

# Cannabis and the Risk of Crash Involvement

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Drugs have long been a focus of law enforcement in Australia but recent legislation in a number of Australian states now requires routine drug testing of drivers (testing for cannabis and methamphetamine), with the stated aim of reducing road crashes. Such legislation is justified if these drugs are known to increase the risk of crashing. Literature concerning cannabis and road crash involvement was reviewed, with emphasis given to studies documenting the relative crash risk associated with driving after use of cannabis. All case-control and culpability studies of cannabis and crashes have been characterised by methodological flaws that make interpretation of the results difficult. Two recent Australian studies analysed the relationship between tetrahydrocannabinol (THC, the psychoactive component of cannabis) measured in the blood and crash culpability. These two studies produced contradictory results. In summary, the risk of crash involvement associated with driving under the influence of cannabis remains to be determined.

## Introduction

Increasing attention has been devoted in recent years to the issue of driving after the use of drugs that are known to affect the functioning of the central nervous system. Researchers have been interested in ascertaining if, and to what extent, such drugs impair driving ability and whether their use increases the risk of crash involvement. Driving under the influence of cannabis has been of particular interest, which is a result of cannabis being found in surveys to be the most frequently used illicit drug (Australian Institute of Health and Welfare 2007; Johnston et al. 2004; Miller and Draper 2001) and to cannabis being the most frequently detected psychoactive substance, after alcohol, among driving populations (Bates and Blakely 1999; Couper and Logan 2004; Kelly et al. 2004; Walsh et al. 2004). Cannabis has also been found in experimental studies to negatively affect skills necessary for safe driving, such as tracking, reaction time, memory and learning, divided attention, sustained attention, perception, thinking and problem solving, and co-ordination (Bates and Blakely 1999; Berghaus and Guo 1995; Couper and Logan 2004; Department of Environment Transport and the Regions 2000; Kelly et al. 2004; Ramaekers et al. 2004). Further studies have been conducted in which participants have performed driving tasks, either in a driving simulator or on the road, after taking doses of cannabis. Impairment of driving performance has been found to last for up to three hours

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(Couper and Logan 2004), with effects being dose-related and greatest for highly automated behaviours, such as road tracking, rather than for the more complex tasks requiring conscious control (Ramaekers et al. 2004). However, the level of impairment in driving studies has been found to be less severe than that found in laboratory studies (Kelly et al. 2004), which may be due to drivers affected by cannabis compensating for their impairment (Smiley 1999).

Although laboratory and on-road studies provide important information regarding the effects of drugs on driving, research into the relationship between recent use of cannabis and involvement in road crashes is of prime importance. If it can be established that driving under the influence of cannabis is clearly related to crash involvement, as is the case for alcohol (Borkenstein et al. 1974; McLean and Holubowycz 1980), then policies and methods of enforcement to reduce driving after cannabis use would be justified. Roadside testing of the oral fluid (saliva) of drivers, to identify those who have recently used cannabis, is already being practised in a number of Australian jurisdictions. The purpose of the present review is to determine whether the currently available scientific evidence provides a clear indication of the existence of a positive relationship between driving under the influence of cannabis and road crash involvement.

There are two main types of studies that can address this issue. First, there are case-control studies, which can be used to estimate crash risks associated with driving under the influence of cannabis by comparing the levels of cannabis in crash-involved drivers with those of non-crash-involved drivers. The second type is a study of the relationship between cannabis use and responsibility (or 'culpability') for crashes. The sum of the evidence provided in these two different types of studies will form the basis for the conclusions that can be drawn at this time regarding whether cannabis use constitutes a road safety problem. First, however, it is necessary to briefly discuss detection of cannabis in body fluids.

## **Detection of cannabis in body fluids**

In order to determine whether a driver has used cannabis prior to driving, it is necessary to detect and measure THC (delta-9-tetrahydrocannabinol, the psychoactive component of cannabis) in the driver's system. There are a number of alternative biological matrices available for drug tests, with the most commonly used being blood, urine and saliva. In order to interpret these

tests, it is necessary to know about the different time courses of THC and its inactive metabolites in the different matrices.

Peak plasma concentrations of THC in blood typically occur within 15 minutes of smoking but decline rapidly due to distribution into body tissues and fat. An hour after consumption, it is rare to get THC plasma concentrations over 10 ng/ml (5 ng/ml in whole blood). Within eight to 12 hours, plasma levels fall below the limits of quantitation in occasional users (Couper and Logan 2004; Drummer 2004).

