



Multi-level analysis of causal attribution of injury to alcohol and modifying effects: Data from two international emergency room projects^{☆, ☆☆}

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Abstract

Although alcohol consumption and injury has received a great deal of attention in the literature, less is known about patient's causal attribution of the injury event to their drinking or factors which modify attribution. Hierarchical linear modeling is used to analyze the relationships of the volume of alcohol consumed prior to injury and feeling drunk at the time of the event with causal attribution, as well as the association of aggregate individual-level and socio-cultural variables on these relationships. Data analyzed are from 1955 ER patients who reported drinking prior to injury included in 35 ERs from 24 studies covering 15 countries from the combined Emergency Room Collaborative Alcohol Analysis Project (ERCAAP) and the WHO Collaborative Study on Alcohol and Injuries. Half of those patients drinking prior to injury attributed a causal association of their injury with alcohol consumption, but the rate of causal attribution varied significantly across studies. When controlling for gender and age, the volume of alcohol consumed and feeling drunk (controlling for volume) were both significantly predictive of attribution and this did not vary across studies. Those who drink at least weekly were less likely to attribute causality at a low volume level, but more likely at high volume levels than less frequent drinkers. Attribution of causality was also less likely at low volume levels in those societies with low detrimental drinking patterns, but more likely at high volume levels or when feeling drunk compared to societies with high detrimental drinking patterns. These findings have important implications for brief intervention in the ER if motivation to change drinking behavior is greater among those attributing a causal association of their drinking with injury.

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1. Introduction

A substantial literature exists which documents the association of alcohol consumption with injury occurrence, the bulk of which comes from studies conducted in hospital emergency rooms (ERs) (Cherpitel, 1993; Romelsjö, 1995). The mechanism

linking alcohol consumption to injury is thought to be associated with a number of factors in combination (one of which is alcohol), with various time relationships to each other and to injury (Romelsjö, 1995). While the association of alcohol consumption and subsequent injury is related, in part, to diminished coordination and balance, increased reaction time, and impaired attention, perception and judgment at the time of injury, it may also be related to the residual effects of drinking (hang-over effects) operating for a period of time following drinking (Cherpitel et al., 1998). Alcohol may also be considered a contributory cause of injury, which, together with other contributory causes, provides a sufficient cause for the event. In this regard, much may also be learned regarding the alcohol-injury link from patients' attribution of their drinking to injury occurrence. While

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relatively little research has been conducted on causal attribution of injury to alcohol consumption, the perceived role of alcohol in the injury event by the patient may be important in tailoring effective intervention and prevention strategies.

Analysis of drinking-in-the-injury event variables, some of which might be thought related to a patient's causal attribution of drinking and injury, have been analyzed in a few ER studies, and include the number of drinks consumed prior to injury, the time lapsed between drinking and the event, and feeling drunk at the time of injury (Cherpitel, 1996, 1998; Cherpitel et al., 1993). These studies have found a substantial proportion of injured report feeling drunk at the time of the event and attribute a causal association of their drinking with the event (Cherpitel, 1996). One study found that while 63% of those who reported feeling drunk at the time of injury attributed a causal association of their drinking with the event, 34% of those who had been drinking but did not feel drunk also attributed a similar causal association (Stephens, 1987).

Causal attribution of injury to drinking in these ER studies has been found to vary across ethnic groups (Cherpitel, 1998) and regions (Cherpitel, 1997) in the U.S., as well as across other cultures and countries (Cherpitel, 1999; Cherpitel et al., 1993). In ER samples from both the U.S. (California) and Spain, almost all of the injured patients who reported feeling very drunk at the time of the event attributed a causal association of their drinking with injury, while only 60% of their counterparts in a sample from Mexico made the same attribution (Cherpitel et al., 1993). Both California and Spain can be considered "wetter" cultures with respect to drinking, reflecting a prevailing pattern of frequent but less heavy usual drinking, while Mexico is considered "drier", where frequent lighter drinking is less common and infrequent heavier, fiesta, drinking more typical (Room, 1989, personal communication).

In addition to drinking-in-the-event factors, usual drinking patterns may also play a part in the likelihood of attribution of injury to drinking. For example, usual heavier or more frequent drinkers may be less likely to attribute a causal association of their drinking with injury than lighter drinkers, at a given volume level; prolonged use of alcohol over time may provide a protective effect if an individual has developed a level of tolerance that allows him to engage in activities while drinking which could result in more severe injury for a less experienced drinker. Other research suggests that social (Cherpitel, 1997; Clark and Hilton, 1991) as well as cultural factors (MacAndrew and Edgerton, 1969) mediate perceptions about the role of alcohol in relation to problem events including injury. For example, drinking and drunkenness may be considered excuses for otherwise socially unacceptable behavior in some cultures (MacAndrew and Edgerton, 1969), resulting in the increased likelihood of attributing injury to alcohol consumption in societies in which alcohol is less well-integrated into the culture. Causal attribution of alcohol with injury may thus be related to both individual-level drinking on the part of the patient as well as to societal-level factors including typical drinking patterns of a culture.

