

ALCOHOL AND RISK OF ACCIDENT

Crash Risk of Alcohol Impaired Driving

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Keywords

Alcohol, Crash Risk, Case-Control Study

Abstract

In order to determine the relative crash risk of drivers at various blood alcohol concentration (BAC) levels a case-control study was conducted in Long Beach, CA and Fort Lauderdale, FL. Data was collected on 4,919 drivers involved in 2,871 crashes of all severities. In addition, two drivers at the same location, day of week and time of day were sampled a week after a crash, which produced 10,066 control drivers. Thus, a total of 14,985 drivers were included in the study. Relative risk models were generated using logistic regression techniques with and without covariates such as driver age, gender, marital status, drinking frequency and ethnicity. The overall result was in agreement with previous studies in showing increasing relative risk as BAC increases, with an accelerated rise at BACs in excess of .10 BAC. After adjustments for missing data (hit-and-run drivers, refusals, etc.) the result was an even more dramatic rise in risk, with increasing BAC that began at lower BACs (above .03 BAC).

Introduction

The role of alcohol in motor vehicle crashes, which was identified as a traffic safety problem by the first decade of this century, remains a major highway safety problem. For example, in the U.S. in 2000, there were 16,653 alcohol-related fatalities, 40% of all traffic fatalities (1). NHTSA defines an alcohol-related fatal crash as one involving either a driver or non-occupant (e.g., pedestrian) who had a BAC of 0.01 grams per deciliter (g/dl) or greater in a police reported crash. While this represents a 25% decline from the 22,084 alcohol-related fatalities reported in 1990 (50% of the total), it is still an unacceptably large number. Moreover, in 2000, some 31% of all traffic fatalities occurred in crashes in which at least one driver or non-occupant had a BAC of 0.10 or greater.

The mechanisms by which alcohol affects individual skills related to safe driving have been studied using well-controlled laboratory experimentation. These laboratory experiments have examined a wide range of BACs from low to relatively high and have found that numerous driving-related skills are degraded beginning at low BACs. The assessment of the risk of crash involvement by drivers at various BACs has been carried out using epidemiological research

methods in which a comparison is made of the BACs of crash-involved drivers and similarly at-risk, non-crash-involved drivers. Perhaps the most widely cited epidemiological study of the crash risk produced by alcohol is the Borkenstein Grand Rapids Study (2). In this, and other similar studies, a relative risk function is determined that indicates the likelihood of a driver at a specified BAC becoming involved in a crash compared to similar drivers under the same conditions at 0.00 BAC. These relative risk functions have been widely used to set the legal limits for driving under the influence of alcohol.

The emphasis of much of this early research on the role of alcohol in contributing to traffic crashes focused on establishing a causal connection between use of alcohol and crash involvement. With the role of alcohol in causing crashes firmly established, attention has shifted to the issue of at what BAC level elevated risk first occurs. While the Grand Rapids study, and other similar epidemiological studies, contributed greatly to our understanding of the role of alcohol in crashes, it is possible to gain an improved understanding of the relative risk at various BACs through more robust research designs and multivariate analytic techniques. For example, in the Grand Rapids study the control drivers were not matched to the time and location or direction of travel of the specific crash-involved drivers. Also, the measurement of BAC level has improved greatly over the last 30+ years, statistical techniques have become much more sophisticated in their ability to take into account potentially confounding variables, and many of the previous studies failed to collect or to include in their analyses of relative risk many key covariates such as age, gender, alcohol consumption patterns and measures of fatigue known or assumed to be related to the use or effects of alcohol. Finally, the extended time since these earlier studies raises the possibility that a change in the driving and/or drinking environments may have influenced relative risk.

Thus, the availability of significantly improved breath alcohol measuring equipment, the possibility of improving the case/control design (based on insights gained over the years), and the potential advantages of modern analytic techniques provided the impetus for the present study. The study was designed to determine the relative risk of crash involvement by BAC level (controlling for other factors like age, gender, alcohol consumption, etc.) and the relative risk for major groups of drivers (e.g., gender and age).

Methods

A case-control study was conducted in Long Beach, CA and Fort Lauderdale, FL in which data was collected on drivers involved in crashes of all severities. Two drivers at the same location, day of week and time of day were sampled a week after the crash to constitute a control group. Relative risk models were generated using logistic regression techniques with and without the inclusion of covariates such as driver age, gender, marital status and alcohol consumption.

Sampling Procedures

Data from crash-involved drivers and matched non-crash-involved (control) drivers was collected in Long Beach, CA from June 1997 through September 1998 and in Fort Lauderdale, FL from September 1998 through September 1999. The collection protocol specified crashes were to be sampled during the late afternoon, evening and nighttime hours (4 PM to 2 AM in Long Beach and 5 PM to 3 AM in Fort Lauderdale) when drinking and driving is most prevalent. Two matched control drivers for each crash-involved driver were sampled by returning to the crash scene one week later at the same time as the crash, and stopping at random drivers on the same

roadway, traveling in the same direction as the crash involved driver. Drivers were first asked to answer a few survey questions (on drinking habits, prior DUI arrests, use of medicines, mileage, fatigue, trip origin, and demographics) and then were asked to provide a breath sample.

