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Review of the literature on cannabis and crash risk

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TITLE

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ABSTRACT

A review of the literature published prior to 2005 concerning cannabis and road crash involvement was conducted, with emphasis given to studies documenting the relative crash risk associated with driving after use of cannabis. Case-control studies that have been conducted into cannabis and road crashes have been characterised by methodological flaws that make the interpretation of the results difficult. 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. However, as for 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 that have analysed the relationship between THC (tetrahydrocannabinol - 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. To resolve the issue, it is necessary to conduct a case-control study similar to those that have been conducted for alcohol. That is, it is necessary to compare the incidence of cannabis in crash-involved drivers with the incidence in non-crash-involved drivers matched for potential confounding factors, such as age, gender, time of day, day of week, and direction of travel.

KEYWORDS

Marijuana, Drug effects, Driver performance, Literature review

Summary

Cannabis is a mostly recreational drug that is known to produce dose-related decrements in performance on a number of laboratory tasks associated with skills that are necessary for driving. Studies of the effects of cannabis on driving performance (measured with on-road driving tests and driving simulators) have revealed that it negatively affects a number of aspects of the driving task but to a lesser degree than it affects performance on laboratory tasks. Although cannabis is found commonly in the blood of crash-involved drivers, second in frequency only to alcohol, this is likely to be due to the fact that it is the second most commonly used drug behind alcohol, and so it is necessary to conduct studies in which the crash risk associated with driving under the influence of cannabis can be determined.

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. However, those studies that have been conducted are characterised by methodological flaws that make the interpretation of the results difficult.

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. These studies treat crash-involved drivers who were *not* culpable for their crashes as a control group against which to compare the drug use of crash-involved drivers who *were* culpable for their crashes. The majority have indicated that cannabis is not associated with an increased likelihood of culpability. However, as for 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 that have analysed the relationship between THC (tetrahydrocannabinol - 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. To resolve the issue, it is necessary to conduct a case-control study similar to those that have been conducted for alcohol. That is, it is necessary to compare the incidence of cannabis in crash-involved drivers with the incidence in non-crash-involved drivers matched for potential confounding factors, such as age, gender, time of day, day of week, direction of travel et cetera. 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. The latter methodological requirement would need the introduction of a system of mandatory roadside drug testing.

Finally, it is important to emphasise that alcohol is still the most important drug in terms of its contribution to crash involvement worldwide. Alcohol is found more frequently than cannabis and other drugs in the blood of crash-involved drivers and analytical studies have found that the crash risk associated with alcohol far exceeds that associated with drugs. Furthermore, drug-impaired drivers are frequently also impaired by alcohol, which makes the risks associated with drugs difficult to isolate from the well-known adverse effects of alcohol. Nonetheless, research is still needed, particularly a case-control study, to determine how great a problem cannabis is and whether steps need to be taken to apprehend those who combine cannabis use and driving.

Contents

1 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, 2003; Johnston, O'Malley, Bachman, & Schulenberg, 2004; Miller & Draper, 2001) and to cannabis being the most frequently detected psychoactive substance, after alcohol, among driving populations (Bates & Blakely, 1999; Couper & Logan, 2004; Kelly, Darke, & Ross, 2004; Walsh, de Gier, Christopherson, & Verstraete, 2004).

Of particular importance is research into the relationship between the recent use of cannabis and involvement in road crashes. 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, Crowther, Schumate, Ziel, & Zylman, 1974; McLean & Holubowycz, 1980), then policies and methods of enforcement to reduce driving after cannabis use would be justified. In Victoria, random roadside testing of the oral fluid (saliva) of drivers, to identify those who have recently used cannabis, is already being practised. 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.

The present review, which outlines the findings of research published prior to 2005, begins with a brief description of cannabis and its effects on aspects of functioning thought to be relevant for driving. Following this, there is a review of the literature concerning studies into cannabis and crash involvement. There are three types of such studies. First, there are studies documenting the incidence of cannabis use among crash-involved drivers. Secondly, there are case-control studies that can be used to estimate crash risks associated with driving under the influence of cannabis, and, thirdly, there are studies into the relationship between cannabis use and responsibility (or 'culpability') for crashes. The sum of the evidence provided in these different types of studies into cannabis use and crash involvement will form the basis for the conclusions that can be drawn at this time regarding whether cannabis constitutes a major road safety problem.

