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# The prevalence of alcohol, cannabinoids, benzodiazepines and stimulants amongst injured drivers and their role in driver culpability
# Part I: the prevalence of drug use in drivers, and characteristics of the drug-positive group

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**Abstract**

Blood samples from 2500 injured drivers were analysed for alcohol, cannabinoids, benzodiazepines and stimulants. Overall, three-quarters of drivers tested negative for drugs. Alcohol was the most frequently detected drug. Cannabinoids were also detected at high rates, but the majority of drivers tested positive for THC-acid, the inactive metabolite of THC. Benzodiazepines and stimulants were detected at low rates, and detection rates for combinations of drugs were also low. Males were more likely to test positive for drugs, especially alcohol and THC, whereas females were more likely to test positive for benzodiazepines. A similar proportion of car drivers and motorcycle riders tested positive for drugs, although riders were more likely to test positive for THC. Single-vehicle crashes were particularly associated with alcohol for both car driver and riders, and for riders, multiple-vehicle crashes were particularly associated with THC. 

*Keywords:* Alcohol; THC; Benzodiazepines; Stimulants; Injured drivers

## 1. Introduction

Of the many causes of road crashes, alcohol and drugs have attracted increasing attention. A Consensus Development Panel in the USA in 1985 concluded: 'most drugs that affect the [Central Nervous System] have the potential to impair driving ability' (Consensus Development Panel, 1985, p. 2618). For many years attention has been primarily focused on alcohol, with the relationship between alcohol and crash risk firmly established and accepted. However, with increasing concerns over the escalating use of licit and illicit drugs in Australia and other countries, this attention also needs to be directed to other drugs.

Research on the prevalence and role of drugs has included experimental and epidemiological approaches. The results of experimental studies show that drugs do impair performance, and hence have the potential to increase the risk of road crashes. However, the link between these results and crash risk is tenuous, and experimental research does not indicate the magnitude of the effect. This will depend on a range of factors including the doses of the drugs used in real-life driving situations.

The information generated by experimental studies thus needs to be verified by obtaining data from epidemiological studies. Henderson (1994), in a review of the effects of drugs on driving performance concluded

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that an understanding of the complex relationship between drugs and road safety could only be achieved through integrating the results of these two approaches. Simpson and Warren (1981) and Terhune (1986) suggested that data from epidemiological studies are required to answer two fundamental questions that experimental studies have not adequately addressed: how frequently do people drive under the influence of drugs under normal driving circumstances, and what is the relationship between the effects of drugs as seen in a laboratory and road crashes.

The present study is concerned with the first of these questions. There have been many reports concerning the prevalence of drugs in drivers. These have varied according to the types of drugs screened, the type of road user (fatally or non-fatally injured) and the size of the sample. Of particular interest here are those studies using relatively large samples of non-fatally injured drivers, which reported the prevalence of the four drugs/drug classes examined in the present study: alcohol, cannabinoids, benzodiazepines and stimulants.

Alcohol has been found to be the most frequently detected drug in injured drivers: the majority of studies reported that between 20 and 40% of drivers tested positive (Holubowycz et al., 1994 and Sugrue et al., 1995 in Australia). Moreover, drivers who tested positive for alcohol tended to have very high blood alcohol concentrations (BACs). Interestingly, the prevalence of alcohol was much higher in studies that examined fatally injured drivers, with percentages ranging between 40 and 60% (Drummer, 1994 and Haworth et al., 1997a,b,c in Australia). Studies have consistently found relationships between the prevalence of alcohol and variables such as driver gender and age, and the type and number of vehicles involved in the crash. Alcohol use was predominantly associated with male drivers aged between 20 and 40 years, involved in single-vehicle crashes. However, no consistent relationship was found between the prevalence of alcohol and the type of vehicle involved.

Cannabinoids have been the most frequently detected drugs in injured drivers after alcohol, with the percentage of drivers testing positive ranging from 7 to 15% (Stoduto et al., 1993 in Canada and Waller et al., 1997 in the USA). In these same studies cannabinoids, unlike alcohol, were more prevalent in non-fatally injured drivers. However, it is important to note the distinction between THC ($\Delta^9$tetrahydrocannabinol, the principal active compound in marijuana), and its major metabolite THC-acid (11-nor-9-carboxy-tetrahydrocannabinol) which is not psychoactive. The aforementioned studies reported the prevalence of all cannabinoids in drivers, without distinguishing between THC and its metabolites. These metabolites persist for some time in the body before they are eliminated, and can therefore be detected long after any psychological effect or impairment has disappeared. Australian studies by Perl et al. (1990) and Starmer et al. (1992) reported the prevalence of THC only in non-fatally injured drivers, and found much lower percentages (2.6 and 4.5%, respectively). As with alcohol, cannabinoid use has been predominantly associated with younger male drivers, and a study by Soderstrom et al. (1993) in the USA found that a higher percentage of motorcycle riders (32%) tested positive for THC compared with car drivers (2.7%).

