A case-control study of the effect of alcohol on the risk of driver fatal injury in New Zealand

Keall M.D., Frith W.J., Patterson T.L.

Land Transport Safety Authority of New Zealand

Abstract

This study presents estimates of the effect of alcohol on the risk of driver crash death in New Zealand. The risk estimates presented in this paper are derived from a case-control study using data from the years 1995 to 2000. The cases were drivers killed in crashes that occurred on Friday and Saturday nights between 9:30pm and 2:30am and the controls were drivers stopped at the roadside and breath-tested, also on Friday and Saturday nights (the main drinking days and times in New Zealand). Estimates of driver fatal injury risk per average driving trip were derived from a log-linear model fitted to the data. The estimated risks increased steeply with increasing BAC, with risks at all BAC levels statistically significantly higher for drivers aged under 20 and for drivers aged 20-29 than for drivers aged 30 and over.

Keywords

Alcohol, Risk, Case-control study

Introduction

A major study into the effect of alcohol on the risk of crashing was conducted by Borkenstein et al. (1) in Grand Rapids, Michigan, the data of which were analysed further by Allsop (2) and Hurst, Harte and Frith (3). Borkenstein et al. compared the BAC of drivers involved in all crashes in Grand Rapids with a control group of drivers selected from the city's traffic at the same locations and times as a random sample of the crashes. It was found that the risk of crashing increased with increasing BAC, the risk curve steepening as higher alcohol levels were reached. McLean et al. (4) found a steeper risk curve in a similar study in urban Adelaide, Australia, related to a more severe crash sample than the Grand Rapids study, and possibly also to the relatively high refusal rate of drivers asked to furnish a breath sample.

Zador, Krawchuk and Voas (5) compared 1995 and 1996 US data for drivers involved in fatal crashes with estimates of exposure from a national roadside breath-testing survey in 1996. The crash and survey data were restricted to Friday and Saturday nights between 10pm and 3am. Using 0-0.5 mg/dl BAC as baseline, they found the relative risk increased steadily with increasing driver BAC, increasing more rapidly for younger drivers (16-20 years) than older drivers (>20 years). For male drivers aged 16 to 20 years the relative risk of fatal crash involvement doubled for each 20mg/dl BAC increase.

Maycock (6) compared crash-involved British drivers at BAC=40 mg/dl and higher with roadside alcohol survey-based exposure estimates for the period 7pm to 2am on Thursday, Friday and Saturday nights. The risk estimates, with an exponential curve extrapolated to lower BAC levels, were scaled so that the risk at zero BAC was one. The estimated average risk at a BAC of 40mg/dl of driver death in a crash was 3.7 times the risk at BAC=0. The risk rose to 12.4 at 80mg/dl and 154 at 160mg/dl.

Methods

Data from two sources were used: control (or exposure) data from roadside breath-testing measurements and case data extracted from traffic crash reports for drivers killed in crashes with BAC results from post-mortem reports.

Control data: roadside breath testing measurements

Data were used from 85,120 drivers who were breath tested at randomly selected roadside sites between 1995 and 2000. In 1995 and 1996 the Police stopped and directed vehicles to the roadside, where civilians breath tested and interviewed the drivers. Participation was voluntary. From 1997 onwards, measurements were taken from compulsory breath testing operations in which the Police breath tested drivers. The information was gathered over late summer and autumn (February to May), on Friday and Saturday nights between 10pm and 2am (the main drinking times), excluding holiday weekends. The roadside sites were chosen randomly from 50km/h areas that were not close to drinking establishments. Data from sites that were conspicuous to oncoming drivers were excluded. Their inclusion would have led to overestimated relative risk estimates of about 10%[1] for the higher BAC levels. Only data from light four-wheeled vehicles were analysed where vehicle type data were available in disaggregated form. For two of the six years' breath-testing data (1996 and 1997), no such disaggregation was possible. Examination of the remaining four years' data indicated that the inclusion of

[1] The estimated percentage of drivers over the legal BAC limit was about 10% higher based on data from the non-conspicuous sites than that from all sites including the conspicuous ones.