2 Cannabis and its effects

Cannabis comes in a variety of forms (marijuana, hashish, hash oil) that are derived from the plant *cannabis sativa*. It is usually smoked but can also be ingested in food (Bates & Blakely, 1999). The chief psychoactive compound found in cannabis is Δ^9 -tetrahydrocannabinol (THC). In most forms of cannabis, the content of THC is between 2 and 8 percent by mass but can reach as high as 20 percent (Drummer, 2004). There is also evidence that the level of THC in marijuana is increasing (ElSohly et al., 2000), with greater use of cultivation techniques designed to produce highly potent cannabis. THC produces a range of effects (stimulating, sedating, hallucinogenic) that make classification of the drug difficult. Although cannabis is used mostly as a recreational drug, the cannabis-derived compound dronabinol is used as an appetite stimulant and antiemetic, preventing weight loss in AIDS patients and treating the nausea and vomiting associated with cancer chemotherapy (Couper & Logan, 2004; Grotenhermen, 2003).

Cannabis has been found to be addicting. It is associated with compulsive craving, drug seeking and continued use even when the user is experiencing negative social and health consequences. A degree of physical dependence can occur with cannabis and abrupt discontinuation of use can cause mild withdrawal symptoms (Couper & Logan, 2004). Such dependency occurs in 7 to 10 percent of users (Kalant, 2004), which represents a lower dependency risk than exists for opioids, tobacco, alcohol and benzodiazepines (Grotenhermen, 2003). Long term negative effects include chronic inflammatory and precancerous changes in airways, possible precipitation of schizophrenia, a possible link with depression, and possible functional problems in the offspring of women who smoke cannabis during pregnancy (Kalant, 2004).

The effects of cannabis peak within 30 minutes of smoking, while the overall 'high' tends to last for two hours. Most effects have dissipated after 3 to 5 hours. Effects following a dose of dronabinol begin 30 to 60 minutes after ingestion and peak between 2 and 4 hours, with appetite being stimulated for up to 24 hours (Couper & Logan, 2004).

Experimental (laboratory-based) studies into the effects of cannabis on skills considered to be necessary for driving have found that it leads to dose-related decrements in tracking, reaction time, memory and learning, divided attention, sustained attention (vigilance), perception, thinking and problem solving, and co-ordination (Bates & Blakely, 1999; Berghaus & Guo, 1995; Couper & Logan, 2004; Department of Environment Transport and the Regions, 2000; Kelly et al., 2004; Ramaekers, Berghaus, van Laar, & Drummer, 2004). However, those affected by cannabis are able to successfully compensate for their impairment when asked to concentrate on simple tasks for brief periods of time (Couper & Logan, 2004).

Experimental studies do suffer from a potential lack of external validity, however. Real world drug use may be very different from that practised in the laboratory, and studies not based on the performance of actual drug users may over-estimate the effects of the drugs investigated (Vingilis & Macdonald, 2002). It is also not possible to equate decrements in performance in laboratory tasks with an increased crash risk (Mathijssen et al., 2002; Mura et al., 2003).

A number of studies have also 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, with decrements found in handling performance, reaction time, time and distance estimation, maintenance of lateral position, co-ordination, and vigilance (Couper & Logan, 2004). Ramaekers et al. (2004) noted that effects are dose-related and that highly automated behaviours, such as road tracking control, were more affected by cannabis than more complex driving tasks that require conscious control.

However, the level of impairment observed in driving studies has been found to be less severe than the impairment of individual cognitive or psychomotor skills observed in laboratory-based studies (Kelly et al., 2004), and a review of driving studies by Smiley (1999) found that drivers affected by cannabis tended to compensate for their impairment. Cannabis-affected drivers have a tendency to drive in a less aggressive manner, to be more cautious, and to be more aware of their impairment than drivers affected by alcohol. Smiley did also note, however, that the reduced ability to react to unexpected emergencies was likely to be a problem for cannabis-affected drivers. It has also been reported that compensation for impairment by drivers affected by cannabis tends to be insufficient, particularly when driving demands are high or during prolonged driving (Bates & Blakely, 1999; Couper & Logan, 2004; Ramaekers et al., 2004). Robbe (1994) also found that compensatory behaviour is less evident following larger doses of cannabis. Furthermore, Ogden and Moskowitz (2004) noted that the compensatory behaviour of cannabis-affected drivers may also be due to distortion of judgement of time and distance, as much as it is a response to conscious recognition of impairment.

A consistent finding has been that impairment of driving is far greater when cannabis is combined with alcohol (Austroads, 2000; Couper & Logan, 2004; Krueger & Vollrath, 2000; Ramaekers, Lamers, Robbe, & O'Hanlon, 2000). One factor contributing to this may be that alcohol is associated with a less cautious driving style and so the compensation often applied by drivers affected by cannabis alone is no longer applied when cannabis is combined with alcohol (Department of Environment Transport and the Regions, 2000).

