Attributable Risk of Injury Associated With Alcohol Use: Cross-National Data From the Emergency Room Collaborative Alcohol Analysis Project

Cheryl J. Cherpitel, DrPH, Yu Ye, MA, and Jason Bond, PhD

A strong association between alcohol use and injuries has been documented in the alcohol and injury literature, and much of this evidence has been derived from emergency room (ER) studies.^{1,2} Less is known about the attributable risk of injury associated with alcohol use (or the proportion of injury that would be eliminated in the absence of exposure to alcohol), an exposure measure that relies on estimates of injury risks among individuals exposed versus not exposed to alcohol. Most trauma studies have consisted of case series reports in which estimates of relative risk are not possible, necessitating the use of other data sources.3,4 Previous estimates of attributable risk have not included data derived from epidemiological studies involving probability samples of injury patients, nor do they reflect age-specific or culture-specific differences in relative risk.5,6

To fill this gap in the literature, we used meta-analytic techniques to analyze data on attributable risks associated with both alcohol consumption before an injury event (assessed via blood alcohol concentration [BAC] and self-reported consumption) and a consumption pattern in which 5 or more drinks at a time are consumed at least monthly ("5+ monthly drinking pattern"). We assessed the resulting effects on injury morbidity rates across 14 studies representing 30 ERs in 7 countries.

We based our attributable risk estimates on comparisons of injured patients with noninjured patients, and we report separate estimates for all-cause injuries and violencerelated injuries.^{7,8} We also report genderspecific and age-specific (younger than 30 years vs 30 years or older) estimates. In addition, we analyzed the extent to which studyspecific sociocultural contextual variables explained observed differences in attributable risk estimates across studies. This analysis provided the opportunity for more fine-grained *Objectives.* We sought to determine gender- and age-specific attributable risks of all-cause and violence-related injuries associated with alcohol use.

Methods. We used meta-analytic techniques to estimate attributable risks observed in emergency room studies conducted in 7 countries (n = 17708).

Results. In the case of both alcohol consumption before the injury event and individual drinking patterns, pooled attributable risk effect sizes for all-cause injuries were significant but minimal (2% to 6%). Effect sizes for violence-related injuries were 43% for drinking before an injury event and 27% for individual drinking pattern. Risks were greater for men, but no age-specific differences were found.

Conclusions. This meta-analysis showed that attributable risk of injury is greater for drinking before the injury event than for drinking pattern; in addition, risks were more pronounced for violence-related injuries. Differences in risk were explained by variables related to sociocultural contexts. (*Am J Public Health.* 2005;95: 266–272. doi: 10.2105/AJPH.2003.031179)

estimates of attributable risks of alcohol use associated with injury morbidity and for evaluation of the extent to which cultural variables explain observed differences across geographic localities.

METHODS

ER Data

The data analyzed here were derived from studies included in the Emergency Room Collaborative Alcohol Analysis Project (ERCAAP) (Table 1). Complete details on the ERCAAP procedures have been provided elsewhere.^{9,10} Data from all of the ER studies included in ERCAAP were collected according to a methodology and protocol similar to that developed by Cherpitel.¹¹ Across the study sites, the design involved probability sampling of ER patients 18 years or older.

Patients were approached and asked to participate; if they agreed, they were interviewed for approximately 25 minutes via a standardized questionnaire. Also, estimates of patients' BAC levels were obtained as soon as possible after patients had been admitted to the ER. Interview completion rates ranged from 68% to 93%; reasons for noncompletion of interviews included refusal, incapacitation, and language barriers. Patients who were too severely injured or ill to be approached in the ER were interviewed once their condition had stabilized.