Unlike alcohol, there is no clear relationship between blood concentrations of THC and impairment (Couper and Logan 2004; Grotenhermen 2003; Kalant 2004), with the time of maximum blood concentration preceding the time of maximum impairment of driving-related abilities (Berghaus et al. 2000). Metabolites of THC last much longer in the blood than THC itself but offer no indication of impairment, only previous exposure (Bates and Blakely 1999; Couper and Logan 2004; Grotenhermen 2003; Ogden and Moskowitz 2004).

Testing of urine will only reveal whether a person has been exposed to cannabis, not whether they are impaired by it. It can take as long as four hours for metabolites to appear in urine in concentrations sufficient to be detected by an immunoassay. Positive results indicate use within the previous one to three days, although this period is longer for heavy chronic users (Couper and Logan 2004; Drummer 2004).

The most accessible matrix for detection of drugs is oral fluid (saliva). The presence of THC in saliva suggests that cannabis has been smoked or eaten in the previous hour or two and is, therefore, more likely to be indicative of impairment (Kalant 2004). However, whilst blood levels decline continuously after the initial peak, a person's oral fluid THC level can continue to fluctuate over time. This makes it impossible to predict blood THC levels from levels of THC detected in oral fluid (Huestis and Cone 2004).

#### *Case control studies*

As noted by Chipman et al. (2003), case-control studies for drugs and crash involvement are difficult to carry out. Samples for toxicological analysis are difficult and expensive to obtain, particularly from non-crash-involved drivers. There can also be legal barriers to the collection of control data in some jurisdictions (Walsh et al. 2004). For these

reasons, there are relatively few case-control studies concerning drug driving that have been reported in the literature.

One recent case-control study was conducted in Quebec, Canada (Brault et al. 2004; Dussault et al. 2002). This study compared the level of drugs detected in fatally injured drivers with that detected in the general driving population using roadside surveys. Both blood and urine were collected from fatally injured drivers (cases), and breath, urine and saliva samples were collected from non-crash-involved drivers (controls). Dussault et al. (2002) compared the results of urine analyses for 354 cases and 5,931 controls. Cannabis was detected in 19.5 per cent of cases and 6.7 per cent of controls. The odds ratio for crash involvement for all drivers who tested positive for cannabis (including those in which other drugs or alcohol were involved) was 4.6 but for cannabis found alone it was 2.2. Among other drugs used alone, greater odds ratios were found for alcohol at a concentration of between 0.05 and 0.08 g/100ml (3.7), alcohol over 0.08 g/100ml (39.2), cocaine (4.9) and benzodiazepines (2.5). Very high risks were associated with combinations of drugs (including alcohol). The authors also emphasised that alcohol remained the most problematic drug, with blood alcohol concentrations in excess of 0.08 g/100ml being found in nearly 30 percent of fatally injured drivers (Dussault et al. 2002).

One possible problem with this study, which was acknowledged by the authors, is that the results may have been affected by selection bias. As noted by Walsh et al. (2004), refusal rates in roadside surveys can have a profound effect on the results. Illicit drugs are used by a small number of drivers and it is common in roadside surveys to get a refusal rate that exceeds the proportion of drivers testing positive for drugs. If drug use is over-represented in the drivers refusing to take part in the survey, there will be a bias towards less drug use being detected in the control group and, hence, greater odds ratios for drug use by crash-involved drivers (Bates and Blakely 1999; Keall and Frith 2004; Ogden and Moskowitz 2004; Walsh et al. 2004). In Dussault et al.'s (2002) study, 84.6% of potential controls agreed to providing a fluid sample (saliva or urine). Although this is a high proportion of motorists agreeing to participate, the proportion not participating is still large enough to markedly affect the results. Jonah et al. (2004) argue that if half of those refusing to participate in this study had been positive for cannabis then the true odds ratio for cannabis would not have been significantly different from 1.0. Dussault et al. (2002) argue that selection bias would not have been

great in their study, citing a high percentage of cannabis detected among young control drivers (24.3% of 16-19 year olds and 22.4% of 20-24 year olds).

An additional problem with Dussault et al.'s (2002) study, which was also acknowledged by the authors, is that cannabis was measured in urine and not blood. Thus, only inactive metabolites of cannabis would have been detected, rather than the active component, THC. This means that the study results for cannabis effectively represent a case-control study for cannabis users rather than for cannabis impairment when driving. That is, it measures the relative risk of crashing for drivers who use cannabis at all, rather than measuring the relative risk of crashing for drivers who are affected by cannabis when driving.

