and objections/invariance (i.e., respondents may refuse to participate because of an unwillingness to trade time [in the TTO task] or risk [in the SG task]). Furthermore, the meaning of SG results has been criticized as rather a measurement of risk attitude than a representation of subjective utility (Lenert and Kaplan 2000). TTO results as a metric for utility have been shown to vary with respondents’ age, education, and current health state (Ayalon and King-Kallimanis 2010; Meropol et al. 2008; Stiggelbout et al. 1996; Voogt et al. 2005). Feasibility of PTO frequently is hampered because people tend to refuse such tasks because of their desire to avoid prejudice and discrimination (Damschroder et al. 2005).

As alternatives to the methods described above, psychometric theory provides paired comparisons, ranking tasks, and visual analogue scales as tools to elicit health-state preferences. These tools are discussed below.

**Paired Comparisons**

In the context of health-state valuation, a paired comparison (PC) task simply means that respondents must choose which of two given states is more disabling, worse, or dominant in some way. Because measuring via PC seems quite simple and feasible (because it is only necessary to present all health states in a consistent descriptive system), it has been applied in various surveys in the general population (Bijlenga et al. 2009; Kind 1982, 2005; Prieto and Alonso 2000; Ratcliffe et al. 2009; Stolk et al. 2010). For a recent application of PCs among an expert panel see Rehm and Frick (2013). Deriving DWs from the resulting pattern of dominance relations, by contrast, constitutes a complex statistical task for which solutions have been formulated from the theory of Thurstone scaling (Thurstone 1927), conditional logistic regression (Hosmer and Lemeshow 2000), and loglinear modeling (Critchlow and Fligner 1991).

Methodological challenges associated with PC stem from logically inconsistent judgments (e.g., A > B and B > C, but C > A) and from rapidly increasing burden of task when comparing larger numbers of health states (i.e., combinatorial explosion). Intransitive judgments (e.g., in comparing 10, 7, and 5, 10 is preferred to 7 and 7 is preferred to 5, but 5 is preferred to 10) may originate in unintended framing effects as well as in imperfect judgment (von Winterfeld and Edwards 1986). Recently published experimental stud-
ies favor the position that excluding inconsistent ratings cannot improve the description of true preferences and therefore might to some degree be an inevitable consequence of the decisionmaking process itself (Linares 2009). To keep the number of judgments at manageable dimensions, several studies have used incomplete factorial designs (Bijlenga et al. 2009; Prieto and Alonso 2000; Ratcliffe et al. 2009).

Asking subjects to rank order several health states, but statistically analyzing rankings as PCs, was used as an alternative in several studies (Krabbe 2008; Ip et al. 2004). Rankings can be transformed into a series of PCs (Francis et al. 2002), which at first glance avoids inconsistent judgments.

### Ranking Tasks

Health-state rankings (i.e., putting several health states into an ordinal sequence of disability), which also provide comparative information, require less cognitive effort for survey respondents. Furthermore, simultaneous comparisons of multiple health states might be less sensitive to biases (e.g., those provoked by arbitrarily labeled endpoints of rating scales) (Maydeu-Olivares and Böckenholt 2008). Although ranking exercises had been included in numerous valuation studies as an external comparison measure for TTO and SG, researchers had not used the resulting ordinal data (McCabe et al. 2006) for construction of DWs before the seminal article by Salomon (2003). Cardinal utilities derived from health-state rankings displayed high agreement to utilities from TTO or SG methods (Craig et al. 2009a, b; Kind 2005) and were more stable in a cross-cultural comparison than weights derived from SG (Ferreira et al. 2011).

From a more theoretical viewpoint, articles by Flynn and colleagues (2010) and Flynn (2010) have raised serious statistical concerns about the use of ranks as a substitution for econometric valuation tasks. Their critique focuses on modeling assumptions and thus seems beyond the scope of this article. Nevertheless, their argument suggests that it can be important to restrict the number of alternatives to be ranked and to pay special attention to how a respondent generates rankings. In addition, Lenert and colleagues (1998) have demonstrated that reported utilities are heavily influenced by the search process used to form a certain judgment. This matches the notion that preferences often are constructed (instead of merely obtained) in the elicitation process (Slovic 1995). Ranking tasks within self-administered questionnaires might be hampered by limited control of the mechanism respondents use to generate the rank order. This introduces at least two issues: First, it remains unclear which reference attributes the respondent uses to generate the rank order, which constrains intersubjective comparability and provokes primacy biases (i.e., the tendency to give more attention to items listed first) (Bowling 2005). Second, from a more technical perspective, statistical ranking models (such as the rank-ordered logit model) assume that rankings were obtained using a particular psychological mechanism (Flynn 2010). For free rankings, however, it remains unclear which statistical model is most appropriate to describe the ranking mechanism. Furthermore, it cannot be ensured that respondents using a self-administered questionnaire judge along repeated best/worst choices, a “ping-pong” method that was shown to produce reliable data (Louviere et al. 2008).

### Visual Analog Scale

To use a visual analog scale (VAS), respondents are asked to specify their level of agreement to a statement by indicating a position along a continuous line between two endpoints. Numerous studies have used VAS responses to derive health-state values in the general population (Björk and Norinder 1999; Cleenput 2010; Devlin et al. 2003; Dolan and Kind 1996; Essink-Bot et al. 1993; Greiner et al. 2003; Johnson and Pickard 2000; Johnson et al. 1998; Leidl and Reitmeir 2011). Krabbe and colleagues (2007) proposed a methodology based on differences in VAS values, where the ranks of pairwise VAS differences are used in a multidimensional scaling analysis to estimate cardinal health-state values. However, other researchers have questioned the validity of VAS data as cardinal values (Bleichrodt and Johannesson 1997; Devlin et al. 2004; van Osch and Stiggelbout 2005) for various reasons. First, VAS tasks in which the top and the bottom endpoints are precisely defined (e.g., death versus perfect health) allow direct comparison between individuals, whereas vague labels such as “worst imaginable” and “best imaginable” hamper an interindividual comparison (Torrance et al. 2001). Second, VAS responses might be affected by a so-called end-aversion bias, the phenomenon of respondents tending to be reluctant to mark positions near the endpoints of the scale (Bleichrodt and Johannesson 1997; Robinson et al. 2001; Torrance et al. 2001). Third, a VAS score for a certain health state may depend on other states presented at the same time (i.e., context bias) (Torrance et al. 2001). Fourth, the accuracy of VAS responses may be influenced by hand preferences and which hand was used (McKechnie and Brodie 2008).
Finally, the orientation of the VAS scale (vertical versus horizontal) itself might affect the shape of the resulting score distribution (e.g., Lundqvist et al. 2009). Taken together, VAS responses therefore should be interpreted on an ordinal scale level only.

Health Valuation: A Social Judgment Perspective

Stiggelbout and de Vogel-Voogt (2008) presented a four-step framework describing respondents’ cognitive processes while valuing health states: perspective/perception of the stimulus, interpretation, judgment, and formation of a manifest response (see also Rehm and Frick 2010). For each step, several mechanisms have been identified, which may affect the final response.

1. Perspective/perception of the stimulus. In a meta-analysis, Dolders and colleagues (2006) reported no significant differences in preferences when patient surveys were compared with those of the general public, whereas a more recent and more extensive meta-analysis by Peeters and Stiggelbout (2010) suggests that patients differ from the general public in their valuations. Frick and colleagues (2012) reported on the importance of social relationships as determinants of health valuation, especially for health professionals. Health states hampering social relationships are judged as more disabling. Ubel and colleagues (2003) described several factors that may contribute to these discrepancies: adaptation effects (i.e., affected patients often adapt physically and emotionally to their health state, resulting in a more positive valuation of the respective state), focusing illusion (i.e., healthy people focus on impaired attributes, largely ignoring unchanged attributes of a certain disease), and contrast effects (i.e., severely ill patients may underestimate the impact of lenient diseases, while healthy people may overestimate this impact). Conducting a survey in the general public will result in a weighted mixture of affected and healthy valuation perspective.

2. Interpretation/primary appraisal. The interpretation of a health state depends on a subject’s values, goals, and beliefs, as well as on the cognitive framing (Kahneman and Tversky 1984) and/or context (Schwarz 1999) of the health-state description.

3. Judgments on health states. Like human judgments in general, these are not formed to fulfill the criteria of an exhaustive information processing. By contrast, they serve as decision rules to govern behavior (e.g., giving an answer in a questionnaire) and follow the principles of parsimony and functional pragmatism rather than coherence and rationality. Stiggelbout and de Vogel-Voogt (2008) identified various sources of biases that might be relevant in the context of health valuation, such as focusing illusion (see step 1), status quo bias (i.e., respondents are more sensitive to changes in their own health state compared with imagined health states), loss aversion (see Tversky and Kahneman 1992), or failure to anticipate negative events (i.e., poor hedonic forecasting). In addition, affects and mood are known to be highly influential during judgmental processing.

4. A deliberate editing of the response. In this last step, for example, a respondent’s attempt to be compatible with perceived norms (e.g., perceived fairness, political correctness, or ethical considerations) further biases a subjective valuation (Rehm and Frick 2010).

Conclusion

Econometric elicitation methods were not originally developed for self-administered questionnaires. Given the many methodological risks of using this data collection mode, TTO, PTO, or SG elicitation methods are not recommended for paper-and-pencil surveys. Understanding the introductory scenarios and autonomously and successively approaching the point of indifference seems too complicated a task for lay respondents. Though VAS scales were developed specifically for self-administered questionnaires, their validity and reliability are too weak to measure the utilities of complex health states on the interval level. Choosing between rankings and PC tasks would mean a tradeoff between economy and validity of the measurement procedure.

Among PCs presented to respondents from the general public, those with the following characteristics seem to be most promising: (1) The number of pairs of health states should be limited (to a number determined by pre-analysis) so that annoyance effects or reactance can mostly be precluded. (2) Cognitive complexity of the health state descriptions should not exceed seven (plus or minus two) judgmental attributes (Miller 1956). However, this does not necessarily mean that health-state descriptions should be limited to seven dimensions or attributes, as respondents tend to
organize redundant information into broader superconcepts. That being said, this ability should also be evaluated prior to the survey. Applying these principles would allow surveys to pose complex vignettes to respondents. (3) To avoid biases due to the direction of a comparison (e.g., A versus B is not the same as B versus A) (Wänke 1996), presentation of health states within one comparison should be randomly balanced. To avoid order effects or carryover effects, factorial design techniques that also preclude repetitive presentations of certain health states (A versus B followed by C versus D and not by A versus C, for instance) should be used in the assignment of comparison tasks to respondents. Complex survey designs like the one proposed here should be randomly balanced. To ensure adequate techniques for statistical analysis (Hox et al. 1991).

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Community Indicators

Assessing the Impact of Alcohol Use on Communities

Andrea Flynn, Ph.D., and Samantha Wells, Ph.D.

Community indicators are used to assess the impact of alcohol on communities. This article reviews the main data sources for community indicators, discusses their strengths and limitations, and discusses indicators used in reference to four main topics relating to alcohol use and problems at the community level: alcohol use, patterns, and problems; alcohol availability; alcohol-related health outcomes/trauma; and alcohol-related crime and enforcement. It also reviews the challenges associated with collecting community indicator data, along with important innovations in the field that have contributed to better knowledge of how to collect and analyze community-level data on the impact of alcohol. Key words: Alcohol use, abuse, and dependence; alcohol burden; problematic alcohol use; harmful drinking; alcohol-related harm; alcohol use patterns; alcohol effects and consequences; alcohol availability; risk factors; environmental impact; crime; community indicators; community monitoring; community epidemiology; data collection; public policy on alcohol

In the United States and other countries around the world, researchers have long been interested in community-level measurement of population health in the form of community indicators. Community indicators are measures that communicate information about a given dimension of a community’s well-being (Beslemé and Mullin 1997). In the United States, the current popularity of community indicators can be traced back to the social-indicators movement of the 1960s and 1970s (see Gross and Straussman 1974; Land and Spilerman 1975; MacRae 1985), which saw growing research attention paid to the measurement of social problems and issues such as divorce, crime, education, and social mobility. Although the social-indicators movement initially focused on issues at the national level, recognition of considerable regional and local variation in the prevalence and causes of social problems led to increased interest in measurement at the local level and, as such, the development of “community indicators.”

Community indicators that assess alcohol use and related harms are of great interest to community stakeholders and researchers. Alcohol use has been identified as a major risk factor for acute and chronic health harms and imparts economic, health, and social costs to individuals, communities, and societies (Rehm et al. 2009). Alcohol intoxication is linked to injury, violence, and traffic crashes (Edwards et al. 1994) and chronic alcohol use increases the risk of liver damage and various cancers, among other health harms (Edwards et al. 1994; Rehm et al. 2003; Room et al. 2005). National surveys have revealed a great deal of variability across different communities in the extent of alcohol use and related harms (Gruenewald et al. 1997). Thus, it may not be practical or fiscally responsible to base local prevention and intervention initiatives on national data that do not reflect patterns or problems within a particular community. Moreover, prevention, treatment, and enforcement activities are commonly enacted at the local level (Gruenewald et al. 1997). Therefore, community-level data on the impact of alcohol use that take into consideration the local economic, social, and policy context are key to guiding local decisionmaking and maximizing the effectiveness of prevention and intervention approaches.

Community indicators have been used extensively for a variety of purposes by both researchers and community stakeholders. For communities, indicator data can be used to inform priority-setting agendas by identifying specific concerns within a community, guide policy and education initiatives, monitor community status on a particular measure over time or in comparison with other communities, and evaluate programs or policies (Beslemé and Mullin 1997; Gabriel 1997; Gruenewald et al. 1997; Mansfield and Wilson 2008; Metzler et al. 2008). Local-level data also are critical for justifying requests for funding and provide a powerful tool for resource allocation within communities (Mansfield and Wilson 2008). For researchers, community indicators are central for improving knowledge of factors influencing community well-being, advancing innovative theoretical models and analytical approaches for use in research and prevention planning (for example, see Holder 1998a), and monitoring and evaluating community prevention/intervention initiatives (Metzler et al. 2008).

This article provides an overview of community indicators of alcohol use and related harms, outlining common sources...
of community indicator data and highlighting the various challenges of collecting data on alcohol at the community level. The literature on community indicators of alcohol use and harms is expansive, spanning a large number of disciplines and extending back for numerous decades. As such, it is beyond the scope of this article to provide a comprehensive review of all the literature and measures pertaining to community indicators on alcohol. Rather, this article provides background information relevant to the use of community indicators in general and in relation to alcohol use and harms, providing examples of some of the most common measures used by alcohol researchers. In addition, the article mentions notable methodological and technological advances that have characterized this field of study over the past few decades, while highlighting the ongoing challenges faced by researchers and community stakeholders interested in assessing alcohol use and alcohol-related harm at the local level. This article draws on extensive knowledge regarding community indicator data on alcohol use and harms that has emerged from key community-based intervention trials, such as the Saving Lives project led by Hingson (Hingson et al. 1996), the Community Trials project led by Holder (Grube 1997; Holder 2000; Holder and Reynolds 1997; Holder and Treno 1997; Holder et al. 1997a, 1997b, 2000; Millar and Gruenewald 1997; Reynolds et al. 1997; Saltz and Stanghetta 1997; Treno and Holder 1997; Voas 1997; Voas et al. 1997), and the Communities Mobilizing for Change on Alcohol (CMCA) project led by Wagenaar (Wagenaar et al. 1994, 1999, 2000a, 2000b). The sections that follow outline some of the main community indicators emerging from this literature and other relevant research in reference to four main topics—alcohol use, patterns, and problems; alcohol availability; alcohol-related health outcomes/trauma; and alcohol-related crime and enforcement.

What Is A Community?

A number of different definitions of community have been proposed and used in the social sciences since the 1800s (for a helpful overview of the various ways in which community has been defined historically, see Holder 1992). Generally speaking, the concept of community implies both geographic and social proximity. Gruenewald and colleagues (1997) define a community as “a contiguous geopolitical area over-seen by a common political structure with common policing and enforcement agencies and common educational and utility systems, and in which individuals are in daily physical contact for the purposes of economic and social exchange” (pp. 10–11). Holder (1992, 1998b) provides a similar definition based on a community-systems perspective and theoretically geared toward the prevention of alcohol problems. Community, in this context, is conceptualized as a dynamic, complex, and adaptive system consisting of “a set or sets of persons engaged in shared socio-cultural-politico-economic processes” (Holder 1998b, p. 12). This definition informs the theoretical premise that reducing alcohol use and alcohol-related problems requires a focus on the community system and structural factors influencing alcohol use rather than on individual-level treatment and prevention (Holder 1998b; Holder et al. 2005; Treno and Lee 2002).

Putting these definitions of community into practice when attempting to define and use community indicators is not without its challenges and has direct implications for data collection. When defining the boundaries of the community for the purpose of generating community indicators, it is necessary to consider data availability, methodological requirements of research (i.e., having sufficient cases for meaningful analyses), the catchment area in terms of service provision, other geographic boundaries according to which data are routinely collected by a community, and local stakeholder perspectives on their understanding of community (Gruenewald et al. 1997). These considerations do not always coincide (e.g., available data may not match the catchment area of interest to community stakeholders), making it necessary to weigh the relative importance of these factors when defining the boundaries of the community under study (Gruenewald et al. 1997).

Data Sources for Community Indicators on Alcohol

Community indicators relating to alcohol use and harms are typically gleaned from two main types of data sources: (1) archival sources collected for purposes other than addressing research questions on the impact of alcohol on communities (e.g., data from police and hospital records; crash data from traffic safety databases); and (2) primary data collected by researchers for the purpose of assessing, understanding, and addressing alcohol use and related harms. These different sources of data have inherent advantages and disadvantages in terms of their utility for assessing the community-level impact of alcohol use.

Archival Data

Archival data are an important source of community indicator data. Examples of these archival data sources include administrative and surveillance databases maintained by local city departments, community organizations, municipal/national agencies, schools, hospitals, and police/law enforcement departments, in addition to larger health data–recording systems and traffic crash databases (e.g., the Healthcare Cost and Utilization Project [HCUP] databases and the Fatality Analysis Reporting System [FARS]). A wide range of indicators produced from archival data are used to assess various alcohol-related issues and harms at the community level (examples and discussion of common indicators are presented in the section Community Indicators on Alcohol and Alcohol-Related Harm; see also the table).

A main benefit of using archival sources to produce community indicators is that they can be a cost-effective means of documenting alcohol use and harms, offering a
large volume of retrospective data. In addition, unlike many of the constructs and measures used in social and epidemiological research, archival data often result in indicators that are straightforward, understandable, and of interest to the community, making them easier to use in community planning (Gabriel 1997; Gruenewald et al. 1997; Mansfield and Wilson 2008). Despite these advantages, there are several limitations associated with using archival data to assess alcohol use/harms in a community. By definition, these data are not gathered for research purposes and thus raise concerns relating to both reliability and validity. Most notably, archival data are subject to various sources of measurement error consequent to the fact that they are not collected according to the systematic and rigorous procedures that characterize social and epidemiological research. In addition, for some measures, the involvement of alcohol may not be explicitly identified. For instance, hospital staff and police typically do not systematically record data on alcohol consumption as part of routine practice (Brinkman et al. 2001; Gruenewald et al. 1997; Stockwell et al. 2000). When alcohol data are recorded in community settings, they may be collected in an inconsistent manner, influenced by subjective judgments and local practices (Brinkman et al. 2001). These limitations affect the extent to which researchers can confidently use existing data such as hospital records or police data to assess alcohol involvement in injury or crime. Moreover, access to such data requires cooperation of local community agencies and/or municipal or regional departments, which may not always be possible.

Another important caveat relates to the use of archival data for conducting community comparisons. Differences across communities in policies and data recording systems (Gruenewald et al. 1997; Brinkman et al. 2001; Stockwell et al. 2000) can make it difficult to conduct comparisons across communities. For example, when using arrest data on alcohol-related crime such as public intoxication or disorderly conduct, the indicator will reflect the definition used by the police department (itself dependent on local or regional statutes) as well as on local enforcement capacity and practices, including levels of police discretion. Thus, data on arrests may not be directly comparable across communities, even if the communities themselves are well matched on demographic or other important baseline measures (Gruenewald et al. 1997). Differences in recording systems or policies also present problems for researchers interested in examining patterns over time within communities. For example, variation over time in the number of alcohol-related arrests may reflect changes in enforcement, recording practices, or policies rather than true variations in alcohol-related crime (Gruenewald et al. 1997).

Events with low levels of incidence present another challenge relating to use of archival data for assessing the impact of alcohol on communities. For instance, although alcohol-related morbidity and mortality are of great interest to communities, these types of indicators may be difficult to provide at the community level, particularly for smaller communities, because of their relatively low baseline rate. Moreover, in the case of health-related indicators, the problem of low incidence is compounded by the fact that most health-related harms associated with alcohol use are only partially attributable to alcohol (Rehm et al. 2003). Although researchers have developed approaches for estimating the proportion of a given outcome that is attributable to alcohol as a specific risk factor (i.e., the attributable fraction, AF) (see English et al. 1995; Martin et al. 2010; Rehm et al. 2003; Single et al. 1999; Stockwell et al. 2000; World Health Organization [WHO] 2000), these types of analyses require a large volume of data and are typically only conducted at higher levels of aggregation (e.g., State, Federal).

**Primary Data**

Given that archival data often are unavailable or insufficient to assess alcohol use and harm at the community level, primary data are collected to enhance knowledge of the community-level impact of alcohol use (Gruenewald et al. 1997; Stockwell et al. 2000). Population or subpopulation surveys are the predominant source of primary data used to produce alcohol-related community indicators. Surveys offer the advantage of allowing researchers to define the constructs of interest and use psychometrically sound measures, including measures that have been used in other community-level, State, or Federal surveys, thereby facilitating comparisons. Surveys also permit the collection of self-report data that cannot be gleaned from archival data, such as individual-level alcohol use patterns; underage access to alcohol; and beliefs, attitudes, and perceptions surrounding alcohol. These data allow for individual and group-level risk factors to be determined and permit analyses on subpopulations of interest, such as adolescents or young adults (Gruenewald et al. 1997; Stockwell et al. 2000).

In some instances, it may be possible to extract community-level data from surveys conducted at higher levels of aggregation (e.g., State or national surveys). However, the time frames of State and national surveys often do not meet community or research needs. For example, timing of data collection is an essential factor when monitoring the impact of local policy changes or community initiatives, which may not coincide with national survey data collection (Mansfield and Wilson 2008). Moreover, when attempting to glean information from national or State-level surveys, sample sizes for smaller communities often are insufficient to permit valid conclusions about specific communities or population subgroups within a community (Gruenewald et al. 1997; Mansfield and Wilson 2008; Stockwell et al. 2000). For these reasons, surveys implemented at the community level are key to developing local indicators of alcohol use and harms. Surveys have been widely used in community-based research projects, including both general population surveys and surveys of particular population groups, such as college students (discussed below in Community Indicators on Alcohol and Alcohol-Related Harm; see also the table).

When conducting surveys to produce community indicators, it is necessary to consider the limitations of the survey method. Recent evidence suggests that population surveys...
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<th>Indicator Category</th>
<th>Examples of Indicators</th>
<th>Indicators from Archival Sources</th>
<th>Strengths</th>
<th>Limitations</th>
<th>Indicators from Primary Data Sources</th>
<th>Examples of Indicators</th>
<th>Strengths</th>
<th>Limitations</th>
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<tr>
<td>Alcohol use, patterns and problems</td>
<td>Per capita alcohol consumption</td>
<td>Generated from available sales data</td>
<td>Does not capture patterns of access or use</td>
<td>Self-reported drinking behavior and problems (youth, adults)</td>
<td>Offer individual- and group-level data unavailable from archival sources that can be aggregated to community level, including drinking pattern</td>
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<td>Excludes “surrogate” alcohols (homemade, illegal, alcohol not intended for consumption)</td>
<td>- age at first use</td>
<td>Ability to implement scientifically valid and reliable measures employed in other communities and other levels of aggregation (State, Federal) for comparison purposes</td>
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<td>Data may not be available at the local level (depends on catchment area of research and definition of “community”)</td>
<td>- drinking prevalence</td>
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<td>- heavy episodic drinking (i.e., binge drinking)</td>
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<td>- hazardous or harmful drinking</td>
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<td>Alcohol availability</td>
<td>Formal access - number of active outlet licenses per 100,000 population - concentration/spatial distribution of outlets - excise taxes on alcoholic beverages - price of alcoholic beverages</td>
<td>Data on outlet licenses are generally maintained with good geographic specificity by Alcohol Control Boards</td>
<td>Data do not capture sales to minors</td>
<td>Alcohol purchase attempts at alcohol outlets by pseudo-underage customers</td>
<td>Capture events not visible in archival data and not affected by self-report biases</td>
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<td>Data do not capture social access</td>
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<td>Useful in evaluations of strategies to reduce youth access to alcohol</td>
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<td>Data do not capture differences between outlets with respect to sales (e.g., small outlets versus large outlets)</td>
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<td>Community estimates may be affected by migratory patterns and purchases in communities of non-residence</td>
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<td>Price data difficult to obtain</td>
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<td>General limitations of surveys and self-report measures</td>
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<td>Social access</td>
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<td>- high cost of surveys</td>
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<td>- possible biases (selection bias, social desirability bias, recall bias, coverage bias)</td>
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<td>Indicator Category</td>
<td>Examples of Indicators</td>
<td>Strengths</td>
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<td>Alcohol-related health and trauma</td>
<td>Hospital discharge data</td>
<td>Capture serious health/trauma outcomes – strong impact for communities</td>
<td>Low base rates of mortality from alcohol at the community level</td>
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<td>- rates of direct alcohol mortality or morbidity: alcohol cardiomyopathy, alcohol cirrhosis of liver, alcoholic psychoses, accidental ethyl alcohol poisoning, etc.</td>
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<td>Multiple causes of death often poorly recorded in archival data</td>
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<td>- rates of indirectly-related alcohol deaths: certain malignant tumors, cirrhosis, pancreatitis, etc.</td>
<td>Nighttime emergency department (ED) presentations and nighttime single vehicle traffic crashes are reliable surrogates of alcohol-involved trauma</td>
<td>Proportion of mortality/morbidity events attributable to alcohol difficult to estimate at the community level</td>
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<td>Hospital/ED cases capture only the most severe cases</td>
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<td>Blood alcohol concentrations (BAC) not routinely recorded in hospital/emergency settings</td>
<td>BAC not always measured in injury-producing/fatal crashes</td>
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<td>Fatal crashes rare at community level</td>
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<td>Alcohol-related crime</td>
<td>Calls to police for nighttime assaults</td>
<td>If cooperation can be obtained, arrest or EMS records are a cost-effective source of data that is meaningful to community members</td>
<td>Heavily dependent on police enforcement and accuracy in recording</td>
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<td>Calls to emergency medical services for alcohol-related injury</td>
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<td>Difficult to determine if changes are due to changes in police enforcement, valid changes in crime, or prevention programs</td>
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<td>Calls to police for public drunkenness or disorderly contact</td>
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<td>In community prevention trials or when communities are interested in comparisons, different statutes or operational policies affect ability to compare communities</td>
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<td>Arrest rates for driving under the influence</td>
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<td>Arrests represent only a proportion of offenses – underestimates harm</td>
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<td>Arrest rates for nighttime assaults</td>
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<td>Alcohol-related arrests as a percentage of total arrests</td>
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### General strengths of surveys and self-report data
- Self-reported health harms and trauma experiences related to alcohol
- ED surveys
- BAC measurement
- Self reported alcohol consumption prior to ED presentation

### General limitations of self-report data and surveys
- Difficulty obtaining permission for ED surveys

### General limitations of self-report data
- Self-reported alcohol consumption shown to be valid measure of alcohol use

### General limitations of self-report data and surveys
- Challenges of implementing roadside surveys
- Self-reported crime captures incidents not reported to police
- BAC provides an objective measure of alcohol consumption

### General limitations of self-report data and surveys
- Can be difficult to find appropriate comparison communities

### General limitations of self-report data and surveys
- Can be difficult to obtain police cooperation
- Generally not random (not representative of community)
can underestimate the prevalence of alcohol use and associated harms because of selection bias, response bias, and coverage bias (e.g., exclusion of homeless people) (Shield and Rehm 2012; see also Curtin et al. 2005; Dillman et al. 2002; Kempf and Remington 2007). The growth in use of voicemail, caller ID, cell phones, and do-not-call lists, along with a growing aversion to aggressive telemarketing (Galesic et al. 2006), have contributed to a notable decline in telephone survey response rates (Dillman et al. 2002; Hartge 1999; Kempf and Remington 2007; see also Galea and Tracy 2007). Young people may be particularly underrepresented in population surveys, given their high reliance on cell phones and nonuse of landlines (Blumberg et al. 2007). Large-scale surveys can also be expensive and time consuming to implement.

When collecting primary data on alcohol use and harms, it is also important to consider the limitations of self-report data on drinking behavior and harms associated with drinking. Although self-report data on alcohol use generally are believed to be adequately valid and reliable and are widely used in social and epidemiological research, they have been found to be susceptible to recall error as well as intentional distortion related in part to social desirability (Del Boca and Darkes 2003).

Despite these limitations, surveys are key to answering specific questions about alcohol use and harms in the absence of suitable archival data and are central for cross-validating data gleaned from other sources. Moreover, extensive work on conducting surveys as part of community prevention trials has led to important methodological and statistical innovations, producing advanced knowledge of how to design and analyze surveys better (see Murray 1998; Murray and Short 1995, 1996; Murray et al. 2004).

In addition to surveys, other forms of primary data used to produce community indicators include pseudo-patron studies designed to assess sales of alcohol to individuals appearing underage in both off-premise and on-premise alcohol outlets (see, for example, Freisthler et al. 2003; Saltz and Stanghetta 1997; Toomey et al. 2008; Treno et al. 2006; Wågenaar et al. 2000a) and roadside breath testing to assess drinking and driving (e.g., McCartt et al. 2009; Roether and Vos 1998). These methods and their strengths and limitations are discussed in later sections on alcohol availability and crime/enforcement, respectively.

Overall, although primary data, particularly surveys, allow for the use of psychometrically sound measures, they suffer from potential biases that researchers must take into account when assessing the impact of alcohol use on a community. Alternatively, archival data sources can provide useful data on alcohol’s effects on local communities but require careful interpretation and application and do not always allow researchers to answer questions of interest. Each data source thus offers unique strengths and limitations, such that triangulation of both types of data is a common approach taken by alcohol researchers when assessing the impact of alcohol on communities.

### Community Indicators on Alcohol and Alcohol-Related Harm

Table 1 provides a summary of common community indicators of alcohol use and related harms measured in community-based research. These indicators are organized into four broad areas: alcohol use, patterns, and problems; alcohol availability; alcohol-related health outcomes/trauma; and alcohol-related crime/enforcement. Although this table does not provide an exhaustive list of all possible measures used to assess alcohol use and alcohol-related harm at the community level, it provides common measures used in community research (see Saltz et al. 1992). For each category, examples of indicators produced using archival and primary data sources are provided, and general strengths and limitations associated with these data are noted.

### Alcohol Use, Patterns, and Problems

At the community level, indicators of alcohol use, patterns, and problems commonly are produced from individual-level self-report (i.e., survey) data. Existing community-based studies have examined a wide range of self-report measures of alcohol use, including, for example, lifetime drinking, drinking frequency, heavy episodic drinking (or binge drinking) and hazardous or harmful drinking, alcohol problems, and alcohol dependence (see Dent et al. 2005; Flewelling et al. 2005; Harrison et al. 2000; Hawkins et al. 2009; Perry et al. 1996, 2000, 2002; Saltz et al. 2009, 2010; Spera et al. 2010; Wågenaar et al. 2006; see table 1). It is beyond the scope of this article to discuss the many different instruments used and all of the methodological challenges associated with measuring self-reported drinking and problems. Choice in how to measure indicators of use, patterns, and problems will depend on the research question being asked and the population under examination. The strengths and limitations of various specific measures of alcohol consumption have been discussed extensively in the literature (see Dawson 2003; Gmel et al. 2006a; Graham et al. 2004; Greenfield 2000; Rehm 1998; Rehm et al. 1999), and recommendations for measurement have been put forward elsewhere (see Dawson and Room 2000).

Drinking behavior among youth often is of particular interest to both researchers and communities. Evidence suggests that youth are more likely than adults to engage in risky patterns of drinking (Adlaf et al. 2005) and to experience harms from drinking, including harms to brain development, physical health, financial well-being, and social life (Adlaf et al. 2005; Kolbe et al. 1993; Toumbourou et al. 2007; White and Swartzwelder 2004). Moreover, drinking at a young age can become an ingrained pattern of behavior, with youth who engage in risky drinking being more likely to exhibit problem drinking later in life (Jeffers et al. 2005). For these reasons, measuring alcohol use and alcohol-related problems among youth often is prioritized in prevention and early-intervention initiatives designed to reduce harm from...
alcohol at both the individual and community levels (see DeJong et al. 2009; Nelson et al. 2010). The well-known prevention initiative CMCA (Wagenaar et al. 1994, 1999, 2000a, b) is notable for its focus on community-level strategies for reducing alcohol use and problems among youth and its development of indicators of alcohol use and harms to evaluate program effectiveness.

Surveys on youth drinking have commonly captured these populations in their educational environments, including elementary, high school, and college or university settings. The priority of addressing alcohol use among college students is well evidenced by the NIAAA's Rapid Response to College Drinking Problems initiative, which produced recommendations for reducing heavy drinking by this subgroup (see DeJong et al. 2009; Nelson et al. 2010). Alcohol use, patterns, and problems have been measured in the implementation and evaluation of alcohol prevention trials in school and college settings (see reviews by Saltz 2011 for college-based prevention approaches and Stigler et al. 2011 for elementary and high school programs). Examples of measures of alcohol use and problems among college and school-age students include self-reported alcohol use (i.e., measures of frequency of drinking, drinking patterns, and binge drinking) (Flewelling et al. 2005; Harrison 2000; Hawkins et al. 2009; Perry et al. 1996, 2000, 2002; Saltz et al. 2009, 2010), the incidence and likelihood of intoxication at off-campus drinking establishments (Saltz et al. 2010), age of onset of drinking (Hawkins et al. 2009), and perceptions and experiences of negative consequences associated with drinking (Flewelling et al. 2005; Saltz et al. 2009, 2010). Significantly, although surveys of college and university students may provide communities with estimates of alcohol use, patterns, and problems among this segment of the population, these surveys are inherently limited to the sampling frame of youth attending these institutions. As a result, they fail to capture youth from the broader community not attending educational institutions and thus cannot offer community prevalence data for that age range.

With respect to archival data on alcohol use, this type of information is less commonly available at the community level compared with higher levels of aggregation. Most notable in this regard is the use of sales data to examine per capita alcohol consumption. WHO (2000) has recommended that alcohol use among populations be monitored using reliable estimates of per capita alcohol consumption derived from alcohol sales data, in addition to monitoring through population surveys of alcohol use. Sales data commonly have been used at the State, regional, and Federal levels to examine the link between per capita alcohol consumption and various health harms, including suicide (Kerr et al. 2011b, Landberg 2009), mortality and morbidity (Kerr et al. 2011a; Nordstrom and Ramstedt 2005; Polednak 2012), and traffic crashes (Gruenewald and Ponicki 1995). These types of analyses, however, generally are restricted to large populations (Dawson 2003) and thus are less applicable to alcohol researchers interested in community indicators (i.e., measures below the State level of aggregation), in part as a result of the low base rate of harms at the community level and in part from challenges associated with obtaining sales data at the community level compared with the State level.

### Availability

Measuring the availability of alcohol at the community level is essential for assessing the impact of policies designed to reduce alcohol use and alcohol-related harms (see Babor et al. 2003). Availability commonly is measured in terms of commercial access (including alcohol outlet density, days and hours of sales, and price of alcohol) as well as social access (i.e., informal sources of alcohol, such as peers).

With respect to commercial access, although the evidence on the effects of limiting alcohol outlet density on alcohol consumption is somewhat mixed (see Livingston et al. 2007), studies generally have found significant positive relationships between alcohol outlet density and a range of problems at the community level, including rates of violence, drinking and driving, motor vehicle accidents, medical harms, and crime (Britt et al. 2005; Campbell et al. 2009; Gruenewald and Remer 2006; Gruenewald et al. 2006; Livingston et al. 2007; Toomey et al. 2012). Evidence also suggests a positive relationship between days (Middleton et al. 2010) and hours (Hahn et al. 2010) of sale and alcohol consumption and alcohol-related harms (see also Edwards et al. 1994). Alcohol prices and taxes are inversely related to alcohol consumption and heavy drinking (Chaloupka et al. 2002; Edwards et al. 1994; Osterberg 2004; Wagenaar et al. 2009), although the extent of the impact of price changes depends to some extent on cultural context (i.e., drinking norms) and prevailing social and economic circumstances, among other factors (Osterberg 2004; see also Babor et al. 2003). Researchers have used indicators of commercial access to evaluate whether changes in State policies have an impact on alcohol use/ problems in communities (see Babor et al. 2003; Edwards et al. 1994; Hahn et al. 2010; Middleton et al. 2010).

Community indicators of economic availability commonly are produced using archival data sources, including alcohol price and tax (excise and sales) data from State departments and alcohol-control boards, although the quality of these data and their utility for research at the community level varies substantially across States (Gruenewald et al. 1997). Archival data on retail alcohol prices are difficult to obtain at the State level, and even more so at the community level. Evidence suggests that available data are prone to substantial measurement error (Young and Bielinska-Kwapisz 2003), leading many researchers to rely on tax data instead. When making comparisons across communities or over time, researchers generally also prefer to use tax rates over price data to avoid conflating price differences with differing tax rates across space and over time. Liquor licensing information from alcohol-control boards commonly is used to generate indicators of commercial availability—namely, number of outlets/population rates and concentration of on- and off-premise outlets (Sherman et al. 1996; see also Gruenewald et al. 1997). However, counts of active licenses represent only...
an indirect measure of alcohol availability and can underestimate alcohol sales (Gruenewald et al. 1992). Geographic Information System (GIS) mapping has emerged as an innovative means of generating community indicators of outlet density (including off- and on-premise outlets) and to examine alcohol outlet density and locations in relation to alcohol-related problems, such as assaults and sale of alcohol to minors (see Gruenewald et al. 2002; Millar and Gruenewald et al. 1997).

One major caveat relating to measures of commercial access to alcohol is that archival data obscure who is making purchases, who is consuming the alcohol purchased, and how (in what patterns) the alcohol is being consumed. Therefore, important information about risky drinking behavior (i.e., binge drinking) and populations who engage in such behavior remains unknown from data on alcohol availability. This limitation is particularly salient for measuring drinking among youth, who commonly obtain alcohol from social rather than commercial sources (see Wagenaar et al. 1993).

In light of this limitation, and the fact that early prevention of alcohol use and alcohol-related problems is often a high priority for communities and researchers, other data collection strategies have been implemented to measure access to alcohol among youth. Access surveys involving pseudo-underage youth purchase attempts have produced indicators of youth commercial access, often as part of the evaluation of community prevention initiatives (see Chen et al. 2010; Grube 1997; McCartt et al. 2009; Paschall et al. 2007; Perry et al. 1996, 2000, 2002; Toomey et al. 2008; Wagenaar et al. 1994, 1999, 2000a, b). Self-reported social access to alcohol has also been measured in school or community surveys of youth, with participants asked to report on sources from which they obtain alcohol (i.e., commercial [on- or off-premise outlets] versus social [friends, family, etc.] sources) (see Dent et al. 2005; Harrison et al. 2000; Hearst et al. 2007; Jones-Webb et al. 1997; Wagenaar et al. 1994). Some studies have also examined perceived availability of alcohol among youth (Flewelling et al. 2005; Perry et al. 1996, 2000, 2002; Treno et al. 2008).

Health Outcomes/Trauma

As stated previously, evidence reveals a strong and consistent association between alcohol consumption and a variety of negative health outcomes, including morbidity, early mortality, and increased risk of trauma such as burns, falls, drowning, and injury from interpersonal violence (Cherpitel 1995; Gmel et al. 2006b; Rehm et al. 2003, 2006; Treno et al. 1997). Collectively, alcohol-related health harms and traumas impose notable demands on local emergency and hospital services. Documenting alcohol-related morbidity, mortality, and trauma is thus often a priority for communities and researchers, with such research informing initiatives geared toward preventing alcohol-related harm and efforts to reduce health costs.

Both archival and primary data have been used to produce community indicators relating to fatal and nonfatal alcohol-involved health harms. Data sources and types of indicators emerging from these data include (1) hospital data, used to produce indicators of hospitalizations and emergency department (ED) visits associated with acute or chronic alcohol use; (2) traffic fatality data, used to estimate alcohol involvement in crashes; and (3) household or subpopulation surveys, used to generate indicators from self-reported data on alcohol-involved injuries (including violence). As shown in table 1, each of these data sources has strengths and limitations pertaining to their utility for producing community indicators on alcohol-related harms.

Hospital and ED Data. Archival hospital data allow for documentation of cases of alcohol-related health outcomes and trauma requiring urgent or emergent care. Such data can provide powerful information for use by communities (e.g., in educational or prevention campaigns) because of their severity and corresponding psychological impact (Stockwell et al. 2000). Despite this appeal, notable challenges exist to using archival data to produce community indicators on health outcomes and trauma associated with alcohol. First, as stated above, one of the major caveats with measuring alcohol-related mortality and morbidity at the community level is the rarity of cases (Giesbrecht et al. 1989; Stockwell et al. 2000), meaning that there may be insufficient numbers for meaningful analysis at the community level. Second, it often is quite difficult to obtain access to hospital or ED data within communities, particularly data of reasonable quality for developing valid and reliable estimates. Third, it is often challenging or impossible to determine the extent of alcohol involvement in health outcomes. As previously noted, many chronic health harms associated with alcohol, including those leading to hospitalization and mortality, are only partially attributable to this risk factor (Rehm et al. 2003). In terms of emergency cases, archival data frequently do not capture alcohol involvement (Giesbrecht et al. 1989; Stockwell et al. 2000). Blood alcohol concentration (BAC) is not routinely assessed in hospitals or urgent-care centers in relation to traumatic presentations, given that staff generally are operating under time and resource constraints that preclude systematic testing for alcohol use. Staff also may be hesitant to make conclusions about intoxication because of insurance and liability concerns (Giesbrecht et al. 1989, 1997; Stockwell et al. 2000; Treno and Holder 1997). As a result, archival data of emergency cases likely underestimate the role of alcohol in trauma requiring emergent care. In cases where BAC is recorded, determining the role of alcohol in a traumatic event is complicated by time elapsed since the incident and by alcohol consumed after the incident (Young et al. 2004).

In the face of challenges associated with lack of documentation of alcohol involvement in archival data, researchers commonly turn to surrogate measures of alcohol-related trauma. Such measures have been well studied using international data. For instance, Young and colleagues (2004) found that being male, unmarried, younger than age 45, and presenting at EDs in the late night or early morning hours on
Traffic Fatality Data. Alcohol-related traffic fatalities are an important form of trauma in the community-indicator literature on alcohol-related harm. Consistent evidence confirms that alcohol is a leading cause of traffic crashes, particularly those resulting in fatal and nonfatal injuries (Hingson and Winter 2003). Research has demonstrated that the relative risk of fatal injury and fatal crash involvement rises with increasing driver BAC (see the classic Grand Rapids study by Borkenstein et al. [1974] and subsequent studies by Hurst [1973]; Krüger and Vollrath [2004]; Mathijsen and Houwing [2005]; Mayhew et al. [1986]; McCarroll and Haddon [1962]; Perrine et al. [1971]; Zador [1991]; and Zador et al. [2000]). Relative risk data such as these have been widely used to support alcohol safety legislation, including the lowering of BAC driving limits (see review by Mann et al. 2001).