Results

Number of Crashes and Crash-Involved Drivers

A total of 2,871 crashes were sampled (1,419 in Long Beach and 1,452 in Fort Lauderdale). Overall, 14,985 drivers were approached for participation in the study. There were 4,919 crash (2,422 in Long Beach and 2,497 in Fort Lauderdale) and 10,066 control drivers (5,006 in Long Beach and 5,060 in Fort Lauderdale).

Sample Participation Rates

Of the crash involved drivers, 6.5% refused to participate in the study. Another 603 were classified as hit-and-run. Ninety-four of these were apprehended within two hours of the crash and provided a breath specimen, resulting in 10.3% (509) lost due to hit-and-runs. Thus, taking into account the un-recovered hit-and-runs and refusals, the overall participation rate for crash involved drivers was 93.5%.

The controls participated at an even higher overall rate of 97.6%.

Analytic Approach

The statistical analyses involved the following sequence: (1) analysis of missing data and potential selection bias due to subject non-participation and non-recoverable hit-and-run drivers; (2) univariate logistic regression of the unadjusted relationship between BAC level and relative crash risk; (3) analysis of site x BAC interactions; (4) multiple logistic regression analyses of the BAC crash risk relationship adjusted for various subsets of covariates, such as age, gender, drinking patterns and socioeconomic status; (5) evaluation of selected BAC x covariate interactions; (6) evaluation of the BAC crash risk relationship for three crash subtypes: late night (10PM or later), single vehicle and severity level (PDO vs. injury/fatality); (7) adjustments of relative risk curves for non-participation bias; (8) assessment of accuracy of the logistic regression equations in classifying drivers by case-control status; (9) calculation of confidence intervals for the relative risk estimates; and (10) calculation of relative risks for specific subgroups of a priori interest.

Relative Risk Models

The result was a family of relative risk models with varying levels of covariate inclusion. Three relative risk models are presented in this paper. They are shown in Table 1, below. The first model contains no covariates. This simple model most closely resembles the relative risk estimates from the Grand Rapids study as reported by Allsop (3), which are also shown in the last column of the table for comparison purposes.

Table 1: Relative Risk Models And Comparison with Grand Rapids Results

BAC Level	No Covariates	Non-Reactive Demographic Covariates	Final Adjusted Estimate	Grand Rapids*
.00	1.00	1.00	1.00	1.00
.01	.91	.94	1.03	.92
.02	.87	.92	1.03	.96
.03	.87	.94	1.06	.80
.04	.92	1.00	1.18	1.08
.05	1.00	1.10	1.38	1.21
.06	1.13	1.25	1.63	1.41
.07	1.32	1.46	2.09	1.52
.08	1.57	1.74	2.69	1.88
.09	1.92	2.12	3.54	1.95
.10	2.37	2.62	4.79	
.11	2.98	3.28	6.41	5.93
.12	3.77	4.14	8.90	
.13	4.78	5.23	12.60	4.94
.14	6.05	6.60	16.36	
.15	7.61	8.31	22.10	10.44
.16	9.48	10.35	29.48	
.17	11.64	12.74	39.05	
.18	14.00	15.43	50.99	
.19	16.45	18.31	65.32	
.20	18.78	21.20	81.79	
.21	20.74	23.85	99.78	
.22	22.07	25.99	117.72	
.23	22.51	27.30	134.26	
.24	21.92	27.55	146.90	
.25+	20.29	26.60	153.68	21.38

*From reporting of Grand Rapids Study data in Table 25 (a) of Allsop (1966).

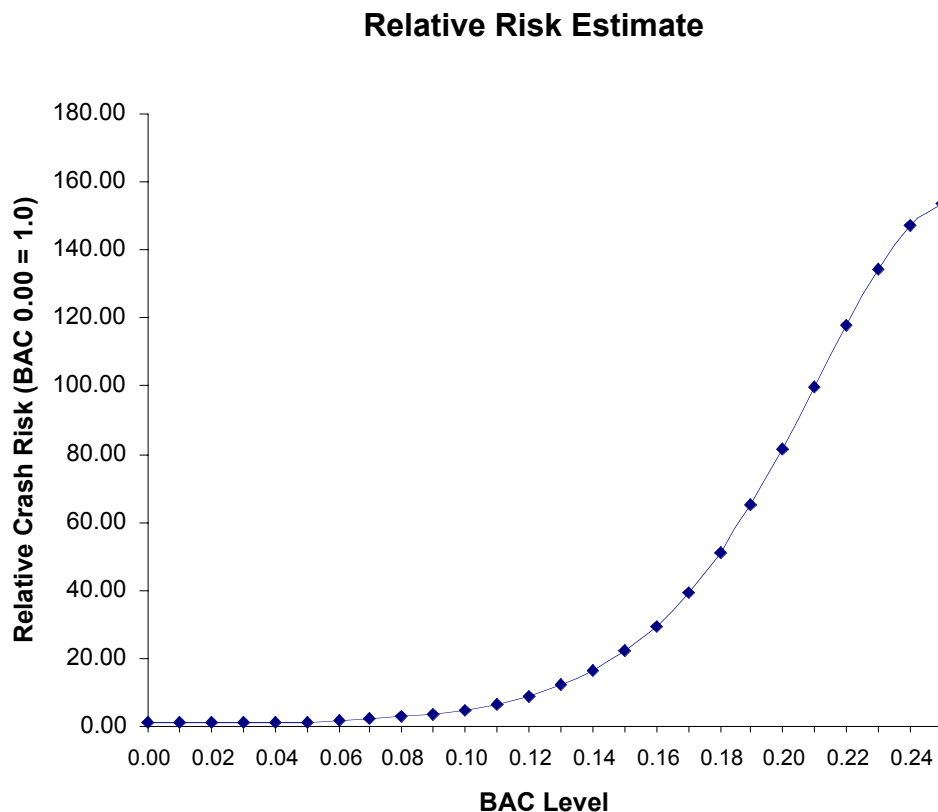
The second model includes age, gender and other demographic and socioeconomic covariates that do not have an obvious direct causal connection with BAC level. Other potentially “reactive” covariates, such as alcohol consumption, were not included in the final model out of concern that their inclusion would artificially inflate the risk estimates based on variables potentially related to the occurrence of alcohol-related crashes. The models with and without covariates generally showed elevated relative risk as BAC increases with a strongly accelerating rise at BACs in excess of 0.10 BAC.

