Methodological Issues in Epidemiological Studies of Alcohol Crash Risk

1 H. Moskowitz, 2 R. Blomberg, 1 M. Burns, 1 D. Fiorentino, 3 R. Peck

1 Southern California Research Institute, Los Angeles, CA, USA, 2 Dunlap & Associates, Inc., Stamford, CT, USA 3 R. C. Peck & Associates, Oakland, CA, USA

Keywords
Alcohol, Epidemiological studies of Traffic Collisions, Logistic regression relative risk.

Abstract
A literature review examined methodological problems which have arisen in epidemiological studies of the role of alcohol in traffic collisions. The methodological problems resulted in varying estimates of collision relative risk as a function of blood alcohol concentration (BAC). Based on the literature review, an improved epidemiological study of crash risk was performed in Long Beach, California and Fort Lauderdale, Florida. The study arrived at considerably higher estimates of the relationship between BAC and collision crash risk than in prior studies.

Methods
The Second International Conference on Alcohol and Road Traffic was held in Toronto, Canada in 1953. In the keynote address, T. K. Ferguson reiterated complaints already voiced at the First International Conference that it was impossible to determine “what percentage of road traffic accidents are due to influence of alcohol. We have heard figures ranging from 1% to 50%….” (1) Many studies had been published describing the percentage of drivers found with alcohol in fatal, injury and property damage collisions. However, without having comparable figures for the prevalence of alcohol among non-collision involved drivers, conclusions could not be reached as to the causal influence of alcohol.

The first published report of a controlled epidemiological study was by Holcomb in 1938 (2) in Evanston, Indiana which compared alcohol levels in injured crash drivers with those from non-crash control drivers. 270 hospitalized injured drivers provided urine samples to be compared with breath alcohol samples from 1,750 control drivers. Forty-six percent of the injured drivers had alcohol present with 14 percent over .15 g/dl. Only 12 percent of the control drivers had alcohol present and 0.4 percent were above .15 g/dl. Holcomb did not perform a relative risk calculation but Hurst (3) subsequently did and concluded that the relative risk of an injury crash was three at .06 g/dl, four at .09 g/dl and ten at .12% g/dl.

Methodological problems limit the conclusions to be drawn from this study. 1) The urine samples from the injured crash drivers were collected for a three-year period by physicians who obtained samples, if they were not otherwise occupied. Thus, not all injured crash drivers in the hospital provided urine samples. 2). The results of the urine alcohol analysis from crash
involved drivers were compared to breath BAC’s from the control driver group. The low
correlation between urine and blood or breath alcohol samples would produce increased
variability.  3) Although the injured crash drivers had collisions at various times, the control
group drivers were obtained only during the evening hours of “greatest alcohol consumption”.  
Sampling of the control drivers occurred at eight locations, half near taverns. Sampling occurred
for one week and resulted in 1,750 control drivers with only 24 refusals to provide breath
samples.  4) Additional information was obtained including the age, sex, time of day and day of
week of the collision for the crash involved drivers. Similar data was also collected for the
control group drivers. The variation of these covariates with BAC was presented in univariate
analysis. However, the relationship of alcohol level to crash probability was not adjusted for
these covariates.

In the Holcomb study, the analysis was of relative risk for an injured driver collision. The
relative risk of involvement differs whether the collision involves only property damage or injury
or fatality or for all types of collisions. U.S. Department of Transportation data for 1999 (4)
indicates that while only 3% of drivers in property damage collisions have alcohol present, 5% of
the drivers in injury crashes and 23% of the drivers in fatal crashes have alcohol present.
Differences in the frequency of presence of alcohol in drivers as a function of crash severity
indicate that relative risk analysis for the different crash conditions will vary greatly. The
literature contains comparisons between studies which have failed to note that the probability of
crash involvement for all crashes is not the same as for only injury or fatal crash involvement.

The U.S. Department of Transportation estimates that alcohol is present in roughly 8% of all
crashes. Thus, even if it is assumed that the presence of alcohol is the causal factor in all alcohol
present collisions, 92% of all traffic collisions remain as due to other factors. The literature has
identified such factors as including age, education, gender, time and place of collision, weather,
etc.