The FARS (formerly the Fatal Accident Reporting System) (see http://www.nhtsa.gov/FARS), initially established in 1975, is a reliable database of all fatal crashes in the United States and includes the BACs of drivers involved in fatal crashes. When chemical tests of driver BACs are not performed in fatal crashes, FARS provides imputed data (see Subramanian 2002). FARS data can be disaggregated to the level of the county (see Voas et al. 1998; Williams 2006). Studies using FARS or State traffic safety department databases have generated indicators of various levels of driver BAC associated with traffic fatalities (e.g., Hingson et al. 2005, et al. 2006; Wagenaar and Wolfson 1995). However, fatal crashes are relatively rare events (Voas et al. 1997), and thus aggregation of events over a long time period may be needed to produce sufficient cases for analysis at the community level (e.g., see Wagenaar et al. 2000a).

Researchers commonly also use fatal single-vehicle nighttime crashes as a surrogate for alcohol-involved traffic fatalities, which can be a useful strategy when data on alcohol involvement in crashes are unavailable for the community of interest or too few cases have been documented. These data have been shown to be a reliable proxy for alcohol-related fatalities. They often are available from local or State sources (e.g., police departments or departments of transportation) and, depending on the size of the community, may occur in sufficient numbers for analysis (see Hingson et al. 1996; Roeppe and Voas 1998; Treno et al. 2006; Wagenaar and Holder 1991; Wagenaar et al. 2000a, 2006). Nevertheless, caution is warranted when interpreting traffic crash data, particularly in the absence of BAC data, given the myriad of other factors that stand to be involved in crashes, including road conditions, speeding, and use of seat belts. The use of multiple data sources for triangulation of data (Gruenewald et al. 1997) can help overcome the limitations of any one measure of alcohol-involved vehicle crashes.

Population Survey Data. Population or community surveys are used to measure self-reported alcohol-related health outcomes and trauma. An advantage of these surveys is that they can detect events not resulting in fatalities or hospital admissions (Gruenewald et al. 1997). These data are thus useful for documenting less severe cases, which are more common than fatal or near-fatal cases. However, the number of self-reported events (e.g., injury) may still be insufficient for analysis, particularly in small communities. General limitations of population surveys apply to these data, including the cost and time required to conduct them, as well as reporting and coverage biases that may result in underestimates of alcohol-related harms.
Crime/Enforcement

Both primary and archival data sources have been used to generate measures of alcohol-related crime in communities. At the community level, household, telephone, and school surveys have been conducted to measure various self-reported crimes, including drinking under the influence (DUI) (e.g., Clapp et al. 2005; Saltz et al. 2009; Wagenaar et al. 2006), underage alcohol purchases (e.g., Harrison et al. 2000), alcohol-related violence (Greenfield and Weisner 1995), and public drunkenness (Greenfield and Weisner 1995). The general strengths and limitations of surveys and self-report measures of alcohol use have been discussed previously. Therefore, this section will focus on roadside surveys and arrest data.

Roadside surveys involve stopping motorists at roadside checkpoints for the purpose of collecting breath alcohol measurements. Two key purposes of roadside surveys are to track drinking and driving trends and to evaluate alcohol safety programs (Lange et al. 1999; Lestina et al. 1999). The majority of roadside studies conducted to track trends in drinking and driving have occurred at the national level (e.g., in the United States, Canada, Britain, Germany, Sweden, Norway, Belgium, and the Netherlands) (see Lacey et al. 2008; Lestina et al. 1999; Lund and Wolfe 1991; Voas et al. 1998; Wolfe 1974 for information on the U.S. National Roadside Surveys). These national surveys typically do not provide sufficient data at the community level for assessment of local drinking and driving because of the exclusion of smaller communities and/or roadways with low daily traffic counts (Voas et al. 1998). At the community level, roadside surveys primarily have been used in the evaluation of community prevention trials (e.g., McCartt et al. 2009; Rooper and Voas 1998). They allow researchers to assess changes in drinking-and-driving behavior in relation to prevention campaigns when fatality and crash data are unavailable (Rooper and Voas 1998). In instances where fatality and crash data are available, roadside survey data may still be useful to confirm that changes in crash data reflect valid changes in drinking-and-driving behavior rather than other changes not related to alcohol consumption (e.g., roadway improvements) (Rooper and Voas 1998).

Two main strategies are used to implement roadside surveys at the community level: (1) “piggybacking” on existing police sobriety check points; and (2) using roadside check points dedicated entirely to research. In both instances, cooperation of local police is imperative, which may create a challenge in communities lacking widespread support for the research (Howard and Barofsky 1992). In addition to the notable cost associated with conducting roadside surveys, there are several limitations and challenges associated with this method of data collection (Lestina et al. 1999). For example, many high-BAC drivers are able to avoid roadside survey check points by driving alternate routes, resulting in underestimates of local levels of drinking and driving (Lestina et al. 1999). Drivers also may refuse to provide a breath sample, and these people may be likely to have higher BACs than those who consent to a breath test (Lestina et al. 1999). Conversely, overestimates of impaired driving may occur if roadways characterized by high volumes of alcohol-related crashes are targeted for surveys (Lestina et al. 1999).

In evaluations of alcohol-safety programs (and other alcohol interventions), it is necessary to compare the intervention community with a comparison community in which the program was not implemented to determine whether changes in drinking and driving can be attributed to the intervention. However, finding adequate comparison sites can be a challenge, given the need for a community with similar population characteristics and policies and the fact that comparison (“non-experimental”) communities may have their own campaigns to reduce drinking and driving (see Voas 1997).

Arrest data on DUI as well as other alcohol-related offenses also represent valuable indicators for communities. Numerous researchers have used archival police and justice records to produce community indicators of alcohol-related crimes, including DUI, liquor law violations, assault, public drunkenness, and disorderly conduct (e.g., Breen et al. 2011; Duncan et al. 2002; Sherman et al. 1996; Treno et al. 2006; Wagenaar et al. 2000a) (see Table 1). When using archival data to assess levels of alcohol-related crime, it is important to recognize that such arrests represent only offenses brought to the attention of the police that they have acted upon. Some criminal events (e.g., violent crime) are not commonly reported to the police, or there may be insufficient cause for police to file an arrest report (Brinkman et al. 2001). Moreover, by definition, arrest data are dependent on local and State statutes and also are highly sensitive to enforcement capacity and practices as well as operational changes and recording practices, including police discretion (Gruenewald et al. 1997). These factors are thus critical to consider when making comparisons over time or across communities. As noted previously, changes in alcohol-related arrests can represent changes in actual crime, changes in enforcement or recording practices, or changes in policies and laws (Gruenewald et al. 1997). In some instances, confounding variables (such as police discretion in making arrests) are difficult if not impossible to measure.

Another problem with police data is that for many types of crime (e.g., violence), police do not formally measure alcohol involvement (i.e., through a breath test). Although some research has measured alcohol-involved crime through archival records of cases that police have flagged for alcohol involvement (Wagenaar et al. 2000a), these data are unlikely to be systematic and rely in large part on police discretion (see discussion by Brinkman et al. 2001). To partially address such concerns, surrogate measures have been used to produce indicators of alcohol-related crime from archival data. For example, nighttime assaults have been used as a proxy for alcohol-related violence, given that temporal data are likely to be recorded in police records and violent assaults during nighttime hours have a high likelihood of being alcohol related (Brinkman et al. 2001).
Indicators of enforcement are also related to measurement of alcohol-related crime at the community level. Some investigators have measured enforcement activities in community-based research projects, often for the purpose of evaluating policy changes or prevention efforts (e.g., Grube 1997; McCart et al. 2009; Voas, Holder and Gruenewald 1997; see also Wagenaar and Wolfson 1995) (see Table 1). Indicators of enforcement can provide communities with data on enforcement capacity and, if tracked over time, can allow for an assessment of the impact of enforcement on reducing alcohol-related crime.

Conclusion

Measuring alcohol use and harm in communities is complex and requires researchers to make choices and find creative ways of assessing the local-level impact of alcohol. The data source and indicator used will depend on data availability, the purpose of the research (e.g., to provide a community with descriptive data versus evaluation of an intervention), and, in many cases, community support for the research to facilitate access to archival data or cooperation in primary data collection efforts.

Whether using archival or primary data to produce community indicators, it is important for both researchers and community stakeholders to be aware of the strengths and potential limitations of the data. They must also recognize the value of combining data from multiple sources when making conclusions about the impact of alcohol on communities. Indeed, many community-based projects have relied on both primary and archival data to assess alcohol use and harms in communities and to evaluate the impact of intervention initiatives. Triangulation of indicators is key for validating measures and thus drawing accurate conclusions about research findings.

Despite the limitations and challenges associated with assessing alcohol use and alcohol-related harms at the community level, many significant advances have been made in the field, including important advances in statistical methods (e.g., Murray 1998; Murray and Short 1995, 1996; Murray et al. 2004), refinement of surrogate measures (e.g., Treno et al. 1994, 1996, 1997), and spatial analysis (e.g., Gruenewald et al. 2002; Millar and Gruenewald 1997). Another example of an innovative approach that currently is being employed to develop community indicators involves use of a mobile research laboratory to collect social, epidemiological, and biological data in diverse communities in the province of Ontario, Canada. Led by a multidisciplinary team of researchers, this project involves collection of local data and the development of a community indicator database relating to mental health and addictions in participating communities, including indicators of alcohol use and harms (see Wells et al. 2011).

Building on these types of innovations and the rich history of social indicators in the United States, a number of communities recently have sought to develop comprehensive community indicator systems consisting of data on a range of factors (e.g., social, economic, and environmental) to allow a detailed examination of influences on community well-being (Beslme and Millin 1997; Ramos and Jones 2005). National initiatives such as the 2008 Community Health Status Indicators (CHSI) project (see Heitgerd et al. 2008; Metzler et al. 2008; see also www.communityhealth.hhs.gov), the Community Assessment Initiative (http://www.cdc.gov/ai/index.html), and the National Neighborhood Indicators Partnership (http://www.neighborhoodindicators.org), for example, have sought to improve access to local data and inform use of data in planning efforts and evaluation of health policies and interventions. At the international level, the Community Indicators Consortium, established in 2003, represents one of the most extensive efforts to engage stakeholders from around the world and to document and share knowledge on community indicators (see Ramos and Jones 2005; http://www.communityindicators.net). Some projects included in the Community Indicators Consortium database of indicator projects specifically include risky alcohol consumption as part of their examination of community well-being (see http://www.communityindicators.net). These types of initiatives suggest that community indicators, including indicators of alcohol use and harm, will continue to grow in the coming years as an area of interest and innovation.

Community indicators are certainly not a panacea for either investigators or community stakeholders. However, when produced with a thorough understanding of the local community system and through thoughtful application of advanced methodological knowledge, they can serve as a powerful tool for understanding, assessing, and addressing alcohol-related problems within their local context.

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Hospital emergency departments (EDs) see many patients with alcohol-related injuries and therefore frequently are used to assess the relationship between alcohol consumption and injury risk. These studies typically use either case-control or case-crossover designs. Case-control studies, which compare injured ED patients with either medical ED patients or the general population, found an increased risk of injury after alcohol consumption, but differences between the case and control subjects partly may account for this effect. Case-crossover designs, which avoid this potential confounding factor by using the injured patients as their own control subjects, also found elevated rates of injury risk after alcohol consumption. However, the degree to which risk is increased can vary depending on the study design used. Other factors influencing injury risk include concurrent use of other drugs and drinking patterns. Additional studies have evaluated cross-country variation in injury risk as well as the risk by type (i.e., intentional vs. unintentional) and cause of the injury. Finally, ED studies have helped determine the alcohol-attributable fraction of injuries, the causal attribution of injuries to drinking, and the impact of others’ drinking. Although these studies have some limitations, they have provided valuable insight into the association between drinking and injury risk. Key words: Alcohol consumption; alcohol-related injury; alcohol and drug related—injury; alcohol-attributable fractions; risk factors; alcohol and other drug–induced risk; hospital; emergency department; emergency room; emergency care; trauma; injury; intentional injury; unintentional injury; patients; case–control studies; case–crossover studies

Alcohol consumption is a leading risk factor for morbidity and mortality related to both intentional (i.e., violence-related) and unintentional injury. In 2000, 16.2 percent of deaths and 13.2 percent of disability-adjusted life years (DALYs) from injuries, worldwide, were estimated to be attributable to alcohol (Rehm et al. 2009). Alcohol affects psychomotor skills, including reaction time, as well as cognitive skills, such as judgment; as a result, people drinking alcohol often place themselves in high-risk situations for injury. Much of the data linking alcohol with nonfatal injuries have come from studies conducted in hospital emergency departments (EDs). As described in this article, in these settings the prevalence of alcohol involvement in the patients’ injuries, as measured by a positive blood alcohol concentration (BAC) at the time of arrival in the ED or self-reported drinking prior to the injury event, is substantial. To accurately assess the relationship between alcohol use and injury risk, ED studies generally have used probability sampling designs, in which all times of day and days of the week are represented equally. This approach circumvents biases associated with sampling that might occur, for example, if samples were identified only on weekend evenings, when a higher prevalence of drinking and, possibly, of injury might be expected. Although the high prevalence rates mentioned above suggest that alcohol is an important risk factor for injury, they do not provide the information necessary to evaluate the actual level of risk for injury at which drinking places the individual.

Data to establish drinking-related risk of both intentional and unintentional injury in ED samples generally have come from two types of study design: case-control studies and case-crossover studies. This article summarizes the findings of these studies and explores specific aspects of the relationship between alcohol use and injury risk.

### Risk of Injury in ED Studies

#### Case–Control Studies

Two types of case–control studies have been used to estimate the risk of injury from drinking for patients treated in the ED. The most commonly used type of case–control study uses noninjured (i.e., medical) patients attending the same ED during the same period of time as quasi-control subjects. These patients presumably come from the same geographic area as the injured patients and likely share other characteristics (e.g., socioeconomic status). Researchers conducted a meta-analysis of 15 ED studies conducted in 7 countries that participated in the Emergency Room Collaborative Alcohol Analysis Project (ERCAAP) (Cherpitel et al. 2003a) and which all used the same methodology and instrumentation. The studies only included those patients who arrived at the ED within 6 hours of the injury event and excluded those medical patients who primarily were admitted to the ED for alcohol intoxication or withdrawal symptoms. The meta-analysis found a pooled odds ratio (OR) of injury associated with a positive BAC (≥0.01 percent) of 2.4 (95% CI = 1.9–3.0); moreover, the OR was higher (OR = 2.9) for patients with higher BAC levels (≥0.10 percent) (Ye and Cherpitel

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1 The OR is the ratio between the risk that a person with a certain characteristic (e.g., positive BAC) experiences a certain outcome (e.g., an injury) and the risk that a person without that characteristic experiences the same outcome. In other words, an OR of 2.4 indicates that people who have a positive BAC are 2.4 times as likely to be injured as people without a positive BAC.

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A similar likelihood of injury (OR = 2.1) was found for patients who reported drinking within 6 hours prior to the injury event, regardless of time of arrival in the ED.

One concern with this approach of using medical patients as control subjects for injured patients is the possibility of underestimating the true risk of drinking associated with injury. Noninjured patients have been found to be heavier drinkers than people in the general population from which they come who do not seek emergency care (Cherpitel 1993). Thus, these patients may be attending the ED for conditions related to their drinking (in addition to those associated with alcohol intoxication or withdrawal).

In the second type of case–control study used to estimate risk of injury from drinking in ED patient samples, people in the general population of the community from which the ED patients come are used as control subjects. These individuals presumably are free of conditions that may be related to their drinking. Only four such studies have been reported to date, including two from Australia (McLeod et al. 1999; Watt et al. 2004) and one each from the United States (Vinson et al. 2003) and Mexico (Borges et al. 1998). In these studies, the ORs ranged from 6.7 in the Mexican study to 3.1 in the U.S. study and around 2.0 in the Australian studies. Moreover, both the U.S. and the Australian studies demonstrated a dose-response relationship.

### Case–Crossover Studies

The second study design used to estimate the risk of injury from alcohol consumption is the case–crossover study (Maclure 1991). This approach is thought to circumvent at least some of the problems raised with the case–control design, such as demographic and others differences between case and control subjects that may be related to both alcohol consumption and likelihood of injury. There are two approaches to the case–crossover design, both of which use injured patients as their own control subjects, thereby theoretically reducing confounding of the alcohol–injury relationship from stable risk factors, such as age and gender.

- **The matched-interval approach.** Studies using the matched-interval approach compare drinking within 6 hours prior to the injury event with drinking during a predetermined control period, such as the same 6-hour period during the previous day or previous week. Such studies have reported ORs ranging from 3.2 (based on any drinking at the same time the previous day) (Vinson et al. 2003) to 5.7 in a 10-country study (based on any drinking at the same time the previous week) (Borges et al. 2006b). Both studies demonstrated a dose-response relationship. Thus, the analysis of Vinson and colleagues (2003) determined ORs ranging from 1.8 with consumption of 1 to 2 drinks prior to injury to 17 with consumption of 7 or more drinks. Likewise, Borges and colleagues (2006b) found ORs ranging from 3.3 with consumption of one to two drinks to 10.1 with consumption of six or more drinks prior to injury.

- **The usual-frequency approach.** This approach compares the patients’ drinking in the 6 hours preceding the injury to their expected drinking during that time, based on their usual frequency of drinking. In a study using this approach that included 28 EDs across 16 countries, the estimated ORs ranged from 1.05 (Canada) to 35.0 (South Africa), with a pooled estimate of 5.69 (95% CI = 4.04–8.00) (Borges et al. 2006a).

### Comparison of Methods to Estimate Risk

The results described above indicate that the estimates of risk of injury in samples from the same country can vary depending on the method used. For example, in analyses across eight countries participating in ERCAAP, analyses using the case–control method found that the pooled OR of injury for self-reported drinking prior to the event was 2.1, compared with an OR of 5.2 when the usual-frequency method of case–crossover analysis was used (Ye and Cherpitel 2009). Furthermore, the World Health Organization (WHO) Collaborative Study on Alcohol and Injury, which used the case–crossover method across 12 countries, found a pooled OR of injury of 6.8 using the usual-frequency approach, compared with 5.7 using the matched-interval approach (Borges et al. 2006b). Case–control designs may underestimate the risk of injury if noninjured control subjects are presenting to the ED with other conditions related to their drinking, whereas both the matched-interval and usual-frequency approaches to the case–crossover design are subject to recall bias of drinking in the past.

### Effects of Other Factors on Risk of Injury

#### Effects of Other Drug Use

None of these estimates of risk of injury related to drinking have taken into consideration other drug use at the time of injury, although multiple substances commonly are used together in ED populations (Buchfuhrer and Radecki 1996). Other drug use might be expected to elevate the risk of injury, either alone or in combination with alcohol; however, this may not be the case. One study found an OR of 3.3 for drinking within 6 hours prior to injury and an OR of 3.0 for drinking in combination with other drug use during the same time; in contrast, drug use alone had no significant effect on risk (Cherpitel et al. 2012b). It is important to consider that in this study the majority of drug users reported using marijuana. However, given their different pharmacological properties, all drugs would not be expected to act in a similar manner, either alone or in combination with alcohol. Consequently, in other populations with different drug use patterns the findings might be different.
**Effects of Usual Drinking Patterns**
The risk of injury from drinking prior to the event (i.e., acute consumption) also is influenced by the drinker’s usual drinking patterns (i.e., chronic consumption). Cherpitel and colleagues (2004) found that the risk of injury from drinking prior to the event was lower among frequent heavy drinkers than among infrequent heavy drinkers, suggesting that heavier drinkers may have developed tolerance against some adverse effects of alcohol that lead to injury. Likewise, in an analysis by Gmel and colleagues (2006), the risk of injury was greater among usual light drinkers who occasionally drink heavily (i.e., report episodic heavy drinking) than among people who usually drink heavily but report no episodic heavy drinking or among people who usually drink heavily as well as report episodic heavy drinking.

**Risk of Alcohol-Related Injury**
Although acute alcohol consumption, modified by drinking pattern, has been found to be associated with risk of injury, drinking pattern also has been found to be associated with risk of an alcohol-related injury\(^2\) (defined as drinking within 6 hours prior to injury), with frequency of drinking among non-heavy drinkers (Cherpitel et al. 2003\(^b\)) and both episodic and frequent heavy drinking predictive of alcohol-related injury (Cherpitel et al. 2012\(^c\)). An analysis of combined data from ERCAAP and from the WHO Collaborative Study on Alcohol and Injury across 16 countries found the pooled risk of alcohol-related injury was increased with heavy episodically drinking (OR = 2.7) as well as with chronic high-volume drinking (OR = 3.5); moreover, the risk was highest for people reporting both patterns of drinking (OR = 6.1) (Ye and Cherpitel 2009).

**Cross-country Variation in Risk of Injury**
A great deal of variation has been found across countries in risk of injury and risk of alcohol-related injury, and this heterogeneity seems to be associated with a country’s level of detrimental drinking pattern (DDP). The DDP score, which is based on aggregate survey data and key informant surveys, is a measure developed for comparative risk assessment in the WHO’s Global Burden of Disease study (Rehm et al. 2004). It includes such indicators of drinking patterns as heavy drinking occasions, drinking with meals, and drinking in public places. The DDP has been assessed in a large number of countries around the world as a measure of the “detrimental impact” on health, and other drinking-related harms, at a given level of alcohol consumption (Rehm et al. 2001, 2003). Countries with a higher level of DDP have been found to have a higher risk of injury related to alcohol than those with lower DDP scores (Cherpitel et al. 2005\(^b\)).

**Risk by Type and Cause of Injury**
Risk of injury from alcohol also varies by type (i.e., intentional vs. unintentional) and cause of injury. For example, Macdonald and colleagues (2006) found that the risk was highest for violence-related (i.e., intentional) injuries. A case–crossover analysis using the usual-frequency approach that included data from 15 countries in the ERCAAP and WHO projects found that greater variations across countries existed in the risk of an intentional injury than in risk of unintentional injury; this difference was at least in part explained by the level of DDP in a country (Cherpitel and Ye 2010). Overall, the pooled OR for intentional injury related to drinking in these countries was 21.5, compared with 3.37 for unintentional injury (Borges et al. 2009). Furthermore, the risk of intentional injury showed a greater dose–response association than the risk of unintentional injury (Borges et al. 2009). Thus, the ORs for intentional injuries ranged from 11.14 for one to two drinks prior to injury to 35.57 for five or more drinks during this time, whereas the ORs for unintentional injuries ranged from 3.86 to 6.4, respectively. Among the unintentional injuries, the risk also varied depending on the cause of the injury. For example, the OR was 5.24 for traffic-related injuries, compared with 3.39 for injuries related to falls.

**Alcohol-Attributable Fraction**
Another variable that has been studied in the context of assessing the risk of injuries after drinking is the alcohol-attributable fraction (AAF). This variable represents the proportional reduction in injury that would be expected if the risk factor (i.e., drinking prior to injury) was absent; it reflects the burden of injury in a given society that results from alcohol use. The AAF also varies across countries in ED studies, because it is related to both the risk of injury and the prevalence of alcohol-related injury. In a case–control study of 14 EDs from six countries in ERCAAP, the AAF based on self-reported drinking within 6 hours prior to the injury event varied from 0.5 percent to 18.5 percent for all types of injury; and from 19.1 percent to 83.3 percent for intentional injury (Cherpitel et al. 2005\(^a\)). The pooled estimate from all EDs for the AAF was 5.8 percent for all types of injury and 42.5 percent for intentional injury. In other words, more than 40 percent of all intentional injuries would not have occurred if the people involved had not been drinking. Moreover, the investigators determined higher AAF estimates for male than female subjects for both unintentional injuries (5.5 percent vs. 1.7 percent) and intentional injuries (50.0 percent vs. 7.7 percent).

\(^2\) As used here, the term “alcohol-related injury” refers to injuries where the patient reported using alcohol in the 6-hour period immediately preceding the injury; in contrast, the term “injury” is used here to refer to any injury, regardless of whether it was preceded by alcohol use or not.
Causal Attribution

The ED studies in the ERCAAP and WHO projects also assessed the patients’ causal attribution of their injuries to their drinking—that is, patients were asked whether they believed the injury would have occurred if they had not been drinking. In an evaluation that included 15 countries, one-half of the patients who reported drinking prior to injury also reported a causal attribution (Cherpitel et al. 2006). This information was used to establish a subjective AAF—an AAF derived from the patient’s own causal attribution of their injury to drinking. This subjective AAF then was compared to the AAF obtained using the standard formula based on the relative risk of injury from alcohol and prevalence of drinking in the 6-hour period (i.e., the objective AAF) from the six ERCAAP countries, as described above. This comparison found that for unintentional injuries, the subjective AAF generally was somewhat higher than the objective AAF. For intentional injuries, however, the subjective AAF was substantially lower (i.e., 5.9 percent to 46.7 percent) than the objective AAF (i.e., 24.9 percent to 83.3 percent) (Bond and Macdonald 2009).

Others’ Drinking

Researchers also increasingly are interested in studying the harm, including injury, resulting from other people’s drinking. Evaluating these so-called externalities is important for a fuller understanding of the burden of alcohol-related injury in society. To assess such externalities, investigators for the ED studies in the WHO project also obtained data on whether the patient being treated for a violence-related injury believed the other person had been drinking. Across the 14 countries, from 14 percent to 73 percent of the victims believed that others definitely had been drinking. Based on these data, the pooled estimate for the AAF was 38.8 percent when both victim and perpetrator were considered, compared with an AAF of 23.9 percent when only the patient was considered (Cherpitel et al. 2012a).

Considerations and Limitations in Estimating Risk of Injury

The data reported here on the risk of injury primarily were derived from patients’ self-reports of drinking prior to injury. Although the ED studies all estimated the patient’s BAC at the time of ED admission based on breath alcohol levels, self-reports seem to be a better measure of drinking, because in many cases a substantial period of time may have lapsed between the patient’s last drink, the injury event, and arrival at the ED. As a result, the BAC may be negative even though the patient reports drinking prior to injury. Indeed, this discrepancy has been found in an analysis of the concordance between self-reported drinking and BAC measurements in the ERCAAP and WHO studies across 16 countries (Cherpitel et al. 2007).

The studies reported here all have been conducted in EDs, rather than in trauma centers that generally treat the most serious injury cases and, consequently, are less conducive to the detailed data collection effort required in studies of alcohol and injury, unless the patient is admitted to the hospital. It is unknown how this may affect the resulting conclusions regarding the rates of the risk of injury from drinking, because the literature has been mixed regarding alcohol’s association with injury severity.

As noted earlier, some limitations also apply to the methods that have been used to estimate the risk of injury related to alcohol consumption. Case–control studies may underestimate this risk because the medical patient controls also may have drinking-related conditions. The matched-interval approach to case–crossover analyses eliminates the heaviest drinkers (i.e., those who report drinking both during the period preceding the injury and during the control period), which may lead to underestimates of the risk of injury for these drinkers. Likewise, the usual-frequency approach may underestimate the risk of injury for heavy drinkers because of the increase in expected drinking occasions for the heaviest drinkers.

In addition, when estimating risk of injury using the case–crossover approach, it is important to consider the activity in which the patient was engaged at the time of injury. For example, for a patient injured in a motor vehicle accident who had been drinking, the comparison with the control time interval only would be valid if the patient also had been in a motor vehicle during the control interval. Otherwise, the patient would not have been exposed to the risk of incurring a motor vehicle–related injury, regardless of whether he or she had been drinking. This is an important consideration in future studies that seek to examine risk of injury related to alcohol.

Lastly, the risk of injury related to drinking likely is affected by a number of individual-level characteristics such as age, gender, and risk-taking disposition, as well as by societal-level characteristics such as detrimental drinking pattern, as discussed above. Estimates of AAFs for injury, which are required for determining the global burden of disease for injury related to alcohol, generally have not taken these variables into consideration, and this is a necessary direction for future research on the burden alcohol-related injury puts on society.

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Chronic Diseases and Conditions Related to Alcohol Use

Kevin D. Shield, M.H.Sc.; Charles Parry, Ph.D.; and Jürgen Rehm, Ph.D.

Alcohol consumption is a risk factor for many chronic diseases and conditions. The average volume of alcohol consumed, consumption patterns, and quality of the alcoholic beverages consumed likely have a causal impact on the mortality and morbidity related to chronic diseases and conditions. Twenty-five chronic disease and condition codes in the International Classification of Disease (ICD)-10 are entirely attributable to alcohol, and alcohol plays a component-risk role in certain cancers, other tumors, neuropsychiatric conditions, and numerous cardiovascular and digestive diseases. Furthermore, alcohol has both beneficial and detrimental impacts on diabetes, ischemic stroke, and ischemic heart disease, depending on the overall volume of alcohol consumed, and, in the case of ischemic diseases, consumption patterns. However, limitations exist to the methods used to calculate the relative risks and alcohol-attributable fractions. Furthermore, new studies and confounders may lead to additional diseases being causally linked to alcohol consumption, or may prove the relationship between alcohol consumption and certain diseases that currently are considered to be causally linked. These limitations do not affect the conclusion that alcohol consumption significantly contributes to the burden of chronic diseases and conditions globally, and that this burden should be a target for intervention. Key words: Alcohol consumption; alcohol use frequency; chronic diseases; disorders; mortality; morbidity; alcohol-attributable fractions (AAF); risk factors; relative risk; AOD-induced risk; cancers; neuropsychiatric disorders; cardiovascular diseases; digestive diseases; diabetes; ischemic stroke; ischemic heart disease; burden of disease

Alcohol has been a part of human culture for all of recorded history, with almost all societies in which alcohol is consumed experiencing net health and social problems (McGovern 2009; Tramacere et al. 2012a, b, c). With the industrialization of alcohol production and the globalization of its marketing and promotion, alcohol consumption and its related harms have increased worldwide (see Alcohol Consumption Trends, in this issue). This has prompted the World Health Organization (WHO) to pass multiple resolutions to address this issue over the past few years, including the World Health Assembly’s Global Strategy to Reduce the Harmful Use of Alcohol, which was passed in May 2010. Of growing concern are noncommunicable chronic diseases and conditions that have been shown to contribute substantially to the alcohol-attributable burden of disease (Rehm et al. 2009). Specifically, in 2004 an estimated 35 million deaths and 603 million disability-adjusted life-years (DALYs) lost were caused by chronic diseases and conditions globally (WHO 2008); alcohol was responsible for 3.4 percent of the deaths and 2.4 percent of DALYs caused by these conditions (Parry et al. 2011). To address the burden of chronic diseases and conditions, the United Nation (UN) General Assembly passed Resolution 64/265 in May of 2010, calling for their prevention and control (UN 2010). This resolution is intended to garner multisectoral commitment and facilitate action on a global scale to address the fact that alcohol (together with tobacco, lack of exercise, and diet) plays a significant role in chronic diseases and conditions. It is noteworthy that cardiovascular diseases, cancers, and diabetes in particular have been highlighted for targeted action (UN 2010) because alcohol is a risk factor for many cardiovascular diseases and cancers and has both beneficial and detrimental effects on diabetes and ischemic cardiovascular diseases, depending on the amount of alcohol consumed and the patterns of consumption.

Building on previous reviews concerning alcohol and disease (Rehm et al. 2003a, 2009), this article presents an up-to-date and in-depth overview of the relationship of alcohol consumption and high-risk drinking patterns and the initia-

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1 Ischemic cardiovascular diseases are those caused by a blockage of blood vessels, resulting in a loss of blood supply to the tissue serviced by the affected blood vessels.
tion/exacerbation and treatment of various chronic diseases and conditions. It also assesses the methods used to calculate the impact of alcohol consumption on chronic diseases and conditions.

**Alcohol Consumption As a Risk Factor for Chronic Diseases and Conditions**

Figure 1 presents a conceptual model of the effects of alcohol consumption on morality and mortality and of the influence of both societal and demographic factors on alcohol consumption and alcohol-related harms resulting in chronic diseases and conditions (adapted from Rehm et al. 2010a). According to this model, two separate, but related, measures of alcohol consumption are responsible for most of the causal impact of alcohol on the burden of chronic diseases and conditions—overall volume of alcohol consumption and patterns of drinking. The overall volume of alcohol consumption plays a role in all alcohol-related diseases, whereas drinking patterns only affect ischemic cardiovascular diseases. In addition to the overall volume and pattern of consumption, the quality of the alcoholic beverages consumed also may influence mortality and morbidity from chronic diseases and conditions. However, this pathway is of less importance from a public health perspective (Lachenmeier and Rehm 2009; Lachenmeier et al. 2007) because it has a much smaller impact than the other two factors.

The effects of overall volume of alcohol consumed, consumption patterns, and quality of the alcoholic beverages consumed on mortality and morbidity from chronic diseases and conditions are mediated by three main mechanisms. These include the following:

- The toxic and beneficial biochemical effects of beverage alcohol (i.e., ethanol) and other compounds found in alcoholic beverages;
- The consequences of intoxication; and
- The consequences of alcohol dependence.

These intermediate mechanisms have been reviewed in more detail by Rehm and colleagues (2003a).

**Chronic Diseases and Conditions Related to Alcohol**

**Chronic Diseases and Conditions Entirely Attributable to Alcohol**

Of the chronic diseases and conditions causally linked with alcohol consumption, many categories have names indicating that alcohol is a necessary cause—that is, that these particular diseases and conditions are 100 percent alcohol attributable. Of these, alcohol use disorders (AUDs)—that is, alcohol dependence and the harmful use of alcohol as defined by the *International Classification of Disease, Tenth Edition* (ICD–10)—certainly are the most important categories, but many other diseases and conditions also are entirely attributable to alcohol (see table 1).

**Chronic Diseases and Conditions for Which Alcohol Is a Component Cause**

Alcohol is a component cause for more than 200 other diseases and conditions with ICD–10 three-digit codes—that
is, alcohol consumption is not necessary for the diseases to develop (Rehm et al. 2010a). For these conditions, alcohol shows a dose-response relationship, where the risk of onset of or death from the disease or condition depends on the total volume of alcohol consumed (Rehm et al. 2003a). Table 2 outlines these chronic diseases and conditions that are associated with alcohol consumption and lists the source of the relative risk (RR) functions if the chronic disease or condition is included as an alcohol-attributable harm in the 2005 Global Burden of Disease (GBD) Study. Several of these chronic diseases and conditions are singled out for further discussion in the following sections to highlight alcohol’s causative or protective role.

**Specific Chronic Diseases and Conditions Associated With Alcohol Consumption**

### Malignant Neoplasms

The relationship between alcohol consumption and cancer already was suggested in the early 20th century, when Lamy (1910) observed that patients with cancer either of the esophagus or of the cardiac region were more likely to be alcoholics. The accumulation of evidence supporting the relationship between ethanol and cancers led the International Agency for Research on Cancer (IARC) to recognize the cancer-inducing potential (i.e., carcinogenicity) of ethanol in animal models and to conclude that alcoholic beverages are carcinogenic to humans (IARC 2008). Specifically, the GBD study found that alcohol increased the risk of cancers of the upper digestive tract (i.e., mouth and oropharynx, esophagus, and larynx), the lower digestive tract (i.e., colon, rectum, and liver), and the female breast (see figure 2). More up-to-date systematic reviews and meta-analyses on alcohol consumption and the risk of developing cancer have been published by Fedirko and colleagues (2011) for colorectal cancer, Islami and colleagues (2011) for esophageal squamous cell carcinoma, Islami and colleagues (2010) for laryngeal cancer, and Tramacere and colleagues (2010) and Turiati and colleagues (2010) for oral and pharyngeal cancers.

A recent meta-analysis also has indicated that alcohol consumption is significantly linked to an increased risk of developing prostate cancer in a dose-dependent manner (Rota et al. 2012); this observation is consistent with previous meta-analyses concluding that alcohol consumption and the risk for prostate cancer are significantly correlated (Dennis 2000; Fillmore et al. 2009). Additional research, however, is required on the biological pathways to prove the role of alcohol required on the biological pathways to prove the role of alcohol

Thus, two recent meta-analyses found no association between alcohol drinking status (i.e., drinkers compared with non-drinkers) and risk of gastric cardia adenocarcinoma (Tramacere et al. 2012a, d). However, one meta-analysis did find an association between heavy alcohol consumption and the risk of this type of cancer (Tramacere et al. 2012a).

For several types of cancer investigators have found a nonsignificant positive association with alcohol consumption, including endometrial (Bagnardi et al. 2001; Rota et al. 2012), ovarian (Bagnardi et al. 2001), and pancreatic cancers (Bagnardi et al. 2001). However, because the relationship at least between alcohol consumption and endometrial and pancreatic cancer is modest (i.e., the point estimates of RR are low, even at high levels of average daily alcohol consumption), additional studies with large numbers of participants are needed to accurately assess the relationship (Bagnardi et al. 2001). The relationship between alcohol consumption and bladder and lung cancers is even less clear, with one meta-analysis finding that alcohol significantly increases the risk for both types of tumors (Bagnardi et al. 2001), whereas

#### Table 1  Chronic Diseases and Conditions That Are, by Definition, Alcohol Attributable (i.e., Require Alcohol Consumption As a Necessary Cause)

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<th>ICD–10 Code</th>
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<td>F10</td>
<td>Mental and behavioral disorders attributed to the use of alcohol</td>
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<tr>
<td>F10.0</td>
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<td>F10.1</td>
<td>Harmful use</td>
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<td>F10.2</td>
<td>Dependence syndrome</td>
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<td>F10.3</td>
<td>Withdrawal state</td>
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<tr>
<td>F10.4</td>
<td>Withdrawal state with delirium</td>
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<td>F10.5</td>
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<td>F10.6</td>
<td>Amnesic syndrome</td>
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<td>F10.7</td>
<td>Residual and late-onset psychotic disorder</td>
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<tr>
<td>G31.2</td>
<td>Degeneration of nervous system attributed to alcohol</td>
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<tr>
<td>G62.1</td>
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<tr>
<td>G72.1</td>
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<td>I42.6</td>
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<td>K85.2</td>
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<td>K86.0</td>
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<td>P04.3</td>
<td>Fetus and newborn affected by maternal use of alcohol</td>
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<tr>
<td>Q86.0</td>
<td>Fetal alcohol syndrome (dysmorphic)</td>
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2 The GBD Study is a project that aims to provide a consistent and comparative description of the global burden of diseases and injuries and the risk factors that cause them.
<table>
<thead>
<tr>
<th>No. of 2005 GBD Code</th>
<th>Disease</th>
<th>ICD–10</th>
<th>Effect</th>
<th>Level of Evidence Meta-Analysis</th>
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<td>IIA13</td>
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<td>IIA19</td>
<td>Kidney and other urinary organ cancers</td>
<td>C64–C66, C68 (except C68.9)</td>
<td>Beneficial (renal cell carcinoma only)</td>
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<td>IIA24</td>
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<td>C82–C85, C96</td>
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<td>IIB</td>
<td>Other neoplasms</td>
<td>D00–D48 (except D09.9, D37.9, D38.6, D39.9, D40.9, D41.9, 48.9)</td>
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<tr>
<td>No. of 2005 GBD Code</td>
<td>Disease</td>
<td>ICD–10</td>
<td>Effect</td>
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<td>Used if Included in the GBD Study</td>
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<td>Causally related</td>
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<td>F01–F03, G30–G31</td>
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<td>Insufficient causal evidence</td>
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<td>G40–G41</td>
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<td>Causally related</td>
<td>(Samokhvalov et al. 2010a)</td>
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<td>Cardiovascular and circulatory diseases</td>
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<td>Hypertensive heart disease</td>
<td>I11–I13</td>
<td>Detrimental (however, this depends on drinking patterns and volume of consumption)</td>
<td>Causally related</td>
<td>(Taylor et al. 2010)</td>
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<tr>
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<td>Ischemic heart disease</td>
<td>I20–I25</td>
<td>Beneficial (however, this depends on drinking patterns and volume of consumption)</td>
<td>Causally related</td>
<td>(Roerecke and Rehm 2010)</td>
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<td>IIH4a</td>
<td>Ischemic stroke</td>
<td>I63–I67, I69.3</td>
<td>Beneficial (however, this depends on drinking patterns and volume of consumption)</td>
<td>Causally related</td>
<td>(Patra et al. 2010)</td>
</tr>
</tbody>
</table>
**A. Neoplasms of the upper digestive tract**

- Mouth and oropharynx cancers
- Esophageal cancer
- Larynx cancer

**B. Neoplasms of the lower digestive tract**

- Colon cancer
- Rectal cancer
- Liver cancer

**C. Other neoplasms**

- Breast cancer (women only)

*Figure 2*  The relationship between increasing amounts of average daily alcohol consumption and the relative risk for cancer, with lifetime abstainers serving as the reference group.

*Source:* Lim et al. 2012.
more recent meta-analyses have found no significant association between alcohol consumption and the risk of bladder cancer (Pelucchi et al. 2012) or the risk of lung cancer in individuals who had never smoked (Bagnardi et al. 2001). These conflicting results may stem from the studies in the more recent meta-analyses adjusting for smoking status when assessing the risk relationship between alcohol and these cancers within individual observational studies (Bagnardi et al. 2001; Pelucchi et al. 2012).

The biological pathways by which alcohol increases the risk of developing cancer depends on the targeted organ and are not yet fully understood. Factors that seem to play a role include the specific variants of alcohol-metabolizing enzymes (i.e., alcohol dehydrogenase [ADH], aldehyde dehydrogenase [ALDH], and cytochrome P450 2E1) a person carries or the concentrations of estrogen as well as changes in folate metabolism and DNA repair (Boffetta and Hashibe 2006). For example, a deficiency in ALDH2 activity in people carrying a gene variant (i.e., allele) called ALDH2 Lys487 contributes to an elevated risk of esophageal cancer from alcohol consumption (Brooks et al. 2009). Because the ALDH2 Lys487 allele is more prevalent in Asian populations (i.e., Japanese, Chinese, and Koreans) (Eng et al. 2007), and ALDH2 is hypothesized to impact the risk associated with alcohol for all cancers, studies should account for the presence of this allele when assessing the risk relationship between alcohol consumption and the development of any form of cancer.

However, it is important to note that alcohol not only increases the risk of cancer but also may lower the risk of certain types of cancer. For example, meta-analyses of observational studies have found that alcohol significantly decreases the risk of renal cell carcinoma (Bellocco et al. 2012; Song et al. 2012), Hodgkin's lymphoma (Tramacere et al. 2012c), and non-Hodgkin's lymphoma (Tramacere et al. 2012c). Alcohol's protective effect for renal cancer is thought to be mediated by an increase in insulin sensitivity, because light to moderate alcohol consumption has been associated with improved insulin sensitivity (Davies et al. 2002; Facchini et al. 1994; Joosten et al. 2008). Insulin resistance may play a key role in the development of renal cancer because people with diabetes, which is characterized by insulin resistance, have an increased risk of renal cancers (Joh et al. 2011; Lindblad et al. 1999). The mechanisms underlying alcohol's protective effect on the risk of developing Hodgkin's lymphoma and non-Hodgkin's lymphoma currently are unknown (Tramacere et al. 2012b, c). Thus, these observed protective effects should be interpreted with caution because the underlying biological mechanisms are not understood and confounding factors and/or misclassification of abstainers within observational studies may be responsible for these effects.

**Diabetes**

Research has found that moderate alcohol consumption is associated with a reduced risk of type 2 diabetes3 (Balunus et al. 2009). Because development of insulin resistance is key in the pathogenesis of type 2 diabetes, it is thought that moderate alcohol consumption protects against the disorder by increasing insulin sensitivity (Hendriks 2007). Such an alcohol-related increase in insulin sensitivity has been found in observational studies as well as in randomized controlled trials (Davies et al. 2002; Kiechl et al. 1996; Lazarus et al. 1997; Mayer et al. 1993; Sierksma et al. 2004). Alternative explanations for the protective effect of moderate alcohol consumption involve an increase in the levels of alcohol metabolites, such as acetaldehyde and acetate (Sarkola et al. 2002); an increase in high-density lipoprotein (HDL)4 (Rimm et al. 1999); and the anti-inflammatory effects of alcohol consumption (Imhof et al. 2001). It is important to note, however, that although there is reason to believe that alcohol consumption is causally linked to reduced risk of type 2 diabetes, it currently is unclear whether alcohol consumption itself is a protective factor or if moderate drinking is a marker for healthy lifestyle choices that may account for some of the observed protective effect.