The third model contains the non-reactive covariates and is adjusted for three sources of bias that were substantiated by the data analysis, namely, differential non-participation rates between the crash and control groups; missing covariate data resulting from differential non-participation rates and subjects failing to complete the interview; and hit-and-run attrition from the crash group.

In all three cases, the effect of the bias was to underestimate the crash risk associated with elevated BAC levels. This occurred because alcohol positive crash drivers were more likely to refuse to participate, and they were also less likely to complete the entire interview. Finally, hit-and-runs were shown to be associated with much higher levels of intoxication than drivers who remained at the scene. Fortunately, the study collected information that was used to estimate the magnitude of these biases and to develop appropriate corrections. The relative risk model using the covariates was therefore adjusted for these biases. The result showed greater risks at all BAC levels but particularly dramatic increases at high BACs.

Figure 1: Shows the final adjusted relative risk estimates displayed graphically

Figure 1



Contrary to a number of previous studies this final model showed no decrease in risk at the very low BAC levels (the Grand Rapids “dip”). This finding is consistent with Hurst’s (4) conclusion that the dip was an artifact of confounding and inadequate statistical analysis. The relative risk of crash involvement was significantly elevated beginning at 0.04 BAC. While the relative risk estimates for BACs below 0.04 were not significantly different from the risk at 0.00 BAC, they were still above 1.0 at each BAC level in the final adjusted model.

Relative risk models were also produced for major subpopulations of drivers (e.g., gender and age). Various univariate and multivariate approaches were used to produce risk estimates for

many variables of interest for which information was available. Space limitations preclude providing these results in this paper. An exhaustive presentation of the data collected and analyses performed are available in the full technical report for this study (5).

Discussion

Sophisticated statistical techniques were used to explore the relationship of numerous driver related variables in terms of relative risk at various BACs. Several covariates were found to significantly affect relative risk (age, gender, drinking variables, marital status, education, etc.). The general correspondence of the univariate risk results from the present study and the Grand Rapids study in the 1960s suggests that the risk of drinking and driving has not changed since that earlier assessment.

The adjustments to the risk estimates that were possible in this study show that the earlier risk estimates were likely a significant underestimate of the true crash risk produced by alcohol. This study provides the clearest up-to-date evidence for the risk associated with driving after drinking (even at relatively low BACs). It is concluded that the ability to adjust the relative risk model for potential biases reduced the attenuation of relative risk and provides a more accurate basis for decision-making.

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Methodological Issues in Epidemiological Studies of Alcohol Crash Risk

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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.

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Effects of Alcohol Countermeasures in Quebec on the Risk of Alcohol-Related Accidents

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Introduction

For almost 40 years, public policies about DUI have relied greatly on the results of the Grand Rapids Study (Borkenstein, 1974), which showed an increased risk of accident as the BAC increase, and on many studies replicating this study revealing age and gender differences.

Since 1997, new licensed drivers in Quebec are subject to a two year probationary period while they have a maximum of four demerits points and 0,00% BAC legal limit. Also we are facing a situation where the proportion of killed drivers with BAC over 0,08% shifted from 40% ten years ago to a minimum of 22,5% in 1999.

Objectives

The goal of this study is to analyze the alcohol-related risk of fatally injured drivers in a context of general decreasing contribution of alcohol to accident and the special case of graduated licensing system.

Methods

A major epidemiological study on the incidence of drugs in fatal collisions began in 1999 in Quebec. This study integrate two different analyses: a case/control study comparing fatally injured drivers (about 700) with a sample of drivers intercepted in a roadside survey (11,574) and a responsibility analysis for killed drivers with the method developed by Terhune (1992). Those two analyses allow to replicate Borkenstein's work and, as in the German study (Kruger et al, 1995), make sure that risk estimates are made only with drivers at-fault in the accident

Results

BAC data from the coroners for year 2000 are available since September and responsibility analysis is on its way and should be finished in late December. So the authors will be able to complete the analysis before March 15th, deadline for the full manuscript.

