PERGAMON

Accident Analysis and Prevention 36 (2004) 239–248

ACCIDENT ANALYSIS & PREVENTION

www.elsevier.com/locate/aap

The involvement of drugs in drivers of motor vehicles killed in Australian road traffic crashes

Olaf H. Drummer [a,*], Jim Gerostamoulos [a], Helen Batziris [a], Mark Chu [a], John Caplehorn [b], Michael D. Robertson [c], Philip Swann [d]

[a] *Department of Forensic Medicine, Victorian Institute of Forensic Medicine, Monash University, 57-83 Kavanagh Street, Southbank, Vic. 3006, Australia*
[b] *School of Public Health, University of Sydney, Sydney, Australia*
[c] *Formerly of Department of Forensic Medicine, Monash University, Melbourne, Australia*
[d] *Road Safety Section, VicRoads, Denmark St, Kew 3101, Australia*

Received 29 May 2002; received in revised form 28 October 2002; accepted 25 November 2002

Abstract

A multi-center case-control study was conducted on 3398 fatally-injured drivers to assess the effect of alcohol and drug use on the likelihood of them being culpable. Crashes investigated were from three Australian states (Victoria, New South Wales and Western Australia). The control group of drug- and alcohol-free drivers comprised 50.1% of the study population. A previously validated method of responsibility analysis was used to classify drivers as either culpable or non-culpable. Cases in which the driver "contributed" to the crash ($n = 188$) were excluded. Logistic regression was used to examine the association of key attributes such as age, gender, type of crash and drug use on the likelihood of culpability. Drivers positive to psychotropic drugs were significantly more likely to be culpable than drug-free drivers. Drivers with Δ^9-tetrahydrocannabinol (THC) in their blood had a significantly higher likelihood of being culpable than drug-free drivers (odds ratio (OR) 2.7, 95% CI 1.02–7.0). For drivers with blood THC concentrations of 5 ng/ml or higher the odds ratio was greater and more statistically significant (OR 6.6, 95% CI 1.5–28.0). The estimated odds ratio is greater than that for drivers with a blood alcohol concentration (BAC) of 0.10–0.15% (OR 3.7, 95% CI 1.5–9.1). A significantly stronger positive association with culpability was seen with drivers positive to THC and with BAC $\geq$0.05% compared with BAC $\geq$0.05 alone (OR 2.9, 95% CI 1.1–7.7). Strong associations were also seen for stimulants, particularly in truck drivers. There were non-significant, weakly positive associations of opiates and benzodiazepines with culpability. Drivers positive to any psychoactive drug were significantly more likely to be culpable (OR 1.8, 95% CI 1.3–2.4). Gender differences were not significant, but differences were apparent with age. Drivers showing the highest culpability rates were in the under 25 and over 65 age groups.

Keywords: Forensic toxicology; Driving; Crash risk; Epidemiology; Alcohol; Drug impairment

1. Introduction

Alcohol is recognised as a leading contributor to road trauma. Alcohol over-involvement in crashes has been clearly demonstrated by a number of studies which show substantial increases in crash risk when the blood alcohol concentration (BAC) exceeds 0.10 g per 100 ml (%) (Borkenstein et al., 1974; Mounce and Pendleton, 1992; Robertson and Drummer, 1994).

While many drugs detected in crash victims are liable to impair driving skills, there is still uncertainty as to whether this translates to an increased crash risk. Likely drugs include cannabis; benzodiazepines; opiate-like drugs, such as heroin, morphine and methadone; and amphetamines and other CNS stimulants.

Experimental studies using instrumented cars have provided much useful information on the role of certain drugs on performance and error rates. Many CNS active drugs, particularly cannabis, benzodiazepines, barbiturates and the sedating antihistamines reduce lane control by increasing the standard deviation of lateral position (SDLP) while the drug is having its peak activity (Brookhuis et al., 1990; Laurell and Tornros, 1986; O'Hanlon et al., 1982; O'Hanlon and Volkerts, 1986; Ramaekers et al., 2000; Robbe, 1994). Logan et al. (2000) examined the extent of driver impairment of carisoprodol, a skeletal muscle relaxant, and its major metabolite meprobamate, which has sedative properties. The authors found that at therapeutic concentrations impairment was possible with symptoms of intoxication similar to alcohol.

* Corresponding author. Tel.: +61-3-9684-4304; fax: +61-3-9682-7353.
E-mail address: olaf.drummer@med.monash.edu.au (O.H. Drummer).

0001-4575/$ – see front matter
doi:10.1016/S0001-4575(02)00153-7

Unfortunately, these experimental studies may not accurately predict the effects of drugs under actual driving conditions. The association of drugs with crashes has been extensively investigated in epidemiological studies but with mixed results. Moreover, while providing evidence of an association, epidemiological methods cannot unequivocally establish that drug use causes adverse driving events.

Early studies of drugs and crash risk concluded that cannabis-users are more likely to be responsible for their crashes than drug-free drivers (Simpson, 1986; Simpson et al., 1982; Warren et al., 1981). The epidemiological design providing the strongest evidence of a causal association of drug use and crash risk is case-control responsibility analysis. Terhune et al. (1992) examined the cases of almost 2000 drivers fatally-injured in the USA to assess the contribution of drugs to crashes and found that the responsibility rate for amphetamine-positive drivers was higher than the drug-free group (Terhune et al., 1992). However, Terhune (Terhune et al., 1992) also found the responsibility rate for all THC-positive drivers was lower than that for the drug-free control group. Williams also found no evidence of an association between cannabis and the risk of injury but the numbers of drivers were small (Williams et al., 1985). In contrast, an investigation of trucker fatalities pointed to an adverse effect of Δ^9-tetrahydrocannabinol (THC) over 1 ng/ml, and other psychotropic drugs, on crash risk (Crouch et al., 1993).

