Risks of driving when affected by cannabis, MDMA (ecstasy) and methamphetamine and the deterrence of such behaviour: a literature review

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Abbreviations and acronyms

BAC  blood alcohol concentration
CBT  compulsory breath testing
CI  confidence interval
DRUID  Driving under the Influence of Drugs, Alcohol and Medicines project
ESR  Environmental Science and Research Ltd
OR  odds ratio
RBT  random breath testing
RDT  random drug testing
ROFT  Victorian roadside oral fluid testing programme
SRSP  supplementary road safety package
THC  tetrahydrocannabinol (the principal psychoactive constituent of cannabis)
UDA  unsafe driving action
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Executive summary

Introduction

Drug use and drugged driving is a concern in New Zealand and around the world. In response to this the New Zealand Government is considering options for roadside testing of drivers for drugs. To inform this consideration, a review and analysis of information in the literature on the relationship between driver impairment, the resultant risks to road users and the deterrence effect of drug-testing drivers was undertaken. The scope of the review was confined to three illicit drugs – cannabis, methamphetamine and MDMA (ecstasy) – commonly tested for in jurisdictions that conduct roadside drug testing. The discussion of deterrence in this report gives context to the discussion of driver impairment risk.

Risks associated with drugged driving

Evidence for risks associated with drugged driving comes primarily from case-control studies, culpability studies (these provide estimates of the risk of a case group of drugged drivers compared with a control group) and behavioural studies using driving simulators. The case control studies and culpability studies differ in the risks they portray depending on whether the controls are a sober group or a group representative of the driving population, such as drivers from a roadside survey that already has a proportion of drugged drivers. All the behavioural studies had control groups composed of people who were sober during the studies. However, the drug use of control group members outside the studies differed at times between studies.

The three drugs under consideration do not have well-defined dose response relationships with crashes, such as that of alcohol. Looking at the studies yields a conclusion that the relative crash risk of cannabis for a sober driver is around 1.5 for lower doses of cannabis and around 2 for higher doses of cannabis. No specific estimates of the increased risk for ecstasy and methamphetamine were available at the time of writing although stimulants, as a group, and amphetamines, as a group, appear riskier than cannabis on its own. Overall the fatality risks are considerably higher than the serious injury risks. For both fatalities and serious injuries, alcohol (at levels greater than 0.05 gm/dl) is the riskiest substance, followed by stimulants (such as amphetamines) with cannabis following as moderately risky on its own. All three drugs form more dangerous combinations with alcohol and other drugs than on their own and impact on a wide range of behaviours in simulated driving. Those discovered from the literature are detailed in the table below.

<table>
<thead>
<tr>
<th>Impacts on simulated driving</th>
<th>Cannabis/synthetic cannabis</th>
<th>Methamphetamine</th>
<th>Ecstasy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased reckless driving</td>
<td></td>
<td>Release the brakes inappropriately when stopping</td>
<td>Higher urban speed</td>
</tr>
<tr>
<td>Slower driving, larger headways</td>
<td></td>
<td>Drive too fast for the traffic conditions</td>
<td>Acceptance of smaller gaps</td>
</tr>
<tr>
<td>More signalling errors</td>
<td></td>
<td>Travel slower on the freeway in an emergency</td>
<td>More simulated crashes</td>
</tr>
<tr>
<td>Impaired control of speed, headway and lateral position</td>
<td></td>
<td>Impaired control of lateral position</td>
<td>More signalling errors</td>
</tr>
<tr>
<td>Decreased car control as task demand increases</td>
<td></td>
<td>Execute right turn against movements with a smaller gap</td>
<td>More skidding</td>
</tr>
<tr>
<td>Decreased performance on road tracking tasks</td>
<td></td>
<td>More aggressive driving</td>
<td>More inappropriate braking</td>
</tr>
<tr>
<td>Decreased psychomotor skills, reaction time, visual functions, attention and encoding</td>
<td></td>
<td>Less safe following distances</td>
<td>More aggressive driving</td>
</tr>
<tr>
<td></td>
<td></td>
<td>More aggressive driving</td>
<td></td>
</tr>
</tbody>
</table>
In addition, cannabis users may try to mitigate their impairment by not overtaking, slowing down and focusing their attention in anticipating an expected event for which a known response is required. Of course, this cannot happen when an event is unexpected. Ecstasy and methamphetamine are both stimulants and can also have indirect impacts by assisting people to become exhausted. If such people then drive, they are more susceptible to fatigue-related crashes.

