

The Role of Alcohol, Cannabinoids, Benzodiazepines and Stimulants in Road Crashes

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Abstract

Blood samples collected from 2500 non-fatally injured drivers involved in road crashes were analysed for the presence of alcohol, cannabinoids, benzodiazepines and stimulants. Evaluation of the causal role of the drug in the crash was based on culpability analysis using data collected at the crash scene. A total of 22.6% of drivers tested positive for at least one drug including alcohol. Either alone or in combination with other drugs, alcohol was found in 12.4% of drivers, cannabinoids in 10.8%, benzodiazepines in 2.7% and stimulants in 1.3%. A small number of cannabinoid-positive drivers tested positive for tetrahydrocannabinol (THC, the main active ingredient in marijuana) while most drivers tested positive for the inactive metabolite. A clear causal role was found for alcohol, with increased culpability rates at high blood alcohol concentrations (BACs). Benzodiazepine use was also associated with higher culpability when those with very low concentrations were excluded. Consistent with earlier research, THC was not associated with increased culpability. Relatively few drivers tested positive for stimulants and there was no clear evidence of greater culpability. Overall, alcohol plays the greatest role in road crashes, but benzodiazepines also have a significant effect.

Introduction

A variety of techniques have been used to assess the role of drugs in road crashes. The aim of this study was to use an objective measure of driver culpability, and methods for analysis of blood samples that permitted determination of the presence and concentration of each drug in blood samples from non-fatally injured drivers. The data collected were analysed to determine the effects of each drug either alone or in combination with other drugs, and the relationship between drug concentration and culpability was also examined.

Culpability analysis is based on the premise that if drugs do contribute to crashes, the proportion of drivers who are judged culpable will be greater among drug-affected drivers than drug-free drivers. The relationship between drug use and crash risk can thus be examined, where crash risk is measured by the percentage of drivers judged culpable for the crash. The culpability of drivers in each crash is assessed using defined criteria, with information from police reports including vehicle condition, weather and lighting, and road characteristics and conditions.

Results from studies using culpability analysis have consistently reported a significant relationship between alcohol and culpability. Studies by Terhune (1982), Terhune *et al.* (1992) and Drummer (1994) found that those who tested positive for alcohol were significantly more likely to be culpable for the crash than drug-free drivers. Moreover, the effect was more marked as BAC increased.

Studies examining the relationship between THC and culpability have generally found that when used alone, THC was associated with *lower* culpability (Williams *et al.*, 1985; Terhune *et al.*, 1992; Drummer, 1994). Conversely, a study by Terhune (1982) found the reverse. However, in all studies the differences were not statistically significant, and the number of drivers testing positive for THC-alone was small.

Where the relationship between benzodiazepines and culpability has been examined the results have been inconsistent. A study by the Benzodiazepine and Driving Collaborative Group (1993) found no statistically significant evidence that benzodiazepines are a risk factor in road crashes. Similarly, Terhune (1982) found that a *lower* percentage of drivers who tested positive for benzodiazepines-alone were culpable for the crash compared with drug-free drivers. Conversely, Drummer (1994) found the reverse and Terhune *et al.* (1992) found that drivers who tested positive for benzodiazepines-alone and drug-free drivers had almost identical culpability rates. However, the differences were not statistically significant. Moreover, the number of drivers who only tested positive for benzodiazepines was small as most also tested positive for alcohol, and the results must be interpreted with caution.

There is some indication that testing positive for stimulants is associated with increased culpability, although small sample sizes make it difficult to derive firm conclusions. Terhune *et al.* (1992) and Drummer (1994) found that a lower percentage of drug-free drivers were culpable for the crash compared with drivers who tested positive for stimulants-alone, although these differences were not statistically significant.

Method

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 & Emergency units following a road crash must provide a blood sample. For the present study, consecutive samples were collected in the periods April 1995 to August 1995, and December 1995 to August 1996. These samples were analysed for the presence of alcohol, cannabinoids (THC and THC-acid), benzodiazepines and stimulants.