Many studies either did not test for the presence of benzodiazepines or stimulants, or did not detect them in the sample. Those that did test for benzodiazepines generally found smaller percentages for these drugs compared with alcohol or cannabinoids, with values ranging from 2 to 5% in fatally injured drivers (Drummer, 1994 in Australia and Jeffrey et al., 1995 in Canada). However, some studies that examined non-fatally injured drivers reported percentages as high as 12% (Christophersen et al., 1995 in Norway; Stoduto et al., 1993 in Canada). Stimulants were reported at similar percentages of 1–4% (Kirby et al., 1990 in the USA; Perl et al., 1990 in Australia). In comparison, studies using fatally injured truck drivers found much higher percentages of stimulants, between 14 and 16% (Crouch et al., 1993 in the USA and Drummer, 1994 in Australia).

The present report describes the prevalence of drugs in a sample of non-fatally injured drivers. Four drugs/drug classes were chosen for analysis based on their prevalence of use and the likelihood of their playing a role in road crashes: alcohol, cannabinoids (measured as the presence of THC and/or THC-acid), benzodiazepines and stimulants. Moreover, the analyses conducted permitted determination of the actual drug and concentration for cannabinoids, benzodiazepines and stimulants. As blood testing of injured drivers is compulsory in South Australia, the data reported here comprise a representative sample. In addition, the sample is much larger than in other comparable studies. This provides a unique opportunity to examine the prevalence of these drugs, and to investigate the relationship between drug use and factors such as gender, age and the type and number of vehicles involved in the crash.

## 2. Method

### 2.1. Sample selection and procedure

Under Section 47(i) of the Road Traffic Act (1961) of South Australia, any person over the age of 14 years who attends one of 70 prescribed hospital Accident and Emergency units following a road crash must provide a blood sample. Blood samples are collected from all

such drivers who attend these hospitals after being involved in a non-fatal road crash, and who survive more than 30 days. For the present study, consecutive samples were collected in the periods from April to August 1995, and December 1995 to August 1996. These samples were analysed for the presence of alcohol, cannabinoids, benzodiazepines and stimulants. The time between the crash and blood sample collection varied across drivers. The mean length of time was 2.7 h, with a standard deviation of 3 h.

Blood test results from 2500 drivers were matched with their crash details from police crash report forms, and information was collected on the gender and age of drivers, and the type and number of vehicles involved in the crash.

### 2.2. Analytical methods

Whole blood samples were initially screened for the presence of cannabinoids (THC and THC-acid), benzodiazepines and stimulants using radioimmunoassay, and samples testing negative were eliminated. The cut-off values were as follows: 40 ng/ml for cannabinoids, 5 ng/ml for benzodiazepines and 50 ng/ml for stimulants. Presumptive positive samples were retained and subjected to further definitive testing to positively identify the drug or drugs present and to determine concentration. THC and THC-acid were analysed using gas chromatography/mass spectrometry, and the approximate limit of detection was 0.5 ng/ml. Benzodiazepines were analysed using gas chromatography or high pressure liquid chromatography, and the approximate limits of detection were 5 ng/ml for diazepam, nordiazepam, clonazepam, alprazolam and nitrazepam, 10 ng/ml for desalkylflurazepam, bromazepam, 7-aminoclonazepam and midazolam, and 100 ng/ml for oxazepam and temazepam. Stimulants were analysed using gas chromatography, and the approximate limits of detection were 5 ng/ml for amphetamine, methamphetamine and phentermine, and 10 ng/ml for ephedrine, pseudoephedrine and MDEA. Blood samples were analysed directly for alcohol using gas chromatography without prior screening. Blood concentrations for benzodiazepines and stimulants were interpreted as therapeutic or toxic, based on the concentration attained following medical use. Therapeutic concentrations are those observed following therapeutically effective doses. Toxic concentrations represent those associated with some form of toxicity. In instances where a toxic range has not been established, but the concentration of the drug exceeded therapeutic levels, or where the concentration fell between the therapeutic and toxic ranges, the concentration was interpreted as above therapeutic. For some stimulants (e.g. MDEA) this was a notional allocation only as these drugs are not used medically.

