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Cannabis and driving: A new perspective

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Abstract

Cannabis and driving is an emerging injury-prevention concern. The incidence of driving while affected by cannabis is rising in parallel with increased cannabis use in the community. Younger drivers are at particular risk. Improvements in research methodology, technology and laboratory testing methods have occurred in the last 10 years. These cast doubt on earlier results and conclusions. Studies now show that cannabis has a significant impairing effect on driving when used alone and that this effect is exaggerated when combined with alcohol. Of particular concern is the presence of cannabis as the sole psychoactive drug in an increasing number of road fatalities and the lack of any structural response to this problem. A review of testing methods, laboratory and real driving studies, and recent epidemiological studies is presented. Suggestions for methods of further data collection and future public policy are made.

Key words: *accidents, cannabis, drugs and driving, injured drivers, marijuana, road trauma, THC, traffic safety.*

Introduction

There are 22 000 persons, on average, hospitalized per year throughout Australia as a direct result of road accidents, with 1817 fatalities for the year 2000.[1] Alcohol, speed and driver fatigue remain the major driver-related issues to be addressed. In the under 40 age group, however, an injury-prevention concern is emerging in the form of cannabis intoxication and driving.

Patterns of drug consumption have changed since the early 1990s, with cannabis use increasing more than any that of any other drug.[2–4] Marijuana has now been tried by almost half of all Australians aged 14–19. Over 40% of 18–19 year olds have used cannabis in the previous year and 1 in 10 of those are using it at least weekly.[5] More people are also driving under the influence of cannabis.[6–11] The vast majority of these are under 40[12] with most finding it acceptable to drive occasionally while impaired.[6,13] Younger drivers seem to be substituting cannabis for alcohol, perhaps to avoid detection by random breath test units.[14,15]

The Australian government has been aware that a potential problem exists since at least 1982.[16] The Federal Office of Road Safety, in conjunction with the Victorian Institute of Forensic Medicine, has established a data base of road fatalities, which shows that cannabis is a greater problem than previously perceived.[17]

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Recent advances in the measurement of active THC (9-delta-tetrahydrocannabinol) have occurred that enable researchers, for the first time, to accurately assess the use of cannabis in the hours preceding a traffic accident or fatality. This paper will briefly review the relevant points of cannabis kinetics and testing, and the acute effects of cannabis on driving. Suggestions for methods of further data collection and future public policy are made.

Cannabis

Cannabis is derived from *Cannabis sativa*, meaning 'useful hemp'. The active ingredient is 9-delta-tetrahydrocannabinol (THC),[18] which for the purpose of this paper will be called active THC.

Potency depends on the content of active THC in the preparation. In Australia, it varies from 0.6% to 13%, with an average of 1–3%. The potency of the plant has not increased but cheaper production methods have resulted in more potent parts of the plant being less expensive.[19] Most cannabis is smoked in a 'joint' (a cigarette) or a 'bong'. Since greater than 98% of cannabis users smoke cannabis,[5] for further discussion smoking as the route of delivery will be assumed.

The duration of effect

Tetrahydrocannabinol is rapidly absorbed when smoked.[20] Plasma levels can reach greater than 100 ng/mL but fall below 20 ng/mL by 1 h and below 10 ng/mL by 4 h.[20,21] Blood levels of active THC correlate poorly with perceived intoxication, mostly due to its high lipid solubility.[18] Its kinetics loosely follow a three compartment model. Peak plasma levels occur 5–8 min after smoking and the onset of clinical effects begin about 5 min after inhalation. The full intensity of effects is delayed for a further 20 min after the peak blood level.[18] Similarly the decline in blood THC levels is not directly linked to the decline in the perception of the drug effect. The delay between decline of effects and decline of plasma concentration makes it difficult to predict the degree of intoxication from plasma levels of THC, especially to exclude an intoxication in the presence of low THC levels.[18] The purpose of measuring active THC levels can only be to detect recent use, or estimate the time of use, rather than directly measure the degree of intoxication.