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.

Analytical methods

Whole blood samples were initially screened using radioimmunoassay, with the exception of alcohol, which was analysed directly without prior screening. Samples testing negative were eliminated, and presumptive positive samples were retained and subjected to further definitive testing to positively identify the drug or drugs present and to determine concentration.

Culpability analysis

Culpability of the injured driver in each crash was assessed using the method developed by Robertson and Drummer (1994). Culpability was assigned by identifying any mitigating factors that

may have reduced responsibility for the crash. A driver was judged culpable if not exonerated by these mitigating factors. If sufficient mitigating factors were identified, a driver was deemed only partly culpable (contributory) or not culpable. The analysis was based on eight mitigating factors: the condition of the road, the condition of the vehicle, general driving conditions, the type of crash, witness observations, road law obedience, the difficulty of the task involved and the level of fatigue.

Results

Prevalence of drug use

A range of drugs and drug combinations were detected (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-alone or stimulants-alone 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. Drivers positive for cannabinoids included samples that showed the presence of THC together with THC-acid (2.8%), and samples positive for THC-acid alone (8%). 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 pseudoephedrine were not treated as drug-positive as this drug has very weak stimulant effects.

Table 1: Percentages of injured drivers testing positive for the various drugs and drug combinations

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%

Table 2 shows the percentage of drivers who tested positive for different BACs, divided into those drivers who tested positive for alcohol-alone, and those who tested positive for alcohol in combination with other drugs. Most drivers (87.6%) had a zero BAC, although 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-alone in each BAC category was similar to that for drivers who tested positive for alcohol in combination with other drugs. Moreover, there was no significant difference in the

mean BAC (0.136% for alcohol-alone and 0.146% for alcohol in combination with other drugs, $t=0.07$, $p>0.05$).

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)

Drugs and culpability

Nearly 55% of drivers were judged culpable for the crash and 39% were not culpable. The proportion of drivers judged to be contributory was small (6.2%) and in subsequent analyses these drivers were omitted. Data from the two drivers for whom there was insufficient information to determine culpability were also omitted.

There were no significant differences between males and females in this sample with respect to culpability (59.7 compared with 56.6%, respectively: $\chi^2_1=2.47$, $p>0.05$). However, there was a statistically significant relationship between age and culpability. Younger drivers (less than 26 years) and older drivers (over 60 years) were more likely to be culpable than other age groups ($\chi^2_3=72.82$, $p<0.001$).

Effect of drugs on culpability

Table 3 shows the percentage of drivers judged culpable for the crash for the various drugs and drug combinations. Culpability rates for the various drug groups were compared with the drug-free group. The groups that differed significantly from the drug-free group were drivers who tested positive for alcohol-alone, benzodiazepines-alone, alcohol and THC, and alcohol and benzodiazepines. The culpability rate of drivers who tested positive for THC-alone did not differ significantly from the drug-free group. Moreover, the culpability rate of drivers who tested positive for alcohol-alone was not significantly different from that of drivers who tested positive for alcohol in combination with either THC or benzodiazepines.

Table 3: Percentages of injured car drivers and riders testing positive for the various drugs and drug combinations according to level of culpability for the crash. Odds-ratios for the drug-positive groups compared with the drug-free group are included in brackets

Drug combination	Percentage culpable
Drug-free (n=1887)	52.8%
Alcohol only (n=250)	90.0% (8.0)
THC only (n=44)	47.7% (0.8)
Alcohol + THC (n=14)	85.7% (5.4)
Benzodiazepines only (n=46)	69.6% (2.0)
Stimulants only (n=16)	68.8% (2.0)
Alcohol + benzodiazepines (n=16)	93.8% (13.4)
Stimulants + THC (n=1)	100.0% (-)
Benzodiazepines + THC (n=2)	100.0% (-)
Other combinations (n=3)	100.0% (-)

Culpability for individual drug classes

The following tables present the concentration-culpability relationship for alcohol, THC, benzodiazepines and stimulants both alone and in combination with other drugs.

