

ORIGINAL ARTICLE

Being "at fault" in traffic crashes: does alcohol, cannabis, cocaine, or polydrug abuse make a difference?

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Objective: To compare associations of alcohol, cannabis, and cocaine abuse and traffic crash risk for "at fault" crashes and all crashes.

Design: A historical cohort study.

Setting: Toronto, Ontario.

Patients or subjects: Subjects beginning treatment at the Centre for Addictions and Mental Health (CAMH) in 1994 for abuse of alcohol, cannabis, cocaine, and all combinations of these substances (n = 590, with 411 drivers). A control group consisted of 518 records from the Ontario registry of registered drivers, frequency matched for age and sex and residence.

Interventions: CAMH subjects took part in therapeutic programs. Pre-intervention (11 115 driver-years) and post-intervention intervals (8550 driver-years) were defined and compared.

Main outcome measures: Crash and collision rates, adjusted relative risks (ARRs) of crash involvement and of "at fault" crashes were computed using Poisson regression to control for variations in time at risk, age, and sex of participants.

Results: Pre-treatment, significant ARRs of 1.49 to 1.79 for all crashes were found for abusers of cannabis, cocaine, or a combination. ARRs increased by 10%–15% for "at fault" crashes. Post-treatment, all associations were very modest for all abuse types. Only younger and male drivers had a significantly increased risk, which was stronger for "at fault" than for all crashes.

Conclusions: Abuse of cannabis and cocaine pre-treatment was more strongly related to "at fault" crashes than to all crashes. Interaction between these substances means that the effects of combined abuse cannot be predicted from simple main effects.

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Alcohol is a familiar and still frequent factor as a cause of injuries.¹ This is especially true for road traffic crashes.²⁻⁶ It is well established that alcohol increases collision risk, and is a major factor in the injuries and deaths, not only of drunk drivers, but of other road users.⁷ This research has resulted in many initiatives to reduce drunk-driving, including legislation to establish legal limits, education, enforcement,⁸ and rehabilitation.^{9, 10}

Other psychoactive substances have also been found in drivers in road traffic crashes,¹¹⁻¹⁶ but the case-control methods that have been so useful in studies of alcohol are more difficult to conduct for other drugs. A major concern has been how to obtain samples for toxicological analysis. These analyses are much more difficult and expensive than those for alcohol, and thus are usually not done on drivers involved in crashes. When specific efforts to obtain these samples are made in the context of research, the problem of obtaining samples from a suitable control group has been extraordinarily difficult. Until a test of breath or saliva, analogous to the breath tests for alcohol, exists for other substances, this situation is unlikely to improve.

Some investigators have used other data sources for information on the potential road safety risks presented by illicit drugs. One very promising approach is to examine the collision experiences of groups of individuals presenting for treatment for abuse of these substances. This approach has provided much valuable information for understanding the effects of alcohol.^{17, 18} There are some promising data on the effects of other drugs in clinical samples, but as yet only a very small number of studies have addressed this question.¹⁹⁻²¹

In particular, the study of clinical subjects may permit the investigation of joint effects, as subjects often present with

polydrug problems. Finally, the experience of these subjects before and after treatment may provide some indication of the ability of treatment to alter crash risk.

We have examined the collision experience of a sample of substance abusers presenting for treatment at the Centre for Addiction and Mental Health (CAMH) in Toronto, Canada. With data from intervals before and after treatment, we have compared the experience of these drivers with a group of control drivers living in the same community. Associations were found, with an increased risk of overall crash involvement for cannabis and cocaine in the pre-treatment period.²² Examination of polydrug abusers revealed a statistically significant negative interaction between cannabis and cocaine. The effects of other combinations (for example, alcohol and cocaine) showed no evidence of interaction.²³

Assessing the effectiveness of treatment involved a direct comparison of crash experience before and after beginning treatment for drivers classified by primary substance abused.²⁴ Significant drops in crash occurrence were found after treatment for alcohol and cocaine, but not for cannabis.

