Risk of 30-day mortality and its association with alcohol concentration level among driver victims of motor vehicle crashes: comparison of population- and hospital-based designs

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ABSTRACT

Background Although blood alcohol concentration (BAC) is undoubtedly associated with increased risk of injury among driver victims involved in motor vehicle crashes (MVCs), some studies noted that high BAC was associated with reduced risk of mortality after injury. In addition, most of the previous studies included only injured patients admitted, which may lead to potential selection bias arising from exclusion of those with minor injury and those who died at the accident scene of MVC.

Method The population-based design included 2586 driver victims with BAC equivalent >0 and 10 307 matched controls (BAC equivalent =0) selected from the Police-reported Traffic Accident Registry from 1 July to 31 December 2016 in Taiwan. The hospital-based design comprised a subset sample, which included 517 driver victims with BAC equivalent >0 and 662 with BAC equivalent =0 hospitalised on the same day the MVCs occurred. Conditional logistic regression models with adjustment for potential confounders were used to estimate the ORs and 95% CIs of 30-day mortality associated with BAC equivalent level.

Results In the population-based design, a positive dose–gradient relationship was observed between BAC equivalent level and 30-day mortality, with a covariate-adjusted OR of 3.77 (95% CI 1.84 to 7.72), 6.19 (95% CI 3.13 to 12.26) and 7.75 (95% CI 4.51 to 13.32) for low, moderate and high BAC equivalent levels, respectively. By contrast, the hospital-based design revealed no significant association between 30-day mortality and alcohol concentration regardless of the BAC equivalent level.

Conclusion The association between BAC equivalent level and short-term mortality could have been overlooked in hospital-based studies that excluded MVC-related deaths outside hospital settings.

INTRODUCTION

The dose–response relationship between the amount of alcohol consumed or the blood alcohol content and the risk of traffic injuries is well established, but with regard to fatal traffic injuries, studies generally do not clarify whether the increase in mortality with increasing alcohol level is due to the fact that alcohol increases the probability of injury occurrence or increases the lethality of injuries or both components.[1] The meta-analysis study conducted by Cherpitel proved the evidence on non-fatal traffic injuries was increased risk with increasing blood alcohol content.[2] However, there is still controversy that surrounds the issue of whether or not blood alcohol concentration (BAC) increases the risk of mortality in drivers involved in motor vehicle crashes (MVCs). Studies have shown that the mortality for hospitalised trauma patients with elevated BAC is significantly lower than that for sober patients with the same injury severity.[3–6] This seemingly protective effect of alcohol is consistent with the argument that 'drunks don't get hurt when they fall because they are so relaxed'.[3 7] However, a study has revealed that drivers with high BAC levels are at a significantly increased risk of encountering serious or fatal injury when injury severity is controlled for as a potential confounder.[8] Other studies have added to the inconsistency by reporting that no significant differences in mortality or length of hospital stay exist between alcohol-intoxicated and non-intoxicated drivers.[9 10]

Studies on the association of BAC with mortality have yielded conflicting results, and one of the factors that might have contributed to this conflict is the different settings in which the studies were performed.[8] To examine the hypothesis that there is a dose–response relationship between blood alcohol content and the lethality of traffic accidents, we performed two independent analyses with different study designs (ie, population-based vs hospital-based design) by using the same data sources.

METHODS

Data source and materials

Data from the Police-reported Traffic Accident Registry (PTAR) and National Health Insurance (NHI) medical claims were analysed. The PTAR is recorded by the National Police Agency in Taiwan. After a road traffic accident is made known to the police, certified police accident investigators examine the accident scene and complete accident reports, which comprise information relevant to the MVC.[11] The NHI data were retrieved from Taiwan's NHI programme, which is implemented and supervised by the National Health Insurance Administration that also performs quarterly expert reviews on random samples of medical claims to ensure the accuracy of such claims.[12] This study was approved by the Institutional Review Board of National Cheng Kung University Hospital (No. A−EX-107-009).

Design and participants

A total of 291 122 individuals involved in MVCs were extracted from PTAR from 1 July to 31 December 2016 (ie, sample enrolment period). These individuals accumulated 330 418 crash episodes. We retained information on the first crash episode for individuals with multiple crashes during sample enrolment. The following exclusion criteria were applied: (1) the individuals involved in the MVCs were pedestrians or passengers (n=38 225); (2) the driver victims had missing information on vehicle type (n=1616); and (3) the alcohol concentration level was unknown or undetected (n=11). Among the remaining 251 270 individuals who were driver victims involved in MVCs, 2614 (1.0%) tested positive for alcohol at the accident scene or in hospitals. We excluded 28 driver victims below 18 years of age (the legal age for obtaining a driver's license in Taiwan). Thus, 2586 driver victims remained and were regarded as having been involved in alcohol-related MVCs (figure 1).

We performed two independent cohort studies using population- and hospital-based designs to assess the association of 30-day mortality with alcohol level. The population-based design included all driver victims involved in MVCs, and the hospital-based design included only those admitted on the same day the MVCs occurred.