Drinking before the injury event was defined as a positive BAC at the time of arrival at the ER or a report of alcohol consumption within 6 hours before the event. In all of the ERCAAP studies other than the Canadian study, the Alco-Sensor III breathalyzer, which has been shown to provide estimates that are highly correlated with chemical blood analyses, was used in estimating BACs.¹² In the Canadian study, BACs were estimated from urine samples that were assessed for ethanol via KDA enzymatic testing and then standardized to the unit measure quantifying BAC estimated from breath samples. As mentioned, alcohol consumption patterns were analyzed in relation to whether patients reported having consumed 5 or more drinks at a time at least monthly during the past year.

Contextual Data

The contextual data gathered included information on sociocultural variables thought

Study No. and Location	Collaborator ^a	Year(s)	No. Sites	No. Participants	Recorded per Capita Ethanol Consumption, L	Legal Drinking Age, y	Legal Intoxication Level, mg/100 mL	Alcohol Use Stigmatization Level	Detrimental Consumption Pattern ^b	Homicide Rate per 100 000 Population
1. San Francisco, Calif	Cherpitel	1984-1985	1	1896	11.94	21	0.10	0	1	10.5
2. Contra Costa, Calif	Cherpitel	1985	4	2400	11.81	21	0.10	1	1	10.5
3. Contra Costa, Calif	Cherpitel	1989	3	961	10.56	21	0.10	1	1	10.5
4. Jackson, Miss	Cherpitel	1992	1	1017	7.91	21	0.10	1	3	12.3
5. Santa Clara, Calif	Cherpitel	1995-1996	1	1334	8.46	21	0.08 ^c	0	1	9.1
6. Mexico City, Mexico	Borges (Rosovsky) ^d	1986	8	2188	4.59	18	0.08	0	4	12.69
7. Acapulco, Mexico	Borges (Garcia) ^d	1987	3	640	4.64	18	0.08	0	4	49.26
8. Pachuca, Mexico	Borges/Cherpitel	1996-1997	3	1417	5.14	18	0.08	0	4	6.8
9. Alberta, Canada	Giesbrecht/Macdonald	1989	1	842	9.76	18	0.08	1	3	1.85
10. Quebec, Canada	Giesbrecht/Macdonald	1989	1	655	8.33	18	0.08	0	1	2.06
11. Barcelona, Spain	Cherpitel (Rodes) ^e	1987	1	2363	5.99	16	0.08	0	1	0.9
12. Trieste, Italy	Cherpitel (Poldrugo) ^d	1990	1	476	9.07	16	0.08	0	1	1.4
13. Mar Del Plata,	Cremonte	2001	1	800	15.30	18	0.05	1	2	5.8
Argentina										
14. Warsaw, Poland	Cherpitel/Moskalewicz ^e / Swiatkiewicz	2001	1	719	13.0	18	0.02	1	3	2.7

TABLE 1—Characteristics of 14 Studies Included in the Emergency Room Collaborative Alcohol Analysis Project

^aInvestigator(s) representing the study in the collaborative project.

^bSee Rehm et al.^{16,18} Distinct within-country regional variations in drinking patterns have been found; as a result, Quebec was assigned a lower pattern level than Canada as a whole, while Alberta was assigned a higher level. In the United States, California was assigned a lower level than the country as a whole, while Mississippi was assigned a higher level.

^cData collection occurred subsequent to a decrease in the legal intoxication level in the United States.

^dPrincipal investigator of study.

^eCo-principal investigator of study.

to be markers of aggregate exposure to alcohol (legal drinking age, legal level of intoxication while driving) and of alcohol's integration into a particular society (per capita consumption, stigmatization of alcohol use, detrimental drinking patterns). Societies in which levels of per capita consumption are high are also characterized by daily light drinking, in which alcohol is integrated with meal functions, and by lower levels of stigmatization associated with alcohol use.^{13–15} Attributable risks of injury associated with alcohol use may be higher in societies involving greater levels of exposure to alcohol and lower in those societies in which alcohol use is integrated into the culture.