Table 1
Percentages of injured drivers testing positivefor the various drugs and drug combinations[a]

| Drug combination | % Positive ($n = 2500$) |
|---|---|
| Drug-free ($n = 1935$) | 77.4 |
| Alcohol only ($n = 214$) | 8.6 |
| Cannabinoids only ($n = 178$) | 7.1 |
| Alcohol + cannabinoids ($n = 74$) | 3.0 |
| Benzodiazepines only ($n = 46$) | 1.8 |
| Stimulants only ($n = 19$) | 0.8 |
| Alcohol + benzodiazepines ($n = 13$) | 0.5 |
| Stimulants + cannabinoids ($n = 7$) | 0.3 |
| Benzodiazepines + cannabinoids ($n = 4$) | 0.2 |
| Alcohol + stimulants ($n = 3$) | 0.1 |
| Stimulants + benzodiazepines ($n = 1$) | 0.03 |
| Alcohol + benzodiazepines + cannabinoids ($n = 3$) | 0.1 |
| Alcohol + stimulants + cannabinoids ($n = 2$) | 0.1 |
| Stimulants + benzodiazepines + cannabinoids ($n = 1$) | 0.03 |
| Alcohol + stimulants + benzodiazepines ($n = 0$) | 0.0 |
| Alcohol + stimulants + benzodiazepines + cannabinoids ($n = 0$) | 0.0 |

[a] This table includes drivers who tested positive for pseudoephedrine and for THC-acid.

## 3. Results

### 3.1. Prevalence of drug use

A range of drugs and drug combinations were detected in the blood samples obtained from the injured drivers (Table 1). However, over 75% of drivers tested negative for both alcohol and other drugs. Alcohol and cannabinoids were the most frequently detected drugs: 8.6% of drivers tested positive for alcohol only, and 7.1% tested positive for cannabinoids only. By comparison, the percentages that tested positive for benzodiazepines only or stimulants only were 1.8 and 0.8%, respectively. For most combinations of drugs, percentages were very low. The alcohol and cannabinoids combination was the most common, with 3% of drivers testing positive. When adding the various drugs and drug combinations, it was found that 22.6% of drivers tested positive for at least one drug including alcohol, and 10.3% tested positive for at least one drug excluding alcohol. Subjects positive for cannabinoids included samples that showed the presence of THC together with THC-acid, and samples positive for THC-acid alone. Cannabinoids were detected in 10.8% of drivers: 8% were positive for THC-acid alone and 2.8% for both THC-acid and THC. As THC-acid is not pharmacologically active, in subsequent analyses only drivers who tested positive for THC were treated as drug-positive. Similarly, drivers who tested positive for the stimulant

Table 2
Blood alcohol concentrations of injured drivers

| BAC (%) | Alcohol only ($n = 275$) | Alcohol + other drugs ($n = 34$) | Total sample ($n = 2500$) |
|---|---|---|---|
| 0.000 | – | – | 87.6% ($n = 2191$) |
| 0.001–0.049 | 15.6% ($n = 43$) | 14.3% ($n = 5$) | 1.9% ($n = 48$) |
| 0 05–0.079 | 9.8% ($n = 27$) | 8.8% ($n = 3$) | 1.2% ($n = 30$) |
| 0.08–0.149 | 33.8% ($n = 93$) | 31.4% ($n = 11$) | 4.2% ($n = 104$) |
| 0.150+ | 40.7% ($n = 112$) | 42.9% ($n = 15$) | 5.1% ($n = 127$) |

pseudoephedrine were not treated as drug-positive as this drug has very weak stimulant effects.

Table 2 shows the percentage of drivers who tested positive for different BACs, divided into those drivers who tested positive for alcohol only, and those who tested positive for alcohol in combination with other drugs. Most drivers (87.6%) had a zero BAC. A positive BAC was found in 12.4% of drivers and a positive and illegal BAC (over 0.05%) was found in 10.4%. Although most drivers recorded a zero BAC, those who did test positive tended to have illegal concentrations: of the 309 drivers who tested positive for alcohol, 84.5% had an illegal BAC, with a mean BAC of 0.132%. The percentage of drivers who tested positive for alcohol only in each BAC category was similar to that for drivers who tested positive for alcohol in combination with other drugs. The difference between these two groups in the distribution of BACs across categories was not statistically significant ($\chi^2_3 = 0.2$, $P > 0.05$).

A range of benzodiazepines were detected in the sample (Table 3). Some are marked as active metabolites of parent drugs. The most common benzodiazepine was nordiazepam (*N*-desmethyldiazepam) followed by diazepam, oxazepam and nitrazepam. Of the positive findings ($n = 111$), 93.7% were at subtherapeutic or therapeutic levels, and 6.3% were above the therapeutic level.