To explore drinking-in-the-event and usual drinking pattern variables related to patients' causal attribution of injury to alcohol, a recent meta-analysis of 13 ER studies covering seven

countries included in the Emergency Room Collaborative Alcohol Analysis Project (ERCAAP) (Cherpitel et al., 2003c) was undertaken. Blood alcohol concentration (BAC), the volume of alcohol consumed prior to injury, and feeling drunk at the time of the event were all significantly predictive of causal attribution, but heterogeneous across studies. Among contextual variables used to predict heterogeneity of the relationship between drinking and causal attribution, detrimental drinking pattern, which is a measure comprised of three societal indicators expected to affect the impact of a given volume of drinking (heavy drinking occasions, drinking with meals, and drinking in public places) (Rehm et al., 2001, 2003b), was found to be most important. A higher volume of consumption was found to be associated with a relatively higher probability of causal attribution in societies with low detrimental drinking patterns compared to those with high detrimental patterns.

Although estimates of the between-study variances are provided in meta-analysis, a formal statistical model is not developed to carry out inferences based on these variances. In order to analytically test hypotheses of the random variation in the rate of causal attribution, controlling for level of consumption, analyses must be performed in a multi-level context, where the rate of causal attribution and its relationship with alcohol consumption can be modeled together, along with specific distributional forms of the error distribution for the study-level variances. Additionally, these analyses expand upon earlier work (Cherpitel et al., 2003c) by explicitly focusing on both the intercept (the rate of causal attribution) as well the slope (the relationship between reported drinking and causal attribution) in a simultaneous model, as well as focusing on individual and study-level variables that modify these relationships. In addition to the role that the intercept and the consumption slope parameters play, it is also important to consider other possible influences on a patient's causal attribution of the accident to their drinking. Such individual-level variables will also be incorporated into the multi-level models as fixed effects parameters.

Reported here is analysis, using hierarchical linear modeling (HLM), of data from 35 emergency room (ER) sites in 24 ER studies across 15 countries comprising the combined ERCAAP study (Cherpitel et al., 2003a) and the WHO Collaborative Study on Alcohol and Injuries (WHO Collaborative Study Group, 2004). Among other aims, the WHO study was designed to quantify the incidence of alcohol-related injuries in emergency rooms across different cultural settings. The additional study sites from inclusion of the 12 WHO studies with the ERCAAP studies makes this HLM analysis of causal attribution of alcohol and injury technically feasible.

Individual-level variables are analyzed, which include age, gender, drinking patterns and event variables (volume consumed, feeling drunk, time lapsed between the last drink and injury), as well as aggregated individual-level variables within a study (volume, and percentage of wine, beer and spirits consumption), and socio-cultural variables (per capita consumption of total ethanol; percentage of per capita consumption, separately for spirits, wine, and beer; legal drinking age; maximum legal BAC level for driving; gross national product (GNP)), stigmatization of alcohol use, and detrimental drinking pattern,

all of which may be thought related to the cultural positioning and integration of alcohol in society (Room, 1989, personal communication; Room and Mäkelä, 2000).

This paper examines the following among injured patients who reported drinking within 6 h prior to injury: (1) the rate of causal attribution and the relationship of volume consumed as well as feeling drunk with causal attribution; (2) the association of aggregated individual-level and socio-cultural variables with the relationship between volume as well as feeling drunk with causal attribution; and (3) whether the relationship between usual consumption and causal attribution of injury to alcohol is influenced by individuals' drinking patterns. In addition, the probability of causal attribution is examined by the time lapsed between drinking and the event, and for traffic-related injuries and violence-related injuries.

2. Methods

2.1. Samples

The ERCAAP data include 24 ERs in 13 ER studies across seven countries (Cherpitel et al., 2003a, 2004), while the WHO Collaborative Study on Alcohol and Injuries includes 11 ER studies in 11 countries (WHO Collaborative Study Group, 2004) (see Table 1). The WHO study collected data only from those

patients arriving at the ER within 6 h of the injury event leading to the emergency room visit. The ERCAAP data analyzed here were selected to include only those meeting this 6-h arrival criterion. Data from ERs in both projects were collected using a similar methodology developed by Cherpitel (1989). Probability samples of patients 18 years and older, reflecting consecutive arrivals to the ER across a representative range of each shift for each day of the week, were approached with an informed consent to participate in the study. Studies where patients were disproportionately sampled were weighted to ensure patients equally represented within a study. Patients were interviewed and an estimate of blood alcohol concentration (BAC) obtained as soon as possible after admission to the emergency room. Completion rates for the ERCAAP studies averaged 72%, while those for the WHO studies averaged 91%. Reasons for non-interviews in both projects included refusal, incapacitation, leaving prior to completing the interview, the patient was in police custody, and language barriers. Patients who were too severely injured or ill to be approached in the ER were followed into the hospital and interviewed once their condition had stabilized in all of the ERCAAP studies and in South Africa. A cohort of trained interviewers obtained the BAC estimate and administered a 25-min questionnaire that included items, among others, regarding the type and cause of injury, drinking in the 6 h prior to the injury, amount consumed, feeling drunk at the time of

Table 1

Sample characteristics and rate of casual attribution among those consumed alcohol before injury across ER studies in the Emergency Room Collaborative Analysis Project (ERCAAP) and the World Health Organization Collaborative Study on Alcohol and Injuries (WHO)