Population-based design

Each alcohol-related driver victim was randomly matched with four non-alcohol driver victims in terms of gender, age, vehicle type and MVC date (within 3 days). A total of 2586 alcohol- and 10 307 non-alcohol-related driver victims were identified (figure 1).

Hospital-based design

A total of 517 (20.0%) out of the 2586 alcohol-related driver victims were admitted on the same day the MVCs occurred, and only a small proportion (6.4%, 662/10 307) of non-alcohol driver victims were hospitalised (figure 1). The hospitalised driver victims from both groups were monitored for 30 days beginning on the date of hospitalisation (or MVCs) for information on all-cause mortality.

Independent variable of interest

Taiwanese law requires those who are involved in traffic accidents and show indications of 'drinking and driving' as determined by certified police accident investigators to undergo an alcohol test. Hence, all suspected or confirmed cases involving alcohol consumption must be reported to the National Policy Agency, and these cases should be registered with PTAR. Driver victims with documented breath or BAC (ie, BAC equivalent) are evaluated to determine if they have violated the law.

Eight categories of BAC equivalent level are listed in the *Road Traffic Management and Penalty Act* of the Taiwan Ministry of Transportation and Communications (supplementary table S1, available as supplementary data at journal online). In this study, we regarded *Categories 1* and *2* as BAC equivalent =0, and divided *Categories 3* to *8* into three BAC levels: low BAC equivalent (*Categories 3–6*), moderate BAC equivalent (*Category 7*) and high BAC equivalent (*Category 8*). We selected a BAC of 0.11 g/L to distinguish between low and moderate BAC equivalent, because previous studies have demonstrated that BAC higher than 0.10 g/L may cause significant deterioration of movement control.[13 14] Additionally, a BAC of 0.16 g/L or higher (equivalent to a BAC of 0.81 mg/L) was regarded as a high BAC equivalent level in this work, because this value is under the highest BAC equivalent category listed in the *Road Traffic Management and Penalty Act*. A sufficient number of drivers belong to this category because of the open-ended upper limit.

Outcome variables

The 30-day mortality was defined by linking the personal identification numbers of the driver victims and their matched controls to the Beneficiary Registry of the NHI Program to obtain information on mortality within 30 days after the date of MVCs, including the deaths occurring 'instantly' during the accident. Only information on all-cause mortality is available in the NHI Beneficiary Registry. We were unable to ascertain whether these deaths were directly or highly related to traffic accidents.

Covariates variables

Several covariates were considered in the analysis, including age (18–29, 30–64 and ≥65 years), gender, type of vehicle, month of accident, comorbid conditions and injuries at various body parts. We regarded the month of MVC occurrence (July–December) and vehicle type (truck, car and motorcycle) as covariates in the analysis, because a previous study revealed that motorcyclists

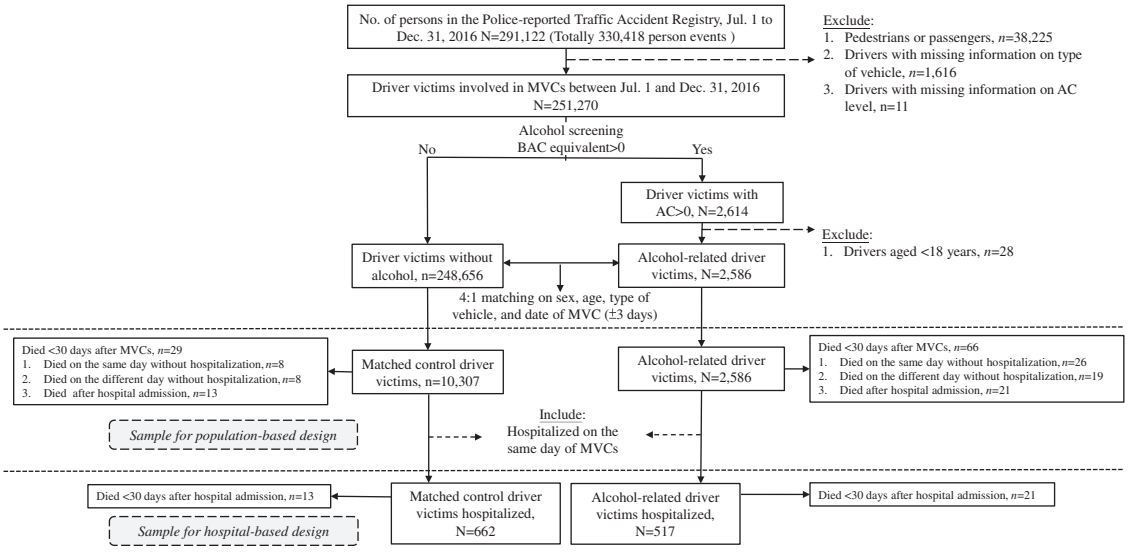

Figure 1 Flow chart of selecting study subjects.

exhibit a seasonal pattern of road accidents that can be explained by air temperature changes over time.[15] Another study showed that vehicle type is associated with the risk of crash-related fatality primarily because of the difference in speed.[16]

Co-morbidities, namely, alcohol dependence (International Classification of Diseases—Tenth Revision, Clinical Modification Codes (ICD-10-CM)=F10), diabetes (ICD-10-CM=E08–E13), depression (ICD-10-CM=F33), hypertension (ICD-10-CM=I10–I15), ischemic heart disease (ICD-10-CM =I20–I25), stroke (ICD-10-CM=H341, I60, I63, I64, G45) and epilepsy/seizure (ICD-10-CM=G40, R569). Information on co-morbidity was obtained from inpatient and outpatient claims filed within 6 months prior to the date of MVCs.