Discussion and Conclusion

Conclusions and discussion will compare risk of accident estimates in Quebec with those of previous studies in the context of graduated licensing system and the major decrease in alcohol related accident in Quebec. Also, source of data for responsibility is different (information from the police for the German study and evaluation by a panel of judges without knowledge of drugs consumption for Quebec study) so results may vary.

Comparing the Involvement of Alcohol in Fatal and Serious Injury Single Vehicle Crashes

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Keywords

Alcohol, single vehicle accident, fatality, drink driving

Abstract

Single vehicle crashes comprise about 30% to 40% of fatal and serious injury crashes in many jurisdictions. The study sought to identify the factors that contribute to an increased risk of occurrence of single vehicle crashes, an important step in developing countermeasures to address the problem. A case-control study was undertaken that compared single vehicle crashes within 200 km of Melbourne, Australia with control data from a random sample of cars and light trucks travelling through the same area. BAC data were essentially complete for drivers in fatal crashes and control drivers but were missing for almost half of the drivers in serious injury crashes. The effect of this missing data is likely to be one of increasing the proportion of positive BAC values in the known data for serious injury crashes and of inflating the calculated odds ratios for the involvement of alcohol in serious injury crashes. Possibly as a result of this factor, the proportion of drivers with BAC > 0.05, among the drivers with BAC known, was almost statistically significantly higher in serious injury than fatal crashes. However, among the drivers with BAC levels greater than zero, drivers in fatal crashes were more likely to have BACs of 0.150 and above than drivers in serious injury crashes. The odds ratio associated with BAC values of 0.001 to 0.050 was higher and statistically significant for serious injury crashes compared with fatal crashes. The extent of missing blood alcohol concentration data for serious injury crashes complicated the interpretation of the prevalence and risks associated with alcohol in fatal and serious injury crashes. There is a clear need for improvements to the collection and recording of blood alcohol data in non-fatal crashes.

Introduction

In Australia, about 40% of fatal crashes each year are single vehicle crashes (1). Overall, single-vehicle collisions comprise approximately 30% of road trauma (2). Single vehicle crashes often involve rollover (3) and impacts with trees and poles (2), which tend to result in high severity of injury. It is relatively difficult to prevent injury in these types of impacts and therefore identifying risk factors to enable prevention of these crashes is a high priority.

A case-control study was undertaken to

1. investigate single vehicle crashes to determine the circumstances and factors contributing to them
2. estimate the over-involvement (relative risk) of these factors
3. identify improvements in procedures for the investigation of road deaths and life threatening injuries
4. provide information from which countermeasures can be developed

Traditionally, Police investigation of these crashes has often been rudimentary, especially for crashes in which the driver has been killed (and so prosecution is not possible). Thus this case-control study aimed to identify the factors which contribute to an increased risk of occurrence of fatal single vehicle crashes, an important step in developing countermeasures to address the problem.

Methods

Single-vehicle crashes involving a collision with a fixed object (on or off road), collision with a parked vehicle or rollover that resulted in fatality or serious injury to occupants of a car or light truck were included in the study. The crashes occurred between 1 December 1995 and 30 November 1996. The study area was restricted to a 200 km radius of Melbourne, Victoria to allow attendance at fatal crashes by the Victoria Police Accident Investigation Section (now Major Collisions Investigation Unit). The population of the area is approximately four million people.

Two sources of crash information were used. For fatal single vehicle crashes, the Victoria Police Accident Investigation Section undertook the investigation, completed a questionnaire developed for the research project and compiled a brief for the Coroner. Copies of briefs and toxicology reports were provided by the State Coroner's Office. For serious injury crashes, a data file was created from the State Traffic Accident Record (STAR) database maintained by VicRoads that included only crashes in the study area during the study period. Single vehicle crashes were identified by use of Definition for Classifying Accident Codes. The resulting data file was modified to reflect the variables and their coding in the data file of control information.

A sample of 100 control sites was structured according to location, road class and time of day. The number of control sites in each of the Melbourne metropolitan area (40), rural roads (50) and rural towns (10) reflected the proportion of single vehicle fatal crashes occurring in the three areas in 1994. The number of control sites on each class of road was chosen to reflect the amount of travel on that type of road. The VicRoads 1994 Exposure Survey was used to estimate how much travel occurred in Melbourne, provincial cities and rural highways during the day and night on weekdays and weekends. Individual sites were selected to fit the above criteria by reference to the VicRoads State Directory.

During the 12 months of the study, drivers of cars (775) and light trucks (72) at the control sites were stopped by members of the Victoria Police Traffic Operations Group who recorded their licence details and conducted a preliminary breath test using a Lion SD-2 device. Motorists were then interviewed by researchers and contact details recorded for a follow-up telephone interview. Follow-up interviews were completed for 70% of motorists stopped.