ID	Year	No. of ERs in the study	Number who reported drinking within 6 h before injury	% of causal attribution ^{a,b}	% of male ^a	% of age ≥30 ^a	
ERCAAP							
1	San Francisco, CA	1984–1985	1	99	40	82	55
2	Jackson, MS	1992	1	40	40	82	42
3	Santa Clara, CA	1995–1996	1	20	70	95	80
4	Mexico City, Mexico	1986	8	263	61	95	38
5	Acapulco, Mexico	1987	3	71	75	77	43
6	Pachuca, Mexico	1996–1997	3	84	77	90	50
7	Edmonton (Alta.), Canada	1989	1	30	50	83	40
8	Quebec City, Canada	1989	1	9	67	78	33
9	Fremantle, Australia	1997	1	99	49	78	37
10	Barcelona, Spain	1987	1	163	29	76	44
11	Mar del Plata, Argentina	2001	1	57	18	84	36
12	Warsaw, Poland	2002	1	15	67	87	79
13	Sosnowiec, Poland	2002–2003	1	49	39	78	69
WHO							
14	Mar del Plata, Argentina	2001	1	105	47	84	47
15	Minsk, Belarus	2001	1	136	64	76	48
16	São Paulo, Brazil	2001	1	56	41	87	39
17	Orangeville (Ont.), Canada	2002	1	13	23	85	85
18	Hunan, China	2001	1	97	57	95	73
19	Prague, Czech Republic	2001	1	40	30	80	55
20	Mexico City, Mexico	2002	1	78	58	88	22
21	Maputo, Mozambique	2001	1	73	44	85	55
22	Auckland, New Zealand	2000	1	55	47	73	55
23	Cape Town, South Africa	2001	1	238	73	82	47
24	Malmö, Sweden	2001	1	65	32	74	62
Total			35	1955	53	84	48

^a Among those reporting drinking prior to the injury event.

^b Percent of those who attributed a causal association between their drinking and the injury event.

injury, and believing the injury would have happened had he not been drinking (causal attribution), quantity and frequency of usual drinking and higher consumption times and demographic characteristics.

The analysis on causal attribution was restricted to those injury patients who reported consuming any beverage containing alcohol within the 6 h before the injury and reported their beverage-specific volume of consumption for this time period. One ERCAAP study was removed from analysis since the time between injury and arrival at the ER was not available (Italy), and three U.S. studies and one WHO study (India) were excluded because the question regarding causal attribution was not asked (India) or asked only of those who reported feeling drunk at the time of the injury event (U.S.).

2.2. Measures

2.2.1. Individual-level ER data

The measure of *causal attribution* of injury to in-the-event drinking is based on the following question among those who reported drinking within 6 h of the injury event, “Do you think it (the injury) would have happened even if you had not been drinking?” Those who answered “no” or “not sure” were coded as attributing the injury to their in-the-event drinking. Those reporting they were not sure whether the injury would have happened if they had not been drinking comprised approximately 14% of the sample. An alternate definition of causal attribution was also explored where “not sure” was coded as not attributing their injury to in-the-event drinking. As an additional measure, patients who reported drinking within the 6 h prior to the event were asked if they were “*feeling drunk*” when the injury happened. The *time lapsed* between the last drink and the injury event in hours was also analyzed.

The *volume of alcohol consumed within the 6 h prior to the injury* was measured by summing the patient’s self-reported beverage-specific number of drinks consumed for wine, beer, and spirits. In the ERCAAP studies, one drink was considered to be a 4-oz glass of wine, a 12-oz can or bottle of beer or 1 oz of hard liquor, each of which has been presumed to contain approximately .5 oz of absolute alcohol. In the WHO studies, amount of alcohol consumed was assessed in equivalent units for a detailed list of different types of alcoholic beverages and were also summed to create an overall consumption measure. All volume measures were converted to standard drinks prior to the creation of an in-the-event volume measure. log volume was used in the analysis as reported volume was somewhat skewed and model fit was improved using log transformations.

BAC was estimated in all but the Canadian ERCAAP studies using a breathalyzer (the Alco-Sensor III breathalyzer and in New Zealand the Alcotec AR1005), which provides estimates that are highly correlated with chemical analysis of blood (Gibb et al., 1984). In Alberta and Quebec, BAC was estimated from urine samples that were assessed for ethanol using KDA enzymatic testing, and standardized to the unit measure quantifying BAC estimated from breath samples.

Quantity and frequency of drinking was obtained from a series of questions having to do with the frequency of any drink-

ing and the frequency of consuming five or more drinks at a time during the last year. For analysis here variables related to *weekly drinking of any amount*, and drinking five or more drinks at a time at least monthly (*5+ monthly*) were analyzed. In a separate question, patients were also asked the amount *usually* consumed when they drank. A dichotomous *heavy drinking* measure was developed using this *usual* question that took the value 1 if the respondent reported usually having five or more drinks when they drank for men and four or more drinks for women (regardless of the frequency of such drinking occasions) and 0 if their usual drinking quantity per occasion was less than this amount.

2.2.2. Study-level contextual data

Study-level contextual variables were used to explain the between-study variation of the relationship between both the amount of alcohol consumed in the 6 h prior to injury (log volume) as well as whether or not the patient felt drunk at the time of the injury with the likelihood of attributing a causal attribution association of the injury to drinking. Contextual variables included both (1) aggregated individual-level variables within a study and (2) socio-cultural variables pertaining to a region or country. The aggregated variables for this analysis consisted of average log volume 6 h prior to the injury across patients within each study separately and the percentage of consumption of wine, beer and spirits separately. The average overall log volume reflects the cross-study differences in drinking behaviors before an acute event like injury that might not be captured by average consumption over the past year. For example, in countries like Mexico with high abstention rates and infrequent heavy drinking, a large average log volume is likely to be observed although the per capita consumption is low. The proportion of beverage-specific consumption might also suggest differential relationships between beverage choice and attribution of injury to alcohol consumption.