motorcyclists and truck drivers in the breath-testing sample for 1996 and 1997 would have a negligible effect on the overall distribution of driver trips by BAC levels. This was related to their relatively low representation (only 5%) in night-time weekend traffic. The 1995 measurements covered about 57% of New Zealand's population, whereas the 1996 to 2000 measurements covered the entire country except areas more than 50km from any population centre with at least 1,000 inhabitants. The data were weighted to account for: variations in the proportions of passing traffic sampled; sampling probabilities applied to potential sites; the length of time information was gathered at each site; how many times a site was used in the given year (usually once or, rarely, twice). In 1995, 63 drivers (1.1% of those requested) refused the voluntary breath test and interview, compared to only three drivers in 1996. From 1997 onwards there was close to full compliance with the compulsory breath tests. Drivers who attempted to turn off before the site were generally pursued and most were found to be over the legal limit (henceforth referred to as DUI – driving under the influence).
Although breath alcohol readings were used to estimate BACs for the vast majority of the control sample, a small number of drivers had evidential blood tests following their refusal of the breath test. Rarely (on less than five occasions), a driver who refused the breath test would also refuse the blood test (resulting in prosecution). For these drivers, a low illegal BAC was imputed. As sites were randomly sampled, weighted counts of sampled drivers could be used as an exposure measure for each driver group (e.g. drivers over the legal limit), estimating the number of all such drivers passing sites in the areas surveyed. The average rural drivers' trip length was an estimated 50% longer than other drivers' trips, according to a household travel survey in 1997/98 (described in Land Transport Safety Authority, (7)). The rural driving trip involves more travel on higher speed limit roads that have a higher fatality risk per km driven. Thus, the exposure units do not provide a meaningful unit of comparison for risks of urban vs. rural drivers. Further, sampling procedures and on-site procedures changed between years, leading to some variation in the number of sites that were inconspicuous (and hence useable). For these reasons, the control data were not used to compare exposure *between* strata defined by the year and the size of the population centre from which the data were gathered.

Case data: drivers killed

The data on fatally injured drivers were from all crashes occurring between 9.30pm and 2.30am on Friday and Saturday nights for the years 1995 to 2000 in the areas covered by the exposure data. The inclusion of the extra half-hours outside the periods covered by the exposure data (10pm to 2am) was justified by the lack of certainty regarding the times of many night-time crashes. The BAC distribution of the dead drivers is unlikely to be much different for crashes that did occur during the extra half-hour periods. The geocoded crash sites were identified on maps and were classified according to the size of the nearest population centre. Crashes that occurred more than 50 km from a population centre of at least 1,000 residents were omitted, as relevant control data (roadside breath testing measurements) were not collected. These comprised 10% of the dead driver sample. If a given year's roadside breath testing measurements excluded parts of the country, crashes from these areas were also excluded. Although control data were only collected on 50km/h roads, data from the 1989/90 travel survey indicated that 92% of night-time trips made by non-main urban area drivers passed through urban speed limit areas that were within the scope of the exposure data collected. Therefore, apart from the geographical exclusions already mentioned, crashes that occurred on 100km/h roads were included in the case sample. However, crashes involving motorcycles and trucks were excluded due to the lack of disaggregated exposure data for two of the six years studied (as described above), and because of their different characteristics vis-à-vis light four-wheeled vehicles: motorcyclists have about 18 times the risk of car drivers per hour driving (Land Transport Safety Authority (7)); truck drivers are much better protected and their driving task is very different.

Of the 102 dead drivers in the sample, only four were killed in crashes involving two vehicles whose drivers were both killed. 78% had BAC results available via post-mortem tests and, of these, 70% were over the legal limit (for the age of the driver)[2]. Police Officers attending the crash site record their judgement of each driver as "suspected" or "not suspected" of being impaired by alcohol. This judgement is recorded as "unknown" where the Police Officer has insufficient information. The Police are generally quite accurate in this judgement: for all dead drivers for the years 1995 to 2000 where BAC results were available, 92% of drivers judged "not suspected" had a BAC=0. Of those judged "suspected", 77% had a BAC over 80mg/dl. The usefulness of this judgement made at the crash site is that it is made independently of the later decision whether to obtain post-mortem BAC information from the driver, a decision that appears to be slightly biased towards the testing of alcohol impaired drivers: in the case of our sample of dead drivers, only 71% of the "not suspected" and "unknown" drivers were tested, whereas 81% of the "suspected" drivers were tested. This correlation between driver impairment and the availability of BAC data is a potential source of bias towards high relative risk estimation that needed to be addressed. Accordingly, zero BAC was imputed for all four drivers judged "not

[2] The legal limits were 80mg/dl for drivers 20 and over and 30mg/dl for drivers under 20.

suspected" who had missing BAC values. This imputation increased the percentage of available BAC values for the "not suspected" and "unknown" group of drivers to 86%. The allocation of zero BAC to the four drivers is likely to be quite accurate, given the accuracy of the Police judgement combined with the apparent bias towards non-testing of non-impaired dead drivers. If any of the drivers with imputed zeros in reality had non-zero BACs then this imputation will lead to *lower* estimates of relative risk than those achieved from the complete data. The slightly higher rate of available BACs (following the imputation) for the "not suspected" and "unknown" group than the "suspected" group would also be expected to lead to slightly lower relative risk estimates.