Another case-control study into drug use and crash involvement was conducted in France by Mura et al. (2003). In that study, blood samples were taken from 900 injured drivers presenting at hospitals (cases) and 900 patients presenting at the same hospitals for non-traumatic medical problems, excluding admission for intoxication (controls). Control group participants all held driver's licences and were matched to the case group according to age and gender. The use of blood enabled detection of THC, so that impairment could be inferred from cannabis-positive results.

It was found that THC was detected in 10 percent of cases and 5 percent of controls. Consistent with reports that cannabis use is more common in younger adults, the percentages for those aged between 18 and 27 were 14.1 percent for cases and 6.7 percent for controls, giving an odds ratio of 2.5. However, cannabis was not over-represented in cases in older age groups. This contrasts with alcohol, which was over-represented among cases for all age groups. Furthermore, odds ratios for crash involvement increased with increasing blood alcohol concentration but, for cannabis detected in participants under the age of 27, the odds ratio for THC concentrations above 1 ng/ml was 2.5 and that for THC concentrations less than 1 ng/ml was 2.7. The authors explained this by referring to the lack of relationship between THC concentrations in blood and impairment and, more specifically, to the fact that peak clinical effects of cannabis occur after blood levels have declined substantially from their peak. Among other findings, alcohol alone ( $> .05$  g/100ml) was found most commonly (17.0% of cases, 5.0% of controls, odds ratio = 3.8), benzodiazepines were found to be common (9.4% of cases and 5.8% of controls, odds ratio = 1.7), morphine was found to have an odds ratio of 8.2 (2.7% of cases, 0.3% of controls), and the

combination of alcohol and cannabis for drivers under the age of 27 was found to have an odds ratio of 4.6 (9.5% of cases, 2.2% of controls) (Mura et al. 2003).

There is one clear methodological flaw with the study by Mura et al. (2003). This is that the control group consisted of non-traumatic patients at hospital rather than non-crash-involved drivers. This violates the case-control study principle that controls should be representative of the population from which the cases arise (Jamrozik and English 1991). The use of hospital patients rather than non-crash-involved drivers is problematic for two reasons. The first of these is that the control group would ideally provide information about the number of drivers on the road who had used drugs prior to driving. The use of a group of patients in a hospital tells us nothing about the drug use of drivers and so does not provide the information required for calculations of the relative risks for crash involvement. The second problem is that individuals presenting at a hospital with non-traumatic medical complaints are likely to be a particular group of people who are not representative of the general population. How this relates to their likelihood of using drugs in the period prior to their appearance at a hospital is unclear. Mura et al.'s (2003) study provides very useful information about the prevalence of drugs in crash-involved French drivers but the fact that the controls were not driving at the time of their blood being taken makes it difficult to interpret the odds ratios derived from the study data.

Another European case-control study was conducted in Tilburg, Netherlands (Mathijssen and Houwing 2005; Movig et al. 2004). In this study, 184 drivers sustained injuries requiring hospitalisation (cases) and 3,374 non-crash-involved drivers recruited at roadside surveys (controls) provided a sample of either urine or blood. Cannabis use was determined on the basis of the presence of THC metabolites, which, as noted earlier, provide no indication of impairment, only previous use. It was found that cannabis by itself was detected in 3.4 percent of cases and 3.9 percent of controls. These percentages, after controlling for potential confounders, produced an odds ratio of 1.45, which was not statistically significantly different from zero. Significantly elevated odds ratios were found for benzodiazepines (2.98), alcohol at a concentration between 0.05 and 0.08 g/100ml (8.28), alcohol between 0.08 and 0.13 (17.6), alcohol over 0.13 (87.2) drug combinations (24), and drugs combined with alcohol (12.9 for alcohol less than 0.08 and 179 for alcohol greater than 0.08) (Mathijssen and Houwing 2005).

A methodological problem with the Dutch study is related to the choice of matrices for drug analysis. The researchers opted to collect either blood or urine, so

that the inclusion of participants was not limited by the choice of only one biological fluid. The problem with this method was that 34 percent of cases provided urine samples and 66 percent provided blood, compared to 85 percent urine and 15 percent blood for the controls. This meant that comparisons between cases and controls were not using the same measurement techniques. It is a principle of case-control studies that any errors in measurement of exposure be non-differential between cases and controls. Failure to achieve this can cause 'information bias' (Wacholder et al. 1992). In the case of cannabis, its metabolites last longer in urine than in blood and the greater use of urine for the control group would lead to a greater chance of cannabis being detected for the controls than for the cases. This, in turn, would be likely to lead to under-estimation of the relative crash risk associated with driving after cannabis use. Movig et al. (2004) deny the likelihood of information bias by referring to the greater detection of cannabis in blood specimens than in urine specimens (the opposite of what would be expected if cannabis was too readily detected in urine compared to blood). However, in a study in which participants decided whether they would provide blood or urine, these results may reflect qualitative differences in the types of control drivers who opted to provide blood compared to the drivers who opted to provide urine.

