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# Hospital-Admitted Injury Attributable to Alcohol

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# Abstract

**Background**—Primary data collection has established that alcohol causes injuries treated in the emergency department. No comparable data exist for injuries admitted to hospital. Data on the injury risks of heavy drinkers relative to other drinkers also are sparse.

**Methods**—We estimated (1) whether regular heavy drinkers have higher hospitalized injury risks than other people when alcohol negative and (2) how much hospitalized injury risk of regular heavy drinkers and other drinkers rises when alcohol positive. We combined national alcohol consumption data with alcohol metabolism rates to estimate hours spent alcohol positive versus alcohol negative during a year for heavy drinkers versus other people. A literature review provided hospitalized non-fatal injury rates for these groups by alcohol involvement.

**Results**—Relative to other alcohol-negative people aged 18 and older, heavy drinkers have an estimated relative risk of hospitalized injury of 1.4 when alcohol negative and 4.3 when alcohol positive. Others have an estimated relative risk of 1.0 when alcohol negative and 6.8 when alcohol positive. Thus alcohol greatly raises injury risk. The excess risk patterns persist for a wide range of sensitivity analysis values. Of hospitalized injuries, an estimated 21% are alcohol attributable including 36% of assaults.

**Conclusions**—Drinking alcohol is a major cause of hospitalized injury. Heavy drinkers lead risky lifestyles. They tolerate alcohol better than most drinkers but their injury risks still triple when they drink. Our approach to attribution is a valuable complement to more costly, more precise approaches that rely heavily on primary data collection. It works for any severity of injury. Applying it only requires an existing alcohol consumption survey plus data on alcohol involvement in targeted injuries.

# Keywords

heavy drinker; burn; spinal cord; assault; cause

# Introduction

Hundreds of studies document the frequent involvement of alcohol in fatal and nonfatal injury (National Institute on Alcohol Abuse and Alcoholism, 1997; Lipsey et al., 1996; Roizen, 1988; Smith et al., 1999a; Smith et al., 1999b). These studies show that people often are injured while drinking and that heavy drinkers have above-average injury risks. Several studies document injury victims' self-reports that alcohol consumption caused injury (Cherpitel et al., 2006; Sommers et al., 2000; Stephens, 1987). Consistent with those reports, laboratory studies show that alcohol impairs coordination and the ability to perceive and respond to hazards (Normand et al., 1994) and that hangover impairs neurocognotive

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performance and pyschomotor vigilance (Howland et al., 2010; Prat et al., 2008; Rohsenow et al., 2010). It is difficult, however, to assess the impact on injury risk in the uncontrolled setting of real life (Hingson et al., 2001; Morrow et al., 1991; Tornros and Laurell, 1991; Wolkenberg et al., 1975; Yesavage and Leirer, 1986).

Early epidemiologic studies estimated how likely a group of people classified by drinking behavior were to suffer injuries relative to a base group. These studies demonstrated linkage only weakly (Heien and Pittman, 1989; Lipsey et al., 1996). They cannot tell us whether the excess injury risk associated with alcohol results from alcohol impairment or from unmeasured factors that differentiate the groups being compared. One factor is a lifestyle that includes risk-taking and heavy drinking. These studies established correlation, not causation. They included injuries that involved alcohol but would have occurred even if the victim were alcohol negative. For example, a drunk driver rear-ended by an alcohol-negative driver while stopped at a red traffic light suffers an alcohol-involved injury unrelated to alcohol. So does a sleeping drunk whose home collapses in an earthquake.

Epidemiologists estimate attributable risk ratios to reduce the effects of such extraneous events (Rothman and Greenland, 2005). Except for impaired driving (Borkenstein et al., 1974; Lloyd, 1992), only recently have studies shown injury risk rises while drinking. Some studies use case-crossover designs. These studies ask injured people in emergency departments (EDs) about their drinking a week prior to and at the time of their injury (Borges et al., 2004a, 2006, 2008; Pledger et al., 2007; Vinson et al., 1995, 2003). An overlapping group of studies use case-control designs with people visiting the ED for reasons other than injury serving as a comparison group (Cherpitel et al., 2005; Kuendig et al., 2008). A third group are case-control studies with a community comparison group (Borges et al., 1998; McLeod et al., 1999; Stockwell et al., 2002; Vinson et al., 2003; Watt et al., 2004). Some studies use alcohol tests in the ED to replace or supplement self-report. Here we introduce a less accurate but also far less expensive approach; we construct the comparison group from existing consumption surveys. The method is especially useful in analyzing attribution for severe injuries that result in death or hospital admission because interviewing the severely injured can be impractical (e.g., those with brain injury, quadriplegia, or a memory gap around the injury incident). It also yields insight into attribution of criminal acts where self-report by apprehended criminals and impressions of victims describes alcohol involvement but comparison with other time periods or with contemporaneous alcohol use by similar people can be difficult.

Some studies assess if injury risk is higher for those who drink or drink heavily. They confound injuries caused by alcohol with injuries caused by lifestyle and risk-taking differences between groups whose drinking patterns differ. Studying whether alcohol causes injury requires differentiating injuries during alcohol-positive and alcohol-negative hours.