As with laboratory studies, there are doubts regarding the validity of driving performance studies. Safe driving is dependent not only on a driver's *ability* to perform but also on that driver's *behaviour*. The former refers to what drivers are capable of doing, whereas the latter refers to what they actually do. Behaviour, in this sense, includes thrill seeking, aggression and acceptance of risk. Although, as noted previously, differences in behaviour related to cannabis use have been found in driving studies, such direct studies of performance are not capable of providing *valid* measures of behaviour. Driving simulators are limited because there is no element of actual risk in a simulated environment. Studies conducted on public roads are better than simulator studies but drivers are still aware of being observed and assessed, and so may behave differently than would be the case in a normal driving situation (Austroads, 2000).

To summarise, cannabis is a popular, largely recreational, drug that reliably produces decrements in performance on a number of laboratory tasks. Cannabis also negatively affects driving performance but is often associated with compensation for impairment in the form of more cautious driving behaviour. Although these laboratory, driving simulator and on-road driving performance studies are informative, they do not provide any indication of the extent of driving under the influence of cannabis, or of the relationship between cannabis use and crash risk. Research that has been conducted to investigate the crash involvement risk associated with driving after using cannabis is described in Section 3.

3 Studies into cannabis and crash involvement

This section describes research undertaken to determine whether driving under the influence of cannabis increases the risk of being involved in a crash. It describes studies measuring the prevalence of recent cannabis use in crash-involved drivers, followed by case-control studies directly assessing the risk of crashing associated with cannabis use, and, finally, studies of the relationship between cannabis use and the likelihood of being responsible for crashes. First, however, it is necessary to provide background information on the methods of testing drivers for cannabis that have been used in studies of cannabis and crash involvement.

3.1 Testing drivers for recent cannabis use

In order to determine whether a driver has used cannabis prior to driving, it is necessary to detect and measure THC in the driver's system. There are a number of alternative biological matrices available for such 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 metabolites in the different matrices.

Peak plasma concentrations of THC in blood typically exceed 50 ng/ml (25 ng/ml in whole blood) 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). A few hours after consumption, blood concentrations are usually below 2 ng/ml (Drummer, 2004). Within 8 to 12 hours, plasma levels fall below the limits of quantitation in occasional users (Couper & Logan, 2004).

Unlike alcohol, there is no clear relationship between blood concentrations of THC and impairment (Couper & Logan, 2004; Grotenhermen, 2003; Kalant, 2004). Illustrating this, Berghaus, Grass and Sticht (2000) conducted a meta-analysis of experimental drug studies and demonstrated that for drugs with short resorption times, such as cannabis, the time of maximum concentration in the blood precedes the time of maximum impairment of abilities necessary for driving. For a 20 mg dose of cannabis, the time to maximum blood concentration is 0.3 hours, whereas the time to the highest decrement in task performance is 0.6 hours (Berghaus et al., 2000). Papafotiou, Carter and Stough (2005) tested driving ability on a simulator 30 minutes and 80 minutes after participants smoked cannabis. It was found that impairment from cannabis was greater 80 minutes after smoking, when THC levels in blood were 5 to 10 percent of their peak. In addition to making it difficult to link THC blood concentrations with the likelihood of impairment, the rapid decline of these concentrations means that in investigations of crash-involved drivers, blood testing of the drivers two hours after the crash will produce underestimates of THC blood levels at the time of the crash (Swann, 2000). Metabolites of THC last much longer in the blood than THC itself and offer no indication of impairment, only previous exposure (Bates & Blakely, 1999; Couper & Logan, 2004; Grotenhermen, 2003; Ogden & 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 4 hours for metabolites to appear in urine in concentrations sufficient to be detected by an immunoassay. Positive results indicate use within the previous 1 to 3 days, although this period is longer for heavy chronic users (Couper & Logan, 2004; Drummer, 2004). The concentration of drugs within a urine sample can also be influenced by the degree of water intake by the person providing the sample (Caplan, 2004).

The most accessible matrix for detection of drugs is oral fluid (saliva), which is comprised of secretions from the submaxillary, parotid and sublingual glands (Walsh, Flegal, Crouch, & Cangianelli, 2004). 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 & Cone, 2004). There can also be difficulties collecting sufficient amounts of oral fluid for testing, as only very small concentrations of cannabis are found in oral fluid (Kintz, Cirimele, & Ludes, 2000; Walsh, de Gier et al., 2004) and cannabis can cause decreased oral fluid production (Verstraete, 2004).