A study of drivers injured in South Australia found drivers who tested positive for alcohol only, benzodiazepines only and the combinations of alcohol and THC and alcohol and benzodiazepines were significantly more likely to be culpable for the crash compared with the drug-free group (Longo et al., 2000, 2001). A small percentage of drivers who only tested positive for THC were culpable for the crash but this was not statistically significant. It was of interest that the blood THC concentrations were particularly low, most being <2 ng/ml. A study of injured drivers who presented to an urban emergency centre in Colorado found only alcohol and alcohol in combination with other drugs increased the likelihood they were responsible for the crash (Lowenstein and Koziol-Mclain, 2001). In this study cannabinoids including THC were only measured in urine limiting the time frame of last cannabis use.

Other case-control designs compare the drug use of drivers involved in crashes with other groups. A nested case-control design of over 200,000 drivers using driver's license files, police reports of injurious crashes, and health insurance records showed an increased risk of motor vehicle crash involvement in the elderly population using long acting benzodiazepines (Hemmelgarn et al., 1997).

A case-control study of persons involved in injurious crashes showed that use of anti-depressants and opioid analgesics by older drivers was associated with increased risk of injurious motor vehicle collisions, but not with benzodiazepines or sedating antihistamines (Leveille et al., 1994). This contrasted with another study that found the relative risk of injurious crash involvement for current users of any psychoactive drug was significantly elevated and the increase was primarily due to benzodiazepines and cyclic antidepressants (Ray et al., 1992).

Other studies of all drivers have failed to find a positive association of drugs with road crashes. A retrospective, hospital-based case-control study failed to find a significant difference between the prevalences of drugs (opiates, cannabinoids and amphetamines) in drivers injured in road crashes and those admitted to a hospital for other reasons (Marquet et al., 1998).

A study which linked prescription records with hospital admissions from road crashes showed that people who used minor tranquilizers in the past 3 months had a five-fold higher risk of a serious road accident (Skegg et al., 1979). A similar study showed the odds ratio was elevated for those persons taking benzodiazepines, particularly within a few weeks of the first prescription (Neutel, 1995). In contrast, a similar study showed no increase in accident risk with the use of benzodiazepines and sedatives (Jick et al., 1981). An association of drug use and motor crashes has also been shown by comparisons of the prevalence of drugs in crash victims with general drug use obtained from surveys. Other studies have surveyed drivers regarding their use of drugs while driving and their perception of possible impairment (Albery et al., 2000; Walsh and Mann, 1999; Wechsler et al., 1984).

Crash investigations have provided evidence of likely mechanisms of causality. Logan (1996) showed that in a population of methamphetamine positive drivers, predominantly culpable for accident causation, the use of methamphetamine most likely contributed to risk-taking behaviour or was a result of withdrawal-related fatigue and hypersomnolence (Logan and Schwilke, 1996).

It is clear that variable conclusions have emanated from various sources of any link of crash risk to use of one or more psychotropic drugs. Some studies have failed to demonstrate a convincing association of drugs with crash risk often because they lacked a suitable control population while others lacked statistical power because of their small population sizes, limited extent of toxicology testing or their assessment of culpability.

The present study was designed to avoid these problems. A validated method of responsibility analysis (Robertson and Drummer, 1994) was used to classify culpability of drivers killed in three Australian states over 10 years. The toxicological data on the incidence of alcohol and the various drugs for the 3398 subjects have been described elsewhere (Drummer et al., 2003).

2. Materials and methods

2.1. Study population

The study population is drivers killed in motor vehicle crashes in the three Australian states of Victoria (Vic.), New

South Wales (NSW) and Western Australia (WA). Only cases that were on-road motor vehicle crashes were included. Crashes that occurred off-road, or those classified as due to natural causes or suicide were excluded.

In Victoria these data were obtained from records kept at the Victorian Institute of Forensic Medicine and the State Coroners Office at Southbank. Drivers were identified on the basis of records obtained from the Victorian Institute of Forensic Medicine. These cases included Victorian drivers killed in road crashes from 1990 to 1999.

In the state of New South Wales (NSW) Coroner's case numbers and names of persons killed in motor vehicle crashes between January 1991 and March 1993, and from 1995 to 1999 were obtained from records kept at the Coroners Courts in Glebe and at Westmead, Sydney. Drivers were identified on the basis of records obtained from the State Coroners Office. Cases from regional NSW were included except for the years 1995 and 1996 when only cases from the wider Sydney metropolitan area were included.

In the state of Western Australia (WA) information on drivers killed in motor vehicle crashes between 1990 and 1992 and from 1995 to 1999 was obtained from records kept at the Perth Coroner's Office. Drivers were identified on the basis of records obtained from the toxicology section of the Chemistry Centre. These cases included all Western Australian drivers killed in road crashes in these periods. Ethics permission to conduct these WA studies was obtained from the Perth Coroner's Office.

There was reasonable consistency in crash investigation between and within the three jurisdictions. Coroner's files (coronial briefs) included all relevant information including that of the investigating police officer(s), witness statements, medical reports, road worthiness inspections, toxicology results and the coroners findings.

2.2. Drug analysis

In each state, a central forensic laboratory performed a full toxicological investigation on all driver fatalities irrespective of type or cause. The toxicology testing was similar in the three states and included testing for alcohol, drugs of abuse (cannabinoids, amphetamines and related stimulants, benzodiazepines, cocaine, opiates) with screens for neutral drugs and basic psychotropic drugs. All three state laboratories took part in proficiency trials during this period, and were accredited by the national accreditation body in Australia (National Accreditation Testing Authority, NATA) in Forensic Science (toxicology).

Drugs administered to the deceased as part of medical treatment after the crash were excluded from consideration. Information on drugs given as ambulance or hospital treatment was obtained from records held within the coroners' files. When a death occurred in hospital, data from the toxicological analysis of ante-mortem specimens were used in the analysis. Cases were excluded where toxicology was not conducted due to unavailability of specimens. Cases were also excluded if the time from the crash to the time of death was more than four hours and a blood specimen was not obtained in hospital within 4 h of the incident.

During the study period, the laboratories reported the presence of Δ^9-tetrahydrocannabinol (THC) and/or its metabolite 11-nor-Δ^9-tetrahydrocannabinol-9-carboxylic acid (carboxy-THC). Since circa 1998 all three state laboratories routinely estimated the concentration of THC in cases where it was detected. Prior to this, the detection of THC was not always followed by the estimation of the THC concentration. We used two categorical variables of THC in our analyses: THC detected versus THC not detected, (with cases in which carboxy-THC alone was detected being classified as THC not detected) and in cases where THC was identified and its concentration estimated, THC concentration <5 and ≥ 5 ng/ml.