Additionally, information on cause-specific injuries in various body parts was obtained from inpatient and outpatient claims filed within 7 days after the MVCs occurred. Injured body parts include head or neck, thorax or abdomen, upper extremity and lower extremity. Injuries were further classified by the main disease diagnosis according to broad ICD-10 classifications, which is shown in supplementary table S2.

Statistical analysis

We compared the characteristics of driver victims with and without BAC equivalent and those of driver victims with low, moderate and high BAC equivalent under the two study designs (population- and hospital-based) by performing a χ^2 test. We calculated the 30-day mortality rate for different BAC equivalent levels and assessed the dose–gradient relationship between BAC equivalent levels (no, low, moderate and high) and mortality rate by using the Cochran–Armitage trend test.[17] The unadjusted and covariate-adjusted ORs (AORs), and the corresponding 95% CIs of 30-day mortality rate in association with BAC equivalent level were estimated with conditional logistic regression models. Statistical analyses were conducted with SAS (version 9.4; SAS Institute, Cary, NC), and the level of significance was set to alpha=0.05.

RESULTS

Table 1 shows a comparison of the characteristics of study subjects in terms of the BAC equivalent level for the population-based design. Driver victims with BAC equivalent >0 were dominated by males (86.3%), in the middle age group (30–64 years) and involved in motorcycle crashes (68.7%). They also showed a significantly high prevalence of alcohol dependence and depression but a low prevalence of diabetes and ischemic heart disease. In the first 7 days after the MVCs, the driver victims with BAC equivalent >0 had a significant high prevalence of clinical visits for head/neck, thorax/abdomen and upper extremity injuries. The high BAC equivalent level among the driver victims with BAC equivalent >0 was significantly associated with old age, motorcycle crashes, high prevalence of alcohol dependence and diabetes, and high prevalence of clinical visits for injuries in various body parts (table 1).

In the hospital-based design, compared with driver victims with BAC equivalent =0, those with BAC equivalent >0 were significantly younger, were involved more often in car crashes, suffered from higher prevalence of alcohol dependence and epilepsy, and had lower prevalence of diabetes and ischemic heart disease; they also had significantly higher prevalence of head/neck injury but lower prevalence of injuries on upper and lower extremities (table 2). The high BAC equivalent level among hospitalised driver victims with BAC equivalent >0 was significantly associated with old age and high prevalence of alcohol dependence and diabetes. The driver victims with a high BAC equivalent level had a significantly high prevalence of head/neck and upper extremity injuries. A significantly high prevalence of thorax/abdomen injury and a low prevalence of extremity injury were observed in driver victims with moderate and low BAC equivalent levels, respectively.

Table 3 shows the number of deaths among driver victims with and without BAC equivalent for both study designs. Under the population-based design, the overall 30-day mortality rate was 0.74% (95/12 893). The corresponding figures for driver victims with BAC equivalent =0 and BAC equivalent >0 were 0.28% (29/10 307) and 2.55% (66/2586), respectively. Among the 95 deaths, only 34 (35.8%) occurred during hospitalisation or after discharge, and the same number (n=34) of diver victims died on the same day the MVCs occurred without hospitalisation. Among the driver victims with BAC equivalent >0, the 30-day mortality rate was the highest for those with a high BAC equivalent level (3.46%), followed by driver victims with moderate (2.39%) and low (1.35%) BAC equivalent levels. The trend test showed a significant increase in the overall mortality rate with increasing BAC equivalent level (ie, from BAC equivalent =0 to high BAC equivalent level) (p<0.0001).

Under the hospital-based design, the overall 30-day mortality rate was 2.88% (34/1179), and a higher rate was observed for victims with BAC equivalent >0 than for victims with BAC equivalent =0 (4.06% vs 1.96%). The trend test also showed a positive and significant dose–response relationship between BAC equivalent level (from BAC equivalent =0 to high BAC equivalent level) and 30-day mortality rate (p=0.0207).

Table 4 and figure 2 show the covariate AORs of 30-day mortality in association with BAC equivalent level for both study designs. Under the population-based design, a positive dose–gradient relationship (p for trend <0.001) was found between BAC equivalent level and 30-day mortality, with an AOR of 3.77 (95% CI 1.84 to 7.72), 6.19 (95% CI 3.13 to 12.26) and 7.75 (95% CI 4.51 to 13.32) for low, moderate and high BAC equivalent levels, respectively. By contrast, the hospital-based design revealed no significant association between 30-day mortality and BAC equivalent regardless of the BAC equivalent level. Stratified analyses according to type of vehicle are presented in supplementary tables S3 and S4. Test for interaction of BAC equivalent and vehicle type showed no significant difference in dose–gradient association of BAC equivalent with 30-day mortality between motorcyclists and truck/driver victims in both population-based (p=0.5463) and hospital-based (p=0.2267) designs.