It is important also to recognise that passive smoking of cannabis can lead to positive tests for all three matrices. Exposure to cannabis being smoked by others has been found to result in positive tests in blood (Cone & Johnson, 1986), urine (Cone et al., 1987) and oral fluid (Niedbala et al., 2004). The positive tests are only found shortly after exposure, however. Niedbala et al., (2004), for example, tested the oral fluid of study participants sitting in an unventilated room in the presence of people smoking cannabis. THC positive tests were found within 30 minutes of exposure to the cannabis smoke. Those who were actively smoking cannabis tested positive at higher concentrations of THC, and for the whole four hour testing session. Therefore, if recent passive exposure can be ruled out, it is likely that THC positive oral fluid tests indicate recent use (Huestis & Cone, 2004).

3.2 Prevalence of cannabis in crash-involved drivers

Epidemiological studies into drugs and road crashes can be divided into two types. The first type is descriptive epidemiology, which involves determining the extent of drug involvement in road crashes. Such studies can provide information about which drugs are the most commonly involved in crashes and repeated evaluations provide information about changes in the patterns of drug use and driving. The second type is analytical epidemiology, which involves determining which drugs are over-represented in road crashes. This is accomplished by the use of control groups and the calculation of relative crash risks according to drug use (de Gier, 2004; Vingilis & Macdonald, 2002). This section is concerned with studies of the first type. Specifically, it is concerned with studies in which researchers have analysed body fluids of crash-involved drivers to determine the presence or absence of indicators of recent cannabis use. Therefore, it excludes studies that have not focussed specifically on drivers (Waller et al., 1997), or that have been based on self-reports of either crash involvement or drug use (Albery, Strang, Gossop, & Griffiths, 2000; Chipman, Macdonald, & Mann, 2003; Darke, Kelly, & Ross, 2004; Fergusson & Horwood, 2001; Gerberich Goodwin et al., 2003; O'Malley & Johnston, 2003).

In most epidemiological studies of drug use and crash involvement, cannabis is the most frequently detected drug, excluding alcohol, and is found with the greatest frequency among young males (Bouchard & Brault, 2004; Drummer et al., 2003; Gerostamoulous et al., 2002; Logan & Schwilke, 2004; Longo, Hunter, Lokan, White, & White, 2000a; Mura et al., 2003; Terhune et al., 1992; Tunbridge, Keigan, & James, 2001). The finding that cannabis is detected more often than other drugs in crash-involved drivers is likely to be a reflection of cannabis being used more commonly than other drugs (Kelly et al., 2004).

However, the matrices used for testing, and the bio-markers used as indications of cannabis, need to be taken into consideration when interpreting the results (Department of Environment Transport and the Regions, 2000; Walsh, de Gier et al., 2004). Higher percentages of cannabis are found when urine is used as the matrix for testing because of the longer detection window for cannabis that is provided by urine. These high percentages do not imply that the driver was impaired by cannabis at the time of the crash, however, because, as noted in Section 3.1, only inactive metabolites are detected in urine. The use of blood as the matrix for drug analysis provides the opportunity for detecting the psychoactive component of cannabis, THC, but many studies in which blood was analysed still reported the presence of cannabis for cases in which only inactive metabolites were detected. When only THC is taken as an indicator of the presence of cannabis, percentages are lower than when metabolite presence is included in cannabis positive cases. Table 3.1 provides a summary of a number of the most recent studies into cannabis and crash involvement. Note that the presence of cannabis tends to be detected in 10 to 20 percent of cases when metabolites are used as indicators of cannabis but that when THC is measured separately

from metabolites (Drummer et al., 2003; Longo et al., 2000a; Mura et al., 2003), percentages are more likely to be less than 10 percent. An unusually high prevalence of THC metabolites was detected in the study by Gerostamoulous et al. (2002), which the authors thought was due to the hospital used in the study being in close proximity to the inner-city entertainment districts. A summary of earlier research is provided in a report by the UK Department of Environment, Transport and the Regions (2000). This report indicated that cannabis is detected in between 4 and 12% of crash-involved drivers, with higher rates for fatally injured drivers and in studies measuring metabolites of THC.

Table 3.1 includes three recent studies in which the active component of cannabis, THC, was measured in the blood of drivers. One was conducted on a sample of fatally injured drivers (Drummer et al., 2003) and two on injured drivers treated at a hospital (Longo et al., 2000a; Mura et al., 2003). In Drummer et al.'s (2003) study, blood samples from 3,398 fatally injured drivers from three states (Victoria, New South Wales, Western Australia) were analysed over a ten year period (1990-1999) for the presence of a number of drugs, including cannabis. However, it was only possible during the last two years to test for THC, and so the sample for THC analyses was 1,420 drivers. Of these 1,420 drivers, 121 (8.5%) tested positive for THC. Fifty eight drivers tested positive for THC only, 43 also tested positive for alcohol and 20 also tested positive for other drugs. THC concentrations in the blood ranged from 1 to 228 ng/ml. Cannabis was detected most frequently among motorcyclists, rather than car or truck drivers, and more frequently in single vehicle, rather than multiple vehicle, crashes. Alcohol over the legal limit of 0.05 g/100ml was detected in 29 percent of drivers.