Due to differences in drinking culture across studies, socio-cultural variable data assessing per capita consumption of ethanol; the percentage of per capita consumption, separately for spirits, wine, and beer; legal drinking age and maximum legal BAC level for driving; were obtained for the locale of each site and time period during which the ER data were collected. Per capita consumption has been found to be positively associated with integration of alcohol and closely linked to the proportion of abstention in a society (Rehm et al., 2004). A higher ethanol concentration is found in a given fluid volume of spirits compared to beer or wine, leading to potentially higher BAC levels which increase the potential for risky outcomes including injury (Pridemore, 2002). Legal BAC limits can be seen as an indicator of how much a society is concerned with alcohol and road safety, while legal drinking age reflects the concern about alcohol as a problematic substance for youth. Since drinking age and maximum legal BAC level for driving are not legally defined in China, it was assigned the most liberal values, 16 for drinking age and .10% for intoxication. The gross national product (GNP) as a measure of purchasing power parity is also included in analyses as a potential confounder, as GNP has been linked to both alcohol consumption and injury rate (Peden et al., 2004; World

Health Organization, 2002) and has been used in prior studies of alcohol-related injury (Cherpitel et al., *in press*).

Stigmatization of alcohol use was obtained from the collaborator for each of the ER studies and was based on their belief regarding the degree to which alcohol use is accepted within a society and the degree which drinking prior to injury may have been under-reported. This variable was found to be a significant predictor of alcohol-related injury in prior meta-analysis of the ERCAAP data (Cherpitel et al., 2003b). Detrimental drinking pattern within a society was based on survey data and a survey of key informants selected by the World Health Organization for each country (Rehm et al., 2001). Responses of key informants were evaluated on validity (Rehm et al., 2001) and ratings were analyzed using optimal scaling analysis (Bijleveld and Van Der Burg, 1998) with one dimension identified and labeled “detrimental impact” (Rehm et al., 2001). The measure includes indicators that would be expected to affect the impact of volume of drinking: heavy drinking occasions (based on the quantity per occasion, proportion of daily drinking, getting drunk, and festive drinking), drinking with meals, and drinking in public places. Detrimental impact scores range from 1 to 4, with higher score indicating a higher postulated detrimental effect of the same per capita consumption of alcohol (Rehm et al., 2003a,b). Since distinct regional variations in drinking patterns have been found within country (as in rates of abstinence), Canada, Quebec was assigned a lower detrimental pattern level than the country as a whole, while Alberta was assigned a higher detrimental pattern. In the U.S., California sites were assigned a lower detrimental pattern level than the country as a whole, while Mississippi was assigned a higher detrimental pattern.

2.3. Analysis

Primary data from each of the ER studies were cleaned and merged into a single data file. Analyses here include current drinkers reporting to the ER with an injury and who reported drinking within 6 h prior to injury. In order to estimate the probability that a drinker attributed their injury to drinking across studies, as well as to explore the relationship between causal attribution and both the amount consumed prior to injury (log volume) and whether the respondent felt drunk at the time of injury, five separate multi-level logistic regressions models were estimated. To study the moderating influence of additional variables in the relationship between volume and rates of causal attribution, two types of variables will be explored. The first type of variable is study-level, or contextual variables, while the second type is individual-level, or patient-level variables.

All hierarchical generalized linear models were estimated in the MLwiN program Version 1.1 (Snijders and Bosker, 1999; Rasbash et al., 2000). The study was chosen as the second-level unit as it retained regional variation within country while avoiding over-representation of ERs from the same region. Five models were used to explore these relationships (specific details for each of these models can be found in the Appendix A). More detail on estimation of model parameters and associated infer-

ences and tests of variance of random effects can be found in Raudenbush and Bryk (2002).

2.3.1. Model 1: intercept only model

The probability of causal attribution was modeled with only a random intercept term such that the intercept was allowed to vary across studies. The pooled estimate of the intercept represents the average rate of causal attribution across studies. In each of the subsequent models with additional predictors, the intercept should be interpreted as the rate of causal attribution at values of zero for all other predictors. A significance test for the variance estimate of the random intercept assessed whether the average rate of causal attribution varied significantly across studies.

2.3.2. Model 2: random intercept and random slope (log volume) model

The probability of causal attribution was predicted from patients' gender, age and their log volume of consumption 6 h prior to injury in order to explore the relationship between causal attribution and the amount of alcohol consumed. Here, the intercept and slope of log volume were assumed to be random across studies and the variance and covariance of the random effects were estimated.

2.3.3. Model 3: random intercept and random slope (feeling drunk) model

Similar to Model 2, self-report feeling drunk was used to predict causal attribution, when controlling gender and age, as well as log volume. Here, the intercept and the slope for the feeling drunk predictor were assumed to be random across studies, while the log volume slope was assumed to be a fixed effect in order to reduce the number of parameters within the model.

2.3.4. Model 4: random intercept and random slope (log volume) model with study-level predictors

This model was built upon Model 2, with study-level variables used to predict both the variation of the intercept and log volume slope across studies. Study-level variables included both those derived from the aggregation of patient-level variables within studies and contextual variables measuring the socio-cultural characteristics of the regions where the ER studies were conducted.

2.3.5. Model 5: random intercept and random slope (feeling drunk) model with study-level predictors

This model was built upon Model 3, with study-level variables used to predict the variation of intercept and feeling drunk coefficients across studies, similar to Model 4.