This study probes the roles of alcohol impairment and lifestyle in the risk of injury. We test three hypotheses: (1) the hospitalized injury risk of both regular heavy drinkers and other drinkers rises when they are alcohol positive (i.e., alcohol appears to cause injury); (2) regular heavy drinkers have higher hospitalized injury risks than other people when alcohol negative (i.e., heavy drinker lifestyle is riskier and/or hangover is risky); and (3) being alcohol positive raises the hospitalized injury risk of people who are not heavy drinkers more than it raises the risk of heavy drinkers (i.e., regular heavy drinkers tolerate alcohol better than most drinkers).

# Methods

The innovative seed for our analysis is simple; dividing existing data on alcohol consumption by data on alcohol metabolism rates yields less costly exposure estimates than breath testing. Once exposure is known, estimating the risk ratios simply requires writing some standard epidemiologic formulas related to relative risk (Szklo and Nieto, 2000). A literature search and some secondary data analyses yield the data for the ratios. Annual hours alcohol positive versus negative are used as the exposure measures. Risk attributable to alcohol is measured by estimated relative risk when alcohol positive versus negative.

The equations used to meet the objectives appear in Table 1. They are developed below. Table 2 lists the best estimates and sensitivity analysis values for the equations. We adjusted the data to achieve better consistency in age range and definition of regular heavy drinkers. Sensitivity analysis assessed how value choices influenced the results. We used wide ranges when available data were weak or inconsistent or values varied over time.

# Relative Risk of Hospitalized Injury when Alcohol Positive versus Alcohol Negative (Equation 1)

The definition of relative risk (Szklo and Nieto, 2000) dictates that the risk of hospitalized injury when alcohol positive relative to when alcohol negative equals the alcohol-positive injury rate divided by the alcohol-negative injury rate. Equation 1 expresses that definition in equation form with the fraction of events that are alcohol negative equal to one minus the the fraction that are alcohol positive. Equation 1 was applied, in turn, to data on all people over age 14, regular heavy drinkers, and more moderate drinkers. To address the role of lifestyle, we applied it to estimate the risk of hospitalized injury of regular heavy drinkers and separately of other people when both groups are alcohol negative. These calculations used fractions of total alcohol-positive hours and injuries by group.

Within a drinking group, a relative risk ratio above 1 means that injury is more likely when alcohol positive than negative. Between groups it means that drinkers have higher injury risks than non-drinkers.

#### Attributable Fraction of the Alcohol-Involved Injuries (Equation 2)

Equation 2 gives the fraction of injuries attributable to alcohol (AF). This equation is the standard epidemiological equation to compute AF from relative risk (Rockhill et al. 1998). It says that alcohol causes at most the fraction of the alcohol-involved injuries associated with the risk differential from the baseline (alcohol-negative) risk of 1. It yields an upper bound because it assumes all the excess risk is caused by alcohol. Instead, some unobserved factor could cause the rates to differ. For example, injury rates could be higher during recreation hours and recreation hours could be alcohol positive more often than other hours. That limitation is important with our methods. Our exposure measure does not differentiate sleeping from waking hours, working from recreational hours, or a prolonged leisurely sipping from a concentrated binge.

# Fraction of Hours Alcohol Positive (Equation 3, used to estimate H in Equation 1)

Equation 3 computes the fraction of hours that a person is alcohol-positive. To estimate the number of hours alcohol-positive, it divides annual alcohol consumption by the alcohol metabolism rate. The number of hours alcohol-positive then is divided by the number of hours in a year. People live 8,766 hours annually (365.25 days x 24 hours; assuming people who die mid-year counterbalance people who are born mid-year). This equation estimates the annual number of hours members of a group have alcohol in their bloodstream. We first applied it to all Americans age 14 and over. On average, people metabolize 0.45 ounces of

ethanol per hour (Ray and Ksir, 1999). Annual per capita ethanol consumption in the early 1990s averaged 303 ounces (LaVallee and Yi., 2010). (NB: per capita ethanol consumption was virtually the same in 2006-2008.) Thus the average person was alcohol positive for an estimated 673 hours per year (303 ounces / 0.45 ounces/hour). That equates to 0.0767 (7.67%) of the year (673 / 8,766).

Regular heavy drinkers metabolize alcohol at elevated rates (Tabakoff et al., 1986). Our best estimate uses a metabolic rate of 0.675 ounces of ethanol per hour to compute H for heavy drinkers (1.5 times the norm). Sensitivity analysis uses 0.9 ounces per hour.

To estimate alcohol consumption per capita (C in equation 3), we combined annual per capita alcohol consumption (LaVallee and Yi, 2010) with 1998 National Household Survey on Drug Abuse (NHSDA; SAMHSA, 1998) data on alcohol consumed by type of drinker. We estimated consumption overall, for regular heavy drinkers, and for moderate drinkers. On average, heavy drinkers consume 1,716 ounces of ethanol annually (291.4 average annual ounces per person ages 18 and over \* 0.53 of alcohol consumed by heavy drinkers / 0.09 of adults are heavy drinkers). They are alcohol positive an estimated 0.291 (29.1%) of the time: (1,716 ounces / 0.675 oz per hour / 8,766 hours). Non-drinkers never are alcohol positive. From NHSDA, 0.446 of people ages 18 and over abstain. Thus moderate drinkers comprise 0.464 of the population (1 – 0.09 -0.446). They are alcohol positive an estimated 0.075 of the time (291.4 \* 0.47 of all alcohol / 0.45 oz per hr) / (0.464 \* 8,766 hours)).