Stigmatization of alcohol use was based on the level to which alcohol use is stigmatized or integrated into a particular society and the degree to which obtaining information about alcohol use in the ER setting may have been underreported by patients (measured on a scale ranging from 0 [low] to 2 [high]). The detrimental drinking pattern variable^{16,17} tapped patterns that might be expected to affect the outcomes associated with a given volume of consumption. This measure included indicators of heavy drinking occasions, drinking with meals, and drinking in public places. Detrimental drinking patterns were based on a survey of key informants in each study country.^{18,19} Detrimental impact scores ranged from 1 to 4, with higher scores indicating higher postulated detrimental effects of the same per capita consumption level.^{16,17}

Because a positive association has been shown between alcohol consumption and homicide rates, homicide rate was also included as a contextual variable in analyses of violence-related injuries.²⁰ All contextual data with the exception of data regarding detrimental drinking patterns were obtained from the ER study collaborators, and they reflect the catchment area of the ER and the time period during which the ER data were collected. Table 1 shows the distribution of contextual variables across the ER studies.

Data Analysis

Primary data from each of the ER studies were cleaned and merged into a single data file. Meta-analytic techniques were applied at the study level (each study could include one or more individual ER sites) rather than the ER level, because the numbers of patients were too small in the case of some ERs to allow reliable estimates. Weights were constructed for each ER within a study to adjust for differential sampling fractions and the length of time over which data were collected. These weights ranged from 0.35 to 1.64.

In the case of each study, attributable risk estimates (attributable fractions) for all-cause and violence-related injuries were calculated separately for the 3 study risk factors (BAC, self-reported consumption 6 hours before the injury, and 5+ monthly drinking pattern),

each of which was coded dichotomously. The following formula²¹ was used in these calculations:

(1) Attributable Fraction = $p_e(\text{RR} - 1)/[p_e(\text{RR} - 1) + 1].$

Here p_e is the proportion of the population exposed to the risk factor and RR is the relative risk of injury, defined as the ratio of the injury rate among those exposed to the risk factor to the injury rate among those not exposed to the risk factor. Attributable risk estimates for all-cause injury were calculated against cases in which there was no injury, and estimates for violence-related injury were calculated against cases involving no injury in combination with all other (nonviolent) causes of injury. Pooled estimates of attributable risk were calculated for the total ER population according to gender and age group (younger than 30 years vs 30 years or older).

We estimated the Q statistic to test the hypothesis of homogeneity of effect sizes (attributable risk) across the ER studies.²² We report fixed pooled effect sizes in cases of homogeneity and random effect sizes in cases of heterogeneity.²³ Add-on programs available in Stata were used in estimating effect sizes and in conducting the metaanalyses.²⁴ When pooled effect size estimates were heterogeneous, we used meta-regression techniques to examine the possible contribution of the study-specific contextual variables to observed differences in effect sizes across studies. In these analyses, the dependent variable was the study-level attributable risk, and covariates were the study-level contextual variables.

Because the number of studies available did not permit simultaneous entry of contextual variables, regressions were performed univariately. Both fixed and random effects regressions are reported. The fixed effects regressions involved the assumption that effect sizes across ER studies varied only as a function of individual study characteristics, while the random effects regressions involved the assumption that differences in effect sizes were due not only to measured characteristics of each ER study but also to an additional component of variance. Cases in which *P* values differed greatly between the 2 types of regressions represented evidence of the contribution of between-study variance, even after control for contextual variables.²⁵

The intent of the present analysis was not to generalize the combined ER populations represented in the data, because the number of available studies was limited and no information was available on ER population distributions. As a consequence of the large estimated between-study variances and the relatively few numbers of studies, the significance levels from the random effects analyses should be viewed as conservative, with actual levels probably residing somewhere between the results shown by the fixed and random effects analyses.

RESULTS

Attributable risks are based on both exposure rates and risks of injury relative to exposure in a given population. Table 2 shows exposure rates, relative risks, and attributable risks for all-cause and violence-related injuries associated with the 3 alcohol exposure variables in each ER study, while Table 3 shows pooled effect sizes for the total ER sample and separately by gender and age group. It can be seen from Table 2 that although attributable risks varied considerably across studies, significant estimates were found in a majority of studies for both all-cause and violence-related injuries for all 3 exposure variables, with estimates higher for violencerelated than for all-cause injuries.