At least one benzodiazepine was detected in 68 drivers. Of these, 37 (54.4%) tested positive for one benzodiazepine only. An additional 21 drivers (30.9%) tested positive for two benzodiazepines, eight drivers (11.8%) for three and two drivers (2.9%) for four. However, as some of the benzodiazepine compounds are metabolites, testing positive for multiple drugs in this group does not necessarily imply consumption of multiple benzodiazepines by the driver. Of the 68 drivers who tested positive, the concentration was above the therapeutic range in nine (13.2%).

Table 4 shows that the most commonly detected stimulant was methamphetamine, followed by pseudoephedrine and amphetamine. Methamphetamine concentrations in the blood samples from the majority of drivers were at or above the therapeutic level, whereas with pseudoephedrine the majority of drivers recorded a subtherapeutic level of the drug. Of those drivers who tested positive for at least one of the stimulants ($n = 40$), 72.5% recorded a subtherapeutic or therapeutic level of the drug and 27.5% were above the therapeutic level.

A number of drivers tested positive for more than one stimulant. Of the 33 drivers who tested positive for stimulants, 28 (84.8%) tested positive for one stimulant only and five (15.2%) for two or more (three for amphetamine and methamphetamine, one for ephedrine and pseudoephedrine, and one for amphetamine, methamphetamine, ephedrine and pseudoephedrine). In total, ten drivers (43.5%) had concentrations of stimulants other than pseudoephedrine above the therapeutic range.

Table 3
Percentage of injured drivers testing positive for each type of benzodiazepine[a]

| Type of benzodiazepine | % Positive ($n = 2500$) | Impairment level |
|---|---|---|
| Alprazolam | 0.04 ($n = 1$) | Subtherapeutic $n = 1$ |
| Bromazepam | 0.08 ($n = 2$) | Therapeutic $n = 1$<br>Toxic $n = 1$ |
| Clonazepam | 0.2 ($n = 5$) | Subtherapeutic $n = 2$<br>Therapeutic $n = 1$<br>Toxic $n = 2$ |
| 7-Amino-clonazepam[b] | 0.1 ($n = 3$) | Therapeutic $n = 3$ |
| Desalkylflurazepam[b] | 0.04 ($n = 1$) | Above therapeutic $n = 1$ |
| Diazepam | 1.2 ($n = 30$) | Subtherapeutic $n = 6$<br>Therapeutic $n = 24$ |
| Midazolam | 0.1 ($n = 3$) | Subtherapeutic $n = 2$<br>Therapeutic $n = 1$ |
| Nitrazepam | 0.4 ($n = 9$) | Subtherapeutic $n = 2$<br>Therapeutic $n = 5$<br>Above therapeutic $n = 1$<br>Toxic $n = 1$ |
| Nordiazepam[b] | 1.8 ($n = 45$) | Subtherapeutic $n = 20$<br>Therapeutic $n = 25$ |
| Oxazepam | 0.4 ($n = 9$) | Subtherapeutic $n = 3$<br>Therapeutic $n = 4$<br>Toxic $n = 2$ |
| Temazepam | 0.1 ($n = 3$) | Subtherapeutic $n = 1$<br>Therapeutic $n = 2$ |

[a] In some cases drivers tested positive for more than one benzodiazepine.
[b] Metabolite.

Table 4
Percentage of injured drivers testing positive for each stimulant type[a]

| Type of stimulant | % Positive ($n = 2500$) | Impairment level |
|---|---|---|
| Amphetamine | 0.2 ($n = 5$) | Therapeutic $n = 5$ |
| Methamphetamine | 0.7 ($n = 18$) | Subtherapeutic $n = 1$<br>Therapeutic $n = 8$<br>Above therapeutic $n = 9$ |
| Phentermine | 0 04 ($n = 1$) | Therapeutic $n = 1$ |
| Ephedrine | 0.1 ($n = 3$) | Subtherapeutic $n = 1$<br>Therapeutic $n = 2$ |
| Pseudoephedrine | 0.5 ($n = 12$) | Subtherapeutic $n = 10$<br>Therapeutic $n = 1$<br>Above therapeutic $n = 1$ |
| MDEA (methylenedioxyethylamphetamine) | 0.04 ($n = 1$) | Above therapeutic $n = 1$ |

[a] In some cases drivers tested positive for more than one stimulant.

