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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

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Abstract

Blood samples from 2500 injured drivers were analysed for alcohol, cannabinoids (measured by the presence of THC), benzodiazepines and stimulants. The relationship between the prevalence and concentration of drugs and the culpability of the driver was examined using an objective method for assessing culpability. There were no significant differences between males and females with respect to culpability. However, there was a relationship between age and culpability: drivers under 26 years and over 60 years were more likely to be culpable. 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. Conversely, a lower percentage of drivers who only tested positive for THC were culpable for the crash compared with drug-free drivers. This difference was not statistically significant. For car drivers in single-vehicle crashes, the majority of drivers were judged culpable irrespective of drug use. In multiple-vehicle crashes, car drivers testing positive for alcohol only or benzodiazepines only were more likely to be culpable for the crash compared with drug-free drivers. For motorcycle riders in both single- and multiple-vehicle crashes, there were no significant differences between the drug-positive and drug-free groups. A higher percentage of drug-free riders in multiple-vehicle crashes were culpable compared with riders who only tested positive for THC, but this difference was not statistically significant. There was a significant concentration-dependent relationship between alcohol and culpability: as blood alcohol concentration increased, so did the percentage of culpable drivers. When THC was used alone, there was no significant increase in culpability. For those drivers with benzodiazepines at therapeutic concentrations and above, there was a significant increase in culpability. The relationship between stimulants and culpability was not significant, although a higher proportion of stimulant-positive drivers were culpable compared with drug-free drivers. The combinations of alcohol and THC, and alcohol and benzodiazepines also produced a significant increase in culpability, but this increase was not significantly greater than that produced by alcohol alone.

Keywords: Alcohol; THC; Benzodiazepines; Stimulants; Injured drivers; Culpability; Drug concentration

1. Introduction

A variety of methods have been used to assess the role of drugs in road crashes. Case-control studies have compared the prevalence of alcohol and other drugs in crash-involved drivers with the prevalence in a control group of drivers who have not been involved in crashes,

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driving under similar conditions and at similar times of the day (Bo et al., 1975 in Norway; Honkanen et al., 1980 in Finland; Ferrara et al., 1990 in Italy). Depending on the study, the control groups have not always accurately represented the general population, and, accordingly, the results may under- or over-estimate the prevalence of drugs in these groups. Moreover, it has to be recognised that the presence of drugs in drivers does not necessarily mean that the drug was a causal factor in the crash. The over-representation of drivers testing positive for drugs could be because of some external factor. For example, those who use drugs may drive more, or in more dangerous locations, or may be younger and less experienced.

An alternative approach is to use culpability or responsibility analysis. Studies using culpability analysis are 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. This method enables the relationship between drug use and crash risk to be examined, where crash risk is measured by the percentage of drivers judged culpable for the crash. The level of objectivity used can differentiate the methods of assessing culpability. Some rely on police evaluations, where drivers are judged as either culpable or not culpable for the crash (e.g. Williams et al., 1985 in the USA; Benzodiazepine and Driving Collaborative Group, 1993 in France). Although these studies have found some significant relationships between drug use and culpability, many only examined this relationship for alcohol (e.g. Maull et al., 1984 and Waller et al., 1995 in the USA; Bailey, 1985 in New Zealand). In addition, the way in which culpability was assigned in these studies often relied heavily on opinions rather than facts.

Other studies have used more objective methods of establishing culpability. Each crash is assessed using defined criteria, with information from police evaluations including contributing factors such as vehicle condition, weather and lighting, and roadway characteristics and conditions (Terhune, 1982; Soderstrom et al., 1991; Terhune et al., 1992 in the USA).

Results from studies using culpability analysis have consistently reported a significant relationship between alcohol and culpability. American studies by Terhune (1982) and Terhune et al. (1992) using non-fatally injured and fatally injured drivers, respectively, and an Australian study by Drummer (1994) using fatally injured drivers 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 blood alcohol concentration (BAC) increased.

Studies examining the relationship between culpability and Δ^9tetrahydrocannabinol (THC) generally found that when used alone, THC was associated with *lower* culpability (Terhune et al., 1992; Drummer, 1994). Conversely, a study by Terhune (1982) found the reverse. However, in all three studies the differences were not statistically significant because the number of drivers testing positive for cannabinoids only was small. Moreover, the majority of drivers in Drummer's study tested positive for the inactive metabolite of THC, which only confirms that marijuana has been used, and does not indicate impairment at the time of the crash.