It remains to examine the question of responsibility for crash occurrence more closely. Impairment of performance may be non-specific—that is, changes in the perception of risk, reaction time, etc may mean that risky circumstances and their consequences are harder for the driver to avoid, whether caused by the driver or not. However, if the impairment results in a suspension of good judgment so

Abbreviations: ARR, adjusted relative risk; CAMH, Centre for Addiction and Mental Health

that greater risks are taken, these drivers may be much more likely to cause the crashes in which they are involved. In the present paper, we wish to examine this question: is the association of abuse on crash risk the same for crashes when drivers are “at fault” as for all crashes in which these drivers are involved?

METHODS

We selected subjects beginning treatment at CAMH in 1994 for abuse of alcohol, cannabis, cocaine, and combinations of these substances. These are among the most commonly used addictive substances in the Ontario population and also the most commonly identified by individuals in substance abuse treatment in the province.^{25, 26} To be eligible, a person had to be aged 20–59 in 1994, live in the greater Toronto area, and have no history of prior treatment at CAMH. This age range was chosen to ensure that there was a reasonable opportunity before starting treatment for an individual to obtain a driver's license and some exposure to the risks of driving. The upper limit was more arbitrary, but few people begin treatment for abuse of these substances at age 60 or more.

The number of subjects admitted each year varies by substance or combination of substances; we chose random samples of 80–100 subjects for each type, seven groups in all. Subjects were selected from a computerized file based on face sheet information from the clinic records. This file contained basic demographic information including full name, birth date, sex, and address, as well as the substances identified by the client as his or her major problem substance(s).

Full name, birth date, and sex are sufficient to determine a unique license number in the Ontario registry of licensed drivers. These provincial records include all licensed drivers in Ontario, and document traffic convictions, reported collisions, and suspensions over time as well as the current level of demerit points, current address, and other information. For unlicensed individuals found to be driving, a record is also generated for administrative purposes, so a small number of drivers without licenses will also be found in this database.

A control group of licensed drivers was randomly selected by the provincial Ministry of Transportation from the registry of drivers just described from drivers living in the greater Toronto area and frequency matched to the total clinical sample by age group and sex.

The name, birth date, and sex of all subjects were used under confidential conditions to obtain driving record data for the interval 1985–2000 inclusive. Great care was taken to ensure the confidentiality of clinical and driver information; individuals in the Ontario Ministry of Transportation responsible for linkage were blind as to the derivation of the sample and the purpose of the study. Details of subjects' history and clinical experience were not available for the analyses of driver records. Identifying information (that is, the actual license number) was stripped from the records after we received the data. Subjects who could not be linked were not included in subsequent analyses.

The history of convictions and collisions for all drivers was examined. Rates were calculated for pre-treatment and post-treatment intervals, defined as follows:

- For *clinical* subjects, the interval from 1 January 1985 or the subject's 16th birthday (whichever came later) was used to define the beginning of the pre-treatment period. The date the subject began treatment defined the end of this period.
- For *control* subjects, the same algorithm was used to define the beginning of the interval. The median date of beginning treatment for all clinical subjects was used as the end of this period for all controls.

- For both *clinical* and *control* subjects, post-treatment began at the end of their pre-treatment interval and ended on 31 December 2000.

Crude crash rates were calculated as number of crashes per 100 person-years at risk. A similar formula was used for conviction rates, although the frequency of convictions was modified. We counted only once for multiple convictions occurring on the same date; in essence this meant that we counted the number of times a driver was stopped and charged with one or more moving traffic violations, not the true conviction frequency.

When the collision record indicated that the driver was charged with a conviction in connection with the crash, the driver was considered to be “at fault” for that collision. Other drivers involved in the crash may also have been charged with traffic violations, but without information on these drivers it was not possible to tell who was more “at fault” in such situations.

Crash frequencies for each driver were modeled by substance, age and sex, using Poisson regression with years at risk as the offset variable.²⁷ The resulting regression coefficients were used to estimate adjusted relative risks (ARRs) of crash occurrence. Separate models were developed for pre-treatment and post-treatment time intervals, for all crashes and for only “at fault” crashes. The 95% confidence intervals for the estimates of ARR were calculated; in the presence of interaction, the standard errors of ARR were calculated from the variance-covariance matrix of the model as described by Armitage and Berry.²⁸

RESULTS

The basic demographic data for drivers in the seven treatment groups and the controls are shown in table 1. Most groups were at or close to the target sample size; however, the group of patients seeking treatment for abuse of all three substances was too small to generate such a large sample, and contained only 49 subjects.