Table 3 indicates that pooled attributable risk estimates were significant for all 3 exposure variables in the case of both all-cause and violence-related injuries. Attributable risks were small for all-cause injuries, ranging from 2% (BAC) to approximately 6% (6-hour self-reported drinking and 5+ monthly drinking pattern). Attributable risks were lower for women than for men in the case of each exposure variable. No differences in attributable risk estimates were found between the 2 age groups assessed.

In the case of all exposure variables, attributable risks were considerably higher for violencerelated injuries, ranging from approximately 28% for BAC and 5+ monthly drinking pattern to 43% for 6-hour self-reported drinking. Effect sizes (attributable risks) associated with BAC were found to be homogeneous. Here again, although no differences were found according to age, attributable risks were higher for men than for women in the case of each exposure variable and reached nearly 50% among men who reported drinking before the injury event.

As can be seen in Table 4, random effects models indicated that, in the case of all-cause injuries, detrimental drinking pattern was a significant predictor of attributable risk associated with BAC, while legal intoxication level approached significance ($P \le .1$). Fixed effects models showed that all variables other than per capita consumption were significant predictors of effect sizes for self-reported drinking. Estimates of between-study variances were larger for models based on self-reports than for those based on BAC, and thus results were more discrepant in the random effects models. In terms of predicting effect sizes for attributable risks associated with 5+ monthly drinking pattern, only legal drinking age and stigmatization were significant in random effects models.

In the case of violence-related injuries, per capita consumption and stigmatization of alcohol use were significant positive predictors of attributable risks associated with self-reported drinking in fixed effects models; in addition, both of these variables, as well as legal drinking age, were positive predictors of the attributable risk effect size for 5+ monthly drinking pattern. Homicide rate was not predictive of effect sizes for either type of alcohol exposure. As a result of the large between-study variances, none of the coefficients were significant in the random effects models. Therefore, the relationships between the predictors and the attributable fractions for each risk factor were stronger for all-cause injuries than for violence-related injuries.

DISCUSSION

The results of this meta-analysis suggest that when all types of injuries are considered together, both alcohol consumption before an injury event and usual consumption pattern contribute little to injury occurrences, especially in the case of women, and effect sizes are not homogeneous across studies. In the case of all-cause injuries, few differences were found between individuals younger than 30

TABLE 2—Risk Exposure Rate (RER), Relative Risk (RR), and Attributable Fraction (AF) for All-Cause Injuries and Violence-Related Injuries