#### Fraction of Hospitalized Injuries Alcohol Positive (I in Equation 1)

To estimate the fraction of hospitalized injuries that are alcohol positive, we started from estimates of alcohol involvement in hospitalized injury overall and by cause (Levy et al., 2002; Miller et al., 2010; Smith et al., 1999b). With Equation 1, we estimated risk of hospitalized injury when alcohol positive versus alcohol negative, overall and by injury category. The denominator (exposure) was the same. The fraction of hospitalized injuries that were alcohol positive (I) varied by injury category.

We estimated the portion of hospitalized injuries that are alcohol positive by drinking category from Soderstrom et al. (1997). That unique study gives the fraction of hospitalized injuries to heavy drinkers age 18 and over that are alcohol positive (0.5667). Using its value assumes the alcohol involvement rates in injuries of diagnosed problem drinkers and the broader set of heavy drinkers are comparable. We combined its estimate with the estimate of overall alcohol-involvement in hospitalized injuries and with findings on the relative risk of 1.82 of injury among heavy drinkers relative to other adolescents/adults (Miller et al., 2001). That let us estimate the proportion of injuries by drinking category and alcohol involvement.

From Smith et al. (1999b), an estimated 0.27 of all injuries involve alcohol. Heavy drinkers comprise 0.09 of the population. They have 1.82 times as many injuries as other people (Soderstrom et al., 1997). Of their injuries, 0.5667 are alcohol positive so their alcohol-positive injuries account for an estimated 0.093 of all injuries (0.09 \* 1.82 \* 0.5667); their alcohol-negative injuries account for another 0.071 of all injuries (0.09 \* 1.82 \* 0.4333). Other drinkers account for the remaining 0.177 (0.27 – 0.093) of alcohol-negative injuries and 0.659 (0.73 – 0.071) of alcohol-negative injuries. To split the alcohol-negative injuries between moderate drinkers and non-drinkers, we assume the two groups have equal injury rates per alcohol-negative hour. Their alcohol negative hours per person average 23.08 (24 \* (0.446 nondrinkers + 0.464 moderate drinkers \* 0.925 of moderate drinkers' hours alcohol negative) / (0.446+0.464)). Nondrinkers account for 0.336 of the alcohol-negative injuries (0.659 \* (24 / 23.08) \* 0.466 / (0.446+0.464)). Moderate drinkers account for the remaining 0.323. These estimates represent I by population group.

To estimate the all-hours risk of injury among heavy drinkers relative to other people (1.82 above) and a sensitivity analysis range, we reran published logistic regression models (Miller et al., 2001) to better match Soderstrom et al.'s (1997) age groups. The models used three years of linked health insurance claims data to analyze injury risk among 12,828 privately insured people with medically identified acute or chronic conditions linked to alcohol abuse and dependence (e.g., alcohol psychosis, portal hypertension, cirrhosis), 2,652 with conditions linked to drug abuse but not alcohol abuse, and 75,331 controls without medically identified substance abuse problems. We assumed injury risk for these heavy drinkers was representative of risk for all heavy drinkers. We ran regression models that adjusted for demographics. We ran models with three different assumptions about cause of the injury: the excess risk is due to alcohol use, and finally, the excess risk is due in part to drugs and in part to alcohol.

We analyzed spinal cord and burn injuries separately. We used the Soderstrom et al. (1997) value as an estimate of I. Further analyzing Miller, Lestina, and Smith's (2001) data, we estimated that medically identified heavy drinkers have 1.80 greater odds of spinal cord injury (diagnosis codes 806 and 952 in the International Classification of Diseases, 9th Edition, Clinical Modification (ICD9)) and 1.27 greater odds of burn injury (ICD9 codes 940-949) relative to controls who do not abuse alcohol. We estimated relative risk and attributable risk as in the all-injury analysis.

#### Relative Risk of Injury when Alcohol Positive versus Alcohol Negative among Heavy Compared to Moderate Drinkers

Finally, we analyzed whether tolerance to the impairing effects of alcohol among heavy drinkers impacts the risk of injury. To do so, we compared the estimated ratio of relative risk of injury when alcohol positive versus negative (computed using equation 1) for heavy versus moderate drinkers.

#### Sensitivity Analysis

The data about heavy drinkers came from a diverse collection of data sources. They defined heavy drinkers differently, varied in time period, and sometimes were not nationally representative. To compensate, we performed extensive sensitivity analysis around the definition of heavy drinker, the proportion of alcohol consumed by heavy drinkers, and the proportion of heavy drinkers' injuries that are alcohol positive.

The sensitivity analysis (Table 5) used values from alternate studies (Table 2) to derive a range of relative risk estimates. We ranged the estimates beyond the values in existing studies. We also computed relative risks with the main estimates for two boundary conditions: all excess injuries of heavy drinkers are due to their lifestyle or all are due to alcohol.