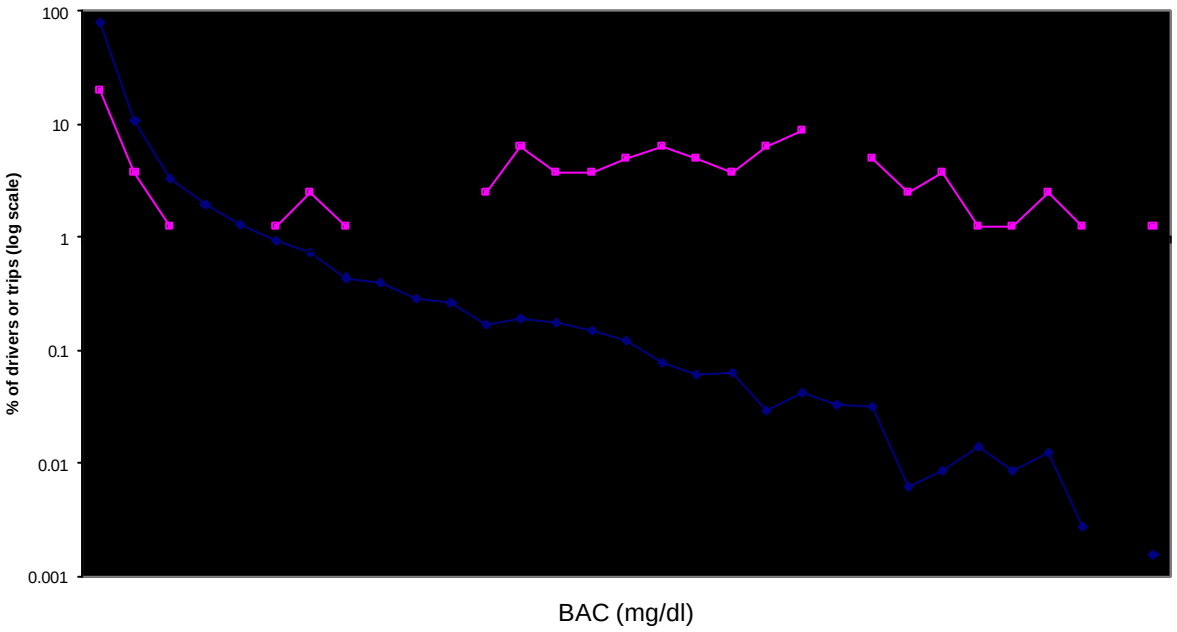
Risk estimation

In this study, the principal estimate produced is the average risk of dying in a road crash on a given type of trip for a given driver group. These were estimated according to a model described below, fitted to raw estimates of risk defined as:

$$risk_j = \left(a_j / c_j \right) \quad \ldots\ldots\ldots\ldots\ldots\ldots (1)$$

where a_j is the count of dead drivers (from the *case* sample) and c_j is the estimate of exposure (number of average driving trips, from the *control* sample) for the driver-exposure[3] combination *j*. The a_j can also be interpreted as a count of trips in driver-exposure class, *j* where the driver was fatally injured in a crash.