Where the relationship between benzodiazepines and culpability has been examined the results have been inconsistent. A study in France by the Benzodiazepine and Driving Collaborative Group (1993) using non-fatally injured drivers 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 only tested positive for benzodiazepines were culpable for the crash compared with drug-free drivers. Conversely, Drummer (1994) found the reverse, and a study by Terhune et al. (1992) found that drivers who only tested positive for benzodiazepines and drug-free drivers had almost identical culpability rates. However, in these last three studies 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. 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 only, although these differences were not statistically significant. As with cannabinoids and benzodiazepines, the number of drivers testing positive for stimulants only was relatively small, and in the earlier study by Terhune (1982) a culpability rate for stimulants could not even be calculated due to the small sample size.

These studies also examined the relationship between drug concentration and culpability. Terhune (1982) found that drivers with higher concentrations of THC were more likely to be culpable than drug-free drivers, but this difference was not statistically significant. The group of drivers with low concentrations of THC had a culpability rate very similar to that of the drug-free group, although this result must be interpreted with caution due to the small sample size. Terhune et al. (1992) divided THC, benzodiazepines and stimulants into four concentration categories: trace, low, high and toxic. In the majority of cases the drugs were detected at trace or low concentrations, with the exception of amphetamine that was found in high to toxic concentrations in 80% of amphetamine-positive drivers. Drivers who tested positive for THC and drivers who tested

positive for diazepam had trace or low concentrations of the drug (< 19 and < 2.5 ng/ml, respectively) in 93 and 100% of cases, respectively. Drummer (1994) rated drug concentrations from 1 (indicating a low therapeutic concentration) to 3 (indicating a high, non-therapeutic concentration). Consistent with prior studies, drugs were almost always detected at low therapeutic concentrations. However, all drivers with medium or high concentrations of benzodiazepines and stimulants were judged culpable.

Although the relationship between drugs other than alcohol and culpability is not entirely clear when these drugs are taken alone, the evidence indicates that the combination of alcohol and these drugs significantly increases culpability (Terhune, 1982; Terhune et al., 1992; Drummer, 1994). However, Terhune et al. (1992) also found that the culpability rate for drivers who tested positive for alcohol and THC, and alcohol and benzodiazepines did not differ significantly from drivers who tested positive for alcohol alone. This suggests that high concentrations of alcohol may explain the increased crash risk.

The aim of this study was to conduct culpability analyses using an objective scoring criterion 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 influence of variables such as gender, age and the type and number of vehicles involved in the crash. The relationship between drug concentration and culpability was also examined. Such an examination may strengthen evidence concerning the effect of a drug or may reveal a relationship hidden when results from all subjects with positive concentrations are combined.

2. Method

2.1. Sample selection and procedure

A detailed description of the sample selection, procedure and analytical methods is contained in Longo et al. (1999a). In brief, blood samples from 2500 injured drivers in South Australia were collected in the periods April 1995 to August 1995, and December 1995 to August 1996. These samples were analysed for alcohol, cannabinoids (THC and THC-acid), benzodiazepines and stimulants. The blood test results were matched with 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. 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.

2.2. 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. The role of the other driver(s) in multiple vehicle crashes is not directly assessed, but influences the final result through the type of crash.

Drivers were assigned a score for each factor reflecting the level of mitigation from 1 (not mitigating, that is, favourable to safe driving) to 4 (mitigating, that is, not favourable to safe driving). Scores for these mitigating factors were added and used to assign subjects to one of three categories denoting their level of culpability for the crash. Scores between 8 and 12 inclusive meant that the crash was due to driver performance (culpable), between 13 and 15 inclusive meant that the crash was due, in part, to driving conditions (contributory), and between 16 and 26 inclusive meant that the crash was due to factors other than the performance of the injured driver (not culpable). Culpability was assessed without knowledge of a driver's drug results.