#### Additional Checks

To assess the quality of our estimates, we also used the formulas to estimate relative risk ratios for injuries treated in US emergency departments and for drivers in fatal motor vehicle crashes. We compared the resulting estimates to published estimates from case-control or case-crossover studies.

## Results

An estimated 27% of hospitalized injury victims are alcohol positive (Table 3); this includes nearly half of hospitalized pedestrian and near-drowning injury victims. The relative risk of

hospitalized injury when alcohol positive versus negative is especially high for assault, pedestrian, and near-drowning incidents. An estimated 21% of hospitalized injuries are attributable to alcohol (from equation 2, 27% \* 3.5/4.5; sensitivity analysis range 14% to 29%). Multiplying the 21% rate times the 1998 injury count from the National Hospital Discharge Survey suggests that almost 540,000 people were hospitalized for injuries caused by alcohol.

Table 4 presents relative risk estimates for hospitalized injury by drinker type. All estimates use other drinkers' hospitalized injury rate when alcohol negative as the comparison rate. Heavy drinkers are an estimated 3.2 times (4.33/1.36) more likely to have a hospitalized injury when alcohol positive than negative. Other drinkers are at even greater risk. When alcohol positive, they are 6.8 times more likely to have a hospitalized injury than when negative. Notably, during alcohol-negative hours, the risk of hospitalized injury among heavy drinkers is an estimated 1.4 times greater than the risk among other alcohol-negative people.

The pattern is similar for hospitalized burn and spinal cord injuries. The estimated risk among heavy drinkers is 3.2 times greater when alcohol-positive than negative. During alcohol-negative hours, heavy drinkers are an estimated 1.6 times more likely to suffer a hospitalized spinal cord injury compared to other alcohol-negative people. The elevated risk of hospitalized spinal cord injury among other drinkers is striking: the estimated risk is 13.6 times higher when alcohol positive than negative.

In sensitivity analysis (Table 5) we computed 578 sets of relative risk estimates using all combinations of alternate values for the variables in Equations 1 and 3 and the injury rate calculation for heavy drinkers versus other people and considered two boundary conditions. The conclusions of the sensitivity analysis are quite strong. Relative to alcohol-negative people who do not regularly drink heavily, heavy drinkers have an elevated risk of hospitalized injury when alcohol negative (true in all but 2.2% of the 578 sets). The injury risk of heavy drinkers rises when alcohol positive. Relative to their risk when alcohol negative, non-heavy drinkers face greater injury risks per alcohol-positive hour than heavy drinkers (true in all but 3.5% of the 578 sets).

Table 6 estimates all-hours relative risk of hospitalized injury by drinker class, regardless of whether alcohol positive or negative. These relative risk estimates were computed with data from Miller, Lestina, and Smith (2001). They control for differences in age, sex, white-versus-blue collar employment, and region of residence between regular heavy drinkers and other adults. The estimated all-hours injury risk of moderate drinkers relative to abstainers is modestly elevated.

### Discussion

The three hypothesized patterns emerge. First, both heavy and moderate drinkers appear to have elevated risks of hospitalized injury when alcohol positive. Prior studies agree that even moderate drinking significantly increased injury risk (Cherpitel, 1999). Second, regular heavy drinkers appear to have higher hospitalized injury risks even when alcohol negative. Third, alcohol appears to raise injury risks of regular heavy drinkers less than that of other drinkers.

This paper's estimates suggest that alcohol causes serious injury. By comparing injury risks of relatively homogeneous groups of drinkers during alcohol-negative hours relative to alcohol-positive hours, we control for lifestyle and demographic differences between heavy drinkers and light to moderate drinkers. Alcohol-impaired psychomotor skills pose a risk. Laboratory studies find that alcohol impairs reaction time, ability to divide attention, motor

skills, coordination, memory, and decision-making (Howland et al., 2010; Normand et al., 1994). Whether the impairment results in injury in real life depends on factors such as the dose and timing of alcohol intake, the drinker's ability to perform under the influence (functional tolerance), and the task being performed.

The elevated injury risk estimate for heavy drinkers when alcohol negative may reflect a risk-taking lifestyle. Cherpitel (1999) examined the relationship between substance use, risk-taking, and injury among a nationally representative sample of Americans. She developed a scale for risk taking disposition. Both substance users and risk-takers were more likely to be injured. However, only risk-taking was a significant predictor of injury after controlling for substance use.

The association of drinking habits with injury also may be due to low residual blood alcohol levels or to hangover (Howland et al., 2001; Morrow et al., 1991; Wolkenberg et al., 1975; Yesavage and Leirer, 1986). Declining health, or other lingering effects of regular heavy drinking, also may elevate the risk of injury when alcohol negative.

As well as estimating overall injury risk, this article estimates risks of hospital-admitted or fatal spinal cord and burn injuries. We chose these risks because the incidents are serious, they are readily identified from diagnosis codes in medical records, and their rate of alcohol involvement has been well documented in an entire state. Our burn estimates, however, have not untangled possible interactions between alcohol-related and smoking-related risks.