Synthetic cannabis is an emerging threat because it is normally an unpredictable and dangerous cocktail of chemicals of which tetrahydrocannabinol (THC) may, or may not, be one. It should not be confused with plant-derived cannabis and generally has a greater impact. Lately, there has been a concerning number of deaths from this in different settings including on the road with Environmental Science and Research Ltd (ESR) reporting that synthetic cannabinoids were linked to 90 deaths referred to the coroner between May 2017 and May 2019.

It is not clear to what extent these potentially diverse, and possibly unique, ingredients can be picked up by a saliva test. The impacts of synthetic cannabis are generally expected to be similar to those of cannabis but more severe.

**Deterrence of drugged driving**

Deterrence of driving after drug ingestion is a crucial tool in combatting the harmful impact of this practice on road safety. It has been shown that to be successful at a societal level such deterrence should include highly visible general deterrence operations to deter the practice in addition to targeted operations aimed at groups known to be at high risk of offending. The operations should be backed up by supportive public education.

Owing to the extra risk associated with combinations of drugs with alcohol, a significant component of drug testing for those who have tested positive for alcohol is worth consideration. This does not need to be at the legal limit for alcohol, as any combination has the potential for a concerning increase in risk.

The results of being caught should be perceived as swift, certain and severe but should not be perceived as unfair.

The impact of deterrence is hard to predict as it depends, among other factors, on the minutiae of Police tactics and the resources available to them.

Similarly, optimal testing levels are hard to quantify. However, it can be said that any successful campaign will include a substantial general deterrence component. General deterrence refers to the impact of enforcement on those not directly affected by the enforcement. These might be people who hear about a campaign by word of mouth, through public education campaigns, the media etc.
Risks of driving when affected by cannabis, MDMA (ecstasy) and methamphetamine and the deterrence of such behaviour: a literature review

Abstract

The New Zealand Government is considering options for roadside testing of drivers for drugs. To inform this, a review and analysis of information in the literature on the relationship between drug use, driver impairment, the resultant risks to road users and the deterrence effect of drug-testing drivers, was undertaken. The scope of the review was confined to cannabis, methamphetamine and MDMA (ecstasy).

The review found that these drugs, when ingested prior to driving, represent a road safety risk especially in combination with alcohol.

Synthetic cannabis is an emerging threat because it is normally an unpredictable and dangerous cocktail of chemicals of which tetrahydrocannabinol may, or may not, be one. It should not be confused with plant derived cannabis and generally has a greater impact.

Deterrence of driving after drug ingestion is a crucial tool in combatting the harmful impact of this practice on road safety. To be successful such deterrence should include general deterrence operations to deter the practice at a societal level in addition to targeted operations aimed at groups known to be at high risk of offending. To achieve a societal impact the deterrence operations should be backed up with appropriately supportive public education.
1 Introduction

Drug use and drugged driving is a concern in New Zealand and around the world. The New Zealand Government is considering options for roadside testing of drivers for drugs. To inform this, a review and analysis of information in the literature on the relationship between drug use, driver impairment, the resultant risks to road users and the deterrence effect of drug-testing drivers, was undertaken. The scope of the review was confined to cannabis, methamphetamine and MDMA (ecstasy). The literature reviewed included peer reviewed journal articles, conference papers and information from jurisdictional sources. These were accessed through search engines, the WSP Opus information Centre and suggestions from colleagues. There were no formal exclusion criteria.
2 The extent of drugged driving in New Zealand

Drug use and drugged driving is a concern in New Zealand and around the world. According to the New Zealand Drug Survey 2012/2013 (Ministry of Health 2015), 11% of adults aged 15 years and over reported using cannabis in the previous 12 months. Cannabis was used by 15% of men and 8% of women.

Of the 11% who had used cannabis in the previous 12 months, 36% of those who drove during that time reported driving under the influence of cannabis. Men (41%) were more likely to have done so than women (27%). Forty-one percent of Māori users did so compared with 44% of Pacific users and 34% of European users but these figures came with wide confidence intervals.

In the survey, 0.9% of respondents reported amphetamine use. Methamphetamine was not reported on specifically. There are no official figures on MDMA (ecstasy) use or the use of ecstasy by drivers, but Victoria University's Professor Susan Schenk and Professor David Harper\(^1\) estimated in 2015 that 13% of New Zealanders had used it in the past year.