### 3.2. Gender and age characteristics of the drug-positive group

The age and gender characteristics of the sample according to drug combination are shown in Table 5. The numbers in the various drug categories differ from those reported in Table 1 as drivers positive for pseudoephedrine or for THC-acid without THC were not included. Overall, the differences in drug use between males and females were statistically significant ($\chi^2_9 = 90.7$, $P < 0.001$). In most drug groups higher proportions of drivers were male, and this was statistically significant for alcohol only and THC only (80.4 and 86% were male, respectively). Conversely, higher proportions of benzodiazepine-positive drivers either alone or in combination with alcohol were female (57.1 and 56.2%, respectively), but the difference was not statistically significant.

There was significant variation in age between the different drug classes and combinations ($F_{92\ 477} = 11.9$, $P < 0.001$). When compared with drug-free drivers, drivers who tested positive for alcohol only, THC only or the combination of alcohol and THC were significantly younger, while drivers who tested positive for benzodiazepines only were significantly older.

The age distributions of males and females for the various drug combinations were only significantly different for drivers testing positive for alcohol and benzodiazepines ($t = 3.0$, $P < 0.01$). That is, there was a statistically significant difference in the mean age of males compared with females, with males positive for alcohol and benzodiazepines very much older.

Table 6 describes the gender and age characteristics for drivers in each BAC range. A higher percentage of males tested positive for alcohol in each range, and the differences were statistically significant ($\chi^2_4 = 60.4$, $P <$

Table 6
Percentages of injured drivers in each BAC range according to gender and age

| BAC (%) | % Male | Mean age (SD) | | |
|---|---|---|---|---|
| | | Males | Females | Total |
| 0.000 ($n = 2191$) | 55.3 | 36.3 (18.7) | 35.4 (17.7) | 35.9 (18.3) |
| 0.001–0.049 ($n = 48$) | 81.3 | 30.7 (14.9) | 42.6 (16.1) | 32.9 (15.7) |
| 0.05–0.079 ($n = 30$) | 70.0 | 24.4 (5.1) | 37.9 (14.2) | 28.4 (10.7) |
| 0.08–0.149 ($n = 104$) | 73.1 | 31.4 (17.7) | 26.1 (8.7) | 30.0 (15.9) |
| 0.150+ ($n = 127$) | 82.7 | 31.4 (11.5) | 32.9 (12.3) | 31.6 (11.6) |

Table 5
Percentages of injured drivers testing positive for each drug and drug combination according to gender and age[a]

| Drug combination | % Male | Mean age (SD) | | |
|---|---|---|---|---|
| | | Males | Females | Total |
| Drug-free ($n = 2070$) | 54.8 | 35.9 (17.5) | 34.5 (16.7) | 35.3 (17.2) |
| Alcohol only($n = 275$) | 80.4 | 29.6 (11.9) | 32.6 (13.4) | 30.2 (12.3)** |
| THC only ($n = 50$) | 86.0 | 23.4 (5.9) | 26.3 (5.9) | 23.8 (5.9)** |
| Alcohol+THC ($n = 15$) | 80.0 | 22.3 (4.4) | 18.7 (1.5) | 21.6 (4.3*) |
| Benzodiazepines only ($n = 49$) | 42.9 | 50.5 (22.3) | 50.9 (21.5) | 50.7 (21.6)** |
| Stimulants only ($n = 16$) | 62.5 | 27.4 (8.4) | 39.3 (18.0) | 31.9 (13.6) |
| Alcohol+benzodiazepines ($n = 16$) | 43.8 | 54.1 (5.4)$^+$ | 34.4 (10.9) | 43.1 (16.1) |
| Stimulants+THC ($n = 3$) | 66.7 | 23.5 (0.7) | 22.0 (0.0) | 23.0 (1.0) |
| Benzodiazepines+THC ($n = 2$) | 50.0 | 21.0 (0.0) | 20.0 (0.0) | 20.5 (0.7) |
| Other combinations ($n = 4$) | 50.0 | 34.5 (13.4) | 26.5 (10.6) | 30.5 (10.9) |

[a] Asterisks denote statistically significant differences in age between the drug-free and drug-positive groups: *$P < 0.05$, **$P < 0.001$, and plus signs denote statistically significant differences between males and females: $^+P < 0.01$.

0.001). There was no significant difference in the mean BAC of males and females (0.133 vs. 0.130%, $t = 0.3$, $P > 0.05$). There was a significant difference in mean age across the different BAC ranges ($F_{42\ 495} = 4.1$, $P < 0.01$). However, only drivers with a BAC between 0.08 and 0.149% were significantly younger when compared with the alcohol-negative group (Tukey–Kramer $q = 4.7$, $P < 0.01$).

### 3.3. Type of vehicles involved

The data were examined in order to explore any differences between car drivers, motorcycle riders and truck drivers ($n = 2488$). Results from bus and ambulance drivers ($n = 12$) are not shown.