A case-control study was conducted in Victoria, Australia by Haworth et al. (1997). This study was concerned with single vehicle crashes within 200 km of Melbourne, Victoria. Driver and vehicle characteristics were recorded for 127 cases and 865 controls, with cannabis measured in urine for cases and by self-report for controls. Cannabis alone was found in the urine of four percent of cases and in combination with alcohol (assessed with breath tests) in 18 percent of cases. Among controls, cannabis was only reported by one percent of drivers. The odds ratio for crash involvement associated with cannabis, after adjustment for age and blood alcohol concentration, was 38. This very high odds ratio, however, is likely to have been inflated by the method used in the study to assess cannabis use. Measurement of cannabis in urine for cases would have resulted in drivers who had consumed cannabis any time in the few days prior to the crash being counted as cannabis cases. For controls, there is a strong possibility that there was under-reporting of cannabis use. Therefore, the likelihood of cannabis use being overestimated among cases and underestimated among controls would have produced an inflated odds ratio.

In summary, there have been a small number of recent case-control studies conducted to determine the relative risks for crash involvement associated with driving

after use of drugs including cannabis. Dussault et al. (2002) found that cannabis use produced an approximately twofold risk of crashing, Mura et al. (2003) found a two to threefold crash risk related to cannabis only for those aged under 27 and Mathijssen and Houwing (2005) failed to detect an increased risk associated with cannabis. In all studies, alcohol and combinations of drugs far exceeded cannabis in their effects on crash risk. However, in all three studies, methodological problems mean that great caution must be exercised in interpreting the results. The studies by Dussault et al. (2002) and Mathijssen and Houwing (2005) were both affected by the use of urine for drug testing, meaning that only the inactive metabolites of THC could be detected. Mura et al.'s (2003) study did not use a sample of drivers for a control group. The most recent Australian case-control study (Haworth et al. 1997) was concerned only with single vehicle crashes and measured drug use in a way that would have greatly inflated the odds ratio for crash involvement associated with cannabis.

These methodological problems emphasise the difficulties of conducting case-control studies designed to investigate drug driving. Compromises are often necessary for studies to proceed. Other case-control studies being conducted in Norway (Assum 2004) and the United Kingdom (Buttress et al. 2004) have run into considerable operational difficulties. These problems associated with case-control studies have led some researchers to investigate the risk of crash involvement associated with drugs by using a methodology that does not rely on collecting control data in addition to case data. These studies, based on assessments of the relationship between drug use and crash responsibility (or 'culpability'), are discussed in the following section.

## **Crash culpability studies**

Studies of this sort involve classifying crash-involved drivers according to their degree of responsibility (or 'culpability') for the crash. The drug use of drivers culpable for their crashes is then compared with the drug use of drivers judged not to be culpable. If greater use of a drug is evident among drivers culpable for their crashes, then that drug is linked to a greater crash risk. Culpability studies treat crash-involved drivers who are not culpable for their crashes as a control group, based on the assumption that a driver's likelihood of being involved in a crash as a non-culpable party is determined by the amount of driving they do. That is, involvement in crashes for which one is not culpable is treated as a measure of driving exposure (Bates and Blakely 1999).

Judgement of culpability is usually based on a set of pre-determined criteria that allow for the effects of mitigating factors (e.g. other drivers' actions, bad weather) to be taken into account. This must be done by assessors blind to the drug use status of the drivers (Robertson and Drummer 1994).