The duration of a perceived 'high' depends on the dose, and the interval between doses. A single 9 mg dose has a perceived drug effect of about 45 min.[18] If multiple doses are given with a dosing interval of 1 h, perceived effects will last 2.5 h after the last joint.[18]

Most studies comparing the effect of THC levels and duration since last dose have measured the perceived 'high' rather than objective impairment. In a placebo-controlled cross-over trial Leirer *et al.* (1991) detected significant impairment in pilots 24 h after a 20 mg cannabis dose in the absence of any perceived 'high'.[22] Objective impairment improved rapidly over the first 4 h, but improved slowly over the subsequent 24 h.[22] This study suggests that cannabis can produce impairment in the absence of a 'high' and that the greatest impairment is in the first 4 h after consumption. A meta-analysis of the effects of cannabis and driving performance showed that THC-related impairment is concentrated in the first 2 h after smoking, but that tracking skills were impaired up to 4 h and simulator driving could be impaired beyond 5 h.[23]

Measuring THC levels

Huestis *et al.* proposed a formula to calculate time of consumption based on serum plasma active THC levels (Equation 1), and another formula to calculate a 95% confidence interval (Equation 2).[24]

$$\log t = -0.698 \log[\mathrm{THC}] + 0.687 \quad (1)$$

$$\mathrm{CI} = \log t \pm \left(0.030 \left(1.006 + \frac{\left(\log[\mathrm{THC}] - 0.996\right)^2}{89.937} \right) \right)^{0.5} \quad (2)$$

It is important to note whether studies report plasma levels such as the above study, or haemolysed whole blood levels. The majority of research and forensic work in Australia is done on haemolysed whole blood. Due to significant plasma protein binding of THC and very low intracellular red cell concentration of THC, haemolysed whole blood concentrations of THC are approximately half those in plasma samples.[25]

A plasma THC level of 1 ng/mL would correspond with a predicted time of 4 h and 52 min. However, Huestis *et al.* did not test their formula for values in plasma below 2 ng/mL.[24] A THC level of 2 ng/mL in plasma, or approximately 1 ng/mL in haemolysed whole blood, would approximate a predicted time of 3 h with an 80% confidence between 1.8 and 5.0 h.

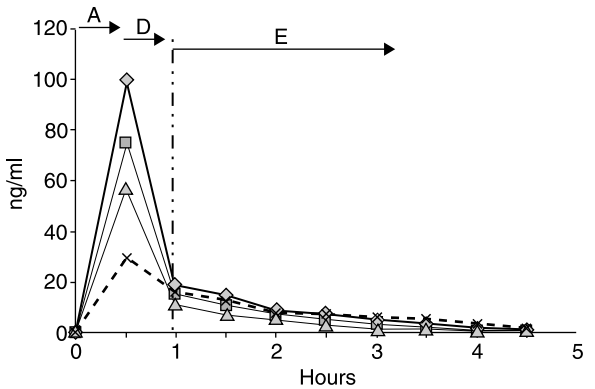

Figure 1. Time course of active THC (9-delta-tetrahydrocannabinol) and THC acid (THC-COOH) concentrations in plasma after smoking marijuana with 15 mg in a 70 kg person. A, absorbtion; D, distribution; E, elimination; ■, maximum; ◇, minimum; △, average; --×--, THC-COOH. (Reprinted with permission, Ward and Dye).[56]

The above equations cannot be applied when predicting the levels of THC after ingestion (eating) of cannabis.[24]

Huestis *et al.* also suggest a formula for predicting time of consumption based on the ratio of THC to its metabolite 11-nor-9-carboxy-delta-9-tetrahydrocannabinol (THC-COOH). This formula was more accurate for cannabis ingestion, and tended to overestimate the time since ingestion for frequent users. It had wider confidence intervals.[24]

Providing there is awareness that cannabis ingestion produces unpredictable absorption of THC, a measure of active THC alone is the authors' preferred option (Fig. 1).