Overall, there were 411 subjects linked to the Ontario registry of drivers (69.7%) among the 590 clients initially selected from clinic records. The proportions of subjects linked varied from a low of 61.4% for alcohol and cocaine to 80.2% for cannabis alone. Drivers abusing alcohol alone had an average age of nearly 40 years, notably older than the other treatment groups with mean ages of 30–32 years. The proportion of males varied from 75.3% to 92.5%.

The crude rates for crashes and convictions and “at fault” crash rates per 100 person-years are shown in table 2 for pre-treatment intervals. There was a total of 610 crashes, 302 of

Table 1 Demographic characteristics by treatment group

Group (sample size)	No (%) with driver record	Mean (SD) age* of drivers	% Drivers who are male
Control (518)	NA	34.0 (7.5)	77.2
Alcohol (100)	71 (71.0)	39.3 (8.1)	78.9
Cannabis (96)	77 (80.2)	29.6 (6.4)	75.3
Cocaine (92)	68 (73.9)	32.4 (7.4)	83.8
Alcohol and cannabis (85)	60 (70.6)	32.4 (6.9)	85.0
Alcohol and cocaine (83)	51 (61.4)	32.3 (6.4)	78.4
Cannabis and cocaine (85)	53 (62.4)	30.3 (5.9)	92.5
Alcohol, cocaine, cannabis (49)	31 (63.3)	32.4 (7.2)	77.4

*Age on date of beginning treatment (treated groups) or median value of these dates (control group).

Table 2 Crude crash, conviction, and “at fault” crash rates per 100 driver-years at risk: pre-treatment

Group	Crashes* per 100 driver-years	Convictions per 100 driver-years	“At fault” crashes per 100 driver-years
Control	5.37	21.89	2.49
Alcohol only	4.89	16.78	1.98
Cannabis only	6.30	22.86	3.21
Cocaine only	7.70	40.93	3.44
Alcohol and cannabis	7.20	20.30	4.32
Alcohol and cocaine	6.51	25.54	4.30
Cannabis and cocaine	6.69	35.98	3.27
All three	6.24	24.30	3.57

*Crashes in which someone has been killed or injured or where there is property damage above \$700 (raised to \$1000 in January, 1998) must be reported to the police. The Police Accident Report is forwarded to the Ministry of Transportation for their records.

which were “at fault” in 11 115 person-years. The rates for control drivers were lower than for drivers in most of the addicted groups, both for all crashes and for “at fault” crashes. Conviction rates were highest for drivers in the cocaine only group, closely followed by drivers in the alcohol and cocaine group. Several groups (cannabis only, alcohol only, alcohol and cannabis) had conviction rates close to or lower than control drivers.

Comparable rates are given in table 3 for post-treatment intervals. There was a total of 366 crashes, 150 of which were “at fault” in 8550 person-years; the rates among drivers in addicted groups were usually lower than for controls, although the difference appears to be less marked for “at fault” crashes than for total crashes. Conviction rates are much lower in the post-treatment interval than in the pre-treatment interval for all groups, including the control group. Groups with higher rates include drivers abusing only cocaine, those abusing only cannabis, and those citing both substances. All other groups have rates close to or below those of control drivers.

These crude rates take no account of drivers’ age or sex, factors known to be strongly associated with crash risk in the general driving population and, in this study, matching variables in the selection of control drivers. Subsequent analyses, using regression techniques, included these factors to estimate ARR of crashing for each substance and combination of substances. Separate models, for total crashes

Table 3 Crude crash, conviction and “at fault” crash rates per 100 driver-years: post-treatment

Group	Crashes* per 100 driver-years	Convictions per 100 driver-years	“At fault” crashes per 100 driver-years
Control	5.29	13.57	2.00
Alcohol only	2.88	8.59	1.37
Cannabis only	5.60	19.89	2.10
Cocaine only	4.08	23.99	1.60
Alcohol and cannabis	3.33	10.28	1.43
Alcohol and cocaine	3.40	10.98	1.78
Cannabis and cocaine	3.72	20.84	2.17
All three	1.90	12.63	0.81

*Crashes in which someone has been killed or injured or where there is property damage above \$700 (raised to \$1000 in January 1998) must be reported to the police. The Police Accident Report is forwarded to the Ministry of Transportation for their records.