Starkey et al (2017) asked survey respondents about cannabis, amphetamine and party drug use while driving. Apart from cannabis, the sample size of positive answers was very small, indicating a low level of use with a large sampling error. For cannabis, 2.55% of people surveyed by telephone reported use within three hours of driving and 14.2% of a younger sample reported similar use via an internet survey.

The recent Ministry of Transport Discussion Document on Drug Driving\(^2\) reports on the analysis of blood samples of drivers killed in crashes between January 2014 and May 2018, carried out by Environmental Science and Research Ltd (ESR). This analysis found:

- 29% had used alcohol
- 27% had used cannabis
- 10% had used methamphetamine
- 15% had used other drugs.

The proportion found clear of all drugs was not stated, but an earlier ESR study reported by the New Zealand AA\(^3\) found that 52% of 1,046 deceased drivers tested in 2010 and 2011 did not have alcohol or drugs detected in their blood.

ESR also analyses blood samples of drivers stopped by Police and determined to be impaired by drugs. Of these drivers, 59% used cannabis and 41% used methamphetamine. Of the drivers caught drink driving in New Zealand, over a quarter also tested positive for recent cannabis use.

Table 2.1 from the NZ Transport Agency’s Crash Analysis System shows the number of fatalities from crashes where a driver was found to have used drugs or alcohol before driving.

\(^1\) www.victoria.ac.nz/news/2015/06/mdma-legalisation-will-be-a-hard-pill-for-new-zealand-to-swalow Viewed 18/6/2019


\(^3\) www.aa.co.nz/assets/about/newsroom/publications/Drugged-Driving.pdf?m=1503531574%22%20class=%22type:%7Bpdf%7D%20size:%7B1.4%20MB%7D%20file
Table 2.1  Road deaths involving drugs or alcohol in one or more drivers involved

<table>
<thead>
<tr>
<th>Year</th>
<th>Deaths involving drugs</th>
<th>Deaths involving alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Above limits/test refused</td>
<td>Below legal limits</td>
</tr>
<tr>
<td>2018</td>
<td>95</td>
<td>80</td>
</tr>
<tr>
<td>2017</td>
<td>88</td>
<td>74</td>
</tr>
<tr>
<td>2016</td>
<td>61</td>
<td>67</td>
</tr>
</tbody>
</table>

Drugged driver deaths exceeded the deaths of drivers over the drink-driving limits (assuming drivers who refused a test were over the limit). The various measures quoted above indicate significant use of alcohol and drugs by drivers killed in crashes or suspected by Police of being impaired, and substantial numbers of road deaths in crashes where one or more drivers had used drugs or alcohol before driving.

As an international comparison, the EU Druid project (Hels et al 2011) found that in the general driving population 1.32% were affected by cannabis and around 0.9% by amphetamines. No separate figures were available for ecstasy or methamphetamine.

These drivers were involved in fatal crashes, not necessarily killed
3 Road safety risks of drug driving

3.1 Introduction

We do not have the luxury of well-defined dose response relationships around drug impairment of drivers as we have for crash risk and breath alcohol. The most useful information linking driver drug use and crash propensity comes from some case control studies and culpability studies of driver risk of crash and some behavioural studies.

Case control studies occur when a sample of crash-involved drivers is compared with a matched sample of non-crash involved drivers. Examples include studies where hospitalised drivers are compared with drivers stopped in roadside surveys conducted in the catchment area of the hospital for crash victims.

Culpability studies occur when a sample of crash-involved drivers is classified into culpable and non-culpable drivers and these two classifications are compared with each other. These studies typically provide higher risk estimates than case control studies. This is may be related to the fact that in this case the controls are totally unpolluted with drivers who fit the criteria for being included in the cases. The results of culpability studies are also influenced by the definitions of culpability used by the authors.

Both kinds of studies are best done when the body fluids of the subjects are sampled using the same methods. For instance, it is preferable for both cases and controls to be sampled by blood or saliva testing, but not by mixed methods. When mixed methods are used, so-called equivalent cut-off points are used for drug detection thresholds. However, the validity of such cut-off points is somewhat controversial and can lead to biased results.