2.4. Patient-level variables

The previously described models only explore the influence of study-level characteristics in moderating the relationships between volume and causal attribution. Models are also examined which explore whether patient-level drinking patterns and heavy drinking also moderate this relationship (Table 4). Other individual-level variables explored include time lapsed between

drinking and injury, traffic-related injuries and violence-related injury.

3. Results

3.1. Model estimation without study-level predictors

Table 2 presents results of Models 1–3. The pooled estimate of the average rate of causal attribution can be derived from Model 1 by taking the inverse logit transformation of the fixed effect parameter intercept estimate. About half (49.8%) of the injury patients who drank in the event attributed their injury to alcohol consumption. The rate estimates varied across studies, as seen from the significance test based on the intercept variance ($p < .01$).

In Model 2, self-reported log volume significantly predicted the likelihood of causal attribution ($p < .001$) while gender and age were not significant predictors. Here also, significant cross-study variation was found for the rate of causal attribution across studies ($p < .05$) but not for the relationship of log volume to causal attribution. Feeling drunk also significantly predicted attribution of injury to alcohol ($p < .001$), after controlling for gender, age, and log volume (Model 3). When feeling drunk was entered, log volume continued to be a significant predictor ($p < .001$). The rate of causal attribution was still found to vary significantly across studies ($p < .05$), while the slope for feeling drunk was not found to vary.

3.2. Model estimation with study-level predictors

Study-level aggregate and contextual variables were used to predict the variation in the intercept and slope of log volume (Model 4) and feeling drunk (Model 5) across studies (Table 3). Although significance tests in Models 2 and 3 (Table 2) indicated that variation in the volume and feeling drunk slopes was not significant at the .05 level, study-level variables were still entered to predict their variances, as (a) variation in the log volume and feeling drunk slopes are of primary interest and (b)

tests of heterogeneity have been found to lack power (especially when a smaller number of level-2 (study-level) units are available and coefficients are close to significance, $p < .10$), resulting in acceptance of homogeneity when heterogeneity may exist across study-level variables (Sutton et al., 2000). Aggregate and socio-cultural study-level variables were entered (univariately) into separate models as a first step for both log volume and feeling drunk. Significant predictors in these separate models for either the intercept or slope were then entered simultaneously.

In the model to explain the variation of relationship between amount of alcohol consumed and the probability of causal attribution when gender, age, and alcohol consumption were controlled, aggregated log volume and detrimental drinking pattern positively predicted the rate of causal attribution across sites and detrimental drinking patterns negatively predicted the relationship of log volume and causal attribution, both univariately and simultaneously. In the simultaneous model, the estimate of the random variance for the intercept and slope dropped to .250 and .058 from .810 and .117, respectively (from Model 2).

The detrimental drinking pattern measure proved to be a highly significant predictor for both the intercept and slope, as seen in Model 4. Interestingly, its positive association with the rate of causal attribution (at zero consumption) and negative association with the relationship of log volume and causal attribution deserve more careful consideration. To illustrate further, a graph of the predicted probability of causal attribution (logit form, vertical axis) against the individual (log) volume of consumption (horizontal axis) by studies and by high or low detrimental drinking patterns can be seen in Fig. 1. The dotted lines show the linear relationship for each study individually, where variation can be seen in both the intercept and slope. The two solid lines show the average linear relationship across those studies with high compared to low detrimental drinking patterns. The intercept for the high detrimental drinking pattern studies was larger than that for the low detrimental drinking pattern studies, indicating a higher rate of causal attribution at low levels of alcohol consumption in those studies with low detri-

Table 2
Multilevel generalized model predicting logit probability of causal attribution among those who consumed alcohol within 6 h before injury

	Model 1	Model 2	Model 3
Fix effect: β (se)			
Intercept	-.008(.135)	-1.056(.261)***	-1.120(.231)***
Male	NA	.140(.134)	.230(.142)
Age >30	NA	.087(.099)	.140(.104)
log volume	NA	.528(.103)***	.254(.074)***
Feeling drunk	NA	NA	.901(.199)***
Random effect: σ^2 (se)			
Intercept	.357(.125)**	.810(.368)*	.421(.176)*
Slope of log volume	NA	.117(.069) ⁺	NA ^a
Slope of feeling drunk	NA	NA	.455(.238) ⁺

^a Forced to be fixed effect.

* $p < .05$ (Wald test).

** $p < .01$ (Wald test).

*** $p < .001$ (Wald test).

⁺ $p < .10$ (Wald test).

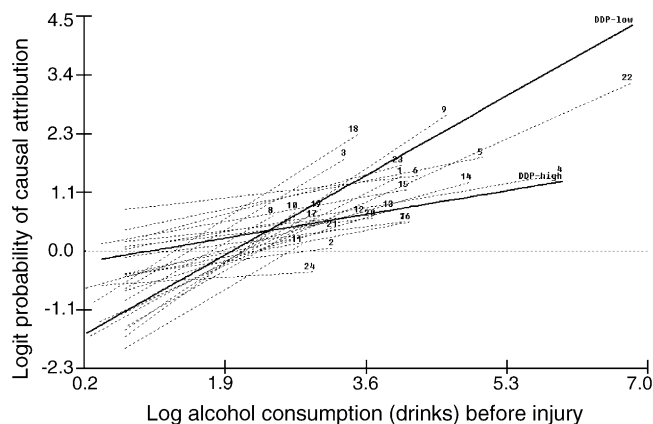


Fig. 1. Relationship of probability of causal attribution and volume of consumption, by studies and detrimental drinking patterns (DDP). Note: Dashed lines—risk curves by study (see study ID in Table 1); horizontal line at 0—OR = 1; solid lines—risk curves by detrimental drinking pattern (DDP): low = 1 and 2, high = 3 and 4.