Table 3.1
Summary of recent studies into the detection of cannabis in crash-involved drivers

Study	Country	Driver sample	Fluid	% cannabis
Drummer et al., 2003	Australia	1,420 fatal	blood	8.5% THC, 7.1% metabolite only
Longo et al, 2000a	Australia	2,500 injured	blood	2.8% THC, 8.0% metabolite only
Mura et al., 2003	France	900 injured	blood	10% THC
Brault, Dussault, Bouchard & Lemire, 2004	Canada	859 fatal	urine	18.5% metabolite
Bouchard & Brault, 2004	Canada	855 fatal	blood	13.1% metabolite
Del Rio, Gomez, Sancho & Alvarez 2002	Spain	5,745 fatal	blood	2.2% metabolite
Gerostamoulous et al., 2002	Australia	358 injured	blood	36% metabolite
Kintz et al., 2000	France	198 injured	urine	11.1% metabolite
Logan & Scwilke, 2004	USA	370 fatal	blood	12.7% metabolite
Lowenstein & Koziol-Mclain, 2001	Canada	414 injured	urine	17% metabolite
Movig et al., 2004	Holland	110 injured	blood or urine	12% metabolite
Tunbridge et al., 2001	UK	1,184 fatal	blood	11.9% metabolite

In Longo et al.'s study (2000a), blood samples of 2,500 drivers treated at hospital following a road crash were analysed for a variety of drugs, including THC. Only 2.8 percent of these samples were positive for THC, with an additional 8.0 percent positive for metabolites of THC. Combining drivers testing positive for THC and those only testing positive for metabolites, 7.1 percent of the total sample tested positive for cannabis only, 3.2 percent also tested positive for alcohol, and the remaining cannabis-positive drivers, 0.5 percent, also tested positive for other drugs but not alcohol. Alcohol was detected in 12.4 percent of drivers, with 10.5 percent at or above the legal limit of 0.05 g/100ml. No range of THC

concentrations was reported for this study, although an earlier report on the data (Hunter, Lokan, Longo, White, & White, 1998) stated that 18.6 percent of THC positive cases had concentrations at or below 1 ng/ml, 42.9 percent of cases were between 1.1 and 2 ng/ml and the remaining 38.6 percent were above 2 ng/ml. As was found by Drummer et al. (2003), motorcyclists more commonly tested positive for THC, as did drivers involved in single vehicle crashes.

Despite this similarity in the findings, Longo et al. clearly found a smaller percentage of drivers testing positive for THC than was found in Drummer et al.'s study, especially as some of the THC positive drivers in the Longo et al. study had THC blood concentrations below 1 ng/ml, which would not have been counted in the study by Drummer et al. One reason for the difference between these studies in levels of cannabis detection may be that Longo et al. tested drivers presenting with injuries at a hospital, whereas Drummer et al. tested drivers who had died in their crashes. It may be that cannabis is more strongly associated with fatal than non-fatal injury crashes or more strongly with the types of drivers more likely to be involved in fatal crashes. A comparison of the crash samples used in the two studies reveals that, compared to Drummer et al.'s sample, the crashes used in Longo et al.'s study were characterised by a lower percentage of male drivers (58% versus 79%). In the Longo et al. study, 86 percent of THC only cases were male, so if more males had been included in the sample, it is likely that the overall percentage of THC cases would have been higher. Another difference was in the percentage of single vehicle crashes. In the Longo et al. study, 29 percent of the car crashes and 32 percent of the motorcycle crashes were single vehicle crashes, while in the Drummer et al. study, the proportion of single vehicle crashes was 51 percent. In both studies, cannabis was detected more commonly in the blood of drivers having single vehicle crashes. A further difference was in the percentage of motorcycle crashes. The study by Longo et al. included 11 percent motorcyclists compared to 19 percent in Drummer et al.'s study. Motorcyclists were found in both studies to be more likely to have used cannabis prior to the crash. Indeed, Drummer et al. found that the highest percentage of cannabis detection for any demographic group was for motorcyclists aged 22 to 30.

Another factor that could have produced differences across the studies, in terms of the percentage of THC positive cases, was the possibility that the THC blood concentrations of the injured drivers in the Longo et al. study decreased between the time of the crash and the time of testing at the hospital. This time period had a mean of 2.7 hours (SD = 3.0). The likely effect of this is that a number of drivers with THC in their blood at the time of the crash would have only tested positive for a metabolite of THC at the time of testing. In Drummer et al.'s study, all blood tests were carried out within four hours of the crash and, in cases in which the driver died instantly or very soon after the crash, there would have been little degradation in the blood THC concentration (Swann, 2000).