DISCUSSION

Main findings

In the population-based analysis, 95 deaths, including 29 with BAC equivalent =0 and 66 with BAC equivalent >0, were identified within 30 days after the MVCs. In the hospital-based analysis, the corresponding figures were 13 (BAC equivalent =0) and 21 (BAC equivalent >0), indicating that a larger proportion of deaths with BAC equivalent >0 occurred outside of hospital settings (45/66=68.2%) compared with deaths with BAC equivalent =0 (16/29=55.2%). Analysis of all driver victims from PTAR showed a significantly high risk of 30-day all-cause mortality in association with BAC equivalent, with a positive and significant dose–gradient relationship. The analysis limited to driver victims who were alive at the accident scene of MVCs and hospitalised soon after showed no significant association between BAC equivalent level and 30-day mortality.

Table 1 Baseline characteristics of study subjects according to BAC equivalent in the population-based design (n=12 893)

	BAC equivalent =0	BAC equivalent >0					
	Total n=10 307 %	Total n=2586 %	P value*	Low n=815 %	Moderate n=585 %	High n=1186 %	P value*
Gender							
Male	86.3	86.3	0.9686	85.9	86.5	86.5	0.9807
Age (years)							
18–29	19.2	19.1	0.9848	27.4	20.3	12.9	<0.0001
30–64	74.0	73.9		65.5	72.3	80.5	
≥65	6.8	6.9		7.1	7.4	6.6	
Mean±SD	43.1±13.9	43.1±14.0		41.0±14.9	43.4±14.4	44.5±12.9	
Vehicle type							
Truck	0.2	0.4	0.1171	0.5	0.5	0.3	<0.0001
Car	31.0	30.9		37.2	35.0	24.6	
Motorcycle	68.8	68.7		62.3	64.4	75.1	
Month of accident							
July	17.8	17.9	1.0000	20.0	17.6	16.7	0.9175
August	14.9	14.8		15.7	14.4	14.4	
September	17.5	17.5		17.9	17.9	17.0	
October	15.8	15.7		15.1	15.2	16.4	
November	17.2	17.2		15.5	18.6	17.7	
December	16.8	16.8		15.8	16.2	17.8	
Comorbid conditions							
Alcohol dependence	0.1	2.2	<0.0001	1.2	1.2	3.3	<0.0001
Diabetes	7.0	5.5	0.0093	4.9	4.8	6.3	0.0277
Depression	0.7	2.0	<0.0001	2.2	1.7	1.9	<0.0001
Hypertension	8.3	8.7	0.5048	6.5	10.8	9.1	0.0278
Ischemic heart disease	1.7	1.0	0.0122	1.2	1.0	0.8	0.0947
Stroke	0.4	0.7	0.0811	0.9	0.7	0.6	0.1961
Epilepsy, seizure	0.4	0.6	0.3333	0.7	0.3	0.6	0.5058
Injury in the body region (ICD-10)†							
Head or neck	13.4	49.5	<0.0001	34.1	49.7	59.9	<0.0001
Skull fracture	1.3	10.2	<0.0001	7.0	8.7	13.1	<0.0001
Intracranial	3.7	22.3	<0.0001	14.2	21.0	28.5	<0.0001
Concussion	2.4	11.3	<0.0001	6.9	12.5	13.8	<0.0001
Superficial	7.0	23.7	<0.0001	16.6	22.7	29.2	<0.0001
Open wound	2.8	18.1	<0.0001	11.3	18.6	22.4	<0.0001
Crush	<0.1	0.4	<0.0001	0.2	0.3	0.5	<0.0001
Others	3.7	10.8	<0.0001	8.2	11.1	12.3	<0.0001
Thorax or abdomen	15.5	23.9	<0.0001	19.5	24.4	26.6	<0.0001
Spinal fracture	0.2	0.4	0.011	0.1	0.7	0.5	0.0027
Rib fracture	1.3	2.8	<0.0001	2.1	2.6	3.4	<0.0001
Pelvic fracture	0.1	0.2	0.3345	0.0	0.3	0.2	0.1939
Open wound	0.5	1.0	0.0004	1.2	0.5	1.2	0.0012
Dislocation, sprain	0.7	0.2	0.0100	0.2	0.3	0.2	0.0142
Crush	0.1	<0.1	0.3102	0.0	0.0	0.1	0.5081
Organ injury	0.9	4.4	<0.0001	2.1	3.8	6.4	<0.0001
Others	13.4	18.7	<0.0001	16.2	20.0	19.8	<0.0001
Upper extremity	31.2	37.1	<0.0001	31.0	38.8	40.5	<0.0001
Open wound	3.9	5.1	0.0084	4.2	6.8	4.8	0.0093
Fracture	3.4	8.7	<0.0001	5.9	8.5	10.7	<0.0001
Crush	0.2	0.1	0.2991	0.1	0.2	0.0	0.2079
Dislocation, sprain, joints, ligaments	1.3	0.5	0.0017	0.6	0.2	0.7	0.0055
Others	25.2	26.3	0.2726	23.3	27.0	27.9	0.0530
Lower extremity	35.3	33.7	0.1426	29.1	35.4	36.1	0.9651
Open wound	6.5	8.4	0.0009	5.4	9.6	9.8	<0.0001

