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ALCOHOL ATTRIBUTABLE FRACTION FOR INJURY MORBIDITY FROM THE DOSE-RESPONSE RELATIONSHIP OF ACUTE ALCOHOL CONSUMPTION: EMERGENCY DEPARTMENT DATA FROM 18 COUNTRIES

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Abstract

Aims—To calculate the alcohol-attributable fraction (AAF) of injury morbidity by volume of consumption prior to injury based on newly reported relative risk (RR) estimates.

Design—AAF estimates based on the dose-response RR estimates obtained from previous pair-matched case-crossover fractional polynomial analysis of mean volume in volume categories were calculated from the prevalence of drinking prior to injury in each volume category.

Setting—Thirty-seven emergency departments (EDs) across 18 countries.

Participants—Probability samples of patients, with equal representation of each shift for each day of the week, totaling 14,026 who arrived at the ED within six hours of injury from ED studies conducted between 2001 and 2011.

Measurements—AAF was analyzed by gender, age (18–30; >30), cause of injury (traffic, assault, fall, other), and country detrimental drinking pattern (DDP).

Declarations of Interest: None

Findings—For the EDs analyzed, 16.4% of all injuries were estimated to be attributable to alcohol, and the AAF did not vary by age but was over twice as large for males (20.6%; 19.3–21.8) than for females (8.6%; 7.5–9.7%). While females were at greater risk of injury than males at higher volume levels, lower prevalence of women drinking at higher levels contributed to overall lower AAF for women. Assault-related injuries showed the largest AAF (44.1%; 37.6–42.6). AAF was slightly higher for injuries from falls (14.3%; 12.9–15.7) than motor vehicle crashes (11.1%; 9.3–12.9). AAF was higher in those countries with a DDP of 3 (18.6; 17.5–19.7) and 4 (19.4%; 17.3–21.6) than those with a DDP of 2 (12.1%; 10.5–13.5).

Conclusions—AAF estimates are higher for males than females, for violence-related injuries compared to other types of injury, and for countries with more detrimental drinking patterns compared to those with less detrimental patterns.

INTRODUCTION

Injuries, globally, constitute a large proportion of alcohol-attributable Disability-Adjusted Life Years (DALYs) (33.2%) and alcohol-attributable mortality (24.4%) [1]. The relative risk (RR) of injury from alcohol consumption is one important component in estimating the proportion of injuries attributable to alcohol consumption, but has been derived primarily from mortality data, and has also been based on chronic rather than acute consumption. While chronic consumption is a useful measure for determining the RR for chronic diseases, it is not as useful for acute events such as injuries, where drinking in the event is a more relevant measure [2]. RR estimates have also assumed a uniform risk across demographic and injury characteristics and have not examined the dose-response relationship of alcohol and injury. Two meta-analytic reviews of alcohol consumption and the dose-response risk of injury have been reported. One review of usual alcohol consumption and 15 disease conditions found a dose-response relationship between RR of injury and grams of alcohol consumed per day [3]. The second review of acute consumption found a non-linear increase in risk of injury with increasing alcohol consumption [4]. Pattern of consumption has also become increasingly recognized as important in relation to injury occurrence. A review of meta-analyses on the relationship of volume and pattern of alcohol consumption with disease conditions including injury found average volume and drinking pattern, each, causally linked to both intentional and unintentional injuries [2]; this finding was supported by a meta-analysis of alcohol-related injury from emergency department (ED) data across 19 countries [5].

Calculation of alcohol-attributable fraction (AAF) of injury, or the proportion of injury which would be eliminated in the absence of the risk factor, has typically not used RR estimates based on acute consumption in the event or on cause of injury, although a distinction is made between motor vehicle and non-motor vehicle crashes in the Global Burden of Disease GBD) estimates [6]. One exception is a study in a Swiss ED in which AAF was calculated for low, medium and high levels of alcohol consumption prior to injury across several mechanisms of injury [7]. A recent study, based on probability samples of ED patients across 18 countries, updated previous RR estimates of injury morbidity, examining the dose-response relationship of acute alcohol consumption and injury overall and by gender, age, cause of injury and country-level drinking pattern [8]. This study, using the fractional polynomial approach to modeling the dose-response relationship of alcohol and injury, based on pair-matched case-crossover analysis of patients' self-reported number of drinks consumed within the six hours prior to injury, found risk to increase with increasing volume of consumption but the relationship was not uniform across demographic or injury subgroups.

