Issues in Estimation of Risk Curves Against Driver BAC Levels

MD Keall
WJ Frith

Land Transport Safety Authority of New Zealand, PO Box 2840, Wellington, New Zealand

Abstract
There are relatively few case-control studies that have generated risk curves against BAC (blood alcohol concentration) level. Reasons for this include the difficulty in obtaining samples of controls (non-crash-involved drivers on the road) representative of the same population of drivers as the cases (crash-involved drivers). This paper discusses how the design and analysis methods used in particular studies may affect resultant risk curves and recommends a methodology for obtaining adequate samples and risk estimates. Particular issues addressed include sample design, accommodation of the sample design by the analysis method, treatment of missing values (imputation methods) and possible biases.

Background
The scarcity of case-control studies producing risk curves of crash involvement or crash injury risk against driver BAC (blood alcohol concentration) is related to methodological difficulties in acquiring adequate data, not to lack of necessity for such estimates to direct road safety policy. Alcohol use by drivers remains a concerning problem for the road safety community and there is a pressing need for research concerning not only the way that risk may increase with alcohol consumption, but how this risk may be modified by other factors, such as age, gender and road type. The most cited study producing risk curves against BAC was carried out by Professor Borkenstein and colleagues (1) in the years 1962-63 in the city of Grand Rapids, Michigan. Drivers involved in some 2000 crashes were the cases, sampled from all city crashes excluding very minor crashes. Control drivers were selected at the same locations, times of day, and day of the year (where possible) as another sample of crashes, but were not matched by these characteristics to the sampled cases. Four control drivers were selected for each sampled crash. Further analysis of these data has been published subsequently (2, 3).

Another case-control study was conducted in the late 1970s in urban Adelaide, Australia by McLean et al. (4), making use of a sample of drivers involved in crashes to which an ambulance was called, with controls interviewed and breath tested at the red phase of traffic lights on or near to the route and direction followed by the respective case driver. Control drivers were also selected to match cases by time of day, day of week, by driver age group and gender.

Recently, an American study (5) employed a study design employing some methodological features of the two studies mentioned above. Each case (crash-involved) drivers was matched to two control drivers sampled at the same location, day of week and time of day (and time of year, as controls were sampled a week after the crash). Unlike the previous studies, the modern statistical analytical technique of logistic regression was used to produce estimated risks. This enabled the risks associated with age, gender and other attributes to be controlled for.

There have also been modern studies making use of controls sampled in the course of surveys of drivers on the road at the same general times and days as cases, but not
matched by precise location. These studies include an American study by Zador et al. (6), a New Zealand study (7) and a German study (8). These studies are characterised by the use of data collected for other purposes (the control data to estimate prevalence of drink driving and the case data collected as normal part of crash data collection) and the use of logistic models to estimate risk curves while controlling for potentially confounding factors.

**Design and Estimation Issues**

The major hurdle for producing risk curves from alcohol case-control data is non-response bias, including bias due to refusal or abscondment of subjects or differential sampling correlated with exposure status (BAC level). Estimation is affected differently according to the nature of this non-response, and whether it occurs amongst cases or controls, discussed in the following.

Non-response among controls may take the form of refusal to participate when stopped by researchers and/or police or avoidance of the data collection altogether. It has been noted by various researchers (e.g., 4, 5) that drinking drivers stopped at the roadside are more likely to refuse to be breath tested than sober drivers. In New Zealand, drivers who attempted to avoid the breath testing site were pursued by police officers and virtually all were found to have high BAC levels (7). The effect of ignoring such non-response is to generate risk curves against BAC that are too steep. Most of the studies cited had response rates that would normally be considered as exceptionally good for voluntary surveys. For example, the lowest stated (Zador et al. (6) gave no response rate in their paper) response rates for controls was 92% in the Australian study (4) and 95% in the German study (8). The highest response rates were obtained in the New Zealand study (7) of close to 100%, 98% in the recent American study (5) and 97% in the Grand Rapids study (1). Nevertheless, the apparently strong relationship between drinking and non-response can still lead to quite substantial bias despite high response rates, as discussed by McLean et al. (4). Most studies did not attempt to impute values for the missing control BAC measures apart from the recent American study (5), which made use of measures from passive alcohol sensors, which were strongly correlated with active breath tests measures.