Wall *et al.* (1983) found no difference in cannabis pharmacokinetics between males and females.[26]

Active THC is metabolized over 1–2 days by the liver to form eventually, 11-nor-delta-9-tetrahydrocannabinol-9-carboxylic acid (THC-COOH). This inactive metabolite is excreted over days to weeks.[20,25]

The distinction between active THC and THC-COOH is an important one to make. Active THC levels drop quickly.[20] Studies only reporting total cannabinoids are of little or no value in assessing a relationship to crash risk, as THC-COOH has no effects on the brain. A level of THC-COOH only indicates usage at some point in the last few weeks. No conclusions about culpability and risk assessments based on a level of THC-COOH are possible.[8,27]

Levels of active THC in a blood sample can be distorted by several factors. Recent work by Mark Chu in Melbourne points to a significant drop in active THC levels if the samples are not stored at −60°C. He suggests a loss of over 50% by 8 weeks.[17] Cannabinoids may also bind significantly to the inner surface of plastic vials.[28] Blood stored in a silanized glass vial at −60°C should accurately reflect the blood levels of active THC at the time of sampling.[17]

The detection of cannabinoids in saliva has been an active area of research for over 20 years. It is based on a swab from the mouth, usually near a salivary duct, and a rapid detection kit. Unlike other drugs (e.g. codeine, alcohol) salivary levels of active THC correlate poorly with blood levels.[29] Salivary THC levels appear to derive mainly from sequestration of the drug in the mouth during intake and may correlate well with impairment.[30] Further evaluation of saliva testing is ongoing in Victoria and Europe.[4,8]

A breath test for the presence of cannabis has been evaluated several times, from as early as 1971. Unfortunately, problems with specificity and sensitivity have not been overcome.[31] The most recent advances in this area came from Tasmania in 1998 but these have not been developed further.

Urine testing remains the most commonly used method for cannabis testing, especially with the police force, work place programs, and sporting events. Problems with sensitivity, new versus old cannabis use, and collection make it less than ideal.[4,32]

Effects of cannabis on driving

Researchers have employed several methods of assessing whether or not cannabis impairs driving. Psychomotor testing is an assessment of the skills involved in driving in isolation, and relies on information processing before a response. Attentiveness, vigilance, perception of time and speed, and use of acquired knowledge are all areas affected acutely by THC consumption.[33–36] A meta-analysis of 60 studies up to 1995 found that 'smoking marijuana causes, to a more or less obvious extent, impairment of every performance area connected with the safe driving of a vehicle'.[23]

Simulator studies use a computer-generated environment to simulate the driving experience with the researchers able to introduce variables, such as an obstacle, at their own choosing. The complexity of tasks is greater than with psychomotor tests. Deficits in the cannabis-affected driver appear more readily

as the complexity of tasks increases, in a dose-dependent manner.[32,36] Consistent abnormalities occur in the degree to which they weave, termed Standard Deviation of Lateral Position (SDLP).[33,37,38] An increase in the number of 'signs' missed and the time it takes to make a decision to start, stop or overtake also occurs.[33,37]

Real driving studies occur with the subject in a real car on a paved road completing a set of tasks, which may be preset or random. These are typically closed circuit driving courses or monitored driving on public roads. Interestingly, driving under the influence of cannabis in real urban situations has been difficult to research, with authors citing risk of accidents and risk to population as ethical considerations.[4,34,37,38]

Again, cannabis-affected drivers show an increase in SDLP. This is also associated with an increase in time out of lane. Also noted has been the attempt by the participants to compensate for their impairments, often by driving slower and maintaining greater headway.[32,33,36–38] Unfortunately, they do not overtake when safe to do so, show decreased attentiveness and forget to complete preassigned tasks.[34,37]

Recognized problems with earlier 'real driving' studies include study design, performance improvements due to vigilance, and the tempering of cannabis doses (typically less than 200 μg/kg) to allow subjects to perform.[4,38] The mean dose required for a 'high' is in the order of 300 μg/kg.[21,37,39]

A more recent study on the effects of cannabis on 'real' driving involved 18 subjects in a randomized, double-blinded trial. Improved technology, methodology and a more realistic dose of cannabis revealed deficits that 'were of sufficient magnitude to warrant concern'.[37]

Epidemiological studies

No good data exist on the epidemiology of cannabis and driving. Some key studies have contributed to the widespread perception that cannabis is not a problem on our roads and contributed to the inertia of the community in dealing with this problem.