Positive BAC ^a				Positive Self-Report				\geq 5 Monthly Drinking Pattern							
Study No.	RER, %	RR: Injury	AF: Injury, %	RR: Violent Injury	AF: Violent Injury, %	RER, %	RR: Injury	AF: Injury, %	RR: Violent Injury	AF: Violent Injury, %	RER, %	RR: Injury	AF: Injury, %	RR: Violent Injury	AF: Violent Injury, %
1	21.4	1.10	2.0	2.77	27.5***	23.0	1.81	15.8***	4.10	41.6***	37.2	1.18	6.3*	1.90	25.0***
2	7.3	1.27	2.0*	4.34	19.6***	16.8	1.16	2.6*	5.34	42.0***	22.3	1.27	5.6***	2.55	25.7***
3	4.0	0.82	-0.7	7.92	21.8	7.9	1.07	0.5	4.06	19.1	10.1	1.57	5.5***	1.36	3.6
4	10.4	1.90	8.5*	5.80	33.5***	10.9	2.38	13.1***	10.40	50.7***	15.7	1.48	7.0**	4.02	32.2***
5	8.6	1.18	1.5	6.32	31.4**	11.4	1.60	6.4**	5.82	35.4***	19.8	1.61	10.1***	2.05	18.6*
6	21.5	1.09	1.9***	3.17	31.7***	22.8	1.20	4.3***	3.63	37.4***	21.8	1.07	1.5*	1.98	17.4***
7 ^b	18.3	1.25	4.3**	2.33	19.9**	21.1	1.54	10.2***	4.61	43.1***					
8	9.1	1.49	4.3***	6.46	32.8***	6.4	1.71	4.3***	8.49	32.0***	10.9	1.53	5.4***	3.81	23.4***
9	14.1	2.01	12.4**	14.11	64.3***	17.5	1.90	13.6***	25.69	81.0***	29.8	1.55	14.1***	6.67	62.8***
10	5.2	1.30	1.5	18.50	47.3	14.7	1.08	1.1	34.84	83.3***	16.8	1.69	10.4***	5.11	40.2
11	12.8	1.06	0.7**	3.84	26.4**	14.9	1.06	0.9	3.23	24.9***	5.0	0.99	-0.0	3.14	9.5*
$12^{c,d}$						33.8	1.24	7.5**			9.6	0.69	-3.1		
13	9.4	1.32	2.9*	2.87	14.5*	16.6	1.45	7.0***	4.56	36.9***	24.1	1.28	6.4**	3.69	39.8***
14	2.8	1.22	0.6*	8.37	16.0	7.4	1.24	1.8**	9.05	36.2***	32.0	1.40	11.4***	4.38	51.5***

Note. BAC = blood alcohol concentration.

^aBAC analysis restricted to those for whom a BAC estimate was made within 6 hours of injury and who reported no drinking after the event.

 $^{\rm b} {\rm Frequency}$ of ≥ 5 monthly drinking pattern not available.

^cTime between occurrence of event and emergency room arrival not available; thus, time-limited BAC could not be obtained.

^dViolence-related injury data not available.

*P<.05; **P<.01; ***P<.001 (*t* test on AF = 0%).

TABLE 3—Pooled Estimates of Attributable Fractions: All-Cause Injury and Violence-Related Injury

Iniury Category and				Age, y			
Risk Factor	Total, Estimate (95% CI)	Women, Estimate (95% CI)	Men, Estimate (95% CI)	< 30, Estimate (95% CI)	\geq 30, Estimate (95% CI)		
All cause							
Positive BAC	0.021 (0.012, 0.030)***, †††	0.005 (0.001, 0.009)*	0.018 (0.008, 0.029)**, ††	0.014 (0.005, 0.024)**, ††	0.021 (0.010, 0.031)***, †		
Positive self-report	0.058 (0.039, 0.077)***, †††	0.017 (0.006, 0.028)**, ††	0.055 (0.034, 0.076)***, †††	0.047 (0.026, 0.068)***, †††	0.058 (0.036, 0.081)***, †††		
\geq 5 drinks monthly	0.058 (0.035, 0.081)***, †††	0.002 (-0.000, 0.005)	0.046 (0.020, 0.072)**, †††	0.044 (0.022, 0.066)**, †††	0.049 (0.025, 0.074)***, †††		
drinking pattern							
Violence							
Positive BAC	0.276 (0.238, 0.314)***	0.123 (0.069, 0.177)***, †††	0.326 (0.249, 0.403)***, †	0.226 (0.127, 0.326)***, †††	0.260 (0.159, 0.361)***, †††		
Positive self-report	0.425 (0.343, 0.507)***, †††	0.078 (0.037, 0.119)***, †††	0.500 (0.417, 0.583)***, †††	0.423 (0.333, 0.514)***, †††	0.395 (0.284, 0.505)***, †††		
\geq 5 drinks monthly	0.267 (0.188, 0.347)***, †††	0.068 (0.017, 0.119)**, †††	0.298 (0.197, 0.399)***, †††	0.226, (0.147, 0.305)***, †	0.257 (0.136, 0.378)***, †††		
drinking pattern							

Note. BAC = blood alcohol concentration.