Building on this, the present paper incorporates these RR estimates in calculating the AAF of injury morbidity by volume of consumption prior to injury for gender, age, cause of injury and country drinking pattern subgroups. These analyses are important for refining estimates of AAF of injury morbidity, a key priority identified by the World Health Assembly [9] and for informing the GBD, highly important since much of this burden is avoidable [10, 11].

METHODS

Samples

Data analyzed include 9 of the EDs included in the 2001–02 WHO collaborative study on alcohol and injuries (Argentina, Belarus, Brazil, Canada, Czech Republic, India, Mexico, New Zealand, Sweden) [12] and additional ED data, collected using the WHO study protocol [13], from Switzerland (2006–07), Ireland (2003–04), China and Korea (2009), and from six countries in the Americas: Dominican Republic, Guatemala, Guyana, Nicaragua, Panama (2010–11) and Canada (2009) [14, 15]. More detailed information on datasets can be found in the Appendix Table 1 (described below) and reported elsewhere [5, 8].

In all studies, probability samples of patients aged 18 years and older arriving consecutively at the ED within six hours of the injury event were obtained, with equal representation of all hours of the day and days of the week. Selected patients were approached with an informed consent to participate in the study, following which a 25-minute structured questionnaire [13] was administrated. Completion rates averaged 87% across all studies (range 59% to 100%). Reasons for non-interviews included refusing, incapacitation, leaving prior to completing the interview, in police custody, and language barriers. Patients who were too severely injured to be approached in the ED were followed into the hospital and interviewed once their condition had stabilized.

Individual measures

Patients were asked about the cause of injury bringing them to the ED (categorized as traffic, assault-related violence, falls, other, since these were the largest injury groupings), drinking within six hours prior to the injury event, and drinking during the same six-hour period the previous week. Both time periods included the beverage-specific number, size and alcohol concentrations of drinks. For each ED study, efforts were made to include all alcoholic beverage types commonly consumed locally with container sizes and alcohol concentrations. Total volume was obtained by summing across all beverage types and converting to the number of drinks, each containing 16 ml (12.8 gms) of ethanol for a given time period.

Country-level drinking pattern

Country-level drinking pattern was determined, based on a country's detrimental drinking pattern (DDP) score which is an indicator of the "detrimental impact" at a given level of alcohol consumption on health and other drinking-related harms. This measure was developed and validated by WHO from indicators of heavy drinking occasions, drinking with meals and drinking in public places, and ranges in score from 1 (least detrimental) to 4 (most detrimental) [16, 17]

Data analysis

RR of injury from drinking was calculated based on pair-matched case-crossover analysis [18, 19] in which each injured patient's alcohol consumption within six hours prior to injury was compared to his or her own alcohol consumption during the same time period the previous week. This method theoretically reduces confounding of the alcohol-injury relationship by controlling for stable risk factors. Fractional polynomial modeling [20] was then used to model the relationship between continuous volume of consumption and the odds of injury, using the conditional logistic model: logit(Prob(injury))=$b_0+b_1x^p+b_2x^q$ (or $b_0+b_1x^p+b_2x^p(\ln x)$ if $p=q$) where p and q are chosen from −2, −1, −0.5, 0, 0.5, 1, 2 and 3 ($x^0=\ln(x)$) and x is some transformed form of volume consumption [8]. Fractional polynomial modeling has several advantages compared to traditional approaches to modeling the dose-response relationship; it is more powerful and flexible than linear models and generate smoother and more efficient estimates than the categorical step-function approach [8], making it more appropriate for RR and the AAF estimation.