2.3. Categorization of drugs

To simplify the statistical analysis of drug-effects, drugs were categorized into drug families. All benzodiazepine drugs were placed in a single group. Substances acting as stimulants were placed into the amphetamine group. This included amphetamine, methamphetamine, methylenedioxymethamphetamine, ephedrine, pseudoephedrine, phentermine and cocaine. The opiate group included morphine, 6-acetylmorphine, codeine, methadone and meperidine (pethidine). The cannabinoid group included cases found to contain either THC or carboxy-THC with cases containing THC given a sub-classification.

All other psychoactive drugs were placed into the psychoactive drug group. This included sedating antihistamines, phenothiazine antipsychotics, tricyclic antidepressants, the anticonvulsants phenytoin and carbamazepine. Any non-psychoactive drug was placed into the miscellaneous group. This included acetaminophen, salicylate, quinine, theophylline, serotonin reuptake inhibitors, etc.

2.4. Responsibility analysis

The responsibility analyses were performed using a method described by Robertson and Drummer (1994). This determines the responsibility of drivers by searching for mitigating evidence among eight factors: condition of the road, condition of the vehicle, driving conditions, type of crash, witnesses' observations, road law obedience, difficulty of task and level of fatigue. Cases in which insufficient information was available to allow a sufficient assessment of culpability were excluded from the analysis.

An index of responsibility was calculated using pre-determined scoring guidelines based on the sum of the scores for each of the eight factors. Using the culpability scores, drivers were grouped into one of three categories—culpable (culpability score <13), contributory (≥ 13 and ≤ 15) or

non-culpable (>15). The proportion of drivers who were culpable (to those not culpable) was calculated for various drug groups including the drug-free group. This proportion is called the "culpability ratio" (cases classified as "contributory" were not included in the statistical analyses).

The results of the toxicological examination were added to the case file only after the responsibility analysis had been completed. All data pertaining to these studies were kept on an Access database.

2.5. Statistical analyses

Differences in the likelihood of culpability between age, gender, multiple- versus single-vehicle crashes, state and year groups were initially investigated using univariate statistical analyses, (χ^2- or Fisher's Exact test, depending on the size of the sample). The relative odds (odds ratio) of culpability for BAC was calculated in six strata (0.01–0.049, 0.05–0.99, 0.10–0.149, 0.150–0.199, 0.20–0.249, >0.249 gm/100 ml) versus no alcohol detected. Further statistical analyses were conducted using logistic regression which models the likelihood (log odds) of culpable driving versus non-culpable driving as a function of crash attributes.

Logistic regression quantifies the effect of the included crash attributes on the likelihood of culpable driving, while adjusting for the effect of the other predictor variables included in the analysis. Logistic regression also allows the statistical testing of interactions between the predictor variables. The interactions test whether the effect of one factor on the likelihood of culpability varies with the value of another predictor, e.g. does the driver's age have a greater effect on the likelihood of culpability in males than females.

The seven crash attributes included in the full logistic regression model were BAC (coded as five dummy variables using the above strata with the two higher strata (0.20–0.249 and >0.249) combined), drug type, driver's gender, driver's age, type of accident (single- or multi-vehicle), location of the crash (Victoria, NSW, WA) and year of crash. The interactions tested were: age × gender; age × (single- versus multi-vehicle); age × BAC strata; age × drug groups; year × (single- versus multi-vehicle); year × BAC strata; year × drug groups; year × state; BAC strata × drug groups. Interaction terms were excluded from the final model if they were not significantly associated with the outcome. The final model contained only one interaction term: year; year × (single- versus multi-vehicle).

Estimates of odd's ratios (OR) were obtained from the final model for the five attributes not involved in the interaction. To estimate ORs for the single- versus multi-vehicle attribute the data was split into the year groups 90–95, 96/97 and 98/99, and a separate model fitted to each. Confidence intervals were calculated for all ORs. An OR of greater than unity indicates a positive association of the attribute with the likelihood of the culpability.

3. Results

3.1. Characteristics of drivers

Three thousand three hundred and ninety eight drivers, were included in the study: 1611 Victorian (47.4% of study population), 1031 NSW (30.3%) and 757 Western Australian drivers (22.3%). This represented 89% (median) of the driver fatalities identified from each jurisdiction. Cases were excluded if the deceased was not a driver, if they died of natural causes or committed suicide, if the crash did not occur on a public road and if either the toxicology data or culpability data were incomplete. The proportions of included cases for the three states are shown in Table 1.

The breakdown of crash types (single and multiple vehicle crashes) and type of vehicle is shown in Table 1. Car drivers, motor cyclists and truckers represented 76.7, 19.1, and 4.1% of the study group, respectively. Single vehicle crashes represented 50.7% of the cases.

The proportion female drivers varied with the type of vehicle: 26.8% in cars, 2.0% in motor cyclists and 0.7% in truckers. The corresponding mean age and age range (in parentheses) in these three vehicle types was 38.5 (13–92), 28.8 (12–79) and 37.7 (17–68) years, respectively.

There were small differences in the distribution of drugs between states, which probably reflected local availability and usage of drugs. Due to the smaller number of cases in each State separate statistical analyses are not presented.