3. Results

The 2500 injured drivers included small samples of truck drivers ($n = 55$) and ambulance and bus drivers ($n = 12$). For these relatively small groups, factors related to culpability and likelihood of injury will be different from car drivers and motorcycle riders. For this reason data from truck, ambulance and bus drivers were not included in the culpability analyses.

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

culpable and not culpable car drivers ($n = 2029$) and motorcycle riders ($n = 250$).

3.1. Gender and age of drivers

There were no significant differences between males and females in this sample with respect to culpability (59.7% compared with 56.6%: $\chi^2_1 = 2.47$, $P > 0.05$). However, there was a significant relationship between age and culpability. Nearly two-thirds of drivers aged less than 26 years were culpable, compared with 50.5% of drivers aged between 26 and 35 years, 48.6% of drivers between 36 and 59 years and 74.4% of drivers aged 60 years or more. Younger and older drivers were thus more likely to be culpable than other age groups ($\chi^2_3 = 72.82$, $P < 0.001$).

3.2. Effects of drugs

Table 1 shows the percentage of car drivers and riders 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 only, benzodiazepines only, alcohol and THC and alcohol and benzodiazepines. It is worth noting that the culpability rate of drivers who only tested positive for THC did not differ significantly from the drug-free group. Moreover, the culpability rate of drivers who only tested positive for alcohol was not significantly different from that of drivers who tested positive for alcohol in combination with either THC or benzodiazepines.

Table 1
Percentages of injured car drivers and riders testing positive for the various drugs and drug combinations according to level of culpability for the crash[a]

Drug combination	Percentage culpable
Drug-free ($n = 1887$)	52.8
Alcohol only ($n = 250$)	90.0 (8.0)**
THC only ($n = 44$)	47.7 (0.82)
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 (–)

[a]Drivers judged contributory or drivers for whom culpability was unknown were excluded. Odds-ratios for the drug-positive groups compared with the drug-free group are included in brackets.

*Denotes statistically significant differences between drug-free and drug-positive groups: $P < 0.05$.

**$P < 0.001$.

Table 2
Percentages of injured car drivers deemed culpable according to the drug combination and number of vehicles involved[a]

Drug combination	Single vehicle (% culpable)	Multiple vehicle (% culpable)
Drug-free	91.2 ($n = 318$)	44.8 ($n = 1361$)
Alcohol only	95.7 ($n = 164$) (2.2)	79.7 ($n = 64$) (4.8)**
THC only	90.9 ($n = 11$) (0.95)	42.9 ($n = 21$) (0.92)
Alcohol + THC	100.0 ($n = 7$) (–)	83.3 ($n = 6$) (6.2)
Benzodiazepines only	80.0 ($n = 10$) (0.38)	65.6 ($n = 32$) (2.4)*
Stimulants only	100.0 ($n = 1$) (–)	66.7 ($n = 12$) (2.5)
Alcohol + benzodiazepines	100.0 ($n = 9$) (–)	85.7 ($n = 7$) (7.4)
Stimulants + THC	–	100.0 ($n = 1$) (–)
Benzodiazepines + THC	–	100.0 ($n = 2$) (–)
Other combinations	100.0 ($n = 2$) (–)	100.0 ($n = 1$) (–)

[a]Drivers judged contributory or drivers for whom culpability was unknown were excluded. Odds-ratios for the drug-positive groups compared with the drug-free group are included in brackets.

*Denotes statistically significant differences between drug-free and drug-positive groups: $P < 0.05$.

**$P < 0.001$.

3.3. Culpability of drivers by number and type of vehicles involved

The following analyses examine driver culpability separately for single- and multiple-vehicles. Car drivers are considered first, followed by motorcycle riders.

Table 2 shows that in single-vehicle crashes, the majority of car drivers were judged culpable for the crash irrespective of the particular drug combination. No statistically significant differences were found in culpability rates between drug-positive and drug-free groups.

In multiple-vehicle crashes, 45% of drug-free car drivers were culpable for the crash. Interestingly, the percentage of car drivers only positive for THC who were culpable was very similar to the percentage of drug-free drivers who were culpable. The groups that differed significantly from the drug-free group were alcohol only and benzodiazepines only. That is, car drivers testing positive for alcohol or benzodiazepines were significantly more likely to be culpable for the crash compared with drug-free drivers.