In the other study in Table 3.1 in which THC was tested for, Mura et al. (2003) collected blood samples from 900 drivers attending a hospital in France following a road crash, and analysed the samples for drugs. THC was found in 10 percent of drivers, with THC being detected alone in 60 percent of cases and combined with alcohol in 32 percent. Alcohol over 0.05 g/100ml was found in 26 percent of drivers overall. The percentage of drivers found with THC in their blood in Mura et al.'s study was higher than that found in Longo et al.'s study, despite the fact that samples for both studies were taken from injured drivers. This may represent real regional differences in the prevalence of driving after cannabis use. However, in Mura et al.'s study, the time between the crash and the taking of blood samples was, on average, 1.8 hours (SD = 0.9), which is a shorter average time than in Longo et al.'s study (M = 2.7, SD = 3.0). Also, the sample of drivers in Mura et al.'s study had a higher percentage of males (74%) than the sample used by Longo et al. (58%). As noted earlier, male crash-involved drivers tend to be more likely to have cannabis detected in their blood.

Therefore, the percentage of crash-involved drivers found to have THC in their blood depends on the nature of the sample, particularly with respect to the proportion of male

drivers, motorcycle riders and single vehicle crashes. The time taken between the occurrence of a crash and the taking of a blood sample from an injured driver would also affect the detection of THC. THC has been found in a significant proportion of crash-involved drivers but not more than 10 percent, and clearly less than the percentage found with illegal levels of alcohol in their blood. Also, a significant proportion of drivers testing positive for cannabis also test positive for alcohol.

Although it is useful to conduct studies in which the extent of drug use by crash-involved drivers is examined, such studies provide no information on the *relative risk* of crash involvement when driving after drug use. In order to determine whether a drug increases the risk of crash involvement, it is necessary also to measure the extent of drug use by drivers not involved in crashes. If the level of a drug detected among crash-involved drivers significantly exceeds the level detected among the general driving population, then it can be concluded that use of the drug increases the risk of crash involvement. Such 'case-control' studies are the focus of the next section.

3.3 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, de Gier 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, Brault, Bouchard, & Lemire, 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% of cases and 6.7% of controls. The odds ratio for all cannabis cases (including those in which other drugs or alcohol were involved) was 4.6 but for cannabis alone it was 2.2. Among other drugs used alone, greater odds ratios were found for alcohol 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) (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 & Blakely, 1999; Keall & Frith, 2004; Ogden & Moskowitz, 2004; Walsh, de Gier et al., 2004). In Dussault et al.'s 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, Boase, Mann, Brands, Macdonald and Stoduto (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. 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 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.

Nonetheless, the study had a number of strengths, such as the measurement of drug use among non-crash-involved drivers, and case-control comparisons using the same matrix for drug detection. The results suggest the possibility of a small increase in crash risk with cannabis but not as great as the risk increase associated with alcohol or with the combination of two or more drugs. The authors also emphasise 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).

Another case-control study into drug use and crash involvement was conducted in France by Mura et al. (2003), using a similar method to that of an earlier French study (Marquet et al., 1998). In the Mura et al. study, referred to previously in Section 3.2, 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 (Mura et al., 2003).

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. Another contrast was found between cannabis and alcohol when results were analysed according to drug concentration. For alcohol, odds ratios for crash involvement increased with increasing blood alcohol concentration. However, 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 & 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 people 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 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 a study conducted by Movig et al. (2004) in Tilburg, Netherlands. In this study, 110 drivers sustaining injuries requiring hospitalisation provided either urine or blood samples (cases) and 816 non-crash-involved drivers were recruited at roadside surveys and provided a sample of urine or blood (controls). Cannabis use was determined on the basis of the presence of THC metabolites, which, as previously noted, provide no indication of impairment. It was found that cannabis was detected in 12 percent of cases and 6 percent of controls. These percentages, after controlling for potential confounders, produced an odds ratio of 1.22, which was not significantly different from zero. Significantly elevated odds ratios were found for benzodiazepines (5.1), alcohol at a concentration between 0.05 and 0.079 g/100ml (5.5), alcohol over 0.08 (15.5), drug combinations (6.1), and drugs combined with alcohol (112.2) (Movig et al., 2004).

Again, there are methodological problems with the study. First is that, similarly to Dussault et al's (2002) study, there may be selection bias. The controls represented 79 percent of those approached to participate. Thus, the percentage not participating (21%) was greater than the percentage testing positive for cannabis (6% of controls). Movig et al. did address this problem, reporting that there were no major differences in the age, gender or alcohol levels of those agreeing to participate and those potential controls who declined. The alcohol levels were available because the roadside drug surveys were done in conjunction with random breath testing sessions operated by the police.