Results

The general characteristics of the crashes are summarised in Table 1. Fatal and serious injury crashes were similar in terms of location and speed limit. Fatal crashes were more likely to involve male drivers, drivers not wearing seat belts and impacts with trees.

Table 1: General characteristics of the fatal and serious injury single vehicle crashes in this study

Crash characteristics	Fatal crashes	Serious injury crashes
Number of crashes	114	960
Number of persons killed/seriously injured	119	1170
Driver killed	73%	
Driver seriously injured		85%
Male driver	78%	66%
Driver not wearing seatbelt	19%	5%
Crash in metropolitan Melbourne	60%	63%
Speed limit 100 km/h or higher	48%	43%
Impact with tree	51%	34%
Impact with pole	29%	25%

BAC data were essentially complete for drivers in fatal crashes and control drivers but were missing for almost half of the drivers in serious injury crashes (see Table 2). The circumstances associated with BAC being recorded or missing for drivers in serious injury single vehicle crashes are summarised in Table 3. BAC data are missing for 132 crashes (14% of crashes and 28% of missing data) where the data file states that a preliminary breath test (but not an evidentiary breath test) was taken. It is highly probable that these BAC readings were not recorded because they showed a level below the legal limit (0.05). In an additional 231 crashes (24% of crashes and 49% of missing data), BAC data was not recorded and the data file states that no breath test was taken because the driver was injured and taken to hospital.

Table 2: Blood alcohol concentration (BAC level) for drivers in fatal and serious injury single vehicle crashes and control drivers

BAC level	Fatal (n=114)		Serious injury (n=960)		Control drivers (n=847)	
	Percent of known	Percent of all	Percent of known	Percent of all	Percent of known	Percent of all
zero	59	57	47	24	97	93
0.001 to 0.050	6	5	8	4	3	3
0.051 to 0.149	10	10	23	12	1	<1
0.150 and over	26	25	23	12	0	0
Missing		4		49		4

BAC data were more likely to be missing in rural than metropolitan crashes, for daytime than night-time crashes, for female drivers than male drivers, and for drivers aged over 35 than

younger drivers (see Table 4). This is consistent with the pattern of crashes in which alcohol is less likely to be involved. The extent of missing data was greater in crashes where the driver was less severely injured. For crashes in which the driver was not injured, only illegal BAC values were present.

Table 3: Circumstances associated with BAC being recorded or missing for drivers in serious injury single vehicle crashes

Circumstances	Number of crashes with BAC recorded	Number of crashes with BAC missing	Total number of crashes
Breath test taken (n=214)			
Evidentiary breath test taken	22	1	23
No evidentiary breath test	59	132	191
No breath test taken (n=745)			
Outside 3 hour limit	6	19	25
Injuries negate test	60	58	118
Injured (taken to hospital)	321	231	552
Nil reading	1	2	3
Not applicable	22	23	45
Evidentiary test taken	1	0	1
Unknown whether breath test taken (n=1)			
Unknown whether evidentiary breath test taken	0	1	1
Total	492	468	960

The pattern of missing BAC data in the serious injury crashes may underlie the finding that, among the drivers with BAC known, the proportion of drivers with BAC>0.05 was almost statistically significantly higher in serious injury than fatal crashes (45% versus 36%, $c^2(1)=3.48$, $p=.06$, see Table 2). However, among the drivers with BAC levels greater than zero, drivers in fatal crashes were more likely to have BACs of 0.150 and above than drivers in serious injury crashes (62% versus 42%, $c^2(2)=6.5$, $p<0.05$).

Crashed male drivers were almost twice as likely than females to have a BAC>0.05, while crashed drivers aged 60 and over were less likely to have BAC>0.05 than other crashed drivers. Crashed drivers in the metropolitan area were more likely to have BAC>0.05 than drivers in the rest of the study area (49% versus 39%).

Odds ratios for several levels of alcohol were calculated against the reference group of zero BAC (see Table 5). The odds ratios associated with having a BAC over 0.05 appeared somewhat larger for serious injury crashes than fatal crashes but were within the same confidence interval. The odds ratios for associated with a BAC between zero and 0.05 were somewhat larger and were statistically significant for serious injury crashes. The odds ratio for crashing with BACs between 0.050 and 0.150 was 33.4 for fatal crashes and 95.6 for serious injury crashes. At each BAC level, adjustments of the odds ratios to account for age and sex had little effect. While a

considerable number of drivers crashed with BACs of 0.150 and above, the odds ratio for this BAC level could not be estimated because there were no control drivers at this level.

Table 4: Extent of missing data by characteristics of serious injury single vehicle crashes

Characteristic	Number	% BAC Unknown
Metropolitan crashes	603	47.1
Rural crashes	357	51.5
Crash time		
Midnight- 6 am	226	28.3
6 am – noon	189	61.9
noon – 6 pm	276	60.8
6 pm - midnight	262	44.7
Male drivers	637	44.3
Female drivers	319	57.1
Sex of driver unknown	4	
Driver seriously injured	812	45.2
Driver other injuries	87	57.5
Driver uninjured	61	83.6
Driver age		
Under 25	419	45.3
25-59	451	50.1
60+	84	56.0

Discussion

Alcohol plays a large role in both fatal and serious injury single vehicle crashes. Illegal BAC levels (>0.050) were found in 36% of drivers of fatal crashes and somewhere between 24% and 46% of drivers in serious injury crashes (lower value if all missing data have BAC below 0.050 and upper figure if all missing data have BAC above 0.050).