In the Holcomb study univariate analysis demonstrated that many factors were differentially
present in crash versus non-crash drivers. However these factors were not controlled in the
analysis of the relationship between alcohol level and crash probability. Comparison of the
alcohol levels in crash and control groups require that the crash and control group be comparable
with respect to all other variables which determine crash probability. Such comparability can
be achieved either by sampling techniques for obtaining unbiased crash and control groups or by
statistical adjustment. Currently the most frequently used statistical method is a logistic
regression utilizing covariate information.

Less than one dozen control studies have been performed to determine the probability of all
crashes, injury crashes or fatal crashes. Only two have examined all crashes as a function of
BAC prior to the study currently reported in this paper. The other studies were performed
utilizing either fatal or injury crashes or both. The two prior studies of all crashes were the
Toronto, Canada study by Lucas, et al. (5) in 1955 and the Grand Rapids, Michigan study by
Borkenstein, et al. (6) in 1962.

The Toronto study involved sampling from December 1951 until November 1952, Monday
through Saturday, from 6:30 p.m. to 10:30 p.m. Breath samples were obtained for crash drivers
and four or more non-accident involved control drivers passing the accident scene at similar
times in vehicles judged to be of similar age. All drivers were asked four questions and also to
complete a more thorough mail questionnaire, but only 60% returned the questionnaire. 433
collision drivers and 2,015 control drivers were enrolled in the study and used for relative risk
assessment on the role of alcohol in crash probability. Unfortunately, Lucas, et al. had
concluded that BAC’s of .05 g/dl or less did not influence crash probability. This was based on
a Toronto study utilizing police estimates of crash responsibility. Therefore, rather than using
only drivers at 0 g/dl as the base comparison for the relative risk analysis, Lucas, et al.
incorporated all drivers with BAC’s from 0.0 g/dl to .05 g/dl in the base comparison group. Lucas reported that drivers with BAC’s between .05 g/dl to .10 g/dl had a relative risk of accident involvement only 1.5 times that of the base group. Similarly, drivers with BAC’s from .10 g/dl to .15 g/dl had a relative risk of 2.5 and above .15 g/dl a relative risk of 9.7 times that of the base group.

The other epidemiological study of all crashes, prior to the current one, was the Grand Rapids, Michigan study reported by Borkenstein, et al., conducted from July 1962 to June 1963. There were 9,353 collision drivers, but due to limits in police and research personnel availability, some 2,764 crash drivers were neither interviewed nor breath tested. Other sources of omission including hit and run drivers and drivers who refused to cooperate.

While a controlled epidemiological study, it was not a matched case control study. The non-accident involved control drivers were not obtained at the same site or times where the collision had occurred for the crash group. Rather, the non-accident involved control drivers were obtained by sampling four drivers at each of 2,000 accident sites selected at random out of a pool of 27,000 accidents during the three years prior to obtaining the crash driver sample group. At each accident site, four control drivers were obtained regardless of the number of drivers in the original collision. The control sites were sampled at the time of day and day of week of the collision from the prior years. The direction of traffic from which the four control drivers were sampled were randomly determined rather than matching the direction of the crash involved drivers.

BAC data was available for 5,985 collision involved drivers and 7,590 control drivers. There was no matching of the collision site, time of day, day of week or direction of travel in the control group. Since there were four control drivers for each control site, regardless of the number of drivers in the original accident, the control driver group was over-represented with drivers from sites of single vehicle crashes. This is of significance since single vehicle and multiple vehicle crash drivers differ in a variety of characteristics including the greater frequency of alcohol in single vehicle accidents.

Other difficulties with the sampling procedure study exist. For example, in the Grand Rapids study 16.6% of the crash group was positive for alcohol. However, no account was taken in the analysis of hit and run drivers. While we cannot know what the hit and run rate in Grand Rapids, Michigan was in 1962 note that when such data has been collected, hit and run rates are a considerable proportion of all collisions. For example, during 1997 through 2000 the California State Highway Patrol (9) reports that 18% of all accidents in California were hit and run. In a study done by police in one California city, La Puente (10), an effort made to apprehend hit and run drivers reported that 65% of the apprehended hit and run drivers had positive BAC’s. If the hit-run rate in Grand Rapids in 1962 were similar to that in California today, 40% of all alcohol related crashes would have not been recorded in the crash related group. Failure to take the hit and run drivers into account leads to a serious underestimation of the alcohol related relative risk.