To estimate how many of the total number of injuries are attributable to a specific volume of alcohol consumption, we calculated specific volume alcohol-attributable fraction (SVAAF) (volume categories shown in Table 1, left hand column) based on the RR estimate for the mean volume of that category and the prevalence of drinking six hours prior to injury in that category: $AAF_i=P_i(Alcohol|injury)\times(1-\frac{1}{RR_i})$, [21] where i refers to a volume category. The total 1 population AAF is thus the summation of SVAAFs:

$AAF_{pop}=\sum P_i(Alcohol|injury)\times(1-\frac{1}{RR_i})$ ([21] formula 4 and see [22] for a heuristic illustration). Bootstrap confidence intervals (CIs) were also estimated based on 1000 replications. The bootstrap approach accounts for variation in both the prevalence rates and function forms fitted for RR estimates, and the CIs derived were found to be highly consistent with those using the analytical approach (as in formula 7 in [21]). SVAAFs and population AAF were estimated for the total sample and separately by gender, by age (18–30, >30), by cause of injury and by DDP. These age categories were chosen because of the number of respondents and their age distribution across studies, and because many countries have found younger adults aged 18–30 to be more frequent heavy drinkers than those older. Comparisons of point-wise CIs were used as the primary method for assessing differences in SVAAF and population AAF across subgroups. Missing data on the volume consumed among those who reported drinking prior to injury were included in AAF calculations and were, conservatively, assigned the estimated RR at 1 drink for the respective subgroup.

Of the total 14,136 injury patients interviewed across the 37 EDs, 110 were missing data on the whether they had consumed any alcohol within 6 hours before injury, the key exposure variable, leading to a total 14,026 valid cases for analysis. See Appendix Table 1 for the sample size by study. Note that the number of valid cases used in this analysis is larger than that in the prior study [8] estimating RR which required valid alcohol volume measures in both the case (6 hours before injury) and control (the same time last week) periods. Also note that given a small number of patients were missing data on gender, age and cause of injury, the total number of cases for subgroup analysis does not sum to the total valid N. Also shown in the appendices are figures of all RR curves generated using the fractional polynomial approach with their functional forms, together with RRs from the categorical step-function approach.

RESULTS

Table 1 shows estimates of the proportion of ED injuries attributable to specific-volume (SVAAF) and the total population AAF, using the combined sample as a whole. SVAAF estimates were fairly consistent up to > 30 drinks, at which point the prevalence of drinking prior to injury and the associated RR was lower than for other volume categories. Summing across dose-response levels, the prevalence of drinking prior to injury was 20.2%, with a total ER *population AAF* of 16.4%. Among the 20.2% exposed (drinking prior to injury) alcohol was an attributable factor for injury in 81% (16.4/20.2; the *exposed AAF*), underscoring the fact that not all exposed injuries are attributable to alcohol, but attribution depends on the RR of injury from drinking.

Table 2 shows SVAAF and population AAF estimates separately by gender and by age. Although RR estimates were higher for females at volume levels above 4 drinks compared to males, due to the smaller prevalence of females drinking at higher volume levels the estimated population AAF for females (8.6%) was less than half of that for males (20.6%). Little difference in population AAF estimates was found for those aged 30 or younger (17.6%) compared to those older (15.5%)

In Table 3, AAF estimates are shown for each cause of injury. The estimated total population AAF was highest for assault-related injury (40.1%), and of the 44.2% of patients who reported drinking prior to injury, 90.7% of their injuries were attributable to alcohol. Population AAF estimates were marginally higher for falls (14.3%) than for motor vehicle crashes (11.1%) or other causes of injury (9.8%).

Table 4 shows AAF estimates by country-level DDP. Since only one country had a DDP score of 1 (Switzerland), estimates are only reported for countries with higher DDP scores. Population AAF estimates were marginally higher (19.4%) for those countries with the highest DDP score (Belarus, Guatemala, Mexico, Nicaragua) compared to countries with a DDP of 3 (18.6%). Among patients who reported drinking prior to injury in countries with a DDP of 3 or 4, 85% and 86.6% of their injuries, respectively, were attributable to alcohol, compared to only 71% of those from those countries with a DDP score of 2.