3.2. Prevalence of alcohol and drugs

The prevalence of alcohol and drugs is described elsewhere (Drummer et al., 2003). However, in general terms the prevalences of alcohol (≥0.05%) and a drug of any type were 29.1 and 26.7%, respectively. Psychoactive drugs were present in 23.5% of drivers. This was made up of cannabinoids (13.5% of drivers), opioids (4.9%), stimulants and benzodiazepines (each 4.1%). Stimulants were present in 23% of all truckers. The prevalence of THC in driver fatalities which occurred in the years in which THC concentration was

Table 1
Breakdown of case type by state

Parameter	Victoria	WA	NSW	All states
No. of cases	1611	757	1030	3398
Proportion of cases analysed[a] (%)	90	90	80	87
Alcohol and drug negative	839	530	335	1704
Alcohol positive (≥0.05%)	422	271	297	990
Drug positive	454	245	208	907
Car drivers	1264	557	788	2609
Motor cyclists	314	164	172	650
Truckers	33	36	70	139
Single vehicle crashes	782	417	523	1722
Multiple vehicle crashes	829	340	507	1676

[a] Proportion of cases in each jurisdiction that were sufficiently complete from an investigation point of view to enable an assessment of responsibility and which included a full toxicological investigation.

estimated was 8.5%. The prevalence of miscellaneous psychoactive drugs was 2.7%. The majority of all drug-positive cases involved more than one psychoactive substance.

3.3. Responsibility analyses

The raw data for each of three responsibility classifications for control cases, age and gender groups, states, crash types, vehicle types, alcohol and drug groups are presented in Table 2. Of the 1704 drug- and alcohol-free drivers, 1214 (71%) were considered "responsible" for the crash or "culpable", (score <13), while 376 (22%) were classified as "not responsible" or "not culpable", (score >15). Of the 1694 drug- or alcohol-positive drivers, 1487 (88%) were "responsible" and 133 (8%) "not responsible" for the crash. The 188 drivers whose actions "contributed" to the crash, 114 controls and 74 drug- or alcohol-positive cases, were excluded from the logistic regression analyses.

3.4. Logistic regression analyses

The only interaction that was found to be significant ($\alpha = 0.05$) was between year and crash type (single/multiple vehicle crashes). This means the relative likelihood of culpability for drivers involved in single- versus multi-vehicle crashes varied somewhat with the year. The final logistic regression model fitted to the overall data set and the various data subsets contains the predictor variables: age; gender; type of crash; alcohol level (in five strata); drug group; state; year; and, the interaction of crash type and year. The estimated odds ratios for these groups and their 95% CIs are presented in Table 3. The odds of culpability for single- versus multi-vehicle incidents varied with the year so three estimates are presented with only one 95% confidence interval. Calculation of the other two confidence intervals requires covariance estimates and these were not available.

3.5. Alcohol odds ratios

In order to study the effect of alcohol on crash risk BACs were categorized into five levels. The ranges were 0.010–0.049, 0.050–0.099, 0.100–0.149, 0.150–0.199 and 0.20% or greater. The odds ratios for each of these groups calculated using the logistic regression model are shown diagrammatically in Fig. 1.

Table 2
Breakdown of responsibility classifications by group

Group	Total drivers	Number in each category			Culpability ratio[a]
		Responsible	Contributory	Not responsible	
Control cases	1704	1214	114	376	3.2
Positive cases[b]	1694	1487	74	133	11.2
Age-group					
0–17	109	96	5	8	12.0
18–25	1098	908	64	126	7.2
26–29	399	315	32	52	6.1
30–39	645	499	36	110	4.5
40–59	680	507	33	140	3.6
≥60	467	376	18	73	5.2
Females	714	559	34	121	4.6
Males	2684	2142	154	388	5.5
NSW	1030	849	53	128	14.4
Vic.	1611	1259	96	256	4.9
WA	757	593	39	125	4.7
SVC	1722	1575	88	59	26.7
MVC	1676	1126	100	450	2.50
Car drivers	2609	2120	126	363	5.8
Truck drivers	139	106	8	25	4.2
Motor cyclists	650	475	54	121	3.9
Drug positive	907	774	39	94	8.2
BAC (≥0.05 g%)	990	922	41	27	34.1
Benzo-only	34	27	1	6	4.5
THC-only	58	51	2	5	10.2
Opioid-only	59	48	0	11	4.4
Other psych-only	51	47	0	4	11.8
Misc-only	95	74	5	16	4.6
Stimulant-only	53	42	5	6	7.0

Abbreviations: SVC: single vehicle crashes, MVC: multiple vehicle crashes, Vic.: Victoria, NSW: New South Wales, WA: West Australia, BAC: blood alcohol concentration, THC: tetrahydrocannabinol.

[a] Ratio of number drivers responsible to those not responsible.

[b] Drug or alcohol positive, or both.

Table 3
Odds ratios and 95% confidence limits of selected factors

Effect vs. baseline	Odds ratio point estimate	95% Confidence limits
Age group		
0–17 vs. 18–25	1.82	0.82–4.00
26–29 vs. 18–25	0.76	0.52–1.12
30–39 vs. 18–25	0.58[a]	0.43–0.79
40–59 vs. 18–25	0.57[a]	0.43–0.79
≥60 vs. 18–25	1.28	0.91–1.79
Female vs. male	1.08	0.83–1.39
SVC vs. MVC (1990–1995)	7.8	4.91–12
SVC vs. MVC (1996–1997)	11.2	4.7–26
SVC vs. MVC (1998–1999)	3.9	2.3–6.7
Drug yes vs. no	1.68[a]	1.29–2.18
Alcohol (≥0.05) yes vs. no	6.0[a]	4.0–9.1
NSW vs. Vic.	1.07	0.83–1.39
WA vs. Vic.	0.74[a]	0.57–0.96

[a] Indicates significant difference at $\alpha = 0.05$ level.

These data show a non-linear increase in odds ratio from 1.2 at a BAC of <0.05% to 25.0 at BACs above 0.20%. This means the odds of a driver with a BAC of 0.01–0.049% being culpable were 1.2 times the odds of a driver who was drug- and alcohol-free. The odds of a driver with a BAC equal to or greater than 0.20% being culpable was 24 times the odds of a drug- and alcohol-free driver being culpable. The estimated odds ratios for BACs above 0.10% were all statistically significant.

3.6. Drugs odds ratios

The detection of any type of drug was found to be significantly associated with culpability (OR 1.7, 95% CI 1.3–2.2, Table 3). The logistic regression estimates of the associations of the major drug types with culpability are presented in Tables 3 and 4. The largest odds ratios were those associated with the presence of THC in blood (OR 6.6), stimulants (OR 2.3) and miscellaneous (other) psychoactive drugs (OR 3.8).