Terhune[40] in 1992 looked at 1882 driver fatalities in seven American states. He found drivers with active THC alone (only 19) were less culpable for their crash than his control group (drug and alcohol-free drivers killed) but this was not statistically significant. He also noted an increase in the risk of an accident of 11-fold if the drivers had active THC and any alcohol. For both conclusions, unfortunately, his numbers were small, with 3500 fatalities needed to provide an adequate sample size. There were other flaws in this study. Drivers who failed to avoid a potential crash, even when they could have done so, were deemed contributory or less, and therefore not recorded as responsible for their accident. The ideal control group would have been the driving population, and not drug and alcohol free drivers involved in an accident. The method of storage of blood samples and time to testing would have decreased the incidence of finding active THC in the sample group. Testing was not done until death, and death occurred from 1–4 h after the accident in 40% of cases. Furthermore the samples were taken up to 96 h after the subjects' death.

Longo *et al.*[27] published their study in 2000. This time, they looked at injured drivers in South Australia, with a sample size of 2447. They found 44 drivers with active THC alone, the majority of whom had levels less than 2 ng/mL. This was a lower number of drivers than found in an earlier study.[41] Again, this group of drivers was deemed less culpable but, due to low numbers, 'results failed to reach statistical significance'. An average of 2.7 h elapsed between the time of the accident and the time when blood was collected. Specimens were stored, before testing, for up to 2 years, at −20°C, and plastic, non-silanized tubes were used to store the blood. When active THC levels were > 2 ng/mL, or when THC was found with other drugs, culpability began to rise above the control group but Longo suggested that 'very few people affected in this way drive a vehicle'. This author and others[6,41] do not share Longo's faith in human responsibility.

Drummer looked at fatalities in Australia from 1990 to 1993.[42] Out of 1045 vehicle controller deaths, 11% were positive for cannabinoids and 33% for alcohol > 0.05. Over half of the cannabinoid group also had alcohol detected. It was found that the cannabis-only group was less culpable than drug and alcohol-free fatalities but due to insufficient numbers, results failed to reach statistical significance. Importantly, they reported on cannabinoids which do not reflect acute cannabis intoxication rather than active THC.[42]

More recent work by Drummer, in conjunction with the Victorian Institute of Forensic Medicine, VIC ROADS, and the Federal Office of Road Safety, has attempted to minimize methodology, testing and storage flaws.[17] They have found that out of a sample of 544 vehicle controller fatalities in NSW since 1995, 24 had active THC alone. Of these 24, 23 were deemed

fully responsible for their accident and one partially responsible. Blood levels of active THC ranged from 5 to 100 ng/mL. They determined a relative risk of having a fatal crash while under the influence of cannabis as six times that of a normal, unimpaired driver.[17]

Alcohol and cannabis combined

Of any single defined factor, alcohol intoxication is the largest contributor to the road toll.[43] Its risks to the driver have been well documented for some time.[44] Roadside testing is available and has been legislated since 1982 in NSW.[45]

The effects of alcohol and cannabis combined are profound. Road tracking, car following, attention and vigilance have been shown to be affected to a greater degree than either drug alone.[39,46] Robbe *et al.* combined low to moderate doses of cannabis with alcohol (blood alcohol concentration (BAC) of 0.04). The impairment effects produced were consistent with a BAC of 0.09 for the low cannabis dose (100 μg/kg) and a BAC of 0.14 for the moderate cannabis dose (200 μg/kg). They conclude that this combination 'has very severe effects on driving' and that drivers partaking in this combination 'would be exceedingly dangerous'.[37]