Note. CI = confidence interval. If test of homogeneity is rejected (P < .05), the pooled effect size is a random effect.

P*<.05; *P*<.01; ****P*<.001 (for pooled effect size).

P < .05; +P < .01; ++P < .001 (for test of homogeneity).

years and those older than 30 years, perhaps because the mix of injury types and causes obscured age differences in attributable risk for all-cause injuries. Substantially larger attributable risk effect sizes were found for violence-related than for all-cause injuries, especially in the case of men, among whom risk levels reached 43% for those reporting consumption of alcohol before the injury event. It may be that men are more likely than women to overreport their consumption before a violence-related

TABLE 4—Effect Sizes for Attributable Fractions (AFs) of All-Cause and Violence-Related Injuries: Meta-Regression Coefficients (95% Confidence Intervals)

	Fixed Effect	Random Effect
	All-cause injuries	
AF by positive BAC		
Per capita consumption	-0.0005 (-0.0020, 0.0011)	-0.0004 (-0.0023, 0.0015)
Legal drinking age	0.0037 (-0.0006, 0.0043)	0.0019 (-0.0013, 0.0052)
Legal intoxication level	0.12 (0.02, 0.22)*	0.15 (-0.01, 0.31)
Stigmatization	-0.006 (-0.013, 0.0001)	-0.009 (-0.018, 0.001)
Detrimental pattern	0.003 (-0.000, 0.006)	0.005 (0.001, 0.010)*
AF by positive self-report		
Per capita consumption	0.0012 (-0.0009, 0.0032)	0.0027 (-0.0063, 0.0116)
Legal drinking age	0.0065 (0.0032, 0.0098)***	0.0062 (-0.0088, 0.0211)
Legal intoxication level	0.25 (0.06, 0.45)**	0.58 (-0.65, 1.81)
Stigmatization	-0.012 (-0.022, -0.002)*	-0.022 (-0.073, 0.028)
Detrimental pattern	0.008 (0.004, 0.012)***	0.008 (-0.013, 0.029)
AF by \geq 5 monthly drinking pattern		
Per capita consumption	0.0080 (0.0056, 0.0105)***	0.0040 (-0.0050, 0.0129)
Legal drinking age	0.0143 (0.0112, 0.0174)***	0.0140 (0.0102, 0.0179)***
Legal intoxication level	-0.67 (-1.09, -0.26)**	-0.56 (-1.74, 0.62)
Stigmatization	0.058 (0.044, 0.071)***	0.045 (0.026, 0.063)***
Detrimental pattern	0.009 (0.005, 0.013)***	0.006 (-0.016, 0.028)
	Violence-related injuries	
AF by positive self-report		
Per capita consumption	0.010 (0.001, 0.020)*	0.005 (-0.025, 0.035)
Legal drinking age	0.010 (-0.010, 0.030)	-0.005 (-0.063, 0.052)
Legal intoxication level	0.74 (-1.32, 2.81)	-0.46 (-4.11, 5.03)
Stigmatization	0.10 (0.03, 0.17)**	0.01 (-0.17, 0.20)
Detrimental pattern	0.004 (-0.019, 0.028)	0.015 (-0.060, 0.089)
Homicide rate	-0.0007 (-0.0036, 0.0022)	-0.0012 (-0.0092, 0.0066)
AF by $\geq\!5$ monthly drinking pattern		
Per capita consumption	0.018 (0.008, 0.028)**	0.016 (-0.013, 0.044)
Legal drinking age	0.021 (0.002, 0.040)*	-0.012 (-0.068, 0.043)
Legal intoxication level	-2.05 (-4.49, 0.39)	-3.52 (-7.78, 0.74)
Stigmatization	-0.13 (0.05, 0.21)**	0.10 (-0.07, 0.27)
Detrimental pattern	-0.010 (-0.013, 0.032)	-0.041 (-0.034, 0.117)
Homicide rate	-0.001 (-0.008, 0.006)	-0.015 (-0.037, 0.007)

Note. BAC = blood alcohol concentration.