Early culpability studies tended to find that cannabis was not associated with an increased crash risk (Drummer 1994, 1995; Terhune 1982; Terhune et al. 1992; Williams et al. 1985). Terhune et al. (1992) conducted a culpability analysis on 1,882 fatal crashes in the United States. Alcohol was present in 51.5 percent of drivers and cannabis was found in 6.7 percent. Two thirds of the cannabis positive drivers were also positive for alcohol. Drivers positive for cannabis only were not found to have an increased likelihood of culpability for the crash (the non-significant trend was actually in the opposite direction). Increased levels of crash culpability were found, however, for alcohol and for alcohol combined with other drugs. The combination of cannabis and alcohol was related to a greater likelihood of crash culpability but no greater than for alcohol alone. It was concluded that the relationship between crash culpability and the combination of cannabis and alcohol was due to the dose-dependent effects of alcohol. Drummer (1994) also looked at fatal crashes, but in Australia, and found that cannabis (found in 11 percent of drivers) was not linked to a greater likelihood of crash culpability. Again, the non-significant trend for cannabis was in the opposite direction, whereas alcohol (36 percent of drivers) and alcohol combined with cannabis were associated with greater crash culpability. Adjusting the odds ratios for age and gender did little to change the results (Drummer 1995). However, all of these studies only measured metabolites of cannabis rather than THC and so, in many cases, the drivers included in the cannabis positive group would not have been impaired at the time of the crash.

More recent culpability studies that have considered the role of cannabis in crashes have been conducted in Australia by Longo et al. (2000) and by Drummer et al. (2004). Studies overseas into cannabis and crash culpability have been conducted in Canada (Dussault et al. 2002), the United States (Bedard et al. 2007; Lowenstein and Koziol-McClain 2001; Soderstrom et al. 2005) and France (Laumon et al. 2005).

Longo et al. (2000) investigated the relationship between drugs and crash culpability in a sample of 2,279 non-fatally injured car drivers and motorcycle riders who were treated at hospital in Adelaide, South Australia. This study used the culpability method devised by Robertson and Drummer (1994), which adjusts

culpability levels according to eight mitigating factors. Compared to the drug-free group, greater culpability was found for those drivers testing positive for alcohol (11% of drivers, OR = 8.0), benzodiazepines (2.0%, OR = 2.0), alcohol combined with cannabis (0.6%, OR = 5.4) and alcohol combined with benzodiazepines (0.7%, OR = 13.4). THC alone (1.9%) was not found to be associated with greater culpability. Instead, similar to the earlier studies noted above, there was a non-significant trend toward lower culpability for THC positive drivers (OR = 0.8). The relationship with culpability for the combination of alcohol and cannabis was no greater than that for alcohol alone, again suggesting that alcohol is the factor increasing crash risk when people drive affected by both alcohol and cannabis. Longo et al. (2000) were able to dismiss any concerns about the small sample size being responsible for not finding a relationship between THC and crash culpability. The failure to find greater crash culpability for the 44 drivers testing positive for THC only was contrasted with the finding of a greater likelihood of culpability for the 46 drivers testing positive for benzodiazepines only. The results also remained the same after adjusting for potentially confounding factors, such as age and gender (Longo 2001).

Another Australian study was conducted by Drummer et al. (2004), using blood analyses of drivers fatally injured in road crashes in Victoria, New South Wales and Western Australia over ten years. Drummer et al. analysed 3,398 blood samples but only 1,420 were analysed for THC, beginning when the necessary technology became available. The drivers with the highest odds ratios for crash culpability (with potentially confounding factors controlled) were those with a blood alcohol concentration above 0.05 g/100ml (29.1% of drivers, OR = 34.1). With regard to cannabis, drivers testing positive for THC alone (3.9%) were found to have an elevated likelihood of being culpable for their crashes (OR = 2.7). The combination of alcohol and THC (2.9%) was also found to have a greater odds ratio for crash culpability than alcohol alone (relative OR = 2.9), which the authors interpreted as indicating that cannabis increases the impairment associated with alcohol. Metabolites of THC were not found to be linked to crash culpability. Drummer et al. (2004) also analysed results according to drug concentration and found an elevated odds ratio for culpability among drivers with blood THC concentrations above 5 ng/ml (OR = 6.6). Furthermore, they argued that the relationship between THC and crash culpability 'showed a biological gradient, similar to that observed for alcohol' (Drummer et al. 2004, p. 245). This last conclusion was based on a comparison of the culpability of 49 drivers with THC blood concentrations above 5 ng/ml and nine drivers with concentrations below that level.

These two studies by Longo et al. (2000) and Drummer et al. (2004) produced very different findings. Drummer et al. argue that the different results are due to the lower THC concentrations detected in the Longo et al. study. According to Drummer et al., the main risk from THC comes when it is consumed in sufficient quantities to produce a blood concentration above 5 ng/ml. Few drivers in the Longo et al. study recorded THC concentrations at this level. In interpreting their results, Longo et al. did note that the majority of THC concentrations found in the blood samples collected were in the very low range relative to the levels that can be reached by cannabis users. Caution was therefore advised in accepting the lack of a relationship between cannabis and crash culpability (Longo et al. 2000).