DISCUSSION

This paper incorporates recent RR estimates [8] based on the dose-response relationship of drinking prior to injury to estimate the AAF of injury morbidity overall and by gender, age, cause of injury and country drinking pattern. Along with the prevalence of the risk factor (drinking prior to the injury event), the RR of injury from alcohol consumption is one important component in estimating the AAF -- that proportion of injury which would be eliminated in the absence of the risk factor. Refined RR estimates have allowed us to estimate the AAF based on the dose-response relationship of alcohol and injury and the heterogeneity of the alcohol-injury risk relationship across demographic and injury subgroups, not previously reported in the literature. Based on a dose-response relationship in the entire sample, the estimated population AAF was 16.4%. A prior analysis estimated the AAF in the U.S. general population, based on any drinking prior to an injury for which treatment was obtained in an ED in the last year, as 2.96% [23], similar to that found at lower levels of consumption here. In that study, the prevalence of drinking prior to injury was based on recall over the past 12 months, however, and was considerably lower (6.45%) than that found here (20.2%). AAF estimates in the present study did not vary greatly by age, but were over twice as large for males (20.6%) than females (8.6%). While females appeared to be at greater risk of injury than males at higher volume levels, the lower prevalence of women drinking at these higher levels contributed to an overall lower AAF estimate for women compared to men, although for both genders, of those who reported drinking before injury, alcohol was an attributable factor for injury (the AAF among those exposed) in over 80%.

Not surprising, assault-related injuries showed the largest estimated population AAFs (44.2%) with alcohol as an attributable factor in over 90% of those who reported drinking prior to the event. Estimated population AAF was slightly higher for injuries from falls (14.3%) than motor vehicle crashes (11.1%). These estimates are similar to those from a Swiss ED study using case-control data to calculate AAF at high, medium and low levels of consumption. Based on drinking within 6 hours prior to injury in that study, AAF was 36% for violence-related injury, 10% for transport injuries and 14% for fall injuries [7]. Unfortunately, in the present study limited numbers did not permit AAF estimation across causes of injury by gender or age, and this may be an area for future research.

AAF estimates here pertain to injury morbidity and not to fatality, for which RR from drinking is higher [2]. In the present paper only 11% of the injuries related to motor vehicle crashes were attributable to alcohol, compare to 35% of fatal motor vehicle crashes in the United States, as measured by a blood alcohol concentration of 0.08 or above [24], and prior comparisons of ED injury data with coroner reports for the same time and jurisdiction have found alcohol involvement to be 6 times greater in fatal than non-fatal injury [25, 26].

Estimated AAFs were higher in those countries with the most detrimental drinking patterns (19.4% and 18.6% for DDP-4 and DDP-3 countries, respectively) and which exhibit a high prevalence of heavy drinking [16], compared to DDP-2 countries (12.0%), and among those reporting drinking prior to the event, over 85% of the injuries in DDP-3 and DDP-4 countries were attributable to alcohol. DDP has been found to predict alcohol-related injury in other ED studies [5, 27], and in the earlier study [28], DDP was found to positively predict AAF estimates. Countries with a similar DDP have been found to cluster in a given region or area; those in Central or Eastern Europe and those in Central America tend to exhibit more detrimental patterns of drinking than those in Western Europe, and these findings suggest that country-level drinking pattern is important to consider in estimating the AAF of injury morbidity.

Differences in AAF estimates for injury morbidity between countries and regions may also be due to other differences in addition to DDP, however. For example, cultural differences between societies in the relationship of alcohol consumption and injury involving both the physical and social context of drinking, such as the proportion of heavy drinking occasions in the overall volume of consumption [2], drunken comportment [29], and disinhibition with alcohol as an excuse for otherwise socially unacceptable behavior [30], may all play an important part, especially for intentional injuries where cultural differences in the meaning of drinking are especially important [31]. Additionally, alcohol control policies which differ greatly, sometimes in the same country, have also been found to predict alcohol-related injury across some of the same countries as those analyzed here [5]. Differences in the composition of injury cases may also differ across countries, with some having a higher prevalence of motor vehicle crashes than others, for example, and we were not able to perform more detailed analyses of AAF by DDP within cause of injury subgroups.

A strength of this study is that RR estimates used in AAF calculations were based on the dose-response relationship of the amount of alcohol consumed prior to the injury event, generated from case-crossover analysis which controls for stable factors such as demographic characteristics, as well as usual drinking patterns which have been found to be predictive of alcohol-related injury in previous analyses [5, 27], and risk taking disposition. The case-crossover design is not without potential bias, however, and findings have been mixed in terms of recall of one’s drinking the previous week [32, 33]. Additionally, potential bias may also arise from the context of drinking in the injury event compared to the previous week [34]. Since context may be independently correlated with both alcohol use and injury risk, bias may occur if context is represented unequally in case and control period comparisons; e.g., to estimate the RR of injury due to drinking as a driver, driving in the control period must be taken into account, since the patient would not be at risk of a traffic crash related to his drinking if he had not been driving, resulting in a lower RR of motor vehicular injury from drinking.