Alcohol

Table 4 shows that as BAC increased, so did the percentage of drivers judged culpable. There was a significant difference in the proportion of culpable drivers who tested positive for alcohol across the BAC ranges (including the drug-free group), for drivers testing positive for alcohol-alone ($\chi^2_4=133$, $p<0.001$), and for alcohol in combination with other drugs ($\chi^2_4=23.6$, $p<0.001$). There was a significant linear relationship for each group ($\chi^2_1=130.5$, $p<0.001$ and $\chi^2_1=21.8$, $p<0.001$, respectively). A comparison was made between the mean BAC of all alcohol-positive drivers according to culpability. It was found that culpable drivers had a significantly higher mean BAC than not culpable drivers (0.144% vs 0.073%, $t=5.2$, $p<0.001$).

Table 4: Culpability of injured drivers and BAC: alone/in combination with other drugs. Odds-ratios for the BAC ranges compared with the drug-free group are included in brackets

BAC (%)	Alcohol alone (n=250)	Alcohol in combination (n=32)
	% culpable	% culpable
Drug-free	52.8% (n=1887)	52.8% (n=1887)
less than 0.05	68.6% (n=35) (1.9)	33.3% (n=3) (0.4)
0.05 - 0.079	87.5% (n=24) (6.2)	66.7% (n=3) (1.8)
0.08 - 0.149	91.7% (n=84) (9.8)	100.0% (n=11) (-)
0.150 +	96.3% (n=107) (23.0)	100.0% (n=15) (-)
	Mean BAC 0.136%	Mean BAC 0.146%

Cannabinoids

Drivers testing positive for cannabinoids had either THC-acid only detected in their blood, or THC-acid and THC in combination. The presence of THC-acid without THC can only confirm that marijuana has been used at some indeterminable point, and is not an indicator of possible impairment at the time of the crash. Table 5 thus indicates the relationship between the presence of cannabinoids and driver culpability for drivers who tested positive for THC, alone or in combination with other drugs.

Table 5: Culpability of injured drivers and THC concentration: alone/in combination with other drugs. Odds-ratios for the THC groups compared with the drug-free group are included in brackets

THC concentration (ng/mL)	Percentage culpable	
	THC alone (n=44)*	THC in combination (n=17)*
Drug-free	52.8% (n=1887)	52.8% (n=1887)
1.0 or less	28.6% (n=7) (0.4)	60.0% (n=5) (1.3)
1.1 - 2.0	36.8% (n=19) (0.5)	100.0% (n=8) (-)
2.1 or more	66.7% (n=18) (1.8)	100.0% (n=4) (-)

*note that these drivers also had THC-acid detected

For those who tested positive for THC-alone, the proportion culpable varied with THC concentration. The percentage of drivers with concentrations less than 2 ng/mL who were culpable was lower than the culpability of drug-free drivers, although a higher percentage of drivers were culpable when concentrations of THC exceeded 2 ng/mL. However, there was no significant

difference in the culpability of drivers across THC concentrations for THC alone ($\chi^2_3=5$, $p>0.05$), and there was no significant linear relationship ($\chi^2_1=0.001$, $p>0.05$). A higher percentage of drivers who tested positive for THC in combination with other drugs were culpable compared with drug-free drivers, irrespective of THC concentration. There was a significant difference in the proportion of culpable drivers across THC concentrations for THC in combination with other drugs ($\chi^2_3=10.7$, $p<0.05$). There was also a significant linear relationship ($\chi^2_1=10$, $p<0.01$). A comparison was made between the mean THC concentration for culpable and not culpable drivers. It was found that culpable drivers had a higher mean THC concentration, but the difference was not statistically significant (2.22 ng/mL vs 1.58 ng/mL, $t=1.9$, $p=0.057$).