Table 4 Adjusted relative risks (95% confidence intervals) of total and “at fault” crashes pre-treatment: Poisson regression analysis

	All crashes	“At fault” crashes only
Alcohol (main effect)	1.14 (0.94 to 1.38)	1.29 (0.99 to 1.69)
Cannabis, no cocaine	1.49 (1.17 to 1.89)	1.68 (1.21 to 2.34)
Cocaine, no cannabis	1.79 (1.42 to 2.25)	1.98 (1.44 to 2.74)
Cannabis and cocaine	1.52 (1.16 to 1.98)	1.69 (1.09 to 2.30)
Age per year	0.98 (0.97 to 0.99)	0.98 (0.96 to 0.99)
Sex (male)	1.75 (1.38 to 2.21)	1.58 (1.14 to 2.19)

and restricted to “at fault” crashes, were computed. The results are shown in table 4 for the pre-treatment interval.

All models initially included interaction terms. The three way interaction between alcohol, cannabis, and cocaine was not significant for models for total crashes ($p = 0.54$ for pre-treatment and $p = 0.99$ for post-treatment) and was removed from each model. There was, however, one significant two way interaction in the pre-treatment model between cannabis and cocaine ($p = 0.004$). In each case, the relative risk was highest for people abusing cocaine and not cannabis, although it was statistically significant for people abusing one or both substances, as indicated by the 95% confidence intervals. Abuse of both substances, if there is no interaction, would increase the crash risk beyond the effect of each substance alone; however, it did not. The relative risk was approximately as high for both as for either substance on its own.

When the analysis is repeated after restricting outcome to “at fault” crashes, a similar pattern emerges. Again, there is no evidence of a three way interaction ($p = 0.47$) but there is evidence of interaction between cannabis and cocaine ($p = 0.015$). In this model the ARRs for the analysis of “at fault” crashes are consistently slightly higher for all abused substances than comparable terms in the analysis of all crashes. The ARR for age is the same, and that for males is reduced. Because the number of “at fault” events is smaller, the precision of these estimates is reduced, indicated by wider confidence intervals.

Alcohol exhibited an increased relative risk for all crashes (ARR = 1.14), which was not statistically significant. As indicated by the 95% confidence interval, the association, if it exists, is unlikely to be stronger than 1.38. Although the association was stronger (ARR = 1.29) when the analysis was limited to at fault crashes, it was not quite significant ($p = 0.06$). The 95% confidence interval suggests the true value of the ARR is unlikely to be less than 0.99 or more than 1.69. Both age and sex were consistently significant, with risks higher for younger drivers and for males.

The results of similar analyses conducted for the post-treatment interval appear in table 5. The pattern of crashes and “at fault” crashes is quite different in this period. None

Table 5 Adjusted relative risks (95% confidence interval) of total and “at fault” crashes post-treatment: Poisson regression analysis

	All crashes	“At fault” crashes only
Alcohol	0.82 (0.62 to 1.08)	0.95 (0.63 to 1.46)
Cannabis	1.05 (0.81 to 1.36)	1.00 (0.68 to 1.49)
Cocaine	0.88 (0.67 to 1.15)	1.01 (0.68 to 1.50)
Age per year	0.98 (0.96 to 0.99)	0.95 (0.93 to 0.98)
Sex (male)	1.50 (1.13 to 2.00)	1.91 (1.18 to 3.11)

of the three substances, either as main effects or interactions, exhibited a significantly different risk of crashes relative to controls. The model in table 5 includes only main effects to reflect the lack of evidence for interaction. The same is true for “at fault” crashes. Age and sex continue to have statistically significant associations with crash risk, and in the usual directions: younger drivers have higher rates and so do male drivers for total crashes and when only “at fault” crashes are considered. The reduction in numbers of crashes, however, means that the estimates of relative risk are less precise, making the 95% confidence intervals wider.