Figure 1: Distribution of case and control data by BAC level



Model for estimating relative risk

A Poisson log-linear model was fitted to the count of dead drivers, a_j, with the respective estimates of exposure, c_j, applied as an offset. The additional variation (beyond that assumed by the Poisson distribution) due to the sampling error of the exposure estimates was incorporated into the fitting algorithm (the SAS procedure, GENMOD (8)). For both crash and roadside breath-testing data, the BAC level was classified into the following intervals (in mg/dl): 0-4.9 as the baseline level and then 5-14.9, 15-24.9, etc (in steps of 10mg/dl). For some of the higher BAC values where control data were available for an interval adjacent to that of cases that lacked corresponding control data, an interval of width 20mg/dl, or sometimes 30mg/dl, was defined within the *year* by *location* stratum concerned. The mid-point of each interval was taken to represent the interval in the regression analysis, with the exception of the lowest interval, that was allocated the value zero, which would potentially

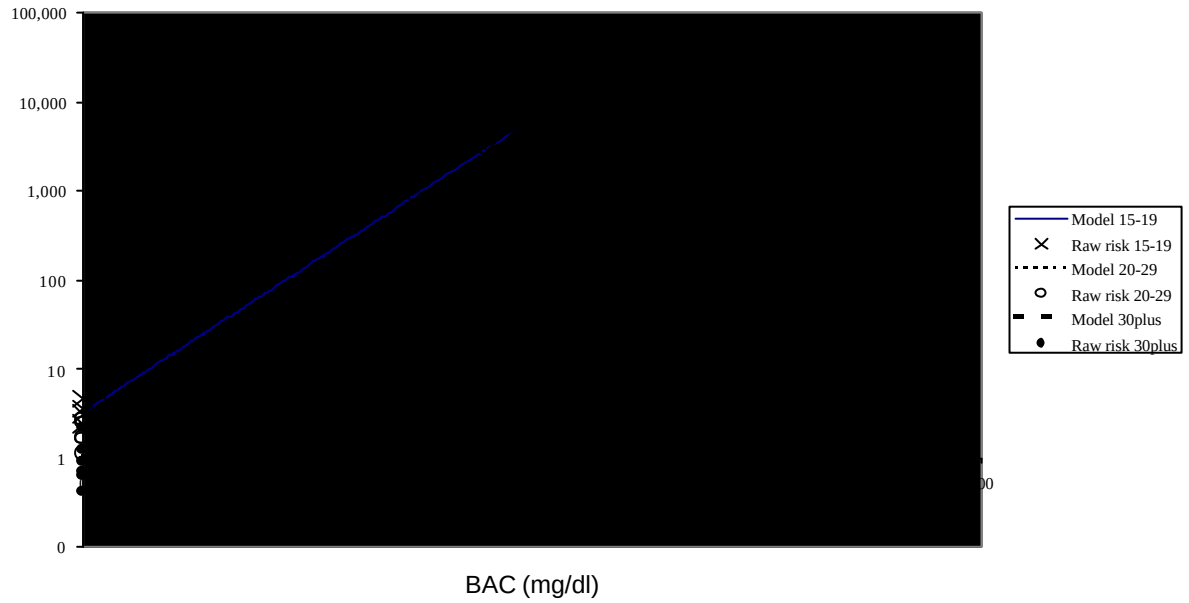
[3] A driver-exposure combination may be "trips made between midnight and 2am by teenage drivers". The single index *j* is used for brevity to define groups cross classified by driver type and exposure type.

lead to underestimated risks if the non-zero BAC levels were associated with risks above those at zero. However, this was not considered to be potential source of bias. Low non-zero BAC levels have historically been included in the baseline BAC level for relative risk estimates in other studies (references (1) to (6)). As the relative exposure between rural and urban areas and between years could not be estimated accurately, as described above, three *location* strata were defined according to the size of the population centre closest to the crash or exposure site: cities with population over 100,000; population centres with population 10,000 to 100,000; and rural centres with population between 1,000 and 10,000. Each of the six years' data analysed was also defined initially as a stratum, although pairs of years were then combined where the pairing had little effect on the estimated coefficients of the model. The following explanatory variables and their interactions were incorporated in the model: BAC, *year*, driver gender, driver age (15-19, 20-24, 25-29 and 30+), time of night (before or after midnight), and *location* (the factor defining the strata according to the size of the population centre closest to the crash or exposure site). Only statistically significant terms were retained in the model: BAC, driver age, age by BAC and the stratum variables, *location*, *year*, and their interaction. As their corresponding estimated coefficients were nearly identical, the age groups 25-29 and 20-24 were combined, which had negligible effect on the fit of the other parameters.

Results

Figure 1 shows the distribution of case (fatally injured drivers) and control (average trips estimated by random roadside breath-testing measurements) data by BAC level. These are presented on a log scale to provide better resolution for the lower percentages. The underrepresentation of the case sample (dead drivers) in comparison with the control sample at lower BAC levels and its overrepresentation at higher levels are clearly shown.