Furthermore, the effects of alcohol consumption on risk of diabetes are dose dependent (see figure 3). Thus, in observational studies consumption of large amounts of alcohol has been related to an increased risk of type 2 diabetes because higher consumption levels may increase body weight, the concentrations of certain fats (i.e., triglycerides) in the blood, and blood pressure (Wannamethee and Shaper 2003; Wannamethee et al. 2003).

**Neuropsychiatric Conditions**

One of the neuropsychiatric conditions associated with alcohol consumption is epilepsy, which is defined as an enduring predisposition for epileptic seizures and requires the occurrence of at least one seizure for a diagnosis. Alcohol consumption is associated with epilepsy, whereas alcohol withdrawal can cause seizures but not epilepsy (Hillbom et al. 2003).5 Observational research has found that a consistent dose-response relationship exists between alcohol consumption and the risk of epilepsy (see figure 3). Multiple possible pathways may underlie this relationship. In particular, alcohol consumption may have a kindling effect, where repeated withdrawals from alcohol consumption by heavy drinkers may lower the threshold for inducing an epileptic episode (Ballenger and Post 1978). Alternatively, heavy alcohol consumption may increase the risk of epilepsy by causing shrinkage of brain tissue (i.e., cerebral atrophy) (Dam et al. 1985), cerebrovascular infarctions, lesions, head traumas, and changes in neurotransmitter systems and ionic balances.

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3 There are two main types of diabetes. Type 1 diabetes results from the body’s failure to produce insulin, and patients therefore regularly must inject insulin. This type also is known as juvenile diabetes because of its early onset, or insulin-independent diabetes. Type 2 diabetes results from insulin resistance, which develops when the cells fail to respond properly to insulin. It develops with age and therefore also is referred to as adult-onset diabetes.

4 HDLs are certain types of compounds consisting of both fat (i.e., lipid) and protein components that are involved in cholesterol metabolism in the body. HDLs also are referred to as “good cholesterol.”

5 Seizures are excluded from the 2005 OBD study definition of epilepsy.
Another neuropsychiatric disorder considered to be causally linked to alcohol consumption is unipolar depressive disorder. This association is supported by the temporal order of the two conditions, consistency of the findings, reversibility with abstinence, biological plausibility, and the identification of a dose-response relationship. One study determined the risk of depressive disorders to be increased two- to threefold in alcohol-dependent people (see Rehm and colleagues [2003a] for an examination of the causal criteria). The alcohol-attributable morbidity and mortality from unipolar depressive disorder currently cannot be calculated because the relationship may be confounded by several factors, including a genetic predisposition, environmental factors (i.e., an underlying disorder or environmental exposure that may contribute to both heavy alcohol use and depressive disorders), and potential self-medication with alcohol by individuals with unipolar depressive disorders (Grant and Pickering 1997; Rehm et al. 2004). Research findings suggest that all of these pathways may play a role. The pathways for the association between alcohol and unipolar depressive disorder in which alcohol does not play a causal role only affect the measurement of the alcohol-based RR for unipolar depressive disorder; however, they do not contradict the notion that alcohol is causally related to the development of unipolar depressive disorder via other pathways. This conclusion results from the observation that depressive symptoms increase markedly during heavy-drinking occasions and disappear or lessen during periods of abstinence (Rehm et al. 2003a).

Numerous studies also have examined the association between alcohol and Alzheimer’s disease and vascular dementia. These analyses generally have determined a beneficial effect of alcohol, which has been attributed to alcohol’s ability to prevent ischemic events in the circulatory system (Peters et al. 2008; Tyas 2001). However, studies of these associations have generated highly heterogeneous results, and the design and statistical analyses of these studies make it impossible to rule out the potential effects of confounding factors (Panza et al. 2008; Peters et al. 2008).

### Cardiovascular and Circulatory Diseases

Alcohol consumption affects multiple aspects of the cardiovascular system, with both harmful and protective effects. These include the following (figure 4):

- Increased risk of hypertension (at all consumption levels for men and at higher consumption levels for women);
- Increased risk of disorders that are caused by abnormalities in the generation and disruption of the electrical signals that coordinate the heart beat (i.e., conduction disorders and other dysrhythmias);
- Increased risk of cardiovascular disease, such as stroke caused by blockage of blood vessels in the brain (i.e., ischemic stroke) (at a higher volume of consumption) or rupture of blood vessels (i.e., hemorrhagic stroke); and
- Protective effects (at lower levels of consumption) against hypertension in women and against ischemic heart disease and ischemic stroke in both men and women.

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**Figure 3** The relationship between increasing amounts of average daily alcohol consumption and the relative risk for diabetes and epilepsy, with lifetime abstainers serving as the reference group.

**Source:** Lim et al. 2012.
Figure 4  The relationship between increasing amounts of average daily alcohol consumption and the relative risk for cardiovascular diseases (i.e., hypertension, conduction disorders, and ischemic and hemorrhagic stroke), with lifetime abstainers serving as the reference group. For both hypertension and hemorrhagic and ischemic stroke, the relationship differs between men and women. Moreover, for both ischemic and hemorrhagic stroke, the influence of alcohol consumption on mortality is much greater than the influence on morbidity, at least in women. In men, no such difference appears to exist.

The specific biological pathways through which alcohol consumption interacts with the cardiovascular system are not always clear, but several mechanisms have been identified that may play a role. These include increased blood concentrations of HDLs, effects on cellular signaling, decreased blood clot formation by platelets, and increased blood clot dissolution through enzyme action (Zakhari 1997). More indirect effects also may play a role. For example, alcohol may increase the risk of hypertension by enhancing the activity of the sympathetic nervous system, which results in greater constriction of the blood vessels and makes the heart contract more strongly. In addition, alcohol possibly decreases the sensitivity of the body’s internal blood pressure sensors (i.e., baroreceptors), thereby diminishing its ability to regulate blood pressure.

Alcohol’s protective effects against the risk of ischemic heart disease as well as against hypertension in women is hypothesized to result from its ability to increase HDL levels and/or reduce platelet aggregation on arterial walls. Differences in the effects of alcohol in men and women may stem from differing drinking patterns, with men more likely to engage in binge drinking, even at low average levels of consumption. These heavy-drinking occasions may lead to an increased risk of hypertension for men compared with women at similar alcohol consumption levels (Rehm et al. 2003b).

Alcohol’s effect on hypertension also contributes to the risk of hemorrhagic stroke (Taylor et al. 2009), with a hypothesized dose-response effect. The mortality and morbidity from alcohol-attributable hemorrhagic stroke differ by sex (see figure 4). As with hypertension, differences in drinking pattern between men and women most likely are responsible for the differing RR functions for hemorrhagic stroke by sex. Three possible explanations have been put forth to explain the effects of drinking pattern on RR:

- Heavy drinkers also may have other comorbidities that may increase the probability of a fatal hemorrhagic stroke.
- Alcohol consumption may worsen the disease course through biological mechanisms and by decreasing compliance with medication regimens.
- Alcohol’s effects on morbidity may be underestimated because of a stigmatization of heavy alcohol consumption in women, thereby potentially decreasing the probability that female heavy drinkers will be treated for stroke.

Large cohort studies and meta-analyses have shown that alcohol consumption leads to an increase in the risk for conduction disorders and dysrhythmias (Samokhvalov et al. 2010b). These effects are caused by changes in the electrical activity of the heart, including direct toxic effects of alcohol on the heart (i.e., cardiotoxicity), excessive activity of the sympathetic nervous system (i.e., hyperadrenergic activity) during drinking and withdrawal, impairment of the parasympathetic nervous system (i.e., of vagal tone), and increase of intra-atrial conduction time (Balbão et al. 2009).

Alcohol interacts with the ischemic system to decrease the risk of ischemic stroke and ischemic heart disease at low levels of consumption; however, this protective effect is not observed at higher levels of consumption. As mentioned above, alcohol exerts these effects mainly by increasing levels of HDL, preventing blood clots, and increasing the rate of breakdown of blood clots. However, binge drinking, even by light to moderate drinkers, leads to an increased risk of ischemic events by increasing the probability of clotting and abnormal contractions of the heart chambers (i.e., ventricular fibrillation). As with hemorrhagic stroke, alcohol has different effects on morbidity than on mortality related to ischemic events (see figure 5). Thus, meta-analyses of alcohol consumption and the risk of ischemic heart disease (Roerecke and Rehm 2012) and ischemic stroke (Taylor et al. 2009) found a larger protective effect for morbidity than for mortality related to these conditions. One possible explanation for this observation, in addition to those listed above for hemorrhagic stroke, is that patients in the morbidity studies may be younger at the time of the stroke than those in mortality studies. Despite the increased risk for ischemic heart disease at higher levels of alcohol consumption noted in observational studies (see Roerecke and Rehm 2012 for the most up-to-date meta-analysis), there was not enough evidence for a detrimental effect of alcohol consumption on ischemic heart disease for it to be modeled in the 2005 GBD study.

Moreover, the observational studies investigating the link between alcohol consumption and ischemic events had several methodological flaws, and the RR functions for ischemic events, especially ischemic heart disease, therefore are not well defined. A meta-analysis conducted by Roerecke and Rehm (2012) observed a substantial degree of heterogeneity among all consumption levels, pointing to a possible confounding effect of heavy drinking. In addition, previous observational studies have been limited by the inclusion of “sick quitters” in the reference groups, who have an increased risk of ischemic events compared with lifetime abstainers.

**Digestive Diseases**

Alcohol is associated with various liver diseases and is most strongly related to fatty liver, alcoholic hepatitis, and cirrhosis. The association between the risk of liver cirrhosis and alcohol consumption has long been recognized (see figure 6). The main biological mechanism contributing to this liver damage likely involves the breakdown of ethanol in the liver through oxidative and nonoxidative pathways that result in the production of free radicals, acetaldehyde, and fatty acid ethyl esters, which then damage liver cells (Tuma and Casey 2003). Given the same amount of alcohol consumption, alcohol increases the risk of mortality from liver cirrhosis more steeply than the risk of morbidity because it worsens the course of liver disease and has a detrimental effect on the immune system (Rehm et al. 2010c).

Alcohol consumption also has been linked to an increase in the risk for acute and chronic pancreatitis. Specifically, heavy alcohol consumption (i.e., more than, on average,
Figure 5 The relationship between increasing amounts of average daily alcohol consumption and the relative risk for ischemic heart disease, with lifetime abstainers serving as the reference group. Low to moderate alcohol consumption has a beneficial effect on both mortality and morbidity from ischemic heart disease. However, the specific effects depend on both the gender and the age of the drinker, with the greatest beneficial effects of low-to-moderate consumption seen on morbidity from ischemic heart disease in women ages 15 to 34.

48 grams pure ethanol, or about two standard drinks, per day) leads to a noticeably elevated risk of pancreatitis, whereas consumption below 48 grams per day is associated with a small increase in risk of pancreatitis (see figure 6). Higher levels of alcohol consumption may affect the risk of pancreatitis through the same pathways that cause liver damage, namely the formation of free radicals, acetaldehyde, and fatty acid ethyl esters during the metabolism of alcohol in damaged pancreatic acinar cells (Vonlaufen et al. 2007).

Psoriasis
Psoriasis is a chronic inflammatory skin disease caused by the body’s own immune system attacking certain cells in the body (i.e., an autoimmune reaction). Although there is insufficient biological evidence to indicate that alcohol is causally linked with psoriasis, many observational studies have determined a detrimental impact of drinking on psoriasis, especially in male patients. Alcohol is hypothesized to induce immune dysfunction that results in relative immunosuppression. In addition, alcohol may increase the production of inflammatory cytokines and cell cycle activators, such as cyclin D1 and keratinocyte growth factor, that could lead to excessive multiplication of skin cells (i.e., epidermal hyperproliferation). Finally, alcohol may exacerbate disease progression by interfering with compliance with treatment regimens (Gupta et al. 1993; Zaghloul and Goodfield 2004).

Alcohol’s Effects on Other Medication Regimens
Alcohol can affect cognitive capacity, leading to impaired judgment and a decreasing ability to remember important information, including when to take medications for other conditions (Braithwaite et al. 2008; Hendershot et al. 2009; Parsons et al. 2008). Although the relationship between alcohol consumption and adherence to treatment regimens mainly has been studied in regards to adherence to HIV antiretroviral treatment (Braithwaite and Bryant 2010; Hendershot et al. 2009; Neuman et al. 2012), research also has shown that alcohol consumption and alcohol misuse impact adherence to medications for other chronic diseases, with significant or almost-significant effects (Bates et al. 2010; Bryson et al. 2008; Coldham et al. 2002; Verdoux et

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**Figure 6** The relationship between increasing amounts of average daily alcohol consumption and the relative risk for digestive diseases (i.e., liver cirrhosis and pancreatitis), with lifetime abstainers serving as the reference group. For liver cirrhosis, alcohol’s effects on mortality are greater than those on morbidity, and slight differences exist between the effects in men and women.

Impact of Sex, Race, and Age on the Association of Alcohol Consumption with Chronic Diseases

Given the same amount of alcohol consumed, men and women can have differing morbidity and mortality from alcohol-related chronic disease and conditions. These differences may be related to pharmacokinetics of alcohol in men and women. Women generally have a lower body water content than men with the same body weight, causing women to reach higher blood alcohol concentrations than men after drinking an equivalent amount of alcohol (Frezza et al. 1990; Taylor et al. 1996). Moreover, women appear to eliminate alcohol from the blood faster than do men, possibly because they have a higher liver volume per unit body mass (Kwo et al. 1998; Lieber 2000). In addition to these pharmacokinetic factors, hormonal differences also may play a role because at least in the case of liver disease, alcohol-attributable harm is modified by estrogen. However, hormonal influences on alcohol-related risks are not yet fully understood (Eagon 2010).

As noted previously, a deficiency of the ALDH2 enzyme in people carrying the ALDH2 Lys487 allele contributes to an elevated risk of cancer from alcohol consumption. Because alcohol metabolism also plays a role in many other chronic diseases, the ALDH2 Lys487 allele also may increase the risk for digestive diseases. The heterogeneity of risk caused by this allele, which is more prevalent in Asian populations, may lead to incorrect measurements of the risk for cancer and digestive disease outcomes in countries with a small Asian population, and will lead to incorrect results if the RRs from these countries are applied to Asian populations (Lewis and Smith 2005; Oze et al. 2011).

Because the pathology of alcohol-related ischemic heart disease is affected by the age of the drinker (Lażeńık et al. 2011), differences also may exist in the risk of ischemic heart disease in different age groups. Preliminary research assessing this issue across multiple studies has found that the association between alcohol consumption and the resulting risk for ischemic heart disease does indeed differ by age (see figure 5). However, no meta-analyses to date have investigated the effects of alcohol consumption on the risk of morbidity and mortality in different age groups for other chronic diseases and conditions. Accordingly, research is needed to assess if the varying relationship between alcohol consumption and ischemic heart disease in different age groups results from biological differences in pathology or from differences in drinking patterns. Additionally, research is needed to assess if age modifies the risk relationships between alcohol and other diseases.

Estimating the Alcohol-Attributable Fractions of Chronic Diseases and Conditions

When assessing the risk of chronic diseases and conditions that are related to alcohol consumption in some, but not all, cases, one of the variables frequently analyzed is the alcohol-attributable fraction (AAF)—that is, the proportion of cases that can be attributed to the patient’s alcohol consumption. Because alcohol consumption can be modeled using a continuous distribution (Kehoe et al. 2012; Rehm et al. 2010b), the calculation of the alcohol-attributable burden of disease uses a continuous RR function. Thus, the AAFs for chronic diseases and conditions can be calculated using the following formula:

$$AAC = \frac{P_{abs} + P_{form} RR_{form} + \int_{0}^{\text{max}} p(x) RR(x) dx - 1}{P_{abs} + P_{form} RR_{form} + \int_{0}^{\text{max}} p(x) RR(x) dx}$$

In this formula, $P_{abs}$ represents the prevalence of the disease among lifetime abstainers, $P_{form}$ is the prevalence among former drinkers, $RR_{form}$ is the relative risk for former drinkers, $p(x)$ is the prevalence among current drinkers with an average daily alcohol consumption of $x$, and $RR(x)$ is the relative risk for current drinkers with an average daily alcohol consumption of $x$. These AAFs vary greatly depending on alcohol exposure levels. (For examples of AAFs and information on the calculation of the 95 percent confidence intervals for chronic diseases and conditions see Gmel and colleagues [2011]).

Limitations of RR Functions for Chronic Diseases and Conditions

The chronic disease RR functions outlined in figures 2 to 6 are derived from the most up-to-date and rigorous meta-analyses in which the risk of a disease (i.e., for mortality and, where possible, morbidity) was provided by alcohol consumption as a continuous function (for more details on the meta-analysis methods used in each study, see the original articles cited in table 2). However, the RR functions and the relationship between alcohol consumption and the risk of chronic diseases and conditions are biased by multiple factors. First, the RRs can be limited by poor measurement of alcohol exposure, outcomes, and confounders. Research on alcohol consumption patterns and disease is scarce, and only few studies have investigated the effects of drinking patterns on chronic diseases and conditions. Thus, the chronic disease and condition RRs presented in this article may be confounded by drinking patterns, which are correlated to overall volume of alcohol consumption. Additionally, the volume of alcohol consumed generally is poorly measured, with many medical epidemiology studies measuring alcohol consumption only at baseline. As a result, these analyses do not include measures
of the volume of alcohol consumed during the medically relevant time period, which may encompass several years. For example, in the case of cancer, the cumulative effects of alcohol may take many years before an outcome is observed. Likewise, many of the larger cohort studies only use single-item, semi-quantitative food questionnaires that measure either frequency or volume of consumption.

Second, medical epidemiology studies typically suffer from poorly defined reference groups (Rehm et al. 2008). Thus, such studies typically only measure alcohol consumption at one point or during one time period in a participant’s life and classify, for example, all people who do not consume alcohol during the reference period as abstainers, even though it is essential to separate ex-drinkers, lifetime abstainers, and very light drinkers. As a result, these measurements of alcohol consumption may lead to incorrect risk estimates because the groups of nondrinkers in these studies have heterogeneous risks for diseases (Shaper and Wannamethee 1998). The potential significance of this issue is underscored by previous research indicating that more than 50 percent of those participants who identified themselves as lifetime abstainers in medical epidemiology studies also had reported lifetime drinking in previous surveys (Rehm et al. 2008).

Third, chronic disease and condition outcomes in medical epidemiology studies also frequently are poorly measured, most often by means of self-reporting. Additionally, other confounding factors, such as relevant, non-substance use–related confounders, often are not controlled for.

Fourth, RR estimates for chronic diseases and conditions resulting from alcohol consumption frequently are hampered by weak study designs that base estimates of alcohol-related risks on nonexperimental designs (i.e., case-control and cohort studies). These study designs are limited by factors that cannot be controlled for and which may lead to incorrect results. For example, experimental studies on the effects of antioxidants have failed to confirm the protective effects of such agents found in observational studies (Bjelakovic et al. 2008). Furthermore, the sampling methodology of many of the cohort studies that were used in the meta-analyses for the above-presented RRs is problematic, especially when studying the effects of alcohol consumption. Many of the cohorts in these studies were from high-income countries and were chosen based on maximizing follow-up rates. Although the chosen cohorts exhibited variation in average daily alcohol consumption, little variation was observed in drinking patterns and other potential moderating lifestyle factors.

The overall effect of these limitations on the RRs and AAFs, and on the estimated burden of mortality and morbidity calculated using these RRs, currently is unclear. In order to investigate the effect of these biases, studies should be undertaken that combine better exposure measures of alcohol consumption with state-of-the-art outcome measures in countries at all levels of economic development. These studies are important, not only for understanding the etiology of alcohol-related chronic diseases and conditions, but also for formulating prevention measures (Stockwell et al. 1997).

Limitations of AAFs for Chronic Diseases and Conditions

In most studies assessing AAFs for chronic diseases and conditions, the AAF for an outcome is calculated as if the health consequences of alcohol consumption are immediate. Indeed, for most chronic diseases and conditions, including even diseases such as cirrhosis, a large degree of the effects caused by changes in alcohol consumption can be seen immediately at the population level (Holmes et al. 2011; Leon et al. 1997; Zatonski et al. 2010; for a general discussion see Norström and Skog 2001; Skog 1988). For cancer, however, the situation is different. The effects of alcohol consumption on the risk of cancer only can be seen after years, and often as long as two decades. Nevertheless, for the purpose of illustrating the entire alcohol-attributable burden of disease it is important to include cancer deaths, because they account for a substantial burden. For example, a recent large study found that in Europe 1 in 10 cancers in men and 1 in 33 cancers in women were alcohol related (Schütze et al. 2011). Therefore, in the interpretation of alcohol’s effect on mortality and burden of disease in this article, the assumption that there has been uniform exposure to alcohol for at least the previous two decades must be kept in mind.

Another limitation to calculating the burden of chronic diseases and conditions attributable to alcohol consumption is the use of mainly unadjusted RRs to determine the AAFs. The RR formulas were developed for risks and were adjusted only for age (see Flegal et al. 2006; Korn and Graubard 1999; Rockhill and Newman 1998), although many other socio-demographic factors are linked with both alcohol consumption and alcohol-related harms (see figure 1). However, two arguments can be made to justify the use of mainly unadjusted RR formulas in the 2005 GBD study. First, in risk analysis studies (Ezzati et al. 2004) almost all of the underlying studies of the different risk factors only report unadjusted risks. Relying on adjusted risks would severely bias the estimated risk functions because only a small proportion of generally older studies could be included. Second, most of the analyses of alcohol and the risk of chronic diseases and conditions show no marked differences after adjustment (see Rehm et al. 2010b). However, the need for adjustment to the RRs may change when other dimensions of alcohol consumption, such as irregular heavy-drinking occasions, are considered with respect to ischemic heart disease.

Conclusions

There are limitations to the current ability to estimate the burden of chronic diseases and conditions attributable to alcohol consumption. The comparative risk assessment study within the GBD study only can determine this burden based on current knowledge of alcohol consumption and risk and mortality patterns at a global level. More detailed, country-specific estimates often are limited by the validity of the available consumption and mortality data. As more stud-
ies are published, it is likely that new confounders will be discovered for some of the relationships between alcohol consumption and various chronic diseases and conditions. The results from such new studies then may be used in meta-analyses of the effect of alcohol in diseases where alcohol only plays a small role, such as bladder, endometrial, and ovarian cancer. New studies also may lead to the recognition of a causal link between alcohol consumption and other diseases. Furthermore, new confounders and new studies may disprove the relationship between alcohol consumption and certain diseases that currently are considered to be causally linked.

Although there are limitations to the current methodology used to estimate the alcohol-attributable burden of chronic diseases and conditions, the limitations discussed in this article do not affect the overall conclusion that alcohol consumption is related to a considerable number of chronic diseases and conditions and contributes to a substantial amount of the global burden of chronic diseases and conditions. Therefore, alcohol consumption should be considered in developing intervention strategies aimed at reducing the burden of chronic diseases and conditions.

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References


Economic Costs of Excessive Alcohol Use

This brief sidebar summarizes recent findings on the economic costs of excessive alcohol use and highlights the primary methods used to estimate these costs. The total economic cost of excessive drinking in 2006, including costs for health care, productivity losses, and costs such as property damage and alcohol-related crime, was estimated to be $223.5 billion (see table) (Bouchery et al. 2011). Prior to this estimate, the last comprehensive analysis reported that the estimated economic costs of excessive drinking were $148 billion in 1992 (Harwood et al. 1998). Data from that report were used to project a cost estimate of $185 billion for 1998 (Harwood 2000). For a review of the global burden of alcohol use, see Rehm and colleagues (2009). For a broader examination of both the methods used to reach the current estimates and details on each of the estimated costs, as well as analysis of the significance and limitations of the study, see Bouchery and colleagues (2011, 2013).

Researchers used updated data, new data sources, and new measurement tools to develop the 2006 estimate. For example, the Alcohol-Related Disease Impact (ARDI) software, created by the Centers for Disease Control and Prevention, was used to assess the relationship between excessive drinking and various health and social outcomes. This software, originally released in 2004, was designed to allow researchers to calculate alcohol-attributable deaths, years of potential life lost, direct health care costs, indirect morbidity and mortality costs, and non–health-sector costs associated with alcohol misuse.

How the Costs of Excessive Alcohol Use Are Estimated

As in the 1992 and 1998 reports of estimated costs, the most recent report was based on the “cost-of-illness” approach. That is, costs for health care, productivity losses, and other expenses for 2006 were obtained from national databases, and alcohol-attributable fractions (AAFs) were applied to assess the proportion of costs that could be attributed to excessive alcohol consumption. For example, researchers would consult a national health care database to determine the cost of liver cirrhosis and multiply this cost by a reasonable AAF to determine the proportion of the total cost that can be attributed to excessive alcohol use. Likewise, costs of lost productivity, crime, and other consequences of excessive alcohol use are estimated based on information from various national databases.

Table: Total Economic Costs of Excessive Alcohol Consumption in the United States, 2006

<table>
<thead>
<tr>
<th>Cost Category</th>
<th>Total Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Health Care Costs</strong></td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse and Dependence</td>
<td>$10,668,457</td>
</tr>
<tr>
<td>Primary Diagnoses Attributable to Alcohol</td>
<td>8,526,822</td>
</tr>
<tr>
<td>Inpatient Hospital</td>
<td>5,115,568</td>
</tr>
<tr>
<td>Physician Office and Hospital Ambulatory Care</td>
<td>1,195,946</td>
</tr>
<tr>
<td>Nursing Home Care</td>
<td>1,002,888</td>
</tr>
<tr>
<td>Retail Pharmacy and Other Health Professional</td>
<td>1,212,420</td>
</tr>
<tr>
<td>Fetal Alcohol Syndrome</td>
<td>2,538,004</td>
</tr>
<tr>
<td>Other Health System Costs</td>
<td>2,822,308</td>
</tr>
<tr>
<td>Prevention and Research</td>
<td>1,207,120</td>
</tr>
<tr>
<td>Training</td>
<td>29,527</td>
</tr>
<tr>
<td>Health Insurance Administration</td>
<td>1,585,660</td>
</tr>
<tr>
<td><strong>Total Health Care Costs</strong></td>
<td>$24,555,591</td>
</tr>
<tr>
<td><strong>Productivity Losses</strong></td>
<td></td>
</tr>
<tr>
<td>Impaired Productivity</td>
<td>$83,695,036</td>
</tr>
<tr>
<td>Traditional Earnings</td>
<td>74,101,827</td>
</tr>
<tr>
<td>Household Productivity</td>
<td>5,355,629</td>
</tr>
<tr>
<td>Absenteeism</td>
<td>4,237,580</td>
</tr>
<tr>
<td>Institutionalization/Hospitalization</td>
<td>2,053,308</td>
</tr>
<tr>
<td>Mortality</td>
<td>65,062,211</td>
</tr>
<tr>
<td>Incarcerations</td>
<td>6,328,915</td>
</tr>
<tr>
<td>Victims of Crime</td>
<td>2,092,886</td>
</tr>
<tr>
<td>Fetal Alcohol Syndrome</td>
<td>2,053,748</td>
</tr>
<tr>
<td><strong>Total Productivity Losses</strong></td>
<td>$161,286,103</td>
</tr>
<tr>
<td><strong>Other Effects on Society</strong></td>
<td></td>
</tr>
<tr>
<td>Crime Victim Property Damage</td>
<td>$439,766</td>
</tr>
<tr>
<td>Criminal Justice System</td>
<td>20,972,690</td>
</tr>
<tr>
<td>Motor Vehicle Crashes</td>
<td>13,718,406</td>
</tr>
<tr>
<td>Fire Losses</td>
<td>2,137,300</td>
</tr>
<tr>
<td>FAS Special Education</td>
<td>368,768</td>
</tr>
<tr>
<td><strong>Total Other Effects</strong></td>
<td>$37,636,930</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>$223,478,624</td>
</tr>
</tbody>
</table>
tion gathered from various national surveys and databases.^

Defining Excessive Alcohol Consumption

The recent report estimated the costs of excessive alcohol consumption, which includes binge drinking (four or more drinks per occasion for a woman and five or more drinks per occasion for a man), heavy drinking (more than one drink per day on average for a woman and more than two drinks per day on average for a man), any alcohol consumption by persons younger than 21, and any alcohol consumption by pregnant women. The surveys used generally ask respondents about the 30 days prior to the survey. The focus on excessive drinking, rather than alcohol use disorders, allows for a more comprehensive look at the health and social consequences that are associated with harmful drinking patterns, including a wide range of acute and chronic health problems, productivity losses due to absenteeism, and crimes committed while intoxicated. In some cases, a history of alcohol abuse or dependence was used as a specific indicator of excessive drinking (e.g., productivity losses based on lost earnings).

Alcohol-Attributable Fractions

AAF, or the proportion of a condition or outcome that is attributed to excessive alcohol consumption, were used to estimate both health-related costs, including deaths and health care expenditures related to excessive drinking, and the costs of specific criminal offenses.

Crime-Specific AAFs. Certain alcohol-related crimes (e.g., driving under the influence of alcohol, public drunkenness, and liquor law violations) are fully attributed to alcohol. For other offenses, researchers estimated the proportion attributable to alcohol based on the percentage of offenders intoxicated at the time of their offense (according to self-reported alcohol-consumption data from surveys of inmates). For homicide, researchers used the AAF from ARDI because it takes into account drinking by both the perpetrator and the victim.

The Costs of Excessive Alcohol Use

Of the total estimated cost of excessive alcohol use for 2006, lost productivity represents 72.2 percent, health care costs represent 11.0 percent, criminal justice system costs make up 9.4 percent, and other consequences make up 7.5 percent (see figure).

Acknowledgement

Source material for this article first appeared in Bouchery et al. 2011, 2013.

References


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For information on the main data sources used in the report, see Bouchery and colleagues (2011, 2013).

Although the methods used to estimate productivity losses attributed to premature mortality are consistent with previous cost-of-illness studies, alternative methods with greater support from economists (i.e., the so-called willingness-to-pay approach) would yield much larger cost estimates.

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Figure
Alcohol and Mortality

Global Alcohol-Attributable Deaths From Cancer, Liver Cirrhosis, and Injury in 2010

Jürgen Rehm, Ph.D., and Kevin D. Shield, MH.Sc.

Alcohol consumption has long been recognized as a risk factor for mortality. By combining data on alcohol per capita consumption, alcohol-drinking status and alcohol-drinking patterns, risk relationships, and mortality, the Comparative Risk Assessment study estimated alcohol-attributable mortality for 1990 and 2010. Alcohol-attributable cancer, liver cirrhosis, and injury were responsible for the majority of the burden of alcohol-attributable mortality in 1990 and 2010. In 2010, alcohol-attributable cancer, liver cirrhosis, and injury caused 1,500,000 deaths (319,500 deaths among women and 1,180,500 deaths among men) and 51,898,400 potential years of life lost (PYLL) (9,214,300 PYLL among women and 42,684,100 PYLL among men). This represents 2.8 percent (1.3 percent for women and 4.1 percent for men) of all deaths and 3.0 percent (1.3 percent for women and 4.3 percent for men) of all PYLL in 2010. The absolute mortality burden of alcohol-attributable cancer, liver cirrhosis, and injury increased from 1990 to 2010 for both genders. In addition, the rates of deaths and PYLL per 100,000 people from alcohol-attributable cancer, liver cirrhosis, and injury increased from 1990 to 2010 (with the exception of liver cirrhosis rates for women). Results of this paper indicate that alcohol is a significant and increasing risk factor for the global burden of mortality. Key words: Alcohol consumption; alcohol burden; alcohol-attributable mortality; alcohol-attributable fractions; global alcohol-attributable mortality; risk factor; cancer; liver cirrhosis; injury; burden of disease; Global Burden of Disease and Injury study

Alcohol and Mortality

Alcohol is causally linked to more than 200 different diseases, conditions, and injuries (as specified in the International Classification of Diseases, Revision 10 [ICD-10] three-digit codes [see Rehm 2011; Rehm et al. 2009; Shield et al., 2013c [pp. 155–173 of this issue]). All of these disease, condition, and injury categories cause mortality and disability, and, thus, alcohol consumption causes a net burden of mortality and disability (Rothman et al. 2008). However, certain patterns of alcohol consumption are protective for ischemic diseases (Roerecke and Rehm 2012a) and diabetes (Baliunas et al. 2009), and, thus, alcohol can prevent death and disability from these causes. The total mortality and disability caused by and prevented by the consumption of alcohol is calculated by comparing the expected mortality under current conditions to a counterfactual scenario where no one has consumed alcohol (Ezzati et al. 2006; Walter 1976). Although the counterfactual scenario seems unrealistic as almost one-half of the global population consumes alcohol (for the most up-to-date statistics on alcohol consumption, see Shield et al. 2013b; World Health Organization 2011a), recent natural experiments in countries where there has been a considerable reduction in alcohol consumption showed a clear reduction in mortality (e.g., Russia) (Leon et al. 1997; Neufeld and Rehm 2013). Accordingly, the calculations of the deaths and disability caused by alcohol consumption seem to correspond to real phenomena and, thus, could predict an approximate level of reduction in mortality if alcohol consumption were to be reduced.

This article outlines the alcohol-attributable mortality burden from three major causes: cancer, liver cirrhosis, and injury. All three categories have long been identified as causally linked to alcohol consumption. With respect to cancer, in 1988 the International Agency for Research on Cancer established alcohol as a carcinogen (International Agency for Research on Cancer 1988), and in its latest monograph has found alcohol consumption to be causally associated with oral cavity, pharynx, larynx, esophagus, liver, colon, rectum, and female breast cancers (International Agency for Research on Cancer 2010, 2012). Studies have shown that stomach cancer may be associated with alcohol consumption, but evidence on the causal relationship between stomach cancer

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and alcohol consumption is not yet conclusive (International Agency for Research on Cancer 2012; Rehm and Shield, in press). Biologically, it has been established that ethanol, and not other ingredients of alcoholic beverages, is the ingredient that mainly causes cancer (Lachenmeier et al. 2012), with acetaldehyde (the first metabolite of ethanol) likely being the most important biological carcinogen (International Agency for Research on Cancer 2010, 2012; Rehm and Shield, in press). In addition, observational studies have found a clear dose-response relationship between alcohol consumption and the risk of cancer, with no observed threshold for the effect of alcohol, as an elevated risk of cancer has been observed even for people who consume relatively low amounts of alcohol (Bagnardi et al. 2013; Rehm et al. 2010a).

Liver cirrhosis has been associated with alcohol consumption, especially heavy consumption, since the seminal work of Benjamin Rush (Rush 1785). The causal link between alcohol consumption and liver cirrhosis is so strong and important that the World Health Organization has created a specific category for alcoholic liver cirrhosis (World Health Organization 2007). As with cancer, there is a dose-response relationship between alcohol consumption and the risk of liver cirrhosis, with no lower threshold being observed (Rehm et al. 2010c); however, the majority of the effect can be seen for heavy drinking (Rehm et al. 2010c).

The risk of injury also has been causally linked to alcohol consumption, with this relationship fulfilling all of the classic Bradford Hill criteria (e.g., consistency of the effect, temporality, a dose-response relationship with the risk of an injury [biological gradient]) (Rehm et al. 2003a). The effect of alcohol on injury is acute; the level of risk for both intentional and unintentional injuries is clearly linked to blood alcohol level (Taylor and Rehm 2012; Taylor et al. 2010), with a very low threshold (Eckardt et al. 1998). There also is an association between average consumption of alcohol and injury (Corrao et al. 2004).

Alcohol-attributable cancer, liver cirrhosis, and injury constitute the majority of the burden of alcohol-attributable mortality. Collectively, they were responsible for 89 percent of the net burden of alcohol-attributable mortality (i.e., the mortality rate after including the beneficial effects of alcohol on ischemic diseases and diabetes) and for 79 percent of the gross burden of alcohol-attributable mortality (Shield et al. 2013a) in the United States in 2005, for people 15 to 64 years of age. Additionally, they were responsible for 91 percent of the net alcohol-attributable mortality and 79 percent of the gross alcohol-attributable mortality in the European Union (Rehm et al. 2012) and 80 percent of the net alcohol-attributable mortality and 72 percent of the gross alcohol-attributable mortality globally (Rehm et al. 2009) in 2004. This article does not review the other causes of alcohol-attributable deaths included in the latest Comparative Risk Assessment (CRA) Study (Lim et al. 2012). The CRA study estimates as published in December contained significant errors in the calculation of alcohol-attributable cardiovascular deaths, estimating that 33 percent of all ischemic heart disease deaths were attributed to alcohol, which is an impossibility as the relationship between alcohol consumption and this disease category is mainly protective (for details on relationship between alcohol and heart disease, see Roerecke and Rehm 2010, 2012b). A comparison with other alcohol-attributable disease and protective effects will thus only be possible once the corrected CRA results are published.

Methodology Underlying the Estimation of the Mortality Burden of Alcohol-Attributable Diseases and Injuries

The number of alcohol-attributable cancer, liver cirrhosis, and injury deaths in 1990 and 2010 were estimated using alcohol-attributable fractions (AAFs) (Benichou 2001; Walter 1976, 1980). AAFs are calculated by comparing the population risk of a disease under current conditions to a counterfactual scenario where no one has consumed alcohol. This is achieved by using information on the distribution of levels of alcohol consumption and the associated relative risks (RRs) (i.e., risks of disease for different levels of alcohol consumption versus abstainers). These calculated AAFs then were applied to mortality data obtained from the 2010 Global Burden of Disease (GBD) Study for 1990 and 2010 (Lim et al. 2012). Mortality data for 1990 and 2010 were modelled using data on mortality from 1980 to 2010. Data on mortality were imputed for those countries with little or no data by using data from other countries and were smoothed over time (in addition to other data corrections procedures that corrected for cause of death recording errors) (Lozano et al. 2012).

Calculating the Alcohol-Attributable Mortality Burden of Cancer and Liver Cirrhosis

Alcohol consumption is causally related to mouth and oropharynx cancers (ICD-10 codes: C00 to C14), esophageal cancer (C15), liver cancer (C22), laryngeal cancer (C32), breast cancer (C50), colon cancer (C18), and rectal cancer (C20). Alcohol RR functions for cancer were obtained from Corrao and colleagues (2004) (For information about the causal relationship between alcohol and cancer, see Baan et al. 2007; International Agency for Research on Cancer 2010.) The alcohol RR for liver cirrhosis (ICD-10 codes: K70 and K74) was obtained from Rehm and colleagues (Rehm et al. 2010c). The above-noted RRs were modelled based on drinking status and average daily alcohol consumption among drinkers. The same RRs were used to estimate the AAFs by country, sex, and age for 1990 and for 2010.

Alcohol-drinking statuses and adult (people 15 years of age and older) per capita consumption data for 1990 were obtained from various population surveys (Shield et al. 2013b), and the Global Information System on Alcohol and Health (available at: http://apps.who.int/ghodata/?theme=GISAH), respectively. Data on drinking status and adult per
capita consumption for 2010 were estimated by projections (performed using regression analyses) using data from years prior to 2010 (Shield et al. 2013b). Average daily alcohol consumption was modelled using a gamma distribution (Rehm et al. 2010b) and data on per capita consumption for 1990, which was projected to 2010 (Shield et al. 2013b).

(For more information on the methodology of how alcohol consumption was modelled, see Kehoe et al. 2012; Rehm et al. 2010b). This paper presents alcohol consumption data from 2005, the latest year with actual data available.

Calculating the Alcohol-Attributable Mortality Burden of Injuries

The burden of injury mortality attributable to alcohol consumption was modelled according to methodology outlined by Shield and colleagues (2012), using risk information obtained from a meta-analysis (Taylor et al. 2010) and alcohol consumption data from 1990 and 2010. The risk of an injury caused to the drinker over a year was calculated based on alcohol consumed during normal drinking occasions and alcohol consumed during binge-drinking occasions. Alcohol-attributable injuries caused to nondrinkers also were estimated (Shield et al. 2012).

Global Consumption of Alcohol

In 2005 adult per capita consumption of alcohol was 6.1 litres of pure alcohol. Figure 1 shows the adult per capita consumption of alcohol by country. Alcohol consumption per drinker in 2005 was 17.1 litres (9.5 litres per female drinker and 20.5 litres per male drinker). Of all adults, 45.8 percent were lifetime abstainers (55.6 percent of female adults and 36.0 percent of male adults), 13.6 percent were former drinkers (13.1 percent of female adults and 14.1 percent of male adults), and 40.6 percent were current drinkers (31.3 percent of female adults and 49.9 percent of male adults). The global pattern of drinking score (a score from 1 to 5 that measures the harmfulness of alcohol drinking patterns, with 1 being the least harmful and 5 being the most harmful [Rehm et al. 2003b]) was 2.6 in 2005 and ranged from 4.9 for Eastern Europe to 1.5 for Western Europe. Thus, alcohol consumption in Eastern Europe can be characterized by fre-

Figure 1  Adult per capita consumption of pure alcohol by country in 2005.

NOTE: More detailed information can be obtained from the author.
quent heavy alcohol consumption outside of meals and drinking to intoxication.

**Global Alcohol-Attributable Mortality From Cancer**

In 2010, alcohol-attributable cancer caused 337,400 deaths (91,500 deaths among women and 245,900 deaths among men) and 8,460,000 PYLL (2,143,000 PYLL among women and 6,317,000 PYLL among men). This burden is equal to 4.9 deaths per 100,000 people (2.7 deaths per 100,000 women and 7.1 deaths per 100,000 men) and 122.8 PYLL per 100,000 people (62.8 PYLL per 100,000 women and 181.9 PYLL per 100,000 men). Stated another way, alcohol-attributable cancer was responsible for 4.2 percent of all cancer deaths in 2010 and 4.6 percent of all PYLL caused by cancer. Figure 2 shows the number of alcohol-attributable cancer deaths per 100,000 people by region in 2010. Eastern Europe had the highest burden of mortality and morbidity from alcohol-attributable cancer, with 10.3 deaths and 272.0 PYLL per 100,000 people. North Africa and the Middle East had the lowest mortality burden of alcohol-attributable cancer, with 0.6 deaths and 17.1 PYLL per 100,000 people.

In 1990, alcohol-attributable cancer caused 243,000 deaths worldwide (70,700 deaths among women and 172,300 deaths among men) and 6,405,700 PYLL (1,762,200 PYLL among women and 4,643,500 PYLL among men). This mortality burden is equal to 4.6 deaths per 100,000 people (2.7 deaths per 100,000 women and 6.5 deaths per 100,000 men) and 120.8 PYLL per 100,000 people (67.0 PYLL per 100,000 women and 173.9 PYLL per 100,000 men) caused by alcohol-attributable cancer. From 1990 to 2010 the absolute mortality burden of alcohol-attributable cancer (measured in deaths and PYLL) and the rates of deaths and PYLL per 100,000 people have each increased.

**Global Alcohol-Attributable Mortality From Liver Cirrhosis**

In 2010, alcohol-attributable liver cirrhosis was responsible for 493,300 deaths worldwide (156,900 deaths among women and 336,400 deaths among men) and 14,327,800 PYLL.
(4,011,100 PYLL among women and 10,316,800 PYLL among men). This mortality burden is equal to 7.2 deaths per 100,000 people (4.6 deaths per 100,000 women and 9.7 deaths per 100,000 men) and 208.0 PYLL per 100,000 people (117.5 PYLL per 100,000 women and 297.0 PYLL per 100,000 men) caused by alcohol-attributable liver cirrhosis in 2010. Overall, in 2010 alcohol-attributable liver cirrhosis was responsible for 47.9 percent of all liver cirrhosis deaths and 47.1 percent of all liver cirrhosis PYLL. Figure 3 outlines the number of alcohol-attributable liver cirrhosis deaths per 100,000 people by region in 2010, showing strong regional variability.