Benzodiazepines

Table 6 shows that a higher proportion of drivers who tested positive for benzodiazepines were culpable compared with drug-free drivers. The difference in the proportion of culpable drivers across benzodiazepine groups (including the drug-free group) was significant for benzodiazepines in combination with other drugs ($\chi^2_3=14.1$, $p<0.01$), but not for benzodiazepines-alone ($\chi^2_3=6.9$, $p>0.05$). However, when comparing the proportion of culpable drivers with therapeutic and above therapeutic/toxic levels of benzodiazepines-alone with drug-free drivers, there was a significant difference ($\chi^2_1=5.6$, $p<0.05$). There was also a significant linear relationship for benzodiazepines both alone and in combination with other drugs ($\chi^2_1=6.5$, $p<0.05$ and $\chi^2_1=13.6$, $p<0.001$, respectively).

Table 6: Culpability of injured drivers and benzodiazepine level: alone/in combination with other drugs. Odds-ratios for the benzodiazepine groups compared with the drug-free group are included in brackets

Benzodiazepine level	Percentage culpable	
	Benzodiazepines alone (n=46)	Benzodiazepines in combination (n=20)
Drug-free	52.8% (n=1887)	52.8% (n=1887)
Sub-therapeutic	59.1% (n=22) (1.3)	75.0% (n=4) (2.7)
Therapeutic	78.9% (n=19) (3.3)	100.0% (n=12) (-)
Above therapeutic or toxic	80.0% (n=5) (3.6)	100.0% (n=4) (-)

Stimulants

As the frequency of stimulant use was low, sub-therapeutic and therapeutic classes were combined. Table 7 shows that a higher proportion of drivers who tested positive for stimulants were culpable compared with those who were drug-free. However, there was no significant difference in the proportion of culpable drivers across stimulant groups (including the drug-free group) for stimulants-alone ($\chi^2_2=1.6$, $p>0.05$), or for stimulants in combination with other drugs ($\chi^2_2=3.6$, $p>0.05$). There was no significant linear relationship for either group ($\chi^2_1=1.3$, $p>0.05$ and $\chi^2_1=3.2$, $p>0.05$, respectively).

Table 7: Culpability of injured drivers and stimulant level: alone/in combination with other drugs. Odds-ratios for the stimulant groups compared with the drug-free group are included in brackets

Stimulant level	Percentage culpable	
	Stimulants alone (n=16)	Stimulants in combination (n=4)
Drug-free	52.8% (n=1887)	52.8% (n=1887)
Sub-therapeutic/therapeutic	70.0% (n=10) (2.1)	100.0% (n=2) (-)
Above therapeutic	66.7% (n=6) (1.8)	100.0% (n=2) (-)

Discussion

The present study found a clear, concentration-dependent relationship between alcohol and culpability. Drivers who tested positive for alcohol were significantly more likely to be culpable than drug-free drivers and this effect was more marked at higher BACs. Moreover, drivers who tested positive for alcohol in combination with either THC or benzodiazepines were significantly more likely to be culpable. However, they did not differ significantly from drivers who tested positive for alcohol-alone, which suggests that there was no increase in culpability beyond that produced by alcohol. These results are in accordance with those from earlier studies showing a strong causal role for alcohol in road crashes (Terhune, 1982; Terhune *et al.*, 1992; Drummer, 1994).

This study also found a significant relationship between benzodiazepines and culpability. Drivers who tested positive for benzodiazepines had a significantly higher culpability rate than drug-free drivers. Prior research has yielded inconsistent results, with some studies finding no significant relationship between benzodiazepines and crash risk (Jick *et al.*, 1981; Benzodiazepine and Driving Collaborative Group, 1993), and others finding a strong relationship (Skegg *et al.*, 1979; Neutel, 1995). The significant positive finding in the present study is, in part, due to the comparatively larger sample size. There was also a significant relationship between benzodiazepine concentration and culpability. Amongst those who had a benzodiazepine concentration at or above the therapeutic level, culpability was significantly greater than for the drug-free group. Within this group the majority of drivers had concentrations within the therapeutic range. Although the effect was not as great in magnitude as the effect of alcohol, the data here represent clear evidence of increased culpability associated with the benzodiazepine group of drugs.