Table 7 shows the percentage of car drivers, riders and truck drivers testing positive for the various drug combinations. Due to the small sample size of truck drivers, their results were not analysed statistically. Although there were fewer drug andlor alcohol positives in this group compared with car drivers and riders, it is notable that 9% of truck drivers had such a positive result.

Overall, the profile of drug use was very similar between car drivers and riders. Although a higher proportion of car drivers tested positive for almost every drug and drug combination, the differences were not statistically significant. However, a higher proportion of riders tested positive for THC only (5.2 vs. 1.7%), and this difference was statistically significant ($\chi^2_1 = 12.4$, $P < 0.001$).

The percentage of riders in each BAC range was very similar to that for car drivers (data not shown). The mean BAC for car drivers was 0.134%, compared with 0.119% for riders. Differences between car drivers and riders in the distribution of BACs across categories were not statistically significant ($\chi^2_3 = 4.4$, $P > 0.05$).

### 3.4. Single- and multiple-vehicle crashes

Although most crashes that resulted in injuries involve more than one vehicle, a substantial number were single-vehicle crashes. The possibility that the involvement of drugs varied according to the number of vehicles involved was examined, and the results are shown in Table 8 for car drivers and riders.

Table 7
Percentages of injured drivers testing positive for the various drugs and drug combinations according to the type of vehicle[a]

| Drug combination | Car drivers (%, $n = 2164$) | Riders (%) | Truck drivers (%, $n = 55$) | Total (%, $n = 2500$) |
|---|---|---|---|---|
| Drug-free | 82.6 | 81.8 | 90.9 | 82.8 |
| Alcohol only | 11.3 | 10.0 | 5.5 | 11.0 |
| THC only | 1.7** | 5.2 | 0.0 | 2.0 |
| Alcohol + THC | 0.7 | 0.4 | 0.0 | 0.6 |
| Benzodiazepines only | 2.0 | 1.5 | 1.8 | 2.0 |
| Stimulants only | 0.6 | 1.1 | 0.0 | 0.6 |
| Alcohol + benzodiazepines | 0.7 | 0.0 | 0.0 | 0.6 |
| Stimulants + THC | 0.1 | 0.0 | 1.8 | 0.1 |
| Benzodiazepines + THC | 0 1 | 0.0 | 0.0 | 0.1 |
| Other combinations | 0.2 | 0.0 | 0.0 | 0.2 |

[a] Asterisks denote statistically significant differences between car drivers and motorcycle riders: $^{**}P < 0.001$.

Table 8
Percentages of injured drivers testing positive for the various drugs and drug combinations according to the number of vehicles involved[a]

| Drug combination | Car drivers | | Riders | |
|---|---|---|---|---|
| | 1 Vehicle (%, $n = 624$) | >1 Vehicle (%, $n = 1540$) | 1 Vehicle (%, $n = 86$) | >1 Vehicle (%, $n = 183$) |
| Drug-free | 63.6** | 90.3 | 73.3* | 85.8% |
| Alcohol only | 28.8** | 4.2 | 19.8** | 5 5% |
| THC only | 2.2* | 1.4 | 2.3 | 6.6% |
| Alcohol + THC | 1.3* | 0.4 | 0.0 | 0.5% |
| Benzodiazepines only | 1.8 | 2.1 | 3.5 | 05% |
| Stimulants only | 0.2 | 0 8 | 1.2 | 1.1% |
| Alcohol + benzodiazepines | 1.4* | 0.5 | 0.0 | 0.0% |
| Stimulants + THC | 0.2 | 0.1 | 0.0 | 0.0% |
| Benzodiazepines + THC | 0.0 | 0.1 | 0.0 | 0.0% |
| Other combinations | 0.5 | 0.1 | 0.0 | 0.0% |

[a] Asterisks denote statistically significant differences between single- and multiple-vehicle crashes: $^{*}P < 0.05$, $^{**}P < 0.001$.

For both car drivers and riders, there was a significant difference in the drug profile of single- compared with multiple-vehicle crashes ($\chi^2_9 = 297.8$, $P < 0.001$ for car drivers and $\chi^2_3 = 18.9$, $P < 0.01$ for riders).

Car drivers involved in single-vehicle crashes were significantly more likely to test positive for drugs than drivers in multiple-vehicle crashes, in particular for alcohol, THC, and alcohol in combination with either THC or benzodiazepines. Similarly, riders involved in single-vehicle crashes were significantly more likely to test positive for alcohol than riders in multiple-vehicle crashes. Although almost three times as many riders in multiple-vehicle crashes tested positive for THC only, the difference was not statistically significant.