In 1990, alcohol-attributable liver cirrhosis was responsible for 373,200 deaths worldwide (125,300 deaths among women and 247,900 deaths among men) and 10,906,200 PYLL (3,253,300 PYLL among women and 7,652,900 PYLL among men). That is, 7.0 deaths per 100,000 people (4.8 deaths per 100,000 women and 9.3 deaths per 100,000 men) and 205.7 PYLL per 100,000 people (123.7 PYLL per 100,000 women and 286.6 PYLL per 100,000 men) were caused by liver cirrhosis attributable to alcohol consumption. From 1990 to 2010, the absolute mortality burden of alcohol-attributable liver cirrhosis (measured in deaths and PYLL) and this mortality burden per 100,000 people have each increased (except for women, where alcohol-attributable liver cirrhosis deaths and PYLL per 100,000 decreased slightly).

Global Alcohol-Attributable Mortality From Injury

Globally in 2010, alcohol-attributable injuries were responsible for 669,300 deaths (71,100 deaths among women and 598,200 deaths among men) and 29,110,600 PYLL (3,060,200 PYLL among women and 26,050,400 PYLL among men). This mortality burden is equal to 9.7 deaths per 100,000 people (2.1 deaths per 100,000 women and 17.2 deaths per 100,000 men) and 422.6 PYLL per 100,000 people (89.6 PYLL per 100,000 women and 750.0 PYLL per 100,000 men). Overall, in 2010 alcohol-attributable injuries were responsible for 13.2 percent of all injury deaths and 12.6 percent of all injury PYLL. Figure 4 outlines the number of alcohol-attributable injury deaths per 100,000 people in 2010. Eastern Europe had the greatest mortality burden of alcohol-attributable injuries, with 76.7 deaths and

Figure 3 Alcohol-attributable liver cirrhosis deaths per 100,000 people in 2010 by global-burden-of-disease region.

NOTE: More detailed information can be obtained from the author.
3,484.7 PYLL per 100,000 people, whereas North Africa and the Middle East had the lowest mortality burden, with 2.0 deaths and 117.2 PYLL per 100,000 people.

In 1990, alcohol-attributable injuries were responsible for 485,100 deaths (54,700 deaths among women and 430,400 deaths among men) and 21,934,800 PYLL (2,409,100 PYLL among women and 19,525,700 PYLL among men), equal to 9.2 deaths (2.1 deaths per 100,000 women and 16.1 deaths per 100,000 men) and 413.8 PYLL per 100,000 people (91.6 PYLL per 100,000 women and 731.3 PYLL per 100,000 men). The absolute number of alcohol-attributable injury deaths and PYLL and the number of alcohol-attributable injury deaths and PYLL per 100,000 people each increased from 1990 to 2010.

Appendix 1 presents the number and percentage of alcohol-attributable cancer, liver cirrhosis, and injury deaths and PYLL by GBD study region for 1990 and 2010. Appendix 2 presents the number of alcohol-attributable cancer, liver cirrhosis, and injury deaths per 100,000 people. Unlike figures 1, 2, and 3, the figures in Appendix 2 use the same scale for each cause of death.

Global Alcohol-Attributable Cancer, Liver Cirrhosis, and Injury Mortality As Part of Overall Mortality

In 2010, alcohol-attributable cancer, liver cirrhosis, and injury caused 1,500,000 deaths (319,500 deaths among women and 1,180,500 deaths among men). This represents 2.8 percent of all deaths (1.3 percent of all deaths among women and 4.1 percent of all deaths among men), or 21.8 deaths per 100,000 people (9.4 deaths per 100,000 women and 34.0 deaths per 100,000 men). In 1990, alcohol-attributable cancer, liver cirrhosis, and injury caused 1,101,400 deaths (250,800 deaths among women and 850,600 deaths among men), representing 20.8 deaths per 100,000 people (9.5 deaths per 100,000 women and 31.9 deaths per 100,000 men). The table outlines the mortality burden (measured in deaths and PYLL) of alcohol-attributable cancer, liver cirrhosis, and injury for 1990 and 2010 by age and by sex. Compared with the mortality burden in 1990, the absolute number of alcohol-attributable deaths from cancer, liver cirrhosis, and injury in 2010 is higher, and the rate of deaths per 100,000 also increased for men but decreased slightly for women in 2010.

Figure 4 Alcohol-attributable injury deaths per 100,000 people in 2010 by global-burden-of-disease region.

NOTE: More detailed information can be obtained from the author.
The burden of mortality from alcohol-attributable cancer, liver cirrhosis, and injury led to 51,898,400 PYLL (9,214,300 PYLL among women and 42,684,100 PYLL among men) in 2010 and 39,246,800 PYLL (7,424,600 PYLL among women and 31,822,100 PYLL among men) in 1990. This mortality burden represents 3.0 percent (1.3 percent for women and 4.3 percent for men) of all PYLL in 2010 and 2.0 percent (0.9 percent for women and 2.9 percent for men) of all PYLL in 1990. In 2010, alcohol-attributable cancer, liver cirrhosis, and injury led to 753.4 PYLL per 100,000 people (269.8 PYLL per 100,000 women and 1,228.9 PYLL per 100,000 men) and to 740.4 PYLL per 100,000 people (282.2 PYLL per 100,000 women and 1,191.9 per 100,000 men) in 1990. Again, the overall rates of PYLL per 100,000 people increased, but this effect was attributed to increases for men, coupled with slight decreases for women.

Measurement Limitations

The methods used to estimate the number of alcohol-attributable cancer, liver cirrhosis, and injury deaths and PYLL have limitations as a result of the available data on mortality and the measurement of alcohol consumption and RRs. Most low- and middle-income countries do not have reliable mortality data and measurement of adult mortality in these countries (through verbal autopsies or surveys) is infrequent. Therefore, estimates of mortality and PYLL have a large degree of uncertainty (Wang et al. 2012). Additionally, for high-income countries, information concerning the cause of death has long been acknowledged as containing inaccuracies (James et al. 1955), and more recent studies have confirmed considerable degrees of error in this information (Nashelsky and Lawrence 2003; Shojania et al. 2003). To adjust for inaccuracies and inconsistencies in mortality data,

### Table 1
Deaths and Years of Life Lost (YLL) From Cancer, Liver Cirrhosis, and Injuries Attributable to Alcohol Consumption in 1990 and 2010

<table>
<thead>
<tr>
<th>Year</th>
<th>Gender</th>
<th>Age (Years)</th>
<th>Deaths</th>
<th>% Of All Deaths</th>
<th>YLL</th>
<th>% Of All YLL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>Women</td>
<td>0 to 15</td>
<td>4,000</td>
<td>0.1</td>
<td>324,400</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 to 34</td>
<td>22,300</td>
<td>1.5</td>
<td>1,349,500</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>35 to 64</td>
<td>128,700</td>
<td>2.9</td>
<td>4,437,000</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>65+</td>
<td>95,800</td>
<td>1.0</td>
<td>1,313,800</td>
<td>1.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total</td>
<td>250,800</td>
<td>1.2</td>
<td>7,424,600</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>0 to 15</td>
<td>6,700</td>
<td>0.1</td>
<td>540,400</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 to 34</td>
<td>174,400</td>
<td>8.4</td>
<td>10,547,900</td>
<td>8.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>35 to 64</td>
<td>502,600</td>
<td>7.4</td>
<td>18,167,100</td>
<td>7.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>65+</td>
<td>166,800</td>
<td>1.8</td>
<td>2,566,700</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total</td>
<td>850,600</td>
<td>3.4</td>
<td>31,822,100</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1,101,400</td>
<td>2.4</td>
<td>39,246,800</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>2010</td>
<td>Women</td>
<td>0 to 15</td>
<td>3,800</td>
<td>0.1</td>
<td>313,800</td>
<td>0.1</td>
</tr>
<tr>
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<td>28,800</td>
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<td>162,000</td>
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<td>124,800</td>
<td>0.9</td>
<td>1,587,900</td>
<td>1.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total</td>
<td>319,500</td>
<td>1.3</td>
<td>9,214,300</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>0 to 15</td>
<td>6,100</td>
<td>0.1</td>
<td>492,400</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 to 34</td>
<td>214,900</td>
<td>8.5</td>
<td>12,972,300</td>
<td>8.5</td>
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<td>709,200</td>
<td>7.9</td>
<td>25,549,800</td>
<td>8.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>65+</td>
<td>250,300</td>
<td>1.9</td>
<td>3,669,500</td>
<td>2.2</td>
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<tr>
<td></td>
<td></td>
<td>Total</td>
<td>1,180,500</td>
<td>4.1</td>
<td>42,684,100</td>
<td>4.3</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1,500,000</td>
<td>2.8</td>
<td>51,898,400</td>
<td>3.0</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** More detailed information can be obtained from the author.
the 2010 GBD study modelled the number of deaths mathematically (Wang et al. 2012).

Survey data measuring alcohol consumption, patterns of alcohol consumption, and the prevalence of lifetime abstainers, former drinkers, and current drinkers also are susceptible to numerous biases (Shield and Rehm 2012). To correct for the undercoverage that is observed when alcohol consumption is measured by population surveys (as compared with per capita consumption of alcohol), alcohol consumption was modelled by triangulating per capita and survey data (see above). Total alcohol consumption was set to 80 percent of per capita consumption in order to account for alcohol produced and/or sold, but not consumed, and to account for the undercoverage of the alcohol consumption typically observed in studies that calculate the alcohol RRs (Rehm et al. 2010b). Additionally, although alcohol was measured using adult per capita consumption and most people 14 years and younger do not consume alcohol or binge regularly, some adolescents 10 to 14 years of age report previously trying alcohol and previously been intoxicated (Windle et al. 2008).

The CRA was based on alcohol RR functions that typically were differentiated by sex and adjusted for age, smoking status, and other potentially confounding factors. The use of adjusted RR functions may introduce bias into the estimated number of deaths and PYLL that would not have occurred if no one had ever consumed alcohol (Flegal et al. 2006; Korn and Graubard 1999; Rockhill and Newman 1998). However, most of the published literature on alcohol-as-a-risk-factor–only reports adjusted RRs, and, thus, the use of unadjusted alcohol RRs for the CRA study would have led to imprecise estimates as a result of leaving out most of the studies. The bias of using adjusted RRs is likely to be small, as most analyses of the estimated RRs show no marked differences after adjustment for the potentially confounding factors and effect measure modifiers. Future CRA studies may require more complex modelling techniques for alcohol if other dimensions of alcohol consumption, such as irregular heavy-drinking occasions, impact RR estimates.

Finally, this analysis did not account for a lag time for the calculation methods used in this paper. This is especially a problem for cancer, which has a lag time of 15 to 20 years (Holmes et al. 2012; Rehm et al. 2007). In other words, the alcohol-attributable deaths and PYLL in 2010 actually are based on consumption patterns from 1990 to 1995, but in this paper were estimated based on consumption in 1990 and 2010. Although liver cirrhosis also is a chronic disease that develops over time like cancer (Rehm et al. 2013a), the impact of population-level consumption on liver cirrhosis deaths can be quite abrupt. For example, Gorbachev’s anti-alcohol campaign was reflected in a clear reduction in Russia’s liver cirrhosis mortality (Leon 1997). Likewise, the German seizure of alcohol in France in World War II led to reduced cirrhosis mortality (Zatonski et al. 2010). Most of the effect of alcohol consumption on liver cirrhosis probably is captured within 1 year (Holmes et al. 2012). For injury, with the exception of suicide, there is no noticeable lag time as the risk of injury is associated with blood alcohol content (Taylor and Rehm 2012; see also Cherpitel 2013).

Implications of Alcohol-Attributable Mortality

In 1990 and in 2010, alcohol consumption had a huge impact on mortality. Regions such as Europe (especially Eastern Europe) and parts of Sub-Saharan Africa (especially south Sub-Saharan Africa) that have a high per capita consumption of alcohol and detrimental drinking patterns are more affected by alcohol consumption compared with other regions. It is important to note that the alcohol-attributable mortality burden is composed of two elements: AAF and the overall mortality burden of the respective disease. Accordingly, the observed overall increase from 1990 to 2010 in alcohol-attributable cancer, liver cirrhosis, and injury deaths and in PYLL can be attributed to two different sources: (1) an increase in the number of cancer, liver cirrhosis, and injury deaths (mainly attributed to increases of these deaths in low- to middle-income countries) (Lozano et al. 2012) and (2) an increase in alcohol consumption in low- to middle-income countries (Shield et al. 2013b).

Low- and middle-income countries have higher rates of alcohol-attributable mortality per 100,000 people, even though these countries typically have lower AAF (as their overall burden of mortality is higher). Economic wealth is correlated with overall mortality (Lozano et al. 2012), and, thus, the mortality burden per litre of alcohol consumed is highest in low-income countries, followed by middle-income countries (Rehm et al. 2009; Schmidt et al. 2010). It follows, therefore, that increases in the alcohol-attributable mortality burden in low- and middle-income countries attributed to economic growth may be able to be reduced or controlled for by implementing alcohol control policies such as taxation (Shield et al. 2011; Sornpaisarn et al. 2012a, b, 2013), bans on advertising, and restrictions on availability (Anderson et al. 2009; World Health Organization 2011b) preferably while maintaining the relatively high levels of abstention in these countries.

The typical causes of death associated with alcohol use disorders are liver cirrhosis and injuries, (i.e., exactly the categories described in this article). Liver cirrhosis and injuries, and to a lesser degree cancer, may primarily be responsible for the high proportion of alcohol-attributable mortality explained by alcohol use disorders (Rehm et al. 2013b); however, additional research is required to empirically confirm this hypothesis. By increasing the rate of treatment for alcohol use disorders (Rehm et al. 2013b), the mortality burden of alcohol-attributable diseases also can be reduced. Recent research has shown that the mortality burden associated with alcohol use disorders, albeit high, has been underestimated (see Harris and Barraclough 1998 for the first meta-analysis; and Callaghan et al. 2012; Campos et al. 2011; Guitart et al. 2011; Hayes et al., 2011; Saieva et al. 2012; Tikkanen et al. 2012; Taylor and Rehm 2012; see also Cherpitel 2013).
2009 for recent papers that observed a markedly higher mortality risk than in the first meta-analysis).

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Neufeld, M., and Rehm, J. Alcohol consumption and mortality in Russia since 2000: Are there any changes following the alcohol policy changes starting in 2006? Alcohol and Alcoholism 48:222-230, 2013. PMID: 23299570


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The Alcohol Policy Information System (APIS) is an NIAAA-sponsored Web site that provides detailed information on alcohol-related public policies at both the State and Federal levels. Updated annually, the APIS information can be used to identify policy changes in 33 policy areas. Up to two-thirds of these policies can be tracked back to 1998, and data on the remaining one-third are available since 2003.

APIS provides a tool for conducting research on the effectiveness of alcohol policies in reducing alcohol-related harm. Natural experiments on the impact of a policy are possible whenever a State makes a change in some aspect of alcohol policy. To conduct such an experiment, scientists obtain a series of regularly collected data on an outcome of interest, compare problem levels before and after a policy change, and report this comparison for both the “experimental” State and a “control” State that did not change its policy. As described in other articles in this issue, measures of policy outcomes can be taken from a number of existing data series that report rates of mortality, morbidity, accidents, crimes, and the like. APIS provides an accurate and consistently maintained source of the “independent variable” of interest in these studies, showing changes in codified statutes and regulations.

As the lead Federal agency responsible for research into alcohol use and its related consequences, NIAAA believes that providing this research tool is critically important. A vigorous program of research on the

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It should be noted that enforcement efforts affect the effectiveness of any given policy. However, there is, at present, no method of measuring State-by-State enforcement efforts that are both broadly recognized as valid by policy researchers or economically feasible to gather. Although the Institute encourages the development of better methods to measure enforcement, APIS currently is not able to provide this information.

Figure The APIS Web site (www.alcoholpolicy.niaaa.nih.gov) features three basic displays.
effects of various drinking-and-driving policies has been a key factor in reducing alcohol-related traffic fatalities to drivers, passengers, and pedestrians, ages 16 to 20 by 50 percent since 1982. By providing information on other aspects of alcohol policy, APIS can lead to similar advances in research knowledge and improvements in public health.


New topics are added periodically and are selected on the basis of the likelihood that they will stimulate new research on policy outcomes. Candidate policy topics should cover areas where recent policymaking activity has occurred or where current discussions imply that impending change is likely. Additional criteria include (1) a scientifically grounded expectation that policy change in the particular candidate area is likely to produce measurable changes in some public health outcome; (2) sufficient data are available to measure these policy outcomes; (3) the legal research involved in locating relevant statutes and regulations and in constructing precise and consistent operational definitions of the laws is a manageable task that falls within the resources available; and (4) case law or administrative decision making that is not captured in statutory or regulatory texts plays a limited role in interpreting the policy.

Documenting the decision rules for categorizing the features of State policies is a critical concern. This documentation must capture the decisions made by the APIS research staff in such a way that consistent decisions can be made across all States, consistent decisions can be made across time as policies change, and the basis for the decisions can be clearly communicated to research users.

In addition to strict documentation, a hallmark of APIS has been careful attention to quality control. Each new law or regulation is reviewed by a minimum of two research lawyers and a senior social scientist. Any conflicting interpretations are then resolved. On occasion, new developments in some detail of a policy reveal a gap or an ambiguity in the coding procedures that were used previously. In such cases, that coding is reviewed and, if necessary, changes are made. Likewise, when errors are encountered in APIS’s legal descriptions, those corrections are made and documented in a “Change Log.” Fortunately, the number of such instances has remained remarkably low.

Policy changes are reviewed each year for each topic and for every State. Shortly after the beginning of each calendar year, legal tools become available that make it possible to identify all of the codified statutory and regulatory activity that has occurred in a given State over the preceding year. APIS legal researchers sift through these policies to see if changes have occurred in any of the 32 policy areas tracked by APIS. Typically, the most difficult aspect of this work is “demonstrating the negative,” that is, verifying that there has not been a change in some specific aspect of a policy. This work is costly and has become the largest part of the APIS contract budget.

The APIS Web site (www.alcoholpolicy.niaaa.nih.gov) features three basic displays (see figure):

- “Data on a Specific Date” shows the features of a given policy as of the date of the last system-wide update (the default value) or as of a specific date selected by the viewer. Any given APIS policy will vary across States in a number of specific features. Understanding these details is important.

- “Changes Over Time” contains entries whenever a State changes its policy. There is one row showing the policy before the change and another showing the policy afterward. Should a State make additional policy changes, even more rows are added.

- “Timeline of Policy Changes” displays calendar years on the horizontal axis and States on the vertical axis. Icons indicate the year (and quarter) when a policy change went into effect. Note that for all APIS information, policies are categorized according to the date when they took effect rather than when they were enacted.

There are a number of other useful Web site features and displays. The “Maps and Charts” feature shows graphic depictions of selected information. An “About This Policy” section gives useful background information on each policy. “State Profiles of Underage Drinking Laws” contains brief State-by-State summaries of policy in 12 topic areas specifically related to underage drinking. “Citations” are given so that the statutory or regulatory text of each policy covered by the Web site can be located. Finally, there are links to NIAAA’s current funding opportunity announcements, inviting researchers to submit proposals to study policy impacts. NIAAA encourages all researchers who are interested in the effect of public policy on alcohol-related health outcomes to visit the APIS Web site and to consider applying for research support.
The Burden of Alcohol Use

Focus on Children and Preadolescents

John E. Donovan, Ph.D.

The study of alcohol use by children ages 12 and younger has been very limited. This article summarizes information from U.S. national and statewide surveys on the prevalence of alcohol use among children in grades 6 and lower, data on health conditions wholly attributable to alcohol, the prevalence of children’s treatment admissions for alcohol abuse, and their rates of presentation at emergency departments for acute alcohol intoxication. Factors hampering the estimation of alcohol burden in this population include the lack of ongoing national surveys of alcohol use and problems in children, the hand-me-down nature of alcohol assessments in this population, and the lack of studies to establish whether there is a causal relationship between childhood-onset drinking and morbidity and mortality in adolescence and later in life that would permit determination of alcohol-attributable fractions. This article concludes that although the alcohol burden in childhood is low, it may be augmented by both referred alcohol burden through parental drinking and alcohol abuse and by deferred alcohol burden from longer-term consequences of early use.

Factors Limiting Estimation of Alcohol Burden

The Absence of Recent National Surveillance Data

Chief among the factors inhibiting the estimation of alcohol burden in children and preadolescents is the absence of ongoing national surveillance data. The prevalence of child alcohol use can theoretically be estimated from either adolescents’ retrospective recall of their alcohol use in childhood or from survey research with children.

Retrospective reports of the age at first drink, however, are not very reliable for this life stage. Typically, reported age of onset of alcohol use increases as a function of the age of the adolescents questioned (Bailey et al. 1992; Engels et al. 1997; Johnson et al. 1998; Labouvie et al. 1997; Parra et al. 2003). For example, in the most recent national data from the 2009 Youth Risk Behavior Survey (YRBS), 28.1 percent of 9th graders reported that they drank alcohol before age 13, compared with 14.2 percent of 12th graders (Eaton et al. 2010). These are not cohort effects but rather evidence of “forward telescoping,” as shown by the fact that although the percentages at all grades have declined over time, a similar pattern can be seen in each of the previous YRBS surveys (1991–2007). This pattern also is evident in the 1993–2010 national data from the Monitoring the Future (MTF) surveys (see figures 6 to 20 in Johnston et al. 2011): in every year, less than one-half as many 12th graders as 8th graders report alcohol use initiation by grade 6. Thus, estimates based on retrospective recall are problematic as a summary of the prevalence of childhood drinking.

Direct surveys of children constitute a more appropriate approach for capturing normative data on child drinking. However, of the three major ongoing Federally sponsored national surveys in the United States—the annual MTF survey, the biennial YRBS, and the annual National Survey on Drug Use and Health (NSDUH)—only the NSDUH includes children who are age 12, and none includes children younger than 12. According to the 2010 NSDUH results (Tables 2.15B and 2.16B in Substance Abuse and Mental Health Services Administration [SAMHSA] 2011), 7.1 percent of 12-year-olds had ever had a drink of alcohol (i.e., a can of beer, a glass of wine, or a shot of liquor) in their life, 4.4 percent had a drink in the past year, 1.6 percent had a drink in the past month, and 0.4 percent had consumed five or more drinks on the same occasion.

Despite the absence of children in these ongoing Federal surveillance studies, preliminary information on the prevalence of alcohol use in children has nevertheless been compiled.
through a comprehensive search of internet sources to locate Nationwide and Statewide surveys of children in grades 6 and below (see Donovan 2007). Based on this review of the four Nationwide and seven Statewide datasets located, it is clear that a substantial number of children have had some exposure to alcohol. Data from the cross-national Health Behaviour of School-Aged Children Study (Nie Gabhainn and François 2000) indicate that in a 1998 national sample of 11-year-old U.S. students, 62 percent of boys and 58 percent of girls had ever tasted alcohol, 8 percent of boys and 7 percent of girls had consumed alcohol at least weekly, and 3 percent of both boys and girls had ever been drunk twice or more. According to the 1999 Partnership Attitude Tracking Study (sponsored by the Partnership for a Drug-Free America), which surveyed a national probability sample of nearly 2,400 U.S. elementary-school students, 9.8 percent of 4th graders, 16.1 percent of 5th graders, and 29.4 percent of 6th graders had had more than just a sip of alcohol in their life. In 2000–2001, the National Survey of Parents and Youth (NSPY) collected alcohol use information on 1,560 9- to 12-year-olds and found that 6.2 percent of 9-year-olds, 5.5 percent of 10-year-olds, 9.2 percent of 11-year-olds, and 15.5 percent of 12-year-olds had had more than a few sips of alcohol in their life. Data on alcohol use in the past year (rather than lifetime) are reported annually by Pride Surveys (see www.pridesurveys.com): according to the 2009–2010 summary of school-district surveys performed across the United States, 4.0 percent of 4th graders, 4.8 percent of 5th graders, and 8.3 percent of 6th graders had drunk alcohol in the past year. Both the Nationwide and Statewide datasets examined showed a decline in the prevalence of child drinking over the past 10 years or so. The datasets located for this review, however, generally are either outdated or nonrepresentative, and their limitations must be recognized in any attempt to estimate the burden of alcohol use in this population. The absence of any recent national survey of alcohol use among children argues for the need to institute ongoing Nationwide surveillance of this population.

It is nevertheless evident, however, that the percentage of children who have experience with alcohol decreases as the intensity of alcohol involvement increases (from a sip or taste to more than a few sips ever to use in the past year, past month, or past week), and that it differs as a function of grade, gender, and ethnicity (see Donovan 2007). Alcohol use rates increased with age, doubling between grades 4 and 6, with the largest jump in prevalence between grades 5 and 6. At each grade level, boys were more likely to have used alcohol than girls. African-American children were nearly as likely as white and Hispanic children to have used alcohol. About one-third as many children reported having had more than a sip of alcohol as reported having had only a sip. In general, around one-third of children who had ever used alcohol reported its use in the past year as well, and use in the past month occurred in only about one-third of those children who reported use in the past year.

There are few current Nationwide data sources on the prevalence of children's experience of problems attributed to alcohol use that could inform estimates of their wholly alcohol-attributable health conditions (i.e., alcohol dependence and acute intoxication). Several community-level studies suggest that rates of alcohol use disorders are close to zero prior to adolescence (Cohen et al. 1993; Giaconia et al. 1994; Sung et al. 2004). The low number of Nationwide admissions for treatment of alcohol abuse at ages 10–12 bears this out (see figures 14 and 15 in SAMHSA 2008). Patients under the age of 15 constitute just 0.5 percent of those admitted for treatment of alcohol abuse alone and 0.7 percent of those admitted primarily for treatment of alcohol abuse who also had abused another drug (Table 3.2a in SAMHSA 2008).

Likewise, in contrast to adolescents, children rarely present at hospital emergency departments for acute intoxication (alcohol poisoning). In 2009, the rate of visits to emergency departments for acute alcohol intoxication was 5.6 per 100,000 for U.S. children ages 0–5 and 1.0 per 100,000 for children ages 6–11 versus 310.8 per 100,000 for adolescents ages 12–17 (Drug Abuse Warning Network 2010). Of all calls to poison-control centers in the United States in 2009 involving children ages 5 or younger, 2.12 percent of cases involved ingestion of alcohol (Bronstein et al. 2010). This probably is an underestimate, as many children ingested products such as cold medicines, cologne, perfume, aftershave, and mouthwash that contain ethanol (see Vogel et al. 1995).

In summary, there are few surveillance studies of alcohol use and alcohol-related problems among children and preadolescents. The extant data indicate that although the rates of alcohol use are low in this population, substantial numbers of children do have experience with alcohol and the rates of wholly alcohol-attributable health conditions are very low in this population. No evidence has been generated regarding the influence of child drinking on other diseases or injuries within childhood.

**Problems of Measurement**

A second major limitation for estimating alcohol burden in this population is the widespread use of “hand-me-down” measures for the assessment of children's alcohol use. Measures originally developed for use with adults have been modified for use with college students; then modified for use with adolescents; and, finally, modified for assessment of children. Reliance on such hand-me-down assessments has resulted, for instance, in only limited research into sipping and tasting of alcohol despite the fact that this is the most common form of children's experience with alcohol (see Caswell 1996; Caswell et al. 1991; Donovan and Molina 2008; Johnson et al. 1997).

The hand-me-down nature of child and adolescent assessments is nowhere more evident than in the case of heavy episodic (binge) drinking, a major contributor to adult morbidity and mortality. In adults, binge drinking has been operationally defined as five or more drinks per occasion for...
men and as four of more drinks per occasion for women (Wechsler et al. 1995); these levels of intake result in blood alcohol concentrations (BAC) of 0.08 percent (the legal definition of intoxication) if consumed within a 2-hour window. Using these definitions for children and adolescents is inappropriate, however, because they weigh less and thus have smaller volumes of total body water than adults. A recent analysis (Donovan 2009) modified the Widmark equation for estimating BAC so it would be more developmentally appropriate. This was done by incorporating formulas for estimating total body water that were derived from children and adolescents and by using ethanol elimination rates derived from child and adolescent presentations for acute alcohol intoxication at emergency departments. BAC estimates were calculated for intake of from one to five standard drinks for boys and girls separately at each age from 9 to 17 to determine how many drinks were required to result in an estimated mean BAC of 0.08 percent or higher. Data from more than 4,700 children and adolescents from the 1999–2002 National Health and Nutrition Examination Survey were analyzed. Girls and boys ages 9–13 had mean estimated BACs of 0.08 percent or higher at three or more drinks, as did girls ages 14–17. Boys ages 14 and 15 had mean estimated BACs of 0.08 percent or higher at four or more drinks, and boys ages 16 and 17 reached this level at five or more drinks. Table 1 summarizes the resulting recommendations for defining binge drinking for children and adolescents by age and gender. Only boys ages 16 or 17 met the adult definition.

In addition to the concern over hand-me-down assessments, there is a lack of consensus on the definition of the various levels of alcohol involvement for both children and adolescents. As is evident in the summary of survey studies above, drinker status was defined variously as consumption of more than a sip, more than a few sips, or a whole drink. This severely hinders the performance of meta-analyses across studies and the description of trends over time. Bacon (1976) noted a similar lack of consensus 35 years ago.

In general, evidence from both test–retest examinations and collateral reports suggests that children’s self-reports of their alcohol use are as valid as adolescent self-reports (Dielman et al. 1995; Donovan et al. 2004). Given their typically low levels of intake and the opportunistic nature of their drinking, misreporting in child reports of their alcohol involvement is unlikely to reflect cognitive overload. More likely, difficulties stem from a lack of familiarity with alcohol beverage types (beer versus liquor, for example) and with estimation of drink volumes consumed. At least one recently developed inventory uses pictorial images to assess alcohol and drug use and their risk factors (see Andrews et al. 2003; Ridenour et al. 2009, 2011).

In addition to making child alcohol assessments more developmentally appropriate and user friendly, surveillance studies of child alcohol use need to be expanded to include questions on the intensity and patterning of their current alcohol use (e.g., frequency of use, usual and greatest intake, frequency of binge drinking, and contexts of use).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Recommended Cut Points (Number of Drinks) for Developmentally Appropriate Definition of Binge Drinking in Children and Adolescents (Donovan 2009)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td><strong>Boys</strong></td>
</tr>
<tr>
<td>9–13</td>
<td>3+</td>
</tr>
<tr>
<td>14–15</td>
<td>4+</td>
</tr>
<tr>
<td>16–17</td>
<td>5+</td>
</tr>
</tbody>
</table>

**Barriers to Collecting Child Data**

Although monitoring the Nationwide prevalence of children’s alcohol use would constitute a step in the right direction, increased research also is needed. It is possible that so few studies have been conducted in this area because of the perception of several barriers to such research (see Donovan 2007). One perceived barrier is that few children drink, so there is little variation to explain. A second is the difficulty of gaining school-district approval to access elementary school populations, necessitating the use of targeted-age directory sampling or household enumeration sampling methods. A third barrier sometimes raised is the misapprehension that parents will be reluctant to consent to their children’s participation in alcohol research.

**Referred Childhood Alcohol Burden Through Parent Drinking**

Parents contribute to the alcohol burden of their children in a variety of ways. First, they model drinking behavior for good or for ill. National surveys show that the majority (87.9 percent) of adults in the United States ages 26 and older have ever drank, 69.0 percent drank in the past year, and 54.9 percent drank in the past month (Table 2.37B in SAMHSA 2011). Children learn about alcohol and its effects and usages from observing their parents drinking or from hearing their parents talk about their drinking, as well as from their exposure to drinking in the larger social environment (e.g., relatives, peers and their families, neighborhood events, alcohol commercials on TV and radio, magazine ads, Internet Web sites, social media, and drinking in movies and even in animated feature films) (see Zucker et al. 2008, 2009). Children whose parents drink are more likely to initiate early use (Donovan and Molina 2008, 2011; Hawkins et al. 1997).

Second, parents actively teach their children about alcohol. Children are first introduced to alcohol use by parents or other relatives in a family context (see Jackson 1997; Jahoda and Cramond 1972; Johnson et al. 1997). Such precocious socialization into alcohol use can reflect either Old World cultural beliefs regarding the role of alcohol as food or as a necessary adjunct for celebrations or the belief that introducing
children to alcohol use as part of family dinners or events serves to inoculate them from later involvement in problem drinking. Research has not yet established, however, whether learning to drink in a family context actually serves to protect children from developing later alcohol problems. The relevant longitudinal research (Dielman et al. 1989; McMorris et al. 2011; van der Vorst et al. 2010) suggests that this is not the case: prior supervised drinking increases the likelihood of unsupervised drinking and more negative consequences later on. In addition, children who were permitted to drink alcohol at home have been found to show increased alcohol involvement and drunkenness over time (Jackson et al. 1999; Komro et al. 2007). Research also shows that European adolescents, who are more often introduced to alcohol in family contexts, typically are more likely to be involved in binge drinking and intoxication than U.S. adolescents of the same age (Currie et al. 2008; Friese and Grube 2010; Grube 2009).

Third, the home environment is the most popular source of alcohol for children. Among 6th-grade students who had ever had alcohol, the largest percentage (32.7 percent) obtained the alcohol from a parent or guardian the last time they drank (Hearst et al. 2007). Other adults become a more important source of alcohol than parents as children move into adolescence. Greater access to alcohol in the home and greater parental provision of alcohol are associated with greater alcohol intake and problems later on (Komro et al. 2007; van den Eijnden et al. 2011).

In addition to their direct impact on child drinking, parental drinking and alcohol abuse may increase child morbidity and mortality through other means as well. Children also may be placed at increased risk through prenatal exposure to maternal drinking (Jacobson and Jacobson 2002; Mattson et al. 2001; Rasmussen 2005; Richardson et al. 2002; Streissguth et al. 1999); through genetic inheritance of liabilities to alcohol abuse and related addictive behaviors (Schuckit 1994; Sher 1991; Zucker et al. 2003); through alcohol-impaired parenting, abuse, and neglect (Bijur et al. 1992; Dube et al. 2001; Kelleher et al. 1994); and through their adoption of parent-socialized alcohol-specific intentions, attitudes, and expectancies (e.g., Donovan et al. 2009; Handley and Chassin 2009; Tildesley and Andrews 2008), leading to both short-term and longer-term consequences. In addition, children are at risk of injury or death through riding in cars driven by an alcohol-impaired parent: in 2009 alone, 14 percent of the children ages 14 and younger killed in traffic crashes were killed in alcohol-impaired driving crashes, and one-half of these children were passengers in vehicles driven by a driver with a BAC of 0.08 percent or higher (U.S. Department of Transportation 2011).

Deferred Childhood Alcohol Burden Through Long-Term Consequences

The measurable burdens of child and preadolescent drinking are for the most part postponed into adolescence and young adulthood. Early onset of alcohol use predicts involvement in alcohol problems, alcohol abuse, and alcohol dependence in adolescence (Gruber et al. 1996; Hawkins et al. 1997; Horton, 2007; McGue and Iacono, 2005; Pederson and Skrondal, 1998; Warner et al. 2007). Early-onset drinking also relates to a variety of other problematic outcomes in adolescence, including absences from school, delinquent behavior, drinking and driving, smoking, marijuana and other illicit drug use, sexual intercourse, and pregnancy (Ellickson et al. 2001; Gruber et al. 1996; McCluskey et al. 2002; Stueve and O’Donnell, 2005).

There also is evidence that early initiation of alcohol use affects a number of outcomes in young adulthood as well. These young-adult outcomes include not only alcohol use disorder (e.g., Hingson et al. 2006; King and Chassin, 2007) but also prescription drug misuse (Hermos et al. 2008), substance use disorders (Hingson et al. 2008; King and Chassin, 2007), employment problems (Ellickson et al. 2003), unintentional injuries (Hingson and Zha 2009; Hingson et al. 2000), and risky driving and drinking and driving (Hingson et al. 2002; Zakrjesk and Shope 2006). Retrospective data from adults also have shown a relationship between earlier onset of drinking and lifetime experience of an alcohol use disorders (e.g., DeWit et al. 2000; Grant and Dawson 1997). Research currently is lacking, however, on whether early-onset drinking relates to psychosocial functioning in other young-adult life areas, such as educational, occupational, marital, social, political, and community functioning, and relationship with parents.

As yet, there are few studies of the mechanisms linking early-onset drinking to young-adult alcohol problems and other negative outcomes. McGue and Iacono (2008) see this linkage as emanating from the interrelations between early drinking and other problem behaviors in adolescence (Donovan and Jessor 1985) and the stability of this syndrome into young adulthood (Jessor et al. 1991), which is seen as reflecting both inherited vulnerability and the influence of early problem behavior on the selection of risky social environments. Identification of such underlying mediating mechanisms is an important component of establishing any causal linkage between early-onset drinking and these later outcomes that would inform estimation of their alcohol-attributable fractions (Rehm et al. 2010). The greater the role of mediating variables in this pathway, the smaller the alcohol-attributable fraction is likely to be.

Conclusions

In summary, there are few surveillance studies of alcohol use and alcohol-related problems among children and preadolescents, a situation that makes estimation of alcohol burden in this population problematic. The available data indicate that whereas the rates of alcohol use are relatively low in this population, substantial numbers of children do in fact have experience with alcohol. With respect to wholly alcohol-
attributable health conditions, the available data suggest very low levels of alcohol abuse and acute intoxication among children. The scattered and inaccessible nature of much of this available data highlights the need for better ongoing surveillance of this population. Although these direct assessments imply that alcohol burden in children is relatively low, their alcohol burden is increased through the alcohol use and abuse of their parents, and through the increased likelihood among early drinkers of alcohol problems and other negative outcomes in adolescence and young adulthood.

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References


Prevalence and Predictors of Adolescent Alcohol Use and Binge Drinking in the United States

Megan E. Patrick, Ph.D., and John E. Schulenberg, Ph.D.

Because alcohol use typically is initiated during adolescence and young adulthood and may have long-term consequences, the Monitoring the Future (MTF) study annually assesses various measures of alcohol use among 8th-, 10th-, and 12th-grade students. These analyses have found that although alcohol use among these age groups overall has been declining since 1975, levels remain high. Thus, in 2011 about one-quarter of 8th graders, one-half of 10th graders, and almost two-thirds of 12th graders reported drinking alcohol in the month preceding the interview. Binge drinking (i.e., consumption of five or more drinks in a row) was also prevalent. Specific rates of drinking, binge drinking, and getting drunk varied among different student subgroups based on gender and race/ethnicity. The MTF study has also identified numerous factors that influence the risk of alcohol use among adolescents, including parents and peers, school and work, religiosity and community attachment, exercise and sports participation, externalizing behavior and other drug use, risk taking and sensation seeking, well-being, and drinking attitudes and reasons for alcohol use. Drinking during adolescence can have long-term effects on a person’s life trajectory. Therefore, these findings have broad implications for prevention and intervention efforts with this population.

Key words: Underage drinking; binge drinking; adolescent; high school student; young adult; prevalence; predictors; causes of alcohol and other drug use; risk factors; school risk factors; environmental risk factors; family risk factors; peer risk factors; gender differences; racial/ethnic differences; Monitoring the Future (MTF) Study; United States

In the United States, alcohol use typically begins and escalates during adolescence and young adulthood. To describe the historical and developmental trends in substance use in this age group, the Monitoring the Future (MTF) study (Johnston et al. 2012) was designed in 1975. Since then, this ongoing national-cohort sequential longitudinal study assessing the epidemiology and etiology of substance use among adolescents and adults has been funded by the National Institute on Drug Abuse (NIDA). This article summarizes findings from the MTF study regarding the prevalence and predictors of alcohol use during adolescence.

The Prevalence of Drinking and Historical Changes

As is true for adults, alcohol is the most commonly abused substance among American youth. The MTF study has been documenting the prevalence and trends in alcohol use frequency and binge drinking (i.e., consumption of five or more drinks in a row) for the past several decades in annual, national samples of American 8th-, 10th-, and 12th-grade students. Using these data, Patrick and Schulenberg (2010) found that very few 8th- and 10th-grade students who reported having ever used alcohol had not used alcohol in the past year, suggesting that most of the alcohol use reported is relatively recent. Therefore, this article focuses on alcohol use in the past 12 months and the past 30 days, as well as self-reported drunkenness in the past 30 days and binge drinking in the past 2 weeks. The prevalence figures for these variables for 2011 are summarized in table 1, broken down by grade level, gender, and racial/ethnic subgroups (for more information, see Johnston et al. 2012).

In 2011, 27 percent of 8th graders, 50 percent of 10th graders, and 64 percent of 12th graders reported having used alcohol in the past 12 months. The corresponding rates for alcohol use in the past 30 days were 13 percent, 27 percent, and 40 percent, respectively. Furthermore, 4 percent of 8th graders, 14 percent of 10th graders, and 25 percent of 12th graders reported having been drunk within the past month. Finally, binge drinking in the past 2 weeks was reported by 6 percent of 8th graders, 15 percent of 10th graders, and 22 percent of 12th graders.

Interestingly, it is more common for students to report binge drinking 2 or more times in the past 2 weeks than to report binge drinking only once in the past 2 weeks; thus, 61 percent of 8th graders and 62 percent of 10th graders who had engaged in binge drinking in the previous 2 weeks did so on multiple occasions (Patrick and Schulenberg 2010). This observation suggests a fast shift to frequent heavier drinking for many young people. In addition, the surveys indicate that extreme binge drinking (i.e., consumption of 10 or more or 15 or more drinks in a row) is a problem among 12th graders (this variable was not assessed among 8th and 10th graders). Thus, 10.5 percent of high school seniors reported consuming 10 or more drinks in a row, and 5.6 percent reported consuming 15 or more drinks in a row in the past 2 weeks (Patrick et al. 2013).

Alcohol use differs not only by age but also by demographic subgroups, including gender and race/ethnicity.

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(see table 1). In 8th grade, girls tend to have somewhat higher rates of alcohol use (i.e., 13 percent in the past 30 days) than do boys (12 percent). Among older students, however, this ratio is reversed, with 38 percent of female and 42 percent of male 12th graders reporting alcohol use in the past 30 days. This gender difference continues into adulthood, with men consistently using alcohol at higher rates compared with women (Johnston et al. 2012; Wilsnack et al. 2000). A similar interaction seems to exist between grade and race/ethnicity (Wallace et al. 2003). Thus, among 8th graders, Hispanic youth tend to report a greater prevalence of alcohol consumption in the last 12 months (36 percent) or last 30 days (18 percent), as well as of being drunk in the last 30 days (6 percent) and binge drinking in the past 2 weeks (10 percent) than do both White and African American youth. By 12th grade, however, White adolescents have the highest prevalence levels of the three racial/ethnic groups on all alcohol use measures, African American adolescents have the lowest levels, and Hispanics have intermediate levels. For example, for binge-drinking, prevalence rates among 12th graders are 26 percent for Whites, 11 percent for African Americans, and 21 percent for Hispanics. Some, but not all, of these race/ethnicity differences in alcohol use among 12th graders are attributable to differential high-school dropout rates among the different groups. Thus, dropout rates tend to be higher among racial/ethnic minority youth, and alcohol and other drug (AOD) use tends to be higher among school dropouts than among those staying in school (Bachman et al. 2008).