Continued

Table 1 Continued

	BAC equivalent =0	BAC equivalent >0					
	Total n=10 307 %	Total n=2586 %	P value*	Low n=815 %	Moderate n=585 %	High n=1186 %	P value*
Fracture	2.7	4.6	<0.0001	5.2	3.4	4.9	<0.0001
Crush	0.3	0.2	0.8533	0.0	0.5	0.3	0.8155
Dislocation, sprain, joints, ligaments	0.6	0.5	0.8964	0.5	1.0	0.3	0.7110
Others	28.7	23.9	<0.0001	21.1	24.4	25.6	0.0005
Others	6.6	8.9	<0.0001	8.3	6.2	10.6	<0.0001

*Based on a χ^2 test.
†ICD-10, International Classification of Diseases—Tenth Revision; head or neck (S00–S19); thorax or abdomen (S20–S39); upper extremity (S40–S69); lower extremity (S70–S99); and others (T00–T14).
BAC equivalent, blood alcohol concentration equivalent.

Table 2 Baseline characteristics of study subjects according to BAC equivalent in the hospital-based design (n=1179)

	BAC equivalent =0	BAC equivalent >0					
	Total n=662 %	Total n=517 %	Low n=119 %	P value*	Moderate n=102 %	High n=296 %	P value*
Gender							
Male	82.6	86.8	0.0471	89.9	89.2	84.8	0.1032
Age (years)							
18–29	10.9	20.5	<0.0001	24.4	27.5	16.6	<0.0001
30–64	73.6	71.8		66.4	64.7	76.4	
≥65	15.6	7.7		9.2	7.8	7.1	
Mean±SD	48.7±14.6	42.9±14.3		41.8±16.0	40.8±14.9	44.0±13.2	
Vehicle type							
Truck	0.0	0.0	<0.0001	0.0	0.0	0.0	<0.0001
Car	2.9	15.5		14.3	17.6	15.2	
Motorcycle	97.1	84.5		85.7	82.4	84.8	
Month of accident							
July	18.1	16.4	0.9093	26.1	10.8	14.5	0.3825
August	16.9	17.0		12.6	19.6	17.9	
September	17.4	17.8		16.0	22.5	16.9	
October	15.9	18.0		15.1	20.6	18.2	
November	14.5	14.7		14.3	11.8	15.9	
December	17.2	16.1		16.0	14.7	16.6	
Comorbid conditions							
Alcohol dependence	0.3	1.5	0.0256	0.8	1.0	2.0	0.0370
Diabetes	10.4	5.6	0.0029	5.9	3.9	6.1	0.0297
Depression	1.4	1.0	0.5986	0.8	1.0	1.0	0.9687
Hypertension	9.1	9.1	1.0000	6.7	9.8	9.8	0.7944
Ischemic heart disease	2.0	0.6	0.0449	0.8	0.0	0.7	0.3125
Stroke	2.6	1.0	0.0510	0.8	1.0	1.0	0.3553
Epilepsy, seizure	0.2	1.4	0.0248	2.5	0.0	1.4	0.0079
Injury in the body region (ICD-10)†							
Head or neck	43.7	81.2	<0.0001	73.1	80.4	84.8	<0.0001
Skull fracture	10.4	30.6	<0.0001	31.9	28.4	30.7	<0.0001
Intracranial	25.2	60.2	<0.0001	54.6	59.8	62.5	<0.0001
Concussion	12.2	21.9	<0.0001	21.0	26.5	20.6	0.0001
Superficial	18.9	29.8	<0.0001	27.7	26.5	31.8	<0.0001
Open wound	12.5	36.9	<0.0001	27.7	40.2	39.5	<0.0001
Crush	0.0	0.6	0.0497	0.8	0.0	0.7	0.0779
Others	6.5	9.1	0.0958	8.4	7.8	9.8	0.0793
Thorax or abdomen	41.5	42.9	0.6292	37.8	50.0	42.6	0.5055
Spinal fracture	1.2	1.4	0.8249	0.0	2.9	1.4	0.6001
Rib fracture	8.0	8.1	0.9412	8.4	8.8	7.8	0.9616