The large amount of missing data (almost 50%) for drivers in serious injury crashes complicates the comparison of the prevalence of alcohol in drivers in fatal and serious injury single vehicle crashes. The effect of this missing data is likely to be one of increasing the proportion of positive BAC values in the known data for serious injury crashes and of inflating the calculated odds ratios for the involvement of alcohol in serious injury crashes. The results reflect this pattern, with the proportion of drivers with $BAC > 0.05$ among the drivers with BAC known, being almost statistically significantly higher in serious injury than in fatal crashes. Despite the extent of missing data, the results provide some evidence that alcohol plays a larger role in fatal than serious injury single vehicle crashes. Among the drivers with BAC levels greater than zero, drivers in fatal crashes were more likely to have BACs of 0.150 and above than drivers in serious injury crashes. Fatal crashes were more likely than serious injury crashes to involve male drivers and non-use of seat belts which are both associated with a higher prevalence of alcohol.

The extent of missing blood alcohol concentration data for serious injury crashes complicated the interpretation of the prevalence and risks associated with alcohol in these crashes. There is a clear need for improvements to the collection and recording of blood alcohol data in non-fatal crashes.

Table 5: Odds ratios for the involvement of alcohol in fatal and serious injury single vehicle crashes. Odds ratios are compared with BAC value of 0.00. Odds ratios in bold text are statistically significant at the 95% level. Confidence intervals are presented in brackets

BAC level	Fatal crashes		Serious injury crashes	
	Odds ratio	Confidence interval	Odds ratio	Confidence interval
BAC>0.05	120.6	(42.0-346.6)	167.1	(61.7-453.1)
adjusted for age group	118.4	(40.1-349.3)	189.4	(69.2-518.6)
adjusted for sex	116.3	(40.4-334.7)	174.1	(64.1-472.9)
BAC 0.001-0.050	2.1	(0.7-6.3)	6.0	(3.5-10.2)
adjusted for age group	1.8	(0.6-7.0)	5.2	(2.9-9.4)
adjusted for sex	1.7	(0.5-5.1)	6.5	(3.8-11.2)
BAC 0.051-0.149	33.4	(10.3-107.8)	95.6	(34.9-262.1)
adjusted for age group	23.3	(6.8-106.9)	98.2	(35.3-273.1)
adjusted for sex	22.3	(6.9-72.2)	103.2	(34.5-283.8)
BAC 0.150 and over	undefined		undefined	
adjusted for age group	undefined		undefined	
adjusted for sex	undefined		undefined	

Acknowledgments

The State Coroner's Office, Victoria Police Accident Investigation Section and Victoria Police Traffic Operations Group made this study possible. The technical input and support of the Project Steering Committee and many colleagues at MUARC is appreciated. Thank you to the on-road interviewers and to the drivers who agreed to be interviewed.

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Interactions between Alcohol, Cannabis and Cocaine in Risks of Traffic Violations and Traffic Crashes

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Abstract

Alcohol is well-known for the increased risk of traffic crashes it confers on drivers. The effects of other psychotropic drugs on crash risk are not so familiar, and have been harder to study, as have the risks faced by people dependent on more than one substance. We have been able to examine the driving records of samples of subjects beginning treatment for substance abuse at the Centre for Addiction and Mental Health (CAMH) during 1994. The sampling scheme resulted in approximately 90 people in each of seven treatment groups: alcohol, cannabis, cocaine, and all possible combinations of these substances. A control group of 518 drivers was selected randomly from the provincial driver records. Crash rates per year of driving were computed for 1985-1993 and 1995-2000. Adjusted relative risks (ARR) were computed for each substance and combination of substances using Poisson regression to control for differences in age and sex. A significant interaction was found for cocaine and cannabis ($P = 0.010$) pre-treatment, such that subjects dependent on both substances had a relative risk substantially lower than what is expected from each substance on its own. Alcohol, cocaine and cannabis all were associated with significant increases in crash risk. A separate model, which excluded the interaction term, gave estimates of ARR consistently below those obtained in the correct model, raising the possibility of mis-interpretation when the interaction is not recognized. A separate model, fitted on the collision frequencies in the post-treatment time interval, had no evidence of interaction; in this interval, no substance was associated with an increase in risk. Only being male and being younger were associated with higher crash risks.

Introduction

The effects of alcohol and illicit drugs on the task of driving have long been a major traffic safety concern. It is clear that alcohol impairs the ability to drive safely and increases collision risk, and there is increasing evidence that at least some illicit drugs do so as well (1,2). Thus, individuals who are heavy users of alcohol and illicit drugs are of special interest from a road safety perspective, and over the years research with these groups has yielded important and useful information (3).