Ramaekers et al. (2004) argued that the lack of a significant finding in the study by Movig et al. (2004) for cannabis was due to a small sample size. Movig et al. acknowledged the small sample size and the resulting wide confidence intervals around the odds ratios but did not think that a greater number of cases would have altered their findings. Specifically, they claimed that the confidence intervals would have decreased in size with a larger sample of cases but that the point estimates of odds ratios would have remained the same. That is, according to the authors, the odds ratio of 1.22 for cannabis would not have changed but the width of the confidence interval (0.55-2.73) would have decreased. Movig and colleagues are continuing with the study to increase the sample.

A more important problem with the study by Movig et al. (2004) 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 39 percent of cases provided urine samples and 61 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, McLaughlin, Silverman, & Mandel, 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 detection of cannabis in 7.3 percent of blood specimens but only 6.3 percent of 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, Vulcan, Bowland and Pronk (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 4 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; Movig et al., 2004; Mura et al., 2003). Dussault et al. found cannabis use to produce approximately a twofold risk of crashing, Mura et al. found a two to threefold crash risk related to cannabis only for those aged under 27, and Movig et al. 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. and Movig et al. were both potentially affected by selection bias and the use of urine for drug testing, meaning that only the inactive metabolites of THC could be detected. Mura et al.'s 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 cannabis 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, Tunbridge, Oliver, Torrance, & Wylie, 2004) have run into considerable operational difficulties. These difficulties 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.

3.4 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 & 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 & 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, Peat, Crouch, Wells, & Finkle, 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, the Drummer 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. (2000b) and by Drummer et al. (2004). Studies overseas into cannabis and crash culpability have been conducted in Canada (Dussault et al., 2002) and the United States (Lowenstein & Koziol-Mclain, 2001).

Longo et al. (2000b) 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. Using this method, 55 percent of the injured drivers were designated as culpable, 39 percent as non-culpable and 6 percent as 'contributory'. To make the analysis simpler, the drivers who merely contributed to the crash but were not judged to be culpable were excluded. Compared to the drug-free group, greater culpability was found for those drivers testing positive for alcohol, benzodiazepines, alcohol combined with cannabis, and alcohol combined with benzodiazepines. THC alone 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. 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 when affected by both alcohol and cannabis. Longo et al. (2000b) were able to dismiss any concerns about a small sample size being responsible for not finding a relationship between THC and crash capability. 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. As noted in Section 3.2, Drummer et al. analysed 3,398 blood samples but only 1,420 were analysed for THC, beginning when the necessary technology became available. For the entire sample, 79 percent were classified as being culpable for the crash, 15 percent were classified as not culpable, and 6 percent as contributory. The latter group was excluded from analyses. 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, and those aged from 18 to 25. With regard to cannabis, drivers testing positive for THC alone were found to have an elevated likelihood of being culpable for their crashes. The combination of alcohol and cannabis was also found to have a greater odds ratio for crash culpability than alcohol alone, 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. 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. 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, p245). This last conclusion was based on a

comparison of the culpability of 49 drivers with THC blood concentrations above 5 ng/ml and 9 drivers with concentrations below that level.

As was the case for the incidence of drugs and driving (see section 3.2), the studies by Longo et al. (2000b) 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., 2000b). However, there are three reasons why some degree of confidence can still be placed in the findings.

First, as previously noted in Section 3.2, 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. Secondly, 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. Thirdly, 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 finding no relationship with crash culpability for metabolites of cannabis (Drummer, 1994, 1995), and later studies finding 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 & Blakely, 1999).

In any case, there does at least 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, p17).

Two other studies that included analyses of cannabis use and crash culpability were those conducted in Canada by Dussault et al. (2002) and in the United States by Lowenstein and Koziol-Mclain (2001). Unfortunately, these two studies are unable to provide a solution to the contradictory results of the two Australian studies. This is because both of these studies 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 in Section 3.3) 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 as indicative of the limitations of culpability analyses and a lack of statistical power.

The other study by Lowenstein and Koziol-Mclain (2001) investigated the culpability and drug use of 414 non-fatally injured drivers. Approximately half of the drivers were deemed to be culpable. 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.

To summarise the findings of recent culpability studies, only Drummer et al. (2004) found an increased likelihood of crash culpability associated with cannabis use. Another Australian study that analysed blood samples, but in hospital treated rather than fatally injured drivers, (Longo et al., 2000b) 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. Drummer et al.'s findings are surprising, given that earlier reports by the same authors, grouping 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 of culpability studies is that the non-culpable driver may still have contributed to the causation of the crash (Austroads, 2000; Keall & Frith, 2004; Lowenstein & Koziol-Mclain, 2001; Vingilis & 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 & Frith, 2004). The likelihood of misclassification of culpability can be reduced by assessing culpability on a gradient and eliminating 'contributory' drivers (Bates & 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.