AAF estimates here are substantially larger than those found from case-control analysis [28]. Similar gender differences, but with much smaller AAF estimates (5.5% vs. 1.7% for males and females, respectively) were found in a prior case-control analysis based on drinking prior to injury in the ED, which did not take the dose-response relationship into account [28], although AAF for assault-related injury (42.5%) was similar to that found here. RR estimates of injury from drinking in the six hours prior to the event generated from the pair-matched case-crossover design, as used here, have been found elsewhere to be larger than those generated from the case-control design, controlling for gender and age [35]. The case-control design may be biased, however, when using non-injured patients as controls, since non-injured ED patients have been found more likely to be heavier drinkers than those in the general population from which they come, resulting in conservative RR estimates for injury [36].

While patient samples in all studies are representative of their respective EDs, they are not necessarily representative of a broader area or jurisdiction, nor are injured patients seeking treatment in the ED necessarily representative of all injured patients, an important consideration in interpreting AAFs. Prior analysis in the general population has found that those seeking treatment in the ED are heavier drinkers than those either seeking other types of treatment or not seeking treatment for their injury [37], and estimates of both the RR of injury based on drinking prior to injury and the AAF have also been found to be larger for those seeking ED treatment in the general population compared to those reporting seeking other kinds of treatment or not seeking treatment [23].

The combined sample of patients consists of a varying number of respondents across countries or regions, and heterogeneity in individual and country-level characteristics may not be fully represented. Subgroup analysis assumes a constant effect for the population within the subgroup, and subgroup differences in the dose-response relationship may also be due to differences in the composition of injuries. However, sample size did not permit AAF analysis for cause of injury by demographic characteristics or by DDP, as noted. Additionally, ED samples were collected over a span of over 10 years, and combining data in analysis assumes a constant relationship overtime. However, temporal heterogeneity may be less an issue compared to variation across individuals and countries.

Data here are based on patients' self-report of their own drinking, and the extent to which someone else's drinking may have contributed to the patient's injury is not known. A 14-country analysis of assault-related injury, including many of these same EDs, found that AAF for assault-related injury was 24% based on the patients' belief that their own drinking was causally related to the event (a subset of the 49% who reported drinking prior to the event), and AAF increased to 39% (a 62% increase) when perpetrators of the event who had been drinking and for whom the patients believed their drinking had been causally related to the event were also included [38].

Limitations related to analytical approaches are also worth noting. Relative risk estimates from the fractional polynomial approach become unstable at high levels of consumption given the sparse data; e.g., for the total sample risk of injury increases exponentially as the volume of consumption increases, but then decreases at the high end consumption. Whether the observed decline in risk is real or a result of measurement error is a question yet to be answered [8]. Alternatively, a threshold effect can be assumed by capping the volume of consumption at 30 drinks. Another limitation is that we were unable to allow for potential heterogeneity of effect across countries and EDs in our model, and failure to adjust for autocorrelation for clustered sample data suggests that CIs may be underestimated.

Despite these limitations, ED samples presently provide the most reliable and comprehensive source of injury morbidity data, and analysis here of new dose-response estimates of AAF of injury for demographic and injury subgroups suggest that attribution of injury to alcohol should be based on gender-, cause- and country-specific calculations. The proportion of injuries related to alcohol consumption was found to vary by cause of injury, and these data are especially important for informing the GBD estimates related to injury cause, which are presently divided into motor vehicular and non-motor vehicular crashes in the GBD. Findings here suggest that non-motor vehicular injuries should further be separated into at least assault-related injury, those related to falls and those resulting from other causes when estimating the burden of disease attributable to alcohol consumption. Uniform AAF estimates across countries or regions with differing drinking patterns may also not be appropriate, and is an area of research requiring future attention. Findings here indicate that the proportion of injury attributable to a specific volume level is greater at lower levels of consumption prior to injury, where prevalence of drinking is higher although RR is lower, compared to higher levels of consumption where RR is higher but prevalence lower, and underscore the importance of interventions aimed at lower levels of consumption as well as those aimed at higher levels of drinking.