Rather than an additive effect, alcohol and cannabis could well act on different areas of the brain to simultaneously impair different functions.[47] Alcohol impairs integrative tasks. Cannabis, on the other hand, seems to affect attention and psychomotor skills primarily.[48,35] This simultaneous impairment would only be evident on complex tasks like 'real driving', when integration of several functions would be required.[36,39,47]

Discussion

As alcohol becomes less of a factor in fatal road trauma in Australia, decreasing from 50% in the late 1970s to 17.9% in 1996,[43] the incidence of fatalities associated with cannabis is probably rising.[8,17,49] Studies performed in the last 10 years show that it does impair driving performance, both in the laboratory and in on-road settings. Key studies contributing to the belief that drivers under the influence of cannabis have a lower risk of having a crash than normal unimpaired drivers have had flaws in methodology, storage and testing. Their reported incidence of drivers with active THC in their blood and the levels found are likely to be substantially underestimated.

Drummer's recent work shows that cannabis now solely accounts for 4–5% of total driver fatalities,[11,17] with that percentage being greater when you focus on drivers under 40. Even then it is likely to be an underestimation.[50]

Nobody is certain as to the degree to which cannabis impairment multiplies your risk of having a fatal crash compared to a normal, unimpaired driver. The most recent estimate by Swann (2000) was 6.4-fold.[17] The impairment with cannabis is also likely to be more pronounced when a situation arises that needs urgent evasive action.[4,37,38]

Government policy on cannabis and driving has been slow to develop and is still inadequate. The Nimbin Hemp Bar, in advising people who smoke cannabis not to drive for 3 h,[51] is providing more comprehensive recommendations about cannabis and driving than any government publication. The Australian Transport and Safety Bureau does not include any questions on illicit drugs and driving in its annual questionnaire to the driving public. No national data bank exists with adequate information on driver fatalities, drug levels and culpability. A coronial database still falls short of being useful in assessing trends in drugs and driving.

Future directions

Funding should be directed towards the development of an accurate, portable roadside-testing device to detect the recent use of cannabis. However, it is not necessary to have such a device available before improving legislation and mounting public campaigns.

The setting of a low per se limit of active THC would also be of benefit as shown overseas.[4,52] This would remove the considerable legal obstacles facing police who suspect a driver of being under the influence of cannabis and ease their responsibility on having to prove impairment. Proving impairment for drivers affected by alcohol was abolished years ago.

Data collection on the incidence of cannabis in road trauma is paramount. Suspicions of the role of alcohol in road trauma in the 1960s was only confirmed after good epidemiological studies.[44] Legislation regarding mobile phones and driving was put in place after epidemiological studies showed an increased risk of having a crash.[53] The use of mobile

phones whilst in control of a vehicle causes, similarly to cannabis, slower driving, weaving and decreased attentiveness.[54] Australian hospitals are obliged by law to take blood samples from vehicle controllers involved in road crashes for later police testing of blood alcohol level. There is the possibility to extend this testing to include a level of active THC. Methodological flaws such as the blood containers and storage issues need to be addressed so as not to underestimate the incidence and levels of active THC in the sample population.

Cannabis use in the community is increasing and its impact on our roads has been underestimated. Moskowitz, in 1985, stated 'Any situation in which safety both for self and others depends upon alertness and capability of control of man–machine interaction precludes the use of marijuana'.[32] The philosophy with all future implementations on cannabis and driving must be 'to remove impaired drivers from the road without delay to provide a safer environment for the community'.[55] Just as motorists know they shouldn't 'drink and drive', so they should also be made aware of the hazards of cannabis and driving through education and enforcement.