Our estimates that heavy drinkers experience a smaller injury risk than other drinkers when alcohol positive but have higher background risks also are consistent with the literature (Cherpitel et al., 2004; Borges et al., 2006; Spurling and Vinson, 2005). Alcohol's smaller estimated impact on hospitalized injury risk for heavy drinkers compared to other drinkers also supports previous findings that regular drinkers develop tolerance to the impairing effects of alcohol (Chesher and Greeley, 1992). Their brain functions adapt to the physical disruption caused by the drug in both behavior and physical function. Consequently, the causative link between alcohol and hospital-admitted injury seems to be stronger for people who are not heavy drinkers.

We estimate 24.2% of hospitalizations for motor vehicle injury are alcohol-attributable. Alcohol involvement in crashes rises with crash severity. In 1994, 38.4% of drivers in fatal crashes were alcohol-positive. Applying the formula used in Table 3, the estimated attributable fraction is 33.3%. By comparison, applying the odds ratios from the classic Borkenstein (1974) data to the 1994 driver blood-alcohol distribution, the attributable fraction is 31.2% (13390 drivers at .08 and above \* 0.895 + 2236 drivers at .04-.079 \* 0.326). So our formula closely approximates the more exact measurement. Our hospitalization estimate also appropriately lies between narrower estimates from Miller et al. (1998) that 26.7% of fatal injuries and 9.5% of nonfatal injuries in U.S. crashes are alcohol attributable injuries to occupants of drinking drivers' vehicles.

We estimate that the relative risk of hospital-admitted injury after drinking in the United States is 4.5. A pooled case-crossover analysis that incorporates data from 28 ED clusters in 16 countries (including 6 U.S. clusters) finds a relative risk of ED-treated injury of 5.2 before statistical adjustment for covariates and 5.7 after adjustment (Borges et al., 2006). Additional large U.S. case-crossover studies in a university town in Missouri find odds ratios of 4.0 to 5.6 for any alcohol in the six hours prior to injury (computed using the discordant pairs method) (Vinson et al., 1995, 2003). A pooled relative risk estimate for all 8 U.S. studies is 3.8 and for all 30 studies is 4.8. Across the six U.S. clusters in Borges et al. (2006), 21.3% of ED-treated injuries were alcohol-involved and the pooled relative risk estimate is 3.8. The formula used in Table 3 yields a relative risk ratio of 3.3 for ED-treated

injury. Thus our estimates are reasonably consistent with but possibly a bit below the casecrossover estimates..

Estimates from case-control methods are less consistent with our estimates for admitted patients. The studies with a community control group arrive at odds ratios of 6.7 for Mexico and 2.1-3.4 for Australia and the U.S. with a pooled odds ratio of 2.8 (Borges et al., 1998; McLeod et al., 1999; Stockwell et al., 2002; Vinson et al., 2003; Watt et al., 2004). Our rough pooling of published data on ED-comparison groups from 19 ED clusters in 8 countries yields odds ratios of 1.6-1.9 for all injury (1.2-1.6 for 6 U.S. studies) depending on whether one relies on blood alcohol level (BAL) test results or on self-report (Cherpitel et al., 2005; Kuendig et al., 2008). In these data, 10.5%-11% of US injury patients were alcohol-positive. Our formula yields a relative risk ratio of 1.4-1.5, which matches the case-control estimates. This method assumes that all patients going to the ED due to acute or chronic illness were well enough to drink normally in the six hours prior to arriving at the ED, which is questionable and may bias the risk ratio. That may explain why the ratios with non-injury ED controls are lower than those from other methods.

Our estimated relative risk ratio for interpersonal violence is 8.4, meaning 88% of assaults were caused by alcohol if the victims were alcohol positive. By comparison, a simple caseweighted average of relative risk ratios from 13 studies with ED controls yielded a relative risk ratio of 7.4 (Cherpitel et al., 2005). The ratio was 5.6 with a range from 4.1 to 10.4 for the subset of five U.S. studies (four of them in California). A Swiss study with ED controls yielded an odds ratio of 9.2 (Kuendig et al., 2008). A case-crossover study in three Latin American countries yielded a matched pair odds ratio of 15.0 (Borges et al., 2008). A U.S. study yielded a community-case control odds ratio of 10 and a case-crossover odds ratio of 34 (Vinson et al., 2003). Thus our estimate is consistent with most others. We attribute an estimated 36.1% of hospitalized assaults to victim alcohol use, with 41% of victims alcohol positive. In the U.S., perpetrators report being alcohol positive in 41% of assaults (Miller et al., 2006). Conventional wisdom has been to conservatively assume half of those were attributable to alcohol. We recommend raising the attributable fraction to 36%, at least for medically attended assaults. That estimate is conservative since surely some alcoholattributable incidents involve an alcohol positive perpetrator and an alcohol negative victim. Indeed, with 24.4% to 44.3% of apprehended offenders qualifying as abusing or dependent on alcohol (Compton et al., 2010), our formulas suggest 30.3%-34.6% of violent crime and 9.0%-14.7% of property crime is partially attributable to perpetrator alcohol use (e.g., H=(. 244 \* 31.156% of heavy drinker's hours alcohol positive + (1-.244) \* 2.845\% of other people's hours alcohol positive) = .0975. I = 0.23 for property crime from Miller et al. (2006). RR= (.23/.0975)/(.77/.9025) = 2.764. AF = .23 \* 1.764/2.764 = 14.7%). Collins and Schlenger (1988) estimated that the relative risk that a crime was violent given alcohol use when it was committed was 1.73, somewhat below our estimate of 2.33 (ratio of relative risks = 6.430/2.764).