Table 3

Parameter estimates of contextual variables predicting the cross-study variation of intercept and slope, built on Model 2 (for log volume) and Model 3 (for feeling drunk) of Table 2

	log volume (Model 4)		Feeling drunk (Model 5)	
	Intercept	Slope of log volume	Intercept	Slope of feeling drunk
Aggregate variables entered univariately				
Group mean of log volume	1.948(.599)**	-.548(.294)	1.316(.391)***	-1.034(.487)*
Percentage of wine consumption	-3.415(1.757)	.763(.860)	-2.337(1.079)*	1.443(1.385)
Percentage of beer consumption	-.543(1.167)	.662(.484)	-.663(.802)	1.769(.826)*
Percentage of spirit consumption	1.787(1.057)	-.887(.444)	1.481(.697)*	-2.155(.704)**
Socio-cultural variables entered univariately				
Per capita consumption	.001(.064)	-.019(.030)	-.022(.044)	-.007(.053)
Percentage spirits per capita	.276(1.200)	.388(.531)	.441(.814)	.581(.930)
Percentage wine per capita	-1.998(1.193)	.431(.580)	-1.582(.740)*	1.148(.939)
Detrimental drinking pattern	.739(.184)***	-.291(.090)**	.411(.125)***	-.523(.148)***
Legal intoxication level	2.98(8.68)	2.32(3.99)	6.14(5.71)	4.46(7.24)
Legal drinking age	.168(.185)	-.152(.080)	-.022(.122)	-.123(.146)
Level of alcohol use stigmatized	.194(.488)	-.318(.214)	-.313(.328)	-.167(.396)
Gross national product (PPP)	-.035(.028)	.005(.013)	-.029(.019)	.025(.023)
Significant predictors above entered simultaneously				
Group mean of log volume	.837(.348)*	Not entered	1.148(.526)*	-.028(.523)
Detrimental drinking pattern	.586(.183)**	-.275(.088)**	.107(.156)	-.483(.179)**
Percentage of wine consumption	Not entered	Not entered	.778(1.494)	Not entered
Percentage of beer consumption	Not entered	Not entered	Not entered	.987(1.250)
Percentage of spirit consumption	Not entered	Not entered	.253(.629)	.039(1.316)
Percentage wine per capita	Not entered	Not entered	-1.123(.894)	Not entered
Random effect: $\sigma^2(se)$.250(.194)	.058(.050)	.138(.083)	.019(.082)

* $p < .05$ (Wald test).

** $p < .01$ (Wald test).

*** $p < .001$ (Wald test).

mental drinking patterns. However, the slope of the relationship between log volume and probability of causal attribution was much flatter for high detrimental drinking pattern studies and steeper for those with lower detrimental drinking patterns, indicating that low detrimental drinking pattern studies were less likely to attribute a causal association of drinking and injury at low consumption levels than high detrimental drinking pattern studies but more likely to attribute an association at higher consumption levels.

Study-level variables were also used to explain the variation of relationship between feeling drunk and the probability of causal attribution when gender, age, and alcohol consumption were controlled, together with the intercept, as presented in Model 5 (Table 3). Although a number of study variables significantly predicted variation of either the intercept or slope when entered separately, the only significant predictors in the simultaneous models were aggregated log volume of consumption (for the intercept) and detrimental drinking pattern (for the slope). In those areas with higher detrimental pattern, patients who were feeling drunk were less likely to attribute their injury to drinking.

3.3. Sensitivity analyses

To validate results in Table 3, two types of sensitivity analyses were performed. First, estimates of BAC were used to replace both individual-level as well as average aggregate-level

self-reported alcohol consumption prior to injury. In these analyses, aggregate study-level average BAC was not a significant predictor for the intercepts in either Model 4 or 5, while detrimental drinking pattern significantly predicted the intercept and was negatively associated with the slope for both models. In the second analysis, those who answered “not sure” for causal attribution were coded as not attributing their injury to their in-the-event drinking. All of the main results presented above were again observed.

3.4. Model estimation with patient-level drinking patterns and event characteristics

In a separately analysis, we explored the modifying effect of the individual's usual drinking pattern on the relationship between drinking prior to the event and causal attribution. Usual drinking variables were evaluated separately for weekly drinking, 5+ monthly, and heavy drinking. As seen in Table 4, only weekly drinking was a significant moderator ($p < .001$), with those who drank any alcohol at least weekly less likely to attribute their injury to drinking at low volume levels and more likely to report a causal association at high volume levels, compared with those who drank less often than weekly.

The likelihood of causal attribution was also evaluated in relation to the time lapsed between drinking and the injury event as well as for two specific types of injury—those related to motor vehicle crashes and those related to violence (Table 4).