It is clear from Table 3 that the absolute number of riders who tested positive for each of the drugs and drug combinations was very low. In both single- and multiple-vehicle crashes, there were no significant di-

fferences between riders testing positive for the various drug classes and combinations, and drug-free riders. Although a much lower percentage of THC-positive riders in multiple-vehicle crashes were culpable compared with the drug-free group (9.1 vs. 39.8%), this difference was not statistically significant.

3.4. 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. Again, the analyses are restricted to car drivers and motorcycle riders ($n = 2279$), and drivers who were judged contributory or for whom culpability was unable to be determined were excluded.

3.5. 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 only for alcohol ($\chi^2_4 = 133$, $P < 0.001$), 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). There was no significant difference in the mean BAC of drivers positive for alcohol alone and drivers positive for alcohol in combination with other drugs (0.136 vs. 0.146%, $t = 0.07$, $P > 0.05$). A comparison was also 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 3
Percentages of injured riders deemed culpable according to the drug combination and number of vehicles involved[a]

Drug combination	Single vehicle (% culpable)	Multiple vehicle (% culpable)
Drug-free	74.1 ($n = 54$)	39.8 ($n = 154$)
Alcohol only	91.7 ($n = 12$) (3.9)	60.0 ($n = 10$) (10.2)
THC only	100.0 ($n = 1$) (–)	9.1 ($n = 11$) (0.17)
Alcohol+THC	–	0.0 ($n = 1$) (–)
Benzodiazepines only	66.7 ($n = 3$) (0.7)	100.0 ($n = 1$) (–)
Stimulants only	100.0 ($n = 1$) (–)	50.0 ($n = 2$) (1.7)
Alcohol+benzodiazepines	–	–
Stimulants+THC	–	–
Benzodiazepines+THC	–	–
Other combinations	–	–

[a] Odds-ratios for the drug-positive groups compared with the drug-free group are included in brackets. Drivers judged contributory or drivers for whom culpability was unknown were excluded.

Table 4
Culpability of injured drivers and BAC: alone/in combination with other drugs[a]

BAC (%)	Percentage culpable	
	Alcohol alone	Alcohol in combination with other drugs
Drug-free	52.8 ($n = 1887$)	52.8 ($n = 1887$)
<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%

[a] Contributory drivers excluded. Odds-ratios for the BAC ranges compared with the drug-free group are included in brackets.

3.6. 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.

For those who tested positive for THC only (although THC-acid was also detected), the proportion culpable varied with THC concentration. The percentage of drivers with concentrations less than 2 ng/ml who were culpable was less than the culpability of drug-free drivers, although a higher percentage of drivers were culpable when the concentration 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 was culpable compared with drug-free drivers, irrespective of the 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 vs. 1.58 ng/ml, $t = 1.9$, $P = 0.057$).

Table 5
Culpability of injured drivers and THC concentration: alone/in combination with other drugs[a]

THC concentration (ng/ml)	Percentage culpable	
	THC alone[b]	THC in combination with other drugs[b]
Drug-free	52.8 ($n=1887$)	52.8 ($n=1887$)
1.0 or less	28.6 ($n=7$) (0.36)	60.0 ($n=5$) (1.3)
1.1–2.0	36.8 ($n=19$) (0.52)	100.0 ($n=8$) (–)
2.1 or more	66.7 ($n=18$) (1.8)	100.0 ($n=4$) (–)

[a] Drivers judged contributory or drivers for whom culpability was unknown were excluded. Odds-ratios for the THC groups compared with the drug-free group are included in brackets.
[b] Note that these drivers also had THC-acid detected.

3.7. 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) (see Table 7).

Table 6
Culpability of injured drivers and and benzodiazepine level: alone/in combination with other drugs[a]

Benzodiazepine level	Percentage culpable	
	Benzodiazepines alone	Benzodiazepines in combination with other drugs
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 ($n=4$) (–)

[a] Drivers judged contributory or drivers for whom culpability was unknown were excluded. Odds-ratios for the benzodiazepine groups compared with the drug-free group are included in brackets.