For suicide acts, our relative risk estimate is 5.1 for hospital-admitted nonfatal cases ages 18 and over (4.5 for those ages 14 and over). It would have been 6.6 had we used the 35.3% alcohol involvement rate reported for the pooled 102 suicide attempters in 7 North American and Australian ED studies (Borges et al., 2004b). Based on a case-crossover design, the relative risk reported for those studies was higher, 9.6, with a 95% confidence interval of 5.7-16.3. Powell et al. (2001) found a 26.5% alcohol involvement rate among 153 near-lethal suicide acts (76.5% of ED-treated acts) in Houston TX. The crude odds ratio was 7.0 relative to community controls. That ratio rose to 8.0 after statistical adjustment for sample differences.

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This paper shares the limitations of the studies that supplied data for the computations. The alcohol involvement rate for hospital-admitted heavy drinkers is for injury victims treated at a single trauma center in Maryland that only sees seriously injured patients (Soderstrom et al., 1997). The injury risks for heavy drinkers relative to controls may be underestimates because heavy drinkers who cannot be identified from a 3-year medical record are misclassified into the lower-risk control group (Miller et al., 2001). Also, this paper assumes the injury profile of heavy drinkers diagnosed with alcohol problems applies to all regular heavy drinkers.

The attribution estimates by injury type require additional assumptions, making them more speculative. Furthermore, the Oklahoma data are restricted to cases exceeding a severity threshold (Levy et al., 2002). Less severe injuries, notably burns not triaged to burn centers may have different causal chains. Still, these estimates are consistent with the all-hours injury estimates. They suggest that, controlling for lifestyle effects, alcohol raises hospitalized injury risk for these subsets.

The relative risks estimated with Equation 1 are spread across all hours in a drinking status, including the minimal risk during hours when someone is sleeping off the alcohol they consumed or at very low positive blood alcohol concentrations (BACs). The alcohol-negative injury risk estimate also includes sleep hours and most working hours. Alcohol impairment is not uniform across those hours; it rises more than linearly with alcohol level (e.g., Kuendig et al., 2008). Moreover, drinking pace affects peak BAC. Drinking sessions often last 4-5 hours (Beirness et al., 2004; Lange and Voas, 2001; Thombs et al., 2003). Consequently, BACs of half of people consuming 5 drinks or more were at .06 or lower when they stopped drinking. Similarly, Cranford et al. (2006) finds that 10% of those who drink 5 or more drinks in a drinking session drink slowly enough that their BACs remain moderate. Data were not available to adjust Equation 1 to reflect impairment variations due to changing alcohol levels. Similarly, exposure to injury hazards is not uniform across those hours. It is lowest when sleeping and relatively low when working.

Except for heavy drinkers, this paper assumes that hospital-admitted injury risks while alcohol negative do not differ systematically with a person's drinking habits. That assumption is weak. For example, some people who are not classified as regular heavy drinkers may drink heavily occasionally. Those episodic drinkers may be risk-takers generally. Hence the relative injury risks for heavy drinkers when alcohol positive versus alcohol negative are the more perfect fingerprints. Controlling for lifestyle by focusing on a homogeneous group of drinkers, the findings demonstrate that average injury risks are elevated during alcohol-positive waking and sleeping hours.

A further limitation is the cobbled nature of our estimates. They are sewn from data from different locations and time periods. To the extent that alcohol consumption in the states represented in the data about alcohol involvement in injury differs from national average consumption, the estimates will be skewed.

These limitations are offset by the robustness of the results in extensive sensitivity analysis on the imperfect parameter estimates. Estimates from the sensitivity analyses are consistent with the main estimates. The estimated size of the relative risks shifts, but not their direction.

Our attribution rate estimates consider only victim alcohol involvement. Some alcoholnegative victims are injured, however, because someone else was drinking (Levy et al., 2002). For example, crashes attributable to alcohol in the U.S. injure an estimated 257,000 alcohol-negative pedestrians, drivers, and passengers per year (Miller et al., 1998). These victims account for 7.6% of deaths, 3.9% of nonfatal injuries, and 25,000 hospitalized

injuries in crashes. Adding them would raise the alcohol-attributable percentage of hospitaladmitted injuries from 21% to 22%.

To refine and validate our estimates, future research needs to measure blood alcohol levels of a more representative sample of hospitalized injury victims and assess their drinking behavior. Segmenting the injury rates by alcohol involvement for those who occasionally drink heavily versus those who always drink moderately seems critical. Broadly representative data are needed on alcohol involvement in the injuries of heavy and dependent drinkers. Breaking out risk by injury cause and drinker demographics, especially sex and race, is important. Most Asians lack secondary alcohol metabolism channels. Research on their relative risks when alcohol positive might be especially accurate and informative because the calculations would be free from metabolism-related uncertainty. Another priority is an analysis for youth accounting for their lower total body water and accelerated rate of ethanol elimination (Donovan, 2009).

Our estimates have strong policy implications. They set the stage for injury warning labels on alcohol bottles. They also suggest what percentage of public injury cost justifiably could be recovered through alcohol taxes.