P*<.05; *P*<.01; ****P*<.001.

event, possibly as an excuse for the occurrence of the event. Across the exposure variables, attributable risks for violence-related injuries among women, while larger than those for all-cause injuries, were relatively small, reaching 12% for a positive BAC. Again, few differences in attributable risk were found according to age. While violencerelated injuries are undoubtedly more common among those younger than 30 years, rates of preinjury alcohol consumption appeared to be similar in the 2 age groups. The Canadian study showed that, although violence rates are relatively low in that country (as partially reflected in the homicide rates reported in Table 1), attributable risks across all 3 exposure variables were substantially higher than those in all of the other ER studies. Attributable risks are based on the prevalence of the risk factor (alcohol) and relative risks, and they have little to do with the prevalence of the event (violence-related injury) in the society under consideration.

While the relative risks of all-cause and violence-related injuries were similar for men and women, the attributable risks of both drinking before the injury event and 5+ monthly drinking pattern were greater among men. This is not surprising given that other ER studies have shown that, in comparison with women, men are more likely to have positive BACs, to report drinking before an injury event, and to report heavy drinking before such an event.^{26,27}

In meta-regression analyses focusing on allcause injuries, detrimental drinking patterns were significantly predictive of the effect sizes associated with all 3 exposure variables in the direction expected, with attributable risks greater in those societies in which detrimental patterns were more prevalent. Legal drinking age was also predictive of both drinking before the injury event and 5+ monthly drinking pattern, but in the opposite direction from that expected. While lower legal drinking age reflects greater exposure to alcohol in a particular society, the countries under consideration here with the lowest legal drinking ages, Spain and Italy, were also those in which alcohol is most integrated in everyday functions, with the majority of consumption occurring at mealtimes.

Level of intoxication was positively predictive of effect sizes associated with drinking before the injury event but negatively predictive of effect sizes associated with 5+ monthly drinking pattern. Conversely, stigmatization was negatively predictive of drinking before the injury event but positively predictive of 5+ monthly drinking. Individuals living in societies where alcohol use is less well accepted may be less likely to report drinking before an injury than they are to report heavy drinking in general. Per capita consumption was predictive only of drinking pattern effect sizes, with societies in which per capita consumption was greater showing larger effect sizes.

The data just described suggest that contextual variables explaining heterogeneity of attributable risks for all-cause injuries are not the same in the case of acute and chronic alcohol use; moreover, they may actually work

in opposite directions, possibly as a result of the influence of other contextual variables as well as other unknown variables. Multivariate regression analyses in which variables are entered simultaneously would shed more light on the manner in which these contextual variables work together; given the nature of the present study, such analyses were not possible here.

The relationships between contextual predictors and the effect sizes of attributable fractions were not as strong for violencerelated injuries as they were for all-cause injuries. Fixed effects models showed that per capita consumption and stigmatization were positively predictive of effect sizes associated with both drinking before the injury event and 5+ monthly drinking pattern. Those societies in which alcohol use is less integrated and in which stigmatization of alcohol use is higher are also those in which alcohol is consumed in a more explosive manner, leading to violence-related events.^{13,14}

Legal drinking age was positively predictive of chronic use effect sizes as well. Spain and Italy, the countries with the lowest drinking age and in which alcohol use is most integrated in daily life, were also the 2 societies with the lowest homicide rates. As expected, homicide rate was not found to be predictive of differences in effect sizes for either drinking before the injury event or 5+ monthly drinking pattern. Associations between alcohol use and homicide rates have been found to vary regionally in some countries, such as Russia,²⁸ where such variation is thought to be due to the traditional pattern of consumption of distilled spirits and binge drinking, both of which lead to rapid intoxication. Similar cultural explanations may help explain the heterogeneity in effect sizes observed across the studies analyzed here, but they were beyond the scope of the present investigation.