The detection of THC was positively associated with culpable driving for all drivers and for motor cyclists (OR 2.7, 95% CI 1.0–7.0 and OR 2.4, 95% CI 0.5–12.5, respectively). The median THC concentration in the THC cases

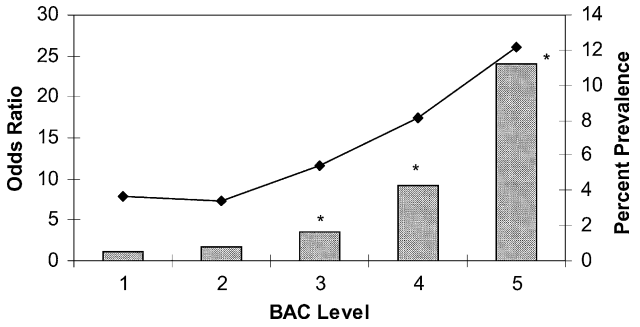

Fig. 1. Odds ratio (bars) and prevalence of the blood alcohol concentrations (BAC) at five ordinal categories: Level 1: 0.010–0.049%; Level 2: 0.050–0.099%; Level 3: 0.100–0.149%; Level 4: 0.150–0.199%; Level 5: ≥0.200%. * $P < 0.05$.

was 12 ng/ml, and 84% of the cases had concentrations ≥5 ng/ml. When only those cases with THC concentrations of 5 ng/ml or higher were considered, the point estimate for all drivers was 6.6 (95% CI OR 1.5–28.0), which is a similar odds ratio to that obtained for BAC-positive cases over 0.15% (note: cases in which carboxy-THC alone was detected were not classified as THC-positive).

Since alcohol was commonly found in THC-positive cases (43%), the effect of THC was also evaluated in the THC plus alcohol cases. Logistic regression modelling showed the odds of culpability in drivers who were THC-positive and had BAC ≥0.05 g% was 2.9 times the odds of drivers who had BAC ≥0.05 g% alone (95% CI OR 1.1–7.7). This relative likelihood is similar to that for THC-positive drivers compared with drug- and alcohol-free drivers (OR 2.7, 95% CI 1.0–7.0). These data strongly suggest that THC does enhance the impairment caused by alcohol. It was of interest that cases in which THC was not detected but carboxy-THC was detected, the odds ratio was 0.9 ($P > 0.05$; i.e. there was no difference in the likelihood of culpability compared with drug- and alcohol-free drivers).

The "other psychoactive" drug group showed a significant OR of 3.8 (95% CI 1.3–10.9) indicating that many if not most of the legally available psychoactive drugs do appear to influence driving safety (Table 5).

In drivers positive for stimulants, the point estimate for all cases showed a strong but statistically insignificant

Table 4
Statistical summary of cannabis data using logistic model

Parameter	*N* (percentage of population)	Point estimate (OR)	95% Confidence limits
Drug and alcohol free	1704 (50.1%)	1.0	–
All drugs[a]	907 (26.7%)	1.7[b]	1.3–2.2
THC-only[c]	58 (1.7%)	2.7[b]	1.02–7.0
THC-only[c] (≥5 ng/ml)	49 (1.4%)	6.6[b]	1.5–28
THC plus BAC (≥0.01 g%) vs. BAC[d]	43 (1.3%)	2.9[b]	1.1–7.7
THC plus BAC (≥0.01 g%) vs. BAC in motor cyclists[d]	33 (5.1%)[c]	2.4	0.5–12

[a] Any cases involving a detected drug (alcohol interactions considered) in all driver types.
[b] Indicates significant difference at $\alpha = 0.05$ level.
[c] No other drugs present in all driver types.
[d] No other drugs or alcohol present in case in all driver types.

Table 5
Statistical summary of drug data using logistic model

Parameter	*N* (percentage of population)	Point estimate (OR)	95% Confidence limits
Drug and alcohol free	1704 (50.1%)	1.0	–
All psychoactive drugs[a]	484 (14.2%)	1.80[d]	1.3–2.4
Psychotropics plus BAC (≥0.05 g%) vs. BAC	285 (8.4%)	1.70[d]	1.3–2.3
Stimulants (all drivers)[b]	53 (1.6%)	2.27	0.9–5.6
Stimulants (truckers)[b,a]	22 (15.8%)[c]	8.83[d]	1.00–78
Benzodiazepines[b]	34 (1.0%)	1.27	0.5–3.3
Opiates[b]	59 (1.7%)	1.41	0.7–2.9
Other psychoactive drugs[b]	51 (1.5%)	3.78[d]	1.3–11
Miscellaneous drugs[b]	95 (2.8%)	1.47	0.8–2.7

Any cases involving a detected drug (alcohol interactions considered).
[a] Any combination of psychoactive drug, but not alcohol.
[b] No other drugs or alcohol present in case.
[c] Percentage of truckers.
[d] Indicates significant difference at $\alpha = 0.05$ level.

association with culpability (OR 2.3, 95% CI 0.9–5.6). If the truckers were considered as a discrete driver type, the OR increased to 8.8 and was of borderline statistical significance (95% CI 1.0–77.8, Table 5).

Benzodiazepines, opiates and miscellaneous drugs, on their own, showed positive but non-significant associations with culpability (Table 5).

However, as other drugs were detected in many cases involving benzodiazepines, this greatly reduced the power of our study. When the THC, stimulant, benzodiazepine, opiate and miscellaneous psychoactive drug groups were combined, there was a strong and significant association with culpability (OR 1.8, 95% CI 1.3–2.4, Table 5). The relative likelihood was similarly increased in the presence of alcohol. Logistic regression modeling showed the odds of culpability in drivers who were psychotropic drug-positive and had BAC ≥0.05 g% was 1.7 times the odds of drivers who had BAC ≥0.05 g% alone (95% CI OR 1.3–2.3).