Figure 2: Risk of driver fatal injury by age group relative to drivers aged 30 and over at BAC=0, controlling for location and year



Equation (2) presents the model for mean risk of driver fatality per average trip during the main drinking times and days, fitted according to the procedure outlined above. The risks thus estimated are relative to the risk of a non-drinking driver aged 30 or more. The nuisance parameters consisting of the intercept and the stratification variables *year*, *location* and their interaction have been omitted here as they do not have meaningful interpretations in terms of risk, as explained above.

$$risk \sim \exp(0.033\,BAC \sim 1.2\,twenties \sim 1.2\,teens \sim 0.017\,BAC \sim teens) \quad \ldots\ldots\ldots\ldots (2)$$

where *teens* takes the value 1 for drivers under 20-years-old and zero otherwise, and likewise *twenties* for drivers aged between 20 and 29. The goodness of fit statistics indicated that the model fitted the data reasonably well (the deviance was 46 for 50 degrees of freedom). However, the relatively few observations that were available at low positive BAC levels for the age group 20-29 were all considerably higher than the fitted risks, indicating that the model underestimates risks at these levels for this age group (see Figure 2). Although the terms BAC^2 and

BAC^3 were also significant, the simpler model was retained, despite the lack of fit for the age group 20-29, to facilitate comparison with other studies. The Grand Rapids study (reference (3), where drinking frequency is controlled for) shows close to exponential growth in risk with increasing BAC; the same is true of McLean et al. (4) (apart from a dip at low BACs) and Zador et al. (5) for teenaged drivers. Thus the dose-response relationship between driver BAC and crash risk may be best represented exponentially, where each additional unit increase in driver BAC is associated with a constant proportional increase in risk. The raw risks, $risk_j$ as defined in equation (1) for which either crash or exposure data were missing were not used in the fitting of the model. Figure 2 shows estimated risks by BAC level and by age (15-19-year-olds, 20-29-year-olds and 30+). The raw risks are represented by crosses and circles and the lines represent the best estimates generated by the model for the mean risk of the relevant age group. The model-based estimates are only displayed within the BAC range of the available data. All quantities have been scaled so that they are relative to the risk of non-drinking drivers aged 30 and over within each stratum (where the strata were defined by *location* - the size of the nearest population centre – and *year*). The vertical axis has a log scale to provide better graphical representation of the risks at lower BAC levels.

Figure 3: Model estimates of risk at low BAC levels on linear scale for driver fatal injury relative to drivers aged 30 and over at BAC=0, controlling for location and year (95% confidence limits for mean risk as dotted lines)