Continued

Table 2 Continued

	BAC equivalent =0	BAC equivalent >0					
	Total n=662 %	Total n=517 %	Low n=119 %	P value*	Moderate n=102 %	High n=296 %	P value*
Pelvic fracture	0.9	0.6	0.5233	0.0	2.0	0.3	0.5590
Open wound	1.5	1.4	0.8229	2.5	1.0	1.0	0.5105
Dislocation, sprain	1.2	0.0	0.0121	0.0	0.0	0.0	0.0233
Crush	0.3	0.2	0.7132	0.0	0.0	0.3	0.9708
Organ injury	10.1	16.1	0.0024	10.1	18.6	17.6	0.0004
Others	29.3	26.9	0.3599	25.2	32.4	25.7	0.3639
Upper extremity	60.4	50.1	0.0004	45.4	51.0	51.7	0.0063
Open wound	4.7	5.8	0.3890	6.7	8.8	4.4	0.8104
Fracture	28.1	25.9	0.4040	23.5	26.5	26.7	0.6216
Crush	0.2	0.4	0.4252	0.8	1.0	0.0	0.9708
Dislocation, sprain, joints, ligaments	3.0	0.4	0.0009	0.0	0.0	0.7	0.0054
Others	31.7	23.2	0.0012	20.2	22.5	24.7	0.0117
Lower extremity	61.3	44.3	<0.0001	53.8	44.1	40.5	<0.0001
Open wound	9.7	11.2	0.3856	10.9	15.7	9.8	0.6187
Fracture	26.4	15.7	<0.0001	26.9	12.7	12.2	<0.0001
Crush	0.5	0.4	0.8619	0.0	0.0	0.7	0.7631
Dislocation, sprain, joints, ligaments	1.7	1.2	0.4739	2.5	0.0	1.0	0.2992
Others	32.3	23.4	0.0008	23.5	21.6	24.0	0.0030
Others	8.9	14.1	0.0049	12.6	12.7	15.2	0.0037

*Based on a χ^2 test.
†ICD-10, International Classification of Diseases—Tenth Revision; head or neck (S00–S19); thorax or abdomen (S20–S39); upper extremity (S40–S69); lower extremity (S70–S99); and others (T00–T14).
BAC equivalent, blood alcohol concentration equivalent.

Table 3 Numbers of death within 30 days after MVCs among driver victims according to study design and BAC equivalent level

	Population-based design					Hospital-based design	
	Total n=12 893	Overall death n=95	Deaths after hospitalisation n=34	Deaths on the same day as the MVC occurrence without hospitalisation n=34	Deaths on a different day after MVC occurrence without hospitalisation n=27	Total n=1179	Deaths during hospitalisation or after discharge n=34
	n	n (%)	n (%)	n (%)	n (%)	n	n (%)
BAC equivalent =0	10 307	29 (0.28)	13 (0.13)	8 (0.08)	8 (0.08)	662	13 (1.96)
BAC equivalent >0	2586	66 (2.55)	21 (0.81)	26 (1.01)	19 (0.73)	517	21 (4.06)
Low	815	11 (1.35)	4 (0.49)	5 (0.61)	2 (0.25)	119	4 (3.36)
Moderate	585	14 (2.39)	3 (0.51)	4 (0.68)	7 (1.2)	102	3 (2.94)
High	1186	41 (3.46)	14 (1.18)	17 (1.43)	10 (0.84)	296	14 (4.73)
P value*		<0.0001					0.0207

*Based on the Cochran–Armitage trend test.
BAC equivalent, blood alcohol concentration equivalent; low=0–0.11 g/L; moderate=0.11–0.16 g/L; high>0.16 g/L.
MVCs, motor vehicle crashes.

Interpretation

Under both study designs, head injury was among the most prevalent conditions for victim drivers with BAC equivalent >0, especially in the sample analysed in the hospital-based study. By contrast, lower extremity injury was mostly prevalent among driver victims with BAC equivalent =0. Our findings are consistent with those of a previous report that indicated that the head is the most common body part injured in traffic accidents.[5 9] One of the possible reasons accounting for a higher prevalence of head injury associated with driving while intoxicated is related to a low level of coordinate ability while crashes happen. Loose objects become flying objects in car accidents, which makes heads and upper body more vulnerable to injury. In fact, Cunningham *et al* found that patients with positive blood alcohol were 2.1-fold more likely to have a more severe head injury as measured on computed axial tomogram scan by the Marshall scores.[18] Another explanation is drivers are less likely to use protective equipment (ie, seat belts used and wear helmets) when they have been drinking compared with sobers. Therefore, the head is higher injured risk while the intoxicated driver does not wear helmets, especially as the motorcycle rider is major victims of crashes in our study. The previous study shows clearly that 17% of alcohol-impaired drivers fail to buckle their seat belts compared with 3% of sober drivers.[19]

Table 4 Association of BAC equivalent with the risk of 30-day mortality among driver victims: comparison of population- and hospital-based study designs

	Population-based design		Hospital-based design	
	Crude OR (95% CI)	Adjusted* OR (95% CI)	Crude OR (95% CI)	Adjusted* OR (95% CI
BAC equivalent =0	Reference	Reference	Reference	Reference
BAC equivalent >0				
Low	4.85 (2.41 to 9.74)	3.77 (1.84 to 7.72)	1.74 (0.56 to 5.42)	1.06 (0.32 to 3.54)
Moderate	8.69 (4.57 to 16.54)	6.19 (3.13 to 12.26)	1.51 (0.42 to 5.40)	0.86 (0.21 to 3.45)
High	12.69 (7.86 to 20.50)	7.75 (4.51 to 13.32)	2.48 (1.15 to 5.34)	1.22 (0.52 to 2.89)
P value†		<0.0001		0.6745

*Adjusted covariates included gender, age, type of vehicle, month of accident, comorbid conditions and injuries in various body parts (including the head or neck, thorax or abdomen, and upper and lower extremities).
†Test for trends in the dose–gradient relationship according to the significance of BAC equivalent level treated as an ordinal variable in conditional logistic regression models.
BAC equivalent, blood alcohol concentration equivalent.