## 4. Discussion

Consistent with other studies of non-fatally injured drivers, alcohol was the most frequently detected drug, although the percentage of drivers who tested positive was relatively low compared with previous results (Holubowycz et al., 1994; Sugrue et al., 1995). However, consistent with these previous studies, drivers who tested positive tended to have illegal BACs. Moreover, the prevalence of alcohol was still much higher than those found in studies using non-crash involved drivers (Bo et al., 1975; Honkanen et al., 1980; Ferrara and Rozza, 1985; Ferrara et al., 1990).

Cannabinoids were the most frequently detected drugs after alcohol. However, most drivers tested positive for THC-acid only, with a much smaller percentage positive for THC. Detection of the metabolite can only be used as an indicator of marijuana use, and it is unlikely that these drivers were impaired at the time of the crash. Again, the percentage of drivers who tested positive was lower than that reported in previous studies using non-fatally injured drivers (Stoduto et al., 1993; Sugrue et al., 1995; Waller et al., 1997). However, many of these studies did not distinguish between THC and its metabolites. The prevalence of THC in the present study was similar to that found in those studies which did make the distinction between THC and metabolites (Perl et al., 1990; Starmer et al., 1992). As with alcohol, the prevalence of THC in this study was higher than in studies using non-crash involved drivers, but again some of these studies did not report the prevalence of THC separately from the metabolises. Over 70% of THC-positive drivers in this study did not test positive for any other drug. This is inconsistent with prior research where a high proportion of drivers who tested positive for THC also tested positive for alcohol (Bailey, 1987; Christophersen et al., 1995). However, the results here show that when drivers tested positive for THC and another drug, the drug was most often alcohol, and these drivers tended to have high BACs.

Consistent with previous studies, the percentages of drivers testing positive for benzodiazepines and stimulants were low compared with alcohol and cannabinoids. Those studies using fatally injured drivers (Everest and Tunbridge, 1990; Drummer, 1994; Jeffrey et al., 1995) typically report lower rates of benzodiazepine and stimulant positive drivers than studies using non-fatally injured drivers (Stoduto et al., 1993; Christophersen et al., 1995).

Diazepam was the most frequently detected benzodiazepine along with nordiazepam, an active metabolite of diazepam and other benzodiazepines. In the Australian context, the benzodiazepines most associated with problems of abuse are reported to be flunitrazepam and diazepam. Flunitrazepam was not detected in this study. Together with the low percentage of concentrations above therapeutic levels, this suggests that benzodiazepine-positive findings were associated with therapeutic use rather than abuse of these drugs. However, the observation that benzodiazepines were usually detected at subtherapeutic or therapeutic levels does not mean that drivers were unimpaired. Experimental studies using psychophysiological tests, driving simulators and on-road driving have found evidence of impairment even at low doses (Linnoila et al., 1990; van Laar et al., 1992; Mattila et al., 1993).

The stimulants detected in this study include a range of prescription compounds, illicit compounds and drugs that have both licit and illicit uses. The most frequently detected drugs were methamphetamine, pseudoephedrine and amphetamine. Pseudoephedrine is used legitimately for the relief of cold and flu symptoms, and has weak stimulant effects. In most cases it was detected at subtherapeutic levels and is unlikely to have caused impairment. Ephedrine and phentermine are also used legitimately for hayfever and appetite suppression, respectively. In most cases they were detected at or above therapeutic levels, but the absolute number of drivers testing positive was very small. In Australia, methamphetamine is not available for therapeutic use. In 50% of cases this drug was detected above the notional therapeutic levels, implying abuse of the drug at the time of the crash. Amphetamine has both licit uses (Attention Deficit Disorder, narcolepsy) and illicit uses. As narcolepsy is a rare condition and ADD tends to be diagnosed in children, legitimate amphetamine use would be expected to be nil in this sample. Two other pieces of evidence suggest that the amphetamines detected here were used illicitly. Amphetamine is a metabolite of methamphetamine and these two were found together in all but one case, and amphetamine was found at or above therapeutic levels in every case. Thus, with the exception of pseudoephedrine, the stimulants detected in this study are likely to have been obtained illicitly and used for non-medical or recreational purposes.

There was a significant relationship betueen the prevalence of drugs and the gender of the driver. Overall, males were more likely to test positive for drugs, in particular for alcohol and THC. Females were more likely to test positive for benzodiazepines. These results accurately reflect patterns of drug use in Australian society, and are also consistent with past studies of non-fatally injured drivers (Soderstrom et al., 1988; Holubowycz, 1989; McLellan et al., 1995). Although a significantly higher percentage of males in this study tested positive for alcohol within each BAC category, the mean BAC of alcohol-positive males compared with alcohol-positive females was not significantly different. This suggests that although drinking and driving still appears to be a predominantly male phenomenon, females who drink and drive also do so at high BACs. This finding is consistent with Haworth et al. (1997a,b,c) who reported that a higher percentage of males tested positive for alcohol, but the proportions of males and females with BACs over 0.15% were not significantly different.