As for the controls, non-response among cases may take the form of refusal to participate when asked to by researchers and/or police, avoidance of the data collection altogether by absconding, or differential testing rates related to the driver's apparent BAC level. The German study (8) appears to be most affected by non-response among cases, with only 65% of cases having BAC measures available. This included refusals from drivers, hit-and-runs and police failure to test. Unfortunately, the uncertainties involved in imputing values for such a large percentage are considerable, particularly as the different forms of non-response produce potential biases in different directions. Refusals and abscondments by potential cases are likely to downwardly bias the risk curve (as drinking drivers may be more likely to refuse or abscond so that higher BACs are underrepresented amongst cases) and the police non-testing will upwardly bias the risk curve (lower BACs are underrepresented as the police don’t bother testing some of those they consider sober, discussed in (8)). The recent American study (5) imputed values for hit-and-run drivers by making use of BAC values from hit-and-run drivers who were later apprehended. This involves the assumption that apprehended hit-and-run drivers are not different in terms of the distribution of their BAC levels (and other relevant characteristics) from drivers who escaped. The New Zealand study (7) was untroubled by absconding drivers as the cases were fatally injured in the crash, but there were problems with differential non-response associated with the subsequent decision whether or not to measure BAC via post-mortem blood tests. There were 20% of case drivers from whom it
was decided (for various reasons) not to obtain such tests. However, police officers attending crashes make judgements about whether drivers had been drinking or not, generally being quite accurate. These data were used to impute BAC values for four drivers, appearing to negate the potential bias as indicated by the secondary measure (7).

Another approach has been suggested for dealing with unusually large proportions of non-responding drivers amongst whom drinking drivers are vastly overrepresented (9). This method derives a risk curve ignoring the BAC measurements of the case drivers while relying on extensive control data and certain assumptions about the shape of the risk curve.

Estimation Issues
Failure to control for confounding variables in the Grand Rapids study produced a dip in the risk curve for low BAC levels. Although hampered by not having access to the original data, Hurst et al. (3) used regression techniques to control for an apparently confounding factor, which removed the dip. Compton et al. (5) found a similar dip in estimates from their data until confounding factors were controlled for. The modern studies considered here had the advantage of the advancements in statistical techniques together with powerful computer technology to be able to account for (and estimate risk associated with) various risk factors simultaneously. A simple example of a confounding factor and its effect on analysis is presented in the following.

Table 1: The effect on odds ratios (odds of crash involvement at BAC>x divided by odds at BAC<x) of pooling data from groups A and B with different exposure (fictitious data)

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th></th>
<th>Group B</th>
<th></th>
<th>Pooled data</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BAC&lt;x</td>
<td>BAC&gt;x</td>
<td>Odds</td>
<td>BAC&lt;x</td>
<td>BAC&gt;x</td>
<td>Odds</td>
</tr>
<tr>
<td>Scenario 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>20</td>
<td>80</td>
<td>8.1</td>
<td>2</td>
<td>98</td>
<td>8.0</td>
</tr>
<tr>
<td>Cases</td>
<td>67</td>
<td>33</td>
<td></td>
<td>14</td>
<td>86</td>
<td></td>
</tr>
<tr>
<td>Scenario 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>2</td>
<td>23</td>
<td>7.7</td>
<td>2</td>
<td>98</td>
<td>8.0</td>
</tr>
<tr>
<td>Cases</td>
<td>10</td>
<td>15</td>
<td></td>
<td>14</td>
<td>86</td>
<td></td>
</tr>
</tbody>
</table>

Table 1 uses fictitious data to show the attenuating effect on odds ratios of failing to identify driver groups where one group (group A in this example) has high levels of exposure. These data were manufactured by allocating the control drivers of each of groups A and B into BAC classes so that group A had proportionately higher levels of exposure (driving at higher levels of BAC) than did group B. Cases were then allocated so that odds ratios (odds of crash involvement at BAC>x divided by odds at BAC<x) were approximately 8 for each group separately. The data were then pooled (such as would happen in an analysis that does not differentiate between groups A and B) and odds ratios for the pooled data were calculated in the last column. Driver group A could be considered as “drivers on the road midnight to 2am” and driver group B could be “drivers on the road 10pm to midnight”, groups that have large differences in levels of drink driving in New Zealand, for example (10). Table 1 shows fictitious numbers of drivers in each category (in Scenario 1, there are 200 drivers in each of groups A and B, divided evenly between cases and controls). The relative odds are calculated for exposure level BAC>x relative to BAC<x. The final three columns show odds calculated for the pooled data. It is clear that for both scenarios, pooling the data attenuates the relative odds, which are close to 8 for the driver groups A and B individually, but are estimated to be 5.5 and 7.2 when the data are pooled for Scenarios 1 and 2 respectively. Attenuation is most severe for Scenario 1,
where groups A and B are equally represented and group A has a much higher rate of driving at BAC>x (20% of controls have BAC>x compared to just 2% for controls in group B). Attenuation is less serious for Scenario 2, where group A has only a quarter the total number of drivers in group B and the difference in control drivers' exposure rate is less (now 8% of group A controls have BAC>x). This latter scenario is more likely to represent a real-life example, where a relatively small group has moderately elevated drink driving rates. So it is less crucial to identify a small group or a group with a modestly elevated exposure rate than to identify a larger group or one with larger exposure differences between cases and controls. If the relative risk measure is used instead of the odds ratio, attenuation is much less severe for Scenario 1 and almost disappears for Scenario 2.