Table 4
Log odds ratios for individual drinking and injury event characteristics on the probability of causal attribution, controlling for gender and age

Variable	β (se)
Modifying effect of usual drinking patterns on the relationship of alcohol consumption before injury with causal attribution	
Sub-models	
(1) Any weekly drinking	-.668(.255)*
log volume \times any weekly drinking	.447(.123)***
(2) Heavy drinking	.518(.268)
log volume \times heavy drinking	-.142(.139)
(3) Monthly_5+	-.229(.257)
log volume \times monthly_5+	.246(.132)
Drinking and injury event characteristics on the probability of causal attribution controlling for log volume	
Sub-models	
(1) Hours between drinking and injury	-.064(.036)
(2) Traffic injury vs. other injuries	-.097(.148)
Violence injury vs. other injuries	-.149(.115)

* $p < .05$ (Wald test).

*** $p < .001$ (Wald test).

Although patients whose drinking occurred in closer proximity to the injury appeared to be more likely to attribute their injury to drinking, the relationship was not significant, when the amount consumed during the 6-h period was controlled. This finding appears to validate the use of a 6-h period for the reporting of alcohol consumption before injury. In addition, Table 4 shows that patients sustaining injuries related to either traffic crashes or violence were no more likely to attribute a causal association of their injury to drinking than those with other types of injury, when alcohol consumption was controlled.

4. Discussion

Half of those patients drinking prior to injury attributed a causal association of their injury with alcohol consumption, but the rate of causal attribution varied significantly across studies. Not surprising, when controlling for gender and age, the volume of alcohol consumed and feeling drunk (controlling for volume) were both significantly predictive of attribution and this did not vary (at the .05 significance level) across studies. In these analyses, moderation of the effect of consumption in the event (log volume) was carried out both for patient-level as well as for contextual-level variables. For the patient-level variables, moderation is represented by direct interactions with log volume whereas for contextual-level variables, the study-level coefficients indicate moderation. Here, both the intercept and the slope were found to be moderated by the patients' usual drinking patterns as well as by contextual variables representing the regional drinking culture.

The likelihood of attributing injury to drinking at a given consumption level appears to be moderated mainly by the frequency of usual consumption. Those who drink at least weekly (and for whom alcohol may be more integrated with everyday life than less frequent drinkers) are less likely to attribute their injury to drinking at a low volume level compared to those who drink less often than weekly. However, at higher volume levels, they are more likely to make such attribution, possibly due to a lower tolerance to heavy drinking compared to less frequent

drinkers who may drink more heavily on fewer occasions. Similarly, in those societies with low detrimental drinking patterns, where drinking with meals is common while public and heavy drinking is less prevalent, individuals are less likely to report a causal attribution at low volume levels, again possibly due to the higher integration of alcohol with their daily lives, while more likely to make attribution at higher volume levels or when feeling drunk. It is also possible that in societies in which alcohol is more highly integrated, causal attribution may less likely be denied (as well as the number of drinks consumed prior to injury or reporting feeling drunk at the time) than in a society in which alcohol is less well accepted, and where punitive measures may be incurred by patients sustaining injuries related to drinking.

Interestingly, the time lapsed between the last drink and injury was not predictive of causal attribution when the amount consumed during the 6-h period was controlled, possibly due to the fact that the bulk of drinking prior to injury occurs within close proximity to the event, as shown in prior research of ER patients (Stephens, 1987). Additionally, when controlling for volume, causal attribution was no more likely to be made for the two causes of injury in which a stronger association of drinking and the event might be expected: traffic injuries and injuries related to violence. It might be thought that those injured in violence-related events may be more likely to attribute a causal association as an excuse for their behavior, while those injured in traffic crashes may be less likely to attribute causality due to the potential legal ramifications and stigma (in some cultures) associated with drinking and driving; however, neither was found to be the case.

Findings reported here extend those from previous analysis of the ERCAAP and WHO data of the association of individual with contextual variables on injury and alcohol-related injury to the patient's attributing a causal association of their drinking with the injury event. Analysis reported here supports that from the previous meta-analysis of the ERCAAP data only which found a robust positive association of acute alcohol use on causal attribution of drinking to injury that was negatively associated with the degree to which a society exhibits harmful drinking

patterns (Cherpitel et al., 2003c). Analysis here of an additional 12 studies representing eight additional countries confirms the association of contextual drinking pattern with the probability of a patient's causal attribution, but also takes into account in this association the amount of alcohol consumed prior to injury, and additionally explores modifying effects of usual drinking patterns on causal attribution, as well as the time lapsed between drinking and the event and potential differential effects by cause of injury.

It should be noted that although patient samples were drawn to be representative of their respective ERs in both the ERCAAP and WHO studies, samples cannot be considered to be representative beyond the ER facilities where the data were collected. Restriction of the sample to those arriving at the ER within 6 h after injury may also pose some limitations to generalizability of the sample, since immediacy of arrival is likely related to severity of injury as well as to other factors that may possibly be related to the likelihood of attributing a causal association of alcohol and injury. Additionally, although contextual variables reflect the same time period during which the respective ER data were collected, they may not adequately represent the geographic level relevant to the specific ER study, since they are generally based on aggregate-level statistics, ranging from county-level to country-level data.

A major strength of the study, however, is that although data collection spanned a 19-year period (1984–2002), in both the ERCAAP and WHO projects comparable study designs and data collection procedures were used with nearly uniform rigor, using a standard questionnaire that helped assure comparability of items across studies. Given the diversity of the countries analyzed and the relatively large and comparable samples across studies, findings here suggest that the volume of alcohol consumed prior to injury and whether or not the patient was feeling drunk at the time, regardless of volume consumed, are important individual-level predictors of attributing a causal association of drinking and injury, but attribution varies by individual drinking patterns as well as by societal drinking patterns. It is important to note, however, that since the data were collected over a protracted period of time, during which there were substantial changes in drinking cultures in some countries (for example, Eastern Europe including Poland), findings related to associations between individual-level and contextual variables found here may require care in interpretation and translation to intervention and prevention strategies.