4. Discussion

This 10-year, multi-centre longitudinal study of driver culpability has shown a number of strong positive associations between use of some psychoactive drugs and responsibility for the crash. Of particular interest is the association of THC with driver culpability. This association showed a biological gradient, similar to that observed for alcohol. The estimated association with culpability of THC in concentrations of at least 5 ng/ml was much greater than the association of all identifiable concentrations of THC (OR 6.6 versus 1.9). The odds ratio for THC concentrations of 5 ng/ml or higher were similar to those for drivers with a BAC of at least 0.15 g%. THC also increased the likelihood of culpability in drivers who had also been drinking alcohol.

While these findings are very suggestive of a causal relationship, they should be interpreted with some caution. Theoretically, it is possible that the presence of THC in a driver's blood is a proxy for a risk-taking lifestyle (or some other factor) and that it is this lifestyle (or other factor) that increases the likelihood of culpability. Neither the size nor the statistical significance of the associations we observed can be used to directly infer causality. However, the available experimental evidence supports the proposition that the relationship is, indeed, causal—that intoxication with THC increases a driver's risk of causing and dying in a crash.

The meta-analysis conducted by Berghaus shows substantial performance decrements at plasma THC concentrations of about 5 ng/ml or higher and significant performance decrements with concentrations of 10 ng/ml (Berghaus et al., 1995; Ramaekers et al., 2002) (a plasma concentration of 10 ng/ml is roughly equivalent to a whole blood concentration of 5 ng/ml).

Driving simulators and on-road driving tests have shown significant adverse effects on the standard deviation of lateral position (SDLP) a measure of lane control following standard smoked cigarettes (100–300 μg/kg doses) (Lamers and Ramaekers, 2001; Ramaekers et al., 2002, 2000; Robbe, 1994, 1998; Robbe and O'Hanlon, 1999). The effects were increased with co-consumption of alcohol (BAC 0.04–0.08 g%) (Robbe and O'Hanlon, 1999). These effects were generally additive, an observation also supported by our studies. Other driving parameters in the above cited papers were either less markedly affected or were variably significant. Increased speed variability has been seen following cannabis use although an increased headway and more conservative driving has been seen (Robbe, 1994; Smiley et al., 1981). More recent studies have shown an adverse effect of cannabis on both the Road Tracking and the Car Following Test, particularly after 200 μg/kg doses, and in combination with alcohol at 0.04 g% (Robbe and O'Hanlon, 1999).

Culpability studies have not always shown an increased risk of cannabis in crashes (Lowenstein and Koziol-Mclain, 2001; Williams et al., 1985), although most have either shown an increase or shown trends for an increase (Longo et al., 2000; Terhune et al., 1992; Warren et al., 1981). This

is probably more to do with the design of the study and the limitations in performing such studies, than the effect of THC on crash risk. For example, our own previous study had focussed on an assessment of cannabis exposure as presence of the carboxy-THC metabolite (Drummer, 1994). We have now shown that past use does not increase crash risk, rather recent use as defined as positive THC blood concentrations, particularly those over 5 ng/ml. The measurement of urinary cannabinoids in a recent study would also reduce the ability to examine short-lived effects of THC (Lowenstein and Koziol-Mclain, 2001). The Williams study (Williams et al., 1985) had found only 19 cases positive to marijuana alone, and 60% of these had THC concentrations <2 ng/ml. Low THC concentrations were also seen in the South Australian injured driver study (Longo et al., 2000). In our study the median THC concentration was 12 ng/ml and in 84% of the THC-only cases the concentration was ≥5 ng/ml. Therefore, on the basis of the increased sample size and the higher THC concentrations our study was more likely to see an effect of THC on crash risk.

The stimulants had the strongest measured association with culpability. Overall, stimulants were detected in 4.1% of all drivers and 23% of truckers. The odds of any drivers using this drug being culpable were 2.3 those of drug- and alcohol-free driver, while the relative odds were 8.8 for truckers on stimulants. While this latter result was only marginally statistically significant at $\alpha = 0.05$ level, it is consistent with the available literature. Previous descriptive studies have found a high rate of aberrant driving among amphetamine users, particularly in the acute intoxication phase and in the rebound fatigue phase (Logan, 2002, 1996). There is also other evidence of the over-involvement of amphetamine users in crash rates (Crouch et al., 1993; NHTSA, 1992; Smart et al., 1969).

The present study revealed a relatively strong and highly significant association between the presence of any drug and the likelihood of the driver being culpable (OR 1.7, Table 3) with the "other psychoactive" group having the strongest association in the group (OR 3.8, Table 5). This strong evidence that the involvement of prescription drugs in road trauma may be much greater than suggested by the number of drivers prosecuted for drug-impaired driving.

Neither benzodiazepines nor opiates showed a strong positive association with culpability. However, this lack of an association may have been due to the present study's lack of power to address the issue. Benzodiazepines have been shown to increase crash risk in a number of epidemiological studies (Barbone et al., 1998; Hemmelgarn et al., 1997; Longo et al., 2001; Neutel, 1995; Ray et al., 1992; Skegg et al., 1979), while others have not found an association (Jick et al., 1981; Leveille et al., 1994; Marquet et al., 1998). Moreover, there is little doubt, however, that benzodiazepines do produce relevant impairment of skills required for safe driving (Drummer, 2002), although chronic use can lead to a reduction of any impairment as tolerance is established.

Similarly, the lack of a statistically significant association in the present study cannot be interpreted as meaning opiates do not increase the risk of a driver being responsible for a crash. This is because 65% of opiate-positive drivers in the study were using other drugs; predominantly benzodiazepines (34%) and cannabis (24%). The exclusion of drivers who had also taken other drugs greatly reduced the statistical power of our testing for an association of opiates with culpability. Furthermore, some drivers (e.g. methadone maintenance patients) would have been tolerant to the effects of opiates and they would have been effectively misclassified as opiate-intoxicated. This would have further reduced the present study's ability to detect a real association between opiates and culpability.

While experimental studies have shown opiates have no or limited effects on a variety of psychometric and other performance studies (Lenné et al., 2000), the effect of narcotic drugs on vigilance is often overlooked in cognitive and psychometric tests. The effect of opiates on driving performance needs further experimental investigation with due consideration given to the effects of tolerance, lack of vigilance and a propensity to falling asleep. Further large epidemiological studies are needed to test the associations observed in the present study between the presence of various drugs with drivers' culpability in fatal and non-fatal road crashes.