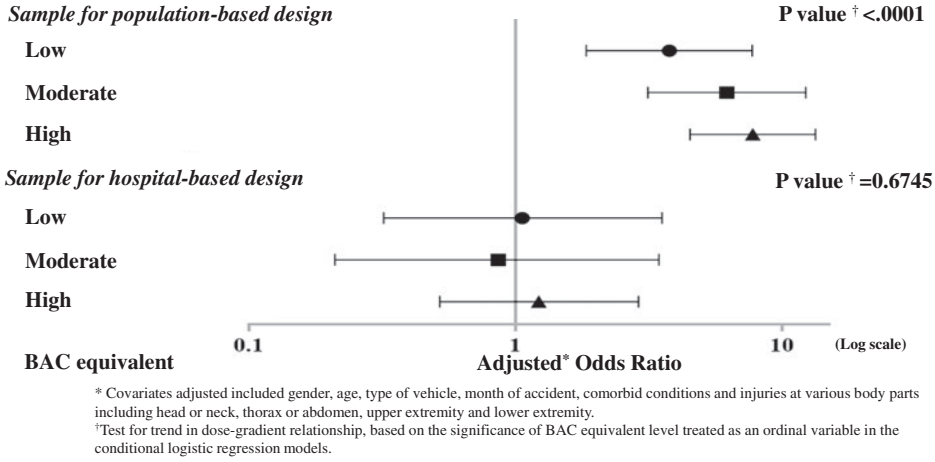

Figure 2 Graphic presentation for the association of blood alcohol concentration (BAC) equivalent with risk of 30-day mortality among driver victims.

Our population-based design demonstrated a positive dose–response relationship between BAC equivalent and mortality from MVCs. Several previous studies also demonstrated a dose–gradient effect of alcohol on driving. Based on the blood samples from 2500 injured drivers in South Australia, Longo *et al* found a significant concentration-dependent relationship between alcohol and culpability: as BAC increased, so did the percentage of culpable drivers.[20] Dubois *et al* conducted a case–control study among drivers aged 20 years or older who had been tested for both drugs and alcohol after involvement in a fatal crash in the US (1991–2008).[21] It showed that each 0.01 BAC unit increased the odds of an unsafe driving action by approximately 9–11%, which was slightly enhanced by a combined use of cannabis.

Factors that contribute to such dose–gradient effect of alcohol can be multifaceted. In the analysis of all people in US fatal automotive accidents, 1994–2008 (n=1 495 667), Phillips and Brewer analysed the severity of automotive injuries associated with BAC in increments of 0.01% and found a strong dose–response relationship for several factors associated with accident severity.[22] Compared with sober drivers, buzzed drivers are significantly more likely to speed, to be improperly seat-belted and to drive the striking vehicle; the greater the BAC, the greater the average speed of the driver and the greater the severity of the accident. In a simulated driving skill game, Calhoun *et al* explored brain activation and behavioural alterations from baseline at two BACs (ie, 0.04 and 0.08, compared with placebo).[23] Dose-dependent functional MRI changes were revealed in orbitofrontal and motor (but not cerebellar) regions; visual and medial frontal regions were unaffected. In addition, cerebellar regions were significantly associated with driving behaviour in a dose-dependent manner. In a literature review, Calhoun *et al* found that alcohol-related effect on cognition may show dose-dependent effects on multiple responses including hand and body steadiness/coordination, increased choice reaction time and time estimation.[24]

Our hospital-based study design found no significant association between BAC equivalent and 30-day mortality. The possible protect mechanisms obtained from several animal studies have suggested that alcohol might exert a neuroprotective effect by inhibiting *N*-methyl-d-aspartame receptor-mediated excitotoxicity and inflammatory neurotransmitter release via the injured neuron.[25] The seemingly protective effect of alcohol has also been observed in previous hospital-based studies that compared the risk of mortality between drinking and non-drinking trauma victims admitted to hospitals.[3 5] However, clinical trials based on proposed mechanisms have been disappointing and have reported conflicting results.[26] Albrecht *et al*[26] evaluated the association between BAC and in-hospital mortality after traumatic brain injury. Using adjusted logistic regression models, the authors noted that the upper level of each BAC categorisation from 0.10 g/L (OR=0.63, 95% CI 0.40 to 0.97) to 0.30 g/L (OR=0.25, 95% CI 0.08 to 0.84) was associated with reduced

risk of mortality after traumatic brain injury compared with individuals with undetectable BAC. The seemingly protective effect of high BAC disappeared in sensitivity analyses of individuals without penetrating brain injuries (mostly due to gunshot wounds), suggesting that the observed protective association between BAC and in-hospital mortality after traumatic brain injury could have resulted from the bias introduced by the inclusion of penetrating injuries.