The refusal rate in the Grand Rapids study for providing either a breath sample or completing a questionnaire was 4.7% for the crash involved drivers versus 2.2% of the control drivers. Analysis of data obtained from questionnaires indicated that the probability of refusal was greatest for drivers reporting the higher drinking frequency. Thus, there is a sampling problem which would lead to an underestimated relative risk.

In addition to the sampling procedure problems, there are issues with the statistical analysis and conclusions. The Grand Rapids study did not compute a relative risk curve for crash involvement as a function of blood alcohol concentration, but generated a figure of the relative risk of causing a crash as a function of BAC. This involved a series of assumptions. 1) The study assumed that all 622 single vehicle crash drivers were responsible for their crash. The
BAC’s of single vehicle crash drivers were known from the breath samples at the site. 2) The authors then assumed that half of the 5,366 drivers involved in multiple vehicle crashes were at fault and half were not. They assumed that the blood alcohol distribution of the not at fault multiple vehicle drivers would have had the same BAC distribution as that of the control group. Therefore, they subtracted the BAC distribution of 2,683 multiple vehicle collision drivers using the control driver’s BAC and assumed that the remaining distribution of BAC’s were those of at fault multiple vehicle drivers. 3) They added the BAC distribution of the 622 single vehicle crash drivers to the BAC distribution of the 2,683 multiple vehicle drivers considered at fault creating a BAC distribution of 3,305 drivers considered at fault. 4) This BAC distribution was compared with the BAC distribution of the non-accident involved control group to permit computation of a relative risk curve of causing an accident.

Several of the assumptions used in producing the relative risk causation curve are questionable. One questionable assumption is that all single vehicle drivers were at fault without considering other possible factors. Another assumption is that half the multiple vehicle collisions drivers were at fault and half were not. Neilsen (7) demonstrated that the BAC distribution of drivers killed in collisions where the police assigned fault to the other drivers, nevertheless were almost twice as likely to have alcohol present than non-collision involved control drivers. This and a similar analysis by Hurst, suggests that drivers who are assigned no fault in collisions, but who have alcohol present, may fail to make avoidance maneuvers which non-alcohol present drivers would have used to avoid crashes. Clearly, assigning fault without analysis of individual collisions is questionable. In any case, producing a relative risk causation curve would result in a function not comparable with all other studies which have determined the relative risk of crash involvement. Using data from the Grand Rapids Study both Allsop (8) and Hurst (2) have produced BAC relative risk collision involvement estimates.

Another difficulty was the failure to take account of the covariate information. The Grand Rapids study performed single variable analysis of the role of age, drinking practice, gender, education level, ethnicity, marital status, occupation, etc., and demonstrated that nearly all these variables influenced accident probability. However, none of these covariates were utilized to ensure that the relative risk analysis of the relationship between alcohol level and crash probability was free of the influence of these variables. The consequence of the failure to control for these covariates in comparing two groups of drivers who vary in many characteristics that determine accident probability, produces a distorted relative risk probability curve. For example, one of the most frequently noted results in the relative risk figure of accident causation of the Grand Rapids study is a lower relative risk at BAC’s from .01 g/dl to .04 g/dl compared to 0 g/dl. Allsop suggested that the purported dip was a consequence of “the danger of comparing ill matched group”. Allsop and Hurst, in performing partial recalculations of the data, took into account some covariates and obtained collision involvement relative risk probabilities which had no dip at low BAC’s.

The literature review also examined epidemiological studies of injured and fatal drivers, and these studies provided additional insights into important methodological issues that were incorporated in the planning for our current study.

The current study was conducted in two cities, Long Beach, California and Fort Lauderdale, Florida. In both cities, specially trained teams of police officers and researchers went to the scenes of traffic collisions to obtain breath alcohol samples and complete questionnaires for the involved drivers. Sampling occurred seven days a week. In Long Beach, accidents were investigated from 4:00 p.m. to 2:00 a.m. and in Fort Lauderdale from 5:00 p.m. to 3:00 a.m. Prior data indicates that this time period reflects roughly 70% of all alcohol related collisions. Data was collected in Long Beach from June 1997 to September 1998, whereas Fort Lauderdale data was collected from September 1998 to September 1999. Approximately equal numbers of crashes were obtained from the two sites. Efforts were made to deal with some of the
methodological problems identified in the literature review. Thus, emphasis was placed on having teams go as rapidly as possible to the crash scene and attempt to apprehend hit and run drivers. Police officers were also equipped with passive alcohol sensing devices incorporated in flashlights so estimates could be made of BAC levels in drivers who would refuse to participate.