DISCUSSION

Our intention in this paper has been to examine the role of “fault” in the traffic crashes of drivers who abuse one or more of alcohol, cannabis, and cocaine. First, there are some additional questions to be addressed related to characteristics of the data, the design of the study, and the interpretation of the results.

Data

As noted earlier in this paper, the registry of licensed drivers in Ontario includes drivers without a valid license who have been convicted of a traffic violation or been involved as a driver in a traffic crash. In other analyses of these data,²⁹ we discovered that approximately 5% of the clinic group matched to driver records had never held a valid driving license. This occurred in the control group as well, but affected less than 1%. Although these proportions are different, each is too small to have much effect on results.

The proportions of CAMH subjects with driver records was lower than expected, based on proportions with a driver's license in the general population. This may mean simply that these subjects are less likely to hold a driver's license, but other explanations were also considered. To see what factors influenced subjects being linked to a driver record, we conducted logistic regression analyses. The only factor that was statistically significant was sex, with men more likely than women to be matched to a driver record ($p = 0.0007$). This may be, in part, a function of women changing their surnames after marriage or divorce, so they may be more difficult to match with driver records.²² It may reflect the fact that women may be less likely to hold a driver's license than men. In general, a failure to match may also reflect problems with the accuracy of clinic information; errors in the spelling of the name, birth date, or sex would result in no linkage with a driver record. For some names (for example, Macdonald *v* McDonald) this may be quite common; it will affect all client groups to the same degree. Although we were concerned about reduced power because of the smaller number of driver records, the width of confidence intervals is narrow enough for many estimates that ARR values of 1.45 or more are statistically significant.

It was not possible to obtain from the abstract of the driver record exact periods of licensure, so that young people obtaining a license later than age 16, drivers living out of the province for extended periods, or drivers complying with license suspensions have an overestimated time at risk. If we can assume that this error is non-differential; that is, that it will affect all groups of drivers in the same way, then the effect on estimates of relative risk will be conservative, so that they appear closer to 1 than they are.³⁰ If it is differential, the effects are harder to predict. Errors in the time at risk may be greater for clients abusing alcohol than for those not abusing alcohol, because the effects of alcohol use on driving receive more attention and are better known than abuse of other substances. Alcohol abusers would have less time at risk if they received more licence suspensions and complied with them; they may therefore appear to have lower crash risks

and weaker associations between alcohol abuse and driving than for other substances. From other analyses of these data the number of suspensions was greater in the records of clients than for controls; however the number charged with driving while the licence was suspended was also substantial, suggesting that the period of suspension is often not a period of non-exposure.³¹ Furthermore, patterns of suspension and evidence of violation of suspension were not consistent for clients abusing alcohol, cannabis, and cocaine. Most studies of alcohol and driving have examined people who are actual drivers, in road side check points or involved in traffic crashes.^{4,5} This study has examined people who have a high use of addictive substances, but no explicit information on whether and how heavily these are used in connection with driving, nor any data on how long they used these substances during the pre-treatment interval. If the period of use or abuse began after this interval had begun, the strength of the association would be under-estimated.

The crude rates for convictions decreased between pre-treatment and post-treatment intervals in all groups, including control drivers. This suggests that external factors affecting all drivers may be responsible: age related changes in the cohort over time, changes in the amount of driving done, or changes in enforcement. All subjects have aged in this interval, and one would expect to see comparable changes in crash rates as in conviction rates if this were responsible. In fact, crash rates rose slightly for control drivers. We have limited data on exposure; when two subgroups of these subjects, 110 from the treatment groups and 104 from the control group, were interviewed, treatment and control subjects reported similar amounts of driving ($p < 0.05$), which did not vary significantly between 1990, 1995, and 2000.³¹ Changes in enforcement practices could produce part of the decrease in conviction rates seen in all groups, and conceivably might explain the slight increase in crash rates for control drivers, if exposure has remained stable. All factors—maturity, exposure, and enforcement—may be acting to some degree; it is not possible to tease them apart in these data.