<table>
<thead>
<tr>
<th>Table 1 Prevalence of Alcohol Use (%) by Demographic Subgroups in 8th, 10th, and 12th Graders, 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Any Use in Past 12 Months</strong></td>
</tr>
<tr>
<td>-----------------------------</td>
</tr>
<tr>
<td><strong>8th Graders</strong></td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Boys</td>
</tr>
<tr>
<td>Girls</td>
</tr>
<tr>
<td>Race/Ethnicity*</td>
</tr>
<tr>
<td>White</td>
</tr>
<tr>
<td>African American</td>
</tr>
<tr>
<td>Hispanic</td>
</tr>
<tr>
<td><strong>10th Graders</strong></td>
</tr>
<tr>
<td>Total</td>
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<tr>
<td>Gender</td>
</tr>
<tr>
<td>Boys</td>
</tr>
<tr>
<td>Girls</td>
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<tr>
<td>Race/Ethnicity</td>
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<tr>
<td>White</td>
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<tr>
<td>African American</td>
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<tr>
<td>Hispanic</td>
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<tr>
<td><strong>12th Graders</strong></td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Boys</td>
</tr>
<tr>
<td>Girls</td>
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<tr>
<td>Race/Ethnicity*</td>
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<tr>
<td>White</td>
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<tr>
<td>African American</td>
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<tr>
<td>Hispanic</td>
</tr>
</tbody>
</table>

*To derive percentages for each racial subgroup, data for the specified year and the previous year were combined to increase subgroup sample sizes and thus provide more stable estimates. NOTE: For 8th graders, the approximate weighted N is 16,000. For 10th graders, the approximate weighted N is 14,900. For 12th graders, the approximate weighted N is 14,100. SOURCE: The Monitoring the Future study, the University of Michigan.
Overall, alcohol use among adolescents and young adults has been declining to the lowest levels in recent decades, as shown by the trends in self-reported alcohol use in the past 12 months and drunkenness in the past 30 days (see figure 1) (Johnston et al. 2012; Patrick and Schulenberg 2010). Similar trends have been observed for alcohol use in the past 30 days and binge drinking in the past 2 weeks. These historical shifts in AOD use can be attributed to multiple influences. For example, changes in the minimum legal drinking age (e.g., Wagenaar et al. 2001) as well as in perceived social norms (e.g., Keyes et al. 2012) have been shown to contribute to changes in alcohol use. Of particular interest are historical shifts that relate to changes in developmental trajectories. Latent growth modeling analyses with multicoHORT data have demonstrated that, compared with earlier cohorts, more recent cohorts exhibit lower initial levels of binge drinking but more rapid increases from age 18 to young adulthood (Jager et al. 2013). This acceleration of alcohol use helps explain the findings that use among adolescents has been decreasing at faster rates than among young adults in recent decades.

**Predictors of Alcohol Use Among Adolescents**

Despite the changes in alcohol use that have occurred over the past three decades, the relevant risk and protective factors tend to remain very stable across historic time, age, gender, and race/ethnicity (e.g., Brown et al. 2001; Donovan et al. 1999; Patrick and Schulenberg 2010). Like many other large-scale studies on adolescent AOD use, the MTF study has cast a wide net in terms of risk and protective factors, correlates, and consequences of substance use. Not only is this approach well suited to placing alcohol use within the larger context of adolescent development, it makes good use of the MTF large-scale survey approach that emphasizes breadth of measurement. Conceptually, the analyses drew from broad multidomain models when examining causes, correlates, and outcomes of adolescent alcohol use (e.g., Brown et al. 2009; Chassin et al. 2009; Maggs and Schulenberg 2005). This section summarizes MTF study findings concerning several domains of predictors of AOD use during adolescence, after considering methodological issues when examining causes and consequences of adolescent alcohol use.

**Methodological Issues in Understanding Risk Factors for and Consequences of Adolescent Alcohol Use**

When considering the correlates of AOD use, any attempt to discern whether these correlates are causes or consequences of substance use is hampered by three factors:

- Firm conclusions about causal connections are difficult without randomly controlled experiments.

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**Figure**  Trends in alcohol use in the past 12 months and in having been drunk in the past 30 days for 8th, 10th, and 12th graders, 1991–2011.
• Alcohol use during adolescence typically is reciprocally related to risk factors across development, such that problems that contribute to alcohol use may get worse with continued alcohol use (e.g., Cairns and Cairns, 1994; Dodge et al. 2009; Schulenberg and Maslowsky 2009).

• Factors that are identified as causes or as consequences of alcohol use during adolescence in the total sample likely do not apply to all young people, given the heterogeneity in developmental course (Schulenberg 2006).

Cross-sectional studies, in which each individual is evaluated only once, typically provide little leverage for concluding whether a given construct is a cause, correlate, or consequence of alcohol use, emphasizing the importance of conceptual guidance, logic, and statistical controls. Furthermore, when adolescents report using multiple substances, it is difficult to determine whether they are using the drugs simultaneously or whether use of one substance leads to use of another. Longitudinal panel studies, in which the same individuals are followed over time, provide more leverage but still leave room for alternative interpretations. For example, these studies may suffer from selection effects—that is, a construct excluded from the analysis actually “causes” both drug use and assumed consequence of drug use, rendering the relationship between cause and consequence spurious. Some recent analytic strategies that have been used with longitudinal data, such as propensity score analyses (Bachman et al. 2011) and fixed effects analysis (Patrick et al. 2012a; Staff et al. 2009), allow for greater control of selection effects and thus better leverage on likely causal connections. Nevertheless, despite such statistical advances, experiments in which participants are randomly assigned to experimental groups remain the gold standard for demonstrating causal connections.

Finally, the use of self-report data may limit the usefulness of study findings because such data rely on participants to remember and accurately perceive their own level of substance use. Nevertheless, most studies like the MTF study rely on these measures, because they have been found to be valid and reliable (Bachman et al. 2011; O’Malley et al. 1983) and because it is very expensive and burdensome to collect physiological data (e.g., blood, urine, or hair) and/or information from multiple reporters (e.g., parents or peers) in large-scale studies.

Influence of Parents and Peers

One developmental transition characteristic of adolescence is the movement away from parents and increasing involvement with peers. Nonetheless, parents still play a pivotal role in adolescent experiences and in fact can sometimes counter the effects of other risk factors for AOD use. Like many other reports in the literature (e.g., Dishion and McMahon 1998; Kiesner et al. 2009), the MTF study found that parental supervision and monitoring relate to lower AOD use among 8th and 10th graders and together are one of the strongest predictors (Dever et al. 2012; Pilgrim et al. 2006). Of particular importance, this effect was equally important (i.e., invariant) across gender and race/ethnicity (Pilgrim et al. 2006). Furthermore, parental monitoring was especially protective against substance use for high-risk–taking adolescents (Dever et al. 2012).

The literature for decades has indicated that peer use is one of the strongest correlates of AOD use. This was confirmed in the MTF; thus, in an analysis of multiple predictors of binge drinking among 8th and 10th graders from 1991 to 2007, having friends who get drunk was the strongest risk factor, regardless of the grade level or cohort analyzed (Patrick and Schulenberg 2010). Moreover, friends’ alcohol use in high school predicted both concurrent binge drinking and future trajectories of binge drinking (Schulenberg et al. 1996). Overall, the frequency of evenings out with friends (unsupervised by adults) was associated with more AOD use (Bachman et al. 2008; Brown et al. 2001; Patrick and Schulenberg 2010). Of course, a central issue when evaluating the role of peer use as a correlate and predictor of alcohol use is the extent to which friends actually influence an individual or the individual select friends who, like them, already drink. During adolescence and the transition to adulthood, both of these processes typically play a role (e.g., Patrick et al. 2012b).

Influence of School and Work

The broad domain of education also significantly relates to AOD use during adolescence (Cronsoe 2011). Studies consistently have found that grades, educational expectations, and school bonding are negatively correlated with AOD use, whereas school disengagement, school failure, school misbehavior, and skipping school are positively correlated with AOD use (Bachman et al. 2008; Bryant et al. 2003; Dever et al. 2012; Patrick and Schulenberg 2010; Pilgrim et al. 2006; Schulenberg et al. 1994). For example, in a longitudinal analysis examining 8th-grade predictors of concurrent and subsequent AOD use, school misbehavior and peer encouragement of misbehavior were positively associated with concurrent substance use and increased substance use over time. Conversely, school bonding, interest, and effort were negatively associated with concurrent and increased substance use, as were academic achievement and parental help with school (Bryant et al. 2003). Positive school attitudes were of particular importance and were especially influential as protective factors against substance use for low-achieving adolescents. The relationship between educational factors and AOD use is bidirectional, and it is clear that AOD use can contribute to educational difficulties. In general, however, it seems that based on MTF study longitudinal data and careful consideration of selection factors, the more common direction of influence is that school difficulties contribute to AOD use during adolescence (Bachman et al. 2008).

By the time they leave high school, most adolescents have worked part time during the school year. Although it has long been recognized that hours of work during adole-
ence are positively related to use of AODs, conclusions about causal connections have remained elusive (Staff et al. 2009). Analysis of MTF study data found that when sociodemographic and educational characteristics are controlled for, the positive relationship between hours of work and AOD use diminishes, suggesting that selection effects exist. In other words, long hours of work and substance use have a common set of causes, particularly disengagement from school (Bachman and Schulenberg 1993; Bachman et al. 2011). The influence of selection effects is further supported by findings that simply wanting to work long hours is associated with heavier AOD use. This is true regardless of actual hours spent working, and especially among those who do not work (Bachman et al. 2003; Staff et al. 2010).

**Religiosity and Community Attachment**

Numerous studies found that religiosity tends to be negatively correlated with AOD use during adolescence (Brown et al. 2001; Wallace et al. 2003, 2007; Wray-Lake et al. 2012). This is true for both African American and White youth. In fact, religiosity does not explain race differences in substance use (Wallace et al. 2003). Religiosity tends to operate at both the individual and contextual levels, because highly religious adolescents attending highly religious schools have lower alcohol use compared with highly religious adolescents attending non–highly religious schools (Wallace et al. 2007). More broadly, community attachments, including religiosity as well as social trust and social responsibility, tend to be negatively correlated with AOD use during adolescence (Wray-Lake et al. 2012).

**Exercise and Sports Participation**

Whereas exercise correlates negatively with alcohol use, participating in team sports correlates positively with alcohol use during high school (Terry-McElrath et al. 2011). This is especially true for males (Dever et al. 2012).

**Externalizing Behaviors and Other Drug Use**

As part of a broader set of problem behaviors, it is not surprising that alcohol use is associated with externalizing behaviors as well as cigarette smoking and illicit drug use during adolescence. In the MTF study, externalizing behaviors overall, and aggressive behavior and theft/property damage in particular, correlated with AOD use during adolescence (Bachman et al. 2008; Brown et al. 2001; Maslowsky and Schulenberg, in press; Patrick and Schulenberg 2010). Disentangling causal connections is difficult, however, and it is likely that alcohol use both contributes to and is caused by externalizing behaviors (Osgood et al. 1988), particularly if these behaviors involve spending unsupervised time with peers (Osgood et al. 1996). Cigarette smoking and other illicit drug use also tend to be highly correlated with alcohol use during adolescence (Patrick and Schulenberg 2010).

**Risk Taking and Sensation Seeking**

The willingness to take risks and high levels of sensation seeking also both correlate with higher levels of AOD use (Dever et al. 2012; Patrick and Schulenberg 2010; Pilgrim et al. 2006; Schulenberg et al. 1996). Among 8th graders and 10th graders, the impact of risk taking on substance use (including alcohol) was partly mediated through school bonding (which negatively affected AOD use) and time with friends (which positively affected AOD use); these effects were largely invariant across race/ethnicity and gender (Pilgrim et al. 2006).

**Well-Being**

Self-esteem tends to be negatively correlated with AOD use and, correspondingly, self-derogation and depressive affect tend to be positively correlated with AOD use during adolescence (Maslowsky and Schulenberg, in press; Patrick and Schulenberg 2010; Schulenberg et al. 1996). When examining the relative contributions of conduct problems, depressive affect, and the interaction of conduct problems and depressive affect on AOD use, depressive affect is not as powerful a predictor as are conduct problems. However, the interaction of the two variables (i.e., high levels of both) is a relatively powerful predictor of alcohol use, especially for younger adolescents (Maslowsky and Schulenberg, in press).

**Drinking Attitudes and Reasons for Using Alcohol**

Attitudes regarding alcohol use and reasons for use are powerful correlates and predictors of drinking behavior. Indeed, disapproval of binge drinking is one of the strongest protective factors against heavy drinking (Patrick and Schulenberg 2010). A long-standing focus of the MTF study has been to show how, at the population level, changes in perceptions of risk about and disapproval of substance use precede changes in substance use (Bachman et al. 1998; Johnston et al. 2012; Keyes et al. 2011). A recent analysis assessed the effects of age, period (i.e., the year in which data were obtained), and cohort effects of population-based social norms regarding heavy alcohol use (i.e., level of disapproval of heavy use) on individual-level heavy drinking during adolescence. The study found that cohort effects predominated, indicating that being part of a birth cohort that reported higher disapproval of heavy drinking set the stage for lower alcohol use (Keyes et al. 2012).

Motivations or reasons for drinking also are associated with alcohol use behaviors and may serve as a marker for the development of problematic behavioral patterns. The reasons for alcohol use typically change across adolescence and into adulthood. MTF study investigators have assessed reasons for drinking using MTF study panel data following high-school seniors into young adulthood. (MTF survey questions regarding motivations are not included in the 8th- and 10th-grade surveys.) Of particular interest here, 12th-grade adolescents tend to report higher motivation for drinking to get drunk (as well as other social and coping reasons for
drinking) than do young adults. Conversely, 12th graders report lower motivations to use alcohol to relax, to sleep, and because it tastes good, all of which increase across the transition to adulthood (Patrick and Schulenberg 2011; Patrick et al. 2011). It is important to understand the reasons for alcohol use among adolescents, because the reasons for use reported in 12th grade, when adolescents are about 18, show long-term longitudinal associations with alcohol use and symptoms of alcohol use disorders decades later (Patrick et al. 2011; Schulenberg et al. 1996).

**Long-Term Consequences of Alcohol Use**

Attempting to discern long-term consequences of adolescent AOD use is fraught with conceptual and methodological complexities (e.g., Schulenberg et al. 2003), yet it is critical for understanding the development (i.e., etiology) of adult alcohol use disorders. Numerous studies have demonstrated that alcohol use in middle school and high school may be an important indicator of later problems. For example, although most students mature out of their heavy alcohol use (Schulenberg and Maggs 2002; Schulenberg and Patrick 2012; Schulenberg et al. 1996), substance use in high school is one of the strongest predictors of substance use in adulthood. Specifically, binge drinking in 12th grade predicts symptoms of alcohol use disorders 17 years later, at age 35 (Merline et al. 2004, 2008; Patrick et al. 2011). Furthermore, trajectories of binge drinking are predictive of alcohol use disorders during middle adulthood (Schulenberg and Patrick 2012), and continued substance use into young adulthood is associated with HIV-related risk behaviors (Patrick et al. 2012). Finally, binge drinking in high school predicts subsequent dropping out of college, although an increase in binge drinking during college is related to not dropping out (Schulenberg and Patrick 2012).

**Implications for Prevention and Intervention**

Studies on the etiology and epidemiology of alcohol use ought to go hand in hand in order to combine the broader approach of epidemiology with the more in-depth emphasis of etiology. As the discussion in this article has shown, there are both historical and developmental predictors related to adolescent AOD use that are changing over time. Understanding the scope of alcohol use during the middle-school and high-school years, and associated long-term problems, is an important step toward effectively intervening to reduce high-risk drinking and its negative consequences. The scope of the problem is underscored by the findings that more than one in five American high-school seniors in the class of 2011 reported binge drinking in the previous 2 weeks. The documented developmental increases in alcohol use across adolescence and young adulthood make this a particularly important time for intervention. In particular, the fast escalation among adolescents from binge drinking once to binge drinking multiple times within a given 2-week period (Patrick and Schulenberg 2010) highlights the importance of preventing early initiation as well as early escalation of AOD use.

Levels of alcohol use have been declining in recent decades, suggesting that past interventions, such as increasing the minimum legal drinking age to 21, have been effective. However, although it is worth recognizing that most adolescents manage to avoid heavy alcohol use and that such use is not an inevitable developmental progression, alcohol remains the most commonly used substance among adolescents, and its use is a leading cause of death and injury (U.S. Department of Health and Human Services 2007). To design effective programs and target prevention efforts toward students most likely to develop problematic levels of alcohol use, it is essential to identify characteristics of individuals at greatest risk. This effort is aided by the fact that the importance of risk and protective factors tends to remain very stable over time. As summarized above, demographic differences in drinking behavior point to important subgroups that should be targeted, including young men and White and Hispanic adolescents. Finally, the findings described here point to several risk and protective factors to consider when designing prevention and intervention programs, including parental involvement, peer influences, academic success, religiosity, externalizing and internalizing behaviors, alcohol attitudes, and self-reported reasons for drinking.

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**References**


New Alcohol Alert

Number 86

Epigenetics—A New Frontier for Alcohol Research

In today’s modern age, with the entire human genetic makeup (the genome) decoded and genetic testing more and more common, most people understand that changes, or mutations, in genes can lead to death, disease, and disorder. But there is another way our genes can impact our health. Increasingly, researchers and physicians are exploring the concept of “epigenetics” — a complex process that determines which genes in the body are active at any given time.

This rapidly emerging area of research suggests that age, the environment, and exposure to drugs and other chemicals, including alcohol, directly affect epigenetics, altering normal genetic patterns and leading to abnormal expression or silencing of essential genes. The research also suggests that these epigenetic changes can, in some instances, be passed from one generation to the next.

This Alert describes how alcohol influences epigenetics and how those influences may be associated with illness and disorders, including fetal alcohol spectrum disorders (FASD), cancer, liver disease and other gastrointestinal disorders, brain development, the body’s internal clock, and its ability to fight disease. This issue also explores therapies that may target those changes caused by epigenetics.

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The Burden of Alcohol Use

Excessive Alcohol Consumption and Related Consequences Among College Students

Aaron White, Ph.D., and Ralph Hingson, Sc.D.

Research shows that multiple factors influence college drinking, from an individual's genetic susceptibility to the positive and negative effects of alcohol, alcohol use during high school, campus norms related to drinking, expectations regarding the benefits and detrimental effects of drinking, penalties for underage drinking, parental attitudes about drinking while at college, whether one is member of a Greek organization or involved in athletics, and conditions within the larger community that determine how accessible and affordable alcohol is. Consequences of college drinking include missed classes and lower grades, injuries, sexual assaults, overdoses, memory blackouts, changes in brain function, lingering cognitive deficits, and death. This article examines recent findings about the causes and consequences of excessive drinking among college students relative to their non-college peers and many of the strategies used to collect and analyze relevant data, as well as the inherent hurdles and limitations of such strategies.

Excessive Drinking At College

Currently, only two active national survey studies are able to characterize the drinking habits of college students in the United States. The National Survey on Drug Use and Health (NSDUH), an annual survey sponsored by the Substance Abuse and Mental Health Services Administration (SAMHSA), involves face-to-face interviews with approximately 67,500 persons ages 12 and older each year regarding use of alcohol and other drugs. Monitoring the Future (MTF) is an annual, paper-and-pencil national survey of alcohol and other drug use with a sample comprising nearly 50,000 students in 8th, 10th, and 12th grades drawn from roughly 420 public and private schools. Approximately 2,400 graduating seniors are resurveyed in subsequent years, allowing for the monitoring of trends in college drinking.

In addition, two prior surveys yielded data on college drinking that remain valuable and relevant. The National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), sponsored by NIAAA, collected data on alcohol and other drug use from a sample of roughly 46,500 citizens 18 and older using face-to-face computer-assisted interviews. Two waves of data (2001 and 2004) were collected from the same sample, and data from an independent sample are scheduled to be collected in 2013. The Harvard College Alcohol Study (CAS), although no longer active, was a land-

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ince 1976, when the National Institute on Alcohol Abuse and Alcoholism (NIAAA) issued its first report on alcohol misuse by college students, research advances have transformed our understanding of excessive drinking on college campuses and the negative outcomes that follow from it. For instance, we now know that a broad array of factors influence whether a particular college student will choose to drink, the types of consequences they suffer from drinking, and how they respond to those consequences.

We have learned that predisposing factors include an individual’s genetic susceptibility to the positive and negative effects of alcohol, alcohol use during high school, campus norms related to drinking, expectations regarding the benefits and detrimental effects of drinking, penalties for underage drinking, parental attitudes about drinking while at college, whether one is member of a Greek organization or involved in athletics, and conditions within the larger community that determine how accessible and affordable alcohol is. Consequences include missed classes and lower grades, injuries, sexual assaults, overdoses, memory blackouts, changes in brain function, lingering cognitive deficits, and death.

This article reviews recent research findings about alcohol consumption by today's college students and the outcomes that follow. It examines what we know about the causes and consequences of excessive drinking among college students relative to their non-college peers and many of the strategies used to collect and analyze relevant data, as well as the inherent hurdles and limitations of such strategies.

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mark paper-and-pencil survey that provided national data (years 1993, 1997, 1999, and 2001) from roughly 15,000 students on more than 100 college campuses each year (Wechsler and Nelson 2008). Data from both NESARC and Harvard CAS remain useful for examining associations between patterns of drinking at college and the frequency and prevalence of alcohol-related consequences for both drinkers and nondrinkers.

Data from NSDUH and MTF suggest that roughly 65 percent of college students drink alcohol in a given month (see figure 1 for data from MTF), and Harvard CAS all suggest that a large percentage of college students who drink do so to excess. Excessive, or “binge,” drinking is defined in NSDUH, MTF, and NESARC as consuming five or more drinks in an evening, although the instruments vary in the specified time frames given (i.e., once or more in the past month for NSDUH, past 2 weeks for MTF, and multiple time periods for NESARC) (Johnston et al. 2001; SAMHSA 2011). The Harvard CAS was the first national study of college students to utilize a gender-specific definition of binge drinking (i.e., four or more drinks in an evening for females or five or more for males in the past 2 weeks) to equate the risk of alcohol-related harms (Wechsler et al. 1995). The Centers for Disease Control and Prevention (CDC) utilizes the same four or more/five or more gender-specific measures but specifies a 30-day time period (Chen et al. 2011). NIAAA uses the four or more/five or more gender-specific measure but specifies a time frame of 2 hours for consumption, as this would generate blood alcohol levels of roughly 0.08 percent, the legal limit for driving, for drinkers of average weight (NIAAA 2004).

According to NSDUH, the percentage of 18- to 22-year-old college students who reported drinking five or more drinks on an occasion in the previous 30 days remained relatively stable from 2002 (44 percent) to 2010 (44 percent) (SAMHSA 2011). Among 18- to 22-year-olds not enrolled in college, the percentage who engaged in binge drinking decreased significantly from 2002 (39 percent) to 2010 (36 percent) (see figure 2).

Looking at a longer time period, data from MTF suggest that there have been significant declines in the percentage of college students consuming five or more drinks in the previous 2 weeks, from 44 percent in 1980 to 36 percent in 2011 (Johnston et al. 2012) (see figure 3). This time frame includes the passage of the National Minimum Drinking Age Act of 1984, which effectively increased the drinking age from 18 to 21 in the United States.

Across the four waves of data collection in the Harvard CAS (1993, 1997, 1999, and 2001), rates of binge drinking remained relatively stable (44, 43, 45, and 44 percent, respectively) (Wechsler et al. 2002) (see figure 4). However, the number of non–binge drinkers decreased, whereas the number
of frequent binge drinkers (three or more binge-drinking episodes in a 2-week period) increased. Wechsler and colleagues (2002) reported that binge drinkers consumed 91 percent of all the alcohol consumed by college students during the study period. Frequent binge drinkers, a group comprising only 1 in 5 college students, accounted for 68 percent of all alcohol consumed (Wechsler and Nelson 2008).

**Individual and Environmental Contributors to Excessive Drinking**

Survey data indicate that males outpace females with regard to binge drinking. According to MTF, in 2011, 43 percent of male and 32 percent of female college students crossed the binge threshold in a given 2-week period. Further, 40 percent of students—more males (44 percent) than females (37 percent)—reported getting drunk in a given month. Research suggests that gender differences in alcohol use by college students have narrowed considerably over the years. In their landmark 1953 report on college drinking, Yale researchers Straus and Bacon indicated that, based on survey data from more than 15,000 students on 27 college campuses, 80 percent of males and 49 percent of females reported having been drunk at some point. Nearly 60 years later, in 2011, data from MTF indicated that 68 percent of males and 68 percent females reported having been drunk. These new, higher levels of drinking among females seem to be ingrained

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**Figure 2** Binge alcohol use among adults aged 18 to 22, by college enrollment: 2002–2011. Survey years are shown on the horizontal axis, and the percentage using in the past month is shown on the vertical axis. For each college enrollment status (enrolled full time in college and not enrolled full time in college), there is a line showing use over the years 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, and 2011. Tests of statistical significance at the .05 level were performed between 2011 and each of the previous years listed; significant results are indicated where appropriate.

Among adults aged 18 to 22 enrolled full time in college, 44.4 percent were past-month binge alcohol users in 2002, 43.5 percent in 2003, 43.4 percent in 2004, 44.8 percent in 2005, 45.6 percent in 2006, 43.6 percent in 2007, 40.7 percent in 2008, 43.6 percent in 2009, 42.2 percent in 2010, and 39.1 percent in 2011. The differences between the 2011 estimate and the 2002, 2003, 2004, 2005, 2006, 2007, 2009, and 2010 estimates were statistically significant.

Among adults aged 18 to 22 not enrolled full time in college, 38.9 percent were past-month binge alcohol users in 2002, 38.7 percent in 2003, 39.4 percent in 2004, 38.3 percent in 2005, 38.5 percent in 2006, 38.6 percent in 2007, 38.2 percent in 2008, 38.0 percent in 2009, 35.4 percent in 2010, and 35.4 percent in 2011. The differences between the 2011 estimate and the 2002 through 2009 estimates were statistically significant.

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in the youth drinking culture. Whereas binge-drinking rates declined significantly among high-school seniors over the last decade, the effect was driven by a decline among males only. Binge-drinking rates among females remained relatively stable (Johnston et al. 2012) (see figure 5).

Beyond gender, survey studies of college drinking reveal a range of characteristics of both individual students and campus environments that influence the likelihood of binge drinking. Data from the Harvard CAS and other studies reveal that males, Caucasians, members of Greek organizations, students on campuses with lower percentages of minority and older students, athletes, students coping with psychological distress, those on campuses near a high density of alcohol outlets, students with access to cheap drink specials, a willingness to endure the consequences of alcohol misuse, and drinking at off-campus parties and bars all contribute to excessive drinking (Mallett et al. 2013; Wechsler and Kuo 2003; Yusko et al. 2008). Further, students living off campus and/or in Greek housing, those who drink to try to fit in, students with inflated beliefs about the proportion of other students who binge drink, and those with positive expectations about the results of drinking are more likely to drink excessively (Scott-Sheldon et al. 2012; Wechsler and Nelson 2008). Importantly, excessive drinking prior to college relative to other college-bound students is predictive of both excessive drinking at college and experiencing alcohol-related consequences (Varvil-Weld et al. 2013; White et al. 2002).

Strengths and Weaknesses of Binge-Drinking Measures

Several studies indicate that crossing commonly used binge-drinking thresholds increases a college student’s risk of experiencing negative alcohol-related consequences. For instance, data from the Harvard CAS indicate that students who binge one or two times during a 2-week period are roughly three times as likely as non–binge drinkers to get behind in school work, do something regretful while drinking, experience a memory blackout, have unplanned sex, fail to use birth control during sex, damage property, get in trouble with police, drive after drinking, or get injured (Wechsler et al. 2000). The more often a student binges, the greater the risk of negative outcomes. Further, the more binge drinking that occurs on a campus, the more likely non–binge drinkers and abstainers are to experience secondhand consequences of alcohol use, such as having studying or sleep disrupted, being a victim of sexual assault, and having property damaged (Wechsler and Nelson 2008).

Figure 3 Alcohol: Trends in 2-week prevalence of consuming five or more drinks in a row among college students vs. others 1 to 4 years beyond high school (12th graders included for comparison).

SOURCE: The Monitoring the Future study, the University of Michigan.
NOTE: Others refers to high school graduates 1 to 4 years beyond high school not currently enrolled full time in college.
Because of the increased risk of consequences to self and others that occurs when a person drinks at or beyond the binge threshold, a great deal of emphasis is placed on tracking the percentage of college students that cross binge thresholds. Although this has proven extremely valuable, as Wechsler and Nelson (2001, p. 289) state, “Alcohol use is a complex behavior. No single measure will capture all the relevant aspects of alcohol use.” One limitation of using a single threshold is that it removes data regarding just how heavily students actually drink (Alexander and Bowen 2004; Read et al. 2008) and assigns the same level of risk to all students who cross the thresholds regardless of how far beyond the threshold they go. This is an important consideration as recent studies suggest that plenty of college students who cross the binge threshold when they drink go far beyond it.

In a study of 10,424 first-semester college freshmen, more than one-half of all males and one-third of all females categorized as binge drinkers drank at levels two or more times the binge threshold (8 or more drinks for women and 10 or more drinks for men) at least once in the 2 weeks before the survey. Indeed, one in four binge-drinking males consumed 15 or more drinks at a time during that period (White et al. 2006). Naimi and colleagues (2010) reported that 18- to 24-year-olds in the United States drink an average of 9.5 drinks per binge episode, nearly twice the standard binge threshold. Data from MTF also reveal that both college students and their non-college peers often drink at levels that exceed the binge threshold. On average, between 2005 and 2011, 7 percent of college females surveyed and 24 percent of college males consumed 10 or more drinks at least once in a 2-week period, compared with 7 percent of females and 18 percent of males not in college. Further, 2 percent of all college females surveyed and 10 percent of college males consumed 15 or more drinks in a 2-week period. Rates among non-college peers were similar, at 2 percent among females and 9 percent among males (Johnston et al. 2012). For a 140-pound female, consuming 15 drinks over a 6-hour period would produce an estimated blood alcohol level above 0.4 percent, a level known to have claimed, directly, several lives on college campuses in recent years. For a 160-pound male, drinking in this way would lead to a blood alcohol level above 0.3 percent, a potentially lethal level associated with memory blackouts and injury deaths.

Data from the Harvard CAS suggested that students who binge drink frequently (three or more times in a 2-week period) are at particularly high risk of negative alcohol-related outcomes. Compared with students who binge drink one or two times in a 2-week period, those who binge three or more times are twice as likely to experience alcohol-induced memory losses (27 percent vs. 54 percent, respectively), not use protection during sex (10 percent vs. 20 percent, respectively), engage in unplanned sex (22 percent vs. 42 percent, respectively), and get hurt or injured (11 percent vs. 27 percent, respectively), and are equally likely to need medical treatment for an overdose (1 percent vs. 1 percent). Whereas binge frequency is associated with an increased risk of negative outcomes, additional research indicates that there is a relationship between how often a student binges and the peak number of drinks he or she consumes. White and colleagues (2006) reported that 19 percent of frequent binge

![Figure 4 Drinking habits of college students from Harvard CAS.](source: Johnston, L.D.; O’Malley, P.M.; Bachman, J.G.; and Schulenberg, J.E. Monitoring the Future National Survey Results on Drug Use, 1975–2011: Volume I: Secondary School Students. Ann Arbor, MI: Institute for Social Research, The University of Michigan.)
drinkers consume three or more times the binge threshold (12 or more drinks for females and 15 or more for males) at least once in a 2-week period compared with only 5 percent of infrequent binge drinkers. As a result of the association between frequency of binge drinking and peak levels of consumption, it is difficult to determine if the increase in risk that comes with frequent bingeing is a result of the number of binge episodes, per se, or the number of drinks consumed in an episode.

Importantly, although evidence suggests that many students drink at levels far beyond the binge threshold, additional research suggests that the majority of alcohol-related harms on college campuses result from drinking at levels near the standard four/five-drink measure. This is related to the well-known prevention paradox in which the majority of health problems, such as alcohol-related consequences, tend to occur among those considered to be at lower risk (Rose 1985). For a particular individual, the odds of experiencing alcohol-related harms increase as the level of consumption increases (Wechsler and Nelson 2001). However, at the population level, far fewer people drink in this manner. As a result, more total consequences occur among those who drink at relatively lower risk levels. For instance, based on data from roughly 9,000 college-student drinkers across 14 college campuses in California, Gruenewald and colleagues (2010) estimated that more than one-half of all alcohol-related consequences resulted from drinking occasions in which four or fewer drinks were consumed. Similarly, using national data from nearly 50,000 students surveyed across the four waves of the Harvard CAS, Weitzman and Nelson (2004) observed that roughly one-quarter to one-third of alcohol-related consequences, including getting injured, vandalizing property, having unprotected sex, and falling behind in school, occurred among students who usually consume three or four drinks per occasion. Such findings raise the possibility that a reduction in high peak levels of consumption might not necessarily result in large overall reductions in alcohol-related consequences on a campus. However, a reduction in high peak levels of drinking would certainly help save the lives of students who drink at these high levels.

In summary, while binge-drinking thresholds are useful for sorting students into categories based on levels of risk, a single threshold cannot adequately characterize the drinking habits of college students or the risks associated with alcohol use on college campuses (Read et al. 2008). It is not uncommon for college students to far exceed standard binge thresholds. Presently, only MTF tracks and reports the incidence of drinking beyond the binge threshold on college campuses. Such data are important as they allow for better tracking of changes in the drinking habits of students. For instance, it is possible that the number of students who drink at extreme levels could increase, whereas the overall percentage of students who binge drink declines or remains stable. Such a phenomenon might help explain why some consequences of excessive alcohol use, like overdoses requiring hospitalization, seem to be on the rise despite relatively stable levels of binge drinking on college campuses across several decades. Finally, although sorting students into binge drinking categories fails to capture high peak levels of consumption among students, a large proportion of harms actually occurs at or near the standard four or more/five or more threshold.

### Do Students Know How to Define Standard Servings?

Despite concerns about the accuracy of self-report data for assessing levels of alcohol use among college students and the general population, such surveys remain the most common tool for assessing alcohol use. One major concern is whether students and other young adults are aware of what constitutes a single serving of alcohol. Research shows that college students and the general public tend to define and pour single servings of alcohol that are significantly larger than standard drinks, suggesting they might underestimate their true levels of consumption on surveys (Devos-Comby and Lange 2008; Kerr and Stockwell 2012). For instance, White and colleagues (2003, 2005) asked students to pour single servings of different types of alcohol beverages into cups of various sizes. Overall, students poured drinks that were too large. When asked to simply define standard drinks in terms of fluid ounces, students tended to overstate the number of ounces in a standard drink. The average number of ounces of liquor in student-defined mixed drinks was 4.5 ounces rather than the 1.5 ounces in actual standard drinks (White et al. 2005). When students were provided with feedback regarding discrepancies between their definitions of single servings and the actual sizes of standard drinks, they tended to revise their self-reported levels of consumption upward, leading to a significant increase in the number of students categorized as binge drinkers (White et al. 2005). Such findings suggest that students underreport their levels of consumption on surveys, raising the possibility that more students drink excessively than survey data indicate.

Although a lack of knowledge regarding standard serving sizes could lead students to underestimate, and thus underestimate, how much they drink, field research suggests that the discrepancy between self-reported and actual levels of consumption might be smaller than expected from lab studies. For instance, Northcote and Livingston (2011) conducted a study in which they monitored the number of drinks consumed by research participants in bars and then asked them to report their consumption a few days later. Reports by study participants were consistent with the observations made by researchers for participants who had consumed less than eight total drinks. Only those who consumed eight drinks or more tended to underestimate their consumption. When comparing estimated blood alcohol concentrations (BAC) based on self-report to actual BAC readings in college students returning to campus from bars, actual BAC levels tended to be lower, rather than higher, than levels calculated using self-reported consumption (Kraus et al. 2005). Similarly, when actual BAC levels are compared with estimated BAC...
levels in bar patrons, estimates are spread evenly between accurate, underestimates, and overestimates (Clapp et al. 2009).

In short, although self-reported drinking data might not be perfect, and college students lack awareness of how standard drink sizes are defined, research does not suggest that the discrepancies between self-reported and actual drinking levels are large enough to question the general findings of college drinking surveys.

**Paper-and-Pencil, Face-To-Face, and Electronic Surveys: Does It Make a Difference?**

National surveys of college drinking often utilize paper-and-pencil questionnaires (e.g., MTF and Harvard CAS) or face-to-face computer-assisted personal interviews (e.g., NSDUH and NESARC). It is now possible to collect survey data electronically via the Internet and also using handheld devices, such as smartphones and personal digital assistants. This raises questions about the comparability between traditional survey methods and electronic data collection.

Several studies comparing traditional (e.g., paper and pencil) and electronic means of data collection suggest that the approaches yield generally similar results from survey participants (Boyer et al. 2002; Jones and Pitt 1999; LaBrie et al. 2006; Lygidakis et al. 2010). For instance, in a comparison of Web-based and paper-and-pencil survey approaches, Knapp and Kirk (2003) found no differences in outcomes, suggesting that Web-based surveys do not diminish the accuracy or honesty of responses. Similarly, LaBrie and colleagues (2006) observed similar outcomes of self-reported alcohol consumption in a paper-and-pencil survey and an electronic survey. However, other studies suggest that students actually feel more comfortable answering personal questions truthfully when completing questionnaires electronically (Turner et al. 1998), which can lead to higher levels of self-reported substance use and other risky behaviors. Both Lygidakis and colleagues (2010) and Wang and colleagues (2005) indicate that adolescents completing electronic surveys reported higher levels of alcohol and other drug use compared with those completing paper-and-pencil versions.

Response rate is an important consideration, with higher response rates increasing the representativeness of the sample and limiting the likelihood that response biases will influence the outcomes. Two national paper-and-pencil surveys mentioned above, MTF and Harvard CAS, report response rates for college students of approximately 59 percent. For MTF, this response rate represents a retention rate, as the participants were followed up after high school. Response rates for the in-person computer-assisted personal interviews, NSDUH and NESARC, which assess college student drinking but are not limited to college students, are roughly 77 percent and 81 percent, respectively. Currently, there is no basis for assessing response rates for national Web-based assessments of college drinking. However, smaller studies suggest that response rates might be comparable, if not higher, than other approaches. McCabe and colleagues (2002) reported that, among 7,000 undergraduate students, one-half of whom were surveyed about alcohol and other drug use via the Internet and half surveyed via paper-and-

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**Figure 5** Percent of 12th-grade male and female students who reported drinking at least once in the prior 2 weeks.

pencil surveys delivered through the mail, the response rates were 63 percent for the Web survey and 40 percent for the paper-and-pencil survey. Further, response rates for Web-based surveys can be improved by sending reminders via e-mail (van Gelder et al. 2010).

In summary, in recent years an increasing number of researchers have utilized electronic survey methods to collect college-drinking data. At present, evidence suggests that these methods can yield results quite similar to those obtained from traditional survey methods and that response rates might actually be higher.

Alcohol-Related Consequences Among College Students

Drinking to intoxication leads to widespread impairments in cognitive abilities, including decisionmaking and impulse control, and impairments in motor skills, such as balance and hand-eye coordination, thereby increasing the risk of injuries and various other harms. Indeed, research suggests that students who report “getting drunk” even just once in a typical week have a higher likelihood of being injured, experiencing falls that require medical treatment, causing injury in traffic crashes, being taken advantage of sexually, and injuring others in various ways (O’Brien et al. 2006). Students who drink with the objective of getting drunk are far more likely to experience a range of consequences, from hangovers to blackouts, than other students who drink (Boekeloo et al. 2011).

National estimates suggest that thousands of college students are injured, killed, or suffer other significant consequences each year as a result of drinking. However, researchers have questioned the manner in which such national estimates are calculated. In many cases, the lack of college identifiers in datasets means that the actual amount of annual alcohol-attributable harm that occurs among college students is unknown. Although the Harvard CAS collected data regarding the consequences of drinking, its final year of administration was 2001. Currently, assessing the damage done, on a national level, by college drinking requires estimating rates of consequences using a variety of data sources. Such assessments are complicated by the fact that outcomes considered to be negative consequences by researchers (e.g., blackouts and hangovers) are not always perceived as negative by students (Mallett et al. 2013). Further, college students often drink off campus, such as during spring breaks and summer vacations, meaning that many alcohol-related consequences experienced by college students are not necessarily associated with college itself. As such, our understanding of alcohol-related consequences among college students remains somewhat cloudy.

In one set of estimates, Hingson and colleagues (2002, 2005, 2009) utilized census data and national datasets regarding traffic crashes and other injury deaths to estimate the prevalence of various alcohol-related harms among all young people aged 18–24. Next, they attributed an amount of harm to college students equal to the proportion of all 18-to 24-year-olds who were enrolled full time in 4-year colleges (33 percent in 2005, the most recent year analyzed) (Hingson et al. 2009). Because college students drink more heavily than their non-college peers, it is possible this approach underestimated the magnitude of alcohol-related consequences on college campuses. Hingson and colleagues (2002, 2005, 2009) also used the percentage of college students who reported various alcohol-related behaviors (e.g., being assaulted by another drinking college student) in national surveys to derive national estimates of the total numbers of college students who experienced these consequences.

Based on the above strategies along with other sources of data, researchers have estimated the following rates and prevalence of alcohol-related harms involving college students:

- **Death:** It is possible that more than 1,800 college students between the ages of 18 and 24 die each year from alcohol-related unintentional injuries, including motor-vehicle crashes (Hingson et al. 2009).
- **Injury:** An estimated 599,000 students between the ages of 18 and 24 are unintentionally injured each year under the influence of alcohol (Hingson et al. 2009).
- **Physical Assault:** Approximately 646,000 students between the ages of 18 and 24 are assaulted each year by another student who has been drinking (Hingson et al. 2009).
- **Sexual Assault:** Perhaps greater than 97,000 students between the ages of 18 and 24 are victims of alcohol-related sexual assault or date rape each year (Hingson et al. 2009).
- **Unsafe Sex:** An estimated 400,000 students between the ages of 18 and 24 had unprotected sex and nearly 110,000 students between the ages of 18 and 24 report having been too intoxicated to know if they consented to having sex (Hingson et al. 2002).
- **Health Problems:** More than 150,000 students develop an alcohol-related health problem each year (Hingson et al. 2002).
- **Suicide Attempts:** Between 1.2 and 1.5 percent of college students indicate that they tried to commit suicide within the past year as a result of drinking or drug use (Presley et al. 1998).
- **Drunk Driving:** Roughly 2.7 million college students between the ages of 18 and 24 drive under the influence of alcohol each year (Hingson et al. 2009).
- **Memory Loss:** National estimates suggest that 10 percent of non–binge drinkers, 27 percent of occasional binge drinkers, and 54 percent of frequent binge drinkers reported at least one incident in the past year of blacking out, defined as having forgotten where they were or what
they die from alcohol each year, in addition to the lack of college identifiers in datasets, another barrier is the fact that levels of alcohol often are not measured in nontraffic fatalities. As such, attributable fractions, based on analyses of existing reports in which alcohol levels were measured postmortem, are used to estimate the number of deaths by various means that likely involved alcohol. The CDC often uses attributable fractions calculated by Smith and colleagues (1999) based upon a review of 331 medical-examiner studies. An updated approach is needed. The combination of including college identifiers in medical records and measuring alcohol levels in all deaths would allow for accurate assessments of the role of alcohol in the deaths of college students and their non-college peers.