An important phenomenon that has occurred in clinical populations of substance abusers over the past couple of decades has been the increasing frequency of multiple drug, or polydrug, abuse (4, 5). Historically, heavy or problem users of individual substances were most commonly

encountered in clinical populations, but more recently clinical groups are more and more likely to report polydrug abuse. This increasing multiple drug use may have important implications for road safety.

Laboratory studies of the effects of individual and multiple drugs are an important source of information about the potential effects of multiple drug use. These studies often reveal drug interactions, such that the effect of two drugs, when combined, is greater than the individual effects of the two drugs (6). Such drug interactions while driving could result in extremely hazardous levels of impairment, and we would predict that individuals who subject themselves to these interactions would be at substantially greater collision risk than individuals who use single drugs, even at an abusive level. Thus, among clinical groups of substance abusers, those who report polydrug abuse are expected to have more collisions than those who report abuse of a single substance. However, as yet no data have appeared to permit an evaluation of this hypothesis.

We report here a test of this prediction, using groups in treatment for the abuse of alcohol, cannabis, cocaine, or any combination of these substances. Our hypothesis is that individuals who report problems with more than one drug or drug class will have higher rates of collisions than drivers who report problems with only a single substance. Furthermore, we predict that the effects of this abuse may not be estimated from the effects of each drug on its own; i.e., that there may be a significant interaction evident for polydrug abusers.

Methods

Subjects were selected from those clients at CAMH beginning treatment for abuse of alcohol, cannabis and/or cocaine, most during the year 1994. (A few from late 1993 or early 1995 were also included.) Based on their declared dependencies when they began treatment, we identified three groups with >simple= dependence on either alcohol, cocaine or cannabis as well as four groups dependent on two or all three of these substances. We obtained information about traffic convictions and collisions for a random sample of approximately 90 people from each group. We also identified a control group of licensed drivers from the records of the provincial Ministry of Transportation, as representative of the general driving population. To be eligible for selection, members of all groups had to be aged 20-59 in 1994 when most subjects were beginning treatment and have an address in the greater Toronto area. Controls were frequency-matched to the age and sex distribution of the combined group of CAMH clients selected for study.

Ontario driving license numbers are based on name, sex and birth date. From this information, we identified the CAMH subjects with an Ontario driving record, stripping the record of all identifying information except sex and year of birth, after matching to obtain a copy of the driving record as far back as the year the person turned 16 or 1985, whichever was later. Records included the occurrence of collisions reported by the police and convictions up to December 31, 2000. This time interval was divided into pre-treatment and post-treatment intervals by the date the subject started treatment. For all control drivers, comparable intervals were taken to begin (post-treatment) and end (pre-treatment) at the median date of all subjects entering treatment. This occurred in June, 1994.

Crash and conviction rates per driver-year were computed for descriptive purposes before and after treatment by group. However, the fundamental analyses involved Poisson regression

models, which allowed us to correct for subjects= differences in age, sex and lengths of time at risk. The frequency of occurrence of events was modeled on the age, sex and abused substance(s) of individuals, using person-years at risk as the offset variable (7). Each subject was coded as dependent or not on each substance. For example, those coded as positive for alcohol would include subjects dependent only on alcohol, those also dependent on cocaine or cannabis and subjects dependent on all three drugs. This is effectively a 2x2x2 factorial design, which can examine the separate effects of alcohol, cannabis and cocaine as well as interactions between them in an efficient way, given the non-experimental nature of the study (8). In all analyses, comparisons with $P < 0.05$ were taken to be statistically significant.

Results

The basic descriptive data of the characteristics of each group appear in Table 1. A total of 590 CAMH clients and 518 frequency-matched controls were identified. Most groups were close to the targeted sample size; the group dependent on all three substances, however, contained only 49 subjects.

In all groups, young males predominate; the mean age is less than 35, with the group dependent on alcohol alone is older. The proportions of people whose driver records were found varied from 62% in the alcohol alone group to 79% in the cannabis alone group. The smaller number of clients with driver records resulted in a reduced sample size, with reduced power for subsequent analyses.

Table 1: Demographic, driving, crash and conviction characteristics by treatment group

Group (N)	Age " SD	% male	n (%) drivers	Collision rate (%)	
				Pre L	Post L
Control (518)	34.0"7.5	77.2	518	5.37	5.29
Alcohol (100)	39.3"8.7	74.0	71 (71.0)	4.89	2.88
Cannabis (96)	29.4"6.4	76.0	77 (80.2)	6.30	5.60
Cocaine (92)	32.5"7.0	78.3	68 (73.9)	7.70	4.08
Alc + Cann (85)	32.3"7.2	82.4	60 (70.6)	7.20	3.33
Alc + Coc (83)	32.6"7.0	73.5	51 (61.5)	6.51	3.40
Cann + Coc (85)	29.8"6.6	81.2	53 (62.4)	6.69	3.72
All three (49)	31.5"6.9	81.6	31 (63.3)	6.24	1.90

The results of Poisson regression appear as adjusted relative risks (ARR) of crashes for abuse of each substance relative to the control group of drivers dependent on neither substance. The results for pre-treatment collisions are reported in Table 2 and for post treatment collisions in Table 3.