Design

From the way groups of subjects have been selected, this study is effectively a $2 \times 2 \times 2$ factorial design; that is, each substance (factor) is at two levels (present/absent) and in all possible combinations. The sample size for each combination of drugs is similar, except for the extremes: the group having problems with all three substances was smaller than any other group, and the control group, assumed to have problems with none of the substances, was much larger. Factorial designs are an efficient way to examine several factors simultaneously, assuming there are no interactions.³² They are also among the best ways to examine interactions, although we did not initially hypothesize that these would be present. In factorial designs, however, it is important to test for the existence of interactions before examining main effects.³² The interaction found suggests that the relationship of these substances with crash risk is more complicated than is often assumed.²³

This is one of the few studies we know of that has been able to examine the joint effects of two or three commonly used addictive substances using a factorial cohort design. Generalizing these results to other users of these substances, however, may be difficult, because subjects seeking treatment may be atypical. Such subjects may be heavier users; they may also be in a better position to take corrective actions to reduce their risk of harm. We considered that some of the elevated crash risk in the pre-treatment interval might be an artifact if the decision to seek treatment was precipitated by traffic crashes and traffic convictions shortly before begin-

ning treatment. However, when we repeated the analyses after excluding the year before treatment and any crashes or convictions that had occurred in that interval, the estimated ARRs were almost identical to the results presented here (data not shown).

Interpretation of results

The social and legal controls for cannabis and cocaine are very different from those for alcohol; while more sanctions exist for illegal substances, few are specific to drivers and the driving environment. The nature of the interaction suggests that, although abuse of either drug is associated with an elevated crash risk, abuse of both does not increase it further. This is consistent with the theory that the people who abuse any socially deviant substance have an elevated risk of traffic crashes that does not depend on the specific substance(s) abused. It is also consistent with other explanations. For example, a subject who abuses both substances cannot be assumed to use them simultaneously. The negative interaction we have observed might occur if most subjects in the groups abusing both drugs do not use them together, or at least not when driving. Further research, in which actual patterns of driving and use of these substances, alone and in combination, can be examined, may clarify what is happening.

There is no evidence of interaction between alcohol and either cannabis or cocaine; nor is it associated with a statistically significant increase in crash risk, either pre-treatment or post-treatment. This may reflect the sanctions that exist in the general driving population for alcohol that are specific to driving. Most studies linking alcohol to crash risk have been case-control studies, and have established high blood alcohol concentrations at the time of the crash. If many of the subjects who seek treatment for abuse of alcohol do not drink and drive, this might explain the weak association we have observed. The examination of these and other relationships will require more direct and personal accounts of exposure to driving and to substance use than are available in these data.

Consistently, the associations with alcohol, cannabis and cocaine were stronger when we looked only at “at fault” crashes than they were for total crashes in the pre-treatment interval. This is consistent with the theory that these drugs not only reduce one’s ability to avoid crashes but also increase one’s propensity to take risks, and to get into more hazardous situations. The consistent increase in ARR for alcohol, cannabis, and cocaine suggests that this alteration in behaviour is not specific to particular substances. If the effects of abuse of these drugs were limited to physiological factors such as slower reaction times, one might expect the crash risk to be increased to the same degree whether or not the driver was “at fault”. The increased crash risk associated with younger and with male drivers does not exhibit the same pattern. The ARR for age is the same for at fault and total crashes, and closer to 1 (though still significant) for male drivers.

In the results for crash risks post-treatment, there is no evidence that the risk of “at fault” crashes is higher from treated clients than for controls. There is also no significant interaction; indeed, none of the substance groups has an elevated risk of any sort. The only statistically significant factors are the traditional demographic ones that affect drivers in the general population—being male and being younger. As reported elsewhere²⁴ we have found lower crash rates post-treatment for some but not all of the substances examined. While it is tempting to suggest this is due to the effectiveness of treatment, we have resisted such conclusions at this stage. Too little is known about changes in driving and other factors in these subjects.