However, there are three reasons why some degree of confidence can still be placed in the findings. First, as previously noted, there is little relationship between THC concentrations in the blood and impairment, so those drivers with low THC readings may have been as impaired as those with higher readings. Second, the long time in many cases between the crash and the taking of blood ( $M = 2.7$  hours,  $SD = 3.0$ ) would have resulted in lower THC concentrations than would have been the case at the time of the crash. Some of the drivers only testing positive for the metabolite may have tested positive for THC at a low concentration if their blood sample had been taken earlier. Therefore, although the study may underestimate the prevalence of THC in crash-involved drivers, it would be more likely to detect a relationship, if one exists, between THC, even at low concentrations, and crash culpability. Third, the results are consistent with previous findings of no increased likelihood of culpability with cannabis use. Although previous studies had chiefly assessed metabolites of cannabis, it is likely that a proportion of drivers positive for cannabinoid metabolites would have been impaired by THC at the time of the crash.

This latter point may also be relevant for raising questions about the findings of Drummer et al.'s (2004) study. Specifically, there is an apparent inconsistency between the earlier study that found no relationship with crash culpability for metabolites of cannabis (Drummer 1994, 1995) and later studies that found the opposite for THC (Drummer 1999; Drummer et al. 2004). As noted in a report by Austroads (2000), it would be expected that some of the drivers in the earlier data set who tested positive for metabolites would also have tested positive for THC if such an analysis had been conducted. If THC is associated with an increased likelihood of crash culpability, as found in the more recent study, then it would be expected that there would have been a

(smaller) relationship between testing positive for cannabis metabolites and greater crash culpability in the earlier study. That this was not the case and instead, that the trend, which was approaching significance, was in the opposite direction, suggests that the cannabis problem, if one exists, must not be a large one (Austroads 2000).

Bates and Blakely (1999) argue that findings such as those from Drummer's early study suggest that THC may reduce the likelihood of crash culpability. In Drummer's early study, drivers only positive for metabolites of THC were classified as cannabis positive when they should have been classified as drug free. As the drug free group was given the culpability odds ratio of 1.0, the wrongful inclusion of any drug free drivers in a drug group would move the odds ratio for the group closer to 1.0. That is, there would be a reduction in the odds ratios for drugs that increase the likelihood of culpability and an increase in the odds ratios for any drugs that reduce the likelihood of culpability. As the odds ratio for culpability for the cannabis group was less than 1.0, and the inclusion of metabolite positive only drivers would have moved the odds ratio closer to 1.0, it is possible that the odds ratio for THC positive drivers was less than that found in the study for the cannabis group. It may have been significantly less than 1.0. That is, it may be that the drivers impaired by THC were significantly less likely to be culpable for their crashes (Bates and Blakely 1999).

In any case, there does appear to be some degree of inconsistency in the two Drummer studies, with the later one finding increased culpability for THC and the earlier one finding no sign of increased culpability for cannabis users (those positive for metabolites, some of whom would likely have been positive for THC). Alternative explanations for this combination of apparently inconsistent results are that the risks associated with THC have changed in a few years, that the proportion of cannabis users choosing to drive when positive for THC (i.e. when actually impaired by cannabis) has increased sharply, or that the incidence of drivers testing positive for THC and who were culpable for the crash in either one of the data sets was 'a statistical aberration' (Austroads 2000, p. 17).

A number of other studies have included analyses of cannabis use and crash culpability but are unable to provide a solution to the contradictory results of the two Australian studies because they have only tested for metabolites of THC in urine.

In the study by Dussault et al. (2002), urine analyses of 354 fatally injured drivers were used to determine the relationship between drug use and crash culpability. The

culpability analysis was done in addition to a case-control analysis (described earlier) so that the case-control results could be checked. The case-control results for cannabis indicated an increased crash risk associated with cannabis use but inconsistent results were found in the culpability analyses, with no evidence of an increased likelihood of crash culpability for drivers testing positive for cannabis metabolites. Although the culpability analyses did confirm the increased case-control crash risks of alcohol and cocaine, the authors attributed the lack of an association between cannabis use and crash culpability to the limitations of culpability analyses and a lack of statistical power.