In Table 2, we contrast the results of a model including the significant interaction between cocaine and cannabis abuse, and a model in which this interaction is ignored. The ARR for clients dependent on cannabis, cocaine and both substances have been estimated for each model.

The three way interaction B the joint effect of alcohol, cannabis and cocaine B was not statistically significant ($P = 0.54$); of the two-way interactions, only the joint effect of cannabis and cocaine was statistically significant ($P = 0.010$). For the model with this interaction, the relative risks for alcohol as a main effect, for cannabis and cocaine alone and in combination range from 1.14 for alcohol to 1.79 for cocaine without cannabis.

Table 2: Adjusted relative risks (ARR) and 95% confidence intervals for pre-treatment collision: models with and without an interaction term for cannabis and cocaine

Substance	With interaction		Without interaction	
	ARR	95% C.I.	ARR	95% C.I.
Alcohol	1.14	0.94, 1.38	1.22	1.01, 1.44
Cannabis, not cocaine	1.49	1.17, 1.89	1.18	0.98, 1.42
Cocaine, not cannabis	1.79	1.42, 2.25	1.44	1.20, 1.73
Cannabis and cocaine	1.52	1.16, 1.98	1.69	1.33, 2.16
Age (per year)	0.98	0.97, 0.99	0.98	0.97, 0.99
Sex (M vs F)	1.75	1.38, 2.21	1.73	1.37, 2.20

Table 3: Adjusted Relative Risks (ARR) and 95% confidence intervals for post-treatment collisions

Substance	ARR	95% C.I.
Alcohol	0.82	0.62, 1.08
Cannabis	1.05	0.81, 1.36
Cocaine	0.88	0.67, 1.15
Age (per year)	0.98	0.96, 0.99
Sex (M vs F)	1.50	1.13, 2.00

In the model which ignores this interaction, the ARR for cannabis without cocaine is 20% lower and not statistically significant ($P = 0.09$); for alcohol as a main effect it is 7% higher and gains significance ($P = 0.04$). For cocaine without cannabis the ARR is also 20% lower, but is still significant. For subjects abusing both cocaine and cannabis the ARR without interaction is 11% higher. (This simply the product of 1.18 and 1.44.) The ARR for age and sex are almost the same, are both significant and in the usual direction; i.e., younger drivers and men have higher risks.

The results for the model of collisions rates in the post-treatment interval is given in Table 3. None of the interaction terms among alcohol, cannabis and cocaine were statistically significant ($P > 0.20$), so this time it is appropriate to include only main effects. In the years post-treatment, none of the abused substances were significantly associated with collision risk. Only being younger (i.e., the relative risk for age being significantly less than 1) and being male were associated with increasing collision risk.

Discussion

Among the subjects in this study men and younger people predominate. This may explain why, even in the control group, collision rates are higher than in the general population of Ontario drivers, which are about 3% per year (9). Given these demographic characteristics the proportions with driver records was relatively low. In analyses reported elsewhere (10), we note that women, and people dependent on alcohol, were most likely not to be found in the driver database. The loss of power associated with a reduced sample size did not, however, affect results. For pre-treatment collision rates, increases in risk of 22% or more were detected as statistically significant, and 95% confidence intervals for ARR were relatively narrow. For post-treatment collisions, confidence intervals were wider, because the total person-years available for the same subjects was shorter for the follow-up interval than for the pre-treatment interval.

Factorial designs are ideal for the exploration of interactions, but they are also an efficient means of examining the effects of several factors in a single study when interaction is not present. In these results, the effects of alcohol abuse do not appear to modify the effects of cocaine or cannabis on collision risk. Since the joint effects of alcohol and other drugs have been the most commonly examined in the past, this is reassuring. The effect of alcohol is modest, however, especially in the alcohol only group.

There is evidence of interaction in these data between cocaine and cannabis; in the model that excludes the interaction term, the estimates of main effects are lower than they should be, so that a genuine effect appears >not significant=. Ignoring the interaction has distorted the relationships of these substances with collision risk. The interaction is, however, a negative effect in that subjects abusing both substances have a lower ARR for collision than might have been predicted from the main effects of these substances in the same model.

How can this happen? Albury et al (11) distinguish between substance abuse and driving under the influence which may apply here. These data cannot say whether subjects abusing both substances use them together or as alternatives, and we have no information on the use of these substance before driving or in the traffic collisions they experienced. Unlike many studies that have tested for alcohol and other drugs in crash victims, this study has used groups of subjects known to abuse these substances in a variety of circumstances, without specific reference to driving. These results are consistent with subjects= abusing drugs and driving as indicated by elevated relative risks, but also with their using only one of cocaine or cannabis, not both, when driving.

Further work is necessary to understand the patterns of use and how they relate to driving, before this interaction can be explained satisfactorily. At present, all that can be done is to take great care in situations such as those where exposure to multiple substances may influence results in ways that are hard to predict.

Acknowledgement

This work has supported in part by a grant from the Canadian Institutes for Health Research.

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