In contrast, the present study found no significant relationship between THC and culpability. While a larger number of injured drivers tested positive for THC compared with other culpability studies (eg Williams *et al.*, 1985; Terhune *et al.*, 1992), their culpability rate was no higher than that of the drug-free group. As in the present study, these past studies found that a higher percentage of drug-free drivers were culpable for the crash compared with drivers who tested positive for THC-alone. However, the results failed to reach statistical significance. Moreover, some studies (eg Warren *et al.*, 1981) were unable to determine a culpability rate for THC-alone due to the small number of drivers testing positive. Another limitation in some past studies has been the failure to separate drivers positive for THC with those only positive for the inactive metabolite THC-acid. For example, Drummer (1994) found that drug-free drivers had a higher culpability rate than drivers positive for cannabinoids, although the difference was not statistically significant. Drummer also acknowledged that only THC-acid was found in the majority of cases, and that results were usually from urine samples, not blood. The presence of THC-acid only confirms that marijuana has been used, and does not indicate impairment at the time of the crash. However, Drummer (1999) reanalysed the relationship between marijuana and culpability using additional data, separating drivers who tested positive for THC from drivers positive for THC-acid. There were 39 drivers

who tested positive for THC-alone, and 37 of these were judged culpable. This was significantly higher than the culpability rate of drug-free drivers. In addition, the THC concentrations were reasonably high (the mean concentration was 21 ng/mL, with a range of 1.4 to 228 ng/mL), suggesting recent use of marijuana. This is the first study to date that has found statistically significant evidence of increased culpability with marijuana, using a relatively large sample.

It is important to recognise that in several earlier studies, as in the present one, the direction of the cannabis effect, while not statistically significant, was indicative of *decreased* rather than increased culpability (Williams *et al.*, 1985; Terhune *et al.*, 1992; Drummer, 1994). Only in Terhune (1982) did the results show a non-significant detrimental effect. Together, these findings suggest that the failure to find an adverse effect of cannabinoids on driving is not simply due to inadequate sample size. The drug may not produce a clear, unequivocal adverse effect on driving performance as is sometimes supposed. However, further examination of the potential impact of THC on crash risk can be obtained by examining the relationship between culpability and drug concentration. The evidence for decreased culpability in this study was most obvious at low THC concentrations and it is possible that at these concentrations the drug alters driving behaviour so as to decrease crash risk. At higher concentrations exceeding 2 ng/mL, THC-positive drivers had a higher culpability rate than drug-free drivers. These results are suggestive of a biphasic effect of THC on crash culpability. However, since none of the differences were statistically significant, this remains an intriguing possibility only. Furthermore, it should be recognised that the vast majority of THC concentrations were in the very low range relative to the values that can be achieved by marijuana smokers. It is important, therefore, to be cautious about relationships between THC concentration and culpability. However, unlike previous studies, the present study had a relatively large number of THC-positive cases (n=44) with no other drugs present. By comparison, there were approximately the same number of benzodiazepine-alone cases (n=46), and for this drug an adverse effect was detected. This suggests that the sample size was sufficient to detect any adverse effect of THC had one been present.

There was some suggestion of increased culpability amongst drivers testing positive for stimulants, but statistical significance was not achieved. A sample much greater than that obtained here would be needed to confirm whether there is such a relationship. However, relatively few drivers tested positive for stimulants other than pseudoephedrine and in many cases stimulants were found at sub-therapeutic or therapeutic levels only. This relationship could be further investigated using a much larger sample than that obtained here, but given the low detection rates in this sample and the evidence that at least some are not culpable, it is reasonable to conclude that stimulants do not play a major role in road crashes.

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