Key points

- Before starting treatment for substance abuse, users of cocaine and cannabis had significantly elevated crash risk, compared to control drivers.
- The associations were 10%–15% higher for “at fault” crashes than when no differentiation was made on who was responsible.
- Drivers abusing both substances (“polydrug” abuse) had the same elevation of risk as drivers abusing just one substance.
- In the post-treatment interval, only being male and being younger, not the substance that had been abused, were associated with elevated crash risk.
- Reasonable detection methods at the roadside, which are available for alcohol, need to be developed to clarify the relationships suggested.

These results suggest that abuse of cannabis and cocaine may raise the risk of crashes. The effect is consistently stronger for crashes in pre-treatment interval for which the driver is likely to be at fault. Driving after using cannabis and other illegal drugs is reported to be low in surveys,³³ but without adequate methods to detect specific substances at the roadside, this cannot be confirmed. Cannabis, in particular, may be crossing the line in Canada between being an illegal drug and guarded respectability; if the increased hazards found in our subjects apply to users in the general population, this trend will make development of good detection methods essential. Further work in circumstances where it is possible to examine patterns of use of these substances and how often use is associated with driving, is essential to help us understand and respond to these tentative relationships.

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REFERENCES

- 1 Chipman ML. Risk factors for injury: similarities and differences for traffic crashes and other causes. *Accid Anal Prev* 1995;**27**:699–706.
- 2 Holcomb RL. Alcohol in relation to traffic accidents. *JAMA* 1938;**111**:1076–85.
- 3 Lucas GWH, Kalow W, McColl JD, et al. Quantitative studies of the relationship between alcohol levels and motor vehicle accidents. *Proceedings of the 2nd International Conference on Alcohol and Road Traffic*. Toronto, Canada: Garden City Press Cooperative, 1955:139–42.
- 4 Borkenstein RF, Crowther RF, Shumate RP, et al. *The role of the drinking driver in traffic accidents*. Bloomington, IN: Department of Police Administration, Indiana University, 1964.
- 5 Haddon W Jr, Valien P, McCarrroll JR, et al. A controlled investigation of the characteristics of adult pedestrians fatally injured by motor vehicle accidents in Manhattan. *J Chron Dis* 1961;**14**:655–78.
- 6 Beirness DJ, Simpson HM, Mayhew DR, et al. Trends in drinking driver fatalities in Canada. *Can J Public Health* 1994;**85**:19–22.
- 7 US Surgeon General. *Surgeon General’s workshop on drunk driving: background papers*. Rockville, MD: US Department of Health and Human Services, 1989.
- 8 Lacey JH, Jones RK, Smith RG. *Evaluation of Checkpoint Tennessee: Tennessee’s statewide sobriety checkpoint program*. Washington, DC: US