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Table 1

Alcohol Attributable Fraction (AAF) estimates and 95% Confidence Intervals (CIs) by levels of volume consumed before injury (total injury sample n = 14,026)

Alcohol intake before injury[1] (in number of drinks)	**n**	**Prevalence of drinking (%)[2]**	**Relative Risks (RR) [3]**	**Attributable Fraction (%)[4]**	**95% CIs**
No drinking	11,181				
≤2	512	3.63	2.58	2.22	(1.90, 2.55)
2.1–4	447	3.14	4.64	2.46	(2.20, 2.72)
4.1–6	446	3.16	6.67	2.69	(2.43, 2.94)
6.1–8	314	2.26	8.27	1.99	(1.75, 2.23)
8.1–10	313	2.23	9.78	2.00	(1.77, 2.23)
10.1–15	354	2.52	11.40	2.30	(2.05, 2.55)
15.1–30	278	1.99	13.32	1.84	(1.61, 2.07)
>30	81	0.57	9.56	0.51	(0.25, 0.77)
Missing[5]	100	0.71	2.26	0.39	(0.30, 0.48)
Total[6]	14,026	20.2		16.4	(15.5, 17.3)

[1] The volume levels are mutual exclusive, thus ≤2 refer to volume larger than zero but no more than two drinks, and 2.1–4 refer to volume larger than two drinks but no more than 4 drinks

[2] Prevalence rates do not match exactly with sample Ns, as several studies were weighted

[3] Relative risks are the fractional polynomial estimates based on the mean volume of each volume category, for example, 1.25 drinks for ≤2 volume category

[4] SVAAF = $P_i*(1-1/RR_i)$, in which P_i is the prevalence of drinking at given volume level among total injured patients (cases) and RR_i the relative risk of injury for given volume level compared to no drinking.

[5] Those who reported drinking before injury but didn't report specific volume of consumption. As a conservative estimate, RR for 1 drink is used for this missing group

[6] The total includes the sum of the prevalence and SVAAF across dose levels

Table 2

Alcohol Attributable Fraction (AAF) estimates and 95% Confidence Intervals (CIs) by levels of volume consumed before injury, by gender and age[1]

Alcohol intake before injury (in number of drinks)	n	Prevalence of drinking (%)	Relative Risks (RR)	Attributable Fraction (%)	95% CIs
MALES (n=9,119)					
No drinking	6,804				
≤2	390	4.23	2.60	2.60	(2.14, 3.07)
2.1–4	346	3.72	4.57	2.91	(2.56, 3.26)
4.1–6	346	3.75	6.47	3.17	(2.83, 3.52)
6.1–8	238	2.65	8.01	2.32	(2.00, 2.63)
8.1–10	268	2.93	9.48	2.62	(2.31, 2.93)
10.1–15	312	3.41	11.03	3.10	(2.74, 3.47)
15.1–30	255	2.81	12.76	2.59	(2.26, 2.92)
>30	78	0.84	8.88	0.74	(0.20, 1.29)
Missing	82	0.89	2.24	0.49	(0.36, 0.63)
Total	9,119	25.2		20.6	(19.3, 21.8)
FEMALES (n=4,893)					
No drinking	4,364				
≤2	122	2.52	2.33	1.44	(0.93, 1.95)
2.1–4	101	2.05	4.59	1.61	(1.24, 1.97)
4.1–6	100	2.06	7.50	1.78	(1.39, 2.17)
6.1–8	76	1.56	10.14	1.40	(1.08, 1.73)
8.1–10	45	0.93	13.12	0.86	(0.61, 1.10)
10.1–15	42	0.85	16.91	0.80	(0.55, 1.05)
>15	26	0.53	32.67	0.51	(0.30, 0.72)
Missing	17	0.34	2.16	0.18	(0.09, 0.28)
Total	4,893	10.8		8.6	(7.5, 9.7)
AGE 18–30 (n=6,189)					