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References

1. Australian Transport Safety Bureau. Statistics. [cited 6 Apr 2001.] Available from: URL. http://www.atsb.gov.au.
2. Australian Institute of Health and Welfare. 1998 National Drug Strategy Household Survey: First results. AIHW cat, no. PHE 15, 1999; Canberra: AIHW (Drug Statistics Series).
3. WHO. Trends in substance use and associated health problems. WHO. Fact sheet: No. 127. [cited 20 Jun 2001.] Available from URL. http://www.who.int/inf-fs/en/fact127.html.
4. European Monitoring Centre for Drugs and Drug Addiction. Literature review on the relation between drug use, impaired driving and traffic accidents. (CT.97EP.14) Lisbon: EMCDDA, February 1999.
5. Reid A, Lynskey M, Copeland J. Cannabis use among Australian adolescents: findings of the. National Drug Strategy Household Survey. *Aust. NZ J. Public Health 2000* 1998; **24**: 596–602.
6. Lenne MG, Fry CLM, Dietze PM, Rumbold GR. *Attitudes towards driving after using cannabis alone and in combination with alcohol among young people in Melbourne*. Proceedings of the 15th International Council on Alcohol, Drugs and Traffic Safety; 22–26 May 2000, Stockholm, Sweden. Stockholm: Economy Press, 2000; 464–9.
7. AAMI. Crash Index 6. AAMI producers. [cited 1 Nov 2000.] Available from URL. http://www.aami.com.au
8. Austroads. *Drugs and driving in Australia*. Austroads Incorporated, Sydney. Publication no. AP-R172/00. Sydney: Superfine Printing, 2000.
9. Joyce D. Illicit Drugs, Driving Impairment and Road Trauma in Western Australian Drivers. Report. Perth (WA): Traffic Board of Western Australia; 1996 Oct 7.
10. Queensland Parliamentary Staysafe Committee. Drug driving in Queensland. Nov. 1998. Issues paper no. 3, Parliament of Queensland.
11. Drummer OH, Gerostamoulos J, Batziris H. *Involvement of Drugs in Accident Causation*. Melbourne: Victorian Institute, of Forensic Medicine and Department of Forensic Medicine, Monash University, 1997.
12. Perl J. Drug law enforcement and legislation in New South Wales. Parliament of Victoria, Road Safety Committee. The Effects of Drugs (Other than Alcohol) on Road Safety. First Report. Incorporating Collected Papers. [cited 1 May 2001.] Available from URL: http://www.parliament.vic.gov.au/parlrsc/inquiry/
13. Stewart K, Cohen A, Taylor E, Sole C. *Values and motivations of young drivers: Key components of impaired driving countermeasures*. Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995: Adelaide, Australia. 1995; 148–52.
14. Sugrue M, Seger M, Dredge G. *et al.* Evaluation of the prevalence of drug and alcohol abuse in motor vehicle trauma in South Western Sydney. *Aust. NZ J. Surg*. 1995; **65**: 853–6.
15. Cameron L, Williams J. *Substitutes or Complements? Alcohol, Cannabis and Tobacco*. Melbourne University, Department of Economics Research paper no. 685. [cited 2 May 2001.] Available from URL: http://www.ecom.unimelb.edu.au/ecowww/research/685.pdf
16. Commonwealth, Question without notice, Senate. (PE Baume, Aboriginal affairs). 16 Feb 1982; 8.
17. Swann P. *The real risk of being killed when driving whilst impaired by cannabis*. Proceedings of the 15th International Council on Alcohol, Drugs and Traffic Safety; 22–26 May 2000, Stockholm, Sweden: Economy Press, 2000; 542–8.
18. Harder S, Reitbrock S. Concentration-effect relationship of delta-9-tetrahydrocannabinol and prediction of psychotropic effects after smoking marijuana. *Int. J. Clin. Pharmacol. Ther*. 1997; **35**: 155–9.
19. Hall W, Swift W. The THC content of cannabis in Australia: evidence and implications. *Aust. NZ J. Public Health*. 2000; **24**: 503–8.
20. Huestis MA, Henningfield JE, Cone EJ. Blood cannabinoids. I. Absorption of THC and formation of 11-OH-THC and THCCOOH during and after smoking marijuana. *J. Anal. Toxicol*. 1992; **16**: 276–82.
21. Sticht G, Kaferstein H. *Pharmacokinetic evaluation of published studies on controlled smoking of marijuana*. Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995, Adelaide, Australia. 1995; 397–401.
22. Leirer VO, Yesavage JA, Morrow DG. Marijuana carry-over effects on aircraft pilot performance. *Aviat. Space Environ. Med*. 1991; **62**: 221–7.
23. Berghaus G, Scheer N, Schmidt P. *Effects of cannabis on psychomotor skills and driving performance: a meta-analysis of*

experimental studies. Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995, Adelaide, Australia. 1995; 403–9.