The study by Lowenstein and Koziol-McLain (2001) investigated the culpability and drug use of 414 non-fatally injured drivers. Cannabis (17%) was detected more frequently than alcohol (14%) in the urine samples of the drivers. However, only alcohol was found to be associated with a greater likelihood of crash culpability. The authors interpreted the findings as indicating that cannabis is not a road safety risk and argued that this may be explained by compensation for impairment. They did, however, note that the sample was primarily of middle-aged drivers with minor or moderate injuries and that a different sample, in terms of age or injury level, may have produced different results.

Soderstrom et al. (2005) analysed urine samples of 2,537 drivers admitted to a trauma centre in Maryland, USA. Culpability was based on the determinations of the investigating police officers. Alcohol, which was easily the most commonly detected drug, and cocaine were both found to be associated with a greater likelihood of culpability (OR = 7.45 and 2.33 respectively) but cannabis was not (OR = 1.18). The authors claimed that the finding added to the literature suggesting the lack of an association between cannabis and crash culpability but, the use of metabolites in urine as the indicator for cannabis, again weakens the argument.

Unlike the three studies above, Laumon et al. (2005) analysed blood samples for the presence of drugs and assessed crash culpability for 10,748 fatally injured drivers in France. They found that THC in the blood was associated with an increased culpability (OR = 3.32) relative to drivers without any drugs or alcohol in their system. The authors also claimed that the odds ratio was higher for drivers with more than 5 ng/ml of THC in their blood than for those with less than 1 ng/ml of THC. However, inspection of the confidence intervals on the culpability ratio estimates reveals that the difference was not statistically significant. The highest culpability odds ratio was for alcohol (15.5), while significantly elevated ratios were also found for amphetamines (3.75) and cocaine (4.44)

but not opiates (0.92). When the authors took the prevalence into account for both cannabis and alcohol, they concluded that 2.5 percent of fatal crashes could be attributed to cannabis, compared to 28.6 percent for alcohol (Laumon et al. 2005).

The most recent of all culpability studies was conducted by Bedard et al. (2007). This study used ten years of Fatal Accident Reporting System (FARS) data from the USA, with the listing of driver-related factors being used as an indicator of culpability. Approximately ten percent of drivers aged 20 to 49 were tested for cannabis, using either blood or urine. Five percent of those drivers were positive for cannabis but not alcohol. Of these, 67.2 percent were culpable for the crash, compared with 59.6 percent for those who were cannabis and alcohol negative. After controlling for confounders, this gave a culpability odds ratio of 1.29. Drivers testing positive for alcohol were far more common and were far more likely to be culpable (Bedard et al. 2007). Problems with this study include the categorisation of drivers as cannabis positive if they tested positive to inactive metabolites of THC and the fact that only ten percent of drivers were tested for cannabis, suggesting that there may have been a bias regarding which drivers were selected for drug testing and were thus eligible for the study.

To summarise the findings of recent culpability studies, Drummer et al. (2004) and Laumon et al. (2005) found an increased likelihood of crash culpability associated with being positive for THC. Another Australian study (Longo et al. 2000) that analysed blood samples, but in hospital treated rather than fatally injured drivers, found no greater likelihood of crash culpability for drivers testing positive for THC. Earlier studies and recent ones measuring metabolites of cannabis found no increased culpability for drivers who used cannabis, although a small increased rate of culpability was found by Bedard et al. (2007). Drummer et al.'s (2004) findings are surprising, given that earlier reports by the same authors, which grouped together drivers testing positive for THC and those testing positive for metabolites only, found no increase in culpability rates for cannabis-using drivers. Nonetheless, the divergent findings of the two recent Australian studies mean that the issue of cannabis and crash culpability remains unresolved.

It is also important to note that crash culpability studies are characterised by a number of limitations. The most obvious limitation is that the non-culpable driver may still have contributed to the causation of the crash (Austroads 2000; Keall and Frith 2004; Lowenstein and Koziol-McLain 2001; Vingilis and Macdonald 2002). For example, a driver not making a mistake may still contribute to an intersection collision by having

not braked quickly enough. If a drug causes a lengthening of reaction time (i.e. slower reactions), it may increase the likelihood of a driver being involved in a crash as the non-culpable party. This would make it less likely that a culpability analysis would find an association between use of this drug and crash culpability. Alternatively, if a drug is associated with a slower, more conservative driving style, it may be that use of that drug will decrease the likelihood of a non-culpable driver striking a vehicle driven by someone making a mistake (such as an unsafe turn across oncoming traffic). In this scenario, it would be more likely that use of the drug would be associated with greater crash culpability. As cannabis has been associated with both longer reaction times and a slower, more conservative driving style, it is unclear whether culpability analyses are more or less likely to identify cannabis use as a contributor to crash involvement. Evidence supporting the possibility that non-culpable crash-involved drivers are not representative of the driving population (contrary to what is assumed in all culpability analyses) comes from studies finding elevated blood alcohol concentrations among this group of drivers (e.g. Neilsen 1965).