Nevertheless, these findings may have important implications for effectiveness of intervention strategies to reduce alcohol-related injuries among those treated in emergency rooms. Attribution of negative outcomes, including injury, to alcohol consumption may be one predictor of a patient's degree of readiness to change drinking behavior, and motivation to change may be greater among those attributing a causal association of their drinking with injury. However, the success of motivational-based interventions may be diminished in those societies exhibiting more detrimental patterns of drinking. The data suggest that a brief intervention linking drinking to the injury may be less effective among those drinking at higher levels prior to injury in countries in which alcohol is less well integrated into society

and in which heavy bouts leading to intoxication and drinking in public places is more common than in those countries displaying lower detrimental scores, in which alcohol is more often consumed with meals and heavy drinking occasions are fewer. While the targeting of intoxicated patients in those countries in which drinking occasions leading to intoxication are less common may prove most effective in a reduction in alcohol-related injuries, intervention and prevention strategies related to culturally-specific drinking patterns is an area of research in need of further research.

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Appendix A

Model 1:

$$\text{Level 1: } \text{logit}(y_{ij}) = \beta_{0j}$$

Level 2:

$$\beta_{0j} = \beta_0 + \mu_{0j}$$

$$[\mu_{0j}] \sim N([0], [\sigma_{\mu_0}^2])$$

where y_{ij} is the probability of causal attribution. Estimates of β_0 and $\sigma_{\mu_0}^2$ are presented in Table 2.

Model 2:

$$\text{Level 1: } \text{logit}(y_{ij}) = \beta_{0j} + \beta_{1j}x_{1ij} + \beta_{2j}\text{sex}_{ij} + \beta_{3j}\text{age}_{ij}$$

Level 2:

$$\beta_{0j} = \beta_0 + \mu_{0j}$$

$$\beta_{1j} = \beta_1 + \mu_{1j}$$

$$\begin{bmatrix} \mu_{0j} \\ \mu_{1j} \end{bmatrix} \sim N \left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{\mu 0}^2 & \sigma_{\mu 01} \\ \sigma_{\mu 01} & \sigma_{\mu 1}^2 \end{bmatrix} \right)$$

where x_{1ij} is the logarithm form of individual self-reported volume of consumption before injury. Of these parameters, estimates of β_0 (intercept), β_1 (volume), β_2 (sex), β_3 (age), $\sigma_{\mu 0}^2$ (intercept variance) and $\sigma_{\mu 1}^2$ (volume) are presented in Table 2.

Model 3:

$$\text{Level 1: } \text{logit}(y_{ij}) = \beta_{0j} + \beta_1 x_{1ij} + \beta_2 \text{sex}_{ij} + \beta_3 \text{age}_{ij} + \beta_{4j} \text{feel_drunk}_{ij}$$

Level 2:

$$\beta_{0j} = \beta_0 + \mu_{0j}$$

$$\beta_{4j} = \beta_4 + \mu_{4j}$$

$$\begin{bmatrix} \mu_{0j} \\ \mu_{4j} \end{bmatrix} \sim N \left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{\mu 0}^2 & \sigma_{\mu 04} \\ \sigma_{\mu 04} & \sigma_{\mu 4}^2 \end{bmatrix} \right)$$

Estimates of β_0 , β_1 , β_2 , β_3 , β_4 (feel drunk), $\sigma_{\mu 0}^2$ and $\sigma_{\mu 4}^2$ (feel drunk variance) are presented in Table 2.

Model 4:

Level 1 is the same as Model 2, but level 2 is:

$$\beta_{0j} = \beta_0 + \beta_5 \text{L2variable}_j + \mu_{0j}$$

$$\beta_{1j} = \beta_1 + \beta_6 \text{L2variable}_j + \mu_{1j}$$

$$\begin{bmatrix} \mu_{0j} \\ \mu_{1j} \end{bmatrix} \sim N \left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{\mu 0}^2 & \sigma_{\mu 01} \\ \sigma_{\mu 01} & \sigma_{\mu 1}^2 \end{bmatrix} \right)$$

where L2 variable refers to study-level (level 2) aggregate or contextual variables.

Estimates of β_5 and β_6 , as well as $\sigma_{\mu 0}^2$ and $\sigma_{\mu 1}^2$ when significant L2 variables were entered simultaneously, are presented in Table 3.

Model 5:

Level 1 is the same as Model 3, but level 2 is:

$$\beta_{0j} = \beta_0 + \beta_5 \text{L2variable}_j + \mu_{0j}$$

$$\beta_{4j} = \beta_4 + \beta_7 \text{L2variable}_j + \mu_{4j}$$

$$\begin{bmatrix} \mu_{0j} \\ \mu_{4j} \end{bmatrix} \sim N \left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_{\mu 0}^2 & \sigma_{\mu 04} \\ \sigma_{\mu 04} & \sigma_{\mu 4}^2 \end{bmatrix} \right)$$

Estimates of β_5 and β_7 , as well as $\sigma_{\mu 0}^2$ and $\sigma_{\mu 1}^2$ when significant L2 variables were entered simultaneously, are presented in Table 3.

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