24. Huestis MA, Henningfield JE, Cone EJ. Blood cannabinoids. II. Models for the prediction of time of marijuana exposure from plasma concentrations of delta-9-tetrahydrocannabinol and 11-nor-9-carboxy-delta-9-tetrahydrocannabinol. *J. Anal. Toxicol.* 1992; **16**: 283–90.
25. Felgate PD, Dinan AC. The determination of delta-9-tetrahydrocannabinol and 11-nor-9-carboxy-delta-9-tetrahydrocannabinol in whole blood using solvent extraction combined with polar solid-phase extraction. *J. Anal. Toxicol.* 2000; **24**: 127–32.
26. Wall ME, Sadler BM, Brine BS *et al.* Metabolism, disposition, and kinetics of delta-tetrahydrocannabinol in men and women. *Clin. Pharmacol. Ther.* 1983; **34**: 352–63.
27. Longo MC, Hunter CE, Lokan RJ. *et al.* The prevalence of alcohol, cannabinoids, benzodiazepines and stimulants amongst injured drivers and their role in driver culpability. *Accid. Anal. Prevention*. 2000; **32**: 613–32.
28. Christophersen AS. Tetrahydrocannabinol stability in whole blood: plastic versus glass containers. *J. Anal. Toxicol.* 1986; **10**: 129–31.
29. Magerl HJ, Schulz E. *Methods of saliva analysis and the relationship between saliva and blood concentration*. Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995, Adelaide, Australia, 1995; 43–8.
30. Menkes DB, Howard RC, Spears GFS, Cairns ER. Salivary THC following cannabis smoking correlates with subjective intoxication and heart rate. *Psychopharmacology* 1991; **103**: 277–9.
31. Verstraete AG. *Perspectives for the detection of cannabis in breath*. Proceedings of the 15th International Council on Alcohol, Drugs and Traffic Safety; 22–26 May 2000, Stockholm, Sweden: Economy Press, 2000; 337–41.
32. Gieringer DH. Marijuana, driving and accident safety. *J. Psychoactive Drugs* 1988; **20**: 93–101.
33. Moskowitz H. Marijuana and driving. *Accid. Anal. Prev.* 1985; **17**: 323–45.
34. Hall W, Solowij N, Lemon J. The health and psychological consequences of cannabis use. National Drug Strategy, Monograph Series no. 25. Canberra: Commonwealth Department of Health and Aged Care, 1998.
35. Kurzthaler I, Hummer M, Miller C *et al.* Effect of cannabis use on cognitive functions and driving ability. *J. Clin. Psychiatry* 1999; **60**: 395–9.
36. Liguori A, Gatto CP, Robinson JH. Effects of marijuana on equilibrium, psychomotor performance, and simulated driving. *Behav. Pharmacol.* 1998; **9**: 599–609.
37. Robbe HWJ, O'Hanlon JF. Marijuana, alcohol and actual driving performance. Washington: National Traffic Safety Administration, 1999. Report no. DOT HS 808, 939. US Department of Transportation.
38. Robbe HWJ. *Marijuana's effects on actual driving performance*. Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995; Adelaide, Australia, 1995; 11–22.
39. Ramaekers JG, Lamers CTJ, Robbe HWJ, O'Hanlon J. *Low doses of marijuana and alcohol severely impair driving when taken together*. Proceedings of the 15th International Council on Alcohol, Drugs and Traffic Safety; 22–26 May 2000; Stockholm, Sweden: Economy Press, 2000: 980–5.
40. Terhune KW, Ippolito CA, Hendriks DL, Michalovic JG. The Incidence and Role of Drugs in Fatally Injured Drivers. Washington: National Highway Traffic Safety Administration, 1992. Report no DOT HS 808 065. US Department of Transportation.