Although the above example appears to be a good argument for including as many covariates as possible in the analysis, there are two important considerations. Firstly, the analysis method may not accommodate many covariates. A rule-of-thumb has been suggested that at least 10 cases (or controls, but normally cases are less numerous in alcohol studies) should be available for each parameter estimated when fitting a logistic regression model to ensure that variances are estimated correctly (11). Of course, inclusion of unnecessary covariates can also reduce the efficiency of the estimation (12). Secondly, the role of each covariate needs to be considered carefully to avoid problems such as overmatching, particularly where the covariate identifies a situation that is intermediate between exposure and crash occurrence (12). A (somewhat ridiculous) example would be to include as a covariate a judgement (made by researchers or police attending crashes and stopping control drivers) as to whether the case of control driver may be affected by alcohol or not. Such a covariate would severely attenuate any estimates of crash risk associated with alcohol.

The case-control studies discussed here all employed some degree of cluster sampling: cases were sampled in clusters of drivers involved in the same crash; controls were sampled in clusters of drivers stopped at the same site at the same time. The analysis should account for this clustering as assumptions of simple random sampling, as are used default to many regression programs, do not hold. Assuming simple random sampling typically leads to underestimated variances for estimators using clustered samples (10).

Etiologically Distinct Outcomes
As alcohol affects driving performance and behaviour, the focus of some case-control studies has been on what may appear to be the most etiologically relevant outcome: crash involvement, in which the driver is deemed to be “at fault” (1,8). Some drivers are assumed to be innocently involved in crashes caused by the actions of other “at fault” drivers, the former drivers’ involvement an etiologically distinct outcome from “at fault” involvement. There are, however, issues regarding the determination of responsibility for the crash, which depends largely on the judgement of the attending police officers and may be influenced by evidence of intoxication (leading to exaggeratedly steep risk curves as drinking cases become over-represented). In terms of crash causation, even the driver deemed innocent may contribute to the circumstances leading to the crash event. Supporting evidence for this includes elevated BAC levels found even amongst crash involved drivers who were deemed not “at fault” (13). Despite the probable bias in the estimation of odds ratios towards the null, combining the etiologically distinct outcomes of “at fault” and not “at fault” crash involvement avoids these issues of definition and accompanying problems in the interpretation of the results.
Sampling Issues
Statistical methods depend on assumptions of random selection. For this reason, it is essential that control data collected to provide estimates of prevalence of drink driving be collected at randomly selected sites, not sites chosen by police officers, for example, who may favour high volume sites, sites that are convenient, or sites used previously for enforcement (and which may be avoided by drinking drivers for this reason). There are various methods to choose sites randomly, including sampling squares from a grid placed on a map (e.g., 10), or listing all possible sites and sampling from the list. In practice, sites generally need to have particular characteristics to be suitable for safe surveying of passing traffic, so the grid method will involve a second step in which some initially sampled sites that are later deemed unsuitable are replaced. Although some sites will also have too low traffic volume (particularly late at night) to be sampled, it is possible that there may be elevated drink driving rates at lower volume sites, as was found in New Zealand (14), so it is desirable to include some low volume sites in order to provide a more complete indication of driver BAC levels on the road network.

Study Base
Related to sampling issues for selecting the controls is consideration of the study base, which is the “segment of person time in which diseased (or injured) subjects become cases” (15). In the context of alcohol case-control studies, “person time” can be interpreted as “person time driving”, which can be approximated by counts of case and control drivers either as counts of crash involvements (for the cases) or counts of control drivers passing roadside points (who are stopped and interviewed). As cases and controls studied should share the same base experience, restriction of control driver sampling to only high volume roads, for example, implies that cases should only be selected from such roads. Likewise, if control data are sampled only on Friday and Saturday nights, then only cases crashing at these times should be included in the study unless there is reliable evidence that drivers sampled at other times are virtually identical in terms of relevant characteristics (most importantly, BAC). If the cases have such restrictions, then it is the controls that need to be similarly restricted.

Summary
Although alcohol case-control studies share the same design and estimation issues as other case-control studies, there are particular problems associated with the very steep dose-response risk curve (where risk of crash involvement increases very steeply with increasing BAC) together with strongly differential response mechanisms, whether generated by the drivers themselves (refusal to participate because they have been drinking and do not wish this to be detected) or by those collecting data (the police may only be interested in testing drivers they suspect may have been drinking, for example). Case-control studies of risk associated with alcohol need to address these issues if further light is to be shed on the nature of alcohol’s effect on crash involvement risk.

Disclaimer
This paper represents the views of its authors and not necessarily those of the Land Transport Safety Authority.
References