Moderate drinking has not traditionally been considered hazardous. From an injury viewpoint, it appears to be more hazardous per drink than regular heavy drinking. Heavy drinkers are alcohol positive for 3 to 6 times as many hours as other drinkers. Thus, they suffer more alcohol-positive injuries. Other drinkers, however, suffer more injuries per hour alcohol positive. Intervention rarely has been targeted to this group, because its high risk was hidden. Revealing that risk sets a difficult public debate into motion. Estimates that moderate drinking with an occasional binge greatly raises hospitalized injury risks raise doubts about U.S. nutritional guidelines that tout moderate wine drinking (U.S. Department of Agriculture, 1990). Given the historical failure of prohibition, harm reduction strategies seem the logical course of action.

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# Bios

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# Equations Used in the Current Study

Equation Number	Objective of Equation	Formula	Variable Definitions
1	To compute the relative risk of injury when alcohol-positive versus negative (RR)	RR = [I / H] / [(1 - I) / (1 - H)]	I = fraction of hospitalized injury victims who are alcohol-positive H = fraction of hours alcohol-positive
2	To compute the upper bound of the fraction of injuries attributable to alcohol (AF)	$AF = I^*(RR - 1) / RR$	RR = Relative risk of injury when alcohol- positive versus negative (see Equation 1)
3	To compute the fraction of annual hours that a group of adults is alcohol positive (H)	H = (C / M) / 8,766 hrs	C = per capita alcohol consumption in ounces M = the average rate alcohol is metabolized = 0.45 oz/ hour across all drinkers and 0.675 oz/hour for heavy drinkers 8,766 hours = the number of hours in a year

### Data Sources and Values Used in the Current Study

Authors, Year	Description of source	Description of how data were used in the current study	Source's definition of heavy drinker (where applicable)
Clark and Hilton, 1991	1984 National Alcohol Survey	% of population who are heavy drinkers (PctPop <sub>HD</sub> =11% in sensitivity analysis) % of alcohol consumed by heavy drinkers (PctAlc <sub>HD</sub> =67% in sensitivity analysis)	Consumption of 5 or more drinks on one occasion at least once a week
LaVallee and Yi, 2010	Estimated annual alcohol consumption nationally and by state	Average alcohol consumption per person in 1989-1994 (c=303 oz)	N.A.
Levy et al., 2002 Plus supplemental analyses	Analysis of Oklahoma data on hospitalized spinal cord, burn, drowning and traumatic brain injuries; victims ages 15 to 64; alcohol involvement noted in >90% of injuries.	See Table 3 for values. Alcohol involvement averaged over all four categories (p=28% in sensitivity analysis)	N.A.
Manning et al., 1991	RAND Health Insurance Experiment 1983 National Health Interview Survey	% of population who are heavy drinkers (PctPop <sub>HD</sub> =10% in sensitivity analysis) % of alcohol consumed by heavy drinkers (PctAlc <sub>HD</sub> =68% in sensitivity analysis)	Consumption of an average of at least 5 drinks per day
Miller et al., 2001 Plus supplemental analyses	A case-control study that compared the odds of injury among people aged 14-64 whose 1987-1989 health insurance claims include treatment for diagnoses that the Alcohol Epidemiologic Data System of the National Institute on Alcohol Abuse and Alcoholism (Cases et al., 1994) classifies as alcohol- related.	Risk of injury among heavy drinkers relative to other people ( $RR_{HD}$ ); reruns at ages 18-64 to better match the Soderstrom et al. (1997) age distribution are used in the main analysis ( $RR_{HD}$ =1.82; sensitivity range 1.67 when drug-only cases are used in the estimation, 2.09 when excess risk for alcohol and drug users is attributed to alcohol, 2.56 for ages 15-64). For spinal cord and burn injuries ( $RR_{HD}$ =1.8 and 1.27, respectively, with sensitivity ranges of 1.63- 1.93 and 1.2-1.4)	Any treatment for alcohol-related diagnoses that include chronic alcohol abuse indicators such as cirrhosis, subacute indicators such as alcoholic psychoses, and acute ethanol poisoning.
Ray and Ksir, 1999	Book on the biological and social aspects of alcohol use and abuse.	People metabolize an average of 0.45 ounces of absolute ethanol per hour.	N.A.
Smith et al., 1999b; Selway et al., 2008; Miller et al., 2010	Smith reviews the literature on the role of alcohol in injury. The other studies fill gaps or provide more robust estimates for selected mechanisms	Alcohol involvement in injury by cause. See Table 3 for values.	N.A.
Soderstrom et al., 1992	Interim report of Soderstrom et al. (1997) study	Percent of hospitalized injuries to heavy drinkers ages 18 and over who were alcohol-positive (PInj <sub>HD</sub> =62% in sensitivity analysis)	Alcohol abuse or dependence according to DSM-III-R
Soderstrom et al., 1997	Examined the percentage of serious trauma patients who were alcohol-positive, by alcohol dependency	Percent of hospitalized injuries to heavy drinkers ages 18 and over that were alcoholpositive (PInj <sub>HD</sub> =56.7%)	Alcohol abuse or dependence according to DSM-III-R
Substance Abuse and Mental Health Services Administration, 1998	U.S. Census Bureau survey that probes alcohol use of a national probability sample of U.S. households	% of population ages 18 and over who did not consume alcohol, past month (44.6%) % of alcohol consumed by people ages 18 and over (96.5%) % of population who are heavy or dependent drinkers (PctPop <sub>HD</sub> = 9%; 6.2%	Drank 5 drinks or more on at least 5 days in the past month (6.2%) or were alcohol-dependent according