Some caution also needs to be exercised when interpreting the strength of the associations reported in the present study. While many of the odds ratios are large, they do not imply a similar increase in relative risk. An odds ratio of 6.0 does not mean the risk of being culpable was increased by a factor of six. This is because the probability of culpability in the drug- and alcohol-free drivers was 0.76, (1214 of 1590 cases included in the logistic regression analyses). Consequently, drivers' consumption of drugs or alcohol could only have increased the probability of culpability by 0.24, or one-third. For instance, while the odds of a driver with a BAC of at least 0.05 g% being culpable were six times those of a drug- and alcohol-free driver (Table 3), the probability of culpability increased from 0.76 to 0.97 and the unadjusted odds from 3.2 to 34.1.

It is unlikely our results were affected by a bias in the selection of cases as this was done by an independent person, the clerk of court or another official. Each jurisdiction had policies of conducting toxicology irrespective of the type of motor vehicle crash and investigated all such cases through a centralised coronial system. Our results are not likely to have been affected by the exclusion of some drivers as we included about 90% of drivers who died as a result of a crash on public roads. Many of the excluded cases had been misclassified in the coroners' offices, e.g. passengers initially thought to be drivers, drivers who died as a result of suicides or from a catastrophic medical event.

Similarly, our results are unlikely to have been influenced by bias in the scoring of culpability as this was done blind to the toxicology results. Moreover, culpability scores were calculated on several different occasions in each

jurisdiction and the associations of culpability with drug groups were relatively consistent across these scoring episodes, i.e. across time. This suggests there was satisfactory inter-rater reliability. It should be noted that factors likely to influence driver performance such as light (night versus day, etc.) visibility, weather, and road surface are taken into account as mitigating factors in the culpability assessment.

The present study presents good evidence that drivers killed in motor vehicle crashes and taking psychoactive drugs, particularly cannabis and strong stimulants, or two or more drugs in combination were more likely to be responsible for the crash than those taking neither drugs nor alcohol. Moreover, the combination of psychoactive drugs with alcohol further increased the likelihood that drivers caused the crash in which they died. We conclude that THC, amphetamines and combinations of psychoactive drugs significantly increase drivers' risk of a serious road crash.

Acknowledgements

We thank the staff of the Victorian Institute of Forensic Medicine for their assistance in these investigations. We also thank the toxicologists in each state for their support and assistance, particularly Mr. Robert Hansson (WA) and Mr. Alan Hodda (NSW), as well as coroners, registrars and clerical assistants at the respective coroners' courts. We acknowledge the financial assistance of VicRoads for financing many of these studies, as well as AustRoads and the NSW roads and traffic authority. Finally, we thank Dr. Tony Wohlers of Statistical Investigations Ltd., for conducting the statistical analyses and his invaluable statistical advice.

References

Albery, I.P., Strang, J., Gossop, M., Griffiths, P., 2000. Illicit drugs and driving: prevalence, beliefs and accident involvement among a cohort of current out-of-treatment drug users. Drug Alcohol Depend. 58, 197–204.

Barbone, F., McMahon, A.D., Davey, P.G., Morris, A.D., Reid, I.C., McDevitt, D.G., MacDonald, T.M., 1998. Association of road-traffic accidents with benzodiazepine use. Lancet 352, 1331–1336.

Berghaus, G., Scheer, N., Schmidt, P., 1995. Effects of cannabis on psychomotor skills and driving performance—a meta analysis of experimental studies. In: Kloeden, C.N., McLean, A.J. (Eds.), Alcohol, Drugs and Traffic Safety. University of Adelaide, Adelaide.

Borkenstein, F.R., Crowther, R.F., Shumate, R.P., Zeil, W.B., Zylman, R., 1974. The role of the drinking driver in traffic accidents. Blutalkohol 11 (Suppl. 1).

Brookhuis, K.A., Volkerts, E.R., O'Hanlon, J.F., 1990. Repeated dose effects of lormetazepam and flurazepam upon driving performance. Eur. J. Clin. Pharmacol. 39, 83–87.

Crouch, D.J., Birky, M.M., Gust, S.W., Rollins, D.E., Walsh, J.M., Moulden, J.V., Quinlan, K.E., Beckel, R.W., 1993. The prevalence of drugs and alcohol in fatally injured truck drivers. J. Forensic Sci. 38, 1342–1353.

Drummer, O.H., 2002. Benzodiazepines—effects on human performance and behavior. Forensic Sci. Rev. 14, 1–14.

Drummer, O.H., 1994. Drugs in drivers killed in Australian road traffic accidents. Monash University Department of Forensic Medicine, Melbourne.

Drummer, O.H., Gerostamoulos, J., Chu, M., Batziris, H., J.R.N., C., Robertson, M.D., 2003. The prevalence of alcohol and drugs in Australia. Forensic Science International, in press.

Hemmelgarn, B., Suissa, S., Huang, A., Boivin, J.F., Pinard, G., 1997. Benzodiazepine use and the risk of motor vehicle crash in the elderly. JAMA 278, 27–31.

Jick, H., Hunter, J.R., Dinan, B.J., Masen, S., Stergachis, A., 1981. Sedating drugs and automobile accidents leading to hospitalization. Am. J. Public Health 71, 1399–1400.

Lamers, C.T.J., Ramaekers, J.G., 2001. Visual search and urban driving under the influence of marijuana and alcohol. Human Psychopharmacol. Clin. Exp. 16, 393–401.

Laurell, H., Tornros, J., 1986. The carry-over effects of triazolam compared with nitrazepam and placebo in acute emergency driving situations and in monotonous simulated driving. Acta Pharmacol. Toxicol. (Copenh) 58, 182–186.

Lenné, M.G., Dietz, P., Rumbold, G., Redman, J.R., Triggs, T.J., 2000. Opioid dependence and driving ability: a review in the context of proposed legislative change in Victoria. Drug Alcohol Rev. 19, 427–439.