Detrimental drinking patterns were a significant predictor of attributable risks for allcause injuries but not for violence-related injuries. This result was surprising given that cultures in which alcohol use is characterized by a dominant pattern of infrequent but very heavy consumption also exhibit high rates of mortality from violence related to heavy drinking.^{13,14} It may be that detrimental drinking patterns predict attributable risks for fatal injuries resulting from violence but are less predictive of nonfatal injuries; this topic also requires further exploration.

Attributable risk estimates for drinking before the injury event were based on patients having a positive BAC at the time of their arrival at the ER and reporting drinking in the 6 hours before the occurrence of the injury. The congruence of these 2 measures is dependent on the rapidity with which patients reach the ER after the injury and may be related, in part, to injury severity.²⁹ Since BAC analyses were restricted to individuals for whom an estimate could be made within 6 hours of the injury and who reported no drinking after the injury, the attributable risks estimated on the basis of this measure are probably conservative. Attributable risks based on self-reported consumption within 6 hours of an injury may also be problematic in terms of accuracy, since a relatively small amount of alcohol consumed the full 6 hours before the injury would probably have little influence of the occurrence of the event. Attributable risk estimates do not take into account differences in intensities of exposure (e.g., amount of alcohol consumed or time between most recent consumption and injury), and total amount of alcohol consumed during the 6-hour period would certainly be of importance in determining injury risks.⁵

The attributable risk estimates described here were based on individuals seeking treatment in an ER relative to individuals using the ER for other reasons. Previous analyses have shown a tendency for injured patients seeking ER treatment to be heavier drinkers than injured patients seeking other types of treatment or no treatment.³⁰ Since it has also been shown that, in general, non-injured ER patients are more frequently heavy drinkers,³¹ basing attributable risks of injury associated with alcohol use on ER populations may result in conservative estimates. In addition, although the ERs analyzed here were representative of those in the catchment areas covered by each study, and the studies included were reflective of the respective societies on which they focused, this group of studies cannot be considered to be representative of the situation in entire countries.

As mentioned, similar study designs and methodologies were used in collecting the

data analyzed in ERCAAP, decreasing the likelihood of problems frequently encountered in meta-analyses such as differences in methods and quality. The primary data available on individual patients, although collected over a 17-year period (1984-2001), were not subject to publication bias and allowed for a unified analysis of the same set of variables. Notwithstanding the limitations of this study, our findings suggest that attributable risks associated with drinking before an injury event may be greater than those associated with overall drinking patterns, especially in the case of violence-related injuries. Our findings also show that the attributable risks of alcohol use associated with violence-related injuries are substantially greater than those associated with all-cause injuries.

The present data support previous findings indicating the important role of alcohol consumption in violence-related injuries. Although differences in attributable risk effect sizes differed in relation to the integration of alcohol in a particular culture, these data may inform future global burden of disease estimates. It is highly likely that attributable risks associated with both drinking before an injury and drinking patterns vary according to the type and cause of injury, and future research should address risks related to alcohol consumption in the case of specific causes of injury in addition to injuries related to violence. Future research should also examine contextual variables simultaneously as a means of further explaining variations in attributable risks across societies and cultures.

About the Authors

The authors are with the Alcohol Research Group, Public Health Institute, Berkeley, Calif.

Requests for reprints should be sent to Cheryl J. Cherpitel, DrPH, Alcohol Research Group, 2000 Hearst Ave, Berkeley, CA 94709 (e-mail: ccherpitel@arg.org).

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Contributors

C.J. Cherpitel conceived the study and was involved in all aspects of data analysis and writing. Y. Ye assisted in data analysis and interpretation. J. Bond assisted in data analysis and interpretation and in the writing of the article.

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Human Participant Protection

This study was approved by the institutional review board of the Public Health Institute. All participants provided informed consent before taking part in the study.

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