The study employed a matched case control design. The control driver group consisting of two control drivers for each crash driver, was obtained by sampling one week after the crash at the same crash location, time of day, day of week and direction of travel of the original crash drivers. The control drivers were obtained by randomly sampling from the traffic stream, going in the appropriate direction, at the appropriate time, at the appropriate site. A comprehensive questionnaire incorporated material utilized in the Grand Rapids questionnaire and additional areas such as sleep and drug use. Subsequently, a relative risk model was created utilizing logistic regression techniques and incorporating adjustments for potential sources of bias.

Results
Collectively, the two sites sampled 2,871 crashes involving 4,919 crash drivers. 603 crash drivers were hit and run. Of these, 104 hit and run drivers were apprehended within two hours of the collision and 94 provided a breath sample. More than 69% of the apprehended hit and run drivers had a positive BAC, typically in the higher ranges. Hit and run drivers constituted 12.26% of the total number of crash drivers.

An adjustment to the data was performed which assumed that the percentage of positive BAC’s in the entire 603 hit and run driver group would have the same relative frequency distribution of positive BAC’s as obtained in the apprehended hit and run drivers. We concluded, therefore, that there would have been 417 hit and run drivers with positive BAC’s. 3,971 crash drivers (excluding hit and run and refusals) included 681 with positive BAC’s or 17% of the non-hit and run drivers. Thus, we estimate that 1,098 drivers had positive BAC’s of which 681 were non-hit and run drivers who cooperated and 417 were the hit and run drivers believed to have positive BAC’s. Thus, the 1,098 positive BAC drivers represented 24% of the 4,574 crash drivers with ascribable BAC’s. The 417 positive BAC hit and run drivers represented 38% of the 1,098 positive BAC drivers.

Another source of methodological variability, which we attempted to control, were drivers who refused to participate. 330 or 7.65% of the crash drivers and 213 or 2.12% of the control drivers refused to participate. Clearly a differential refusal rate for crash involved drivers. In our work with the passive alcohol sensors, we established that the sensor scores had a .82 correlation with the BAC score in drivers who cooperated by giving BAC samples. Based on this information we were also able to impute BAC scores for those subjects who refused to participate, either in the control or crash driver group.

The statistical analysis which was undertaken had three major sources of correction to the obtained original raw data which produced an adjusted relative risk curve. The three sources of correction were adjustments for hit and run drivers, for non-cooperating drivers, and for the information obtained in all the covariates both from the questionnaire and from the sampling procedure. An initial relative risk analysis was performed of crash involvement utilizing the raw data without reference to any covariate or other source of error. Of interest was that this produced a relative risk function which is fairly similar to that found by re-analyzing the Grand Rapids data to determine relative risk of crash involvement rather than crash causation.

Subsequently, we corrected the raw data by performing a logistic regression incorporating all the statistically significant covariates and determined that the relative risk function was greater than without the correction. Following the correction for the covariates we incorporated corrections for the missing data for the hit and run drivers and the non-cooperating drivers. This produced a very large increase in the relative risk function.
In the current study there was no evidence of any dip in the relative risk curve at low BAC’s. Any departure from 0 g/dl including .01 g/dl had a relative risk greater than one. Relative risk estimates at .02 g/dl, .06 g/dl, .10 g/dl, .15 g/dl, .20 g/dl, and .25 g/dl for the raw data without corrections were respectively .87, 1.13, 2.37, 7.61, 18.78, and 20.29. For the same BAC levels the corrected relative risk function which takes into account both covariates and missing data were 1.03, 1.63, 4.79, 22.1, 81.79 and 153.68.

These results are a validation of the importance of attending to methodological issues in sampling procedures and analysis of epidemiological studies. This study will not claim to have solved all sampling problems associated with such a large endeavor. For example, we had difficulty obtaining BAC’s for injured drivers from non-cooperating hospitals, and not every crash that occurred was sampled due to limitations in resources. Thus, it is likely that if one were willing to devote even more resources to executing a similar study in the future, the obtained relative risk function would be even higher.

References