The results of our hospital-based analysis are essentially similar to those of several clinical studies that failed to demonstrate a harmful effect of alcohol on patient mortality.[6 9 10] There are several methodological considerations in our hospital-based design. First, driver victims with high BAC levels were likely admitted to a trauma centre due to their low Glasgow Coma Scale scores, which could have been due to alcohol instead of severe traumatic brain injury.[27] Care at trauma centres has been shown to decrease the short- and long-term risks of death and improve functional outcomes after injury.[28 29] Thus, the null results observed in our hospital-based design might be due to the large proportion of driver victims with BAC equivalent >0 who were transferred to trauma centres because of their intoxicated status,[30] which compromised the adverse effect of alcohol. Second, potential selection bias could have been present in our hospital-based analysis, which did not include information on driver victims outside hospital settings. Hospital-based studies usually do not include patients who do not require hospitalisation, patients treated at nonparticipating hospitals and individuals who do not seek treatment.[31–33] Therefore, analyses limited to hospitalised driver victims might underestimate the risk of mortality in cases where the risk of death is disproportional to the BAC equivalent level. Our data showed a high proportion of pre-hospital deaths in driver victims with BAC equivalent >0. Nonetheless, the non-significant dose–response relationship between BAC equivalent level and risk of mortality in hospital-based design could also be due to a small number of death observed in study subjects with low (4 deaths/119 victims=3.36%) and moderate (3 deaths/102 victims=2.94%) BAC equivalent level. Thus, interpretation of such non-significant findings should proceed with caution.

Strengths and limitations

This study has several methodological strengths. First, our study sample for the population-based design included driver victims who did not seek treatment or who died soon after traffic crashes prior to receiving treatment in hospitals; thus, our study is unlikely to suffer from prevalence–incidence bias. Second, our study sample was selected from nationwide databases with fairly large populations. This characteristic allowed for analyses of the dose–gradient relationship between BAC equivalent levels and risk of mortality without compromising statistical power. Third, we assessed the association of 30-day mortality with BAC equivalent level by using two cohort study designs and the same research data. This condition helped reveal the potential methodological problems of hospital-based design in assessing the association of BAC with mortality.

Despite these strengths, several study limitations should be noted. First, information on injury severity was unavailable from the databases that we used, but we managed to control for the body parts injured. Residual confounding could occur if injury severity varied with BAC equivalent level among driver victims. Second, no information on when BAC equivalent was measured was available. The BAC equivalent level may decrease with the amount of time that passes after crashes because of the human body's absorption, distribution and metabolism of alcohol.[31 34 35] Third, the deaths that occurred within 30 days after the MVCs may not necessarily be traffic injury-related. We were unable to ascertain whether these deaths were directly or highly related to traffic accidents, because we only analysed all-cause mortality and were not allowed to contact the patients or their family members. Fourth, it is expected that while most BAC equivalent was assessed by breath testing (especially for those who were not injured) in the population-based design, the majority of BAC equivalent was measured by blood testing among study subjects analysed in the hospital-based design. Despite that, the information concerning the type of BAC equivalent measurement is not available from our data sets, which leaves room for information bias. Given that breath testing could insert a greater degree of BAC level misclassification than blood testing, we expected that the study results obtained from population-based design are more likely to suffer from BAC exposure misclassification. However, the errors involved in breath testing are unlikely to be systematic, which leads to a non-differential BAC misclassification, which in turn may have resulted in underestimation rather than over-estimation of the associations between BAC equivalent and mortality. The true magnitude of ORs is expected to be greater than what is presented.

CONCLUSION

The hospital-based study could have overlooked the association between BAC and short-term mortality due to the exclusion of deaths outside hospital settings. The legal limit of BAC prior to driving is a critical issue from a public health perspective. Our population-based design demonstrated a significant association between BAC equivalent and 30-day mortality with a dose–gradient pattern. Thus, BAC is still a reliable predictor of short-term death from MVCs among driver victims. Health policymakers should consider other strategies that can effectively reduce drink-and-drive behaviours.

What is already known on this subject

- The dose-response relationship between the amount of alcohol consumed or the blood alcohol content and the risk of traffic injuries is well established.
- Studies generally do not clarify whether the increase in mortality with increasing alcohol level is because alcohol increases the probability of injury occurrence or increases the lethality of injuries or both components.

What this study adds

- In the population-based design, a positive dose–gradient relationship was observed between BAC equivalent level and 30-day mortality, with an adjusted OR of 3.77, 6.19 and 7.75 for low, moderate and high BAC equivalent levels, respectively.
- In the hospital-based design, no significant associations between 30-day mortality and BAC equivalent level were found.
- Population-based studies should be able to include all driver deaths involved in MVCs to reduce selection bias.
- The risk of 30-day mortality among driver victims of vehicle crashes could have been overlooked in hospital-based studies that excluded deaths outside hospital settings.

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