Alcohol intake before injury (in number of drinks)	n	Prevalence of drinking (%)	Relative Risks (RR)	Attributable Fraction (%)	95% CIs
No drinking	4,856				
≤2	211	3.38	2.67	2.11	(1.64, 2.59)
2.1–4	215	3.41	4.83	2.71	(2.28, 3.14)
4.1–6	208	3.31	6.88	2.83	(2.44, 3.23)
6.1–8	155	2.54	8.37	2.23	(1.85, 2.62)
8.1–10	141	2.27	9.63	2.03	(1.69, 2.37)
10.1–15	179	2.88	10.84	2.62	(2.23, 3.01)
15.1–30	137	2.24	11.78	2.05	(1.69, 2.41)
>30	41	0.64	7.68	0.56	(0.36, 0.76)
Missing	46	0.73	2.34	0.41	(0.27, 0.56)
Total	6,189	21.4		17.6	(16.3, 18.8)
		AGE 31+ (n=7,768)			
No drinking	6,270				
≤2	299	3.84	2.47	2.29	(1.80, 2.78)
2.1–4	231	2.93	4.43	2.27	(1.93, 2.62)
4.1–6	237	3.05	6.48	2.58	(2.23, 2.93)
6.1–8	159	2.07	8.25	1.82	(1.52, 2.11)
8.1–10	168	2.17	10.14	1.96	(1.64, 2.27)
10.1–15	174	2.24	12.41	2.06	(1.73, 2.39)
15.1–30	140	1.80	15.97	1.69	(1.40, 1.98)
>30	40	0.51	19.31	0.48	(0.27, 0.70)
Missing	50	0.65	2.18	0.35	(0.23, 0.47)
Total	7,768	19.3		15.5	(14.4, 16.6)

[1] See footnotes in Table 1

Table 3

Alcohol Attributable Fraction (AAF) estimates and 95% Confidence Intervals (CIs) by levels of volume consumed before injury, by cause of injury[1]

Alcohol intake before injury (in number of drinks)	n	Prevalence of drinking (%)	Relative Risks (RR)	Attributable Fraction (%)	95% CIs
Motor Vehicle Crash (n=2,686)					
No drinking	2,272				
2	88	3.29	1.97	1.61	(0.82, 2.41)
2.1–4	68	2.46	3.20	1.69	(1.18, 2.20)
4.1–6	70	2.58	4.41	2.00	(1.44, 2.55)
6.1–8	37	1.36	5.67	1.12	(0.75, 1.49)
8.1–10	28	1.02	6.91	0.88	(0.53, 1.22)
10.1–15	37	1.36	8.85	1.21	(0.79, 1.63)
15.1–30	44	1.63	12.72	1.50	(1.02,1.98)
>30	20	0.73	41.28	0.71	(0.40, 1.03)
Missing	22	0.82	1.79	0.36	(0.13, 0.59)
Total	2,686	15.3		11.1	(9.3, 12.9)
Assault-related Injury (n=2,109)					
No drinking	1,174				
2	117	5.50	4.39	4.24	(3.24, 5.25)
2.1–4	133	6.26	8.32	5.51	(4.46, 6.56)
4.1–6	143	6.75	12.25	6.20	(5.20, 7.21)
6.1–8	118	5.65	15.19	5.27	(4.30, 6.24)
8.1–10	113	5.35	18.32	5.06	(4.15, 5.96)
10.1–15	138	6.55	21.76	6.25	(5.22, 7.28)
15.1–30	102	4.85	27.44	4.68	(3.78, 5.57)
>30	41	1.91	44.53	1.87	(1.27, 2.46)
Missing	30	1.40	3.46	1.00	(0.60, 1.40)
Total	2,109	44.2		40.1	(37.6, 42.6)