A final 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 & 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. (2000b) 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., 2000b).

4 Summary and conclusions

Cannabis is a mostly recreational drug that is known to produce dose-related decrements in performance on a number of laboratory tasks associated with skills necessary for driving (e.g. Couper & Logan, 2004). Studies of the effects of cannabis on driving performance (measured with on-road driving tests and driving simulators) have revealed that it negatively affects a number of aspects of the driving task but to a lesser degree than it affects performance in laboratory tasks (Smiley, 1999). Although cannabis is found commonly in the blood of crash-involved drivers, second in frequency only to alcohol, this is likely to be due to the fact that it is the second most commonly used drug behind alcohol (Kelly et al., 2004), and so it is necessary to conduct studies in which the crash risk associated with driving under the influence of cannabis can be determined.

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. However, those studies that have been conducted are characterised by methodological flaws that make the interpretation of the results difficult.

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. These studies treat crash-involved drivers who were *not* culpable for their crashes as a control group against which to compare the drug use of crash-involved drivers who *were* culpable for their crashes. The majority have indicated that cannabis is not associated with an increased likelihood of culpability. However, as for 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., 2003; Longo et al., 2000b) that have analysed the relationship between THC 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. A number of recent studies have found an increased risk of crashing related to the use of cannabis (Drummer et al., 2003; Dussault et al., 2002; Mura et al., 2003), while others have found no increased risk (Longo et al., 2000b; Lowenstein & Koziol-Mclain, 2001; Movig et al., 2004). To resolve the issue, it is necessary to conduct a case-control study similar to those that have been conducted for alcohol (Borkenstein et al., 1974; McLean & Holubowycz, 1980). That is, it is necessary to compare the incidence of cannabis in crash-involved drivers with the incidence in non-crash-involved drivers matched for potential confounding factors, such as age, gender, time of day, day of week, direction of travel et cetera (Austroads, 2000; Department of Environment Transport and the Regions, 2000; Jones, Shinar, & Walsh, 2003; Kalant, 2004). Although attempts have been made to conduct such studies, they have been beset by methodological flaws potentially resulting in 'selection bias' and 'information 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. The latter methodological requirement would need the introduction of a system of mandatory roadside drug testing.

Controlling for potential confounders is important because it has been suggested by some authors (Department of Environment Transport and the Regions, 2000; Lowenstein & Koziol-Mclain, 2001; Vingilis & Macdonald, 2002; Walsh & Mann, 1999) that any over representation of indicators of cannabis use among crash-involved drivers may merely reflect a tendency toward cannabis use for certain groups of drivers (young, male, risk-taking) who have a high risk of crash involvement irrespective of any drug use. That is, the characteristics of these drivers may lead them to be more likely to crash and also to be more likely to use cannabis, rather than the cannabis use being responsible for the crash involvement. Fergusson and Horwood (2001) investigated this theory in a three year follow-up study of a cohort of 907 drivers in New Zealand who were aged 18 at the beginning of

the study. An association was found between self-reported levels of cannabis use and involvement in at-fault crashes. However, this association disappeared after statistical control of various driver characteristics (drink driving behaviour, other risky or illegal driving behaviour, driver attitudes, and gender). The authors concluded that the increased risks of crash involvement for cannabis users reflected "the characteristics of the young people who used cannabis rather than the effects of cannabis use on driver performance" (Fergusson & Horwood, 2001, p703).

Finally, it is important to emphasise that alcohol is still the most important drug in terms of its contribution to crash involvement worldwide (Alvarez, Del Rio, Sancho, Rams, & Gonzalez-Luque, 2000; Austroads, 2000; Dussault et al., 2002; Jones et al., 2003; Mathijssen et al., 2002). Alcohol is found more frequently than cannabis and other drugs in the blood of crash-involved drivers and analytical studies have found that the crash risk associated with alcohol far exceeds that associated with drugs. Furthermore, drug-impaired drivers are frequently also impaired by alcohol, which makes the risks associated with drugs difficult to isolate from the well-known adverse effects of alcohol. Nonetheless, as noted in the report by Austroads (2000, piii), cannabis and other drugs "present less of a problem than alcohol, but this does not mean that they are no problem." Cannabis may not be as great a problem as alcohol, but research is still needed, particularly a case-control study, to determine how great a problem it is and whether steps need to be taken to apprehend those who combine cannabis use and driving.

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