Another possible problem with culpability analyses is that there is a subjective element involved in the attribution of culpability (Movig et al. 2004). Studies in which the assessment of culpability relies to some extent on the judgement of police may be biased by the greater likelihood of police attributing culpability to an impaired driver. This would result in drugs being more likely to be associated with crash culpability (Keall and Frith 2004). The likelihood of misclassification of culpability can be reduced by assessing culpability on a gradient and eliminating 'contributory' drivers (Bates and Blakely 1999), as was done by both of the recent Australian studies, and by relying on multiple sources of information for the determination of culpability.

Another problem with culpability studies is that the lack of a non-crash-involved control group means a reduction in the sample of crash-involved drivers that can be treated as 'cases'. This, in turn, reduces the statistical power available to assess drug effects (Movig et al. 2004). Samples for analysis can also be reduced by the tendency for drugs to be found in combination in drivers' samples. Those who drive after drug use often do so after consuming multiple drugs or drugs in combination with alcohol, meaning that the samples for drugs used in isolation are often small (Austroads 2000; Bates and Blakely 1999). A further difficulty associated with culpability studies using fatally injured drivers is that there is a high baseline for culpability even among the drug free group, with single vehicle crashes being over-represented in fatal crash data.

Longo et al. (2000) note that, in their study of injured drivers, 53 percent of drug free drivers were culpable for their crashes, compared to 68 percent of the fatally injured drivers in the study by Terhune et al. (1992) and 70 percent in Drummer's (1994) study. This also makes it difficult to demonstrate an increased likelihood of crash culpability with drug use (Dussault et al. 2002; Longo et al. 2000).

## Conclusions and Discussion

The best way of determining whether a drug is associated with an increased risk of crash involvement is to conduct a case-control study in which the drug levels detected in crash-involved drivers are compared with the levels detected in a matched sample of non-crash-involved drivers, such has been done for alcohol (Borkenstein et al. 1974; McLean and Holubowycz 1980). Although attempts have been made to conduct such studies, they have been beset by methodological flaws, potentially resulting in 'information bias' and 'selection bias'. Ideally, the drug use of cases and controls would be compared using the same biological matrix and potential control group drivers would not be given the option of not participating.

Partly as a response to the difficulty of conducting case-control studies, some researchers have used culpability studies to determine whether cannabis use contributes to crash involvement. The majority have indicated that cannabis is not associated with an increased likelihood of culpability. However, similar to case-control studies into cannabis and crash involvement, many culpability studies are difficult to interpret because of methodological problems. There have been two recent Australian studies (Drummer et al. 2004; Longo et al. 2000) that have analysed the relationship between THC measured in the blood and crash culpability. These two studies produced contradictory results. The findings of a French study analysing THC in blood agreed with those of Drummer et al. (Laumon et al. 2005).

In a review of international evidence as part of the European IMMORTAL (Impaired Motorists, Methods of Roadside Testing and Assessment for Licensing) program, Klemenjak et al. (2005) concluded that cannabis was a drug of 'moderate' crash risk, with alcohol and benzodiazepines the only drugs that, when taken alone, could be classified as constituting a high risk and requiring high priority action. The authors claimed (page iv) that for 'illegal drugs taken alone... zero tolerance legislation would... seem to be an overreaction resulting in very high cost and fewer road safety

benefits.' In a number of Australian jurisdictions, programs of roadside drug testing have already commenced. It will be of considerable interest to assess the impact of this testing on drug-related road crashes. Random breath testing (RBT) appears to have been associated with decreases in drink driving and alcohol-related crashes (Peek-Asa 1999) but it is claimed that there is still a 'hard-core' of drink drivers who are difficult to deter from this behaviour (Simpson et al. 2004). If drug drivers are similar to these 'hard-core' drink drivers, which is possible given that drug use is not as normative as alcohol consumption (Australian Institute of Health and Welfare 2007), then roadside drug testing may not be as effective as RBT. It is also very important to note that alcohol is still found to be the most prevalent and dangerous drug in studies of drugs and road crashes. Roadside drug testing could prove counter-productive if it reduces the resources available for RBT.

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