Table 7
Culpability of injured drivers and stimulant level: alone/in combination with other drugs[a]

Stimulant level	Percentage culpable	
	Stimulants alone	Stimulants in combination with other drugs
Drug-free	52.8 ($n=1887$)	52.8 ($n=1887$)
Sub-therapeutic or therapeutic	70.0 ($n=10$) (2.1)	100.0 ($n=2$) (–)
Above therapeutic	66.7 ($n=6$) (1.8)	100.0 ($n=2$) (–)

[a] Drivers judged contributory or drivers for whom culpability was unknown were excluded. Odds-ratios for the stimulant groups compared with the drug-free group are included in brackets.

3.8. Stimulants

As the frequency of stimulant use was low, sub-therapeutic and therapeutic classes were combined. The tables show 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).

3.9. Alcohol and THC

There were 15 cases where car drivers or riders tested positive for both alcohol and THC. These drivers were judged culpable for the crash in 85.7% of cases, and it is interesting to determine whether this culpability could be attributed to alcohol alone. The majority of these drivers were male (80%), less than 26 years of age (93.3%), with a mean age of 21.6 years. In comparison, 80.1% of drivers who only tested positive for alcohol were male, and 46.5% were less than 26 years of age. The mean age for these drivers was 30 years.

There was no significant difference in the mean BAC between drivers who only tested positive for alcohol, and those who tested positive for alcohol and THC (0.132 vs. 0.117%, respectively: $t = 0.76$, $P > 0.05$). There was also no significant difference in culpability between the two groups. Table 4 shows that 90% of drivers who only tested positive for alcohol were culpable compared with 85.7% of drivers who tested positive for alcohol and THC ($\chi^2_1 = 0.004$, $P > 0.05$). This suggests that the effect of alcohol and THC was due mainly to the effect of alcohol. However, these results should be interpreted with caution as the number of

drivers who tested positive for alcohol and THC was small.

3.10. Alcohol and benzodiazepines

There were 16 cases where drivers tested positive for both alcohol and benzodiazepines and in all but one case the driver was judged culpable. Just over half of these 16 drivers were female (56.3%) and the majority were over 35 years of age (68.8%), with a mean age of 38.7 years. In comparison, only 19.9% of drivers who tested positive for alcohol only were female, and 23.6% were over 35 years of age. The mean age for these drivers was 30 years.

There was a significant difference in the mean BAC between drivers who only tested positive for alcohol, and those who tested positive for alcohol and benzodiazepines (0.132 vs. 0.169%, respectively: $t=2$, $P<0.05$). However, there was no significant difference in culpability between the two groups. Table 4 shows that 90% of drivers who only tested positive for alcohol were culpable compared with 93.8% of drivers who tested positive for alcohol and benzodiazepines ($\chi^2_1=0.003$, $P>0.05$). As was the case with alcohol and THC, the small number of drivers who tested positive for alcohol and benzodiazepines precludes any meaningful conclusions.

4. Discussion

A major limitation of culpability studies using fatally injured drivers has been the high percentage of culpable drivers among the drug-free group. This high baseline means that it is difficult to find statistically significant differences between drug-free and drug-positive drivers with respect to their level of culpability. One of the benefits of using non-fatally injured drivers is that the percentage of drug-free drivers judged culpable for the crash is generally much lower. This was the case in the present study. It was found that 52.8% of drug-free drivers were judged culpable for the crash, which is much lower than the culpability rate reported in earlier studies using fatally injured drivers. For example, Terhune et al. (1992) found that 68% of drug-free drivers were culpable, and the percentage rose to 70% in a study by Drummer (1994).

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 only tested positive for alcohol, 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) using non-fatally injured drivers, and Terhune et al. (1992) using fatally injured drivers found that a significantly higher percentage of alcohol-positive drivers were culpable compared with drug-free drivers. Similarly, Drummer (1994) using fatally injured drivers found that 94% of drivers who only tested positive for alcohol alone were culpable compared with 70% of drug-free drivers.