### Academic Performance

About 25 percent of college students report academic consequences of their drinking, including missing class, falling behind in class, doing poorly on exams or papers, and receiving lower grades overall (Engs et al. 1996; Presley et al. 1996a, b; Wechsler et al. 2002). Although some published research studies have not found a statistically significant association between binge drinking and academic performance (Gill 2002; Howland et al. 2010; Paschall and Freisthler 2003; Williams 2003; Wood et al. 1997), studies linking binge drinking to poorer academic performance outnumber the former studies two to one. Presley and Pimentel (2006) reported that in a national survey of college students, those who engaged in binge drinking and drank at least three times per week were 5.9 times more likely than those who drank but never binged to perform poorly on a test or project as a result of drinking (40.2 vs. 6.8 percent), 5.4 times more likely to have missed a class (64.4 vs. 11.9 percent), and 4.2 times more likely to have had memory loss (64.2 vs. 15.3 percent) (Thombs et al. 2009). Singleton and colleagues (2007, 2009), in separate prospective studies, found negative associations between heavy alcohol use and grade point average. Jennison (2004), based on a national prospective study, reported binge drinkers in college were more likely to drop out of college, work in less prestigious jobs, and experience alcohol dependence 10 years later. Wechsler and colleagues (2000) and Powell and colleagues (2004), based on the Harvard CAS, found frequent binge drinkers were six times more likely than non–binge drinkers to miss class and five times more likely to fall behind in school. White and colleagues (2002) observed that the number of blackouts, a consequence of heavy drinking, was negatively associated with grade point average (GPA). It is important to note that although data regarding GPA often are collected via self-report, the negative association between alcohol consumption and GPA holds even when official records are obtained (Singleton 2007). Collectively, the existing research suggests that heavy drinking is associated with poorer academic success in college.

### Alcohol Blackouts

Excessive drinking can lead to a form of memory impairment known as a blackout. Blackouts are periods of amnesia during which a person actively engages in behaviors (e.g., walking, talking) but the brain is unable to create memories for the events. Blackouts are different from passing out, which means either falling asleep or becoming unconscious from excessive drinking. During blackouts, people are capable of participating in events ranging from the mundane, such as eating food, to the emotionally charged, such as fights and even sexual intercourse, with little or no recall (Goodwin 1995). Like milder alcohol–induced short-term memory impairments caused by one or two drinks, blackouts primarily are anterograde, meaning they involve problems with the formation and storage of new memories rather than problems recalling memories formed prior to intoxication. Further, short-term memory often is left partially intact. As such, during a blackout, an intoxicated person is able to discuss events that happened prior to the onset of the blackout and to hold new information in short-term storage long enough to have detailed conversations. They will not, however, be able to transfer new information into long-term storage, leaving holes in their memory. Because of the nature of
blackouts, it can be difficult or impossible to know when a drinker in the midst of one (Goodwin 1995).

There are two general types of blackouts based on the severity of the memory impairments. Fragmentary blackouts, sometimes referred to as gray outs or brown outs, are a form of amnesia in which memory for events is spotty but not completely absent. This form is the most common. En bloc blackouts, on the other hand, represent complete amnesia for events (Goodwin 1995).

Blackouts surprisingly are common among college students who drink alcohol. White and colleagues (2002) reported that one-half (51 percent) of roughly 800 college students who had ever consumed alcohol at any point in their lives reported experiencing at least one alcohol-induced blackout, defined as awakening in the morning not able to recall things one did or places one went while under the influence. The average number of total blackouts in those who experienced them was six. Of those who had consumed alcohol during the 2 weeks before the survey was administered, 9 percent reported blacking out. Based on data from 4,539 inbound college students during the summer between high school graduation and the start of the freshmen year, 12 percent of males and females who drank in the previous 2 weeks experienced a blackout during that time (White and Swartzwelder 2009). Data from the Harvard CAS indicate that blackouts were experienced in a 30-day period by 25 percent of students in 1993 and 27 percent of students in 1997, 1999, and 2001 (Wechsler et al. 2002). A small study by White and colleagues (2004), in which 50 students with histories of blackouts were interviewed, suggests that fragmentary blackouts are far more common than en bloc blackouts. Roughly 80 percent of students described their last blackout as fragmentary.

Blackouts tend to occur following consumption of relatively large doses of alcohol and are more likely if one drinks quickly and on an empty stomach, both of which cause a rapid rise and high peak in BAC (Goodwin 1995; Perry et al. 2006). For this reason, pregaming, or prepartying, which typically involves fast-paced drinking prior to going out to an event, increases the risk of blacking out. Labrie and colleagues (2011) reported that 25 percent of 2,546 students who engaged in prepartying experienced at least one blackout in the previous month. Playing drinking games and drinking shots were risk factors. Further, skipping meals to restrict calories on drinking days is associated with an increased risk of blackouts and other consequences (Giles et al. 2009).

Because blackouts typically follow high peak levels of drinking, it is not surprising that they are predictive of other alcohol-related consequences. Mundt and colleagues (2012) examined past-year blackouts in a sample of more than 900 students in a randomized trial of a screening and brief intervention for problem alcohol use and found that blackouts predicted alcohol-related injuries over a subsequent 2-year period. Compared with students who had no history of blackouts, those who reported one to two blackouts at baseline were 1.5 times more likely to experience an alcohol-related injury, whereas those with six or more blackouts were 2.5 times more likely. In a follow-up report based on the same sample, Mundt and Zakletskaya (2012) estimated that among study participants, one in eight emergency-department (ED) visits for alcohol-related injuries involved a blackout. On a campus of 40,000 students, this would translate into roughly $500,000 in annual costs related to blackout-associated ED visits.

In the study of 50 students with blackout histories by White and colleagues (2004), estimated peak BACs during the night of the last blackout generally were similar for males (0.30 percent) and females (0.35 percent), although it is unlikely that self-reported alcohol consumption during nights in which blackouts occur is highly accurate. A study of amnesia in people arrested for either public intoxication, driving under the influence, or underage drinking found that the probability of a fragmentary or en bloc blackout was 50/50 at a BAC of 0.22 percent and the probability of an en bloc blackout, specifically, was 50/50 at a BAC of 0.31 percent, based on breath alcohol readings (Perry et al. 2006). In their study of blackouts in college students, Hartzler and Fromme (2003a) noted a steep increase in the likelihood of blackouts above a BAC of 0.25 percent, calculated from self-reported consumption. Thus, from existing research, it seems that the odds of blacking out increase as BAC levels climb and that blackouts become quite common at BAC levels approaching or exceeding 0.30 percent. As such, the high prevalence of blackouts in college students points to the magnitude of excessive consumption that occurs in the college environment. It should be noted, however, that BAC levels calculated based on self-reported consumption are unlikely to be accurate given the presence of partial or complete amnesia during the drinking occasion.

It seems that some people are more sensitive to the effects of alcohol on memory than others and are therefore at increased risk of experiencing blackouts. Wetherill and Fromme (2011) examined the effects of alcohol on contextual memory in college students with and without a history of blackouts. Performance on a task was similar while the groups were sober, but students with a history of blackouts performed more poorly when intoxicated than those without a history of blackouts. Similarly, Hartzler and Fromme (2003b) reported that when mildly intoxicated, study participants with a history of blackouts performed more poorly on a narrative recall task than those without a history of blackouts. When performing a memory task while sober, brain activity measured with functional magnetic resonance imaging is similar in people with a history of blackouts and those without such a history (Wetherill et al. 2012). However, when intoxicated, those with a history of blackouts exhibit lower levels of activity in several regions of the frontal lobes compared with subjects without a history of blackouts.

Thus, studies suggest that there are differences in the effects of alcohol on memory and brain function between those who experience blackouts and those who do not. Research by Nelson and colleagues (2004), using data from monozygotic twins, suggests that there could be a significant
Alcohol Overdoses

When consumed in large quantities during a single occasion, such as a binge episode, alcohol can cause death directly by suppressing brain stem nuclei that control vital reflexes, like breathing and gagging to clear the airway (Miller and Gold 1991). Even a single session of binge drinking causes inflammation and transient damage to the heart (Zagrosek et al. 2010). The acute toxic effects of alcohol in the body can manifest in symptoms of alcohol poisoning, such as facial flushing and increased heart rate, which is responsible for the breakdown of acetaldehyde. The accumulation of acetaldehyde after drinking alcohol leads to symptoms of acetaldehyde poisoning, such as facial flushing and increased heart and respiratory rates.

In total, nearly 30,000 young people in this age-group, more males (19,847) than females (9,525) were hospitalized for alcohol overdoses with no other drugs involved in 2008. Hospitalizations for overdoses involving other drugs but not alcohol increased 55 percent over the same time period, while those involving alcohol and drugs in combination rose 76 percent. In total, there were 59,000 hospitalizations in 2008 among 18- to 24-year-olds for alcohol overdoses only or in combination with other drugs. Given that 33 percent of people in this age-group were full-time college students at 4-year colleges in 2008, a conservative estimate would suggest approximately 20,000 hospitalizations for alcohol overdoses alone or in combination with other drugs involved college students, although the exact number is not known.

Data from the Drug Abuse Warning Network (DAWN) indicate that ED visits for alcohol-related events increased in a similar fashion as those observed for inpatient hospitalizations. Among those ages 18 to 20, ED visits for alcohol-related events with no other drugs increased 19 percent, from 67,382 cases in 2005 to 82,786 cases in 2009. Visits related to combined use of alcohol and other drugs increased 27 percent, from 27,784 cases in 2005 to 38,067 cases in 2009. In 2009, 12 percent of ED visits related to alcohol involved use of alcohol in combination with other drugs (SAMHSA 2011).

Alcohol interacts with a wide variety of illicit and prescription drugs, including opioids and related narcotic analgesics, sedatives, and tranquilizers (NIAAA 2007a; Tanaka 2002). Importantly, BAC required for fatal overdoses are lower when alcohol is combined with prescription drugs. An analysis of 1,006 fatal poisonings attributed to alcohol alone or in combination with other drugs revealed that the median postmortem BACs in those who overdosed on alcohol alone was 0.33 percent, compared with 0.13 percent to 0.17 percent among those who overdosed on a combination of alcohol and prescription drugs (Koski et al. 2003, 2005). The combined use of alcohol and other drugs peaks in the 18- to 24-year-old age range (McCabe et al. 2006), suggesting that college-aged young adults are at particularly high risk of suffering consequences from alcohol-and-other-drug combinations.

The above findings reflect the fact that heavy consumption of alcohol quickly can become a medical emergency. One does not need to get behind the wheel of a car after drinking or jump off a balcony into a swimming pool on a dare to risk serious harm. Simply drinking too much alcohol is enough to require hospitalization and potentially cause death. Further, combining alcohol with other drugs can increase the risk of requiring medical intervention substantially. Thus, efforts to minimize the consequences of alcohol-related harms on college campuses should not lose sight of the fact that alcohol often is combined with other drugs and, when this is the case, the risks can be greater than when alcohol or drugs are used alone.

1 The ALDH2*2 allele results in decreased action by the enzyme acetaldehyde dehydrogenase, which is responsible for the breakdown of acetaldehyde. The accumulation of acetaldehyde after drinking alcohol leads to symptoms of acetaldehyde poisoning, such as facial flushing and increased heart and respiratory rates.
Measuring the true scope of medical treatment for alcohol overdoses among college students is difficult for several reasons. First, in datasets such as the Nationwide Emergency Department Sample (NEDS) and the Nationwide Inpatient Sample (NIS), no college identifiers are included to indicate whether a young person treated for an alcohol overdose is enrolled in college. Many schools do not track or report the number of students treated for an alcohol overdose, and many students drink excessively when away from campus. Further, schools that implement Good Samaritan or Amnesty policies, which allow students to get help for overly intoxicated peers without fear of sanctions, could create the false impression that overdoses are on the rise. For instance, after Cornell University implemented an amnesty policy, they witnessed an increase in calls to residence assistants and 911 for help dealing with an intoxicated friend (Lewis and Marchell 2006). Given the dangerous nature of alcohol overdoses, with or without other drugs involved, it is important to improve the tracking of these events at colleges and in the larger community.

Sexual Assault

Sexual assault is a pervasive problem on college campuses, and alcohol plays a central role in it. A study of roughly 5,500 college females on two campuses revealed that nearly 20 percent experienced some form of sexual assault while at college (Krebs et al. 2009). Data from the Harvard CAS suggested that 5 percent of women surveyed were raped while at college (Mohler-Kuo et al. 2004). In a national sample of students who completed the Core Alcohol and Drug Survey in 2005, 82 percent of students who experienced unwanted sexual intercourse were intoxicated at the time. Similarly, nearly three-quarters (72 percent) of respondents to the Harvard CAS study who reported being raped were intoxicated at the time. In many cases, rape victims are incapacitated by alcohol. In one study, 3.4 percent of rape victims reported being so intoxicated they were unable to consent (Mohler-Kuo et al. 2004). In a study of 1,238 college students on three campuses over a 3-year period, 6 percent of students reported being raped while incapacitated by alcohol (Kaysen et al. 2006).

Research suggests that the involvement of alcohol increases the risk of being victimized in several ways, such as by impairing perceptions that one is in danger and by reducing the ability to respond effectively to sexual aggression (Abbey 2002; McCauley et al. 2010; Testa and Livingston 2009). Further, alcohol might increase the chances that a male will commit a sexual assault by leading them to misinterpret a female’s friendly gestures or flirtation as interest in sex and by increasing sexual aggression (Abbey 2002). When asked to read a story about a potential date rape involving intoxicated college students, both male and female subjects who are intoxicated were more likely to view the female as sexually aroused and the male as acting appropriately (Abbey et al. 2003).

It is widely held that sexual assaults, with and without alcohol involvement, are underreported on college campuses. Title IX of the Education Amendments Act of 1972, a Federal civil rights law, requires universities to address sexual harassment and sexual violence. However, universities vary with regard to how they handle such cases, and a student’s perception of safety and protection can influence the likelihood of reporting a sexual assault. Indeed, many universities have indicated changes in rates of reports of assaults consistent with changes in campus policies regarding how such cases are handled. As such, although it is clear that alcohol often is involved in sexual assaults on college campuses, questions about the frequency and nature of such assaults remain.

Spring Break and 21st Birthday Celebrations—Event-Specific Drinking Occasions

More college students drink, and drink more heavily, during specific celebratory events, such as spring break and 21st birthday celebrations, than during a typical week. Spring break is a roughly weeklong recess from school that takes place in the spring at colleges throughout the United States. While some students continue to work, travel home, or simply relax, others use the opportunity to travel to beaches and other party destinations. During spring break, approximately 42 percent of students get drunk on at least 1 day, 11 percent drink to the point of blacking out or passing out, 32 percent report hangovers, and 2 percent get into trouble with the police (Litt et al. 2013). Students with a history of binge drinking and those intending to get drunk tend to drink the heaviest (Patrick et al. 2013), suggesting that prevention efforts aimed at altering students’ intentions to get drunk while on spring break might lead to a reduction in peak drinking and the consequences that follow (Mallett et al. 2013). Interestingly, students who typically are light drinkers are more likely than those who typically are binge drinkers to experience consequences from excessive drinking during spring break (Lee et al. 2009).

In addition to spring break, 21st birthday celebrations are another event-specific opportunity for students to drink excessively. An estimated 4 out of 5 college students drink alcohol to celebrate their 21st birthdays (Rutledge et al., 2008) and many students drink more than they plan. Of 150 male and female college students surveyed about their intentions to drink during their upcoming 21st birthday celebrations, 68 percent consumed more than they anticipated while only 21 percent drank less and 11 percent were accurate. On average, males intended to consume 8.5 drinks but consumed 12.5, while females expected to drink 7 but had 9 (Brister et al., 2010). As with spring-break drinking, students with a history of binge drinking and those who intended to drink heavily on their 21st birthday consumed the most (Brister et al., 2011). In one study, roughly 12 percent of students reported consuming 21 or more drinks while celebrating, and one-third of females (35 percent) and nearly half of males (49 percent) reached estimated BACs...
above 0.25 percent (Rutledge et al., 2008). Such high levels of consumption substantially increase the odds of sexual assaults, fights, injuries, and death (Mallett et al., 2013). Research indicates that brief interventions conducted in the week leading up to the 21st birthday celebration can reduce levels of consumption and associated consequences, suggesting that the risks of experiencing alcohol-related consequences stemming from 21st birthday celebrations could be partially mitigated through specifically timed prevention efforts (Neighbors et al. 2009, 2012).

Summary

We have learned a considerable amount about the drinking habits of college students and the consequences that follow since NIAAA first reported on the matter in 1976. Surprisingly, drinking levels have remained relatively stable on and around college campuses over the last 30 years, with roughly two out of five male and female students engaging in excessive, or binge, drinking. Excessive drinking results in a wide range of consequences, including injuries, assaults, car crashes, memory blackouts, lower grades, sexual assaults, overdoses and death. Further, secondhand effects from excessive drinking place non–binge-drinking students at higher risk of injury, sexual assaults, and having their studying disrupted.

Estimates of the rates of alcohol use and related consequences are imperfect. Lack of knowledge of standard drink sizes and the effects of alcohol on memory formation all complicate the collection of accurate data from traditional self-report surveys. Underreporting of sexual assaults leads to difficulty in estimating the true extent of the problem. Lack of college identifiers in mortality records and the fact that alcohol levels are tested too infrequently in non–traffic-related deaths leaves uncertainty regarding the actual number of college students who die each year from alcohol-related deaths. Furthermore, the lack of knowledge of standard drink size as a means of reducing excessive alcohol consumption and alcohol-related harms (Neighbors et al. 2009, 2012).

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Focus On: Women and the Costs of Alcohol Use

Sharon C. Wilsnack, Ph.D.; Richard W. Wilsnack, Ph.D.; and Lori Wolfgang Kantor, M.A.

Although light-to-moderate drinking among women is associated with reduced risks of some cardiovascular problems, strokes, and weakening of bones, such levels of drinking also are associated with increased risks of breast cancer and liver problems, and heavy drinking increases risks of hypertension and bone fractures and injuries. Women’s heavy-drinking patterns and alcohol use disorders are associated with increased likelihood of many psychiatric problems, including depression, posttraumatic stress disorder, eating disorders, and suicidality, as well as increased risks of intimate partner violence and sexual assault, although causality in the associations of drinking with psychiatric disorders and with violence remains unclear. It is important for women to be aware of the risks associated with alcohol use, especially because gaps between U.S. men’s and women’s drinking may have narrowed. However, analyses of health risks and benefits need improvement to avoid giving women oversimplified advice about drinking. Key words: Alcohol consumption; alcohol use, abuse, and dependence; alcohol use disorder; alcohol burden; drinking patterns; prevalence; alcohol burden; alcohol-related problems; alcohol-related injuries; women; pregnancy; cardiovascular disease; stroke; bone mass density; breast cancer; liver disease; psychiatric disorders; posttraumatic stress disorder (PTSD); depression; eating disorders; suicidal behavior; intimate partner violence; sexual assault

Even though the prevalence of alcohol use in the United States generally is lower among women compared with men (Substance Abuse and Mental Health Services Administration [SAMHSA] 2011), this gap has narrowed (Grucza et al. 2008). Furthermore, although women consume alcohol at lower levels than men, their body composition puts them at higher risk than men of developing some alcohol-related problems, both acutely (because of higher blood alcohol levels from a given amount of alcohol) and chronically (from alcohol-related organ damage). This article examines alcohol-use patterns (with particular attention to midlife) and how they differ for men and women and summarizes recent evidence on associations between women’s alcohol consumption and their physical and mental health.

Drinking Practices and Patterns Among Women in Midlife

Rates of drinking decline with age for both men and women in the United States, and drinking remains less prevalent among women compared with men. In 2010, the proportion of people reporting at least one drink in the previous 30 days (i.e., current drinkers) decreased from 70 percent among 21- to 25-year-olds to 61.1 percent among 40- to 44-year-olds and 51.6 percent among 60- to 64-year-olds (SAMHSA 2011). The same survey also found that approximately 57.4 percent of males aged 12 or older were current drinkers, compared with 46.5 percent of females of the same age range (SAMHSA 2011).

Rates of binge drinking also are higher among men than women (Centers for Disease Control and Prevention [CDC] 2012). One survey (National Institute on Alcohol Abuse and Alcoholism [NIAAA] 2012) reported that 28.8 percent of women and 43.1 percent of men reported binge drinking (i.e., consuming within 2 hours four or more drinks for women and five or more drinks for men) in the previous year. In a multinational study of 35 countries, Wilsnack and colleagues (2009) reported that, as expected, men consistently drank more than women and were more likely to engage in high-volume drinking and high-frequency drinking. Women were more likely to be lifetime nondrinkers and to be former drinkers. The authors suggest that women may find it easier than men to quit drinking because (1) women generally are lighter drinkers than men; (2) drinking is not as important to women’s social roles as it is to men’s; and/or (3) women who stop drinking during pregnancy and early childrearing may not resume drinking later on.

Despite these findings, Grucza and colleagues (2008) reported significant increases between 1990–1991 and 2000–2001 in the lifetime prevalence of drinking for women aged 38–47 in the United States. There also was an increase in lifetime prevalence of alcohol dependence among women drinkers aged 38–47. Similar increases were not found for male drinkers, suggesting that the gender gap in alcohol use and dependence is narrowing, at least in these age groups.

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Footnotes:
1 Because women’s bodies generally have less water than men’s bodies, alcohol becomes less diluted, and women therefore reach higher blood alcohol levels than men even if both are drinking the same amount.

2 Former drinkers reported drinking in the past but not in the last 12 months.
Drinking During Pregnancy: Patterns and Predictors

Women who become pregnant in their thirties and forties may be more likely to drink during pregnancy than younger women. From 2001 to 2005, 17.7 percent of 35- to 44-year-old women reported drinking during pregnancy, compared with 8.6 percent of pregnant women aged 18–24 (Denny et al. 2009). Among women in eight States who gave birth between 1997 and 2002, 30.3 percent reported drinking during pregnancy, and 8.3 percent reported binge drinking (four or more drinks on one occasion). Whereas 22.5 percent of the women reported drinking during the first month of pregnancy, drinking declined during pregnancy; only 7.9 percent of women reported drinking during the third trimester, and only 2.7 percent reported drinking during all trimesters. Drinking during pregnancy was more prevalent among women over 30 (more than 30 percent drank) than among younger women (Ethen et al. 2009).

Understanding the predictors of drinking during pregnancy may help target prevention efforts. The eight-State study by Ethen and colleagues (2009) found that both drinking and binge drinking during pregnancy were predicted by prepregnancy binge drinking. Drinking and binge drinking during pregnancy also were more prevalent among women who were non-Hispanic whites, who smoked during pregnancy, and whose pregnancy was unintended. A recent review of 14 studies of drinking during pregnancy in nine countries (Skagerström et al. 2011) found that drinking during pregnancy was associated with heavier drinking prior to pregnancy in all seven studies that measured this; smaller numbers of studies consistently found that drinking during pregnancy was associated with higher income/social class and with histories of abuse or exposure to violence and histories of drinking problems.

Physical Health Effects of Women’s Drinking

Light to moderate alcohol use has been found to generally be beneficial for many health outcomes and is associated with decreased mortality. Heavier use, however, is associated with poorer health and increased mortality. One meta-analysis of 34 studies in 13 countries found that, compared with abstaining, drinking less than two drinks per day among women and drinking less than four drinks per day among men was associated with significantly reduced total mortality, but higher levels of alcohol use were associated with increased mortality (Di Castelnuovo et al. 2006). These findings should not encourage people to start drinking alcohol for its health benefits, because of the significant health problems associated with heavier use, as described below.

Another study used data from a large survey of middle-aged (median age 58) female nurses in the United States and assessed the health of participants who lived to age 70 and older. The study found that light to moderate alcohol consumption at midlife was associated with modestly increased odds of good health at age 70 or older (no chronic illnesses, physical impairment, or mental problems). That is, women who averaged between one-third and one drink per day had about 20 percent higher odds than nondrinkers of good health at age 70 and older. Also, the women who drank frequently during the week (5 to 7 days) had better odds of good health at age 70 and older than the women who drank only once or twice a week (Sun et al. 2011). However, these findings should be interpreted with caution because the measures of alcohol consumption were quite limited.

Effects of Women’s Drinking on Cardiovascular Health

Many studies have found that light to moderate alcohol use is associated with lower risks of cardiovascular disease and mortality, but these studies often have not reported specifically on women’s drinking. However, studies of coronary heart disease risk in Denmark (Tolstrup et al. 2006) and England (Ward et al. 2011) found that the risks were lower in women who consumed more alcohol. In the United States, pooled data from nine National Health Interview Surveys (1987–2000) showed that women drinking up to seven drinks per week had lower risks of cardiovascular mortality than lifetime abstainers (Mukamal et al. 2010).

Light-to-moderate drinking also may be associated with lower risks of sudden cardiac death (SCD). The study of nurses in the United States, which examined heart problems in 4-year periods after reported drinking or abstaining, found the lowest risk of SCD among women who averaged approximately one-half to one drink per day. Women who drank more heavily (more than 30 g or two drinks per day) had SCD risks similar to risks of abstainers, but the number of SCD cases among women who consumed more than 30 g per day was limited (Chiave et al. 2010). As noted earlier, however, these findings are based on limited measures of drinking.

In contrast to studies finding beneficial effects, a meta-analysis of six studies (Samokhvalov et al. 2010) found that women’s risks of atrial fibrillation (AF) increased steadily with increasing alcohol consumption. Whereas women who averaged up to two drinks a day did not have significantly higher risks than abstainers, women who consumed more than two to three drinks daily had a 17 percent increased risk of AF, and women who consumed more than four drinks daily had twice the risk of AF.

Women’s risk of hypertension also may increase steadily as alcohol consumption increases. A meta-analysis of eight studies indicated that the risk was reduced somewhat among women drinking lightly (averaging less than a drink a day), but the risk then rose steadily with higher levels of consumption. Compared with abstainers, women who averaged roughly four drinks a day had nearly twice the risk of hypertension, and women averaging roughly eight drinks a day had nearly three times the risk (Taylor et al. 2009).

Effects of Women’s Drinking on Stroke Risk

The risk of stroke is lower among women who are light-to-moderate drinkers. The U.S. nurses’ study found lower risk of
strokes among women who were recent light drinkers, averaging approximately one drink a day (Jimenez et al. 2012). Among 45,449 Swedish women aged 30 to 50 who were followed up approximately 11 years later, risks of ischemic stroke were significantly lower among women averaging less than one drink a day (compared with abstainers). The numbers of women with hemorrhagic strokes and/or strokes after drinking more heavily were too small for reliable evaluation (Lu et al. 2008). Meta-analyses of five to nine other studies found that women’s light-to-moderate drinking was protective against both ischemic and hemorrhagic strokes (with lowest risks in women averaging about one drink a day), but risks of morbidity and mortality from both types of strokes increased rapidly as women’s consumption rose above three to four drinks a day (Patra et al. 2010).

**Effects of Women’s Drinking on Liver Disease**

Women apparently are more vulnerable than men to liver cirrhosis and other liver injury from alcohol use, possibly because of estrogens, although the mechanisms are as yet unclear (Eagon 2010). A meta-analysis of 12 studies found that women’s risks of morbidity and mortality from liver cirrhosis increased steadily with higher levels of alcohol consumption, with no protective effect of light to moderate drinking, and the risks increased more rapidly for women than for men (Rehm et al. 2010). These risks may be increased by other personal characteristics and by drinking patterns. In a very large sample of women in the United Kingdom, followed up for an average of 6.2 years, risks of cirrhosis among women averaging two or more drinks a day increased greatly if their body mass indexes were greater than 28 kg/m² (Liu et al. 2010). In a large study of women in New York State, levels of γ-glutamyl-transferase (GGT), a liver enzyme that increases in all forms of liver disease (Niemelä and Alatalo 2010), were highest not only in women who averaged more than a drink a day but also in women who did their drinking only on weekends and without food (Stranges et al. 2004).

**Effects of Women’s Drinking on Breast Cancer Risk**

Even moderate alcohol consumption increases breast cancer risk, and the risk rises as drinking increases. A multinational meta-analysis of 98 studies found that the risk of breast cancer increased an average of 10 percent for every increase of 10 grams per day in alcohol consumption (Key et al. 2006). A 10-year follow-up study of more than 38,000 U.S. women aged 45 and older found a significant trend of increased risk of invasive breast cancer associated with increased alcohol consumption at baseline, with the greatest risk among women averaging at least 30 grams of alcohol per day (Zhang et al. 2007). The risks from alcohol consumption were clearest for estrogen- and progesterone-receptor-positive tumors and for women currently taking postmenopausal hormones, consistent with the hypothesis that part of alcohol’s effect on breast cancer is to increase estrogen exposure (Garcia-Closas et al. 2002; Onland-Moret et al. 2005). Another U.S. study, based on data from 184,418 postmenopausal women aged 50 to 71, reported similar findings (Lew et al. 2009). After 7 years of follow-up, the researchers found that risks of breast cancer increased steadily the more women drank. Risks were highest for estrogen- and progesterone-receptor-positive tumors, with risks of these tumors 46 percent higher for women drinking more than 35 grams of alcohol (more than two drinks) a day. However, when Suzuki and colleagues (2010) followed up 50,757 Japanese women (aged 40 to 69) over 13 years, they found that breast cancer risk increased 6 percent with every additional 10 grams per day of alcohol consumption, but the observed association was not modified by menopausal status or use of exogenous estrogens. These findings suggest that breast cancer risks associated with alcohol consumption involve more than just estrogen levels.

**Effects of Women’s Drinking on Bone Health**

Higher bone-mineral density (BMD) is associated with resistance to fracture. A recent review of research relevant to 40- to 60-year-old women concluded that there was fair evidence that moderate drinking did no harm to BMD (Waugh et al. 2009). In fact, a number of studies have found that light to moderate drinking is associated with increased BMD, at least among postmenopausal women (Maurel et al. 2012). For example, Tucker and colleagues (2009) found that, in women from the Framingham Offspring cohort, hip and spine BMD were 5.0 to 8.3 percent greater in postmenopausal women who consumed more than two drinks per day than in nondrinkers. A study of 2,043 postmenopausal women in the United States found that BMD was 3.8 percent higher in women who had more than 29 drinking occasions per month than those who abstained, although this finding only was marginally significant (because of small numbers of daily drinkers) (Wosje and Kalkwarf 2007). Finally, a study in Scotland of 3,218 women aged 50 to 62 found significant increases in BMD in the femoral neck and lumbar spine in women who averaged more than one drink a day, compared with lifetime abstainers (McLernon et al. 2012). However, in general these studies were unable to evaluate effects of heavy drinking, and the processes by which alcohol affects BMD remain uncertain but may involve effects of increased levels of estrogen and calcitonin (Maurel et al. 2012). In contrast, the prevailing wisdom is that heavy drinking (averaging multiple drinks per day) increases women’s risks of fractures, such as from falls (Epstein et al. 2007). In a combined study of 11,032 women in Canada, Australia, and the Netherlands, the risks of hip fractures and osteoporotic fractures were higher in women averaging two or more drinks a day than in women averaging up to one drink a day (Kanis et al. 2005). In Sweden, a study of 10,902 middle-aged women showed that low-energy fractures were more likely in women who had higher levels of GGT, which is associated with chronic heavy drinking (Holmberg et al. 2006).
Women’s Drinking and Psychiatric Disorders

Alcohol Use Disorders
In addition to physical health risks associated with alcohol use, women's risks of mental health problems also are related to their drinking. It is clear that women's heavy and binge drinking is associated with alcohol use disorders (AUDs). For example, U.S. data show that among women aged 50 or older, those who engage in binge drinking (four or more drinks on a drinking occasion) have more than three times greater risks of alcohol abuse, and more than five times greater risks of alcohol dependence, than women who drink but do not engage in binge drinking (Chou et al. 2011).

However, there has otherwise been limited attention to gender-specific ways in which women's drinking may be related to AUDs. One exception is that women, like men, are at greater risk of AUDs if they begin drinking at early ages. A large study in Missouri has found elevated risks of AUDs in women who began drinking before age 18 (Jenkins et al. 2011), confirming findings from U.S. national surveys (Dawson et al. 2008). A second exception is that it has long been thought that development of AUDs is “telescoped” in women compared with men, occurring in a shorter period of time after women begin to drink (Greenfield 2002). However, this pattern was identified in women in treatment for AUDs, and U.S. survey data now indicate that telescoping does not occur in women drinkers in the general population (Keyes et al. 2010) but may be related to the experiences that bring women to treatment.

Psychiatric Disorders Other Than AUDs
General-population studies often have found links between women's drinking and psychiatric disorders, but the time order and causes of these linkages are often unclear. For example, a German survey found that women with alcohol abuse or dependence, or women who drank an average of at least 20 to 30 grams of alcohol per day, were more likely than other women to have a variety of psychiatric disorders (affective, anxiety, or somatoform), and the connections between drinking and disorders were stronger for women than for men (Bott et al. 2005). A Danish survey found that any psychiatric disorders were more likely in women averaging more than three drinks a day, and anxiety disorders were specifically more likely among women averaging more than two drinks a day, compared with nondrinkers (Flensborg-Madsen et al. 2011). In addition, U.S. data on women aged 50 and older showed higher risks of both panic disorder and posttraumatic stress disorder (PTSD) in women who engaged in any binge drinking, compared with non–binge drinkers (Chou et al. 2011).

Unlike the preceding studies, which linked drinking patterns to increased risks of general psychiatric comorbidity, most studies of women's alcohol use and psychiatric disorders have focused on comorbidity of specific disorders with AUDs and risky drinking patterns. These more specific linkages are discussed in the sections that follow.

Depression. Research clearly has established that depressive disorders and symptoms are more likely among people with AUDs (e.g., Grant et al. 2004), but studies have not always examined this connection specifically among women. However, a large U.S. twin study found that diagnoses of major depression and alcohol dependence were correlated among women (Prescott et al. 2000), and data from the large National Epidemiologic Study on Alcohol and Related Conditions (NESARC) showed that women with major depressive disorder were more likely to report multiple criteria for alcohol abuse and dependence (Lynskey and Agrawal 2008). Research also has repeatedly found associations of women's depression with binge drinking. For example, in a major Canadian survey, women's binge drinking (five or more, or eight or more, drinks per day) was associated with measures of recent and longer-term depression (Graham et al. 2007), and data from the large U.S. Behavioral Risk Factor Surveillance System surveys showed that lifetime depression was significantly more likely in women who engaged in binge drinking (four or more drinks in a day) (Strine et al. 2008).

PTSD. AUDs often have been associated with symptoms or diagnoses of PTSD. For example, in young adults followed up from the U.S. National Survey of Adolescents, women with PTSD in the past 6 months were more than twice as likely as other women to meet criteria for a Diagnostic and Statistical Manual of Mental Disorders, 4th Edition diagnosis of alcohol abuse (Danielson et al. 2009). Among women from the large Missouri Adolescent Female Twin Study, PTSD was associated with a greater likelihood of AUDs (Sartor et al. 2010). In surveys of three Mexican cities, lifetime PTSD was more prevalent in women who misused alcohol (with at least one indicator of alcohol abuse or dependence) (Slone et al. 2006). In addition, in the large California Women’s Health Survey, having symptoms of PTSD doubled the odds that women engaged in binge drinking (Timko et al. 2008). However, most of these studies have not found any effects of PTSD beyond the effects of the traumatic experiences that led to PTSD, a pattern also reported in other recent studies of women who have experienced sexual assaults (Najdowski and Ullman 2009; Testa et al. 2007). Therefore, PTSD may be an indicator of experiences distressful enough to lead women to drink to excess, but PTSD itself may not necessarily be a cause of such drinking.

Alcohol and Eating Disorders. Research often has found that eating disorders in women are associated with problem drinking. The strongest recent evidence is in a meta-analysis of 41 studies, mainly in the U.S. and Canada, in which women’s eating disorders consistently were associated with AUDs (Gadalla and Piran 2007). The meta-analysis included a very large Canadian general-population survey in which risks of eating disorders also were associated with heavier weekly drinking among women ages 15 to 44 (Piran and
Alcohol and Suicidal Behavior. Although research often has reported on factors affecting rates of suicide among women, only rarely have studies been able to show how individual women's drinking patterns are related to suicidal behavior. An exception was a 20-year follow-up of a large sample of Swedish women hospitalized because of suicidal behavior; those women diagnosed also with alcohol abuse or dependence had a higher risk of later committing suicide (Tidemalm et al. 2008). Most general-population surveys of individual women have shown that suicidal ideation (thinking about committing suicide) was associated with heavier, more frequent, or more hazardous drinking. In the United States, for example, women's suicidal ideation was associated with hazardous drinking patterns in a longitudinal study of women aged 26 to 54 (Wilsnack et al. 2004) and was associated with alcohol dependence in the large National Longitudinal Alcohol Epidemiologic Survey (Grant and Hasin 1999). A large study of active-duty U.S. Air Force personnel also found that women's suicidal ideation was associated with higher levels of alcohol problems, but only among women who were not mothers (Langhinrichsen-Rohling et al. 2011). In Seoul, Korea, women aged 18 to 64 showed a strong association of suicidal ideation with drinking nearly daily (Park et al. 2010). Finally, a French survey of women aged 18 to 30 found that suicidal ideation was more common in heavier drinkers, although the relationship no longer was statistically significant after controlling for effects of depression and other adverse experiences (Legleye et al. 2010).

Alcohol-Related Injuries

Similar to research on women's suicidality, research on women's alcohol-related injuries has given more attention to gender differences in injury rates and how women's injury rates are related to population drinking patterns and less attention to how drinking is related to the risks of injury in individual women. However, studies have reported two consistent findings about how individual drinking patterns are linked to injuries.

First, risks of injury increase among women who have consumed alcohol in the 6 hours before being injured; women's injury risks associated with drinking occur relatively rapidly. This conclusion has been confirmed by a combined analysis of 28 hospital emergency-department studies in 16 countries (Borges et al. 2006). Additional confirmation has come from a large emergency-department survey in Sydney, Australia, where the risk was greatest in women who had consumed more than 90 grams of alcohol in the 6 hours before being injured (Williams et al. 2011).

The other consistent finding is that risks of injury are greatest among women whose drinking patterns are particularly heavy or hazardous. A study of women outpatients at a Veterans Administration hospital found that the likelihood of multiple recent injuries was nearly doubled in the heaviest versus the lightest drinkers (Chavez et al. 2012).

Intimate Partner Violence

Associations between alcohol use and intimate partner violence (IPV) have been well documented in research in North America. Male-to-female IPV perpetration consistently has been linked to heavy and problem drinking by men (Caetano et al. 2000; Thompson and Kingree 2006). The large-scale NESARC survey found that past-year IPV victimization was more likely in women who have symptoms of alcohol abuse or dependence (La Flair et al. 2012), and meta-analysis of six surveys of adolescents and young adults showed that women's frequency and/or quantity of drinking was positively related to their perpetration of IPV (Rothman et al. 2012). Furthermore, a comparative study of alcohol consumption and IPV in Canada, the United States, and eight countries in Latin America found that in all 10 countries, rates of physical partner aggression were higher among drinkers than nondrinkers (men and women); and among drinkers, rates were higher among persons who reported drinking larger amounts per occasion. Women reported being victims of more severe aggression than men, and men were more likely than women to be drinking at the time of an incident of physical aggres-

3 The CAGE is a screening instrument (Ewing 1984) consisting of the following four questions: Have you ever felt you should cut down on your drinking? Have people annoyed you by criticizing your drinking? Have you ever felt bad or guilty about your drinking? Eye opener: Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover? Two positive responses are considered a positive test and indicate further assessment is warranted.
sion (Graham et al. 2008). Other multinational studies have shown that odds of IPV were greater where one or both partners had alcohol problems (Abramsky et al. 2011) and that aggression severity was significantly higher if one or both partners had been drinking when the aggression occurred (Graham et al. 2011). However, in all this research, it is unclear to what extent drinking is a cause or an effect of IPV, or both.

Alcohol and Sexual Assault

It has been known for some time that women’s drinking is positively associated with their risks of sexual assault, but how and why this association occurs remains unsettled (Abbey et al. 2004). Part of the association results because women often drink with men who drink, and the men’s intoxication makes them more likely to be sexually aggressive toward women (Abbey 2011). Other links between women’s drinking and sexual assaults are harder to interpret because investigators often lack time-ordered data, they differ in the types of sexual activity they evaluate (ranging from rape to much broader categories of unwanted sexual advances), and most of their studies are limited to college women (as a high-risk group).

Nevertheless, certain patterns have become clear in recent years. First, risks of sexual assault are most clearly higher in women who have established patterns of binge drinking or problem drinking. For example, in a large national survey of college women in 1999, women with alcohol problems were more likely to report experiencing unwanted sexual advances (Pino and Johnson-Johns 2009). At a large New York State university, women who increased their drinking during their first year in college (and who averaged more than four drinks per drinking occasion, with frequent such occasions) had higher odds of sexual victimization (Parks et al. 2008).

Second, women are more likely to experience rape or other severe sexual assault if they become intoxicated, at the time of the assault or as a typical drinking pattern. A large U.S. survey of college women found that the percentage who had been raped was high in women with any recent experience of binge drinking (four or more drinks per occasion) and that more than two-thirds of the women who had been raped reported being intoxicated at the time (Mohler-Kuo et al. 2004). A study of more than 300 young women who had been sexually assaulted since age 14 found that the odds of sexual penetration were greater only among women reporting high levels of intoxication (Testa et al. 2004). An earlier national survey of college women who had experienced sexual victimization found that the severity of the assault was predicted in part by the women’s frequency of intoxication (Ullman et al. 1999).

Findings like these have led some investigators to conclude that one reason why drinking may increase women’s risks of

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**Figure**  Female intimate-partner violence victimization by women’s past-12-month heavy episodic drinking (HED) (10 countries of the Americas).

**NOTE:** * p < .05 for logistic regression, controlling for age.

sexual assault is that highly intoxicated women may be incapacitated, unable to resist unwanted sexual advances. A national survey of college women found that a past-year history of binge drinking (five or more drinks at a sitting) was specifically associated with experiencing incapacitated rape (McCauley et al. 2009). A study of first-year college students found that reported maximum consumption per occasion during the fall semester was strongly associated with experiencing incapacitated rape (Testa and Livingston 2009) led to the conclusions that in many rapes, especially of college students, women are incapacitated by some form of substance use, and that many rapes associated with alcohol use involve incapacitation.

Conclusions

Because alcohol consumption has become a more normal activity for women, it is important for women to have science-based information to help them decide whether and when to drink, and in what amounts, based on potential risks or benefits of drinking. Such past and current information has had some important limitations. Some of these limitations have been addressed in recent decades. In most recent studies (e.g., Mukamal et al. 2010; Patra et al. 2010), apparent health benefits of moderate drinking now are based on comparisons with lifetime abstainers, excluding potentially sicker ex-drinkers who were part of some earlier comparisons. Also, long-term studies of alcohol consumption in women now are likely to include more detailed measures of baseline drinking (Moore et al. 2005; Wilsnack et al. 2006) than earlier studies used (Stampfer et al. 1988). However, some research findings are still presented in terms of rates of health outcomes in whole groups of women (such as for injuries and suicidality; Landberg 2010; Ramstedt 2005), which can be misleading if these results are used to draw conclusions about the effects of drinking on individuals. Finally, research on long-term health effects of women's drinking can measure only some of the lifestyle characteristics (such as eating patterns and exercise) that may be associated with how women drink and that may account for some of the apparent effects of drinking (Mukamal et al. 2010; Rimm & Moats 2007).

A major current limitation of information about alcohol effects is that such effects often are reported, in scientific papers but particularly in the news media, as simple associations (this drinking pattern is associated with that health outcome). Less is said about how large the effects are (not very large for some cardiovascular benefits of moderate drinking), and adverse effects often are implied to increase in a linear way with each unit increase in drinking. There is too little attention paid to how the effects of drinking may not be linear (with the exception of research on cardiovascular benefits versus hazards at different levels of drinking). There also is too little attention paid to how drinking may be both a cause and an effect of some adverse health and behavioral outcomes (such as psychiatric disorders and intimate partner violence). Finally, research findings often are presented as if they applied similarly to all women drinkers, without discussing how other conditions and contexts (such as a drinker's other health conditions) might modify how alcohol affects health. (One exception, for example, is the research by Liu et al. [2010] showing that risks of cirrhosis from relatively heavy drinking are greater in women with high body mass indices.) Therefore, what we should strive for is information about health effects of women's drinking that shows not only the effect sizes, but also when and where and among which women the effects are greatest.