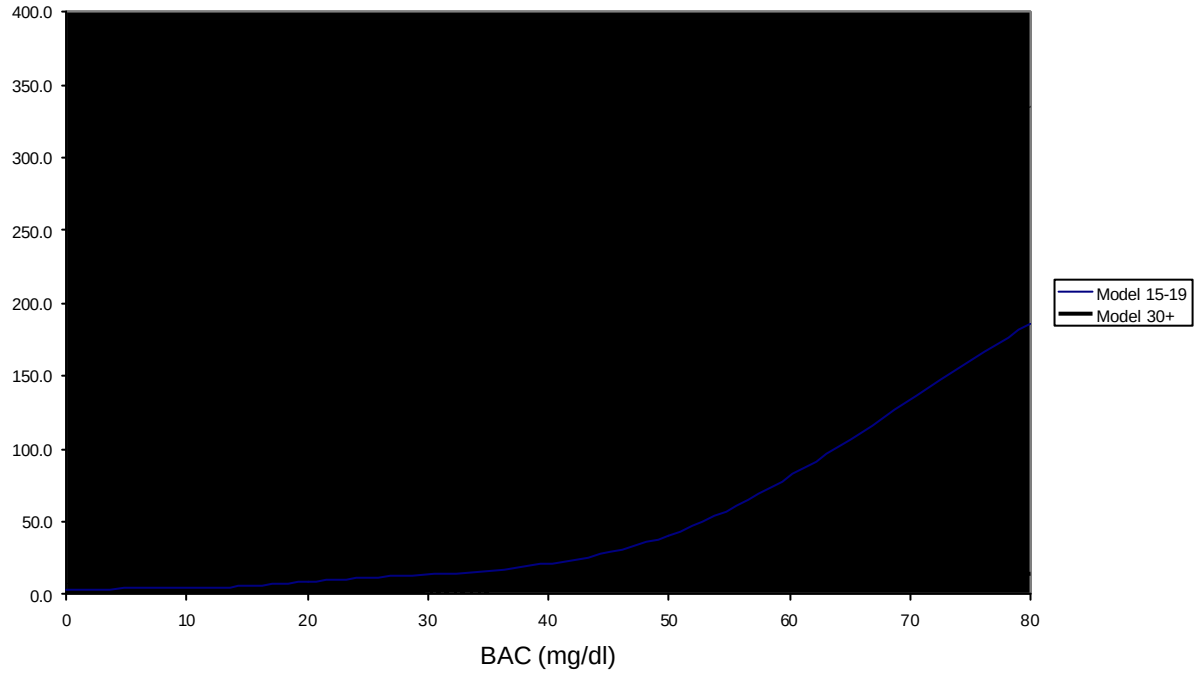


Figure 3 shows the same model-based estimates of risk as Figure 2 for the oldest and youngest driver age groups, but presents the estimates on a linear rather than a log scale for the lower BAC levels. It is clear that the risks for the younger drivers increase much more steeply with increasing BAC than those of the older drivers.

The model provides the following estimates:

?? By as low as 20mg/dl, drivers aged over 19 already have on average a significantly higher risk of fatal injury than at BAC=0. This risk is estimated to be twice their risk at BAC=0. For teenaged drivers, the risk at 20mg/dl is already almost three times their risk at BAC=0.

?? Drivers in their 20's have 3.3 times the risk of drivers aged 30 and over, this difference statistically significant for all BAC levels with a 95% confidence interval (1.3, 8.4).

?? Although risk increases multiplicatively at the same rate for drivers aged 20-29 and drivers aged 30 plus, the excess risk increases much more quickly for the younger drivers. For example at BAC=30, drivers in their twenties have 3.3 times the risk of drivers aged over 30 at the same BAC, but an *additional* risk of 6 (where 6 represents 6 times the risk of a non-drinking driver aged 30 and over).

?? Risk increases much more steeply for teenaged drivers. This can be seen most clearly in Figure 3. At BAC=30, drivers in their teens have 5.5 times the risk of drivers aged over 30 at the same BAC, but an *additional* risk of 12 (where 12 represents 12 times the risk of a non-drinking driver aged 30 and over).

Discussion

Previous studies of crash or casualty risk in relation to driver BAC level (references (1) to (6)) show more steeply increasing risk curves for more severe crashes, steeper curves for single vehicle crashes than for multi-vehicle crashes, and steeper curves for driver fatality than driver fatal crash involvement. This last relationship may be associated with the increased vulnerability to trauma due to alcohol ingestion (see Evans and Frick (9)). It is also possible that the relative risks at night are higher due to interactions of risks associated with alcohol with risks due to fatigue (see Arnedt at al. (10)). Given these considerations, the crash sample used in this study could be expected to correspond to relatively steep risk curves. Possibly related to New Zealand's low levels of congestion (particularly late at night) and the grade of the road network, there was a high proportion of single-vehicle crashes (64% of the sample). The risks estimated here are higher (for equivalent age groups) than those estimated by Zador et al. (5) for single vehicle driver fatal injury, however their sample was restricted to higher traffic flow roads and to larger population centres, which may have reduced their estimated risks relative to our sample as lower grade roads may present relatively higher demands on drinking than sober drivers. Maycock's UK results (6) are similar to those presented here (for example his estimated risk at BAC=40mg/dl is 3.7 for all drivers compared to our estimate of 3.8 for drivers aged over 19), although his estimates were not disaggregated by age. A steeper risk curve for younger drivers than for older drivers has also been found in all the studies referenced (eg Allsop (2), Zador et al. (5)) where the available data have allowed such a disaggregation.

Conclusions

The New Zealand risk of driver fatal injury during the main drinking times increases steeply with increasing BAC. At BAC=20 mg/dl, an average risk for drivers aged over 19 is twice his/her risk at BAC=0. At BAC=20 mg/dl, the estimated risk for the average teenaged driver is almost three times their non-drinking risk. Risks are significantly higher at all BAC levels for drivers aged under 20 and drivers aged 20-29 than for drivers 30 and over. The risk estimates presented here are generally higher than for other comparable studies in other countries, which may reflect driving conditions in New Zealand as well as the nature of the crash sample used where almost two out of every three fatally injured drivers died in a single-vehicle crash.

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