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; Leveille et al., 1994). Other studies have been suggestive (Honkanen et al., 1980; Drummer, 1994) and studies by Skegg et al. (1979), Ray et al. (1992) and Neutel (1995) found a strong relationship between prescription of a benzodiazepine and crash risk. However, this does not preclude a variety of mechanisms other than a direct impairing effect of the drug. With the exception of Neutel (1995), these studies did not differentiate between people who were prescribed a benzodiazepine for the first time and chronic users, and drivers may not necessarily have been using the drug prior to the crash. Conversely, a culpability study by Terhune (1982) found a non-significant decrease in the proportion culpable amongst drivers testing positive only for benzodiazepines and Terhune et al. (1992) found no difference. 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 (for benzodiazepines alone the proportion of culpable drivers was approximately 79% for those in therapeutic ranges and above), the data clearly indicate an adverse effect of this drug class. Terhune et al. (1992) may have had an insufficient number of subjects with high benzodiazepine concentrations to observe such an effect. While Drummer (1994) did show increased culpability amongst the benzodiazepine-positive drivers in his sample, the numbers were too small for statistical significance. The largest study of benzodiazepines and road crashes was carried out in France (Benzodiazepine and

Driving Collaborative Group, 1993). Concentrations of alcohol and the presence of benzodiazepines in blood were determined and culpability measured for 2852 injured drivers. A relationship was found between alcohol and culpability. However, there was no effect of benzodiazepines. There were several limitations of this study. No correction was made for other drugs (e.g. cannabinoids) as these were not measured. An EMIT immunoenzymatic assay was used for benzodiazepines. This method provides only qualitative data and samples with very low concentrations would be included as positive results.

The results here thus represent clear evidence of increased culpability associated with the benzodiazepine class of drugs. Such effects of benzodiazepines are not entirely surprising. These drugs show some commonality in mechanism of action and effects with alcohol. Laboratory studies have shown performance impairment similar to, although of lesser magnitude, than alcohol (Linnoila et al., 1990; Moskowitz and Burns, 1977). However, these studies have used healthy volunteers as subjects. It is reasonable to assume most injured drivers testing positive for benzodiazepines would have been prescribed the drugs for anxiety or insomnia. We do not know the effects of benzodiazepines on the driving performance of people with these conditions, or whether they would be more impaired without taking the drugs. While some warnings about the deleterious effects of benzodiazepines are issued when they are prescribed, it may be useful to reinforce and corroborate these warnings with the empirical findings presented here.

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 (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 only. However, the results failed to reach statistical significance. Moreover, some studies (Warren et al., 1981; Garriott et al., 1986) 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. However, 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. There were only ten drivers who tested positive for THC alone, a number too small to obtain accurate culpability rates (Drummer, 1999). The present study thus has important implications in clarifying the relationship between THC and culpability, with the results confirming previous research suggesting that THC alone may not increase crash risk. Moreover, unlike some previous studies (e.g. Drummer, 1994), drivers in this study who tested positive for THC-acid only (which does not suggest recent use of marijuana) were excluded from the culpability analyses.

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 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.

Finally, the data here and in other culpability studies do not exclude the possibility of an adverse effect of cannabinoids if THC concentration is sufficiently high. At the extreme, cannabinoids are capable of producing hallucinations at very high doses (well above those usually employed by users of the drug). A person affected in this way would clearly exhibit worsened driving performance. Even lower concentrations than

these, but at the high end of those achieved by most users, may have an adverse effect. However, it may also be true that very few people affected in this way drive a vehicle. What can be said from the results here is that at the THC concentrations found in these injured drivers there was no general effect of THC on culpability.

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.

While not the subject of the present research, it is possible to speculate on the possible mechanisms that result in the marked effect of alcohol, somewhat smaller effect of benzodiazepines and no effect of cannabinoids on road crashes. While there is evidence of at least some psychomotor impairment for cannabinoids (Smiley et al., 1981; Chesher et al., 1986) they do not have the same profile of effects as alcohol. One of the major differences observed between the effects of alcohol and cannabinoids is a decrease in risk-taking behaviour with cannabinoids (Smiley et al., 1987; Stein and Allen, 1987; Robbe, 1995). It may be that for road crashes this is the most critical aspect of alcohol-induced impairment. In this respect benzodiazepines can be regarded as a 'weaker' form of alcohol: they can produce increased risk taking, but the effect is of lesser magnitude and occurs under more limited conditions (Linnoila and Hakkinen, 1973; Linnoila and Mattila, 1973; van Laar et al., 1992).

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