Keeping those limitations in mind, the findings summarized here may offer some guidelines for women making personal decisions about drinking in midlife. Light-to-moderate drinking is associated to some extent with reduced risks of some cardiovascular problems, strokes, and weakening of bones. On the other hand, even low levels of alcohol consumption may cause women some increase in risks of breast cancer and liver problems, and heavy drinking also increases risks of hypertension and bone fractures and injuries. Women's heavy drinking patterns and AUDs are associated with increased likelihood of many psychiatric problems, including depression, PTSD, eating disorders, and suicidality. Women's heavy drinking and AUDs also are associated with increased risks of intimate partner violence and sexual assault, although causality in the associations of drinking with psychiatric disorders and with violence remains unclear. On balance, the evidence summarized here suggests that, for those women who choose to drink, moderation in consumption is the safest or least costly strategy to adopt toward alcohol.

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Focus On: Ethnicity and the Social and Health Harms From Drinking

Karen G. Chartier, Ph.D.; Patrice A.C. Vaeth, Dr.P.H.; and Raul Caetano, M.D., Ph.D.

Alcohol consumption is differentially associated with social and health harms across U.S. ethnic groups. Native Americans, Hispanics, and Blacks are disadvantaged by alcohol-attributed harms compared with Whites and Asians. Ethnicities with higher rates of risky drinking experience higher rates of drinking harms. Other factors that could contribute to the different effects of alcohol by ethnicity are social disadvantage, acculturation, drink preferences, and alcohol metabolism. This article examines the relationship of ethnicity and drinking to (1) unintentional injuries, (2) intentional injuries, (3) fetal alcohol syndrome (FAS), (4) gastrointestinal diseases, (5) cardiovascular diseases, (6) cancers, (7) diabetes, and (8) infectious diseases. Reviewed evidence shows that Native Americans have a disproportionate risk for alcohol-related motor vehicle fatalities, suicides and violence, FAS, and liver disease mortality. Hispanics are at increased risk for alcohol-related motor vehicle fatalities, suicide, liver disease, and cirrhosis mortality; and Blacks have increased risk for alcohol-related relationship violence, FAS, heart disease, and some cancers. However, the scientific evidence is incomplete for each of these harms. More research is needed on the relationship of alcohol consumption to cancers, diabetes, and HIV/AIDS across ethnic groups. Studies also are needed to delineate the mechanisms that give rise to and sustain these disparities in order to inform prevention strategies. Key words: Alcohol consumption; alcohol-attributable fractions; alcohol burden; harmful drinking; alcohol and other drug–induced risk; risk factors; ethnicity; ethnic groups; racial groups; cultural patterns of drinking; Native Americans; Hispanics; Blacks; African Americans; Asian Americans; Whites; Caucasians; injury; intentional injury; unintentional injury; fetal alcohol syndrome; gastrointestinal diseases; cardiovascular diseases; cancers; diabetes; infectious diseases

Drinking Patterns and Other Determinants of Risk for Alcohol-Related Harms

Heavy drinking and binge drinking contribute to a variety of alcohol-attributed social and health harms (Naimi et al. 2003; Rehm et al. 2010). Heavy alcohol use, as defined by the National Institute on Alcohol Abuse and Alcoholism’s (NIAAA’s) Helping Patients Who Drink Too Much: A Clinician’s Guide (NIAAA 2005), is defined as consuming more than 4 standard drinks per day (or more than 14 per week) for men and more than 3 per day (or more than 7 per week) for women. One standard drink is equivalent to 12 ounces of...
Beer, 5 ounces of wine, or 1.5 ounces of 80-proof spirits. Binge drinking is defined as consuming five or more drinks in approximately 2 hours for men and four or more drinks for women (NIAAA 2004).

Other than these patterns of consumption, the volume of alcohol intake, defined as the total alcohol consumed over a time period, is linked to social and health harms. Most diseases (e.g., injury, some cancers, and liver cirrhosis) have a detrimental dose-response relationship with alcohol as risk increases with higher-volume alcohol consumption, whereas coronary heart disease and diabetes display a J- or U-shaped relationship (Howard et al. 2004; Rehm et al. 2010; Roerecke and Rehm 2012). The J and U shapes are characterized by both detrimental and beneficial (e.g., increased high-density lipoprotein “good cholesterol”) (Goldberg and Soles 2001) effects of alcohol use, with higher risks for abstainers and heavy drinkers compared with light or moderate drinkers. However, this relationship is complex and varies by age, gender, and ethnicity (Roerecke and Rehm 2012). Drinking levels that may be protective of cardiovascular health among men also may increase the risk for other harms such as injury, violence, gastrointestinal disease, and some cancers.

Epidemiological studies show that these high-risk patterns of drinking and drinking volume vary by U.S. ethnic group. Ethnicities with greater drinking volume and higher rates of daily and weekly heavy drinking could be at greater risk for experiencing alcohol-attributed harms. Among adult drinkers in the United States, based on the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) (Chen et al. 2006), Native Americans and Hispanics have greater alcohol consumption than other ethnic minority groups. Rates of daily heavy drinking were higher among Hispanics (33.9 percent), Native Americans (28.4 percent), and Whites (27.3 percent) compared with Blacks (22.5 percent) and Asians (19.2 percent). Weekly heavy drinking was highest among Native Americans (21.9 percent), followed by Blacks (16.4 percent), Whites (16.3 percent), Hispanics (11.8 percent), and Asians (9.8 percent). Based on the 2001–2002 NESARC data, Caetano and colleagues (2010) reported that White men consumed a higher volume of alcohol (22.3 drinks per month) than Black men (18.9 drinks per month) and Hispanic men (17.8 drinks per month) and that White women consumed more (6.2 drinks per month) compared with Black women (4.9 drinks per month) and Hispanic women (3.9 drinks per month). The sample for these estimates of drinking volume was the U.S. population of Whites, Blacks, and Hispanics and included abstainers. However, a study by Mulia and colleagues (2009) of current drinkers in the United States showed that Whites consumed less alcohol than Hispanics and more than Blacks. The differences between these two studies could reflect a higher rate of abstinence from alcohol among Hispanics (25.7 percent) compared with Whites (13.4 percent) in the U.S. population (Chen et al. 2006). The study that included abstainers (Caetano et al. 2010), who by definition consume zero drinks, showed higher drinking volume for Whites, whereas the study excluding abstainers (Mulia et al. 2009) reported higher volume for Hispanics. Other ethnic minority groups with higher abstinence rates include Blacks (24.7 percent) and Asians (39.1 percent). Native Americans (17.14 percent) have lower rates of abstinence than other minority groups.

Alternatively, the negative effects from drinking could be explained by factors other than alcohol consumption. Mulia and colleagues (2009) showed that Black and Hispanic adult drinkers were more likely than White drinkers to report alcohol dependence symptoms and social problems from drinking at the no/low level of heavy drinking. Blacks also experience negative health effects from alcohol use despite showing a later onset of use and levels of use often comparable with, if not lower than, Whites (Chartier et al. 2011; Chen et al. 2006; Russo et al. 2004). Other factors associated with ethnic disparities in alcohol-related harms include social disadvantage, characterized by lower socioeconomic status, neighborhood poverty, greater neighborhood alcohol availability, reduced alcohol treatment utilization, and unfair treatment or discrimination (Chae et al. 2008; Chartier and Caetano 2011; Cunradi et al. 2000; Mulia et al. 2008; Nielsen et al. 2005; Zemore et al. 2011). Some ethnic subgroups are more likely to consume high-alcohol-content beverages (e.g., malt liquor), which could result in greater social and health harms (Vilamovska et al. 2009). Preference for such beverages seems to be more common in lower-income ethnic minority communities (Bluthenthal et al. 2005). Some ethnic minority groups also face stressors related to the acculturation process. Higher acculturation, U.S.-born nativity, and longer residence in the United States are risk factors associated with alcohol use disorders and alcohol-related social problems among Hispanics, particularly women (Alegria et al. 2007, 2008; Caetano et al. 2009, 2012; Zemore 2007). Another potential contributor is ethnic differences in the alcohol content of poured drinks. Kerr and colleagues (2009) showed that Black men had drink sizes with larger average alcohol content compared with other groups, which partially could explain the higher risks for alcohol-related harms. Genes responsible for alcohol metabolism also vary across ethnic groups and could be associated with susceptibility for alcohol-related diseases. Among Whites, Blacks, and Asians, alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) genotypes have been linked in combination with drinking to alcohol-related cancers, birth defects, and pancreatitis (Yin and Agarwal 2001).

Ethnicity and Alcohol-Attributed Harms

Alcohol-attributed harms can be both acute and chronic conditions that are wholly caused (e.g., alcoholic liver cirrhosis) or associated with alcohol use via intoxication, alcohol dependence, and the toxic effects of alcohol (Rehm et al. 2010). The major injury and disease categories linked to alcohol consumption include (1) unintentional injuries, (2) intentional injuries, (3) FAS, (4) gastrointestinal diseases, (5) cardiovascular diseases, (6) cancers, (7) diabetes, and (8) infectious diseases (World Health Organization [WHO] 2011). Evidence
is incomplete on the relationship between ethnicity, drinking, and each of these categories. Below, those alcohol-related harms are described that have available findings by ethnic group in addition to important gaps in this scientific literature. Alcohol use disorders are causally linked to drinking and vary by ethnicity (i.e., more likely in Native Americans and Whites) (Hasin et al. 2007), but this disease category is not described here.

**Unintentional Injuries**

Unintentional injuries associated with alcohol use include falls, drowning, and poisoning (WHO 2011). However, most available research on ethnicity, alcohol use, and injuries is focused on motor vehicle crashes. Alcohol-impaired driving and crash fatalities vary by ethnicity, with Native Americans and Hispanics being at higher risk than other ethnic minority groups. Past-year driving under the influence (DUI) estimates based on the 2007 National Survey on Drug Use and Health were highest for Whites (15.6 percent) and Native Americans (13.3 percent) relative to Blacks (10.0 percent), Hispanics (9.3 percent), and Asians (7.0 percent) (Substance Abuse and Mental Health Services Administration [SAMHSA] 2008). National surveys generally show lower DUI rates for Hispanics than Whites, but studies based on arrest data identify Hispanics as another high-risk group for DUI involvement (Caetano and McGrath 2005; SAMHSA 2005). The DUI arrest rate for Native Americans in 2001, according to the U.S. Department of Justice (Perry 2004), was 479 arrestees per 100,000 residents compared with 332 for all other U.S. ethnic groups.

Based on a 1999–2004 report from the National Highway Traffic Safety Administration (Hilton 2006), rates of intoxication (i.e., blood alcohol concentration [BAC] more than or equal to 0.08 percent) for drivers who were fatally injured in a motor vehicle crash were highest for Native Americans (57 percent) and Hispanics (47 percent) and lowest for Asians (approximately 20 percent), with Whites and Blacks falling in between. Across ethnic groups, most drinking drivers killed were male, although the proportion of female drivers who were intoxicated among fatally injured drivers was highest (i.e., more than 40 percent) for Native Americans. Centers for Disease Control and Prevention (CDC) (2009b) statistics on alcohol-related motor vehicle crash deaths also point to an important subgroup difference for Asians. In 2006, the overall death rate among Asians (1.8 per 100,000 people) obscured the death rate among Native Hawaiians and other Pacific Islanders (5.9), which was less than the rate for Native Americans but similar to that for Hispanics (14.5 and 5.2, respectively).

**Intentional Injuries**

**Suicide**

Native Americans are overrepresented in national estimates of alcohol-involved suicides. A CDC report (2009a) based on 2005–2006 data from the National Violent Death Reporting System presented findings on alcohol and suicide across ethnic groups. Recent alcohol use was reported among suicides in 46 percent of Native Americans, 30 percent of Hispanics, 26 percent of Whites, 16 percent of Blacks, and 15 percent of Asians. Among those tested for alcohol, the rates of intoxication (BAC higher than or equal to 0.08) were highest for Native Americans (37 percent), followed by Hispanics (29 percent), Whites (24 percent), Blacks (14 percent), and Asians (12 percent). Age-groups identified as being at high risk for alcohol-involved suicide included Native Americans ages 30 to 39 (54 percent of suicide victims had BACs higher than or equal to 0.08), Native Americans and Hispanics ages 20 to 29 (50 percent and 37 percent, respectively), and Asians ages 10 to 19 (29 percent). Males were at higher risk than female drinkers in all ethnic groups except Native Americans; the percentages of alcohol intoxication among Native American suicides were equal for males and females (37 percent).

**Violence**

Ethnic groups are differentially affected by alcohol-attributed violence, including intimate-partner violence (IPV). Alcohol plays an important role in IPV and other types of relationship conflicts (Field and Caetano 2004; Leonard and Eiden 2007). Based on data from the National Study of Couples, general rates of male-to-female partner violence (MFPV) and female-to-male partner violence (FMPV), are highest among Black couples (23 percent and 30 percent, respectively), followed by Hispanic (17 percent and 21 percent) and White (12 percent and 16 percent) couples (Caetano et al. 2000). The National Study of Couples provides general population data on IPV, which includes mostly moderate violence and may differ from other studies of severe violence. In this study, regardless of ethnicity, men were more likely than women to report drinking during partner violence. Drinking during a violent episode by the male or the female partner, respectively, was more frequent among Blacks (MFPV: 41.4 percent and 23.6 percent; FMPV: 33.7 percent and 22.4 percent) than among Whites (MFPV: 29.4 percent and 11.4 percent; FMPV: 27.1 percent and 14.7 percent) and Hispanics (MFPV: 29.1 percent and 5.4 percent; FMPV: 28.4 percent and 3.8 percent). Longitudinal findings, using 5-year National Study of Couples data, identified female-partner alcohol problems (i.e., alcohol dependence symptoms and social problems) in Black couples and male- and female-partner alcohol consumption in White couples as risk factors for IPV (Field and Caetano 2003). Some evidence also suggests that interethnic couples, involving White, Black, and Hispanic partners of different ethnic backgrounds, are a high-risk group for relationship violence. Relative to intraracial couples, these interethnic couples had higher prevalence rates of IPV, which was associated with binge drinking and alcohol problems among male partners (Chartier and Caetano 2012). Alcohol also contributes to violence victimization among Native Americans (Yuan et al. 2006). Several studies...
indicate that Native Americans are at greater risk for alcohol-related trauma (e.g., IPV, rape, and assault) compared with other U.S. ethnic groups (Oetzel and Duran 2004; Wahab and Olson 2004). Based on 1992–2001 National Crime Victimization Survey data, the U.S. Department of Justice (Perry 2004) reported that 42 percent of all violent crimes (i.e., rape, sexual assault, robbery, aggravated assault, and simple assault) were committed by an offender who was under the influence of alcohol. In particular, Native American violent crime victims were more likely (62 percent) than other violent crime victims to report alcohol use by their offender, including Whites (43 percent), Blacks (35 percent), and Asians (33 percent).

Fetal Alcohol Syndrome

Using data from the 2001–2002 NESARC, Caetano and colleagues (2006) examined alcohol consumption, binge drinking, and alcohol abuse and dependence among women who were pregnant during the past year. Most women (88 percent) who reported being pregnant and also a drinker at any point in the past 12 months indicated that they did not drink during pregnancy. Rates of past-year alcohol abuse (0.8 percent to 2.3 percent) and dependence (1.2 percent to 2.8 percent) were similar and low in White, Black, Hispanic, and Asian pregnant women. Binge drinking and alcohol consumption without binge drinking among pregnant women were highest in Whites (21.1 percent and 45.0 percent, respectively) compared with other ethnic groups (0 percent to 10.7 percent and 21.0 percent to 37.3 percent). White women in this study were at greater risk for an alcohol-exposed pregnancy. However, other studies found that Black, Hispanic, and Asian women were less likely to reduce or quit heavy drinking after becoming pregnant (Morris et al. 2008; Tenkku et al. 2009). Blacks and Native Americans are at greater risk than Whites for FAS and fetal alcohol spectrum disorders (Russo et al. 2004). From 1995 to 1997, FAS rates averaged 0.4 per 1,000 live births across data-collection sites for the Fetal Alcohol Syndrome Surveillance Network and were highest for Black (1.1 percent) and Native American (3.2 percent) populations (CDC 2002).

Gastrointestinal Diseases

Liver disease is an often-cited example of the disproportionate effect of alcohol on health across ethnic groups. Native Americans have higher mortality rates for alcoholic liver disease than other U.S. ethnic groups (see figure). According to the National Vital Statistical Reports (Miniño et al. 2011) on 2008 U.S. deaths, age-adjusted death rates attributed to alcoholic liver disease for Native American men and women were 20.4 and 15.3 per 100,000 people, respectively, compared with 6.9 and 2.4 per 100,000 for men and women in the general population. Blacks and Hispanics have greater risk for developing liver disease compared with Whites (Flores et al. 2008), and

![Figure](image-url)
death rates attributed to alcohol-related cirrhosis across populations of Whites, Blacks, and Hispanics are highest for White Hispanic men (Yoon and Yi 2008). Blacks show a greater susceptibility than Whites to alcohol-related liver damage, with risk differences amplified at higher levels of consumption (Stranges et al. 2004). Based on data from the National Center for Health Statistics, 1991–1997, mortality rates for cirrhosis with mention of alcohol were higher in White Hispanics and Black non-Hispanics compared with White non-Hispanics (Stinson et al. 2001). Male mortality rates for alcohol-related cirrhosis in White Hispanics and non-Hispanic Blacks were 114 percent and 24 percent higher, respectively, than the overall male rate (5.9 deaths per 100,000 people); female rates in White Hispanics and non-Hispanic Blacks were 16 percent and 47 percent higher than the overall female rate (1.9 deaths per 100,000 people). In contrast, death rates for White non-Hispanic and Black Hispanic males and females were lower than overall rates for each gender. In addition, there is considerable variation in deaths from liver cirrhosis across Hispanic subgroups, with mortality rates highest in Puerto Ricans and Mexicans and lowest in Cubans (Yoon and Yi 2008).

**Cardiovascular Diseases**

Although moderate alcohol consumption has been associated with a reduced risk for coronary heart disease (CHD) (Goldberg and Soleas 2001), there is some evidence that ethnic groups differ in terms of this protective effect, particularly for Blacks compared with Whites. Semoes and colleagues (2003) found no protective health effect for moderate drinking in Blacks for all-cause mortality, as previously reported in Whites. Kerr and colleagues (2011) reported the absence of this protective effect for all-cause mortality in Blacks and Hispanics. Similar findings have been described for hypertension and CHD risks in Black men compared with White men and women (Fuchs et al. 2001, 2004) and for mortality among Black women without hypertension (Freiberg et al. 2009). Mukamal and colleagues (2010) also showed that the protective effects of light and moderate drinking in cardiovascular mortality were stronger among Whites than non-Whites. Pletcher and colleagues (2005) found evidence that the dose-response relationship between alcohol consumption and increased coronary calcification, a marker for CHD, was strongest among Black men.

**Cancers**

In 1988, the WHO International Agency for Research on Cancer (IARC) reviewed the epidemiologic evidence on the association between alcohol consumption and cancer and found a consistent association between alcohol consumption and increased risk for cancers of the oral cavity, pharynx, larynx, esophagus, and liver (IARC 1988). Regardless of ethnicity, the risk of developing these cancers is significantly higher among men than women (National Cancer Institute 2011c, d, e). The incidence and mortality rates for these cancers also vary across ethnic groups. Regarding cancers of the oral cavity and pharynx, incidence rates among White and Black men are comparable (16.1 and 15.6 per 100,000, respectively); however, mortality rates are higher among Black men (6.0 versus 3.7 per 100,000 for White men) (National Cancer Institute 2011c). For cancer of the larynx, both incidence and mortality rates are higher among Black men than among White men (incidence, 9.8 and 6.0; mortality, 4.4 and 2.0) (National Cancer Institute 2011c). Although these differences may be explained by differential use of alcohol and tobacco in relation to gender and ethnicity, there is some evidence that even after controlling for alcohol and tobacco use, Blacks continue to be at increased risk for squamous cell esophageal cancer and cancers of the oral cavity and pharynx (Brown et al. 1994; Day et al. 1993).

The majority (approximately 90 percent) of all primary liver cancers are hepatocellular carcinomas (HCC) (Altekruse et al. 2009). Alcohol-related and non–alcohol-related liver cirrhosis usually precede HCC and are the two most common risk factors (Altekruse et al. 2009; El-Serag 2011; Pelucchi et al. 2006). The relative risk for developing this cancer increases with increased levels of alcohol consumption (Pelucchi et al. 2006). By ethnic group, 2003–2005 age-adjusted incidence rates for HCC per 100,000 persons were highest among Asians (11.7), followed by Hispanics (8.0), Blacks (7.0), Native Americans (6.6), and Whites (3.9) (Altekruse et al. 2009). Death rates for HCC per 100,000 people also are higher among minority groups (i.e., 8.9, 6.7, 5.8, 4.9, and 3.5 for Asians, Hispanics, Blacks, Native Americans, and Whites, respectively).

In 2007, the IARC reconvened and added breast and colorectal cancers to the list of cancers related to alcohol use (Baan et al. 2007). Research has demonstrated consistent, albeit weak, dose-response relationships between alcohol consumption and these cancers (Cho et al. 2004; Collaborative Group on Hormonal Factors in Breast Cancer 2002; Moskal et al. 2007; Singletary and Gapstur 2001). Alcohol consumption also contributes to the stage at which breast cancer is diagnosed (Hebert et al. 1998; Trentham-Dietz et al. 2000; Vaeth and Satariano 1998; Weiss et al. 1996). This could be because of the timing of disease detection, since heavy drinking has been associated with a lack of mammography utilization (Cryer et al. 1999). Alcohol consumption also may contribute to more rapid tumor proliferation (Singletary and Gapstur 2001; Weiss et al. 1996). Data from the Surveillance, Epidemiology, and End Results (SEER) Program indicate that White women, relative to women from ethnic minority groups, have higher incidence rates of breast cancer (i.e., Whites, 127.3; Blacks, 119.9; Asians, 93.7; Native Americans, 92.1; and Hispanics, 77.9 per 100,000 people) (National Cancer Institute 2011a). Black women, however, are more likely to be diagnosed with advanced disease (Chlebowski et al. 2005) and have significantly higher mortality rates than White women (i.e., 32.0
per 100,000 versus 22.8 per 100,000 people) (Chlebowski et al. 2005; National Cancer Institute 2011a). Regarding colorectal cancer, Blacks have higher incidence (67.7) and mortality (51.2) rates than all ethnic groups combined (55.0 and 41.0, respectively) (National Cancer Institute 2011b). Unfortunately, little is known about how drinking differentially affects ethnic differences in breast and colorectal cancers.

Infectious Diseases

Among the infectious diseases attributable to alcohol (e.g., pneumonia, tuberculosis) (WHO 2011), human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) are most relevant to U.S. ethnic health disparities. In 2009, Blacks represented 44 percent of new HIV infections and Hispanics represented 20 percent. Infection rates by gender for Blacks were 15 times (for men) and 6.5 times (for women) those of Whites, and rates for Hispanics were 4.5 times for men and 2.5 times for women, compared with rates for Whites (CDC 2011). In addition, alcohol consumption has been associated with increased HIV infection risk (Bryant et al. 2010). Caetano and Hines (1995) showed that heavy drinking predicted high-risk sexual behaviors in White, Black, and Hispanic men and women, with more Blacks than Whites and Hispanics reporting risky sexual behaviors. Among HIV-infected patients, there also is evidence that increased alcohol consumption negatively affects adherence to antiretroviral medication regimens (Chander et al. 2006; Cook et al. 2001; Samet et al. 2004) and HIV disease progression (Conigliaro et al. 2003; Samet et al. 2003). Despite these strong individual associations between ethnicity and HIV/AIDS and alcohol and HIV/AIDS, there is limited research across ethnicities on alcohol use and HIV infection or disease progression.

Diabetes

In 2010, the prevalence of diabetes was 7.1 percent, 12.6 percent, 11.8 percent, and 8.4 percent among Whites, Blacks, Hispanics, and Asians, respectively (National Institute of Diabetes and Digestive and Kidney Diseases 2011). Age-adjusted mortality rates in 2007 were 20.5, 42.8, 28.9, and 16.2 per 100,000 people among Whites, Blacks, Hispanics, and Asians (National Center for Health Statistics 2011). Data on mortality rates for diabetes among Hispanics may be underreported as a result of inconsistencies in the reporting of Hispanic origin on death certificates (Heron et al. 2009). Despite higher risks for the development of and death from diabetes in Hispanics and Blacks compared with Whites, little evidence is available to delineate the relationship of alcohol to diabetes across ethnic groups. Studies among both diabetics and nondiabetics demonstrate a J- or U-shaped curve between alcohol consumption and insulin sensitivity (Bell et al. 2000; Davies et al. 2002; Greenfield et al. 2003; Kronke et al. 2003). Likewise, two large epidemiologic studies among diabetic subjects show that moderate alcohol consumption is associated with better glycemic control (Ahmed et al. 2008; Mackenzie et al. 2006). An important limitation of these studies, however, is that few included ethnic minority groups or failed to emphasize possible differences in relation to ethnicity in their analyses.

Conclusions

This article identifies U.S. ethnic-group differences in alcohol-attributed social and health-related harms. Three minority ethnicities are particularly disadvantaged by alcohol-related harms. Native Americans, relative to other ethnic groups, have higher rates of alcohol-related motor vehicle fatalities, suicide, violence, FAS, and liver disease mortality. Unlike other ethnic groups, in which men are primarily at risk for alcohol-related harms, both Native American men and women are high-risk groups. Hispanics have higher rates of alcohol-related motor vehicle fatalities, suicide, and cirrhosis mortality. Blacks have higher rates of FAS, intimate partner violence, and some head and neck cancers, and there is limited empirical support in Blacks for a protective health effect from moderate drinking. These patterns of findings provide recognition of the health disparities in alcohol-attributed harms across U.S. ethnicities. However, further research is needed to identify the mechanisms that give rise to and sustain these disparities in order to develop prevention strategies. The contributing factors include the higher rates of consumption found in Native Americans and Hispanics, but more broadly range from biological factors to the social environment. More research on the relationship of alcohol to some cancers, diabetes, and HIV/AIDS across ethnic groups is also needed. There is limited evidence for how drinking differentially affects ethnic differences in breast and colorectal cancers and in diabetes and HIV/AIDS onset and care, and few findings for how alcohol-attributed harms vary across ethnic subgroups.

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References


PETCHER, M.J.; VARDY, P.; KEEF, C.I.; ET AL. Alcohol consumption, binge drinking, and early coronary calcification: Findings from the Coronary Artery Risk Development in


Heavy drinking causes significant morbidity, premature mortality, and other social and economic burdens on society, prompting numerous prevention and treatment efforts to avoid or ameliorate the prevalence of heavy drinking and its consequences. However, the impact on public health of current selective (i.e., clinical) prevention and treatment strategies is unclear. Screening and brief counseling for at-risk drinkers in ambulatory primary care has the strongest evidence for efficacy, and some evidence indicates this approach is cost-effective and reduces excess morbidity and dysfunction. Widespread implementation of screening and brief counseling of nondependent heavy drinkers outside of the medical context has the potential to have a large public health impact. For people with functional dependence, no appropriate treatment and prevention approaches currently exist, although such strategies might be able to prevent or reduce the morbidity and other harmful consequences associated with the condition before its eventual natural resolution. For people with alcohol use disorders, particularly severe and recurrent dependence, treatment studies have shown improvement in the short term. However, there is no compelling evidence that treatment of alcohol use disorders has resulted in reductions in overall disease burden. More research is needed on ways to address functional alcohol dependence as well as severe and recurrent alcohol dependence. Key words: Alcohol use, abuse, and dependence; heavy drinking; alcohol use disorders (AUDs); alcohol-related problems; alcohol burden; burden of disease; morbidity; mortality; prevention; treatment; prevention strategy; treatment strategy; screening and brief intervention; primary care; cost-effectiveness of AOD health services

Selective Prevention and Treatment: Effectiveness, Cost-Effectiveness, and Disease Burden

Screening and brief advice for at-risk (i.e., nondependent) drinkers, commonly known as screening and brief intervention (SBI), is effective at reducing drinking for a year or more and in many studies also has been shown to reduce alcohol-related harms, such as motor-vehicle crashes and driving violations. Its efficacy is supported by numerous randomized controlled trials and multiple meta-analyses; as a result, the U.S. Prevention Task Force has listed it as a Type B recommendation for medical prevention services (Babor et al. 2007; Whitlock et al. 2004). The evidence is strongest for nondependent heavy drinkers who present for primary care services in ambulatory settings. Unfortunately, a recent meta-analysis of studies of SBI in primary care settings failed to show significant reductions in subsequent health care utilization (Bray et al. 2011). The efficacy of SBI in other settings, such as emergency departments (EDs) or hospitals, has not been established, although several randomized controlled trials have been conducted (Field et al. 2010). One explanation for the observed differences may be the patient populations analyzed. Thus, in most of the outpatient primary care studies, participants with alcohol dependence were excluded from the analysis, whereas that generally was not the case in ambulatory care studies.
the case for studies conducted in EDs or hospital settings. Moreover, patients with alcohol dependence are much more commonly encountered in ED and hospital settings than in primary ambulatory care. In summary, at this time, SBI in primary care ambulatory settings for adults can be strongly recommended as highly efficacious, whereas SBI in EDs or hospitals cannot.

SBI also seems to be effective among select groups when delivered through internet-based or computerized applications. In particular, there is strong evidence that digital SBI can effectively reduce drinking and associated consequences among college students (Moreira et al. 2009). It is not clear whether or to what extent this finding might generalize to other population subgroups, but it is certainly plausible that it could, provided the target population has easy access to computers and is computer literate. The same holds true for other methods, such as telephone-based SBI or use of the relatively new publication and Web site called Rethinking Drinking, which is published by the National Institute on Alcohol Abuse and Alcoholism (NIAAA).

Despite the evidence supporting its effectiveness, SBI is not yet being implemented widely (Hingson et al. 2012). Widespread dissemination of information about recommended drinking limits and easy access to screening and brief counseling has the potential to make a significant public health impact. Because at-risk drinkers are much more numerous than alcohol-dependent people, at-risk drinking contributes a much greater disease burden than alcohol dependence. Accordingly, widespread implementation of SBI has the potential to reduce a greater proportion of disease burden than even very effective treatment, a concept known as the prevention paradox (Rose 1981). Therefore, more research is needed to expand the implementation of SBI in the at-risk population and further increase its effectiveness.

Estimating the effectiveness and cost-effectiveness of treatment is more complex. Most reviews conclude that treatment is effective at reducing drinking and associated consequences. Multiple behavioral treatment approaches—such as cognitive behavioral therapy, motivational enhancement therapy, 12-step facilitation, behavioral marital therapy, and community reinforcement—have similar and relatively high levels of short-term success in reducing drinking and associated consequences, at least when treatment is provided by the highly trained, motivated, and closely supervised clinicians participating in clinical efficacy trials (Project MATCH Research Group 1998). Why these technically diverse counseling techniques produce almost identical drinking outcomes is unclear. Three alternative explanations have been offered:

- The specific technique is less important than other, mostly unidentified, factors associated with psychotherapy.
- Each approach works via different mechanisms but produces similar results on average, much like different antidepressants acting through different mechanisms produce similar outcomes in the treatment of depression.
- Professional treatment only has a small effect in determining outcome compared with other, nontreatment factors, such as social control (e.g., driving-while-intoxicated laws, family pressure, or employer mandate), natural history of alcohol dependence, and the tendency to revert to usual levels of drinking following resolution of a crisis where drinking had peaked (i.e., regression to the mean).

This last explanation is supported by recent research demonstrating that changes in drinking habits begin weeks before treatment entry (Penberthy et al. 2007). Likewise, in another study of treatment of alcohol dependence that examined events leading to treatment seeking (Orford et al. 2006), the findings suggested that the change point occurred prior to treatment entry. Thus, it is unclear how much of the positive change can be attributed to the treatment processes themselves as opposed to other factors leading to and following treatment seeking.

What is clear, however, is that researchers and clinicians do not yet understand how or why some people change in response to treatment and others do not. To address this issue, NIAAA led the way at the National Institutes of Health (NIH) in shifting the focus of behavioral treatment research to identifying the mechanisms of behavior change rather than encouraging more comparisons of different psychotherapy approaches (Willenbring 2007). The NIH subsequently developed a major initiative on basic behavioral research (Li 2009). This research initiative provides an opportunity to investigate many obvious questions. For example, what are the social forces that either support or impede positive health behavior change? What determines their impact, in terms of the response of the individual? Why and how do people begin to change, and what determines the resilience of that change? What is the basic science underlying behavior change, at all levels from genetic and genomic to cellular, organic, individual, and social interactions? Research elucidating the basic science of behavior change is an exciting and promising area that has the potential to substantially change the types of interventions that are available, making them more powerful, available, and cost-effective.

The lack of clarity about what causes change in drinking behavior also results in uncertainty as to whether treatment of alcohol dependence reduces disease burden. The community prevalence of alcohol dependence, which is about 4 percent in any year, has not changed substantially in recent years (Substance Abuse and Mental Health Services Administration 2011). Earlier studies found a cost offset of treatment—that is, lower health care costs after treatment than before treatment (Holder 1998). More recent studies, however, have found that heavy drinkers who are not in crisis underutilize health care, at least in an employed population, suggesting that the observed cost reduction is more a reflection of the natural history of drinking behavior and of a regression to the mean (Finney 2008; Zarkin et al. 2004). In other words, people suffering from any disease tend to seek treatment when their condition is most severe. In the case of alcohol dependence, treatment seeking therefore would be preceded by an escala-
tion of drinking, complications, and utilization of medical services and, consequently, high costs before treatment entry. Because chronic conditions such as alcohol dependence wax and wane, most people will tend to improve after a period of greater severity, even without effective treatment, so that subsequent reduced costs may not necessarily be associated with treatment. Also, every patient’s disease trajectory is different, so that when drinkers are assessed before and after treatment, some of them will be well at followup, whereas for others their condition will be more severe. The average severity, however, will be less following treatment, because for all patients studied, their disease severity at treatment entry will have been high. The most rigorous study of cost-effectiveness of alcoholism treatment, the COMBINE trial, found that treatment was cost-effective, especially pharmacotherapy with medical management (Zarkin et al. 2008, 2010). The interpretation of these findings is limited, however, by the study’s highly rigorous trial design, intensive follow up, and exclusion criteria (Anton et al. 2006), and it is unknown to what extent these findings generalize to community treatment programs and participants.

Another limitation when estimating the effects of treatment on public health is that relatively few affected people seek treatment. For example, among people who develop alcohol dependence at some point in their lives only 12 percent seek treatment in a specialty treatment program (Hasin et al. 2007). Among people who have AUDs and who perceive a need for treatment, almost two-thirds (i.e., 65 percent) fail to obtain it because they are not ready to stop drinking or feel they can handle it on their own. Other common reasons for the failure to seek treatment include practical barriers, such as lack of health insurance, the cost of treatment, and lack of transportation or access to treatment, which are reported by 59 percent of respondents, and stigma, which is reported by 31 percent (Center for Behavioral Health Statistics and Quality 2012). Thus, more people might seek treatment if it was less expensive, stigmatizing, and disruptive than most treatment approaches. Efforts to improve access, affordability, and attractiveness of treatment, especially for individuals with less severe AUDs should be encouraged.

Despite these limitations, some tentative conclusions can be drawn as to which approaches to treating alcohol dependence are more cost effective. Studies found no significant difference in outcomes between residential and outpatient treatment and no clear relationship between intensity of treatment and outcome (Fink et al. 1985; Longabaugh et al. 1983; McCrady 1986). For example, medical management plus pharmacotherapy with naltrexone generated similar outcomes to more expensive counseling approaches, even when counseling was performed once weekly and on an outpatient basis (Anton et al. 2006; O’Malley et al. 2003). These studies suggest that a more individualized, outpatient, and medically based approach may provide a cost-effective alternative to approaches favoring intensive psycho-education, which often are provided in residential settings. Treatment provided in residential rather than outpatient settings may add considerable expense without a commensurate improve-ment in outcomes. In addition, confidential treatment by their usual primary care physician involving only routine clinic visits may attract more people, thus expanding access to effective treatments.

Gaps in the Continuum of Care

There are several gaps in the continuum of care that deserve attention, affecting drinkers across the spectrum of alcohol involvement. Recent epidemiological research has demonstrated that alcohol involvement varies along a continuum ranging from asymptomatic heavy drinking (i.e., at-risk drinking), through functional alcohol dependence, and to severe and recurrent alcohol dependence (Willenbring et al. 2009). The continuum of care ideally should correspond to this epidemiology but does not at this time. Most studies and treatment approaches have focused on the more severe end of the spectrum—that is, people with severe, recurrent dependence. However, the vast majority of heavy drinkers either do not have alcohol dependence or has a relatively milder, self-limiting form (Moss et al. 2007). This spectrum of severity is similar to that for other chronic diseases, such as asthma. Likewise, examining treatment seekers in the current system of care yields similar results to studying hospitalized asthmatics: thus, heavy drinkers in treatment exhibit more severe dependence, more comorbidity, less response to treatment, and a less supportive social network compared with people who do not seek intensive treatment (Bischof et al. 2003; Dawson et al. 2005; Sobell et al. 2000). In contrast, people with functional alcohol dependence predominantly exhibit “internal” symptoms, such as impaired control; a persistent desire to cut down on their drinking but finding it hard to do; and alcohol use despite internal symptoms such as insomnia, nausea, or hangover. These individuals generally drink much less than more seriously affected people (Moss et al. 2007). Functional alcohol dependence typically resolves after a few years, mostly without requiring specialty treatment (Hasin et al. 2007). Large gaps in services exist for people at both ends of the spectrum of dependence severity—that is, both for people at the milder end of the spectrum (i.e., at-risk drinkers and people with functional alcohol dependence) and for those at the most severe end (i.e., with recurrent, treatment-refractory dependence).

There currently are few services for at-risk drinkers and people with functional alcohol dependence. In primary medical care, very few patients are screened and positive screening results addressed (McGlynn et al. 2003). Furthermore, functional alcohol dependence largely is ignored because although these individuals meet diagnostic criteria for dependence, they rarely seek treatment in the current system (Moss et al. 2007). These gaps are significant from a public health perspective because the prevalence of at-risk drinking...
and functional dependence is much higher than that of more severe disorders and these conditions therefore account for the majority of excess morbidity, mortality, and associated costs attributable to alcohol consumption (Centers for Disease Control and Prevention 2012). Whether wider implementation of SBI would result in a reduction in disease burden is not known at this time. However, enhancement of these approaches, especially among young people and community-dwelling heavy drinkers not seeking medical care, might reduce disease burden, although the two populations require somewhat distinct approaches. More studies of secondary prevention efforts outside of medical settings therefore are needed.

SBI in primary care settings to identify people with AUDs at the milder end of the severity spectrum is effective and may be cost-effective (Solberg et al. 2008), but many questions remain. For example, is it more cost-effective to target higher-risk groups (e.g., young people) for routine screening or is universal screening better overall? And when should screening occur (e.g., only during annual prevention visits or at every new patient visit) and how often should it be repeated? However, the biggest problem remains that effective selective prevention interventions such as SBI are not widely implemented. Although implementation has worked well in situations where additional grant funds were available, it still is unknown whether physicians will engage in this widely or how to best facilitate implementation. The Veterans Affairs health services system has been the most effective at implementing annual screening, but this system is unique in its structure and hierarchical nature. Implementation of such approaches in private health care organizations is much more complex and difficult. Therefore, more research is needed on low-cost ways to encourage wider adoption of SBI in primary care settings. Additional research should focus on SBI in other medical settings, especially mental health settings and medical specialties particularly affected by heavy drinking, such as gastroenterology (with patients with alcohol-related liver disease, gastritis, and pancreatitis) and otolaryngology (with patients with alcohol-related head and neck cancers).

Because so many hospitalized heavy drinkers have dependence, SBI is much less effective in this group (Saitz et al. 2007) and its effectiveness with patients in EDs or trauma centers also is unknown. Although some early studies showed positive results, subsequent research has yielded as many negative as positive findings (Field et al. 2010). Current efforts to implement SBI in these more acute-care settings therefore are premature, and more research is needed to determine if heavy drinkers encountered in such settings require more intensive services, linkage to ambulatory care services, or both.

People with functional alcohol dependence likely require more than brief counseling, but there is a major gap in research concerning optimal treatment strategies. Currently, few, if any, services are available for this group because they fall between at-risk drinkers and those with severe recurrent alcohol dependence (who are most likely to enter the current specialty treatment system). Pharmacotherapy (e.g., antirelapse medications) combined with medical management offers an attractive possible approach for this group, and evidence suggests that this combination yields comparable results to state-of-the-art counseling (Anton et al. 2006; O’Malley et al. 2003). Such an approach would allow most people with functional dependence to be treated in primary care and mental health care settings, similar to people with mild to moderate depression. More research, especially regarding effectiveness and implementation, is needed on this approach. Although most people with functional alcohol dependence eventually recover without any treatment (Hasin et al. 2007; Moss et al. 2007), their period of illness is associated with less severe but still significant dysfunction, such as absenteeism, attending work or school while sick (i.e., presenteeism), and reduced productivity. Early identification and treatment could reduce or hopefully eliminate these costs to the affected individuals and society.

Gaps in treatment also exist for people with severe recurrent alcohol dependence—the group that most people tend to think of when they think of “alcoholism.” A recent exhaustive report examining the current treatment system concluded that “Most of those who are providing addiction treatment are not medical professionals and are not equipped with the knowledge, skills or credentials necessary to provide the full range of evidence-based services to address addiction effectively” (p. 3) and that “Addiction treatment facilities and programs are not adequately regulated or held accountable for providing treatment consistent with medical standards and proven treatment practices.” (National Center on Addiction and Substance Abuse at Columbia University 2012, pp. 3–4). The current addiction treatment system first was conceptualized in the middle of the last century, as documented by White (2002), and has changed little since. No other chronic disease is treated with brief stints in a program with limited follow up care. Instead, for other chronic conditions patients are followed closely by physicians and other professionals over long periods of time, with the goal of minimizing symptoms and relapses, treating complications, and maximizing function. In these cases, care is provided indefinitely, often for life. Such a longitudinal-care approach also offers considerable promise in treating people with severe recurrent alcohol dependence. Several studies have found a highly significant positive effect for longitudinal care in people who have one or more medical complications of alcohol dependence (Kristenson et al. 1984; Lieber et al. 2003), including two studies that found significant reduction in 2-year mortality (Willenbring and Olsen 1999; Willenbring et al. 1995). Some findings also indicate that integrating treatment for substance use disorders into that for severe and persistent mental illness may be effective at reducing substance use, although no high-quality randomized controlled trials of this approach have been published (Drake et al. 2006). Pharmacotherapy for AUDs also may be effective in people with severe mental illnesses (Petrakis et al. 2004, 2005, 2006; Salloum et al. 2005). Finally, the ongoing need for recovery support and maintenance should be addressed.
Thus, more research is needed on the best long-term management strategies for recurrent alcohol dependence.

Conclusion

At this time no solid conclusions can be drawn as to whether current approaches to prevention of and treatment for AUDs reduce the disease burden attributable to heavy drinking, although these strategies have shown positive outcomes in the short term. SBI for at-risk drinkers in ambulatory primary care settings has the strongest evidence for efficacy, and some evidence supports its cost-effectiveness and associated reduction in excess morbidity and dysfunction. However, these benefits do not necessarily indicate that health care costs for these patients are reduced. Widespread implementation of SBI for nondependent heavy drinkers outside of the medical context has the potential to have a large public health impact. For heavy drinkers with more severe conditions (i.e., recurrent alcohol dependence), time-limited counseling may improve short-term recovery rates, but its long-term impact is less clear. Moreover, recent research findings have not been widely implemented. Scientifically based, medically anchored treatment approaches may provide a more attractive and cost-effective approach than the current intensive but time-limited treatment. More research is needed on ways to address functional alcohol dependence as well as severe and recurrent alcohol dependence.