Leveille, S.G., Buchner, D.M., Koepsell, T.D., McCloskey, L.W., Wolf, M.E., Wagner, E.H., 1994. Psychoactive medications and injurious motor vehicle collisions involving older drivers. Epidemiology 5, 591–598.

Logan, B.K., 2002. Methamphetamine—effects on human performance and behavior. Forensic Sci. Rev. 14, 133–151.

Logan, B.K., 1996. Methamphetamine and driving impairment. J. Forensic Sci. 41, 457–464.

Logan, B.K., Schwilke, E.W., 1996. Drug and alcohol use in fatally injured drivers in Washington State. J. Forensic Sci. 41, 505–510.

Logan, B.K., Case, G.A., Gordon, M.A., 2000. Carisprodol, meprobamate and driving impairment. J. Forensic Sci. 45, 619–623.

Longo, M.C., Hunter, C.E., Lokan, R.J., White, J.M., White, M.A., 2000. The prevalence of alcohol, cannabinoids, benzodiazepines and stimulants amongst injured drivers and their role in driver culpability. Part II. The relationship between drug prevalence and drug concentration, and driver culpability. Accid. Anal. Prev. 32, 623–632.

Longo, M.C., Lokan, R.J., White, J.M., 2001. The relationship between blood benzodiazepine concentration and vehicle crash culpability. J. Traffic Med. 29, 36–43.

Lowenstein, S.R., Koziol-Mclain, J., 2001. Drugs and traffic crash responsibility: a study of injured motorists in Colorado. J. Trauma 50, 313–320.

Marquet, P., Delpla, P.A., Kerguelen, S., Bremond, J., Facy, F., Garnier, M., Guery, B., Lhermitte, M., Mathe, D., Pelissier, A.L., Renaudeau, C., Vest, P., Seguela, J.P., 1998. Prevalence of drugs of abuse in urine of drivers involved in road accidents in France: a collaborative study. J. Forensic Sci. 43, 806–811.

Mounce, N.H., Pendleton, O.J., 1992. The relationship between blood alcohol concentration and crash responsibility for fatally injured drivers. Accid. Anal. Prev. 24, 201–210.

Neutel, C.I., 1995. Risk of traffic accident injury after a prescription for a benzodiazepine. Ann. Epidemiol. 5, 239–244.

NHTSA, 1992. The incidence and role of drugs in fatally injured drivers. National Highway Traffic Safety Administration.

O'Hanlon, J.F., Volkerts, E.R., 1986. Hypnotics and actual driving performance. Acta Psychiatr. Scand. Suppl. 332, 95–104.

O'Hanlon, J.F., Haak, T.W., Blaauw, G.J., Riemersma, J.B., 1982. Diazepam impairs lateral position control in highway driving. Science 217, 79–81.

Ramaekers, J.G., Robbe, H.W.J., O'Hanlon, J.J., 2000. Marijuana. Human Psychopharmacol. 15, 551–558.

Ramaekers, J.G., Berghaus, G., van Laar, M., Drummer, O.H., 2002. Performance impairment and risk of motor vehicle crashes after

cannabis. In: Spruit, I.P. (Ed.), Cannabis 2002 Report. Brussels, Belgium.

Ray, W.A., Fought, R.L., Decker, M.D., 1992. Psychoactive drugs and the risk of injurious motor vehicle crashes in elderly drivers. Am. J. Epidemiol. 136, 873–883.

Robbe, H.W.J., 1994. Influence of marijuana on driving. CIP-Data Koninklinik Bibliothek, The Hague, Maastricht, The Netherlands.

Robbe, H.W.J., 1998. Marijuana's impairing effects on driving are moderate when taken alone but severe when combined with alcohol. Human Psychopharmacol. 13, S70–S78.

Robbe, H.W.J., O'Hanlon, J.F., 1999. Marijuana, alcohol and actual driving performance. National Highway Traffic Safety Adminsitration, Maastrict, The Netherlands, p. 43.

Robertson, M.D., Drummer, O.H., 1994. Responsibility analysis: a methodology to study the effects of drugs in driving. Accid. Anal. Prev. 26, 243–247.

Simpson, H.M., 1986. Epidemiology of road accidents involving marijuana. Alcohol Drugs Driving 2, 15–30.

Simpson, H.M., Mayhew, D.R., Warren, R.A., 1982. Epidemiology of road accidents involving young adults: alcohol. Drug Alcohol Depend. 10, 35–63.

Skegg, D.C.G., Ricards, S.M., Doll, R., 1979. Minor tranquillisers and road accidents. Br. Med. J. 1, 917–919.

Smart, R.G., Schmidt, W., Bateman, K., 1969. Psychoactive drugs and traffic accidents. J. Safety Res. 1, 67–72.

Smiley, A.M., Moskowitz, H., Ziedman, K., 1981. Driving simulator studies of marijuana alone and in combination with alcohol. In: Proceedings of the 25th American Association of Automotive Medicine, pp. 107–116.

Terhune, K.W., Ippolito, C.A., Hendricks, D.L., Michalovic, J.G., Bogema, S.C., Santinga, P., Blomber, R., Preusser, D.F., 1992. The Incidence and Role of Drugs in Fatally Injured Drivers. US Department of Transportation, National Highway Traffic Safety Administration, Washington.

Walsh, G.W., Mann, R.E., 1999. On the high road: driving under the influence of cannabis in Ontario. Can. J. Public Health 90, 260–263.

Warren, R.A., Simpson, H.M., Hilchie, J., Cimbura, G., Lucas, D., Bennett, R., 1981. Drugs detected in fatally injured drivers in the Province of Ontario. In: Goldberg, L. (Ed.), Alcohol, Drugs and Traffic Safety. Almqvist and Wiksell International Stockholm, Sweden, Stockholm, pp. 203–217.

Wechsler, H., Rohman, M., Kotch, J.B., Idelson, R.K., 1984. Alcohol and other drug use and automobile safety: a survey of Boston area teen-agers. J. Sch. Health 54, 201–203.

Williams, A.F., Peat, M.A., Crouch, D.J., Wells, J.K., Finkle, B.S., 1985. Drugs in fatally injured young male drivers. Public Health Rep. 100, 19–25.