The relationship between the prevalence of drugs and driver age was also consistent with previous studies, with significant differences found in age depending on the type of drug being used (Warren et al., 1981; Cimbura et al., 1982; Holubowycz et al., 1994; Haworth et al., 1997a,b,c). Use of THC, either alone or in combination with alcohol, was significantly associated with younger drivers and use of benzodiazepines was significantly associated with older drivers. Moreover, younger drivers were more likely to test positive for alcohol compared with older drivers. Differences in the age and gender profile of drug-affected drivers have implications in identifying the groups of drivers that should be targeted in public health programs, and in developing suitable countermeasures to reduce the prevalence of drink- and drug-driving.

The prevalence of drugs was similar for car drivers and motorcycle riders, although riders were significantly more likely to test positive for THC. This is consistent with past research looking at non-fatally injured road users (Bailey, 1987; Soderstrom et al., 1993). It was also found that the percentages of drivers and riders testing positive for alcohol only were very similar, although the mean BAC was higher for drivers.

There are very few reports of the prevalence of drug use in non-fatally injured truck drivers (for example, Hendtlass et al., 1981). Studies of fatally injured truck drivers have found a high prevalence of alcohol, cannabinoids and stimulants (both licit and illicit) although benzodiazepines are rarely detected. The most frequently detected stimulants in these studies include methamphetamine, amphetamine, ephedrine and pseudoephedrine (Sweedler and Quinlan, 1990; Crouch et al., 1993; Drummer, 1994). Conversely, the truck drivers in this study had a low prevalence of drug use both overall and when compared with drivers and riders, and only one truck driver tested positive for stimulants. It is important to note that the number of truck drivers in this study was smaller than in previous studies. Moreover, these studies used fatally injured drivers, and the pattern of drug use may be different in those who survive road crashes.

This study also examined the relationship between the prevalence of drugs and the number of vehicles involved in the crash. In single-vehicle crashes the driver is usually responsible, whereas in multiple-vehicle crashes responsibility is more likely to be mitigated by factors such as other drivers, road, weather and vehicle conditions. If a drug does play a role in crashes, it would be expected that this would be reflected in a greater proportion of drug-positive drivers in single-vehicle crashes. For drivers, this was the case for alcohol, both alone and in combination with THC. For riders, this was true for alcohol, but for THC the converse was true. The results found for alcohol and THC are consistent with previous studies using non-fatally injured drivers (Stoduto et al., 1993; Waller et al., 1997).

It is important to note that many of the studies cited here reported on the drug prevalence in drivers from countries other than Australia. It is thus possible that the lower prevalence of drugs found in this study reflects different patterns of drug use in Australia. However, a study by Maxwell et al. (1997) compared the patterns of drug use in Australia and the Unites States and found them to be identical for marijuana and very similar for benzodiazepines and stimulants, although alcohol use was slightly higher in Australia.

A possible limitation in this study is the fact that there was a time-delay between the crash and blood sample collection. As blood concentrations of drugs change over time this can potentially confound interpretation of the data, especially where there was a substantial delay. Despite this limitation, the present study has several advantages over previous research conducted in this area. The sample is large and representative of injured drivers. The analytical methods used to test for drugs are also valid and reliable, comprising both screening and confirmatory tests. Many past studies have used screening tests only, which may either under- or over-estimate the prevalence of drugs in drivers. There have also been problems with the analyses performed to detect cannabinoids in drivers. Some only tested for the presence of cannabinoid metabolites, which can remain in the body for many days. The prevalence of these metabolites in drivers may not indicate recent use and consequently may not reflect impairment at the time of the crash. Moreover, some studies made no mention of the methods used to screen for cannabinoids, or reported results from screening tests only without confirmation and quantification of these results. Conversely, the present study

used confirmatory testing of initial screening results, and also quantified the results in order to determine actual drug concentrations. However, it is important to note that this study does not provide information on the relationship between drugs and crash risk. Reporting the presence of drugs in drivers does not necessarily mean that the drug contributed to the crash. Thus, further examination of the relationship between the prevalence and concentration of drugs and crash culpability is required in order to elucidate whether drivers who test positive for drugs are more likely to have been responsible for the crash compared with drug-free drivers. This is explored in Part II of this paper (Longo et al., 2000).

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