Authors, Year	Description of source	Description of how data were used in the current study	Source's definition of heavy drinker (where applicable)
		heavy drinkers in sensitivity analysis) % of alcohol consumed by heavy and dependent drinkers (PctAlc <sub>HD</sub> =53%; 47.5% heavy drinkers in sensitivity analysis)	to an abridged version of the DSM- IV (3.6%) or were treated for alcohol problems in the past year (0.3%)

Percentage of Hospitalized and Fatal Injury Victims Age 18 and Over Who Are Alcohol-Positive and Estimated Injury Risk When Alcohol-Positive Relative to Alcohol-Negative, by Injury Category

Injury category	% BAL + = I	Relative Risk	% Attributable
Motor vehicle	30%	5.2	24%
Pedestrian	44%	9.5	39%
Assault	41%	8.4	36%
Suicide attempt <sup>a</sup>	29.4%	5.1	16.6%
Other spinal cord	38.6%	7.6	33.5%
Other traumatic brain	23.0%	3.6	16.6%
Other burn	16.8%	2.4	9.9%
Other near drowning	43.4%	9.2	38.7%
Fall	25%	4.1	19%
Ages 15-64	54.4%	14.4	51%
Age 65 & over	13%	1.8	6%
All others $a$	25%	4.0	19%
ALL	27%	4.5	21%

<sup>a</sup>Excludes fatalities; 26.7% and 4.5 for ages 14 and over

Note: Relative risk estimated using equation 1 with H=0.0767. For example, for all injury, relative risk = (.27/.0767) / (.73/.9233) = 4.5.% attributable estimated using equation 2.

Estimated Relative Risk of Hospitalized Injury by Whether Alcohol-Positive, Type of Injury, and Drinker Class

	Alcohol-Positive	Alcohol-Negative
ANY INJURY		
Regular heavy drinker	4.33	1.36
Other person age 18 and over	6.76	1.00
BURN		
Regular heavy drinker	2.70	0.85
Other person age 18 and over	3.31	1.00
SPINAL CORD INJURY		
Regular heavy drinker	5.20	1.63
Other person age 18 and over	13.60	1.00

Note: Estimated using equations 1 and 3 with 27% of all injuries alcohol-involved from Table 3, 56.7% of heavy drinkers' injuries alcoholinvolved, a 1.82 relative risk of injury for heavy drinkers versus other people, 53% of the alcohol consumed by the 9% of people ages 18 and over who are regular heavy or dependent drinkers, and metabolism rates of 0.675 ounces of absolute ethanol per hour for regular heavy or dependent drinkers and 0.45 ounces per hour for other drinkers.

Sensitivity Analysis on Relative Risk of Hospitalized Injury by Whether Alcohol-Positive, Type of Injury, and Drinker Class

	Alcohol-Positive	Alcohol-Negative
ANY INJURY		
Regular heavy drinker	2.03-9.30	0.94-3.27
Other person age 18 and over	3.81-12.10	1.00
BURN		
Regular heavy drinker	1.33-4.27	0.63-1.54
Other person age 18 and over	2.88-4.26	1.00
SPINAL CORD INJURY		
Regular heavy drinker	2.46-7.96	1.16-2.90
Other person age 18 and over	12.69-18.78	1.00

Note: Estimated using equations 1 and 3 with 25%-30% of injuries alcohol-involved; 56.7%-62% of heavy drinkers' injuries alcohol-involved; relative risk of injury for regular heavy drinkers versus other people ranging from 1.67 to 2.56 (1.2 to 1.4 for burns, 1.63 to 1.93 for spinal cord injury); 50%, 53%, or 60% of the alcohol consumed by the 9% of people 18 and over who are regular heavy drinkers or 47.5% consumed by 6.2% or 68% consumed by 10% or 67% consumed by 11%; and metabolism rates of 0.45 to 0.9 ounces of absolute ethanol per hour for regular heavy drinkers. Boundary conditions applied to the values from Table 4 forced the relative risk of alcohol-negative heavy drinkers to equal 1.0 or equal their alcohol-positive risk.

All-Hour Hospitalized Injury Risk by Type of Injury and Drinker Class Relative to Non-drinkers

	Any Injury	Burn	Spinal Cord
Regular heavy drinker	2.22	1.31	2.43
Other drinker	1.43	1.18	1.96
Non-drinker	1.00	1.00	1.00

Note: Estimated from estimates in Table 4 and hours alcohol-positive. For example, for any injury of regular heavy drinkers, the risk is  $(4.33 \pm 6.98 \text{ hours positive} + 1.36 \pm 17.02 \text{ hours negative}) / 24 \text{ hours} = 2.22$ . Other drinkers are alcohol-positive for an average of 1.8 hours daily.