Alcohol intake before injury (in number of drinks)	n	Prevalence of drinking (%)	Relative Risks (RR)	Attributable Fraction (%)	95% CIs
Fall Injury (n=4,250)					
No drinking	3,461				
2	140	3.26	1.95	1.59	(1.05, 2.14)
2.1–4	124	2.85	3.52	2.04	(1.59, 2.50)
4.1–6	138	3.23	5.60	2.65	(2.16, 3.14)
6.1–8	83	2.02	7.63	1.75	(1.36, 2.15)
8.1–10	85	2.01	9.96	1.81	(1.41, 2.20)
10.1–15	100	2.34	13.00	2.16	(1.71, 2.61)
15.1–30	84	1.96	17.28	1.85	(1.44, 2.25)
>30	12	0.27	7.92	0.24	(0.06, 0.41)
Missing	23	0.53	1.76	0.23	(0.11, 0.35)
Total	4,250	18.5		14.3	(12.9, 15.7)
Other Injury (n=4,848)					
No drinking	4,203				
2	164	3.37	2.42	1.98	(1.35, 2.61)
2.1–4	115	2.35	3.80	1.73	(1.36, 2.11)
4.1–6	91	1.87	4.76	1.48	(1.15, 1.80)
6.1–8	69	1.42	5.37	1.16	(0.87, 1.44)
8.1–10	79	1.64	5.74	1.35	(1.01, 1.69)
10.1–15	58	1.21	6.03	1.01	(0.71, 1.31)
>15	50	1.09	4.23	0.83	(0.16, 1.50)
Missing	19	0.39	2.24	0.21	(0.10, 0.33)
Total	4,848	13.3		9.8	(8.4, 11.2)

[1] See footnotes in Table 1

Table 4

Alcohol Attributable Fraction (AAF) estimates and 95% Confidence Intervals (CIs) by levels of volume consumed before injury, by DDP[1,2]

Alcohol intake before injury (in number of drinks)	n	Prevalence of drinking (%)	Relative Risks (RR)	Attributable Fraction (%)	95% CIs
		DDP=2 (n=5,133)			
No drinking	4,247				
≤2	275	5.30	2.08	2.76	(1.92, 3.59)
2.1–4	191	3.59	3.51	2.57	(2.10, 3.03)
4.1–6	129	2.46	4.91	1.96	(1.58, 2.33)
6.1–8	78	1.59	5.97	1.32	(1.01, 1.63)
8.1–10	62	1.20	6.99	1.03	(0.77, 1.30)
10.1–15	49	0.94	8.52	0.83	(0.59, 1.07)
15.1–30	45	0.91	11.06	0.83	(0.57, 1.09)
>30	29	0.53	10.43	0.48	(0.14, 0.82)
Missing	28	0.53	1.86	0.24	(0.12, 0.36)
Total	5,133	17.1		12.0	(10.5, 13.5)
		DDP=3 (n=6,642)			
No drinking	5,197				
≤2	147	2.21	2.08	1.35	(1.00, 1.70)
2.1–4	159	2.39	3.51	1.88	(1.53, 2.23)
4.1–6	245	3.69	4.91	3.20	(2.78, 3.62)
6.1–8	181	2.73	5.97	2.46	(2.09, 2.83)
8.1–10	208	3.13	6.99	2.90	(2.50, 3.30)
10.1–15	248	3.73	8.52	3.51	(3.08, 3.95)
15.1–30	180	2.71	11.06	2.58	(2.19, 2.97)
>30	18	0.27	10.43	0.24	(0.12, 0.37)
Missing	59	0.89	1.86	0.49	(0.34, 0.65)
Total	6,642	21.8		18.6	(17.5, 19.7)

Alcohol intake before injury (in number of drinks)	n	Prevalence of drinking (%)	Relative Risks (RR)	Attributable Fraction (%)	95% CIs
		DDP=4 (n=1,928)			
No drinking	1,496				
≤2	55	2.85	2.08	2.35	(1.62, 3.07)
2.1–4	80	4.15	3.51	3.59	(2.74, 4.43)
4.1–6	64	3.32	4.91	2.90	(2.19, 3.62)
6.1–8	51	2.65	5.97	2.32	(1.67, 2.97)
8.1–10	37	1.92	6.99	1.69	(1.12, 2.25)
10.1–15	51	2.65	8.52	2.33	(1.66, 3.00)
15.1–30	49	2.54	11.06	2.24	(1.59, 2.89)
>30	34	1.76	10.43	1.56	(0.97, 2.16)
Missing	11	0.57	1.86	0.46	(0.18, 0.73)
Total	1,928	22.4		19.4	(17.3, 21.6)

[1] See footnotes in Table 1

[2] DDP=2: Argentina, Canada, China, Czech Republic, Dominican Republic and New Zealand;

DDP=3: Brazil, Guyana, India, Ireland, Korea, Panama and Sweden;

DDP=4: Belarus, Guatemala, Mexico, Nicaragua