41. Starmer GA, Cairns D, Mascord DJ *et al.* Effects of psychoactive drugs on car driving performance: drugs in crash-involved drivers. *Clin. Neuropharm.* 1992; **15** (Suppl. 1): Part A. 598–600.
42. Drummer OHA. review of the contribution of drugs in drivers to road accidents. Parliament of Victoria, Road Safety Committee. The Effects of Drugs (Other than Alcohol) on Road Safety. First Report Incorporating Collected Papers. [cited 5 Apr 2001.] Available from URL. http://www.parliament.vic.gov.au/parlrsc/inquiry/
43. Austroads. *RoadFacts 2000*. Publication, no. AP-G18/00. Sydney: Austroads Incorporated, 2000.
44. Borkenstein RF, Crowther FR, Shumate RP *et al. The role of the drinking driver in traffic accidents*. Indiana University: Department of Police Administration, 1964.
45. Moynham AF, Perl J, Anderson SG *et al. New South Wales road fatalities and legislation on alcohol, drugs and driving since 1950*. Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995, Adelaide, Australia. 1995; 498–502.
46. Jolly BT. NHTSA Notes Commentary: Drugged driving — different spin on an old problem. *Ann. Emerg. Med.* 2000; **35**: 399–400.
47. Chesher GB. The effects of alcohol and marijuana in combination: a review. *Alcohol, Drugs Driving: Abstracts and Reviews* 1986; **2**: 105–20.
48. Kruger HP, Berghaus G. *Behavioural effects of alcohol and cannabis: can equipotencies be established?* Proceedings of the 13th International Council on Alcohol, Drugs and Traffic Safety; 13–18 Aug 1995, Adelaide, Australia. 1995; 410–15.
49. Tutt D, Bauer L, Arms J, Perera C. Cannabis and road death: an emerging injury prevention concern. *Health Prom. J. Aust.* 2001; **12**: 159–62.
50. Collins DJ, Lapsley HM. Economic aspects of drug taking and road safety. Parliament of Victoria, Road Safety Committee. The Effects of Drugs (Other than Alcohol) on Road Safety. First Report, Incorporating Collected Papers. [cited 15 May 2001.] Available from URL. http://www.parliament.vic.gov.au/parlrsc/inquiry/
51. Nimbin Hemp Bar. Mission statement 2000, April 24. [cited 6 Apr 2001.] Available from: URL: http://www.nimbinaustralia.com/hempbar/mission.html
52. Moeller MR, Steinmeyer S, Bregel S, Wilske J. *Zero-limit versus impairment: new approaches for drugs and driving in Germany*. Proceedings of the 15th International Council on Alcohol, Drugs and Traffic Safety; 22–26 May 2000; Stockholm, Sweden. Stockholm: Economy Press, 2000; 808–12.
53. Redelmeier DA, Tirshirani RJ. Association between cellular-telephone calla and motor vehicle collisions. *N. Engl. J. Med.* 1997; **336**: 453–8.

54. Hour MP. Hanging up on driving with mobile phones. Melbourne: Royal Automobile Club of Victoria. [cited 3 Jun 2001.] Available from URL: http://203.89.198.226/racvm/roundabout/tales.cfm

55. Victorian Road Safety Committee. Inquiry into the effects of drugs (other than alcohol) on road safety. Final Report, 1. November 1996; 6: 114. Parliament of Victoria. [cited 6 Apr 2001.] Available from URL: http://www.parliament.vic.gov.au/parlrsc/inquiry/

56. Ward NJ, Dye L. Cannabis and driving: a review of the literature and commentary. Road Safety Research Report no. 12. (S219 m), Transport and the Regions, UK: Road Safety Division, Department of the Environment, May, 2000.