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With growing awareness of the impact of alcohol consumption on global health (Rehm et al. 2004; World Health Organization [WHO] 2002, 2009) the demand for global information on alcohol consumption and alcohol-attributable and alcohol-related harm as well as related policy responses has increased significantly. Public health problems attributable to harmful alcohol consumption have become the focus of several World Health Assembly resolutions, including one adopted in 2005 that requested the Director-General of the WHO “to strengthen global and regional information systems through further collection and analysis of data on alcohol consumption and its health and social consequences, providing technical support to Member States and promoting research where such data are not available” (WHO 2005).

Monitoring and surveillance are crucial in setting objectives for national alcohol plans and in evaluating success (for more details see Rehm and Scafato 2011). In recognition of the increasing demand from WHO Member States for global health information, the WHO’s 11th General Programme of Work called for monitoring health situations and assessing trends as one of six core functions for the period 2006–2015 (WHO 2006). Monitoring and surveillance are crucial in setting objectives for national alcohol plans and in evaluating success (for more details see Rehm and Scafato 2011). In recognition of the increasing demand from WHO Member States for global health information, the WHO’s 11th General Programme of Work called for monitoring health situations and assessing trends as one of six core functions for the period 2006–2015 (WHO 2006). In 2010, the World Health Assembly endorsed the Global Strategy to Reduce the Harmful Use of Alcohol (WHO 2010), which targeted the monitoring and surveillance of harmful alcohol consumption and alcohol-attributable harm as one of 10 areas for action. The Global Strategy also identified production and dissemination of knowledge as one of the key components for global action (WHO 2010). Most recently, the Political Declaration of the High-level Meeting of the United Nations General Assembly on the Prevention and Control of Non-Communicable Diseases (NCDs) mandated the development of a global monitoring framework, including indicators, and a set of voluntary global targets for the prevention and control of NCDs. This mandate explicitly mentioned the harmful use of alcohol as one of the four common risk factors for NCDs along with tobacco use, unhealthy diet, and lack of physical activity (United Nations 2011). This work yielded a set of nine voluntary targets, including at least a 10 percent relative reduction in the harmful use of alcohol and a set of 25 indicators, including the following possible indicators for monitoring the harmful use of alcohol as appropriate, within the national context: (1) total (recorded and unrecorded) alcohol per capita consumption (among those ages 15 and older) within a calendar year in liters of pure alcohol; (2) age-standardized prevalence of heavy episodic drinking among adolescents and adults; and (3) alcohol-related morbidity and mortality among adolescents and adults (WHO 2012). Inclusion of the alcohol target and indicators in the global monitoring framework for NCDs and their risk factors will increase the demand for high-quality global data on alcohol consumption and alcohol-related harm and attention to the WHO monitoring activities in this area.

History of the WHO Global Monitoring System on Alcohol and Health

The WHO Program on Substance Abuse established the Global Alcohol Database in 1996, creating the world’s largest single source of information.
on levels and patterns of alcohol consumption, its health consequences, and policy responses in WHO Member States. Prior to this, WHO monitoring of alcohol and health activities largely was focused on collecting countries’ alcohol policy and prevention program data (Moser 1974, 1980, 1992). With the establishment of the Global Alcohol Database, the WHO Secretariat started to implement regular global questionnaire surveys on alcohol and health among the governmental officials of WHO Member States nominated to provide information to WHO in the areas of alcohol consumption, alcohol-related harm, and policy responses. The data collection tools were developed by WHO staff in collaboration with external experts. The first Global Status Report on Alcohol was published in 1999 (WHO 1999), followed by the Global Status Report on Alcohol and Young People (WHO 2001). In 2004, the WHO produced two global status reports based on the data collected from Member States and other sources during 2002: one on alcohol consumption and related harm (WHO 2004a) and the second focused on alcohol policy (WHO 2004b). The latest Global Status Report On Alcohol and Health contains newly developed country profiles (see figure) based on 30 key indicators related to alcohol consumption, health consequences, and policy responses (WHO 2011). The reports also provided valuable information on levels and patterns of alcohol consumption at global and regional levels, and contained estimates of alcohol-attributable disease burden. In 2006, the WHO Expert Committee on Problems Related to Alcohol Consumption recommended the establishment of a global information system on alcohol, based on the current WHO Global Alcohol Database, to continue efforts to collect, compile, and analyze alcohol monitoring and surveillance information based on comparable data and agreed definitions (WHO 2007).

The WHO created the Global Information System on Alcohol and Health.
Health (GISAH) to collect, compile, analyze, and disseminate global information on alcohol and health. From the very beginning of its development by the WHO Department of Mental Health and Substance Abuse in collaboration with the Centre for Addiction and Mental Health (CAMH) in Canada, the global information system was conceived as integrated with the regional information systems on alcohol, although at that time such a system existed only in the WHO European region. GISAH now is part of the WHO Global Health Observatory and integrates four regional information systems from countries in the Americas, Europe, Southeast Asia, and Western Pacific regions (http://www.who.int/gho/alcohol/en/index.html). The GISAH functions as one single data repository, with common data collection and data quality control procedures to prevent discrepancies between the global and regional information systems on alcohol and health.

Within GISAH, data are organized under a broad set of seven categories of indicators: levels of alcohol consumption; patterns of consumption; harms and consequences; economic aspects; alcohol control policies; prevention, research, and treatment resources; and youth and alcohol.

GISAH currently encompasses more than 150 alcohol-related indicators, with data for more than 225 countries and territories and includes indicators that are comparable across countries. The information on prevention and treatment resources is presented in another information system (i.e., Resources for the Prevention and Treatment of Substance Use Disorders) (http://www.who.int/gho/substance_abuse/en/index.html), which also is a part of the WHO Global Health Observatory.

Since its development, the GISAH and its regional components have become the central global information tool for dynamic presentation of worldwide data on levels and patterns of alcohol consumption, alcohol-attributable health and social consequences, and policy responses at all levels. The WHO Global Strategy to reduce the harmful use of alcohol explicitly mentions strengthening the GISAH and developing or refining appropriate data-collection mechanisms, based on comparable data and agreed indicators and definitions, as the key activity of the WHO Secretariat in support of WHO Member States in producing and disseminating knowledge on alcohol and health (WHO 2010).

Among the remaining key challenges for improving international comparisons of data on alcohol consumption and alcohol-attributable health consequences are the following: (1) national monitoring systems on alcohol and health in many countries are weak, fragmented or lacking; (2) difficulties exist in estimating consumption of informally and illicitly produced alcohol; (3) poor comparability of indicators used in different jurisdictions; (4) limited geographical representation of studies on the association of alcohol consumption with health outcomes; and (5) a paucity of international multi-country research projects on alcohol epidemiology using common research protocols.

**Processes and Procedures Underlying the WHO GISAH**

Data sources for the GISAH include results of the WHO Global Survey on Alcohol and Health; government documents and national statistics available in the public domain; data from the Global Burden of Disease Project; data from national and international surveys including questions on alcohol consumption and related harm from the WHO STEPS (http://www.who.int/chp/steps/instrument/en/index.html) survey instrument; and data in the public domain from economic operators in alcohol production and trade, including industry-supported organizations, published scientific articles, data from the United Nations (UN) Food and Agricultural Organization (FAO) and other UN agencies, and intergovernmental organizations such as Organization International de la Vigne et du Vin. The Canadian CAMH conducts passive surveillance of the relevant published as well as grey literature. The WHO Secretariat convenes regular meetings with key data providers on alcohol consumption to discuss and triangulate available data for achieving better estimates when national data are either unavailable or incomplete.

The WHO Global Survey on Alcohol and Health, a key data-collection mechanism, is implemented in collaboration with WHO regional and country offices, the Canadian CAMH and several other academic centers and institutions. The WHO Global Survey Instrument on Alcohol and Health, developed by WHO in collaboration with all partners involved in the survey, is forwarded to all WHO Member States through the WHO regional and country offices for completion by focal points and national counterparts explicitly nominated by governments to collaborate with WHO on this activity. For countries belonging to the European Union (EU), the survey is implemented in collaboration with and support from the European Commission. In 2008, the survey instrument contained 69 questions grouped into three sections: (1) alcohol policy; (2) alcohol consumption; and
(3) alcohol-related health indicators. The questionnaire was developed in English and translated into French, Portuguese, Russian, and Spanish. In 2008, completed questionnaires were received from 84 percent of WHO Member States, representing 97 percent of the world’s population. In 2012, 177 Member States participated in the survey, which represented a 90 percent response rate and covered 98 percent of the world population.

In 2012, the survey tool was modified to strengthen the alcohol policy section in line with the main suggested areas for national action specified in the WHO Global Strategy to reduce the harmful use of alcohol. In 2012 the survey was partially implemented using the Web-based data-collection tool.

**Alcohol Per Capita Consumption**

One of the most important indicators of alcohol consumption in the Global Survey on Alcohol and Health is per capita consumption (among those aged 15 and older) in liters of pure alcohol. Notwithstanding some limitations associated with its aggregate-level nature (Bloomfield et al. 2003), alcohol per capita consumption is a key indicator for measuring levels of alcohol exposure in populations (WHO 2000, 2007). Despite the potential measurement bias in unrecorded consumption, per capita consumption is considered the most reliable and valid indicator for alcohol consumption in a population (Gmel and Rehm 2004) and is particularly appropriate for monitoring purposes. Population-based survey data are extremely important for further estimates of alcohol consumption in different age and gender groups but currently cannot be considered as a valid and reliable basis for estimates of alcohol per capita consumption at country, regional, and global levels. Surveys are thought to underestimate per capita consumption by more than 50 percent (Midanik 1988, 1982; Rehm et al. 2007) and survey errors are larger (Shield and Rehm 2012).

The alcohol per capita consumption indicator is based on the estimates of per capita consumption of recorded and unrecorded alcohol, the latter referring to alcohol that is not taxed and is outside the usual system of governmental control, because it is produced, distributed, and sold outside formal channels and, therefore, not registered by routine data collection (Rehm et al. 2003, 2007). It is critical to include unrecorded consumption in the estimates of overall levels of alcohol exposure in populations, because more than one-fourth of global consumption stems from unrecorded alcohol (WHO 2011). However, contrary to some conjectures, unrecorded consumption does not seem to be linked to more health problems than recorded consumption, if volume and patterns of drinking are controlled for (Rehm et al. 2010). Recorded consumption can be measured via sales and taxation or via production, export, and import. Many national governments regularly monitor alcohol per capita consumption, and reliable data is available from a significant number of countries, though predominantly high-income. These national statistics, if based on validated methodology, are given highest preference in reporting in GISAH. However, even if data on alcohol consumption are unavailable from national statistics, per capita consumption can be estimated, either via industry data in the public domain, or by using data supplied from by the FAO and its statistical database (FAOSTAT) (http://faostat3.fao.org/home/index.html). An algorithm is used by the WHO Secretariat to decide which statistics to give preference to, depending on the validity of the data (see http://who.int/gho/gisah).

Unrecorded consumption obviously is harder to estimate and monitor at the country level. Only a few countries have regular monitoring of unrecorded consumption. For all others, unrecorded alcohol consumption is estimated based on one-time studies and expert opinion. For the 2012 Global Survey on Alcohol and Health an additional questionnaire component on unrecorded alcohol consumption has been developed and implemented based on the principles of the Delphi survey methodology (for a description, see Linstone and Turoff 1975; Rehm and Gadenne 1990). The questionnaire in this component covers estimates of unrecorded alcohol consumption in its major categories, such as home production (of spirits, wine, and beer), alcohol brought over the border (smuggling, duty free, and cross-border shopping), illegal production (including counterfeit alcoholic beverages), and surrogate alcohol (liquids usually containing ethanol and industrial spirits not intended for consumption as beverages). The questionnaire also addresses perceived importance of unrecorded alcohol consumption from a public health perspective as well as the measures implemented at the country level to reduce the public health impact of illicit and informally produced alcohol in line with a set of policy options and interventions listed in the Global Strategy to reduce the harmful use of alcohol (WHO 2010).

Tourist consumption also is being considered in estimating alcohol per capita consumption in populations, where tourist consumption is significant (because the number of tourists per year is at least the num-
ber of inhabitants) and is not balanced by drinking by national inhabitants abroad during vacations. This mainly is the case for smaller countries.

Alcohol per capita consumption is one example of the approximately 200 indicators monitored via the GISAH at the country, regional, and global levels.

Data Validation

Before releasing the national, regional, and global data on alcohol consumption, alcohol-related harm, and policy responses, the WHO Secretariat undertakes an intensive process of data validation by compiling country profiles with all the available data for key indicators and forwarding the country profiles to each country for validation. At this stage, any discrepancies are resolved by considering new data for the periods covered in the survey, further triangulation of available information, and building consensus around disputed qualitative indicators. After the validation process, the data are uploaded in the WHO GISAH and subsequently presented in the WHO global status reports on alcohol and health.

Further Developments

Both the depth of the GISAH and the rigor of data collection and validation make it an indispensable tool for policy development and evaluation, as well as for global research (e.g., the Global Burden of Disease 2010 estimates were based on WHO global monitoring [Lim et al. 2012; Shield et al. 2013]). One of the key tasks for the WHO in the area of global monitoring and surveillance on alcohol and health is to support the development of effective national systems for monitoring alcohol consumption, its health and social consequences and related policy responses, while also strengthening national capacity for analyzing and disseminating the information, also through the WHO’s global and regional information systems on alcohol and health. To further improve comparability of data generated in countries, consistent data collection mechanisms, agreed indicators and definitions, and enhanced dissemination of data is needed. The new alcohol module in the WHO STEPS instrument (http://www.who.int/chp/steps/instrument/en/index.html), which is the main data collection tool used in WHO surveillance activities on risk factors for chronic diseases, is an attempt to prioritize some well-defined key indicators and improve consistency between the relevant national surveillance activities and the WHO global monitoring system on alcohol and health. Work continues on WHO tools for alcohol epidemiology and monitoring, such as the International Guide for Monitoring Alcohol Consumption and Related Harm (WHO 2000).

One of the challenges for the WHO global monitoring system on alcohol and health continues to be a time lag between the alcohol exposure data collected from countries and their dissemination through GISAH and WHO global and regional status reports on alcohol and health. Efforts to reduce this time lag will involve data collection through Web-based data collection tools, optimizing data validation and dissemination procedures, as well as strengthening partnerships and resource mobilization for effective functioning of the global monitoring system.

The ultimate objective for the WHO global monitoring system on alcohol and health is strengthening the link between monitoring activities and policy development and evaluation. This system, which includes the global surveys, GISAH, and WHO global status reports on alcohol and health, is the central mechanism for monitoring implementation of the WHO global strategy to reduce the harmful use of alcohol and report on its implementation to WHO Member States (WHO 2013), other constituencies, and the public health community at large.

Note: The views expressed in this article are those of the authors and, except as specifically noted, do not represent the official policies or positions of the WHO.

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Measuring the Burden—Current and Future Research Trends

Results From the NIAAA Expert Panel on Alcohol and Chronic Disease Epidemiology

Rosalind A. Breslow, Ph.D., M.P.H., R.D., and Kenneth J. Mukamal, M.D.

Alcohol has a significant impact on health and well-being, from the beneficial aspects of moderate drinking to the detrimental effects of alcoholism. The broad implications of alcohol use on public health have been addressed through a wide range of epidemiological and clinical studies, many of which are described in this issue of Alcohol Research: Current Reviews. Where chronic disease is involved, alcohol use can be a risk factor that not only affects the onset of various chronic diseases but also exacerbates the ongoing extent and severity of those diseases. Lifestyle choices and genetic influences also contribute to, or help to alleviate, that risk. Key Words: NIAAA Expert Panel on Alcohol and Chronic Disease Epidemiology; alcohol consumption; alcohol burden; chronic disease; risk factors; epidemiology; research; diabetes; cardiovascular disease; cancer; stroke; liver disease; genetic factors; eating behaviors; clinical trials

Research is continuing to investigate how alcohol impacts chronic disease. The National Institute on Alcohol Abuse and Alcoholism (NIAAA) hosted a 2-day Expert Panel on Alcohol and Chronic Disease Epidemiology in August 2011 to review the state of the field on alcohol and chronic disease. The panel was chaired by Kenneth J. Mukamal, M.D., and Rosalind A. Breslow, Ph.D., M.P.H., R.D., and was convened by NIAAA’s Division of Epidemiology and Prevention Research.

Panel members (see textbox) represented a wide range of backgrounds and expertise, ranging from alcohol-related chronic diseases and risk factors to methods and technology. Among the chronic diseases addressed were diabetes, cardiovascular disease, cancer, stroke, and liver disease. The broader aspects of the design and implementation of clinical trials and the implication of technological advances for research also were considered. Other topics included the links between genetics and other lifestyle factors, such as eating behavior, and the relationship between drinking and various chronic diseases. Taken together, these summaries provide unique insight into the current state of research on alcohol’s role in chronic disease and the direction these investigations may take in the future. (For more information on the epidemiological challenges of elucidating the effects of alcohol consumption and drinking as they relate to the initiation/exacerbation and treatment of chronic diseases, see the article by Shield and colleagues [pp. 155–173]). Panel members also were asked what research they would most strongly support if funds were unlimited and how they might scale back that research if funding were limited (see Future Ideas textbox). Highlights from this panel are presented below and specific recommendations are listed in the accompanying sidebar.

Clinical Trials

Clinical studies include clinical nutrition studies, controlled feeding studies, and metabolic studies. This type of research has numerous strengths for studying alcohol and chronic disease, including the ability to control alcohol dose and diet, collect abundant biologic samples from a variety of tissues, assess cause and effect, and examine mechanisms—all with a relatively small number of participants enrolled for a short period of time.

Clinical study end points typically are surrogate markers for chronic diseases because the disease itself may take years or even decades to develop. For example, lipoproteins and markers of inflammation have been used as surrogates for cardiovascular disease, insulin sensitivity for diabetes, and DNA damage for cancer.

According to Dr. David J. Baer, considerable need for controlled clinical studies on alcohol and chronic disease still exists. There have been few clinical studies, even on cardiovascular disease (Brien et al. 2011), which is the focus of most alcohol-related chronic disease research. He also noted the relatively few controlled clinical studies of alcohol and obesity (Sayon-Orea et al. 2011) that were advocated by the

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Kenneth J. Mukamal, M.D., is associate professor of medicine, Harvard Medical School, Division of General Medicine & Primary Care, Beth Israel Deaconess Medical Center, Boston, Massachusetts.
Dr. Baer suggested the following future opportunities for alcohol and chronic disease research:

- Drinking patterns;
- Effects on metabolism and disease risk;
- Non-ethanol components of alcoholic beverages;
- Possible effects on cardiovascular disease, diabetes (insulin sensitivity), cancer, and bone metabolism;
- Gender and age differences (pre- and postmenopausal women, men);
- Genetic basis for response of chronic disease surrogate markers to alcohol;
- Energy metabolism, body weight regulation, and insulin sensitivity;
- Interaction of alcohol with lower-fat or higher-protein diets; and
- Bone metabolism.

**Cardiovascular Disease**

Studies on alcohol and cardiovascular disease have yielded important findings with regard to public health. For example, we now know that the association of alcohol use within recommended limits with lower risk of heart disease depends more on the frequency with which alcohol is consumed and not on the type (Cleophas 1999). Wine, beer, and spirits all have been associated with reduced risk of myocardial infarction. Modest differences in the effects of those different types of alcohol are thought to be more a result of lifestyle differences among drinkers rather than a direct link to a specific type of alcohol. How often people drink alcohol has a larger impact on cardiovascular disease. Among men, drinking more frequently seems to have a greater impact than the actual amount consumed (Mukamal et al. 2003); effects are less clear among women. The beneficial effects of alcohol also have been shown to be similar for people with existing cardiovascular disease or diabetes (Costanzo et al. 2010; Koppes et al. 2006) and those in the general population. In addition to its beneficial effects on coronary heart disease, moderate drinking has been found to reduce the risk of ischemic stroke but at a lesser magnitude and with lower levels of consumption (Klatsky et al. 2001).

Although the exact mechanisms involved in these cardioprotective effects still are under investigation, the putative benefits on cardiovascular disease likely are the result of alcohol's effects on lipids and insulin sensitivity (Dijousse et al. 2009).

In his presentation, Dr. Kenneth J. Mukamal noted that standard epidemiologic studies of alcohol consumption and coronary heart disease incidence or mortality are no longer useful, as virtually all prospective studies performed since 1980 have shown that moderate drinking reduces risk (Corrao et al. 2000; Mukamal et al. 2010; Ronksley et al. 2011). Recent analytic strategies have resulted in more precise statistical estimates, but the conclusion is unchanged. In essence, he stated, “We’ve been doing the same epidemiology since 1992.”

Dr. Mukamal suggested the following future opportunities for alcohol and cardiovascular disease research:

- Effects of heavy and binge drinking;
- Effects of changes in alcohol consumption over time;
- Differences in effect of gender-specific drinking patterns;
- Genetic interactions;
- Studies of new mechanisms directly related to alcohol's effects (for example, cholesterol efflux capacity) (Khera et al. 2011);
- Pooling projects for questions that require large samples; and
- Use of case crossover designs to account for both triggering events and chronic use (Mostofsky 2011).

**Cancer**

Alcohol consumption increases the risk for several cancers, including breast, colon, liver, and upper aero-digestive cancers (oral, pharynx, larynx, and esophagus) (Schutze et al. 2011; World Cancer Research Fund 2007). The potential mechanisms underlying alcohol's effects include the carcinogenicity of acetaldehyde (for colorectal cancer and upper aero-digestive tract cancers), which is an intermediate product of alcohol metabolism; impairment of the one-carbon nutrient metabolism (for colorectal cancer); alteration of hormone levels (for breast cancer); and oxidative stress resulting from alcohol metabolism.

Dr. Edward Giovannucci noted the paucity of research on drinking patterns and cancer. He acknowledged too that studies can yield disparate findings, describing a study that initially showed no relationship between average alcohol consumption and prostate cancer but which in a posteriori analyses hinted at a possible relationship with high-quantity/low-frequency drinking (Platz et al. 2004).
In identifying areas for future research, Dr. Giovannucci discussed the importance of studying cancer–nutrient interactions, particularly for colon cancer. For example, the epidemiologic literature has consistently shown an interaction between alcohol and folate, a nutrient that seems to be protective at higher levels of drinking (Ferrari et al. 2007; Jiang et al. 2003). This suggests that the excess risk of cancer resulting from alcohol use potentially could be modified by a nutrient or combination of nutrients.

Further study also is needed to better understand the role of genetics and family history in cancer risk. The genes involved in alcohol metabolism (Yokoyama et al. 2001) and nutrient metabolism (for example, the gene methylenetetrahydrofolate reductase [MTHFR] for folate as well as other genes involved in the one-carbon metabolism pathway) are other areas that warrant additional study. Determining the molecular characteristics of tumors, such as tumor subtypes classified by level of methylation, which might reflect defects in one-carbon metabolism (Scherhammer et al. 2010), is another area that requires further investigation. In addition, little research has been conducted with cancer survivors, a group that may be especially willing to modify their drinking habits.

Finally, as noted by Dr. Giovannucci, alcohol increases the risk for many cancers, but not all. Recent studies have found that alcohol is associated with a lower risk of kidney cancer (Lee et al. 2007) and non-Hodgkins lymphoma.

### Future Research Ideas, Large and Small, for Consideration

In addition to the full panel discussions, panelists were asked to consider directions for future studies—both large and small. Specifically, the panelists described what studies they would suggest for future research and how they would refine those visions when funds are limited. Selected noteworthy examples are described below.

- A randomized trial to evaluate alcohol consumption and risk of multiple clinical outcomes with sufficient power to evaluate prespecified genetic environmental interactions would be ideal. However, with limited resources, it might be more realistic to use a hybrid design, with a prospective cohort study and a smaller nested trial. For example, a trial might evaluate if recommending moderate alcohol consumption, versus no recommendation, had an effect on cardiovascular and stroke outcomes among patients with a high risk for vascular problems.

- Clinical trials to establish the effects of alcohol consumption on clinical cardiovascular and cancer outcomes. A large-scale trial using high-risk populations with standardized exposure to alcohol would be ideal. A more practical approach would be to conduct shorter trials with subclinical measures of both cardiovascular disease and, to a lesser degree, cancer, using such techniques as serial computed tomography angiography and colonography.

- Studies to identify factors that influence the risk for liver disease among moderate drinkers. A large, prospective study would be ideal and would include serial measures of genomic, dietary, anthropometric, and behavioral risk factors obtained as objectively as possible, coupled with serial noninvasive measures of liver disease using magnetic resonance imaging for fat and fibroscan for fibrosis. Such a cohort could additionally fold in cardiovascular disease risk factors and clinical and subclinical cardiovascular disease. Among other things, this study would help to address the simultaneous associations of alcohol consumption with lower risk of cardiovascular disease but higher risk of fatty liver, which is associated with a higher risk for cardiovascular disease. Although of more limited utility, a cross-sectional study with the same measures would also be of clear import.

- Studies to verify estimates of drinking patterns. This is particularly important as self-reported estimates form the basis for epidemiological studies but have yet to be validated, particularly in the context of eating patterns, portion sizes, and health beliefs.

- Studies of how alcohol ingestion impacts energy balance in both moderate and binge drinkers.

- Studies to better understand the risk factors underlying alcohol-related chronic disease. These factors range from fixed characteristics, such as genetics and ethnic background, to broader modifiable behaviors, such as diet, exercise, or smoking. An ideal study would be multifaceted and include both disease-specific and composite global endpoints, such as healthy aging or survival free of chronic disease. A more limited study could simply compile data from the dozens of cohort studies worldwide where much of this data already have been collected. A more comprehensive effort would use ongoing studies prospectively to incorporate novel measures of drinking patterns, biomarkers of health status, or greater assessment of quality of life and mental health.
(Kroll et al. 2012). Understanding how these two cancers differ from others is another area requiring additional research.

Dr. Giovannucci suggested the following future opportunities for alcohol and cancer research:

- Effects of drinking patterns on cancer risk;
- Nutrient interactions;
- Genetic susceptibility (genes related to alcohol metabolism, genes related to one-carbon metabolism);
- Tumor subtypes;
- Cancer survivors; and
- Pathways that might explain the limited protective aspects of alcohol consumption.

**Diabetes**

Evidence that alcohol can impact diabetes has been consistent over several studies. Results from the Nurses’ Health Study (Stampfer et al. 1988), the Health Professionals Follow-up Study (Conigrave et al. 2001), a systematic review (Howard et al. 2004), and two meta-analyses (Baliunas et al. 2009; Koppe et al. 2005) all show that moderate drinking is associated with a lower risk of diabetes. Heavy drinking, on the other hand, seems to lead to an increased risk of diabetes, although sample sizes generally have been too small to draw firm conclusions.

Dr. Eric Rimm described specific areas of research that warrant further study. For example, only about 30 to 50 percent of alcohol's beneficial effects on diabetes can be linked to biomarkers studied to date. In addition to its overall effect on insulin sensitivity (Davies et al. 2002), moderate alcohol consumption improves adiponectin, a fat-tissue hormone associated with insulin sensitivity; inflammatory status (Joosten et al. 2008); and HDL cholesterol. With regard to metabolic studies, he noted the value of using short-term feeding studies because they provide an opportunity to control and simultaneously examine drinking (for example, with meals or without) and diet (for example, high versus low glycemic load) (Mekary et al. 2011). He also discussed the importance of studying genetic predisposition (Beulens et al. 2007).

In addition to these areas, Dr. Rimm suggested several future opportunities for alcohol and type 2 diabetes research:

- Pool data from large cohort studies with genetic information on alcohol metabolizing and diabetes-related genes to examine the interactions between alcohol, genetic predisposition, and diabetes risk.
- Conduct metabolic studies specifically within subgroups to examine how alcohol modifies risk based on lifestyle characteristics, such as body mass index, diet, and physical activity.

**Stroke and Cognition**

Several important findings on the effects of alcohol consumption on the incidence of stroke have emerged from the Northern Manhattan Study, a prospective, multiethnic cohort study (Elkind et al. 2006; Sacco et al. 1999). In that study, subjects with the lowest risk for ischemic stroke consumed, on average, two drinks per day. Those effects were similar among drinkers of wine, beer, and liquor. In contrast, no protective effect was found for hemorrhagic stroke.

The study’s principal investigator, Dr. Ralph Sacco, presented the results of two meta-analyses. One found the greatest protection against all strokes combined was most evident at a lower level of drinking, less than or equal to one drink per day (Ronksley et al. 2011). Other analyses compared results from ischemic with hemorrhagic strokes (Reynolds et al. 2003). For ischemic stroke, moderate drinking was protective, whereas heavy drinking was associated with an increased risk; for hemorrhagic stroke, heavy drinking increased risk (although sample size was insufficient to study the effects of moderate drinking on hemorrhagic stroke).

The heterogeneity of strokes underscores the importance of studying stroke subtypes. Both ischemic strokes (the majority of all strokes) and hemorrhagic strokes (about 17 percent of all strokes) have subtypes with differing etiologies that may respond differently to alcohol consumption. Little research has been conducted on these subtypes, partly because of the small numbers of each that occur within most studies and the need for relatively large samples to obtain sufficiently precise estimates of risk. Numerous subclinical markers of stroke, such as endothelial function, currently are being pursued by researchers (Suzuki et al. 2009).

**Cognition**

The prevalence of cognitive impairment is growing rapidly as the population ages, and, like stroke, cognitive impairment is not a single disease or condition. Studies of alcohol use and cognition have examined a variety of outcomes, including Alzheimer’s disease, cognitive function, dementia, and mild cognitive impairment (Lee et al. 2010). Studies and meta-analyses generally show that moderate drinking is associated with a decreased risk of dementia (Mukamal et al. 2003b; Peters et al. 2008), Alzheimer’s disease (Peters et al. 2008), vascular dementia (Peters et al. 2008), and cognitive...
n addition to offering ideas for future studies, the
Expert Panel also made recommendations for strength-
ing research in the field. Specific suggestions include:

1. Standardize alcohol consumption measurement in
    prospective and retrospective studies of alcohol and
    chronic disease to the greatest degree possible.
    Standardized measures:
    a. Should include consumption quantity, frequency,
       and binge drinking (i.e., basic drinking patterns).
    b. Should consider drinking over the lifespan (for
       example, during youth, middle age, menopause,
       and during time of heaviest drinking) as the criti-
       cal time periods for effects of alcohol on chronic
       disease development are uncertain.
    c. Are available from NIAAA and from the
       NIH/National Human Genome Research Institute
       Research Resources/Pages/TaskForce.aspx;

2. Strongly encourage collection of biological material
    and broad consent for genetic studies in all clinical
    trials and in as many population studies as possible.

3. Objectively validate standardized alcohol measures
    using novel technologies as they become available.
    Examples may include implantable biosensors and
    point-of-care devices with wireless transmission capability.

4. Develop new biomarkers for moderate alcohol con-
    sumption to complement those used for heavy drinking.

5. Identify surrogate markers for chronic disease
    (including measures of subclinical disease) that will
    have utility in small-scale studies and for elucidating
    mechanisms and pathways linking alcohol to chronic
    disease.

6. Pool data from existing cohort studies to facilitate
    examination by population subgroups, including
    but not limited to age, lifespan phase, race/ethnicity,
    menopausal status, body mass index/anthropomet-
    rics, dietary intake/nutritional status, smoking status,
    physical activity/fitness, cancer survivorship, and age
    of drinking onset. Pooled data also may facilitate
    studies of rare or understudied outcomes such as
    liver disease.
    a. Standardized alcohol questions should be used
       where possible.
    b. Confounding and interaction should be consid-
       ered to ensure robust estimates and define suscep-
       tible subgroups.
    c. Targeted sub-studies within large cohorts should
       be considered as a cost-efficient way to better
       understand and explain results in the full cohort.
       For example, when data on alcohol consumption
       are not gathered in enough detail in the original
       study, targeted follow-up studies may be used among
       stratified subsets of subjects to collect biological
       samples and to obtain more detailed data on con-
       sumption for extrapolating to the parent study.

7. Include associations between alcohol dependence/
    abuse and chronic disease outcomes. Studies using
    pooled data or sub-studies within large cohorts may
    have the power to address these drinking problems.
    Data on period of maximum drinking could be
    important, particularly given the marked variation
    in alcohol intake during the lifespan.

8. Perform studies in understudied areas, including
    but not limited to the effects of alcohol on diabetes,
    obesity, cognition, healthy aging, and food intake.

9. Focus on relationships between drinking patterns
    and chronic disease. Drinking patterns include but
    are not limited to basic patterns such as usual quan-
    tity, frequency, and binge drinking as well as when,
    where, and with whom alcohol was consumed and
    whether it was consumed with a meal.

10. Encourage clinical trials across the spectrum of
    chronic disease from studies that examine key physi-
    ological parameters and intermediate studies such as
    feeding studies that examine surrogates or subclinical
phenotypes to practical trials that examine chronic disease outcomes.

a. Physiologic studies are preferred when epidemiologic evidence is relatively limited.

b. Practical trials are preferred when there is extensive evidence from physiological and epidemiological studies.

11. Encourage studies examining the interactions between the genetics that predispose individuals to drink and the genetics that modify how alcohol affects chronic disease.

12. Encourage studies of carefully defined homogeneous phenotypes. For example, studies are needed to clarify the effects of alcohol on thrombotic versus embolic ischemic stroke, Alzheimer’s disease versus other dementias, specific eye diseases, etc.

13. Encourage studies on moderate drinking patterns and metabolism ranging from total energy and macronutrient metabolism to specific metabolic pathways for small molecules such as vitamins, amino acids, sugars, and steroids and their products and precursors.

14. Examine the effectiveness of communication messages about drinking. Studies may include, but are not limited to, how to disseminate cost-benefit messages, individualized messages based on patient demographic and clinical history, and guidance for health care professionals on how to advise patients.

15. Encourage the use of natural experiments to examine whether policy interventions or alcohol intervention studies might change the relationship between alcohol and chronic disease.

decline (Peters et al. 2008). According to Dr. Sacco, there currently is great interest in vascular risk factors for dementia, yet little alcohol research has been done in that area. Other future opportunities for research into alcohol and chronic neurological disease noted by Dr. Sacco include the following:

- Cohort studies with careful end point adjudication to separate ischemic stroke subtypes and different etiologies of dementia and cognitive impairment;

- Examination of interactions with race and ethnicity and other neurological risk factors;

- Comparison of associations across beverage types for neurological outcomes; and

- Understanding protective alcohol mechanisms including inflammatory relationships, subclinical measures and biomarkers, and gene–environment interactions.

Chronic Liver Disease

Chronic liver disease has long been associated with alcohol consumption and includes alcoholic liver disease, hepatitis C, and nonalcoholic steatohepatitis. Despite this clear association, however, there is a lack of strong clinical measures to describe and predict the progression of chronic liver disease. Dr. James Everhart noted that the course of alcoholic liver disease is several decades in duration and begins as simple steatosis (fatty liver) before progressing to more advanced stages including steatohepatitis, alcoholic cirrhosis, and, eventually, liver failure. Dr. Everhart noted that alcoholic liver disease may be overrepresented in terms of mortality because of the current classification system. Histologically, alcoholic fatty liver and nonalcoholic fatty liver look similar (Scaglioni et al. 2011), and patients with otherwise similar multiple risk factors and histology may be classified as having alcoholic liver disease rather than nonalcoholic steatohepatitis simply because they do or do not drink. According to Dr. Everhart, the current strict separation of alcoholic and nonalcoholic fatty liver disease limits epidemiology, public health, and clinical understanding.

In examining the effects of drinking amounts on liver disease, little association has been found between moderate drinking and alcoholic liver disease, and only a minority of very heavy drinkers develops alcoholic liver disease, although the reason is not clear. It is possible that drinking patterns and diet each play a role in risk. More information also is needed to determine if drinking at times other than during meals could increase risk.

Other factors that put people at higher risk for liver disease include being obese, using cannabis, having diabetes,
and being female (Hart et al. 2010). Conversely, coffee consumption seems to lower risk and smoking seems to have no effect on the development of chronic liver disease. Genetic susceptibility is another important risk factor for liver disease. For example, a variant in one gene, *PNPLA3*, originally associated with fatty liver, has been strongly associated with alcoholic liver disease. Again, additional research is needed to determine how these factors influence alcohol’s effects.

Dr. Everhart suggested several future opportunities for alcohol and chronic liver disease research:

- Improve the current chronic liver disease classification scheme;
- Develop reliable and accurate measures of progressive liver disease that can be applied serially;
- Implement better measures of alcohol consumption and its patterns to study drinking patterns and interactions between drinking and diet;
- Evaluate how genetics may influence the link between alcohol consumption and the risk of liver disease; and
- Identify determinants of chronic liver disease among heavy drinkers.

**Genetics**

Chronic diseases tend to run in families yet do not follow a simple genetic pattern; that is, they are complex and polygenic. Identifying the genes that affect chronic disease risk can be hampered by multiple factors, including phenotypic complexity, multiple genes with small effects, environmental variability, gene–gene interactions, and gene–environment interactions. Alcohol’s role in chronic disease likely reflects a gene–environment interaction in which risk is influenced by genes, by lifestyle choices, and by a combination of both. In addition, as noted by Dr. Howard J. Edenberg, most of the variations in genes related to alcohol and chronic disease likely have only small effects, making those genetic influences especially difficult to identify.

One way of overcoming these difficulties, as proposed by Dr. Edenberg, is to obtain large sample sizes by combining data from multiple epidemiologic studies. This enables investigators to examine gene–environmental associations using secondary data analyses. The drawback is that studies typically ask different questions about alcohol use and often include different time frames, often collect no data on drinking problems, and may not obtain appropriate consent for genetic testing. Dr. Edenberg suggested a number of strategies to manage these obstacles. For example, investigators could be encouraged to incorporate standardized alcohol consumption questions, particularly for patterns of consumption, and to obtain DNA samples using proper consent for genetic studies, where appropriate. Existing studies also could be enhanced through targeted ancillary studies in which key subsets of subjects are re-contacted to provide more detailed or standardized information. The payoffs from such steps could lead to the discovery of key genes and pathways that reveal mechanisms and potential targets for therapy. Even if the effect of a variant is small, the pathway it leads to could be of major importance.

Dr. Edenberg suggested several future opportunities for the genetics of alcohol and chronic disease research:

- Design and incorporate more detailed alcohol exposure measures that include patterns of consumption and drinking problems;
- Search out ongoing and planned studies to;
  - Partner to incorporate exposure measures as early as possible;
  - Target follow-up and additional studies to gather more detailed exposure information and genetic samples; and
  - Encourage collection of samples with consent for genetic studies.

**Eating Behaviors**

The link between alcohol intake and eating behaviors is not well known. Studies generally show that alcohol calories, when added to the diet, increase total energy intake (Yeomans 2010). Yet despite the fact that alcohol is an energy source, is largely uncompensated (i.e., supplements rather than replaces other calories), may weaken feeding controls, and spares fat for storage, little evidence exists that moderate drinking is associated with increased body mass index or weight gain (Liangpunsakul 2010; Liu et al. 1994; Wang et al. 2010) (although a French study did show such an effect [Lukasiewicz et al. 2005]). On the other hand, certain drinking patterns, particularly binge drinking, have been associated with higher body mass index (Arif and Rohrer 2005; Breslow and Smothers 2005), although impulsivity related to both eating and drinking could be an alternative explanation. According to Dr. Richard Mattes, determining alcohol’s effects on eating behaviors is further confounded by beverage consumption itself and the fact that energy compensation for fluids is less than for semisolid or solid foods (Mattes 1996; Mourao et al. 2007).

He also suggested that what people think they are eating may be more important in terms of appetitive sensations than its true energy value, noting current research showing that manipulating food form (liquid or solid) can alter a person’s expectation of how filling that food will be.

Dr. Mattes suggested several research opportunities for future studies on ingestive behavior and alcohol-related
chronic disease research, particularly in controlled experimental designs:

• Clarify the role of moderate alcohol consumption on energy balance;
• Assess which properties of alcohol contribute to hunger and satiety;
• Ascertain the true biological energy value of alcohol;
• Test the role of drinking patterns on energy balance; and
• Determine the effects of different levels of alcohol consumption on body composition and energy balance.

Technology

A number of promising technologies and medical devices currently are under development by the National Institute of Biomedical Imaging and Bioengineering and others that may enhance alcohol-related chronic disease research in the future. Dr. John Haller reviewed the research on three areas: sensors, point-of-care (POC) diagnostic devices, and imaging technologies and bioinformatics tools.

Sensors are used to detect and quantitate clinically relevant analytes. Examples include BioMEMs, microfluidics (Chin et al. 2011), and nanoscale technologies, including micro-total analysis systems, arrays, and biochips. These multifunctional devices can measure multiple analytes across a variety of diseases using a platform the size of a credit card. Such technologies then can be combined into POC tests, which are defined as diagnostic testing at or near the site of patient care (rather than at centralized laboratories). Benefits include earlier diagnosis of disease and the ability to monitor patients at home. For example, POC tests for alcohol include a breath test and saliva-testing devices (http://www.aacc.org/events/online_progs/documents/AlcoholTesting1.2.pdf); SpectRx, a wristwatch-type device; and Giner, a WrisTas transdermal sensor for measuring alcohol consumption (Marques and McKnight 2009). Dr. Haller also reviewed implantable monitors and a tattoo using nanosensors that reside under the skin. By shining a light on the tattoo the subject enables tracking of sodium and glucose levels by portable digital devices, including smartphones. In the future, such a technology could be used to track alcohol consumption.

Biomedical imaging of the brain is another area where advances could be applied to the study of alcohol and chronic disease. Most radiology images (e.g., magnetic resonance imaging [MRI], computerized tomography) show anatomy/morphology. These images generally capture the late stages of chronic disease. An alternative approach would be to examine the physiological function (e.g., neuroreceptors) using nuclear imaging (e.g., positron emission tomography and single-photon emission computed tomography). Magnetic resonance spectroscopy can image relative chemical composition. MRI diffusion tensor imaging can image white matter tracts (connectivity), and functional MRI can image relative blood flow, a marker of neural activity. These structural and functional neuroimaging methods currently are being used in alcohol research (Buhler and Mann 2011). Dr. Haller noted that informatics (data modeling, simulation, and analysis) also will have a significant role in making sense of the large amounts of high-dimension data now available.

Dr. Haller had the following suggestions regarding alcohol-related chronic disease research:

• Among the variety of technologies and medical devices that exist for the study of individuals and populations, those of particular interest might include sensors, POC diagnostic devices, imaging technologies, and bioinformatics tools;
• A better alternative to the “hammer-in-search-of-a-nail” approach in imaging is to define the clinical problem of interest first, then find the appropriate tools to address the problem or chronic disease under study;
• Alcohol and chronic disease epidemiology could be improved through the use of new sensors (including POC diagnostics, sensors embedded in the home or implanted in the body) to enhance alcohol measurement and by techniques that can image physiological function early in the course of chronic disease; and
• Technological advances will inevitably produce vast amounts of data about individuals and populations, but they require new informatics tools that enable meaningful use of the data in wide varieties of research settings.

Summary

This NIAAA workshop provided an excellent forum for summarizing the current state of the field and for identifying future research opportunities. Although by no means exhaustive, the ideas provided here highlight areas in need of additional study and offer a roadmap for moving forward across a variety of methodological approaches and content areas. NIAAA would like to thank all of the presenters for their insight and for taking the time to participate in this unique workshop. Our hope is that the ideas presented here will stimulate additional research and further advance our understanding of the role of alcohol in chronic disease.

Additional Resources

The agenda, roster of speakers, and speaker’s abstracts can be obtained from the author. A copy of the meeting transcript also is available from the author, upon request.
Financial Disclosure

The authors declare that they have no competing financial interests.

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