- Department of Transportation, National Highway Traffic Safety Administration, 1999.
- 9 **Mann RE**, Anglin L, Wilkins K, *et al*. Rehabilitation for convicted drinking drivers (second offenders): effects on mortality. *J Stud Alcohol* 1994;**55**:372-4.
 - 10 **Wells-Parker E**, Bangert-Drawns R, McMillen R, *et al*. Final results from a meta-analysis of remedial interventions with drink/drive offenders. *Addiction* 1995;**90**:907-26.
 - 11 **Cimbura G**, Lucas DM, Bennett RC, *et al*. Incidence and toxicological aspects of drugs detected in 484 fatally injured drivers and pedestrians in Ontario. *J Forensic Sci* 1982;**27**:855-67.
 - 12 **Ferrara SD**, Zancaner S, Snenghi R, *et al*. Psychoactive drug involvement in traffic accidents in Italy. *Proceedings of the 11th International Conference on Alcohol, Drugs and Traffic Safety*. Chicago, IL: 1989:260-4.
 - 13 **Marzuk PM**, Tardiff K, Leon AC, *et al*. Prevalence of recent cocaine use among motor vehicle fatalities in New York City. *JAMA* 1990;**263**:250-6.
 - 14 **Stoduto G**, Vingilis ER, Kapur B, *et al*. Alcohol and drug use among motor vehicle collision victims admitted to a regional trauma unit: demographic, injury and crash characteristics. *Accid Anal Prev* 1993;**25**:411-20.
 - 15 **Brookoff D**, Cook CS, Williams C, *et al*. Testing reckless drivers for cocaine and marijuana. *N Engl J Med* 1994;**331**:518-22.
 - 16 **Walsh G**, Mann RE. On the high-road: driving under the influence of cannabis in Ontario. *Can J Public Health* 1999;**90**:260-3.
 - 17 **Schmidt W**, Smart RG, Popham RE. The role of alcoholism in motor vehicle traffic accidents. *Traffic Safety Res Rev* 1962;**6**:21-8.
 - 18 **Macdonald S**, Pederson LL. Occurrence and patterns of driving behavior for alcoholics in treatment. *Drug Alcohol Depend* 1988;**22**:15-25.
 - 19 **Smart RG**, Schmidt W, Bateman K. Psychoactive drugs and traffic accidents. *J Safety Res* 1969;**1**:67-73.
 - 20 **Mann RE**, Anglin L, Vingilis E, *et al*. Self-reported driving risks in a clinical sample of substance abusers. *Proceedings of the 12th International Conference on Alcohol, Drugs and Traffic Safety*. Cologne, Germany: 1993:860-5.
 - 21 **Mann RE**, Anglin L, Rahman S, *et al*. Does treatment for substance abuse improve traffic safety? A preliminary evaluation. *Proceedings of the 13th International Conference on Alcohol, Drugs and Traffic Safety*. Adelaide, Australia: 1995:817-20.
 - 22 **Chipman ML**, Mann RE, Macdonald S. The effects of addiction on risk of traffic crashes and convictions: a study of clients in addiction treatment programs. *Proceedings of the 12th Canadian Multidisciplinary Road Safety Conference*. London, ON, June 2001.
 - 23 **Chipman ML**, Mann RE, Macdonald S. Interactions between alcohol, cannabis and cocaine in risks of traffic violations and traffic crashes. *Proceedings of the 16th International Conference on Alcohol, Drugs and Traffic Safety*. Montreal, PQ, August 2002.
 - 24 **Macdonald S**, Mann RE, Chipman M, *et al*. Collisions and traffic violations of alcohol, cannabis and cocaine abuse clients before and after treatment. *Accid Anal Prev* (in press).
 - 25 **Ellis K**, Rush B. *Alcohol and other drug services in Ontario: results of a provincial survey, 1992*. Toronto, ON: Addiction Research Foundation, 1993.
 - 26 **Adlaf EM**, Paglia A. *1998 Ontario drug monitor: alcohol, tobacco and illicit drug use*. Toronto, ON: Centre for Addiction and Mental Health, 1999.
 - 27 **Stokes ME**, Davis CS, Koch GG. *Categorical data analysis using the SAS system*. Cary, NC: SAS Institute, 1995:208-12, 471-5.
 - 28 **Armitage P**, Berry G. *Statistical methods in medical research*. Oxford, UK: Blackwell Science, 1994:90-1.
 - 29 **Macdonald S**, Mann RE, Chipman ML, *et al*. Collisions and traffic violations of treatment groups for alcohol, cocaine and cannabis abuse, and a matched control group. Paper presented at the 24th Alcohol Epidemiological Symposium of the Kettil Bruun Society. Toronto: 28 May-1 June 2001.
 - 30 **Armstrong BK**, White E, Saracci R. *Principles of exposure measurement in epidemiology*. Oxford, UK: Oxford University Press, 1994:59-64.
 - 31 **Macdonald S**, DeSouza A, Mann R, *et al*. Driving behaviour of alcohol, cannabis and cocaine abuse treatment clients and population controls. *Am J Drug Alc Abuse* (in press).
 - 32 **Armitage P**, Berry G. *Statistical methods in medical research*. Oxford, UK: Blackwell Science, 1994:249-50.
 - 33 **Bierness DJ**, Simpson HM, Desmond K. *The Road Safety Monitor 2002: drugs and driving*. Ottawa, Canada: Traffic Injury Research Foundation, 2003.



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