Both types of studies deliver odds ratios (ORs) which measure the odds of a case being involved in a crash vis-a-vis a control. Where the number of cases is minor compared with the number of controls (as in most of these studies) this approximates to the relative risk of crash between the cases and controls. The basis for this is explained in Hels et al (2011). In most culpability studies, the number of cases is not minor compared with the number of controls.

Both kinds of studies may have drug free control groups or control groups where drugs are not excluded. Where drugs are not excluded from the control group, as when they are taken from roadside survey respondents without screening out drug users, relative risks or ORs may come out smaller as the controls will contain a proportion of drug users.

Behavioural studies are carried out by diverse methods, each with its own set of assumptions and limitations. Recently, they have been mainly studies using various types of driving simulator.

Succeeding sections will endeavour to form a picture of the impact of cannabis, methamphetamine and ecstasy on driving, using the results of some of these studies.

3.2 Case control studies

These have been carried out recently in Europe and North America with varying degrees of precision and varying types of cases and controls. Li et al (2013) compared 737 drivers involved in fatal US crashes with a control group of 7,719 participants in the 2007 National Roadside Survey of Alcohol and Drug Use by Drivers. The participants were those who chose to provide a saliva sample out of a total of 13,069 drivers who were signalled to enter the off-road survey site (Lacey et al 2009). Some drivers who were initially non-responders were eventually persuaded to take part. These drivers were found to have more impairing substances in their systems than the overall sample, suggesting that refusal may have been associated with
heightened impairment. The prevalence data for the controls was corrected for non-participation (Lacey et al 2009). Overall, 31.9% of the cases and 13.7% of the controls tested positive for at least one non-alcohol drug. The estimated ORs of fatal crash involvement for relevant drug types were:

- cannabis: 1.83 [95% confidence interval (CI): 1.39, 2.39]
- stimulant (including amphetamines and ecstasy): 3.57 (95% CI: 2.63, 4.76).

Those positive for both alcohol and drugs had a much greater OR of being in a fatal crash than the others (OR = 23.24; 95% CI: 17.79, 30.28). This compares with those positive for alcohol but drug free where the OR was 13.64 (95% CI: 11.12, 16.72) The results indicated that drug use was associated with heightened crash risk, particularly in combination with alcohol but no dose response figures were given. The threshold for alcohol impairment was a very modest 0.01 gm/100 ml and for cannabis ‘presence’ in saliva. Only a crude OR was quoted for the higher level of 0.08 gm/100 ml; 61.69 (95% CI: 48.54, 78.45).

Using the data from a case control study of crashes in Virginia Beach, Compton and Berning (2015) reported that neither cannabis nor stimulants had demographically adjusted ORs differing significantly from 1. Their crash data was all Police reported crashes, of which a third involved no injury.

A different type of case control study was carried out in rural Norway by Jamt et al (2019). There the 612 cases were crash-involved drivers arrested under suspicion of being under the influence and the 3,027 controls who were randomly selected drivers recruited to a roadside survey in normal traffic. Overall, 81.7% of the cases and 1.6% of the controls had ingested psychoactive substances. Combinations of substances including psychoactive substances and alcohol occurred in 18% of the cases and 0.3% of the controls. This study was limited in its application to risk as the cases were subject to obvious selection bias and the ORs the study produced related to involvement in a crash investigated for suspected drug involvement rather than all crashes. The high presence of psychoactive substances in the crashes indicates the Police judgements were good.

The study yielded the significant ORs depicted in table 3.1, relating to the risk of crash drivers identified as impaired by the Police compared with drivers in the control group. It indicates that it is possible for police to identify substance impaired drivers with a very high relative risk from a group of crash-involved drivers. It is not known how the skills of Norwegian Police in this aspect of Policing would compare with those of New Zealand Police.

### Table 3.1 Statistically significant odds ratios (Jamt et al 2018)

<table>
<thead>
<tr>
<th>Substance</th>
<th>Odds ratio</th>
<th>Lower 95% CI</th>
<th>Upper 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>1.011.1</td>
<td>466.2</td>
<td>2,192.8</td>
</tr>
<tr>
<td>Amphetamine/methamphetamine</td>
<td>171</td>
<td>3 29.7</td>
<td>990.1</td>
</tr>
<tr>
<td>THC (cannabis)(a)</td>
<td>15.3</td>
<td>6.0</td>
<td>39.0c</td>
</tr>
</tbody>
</table>

\(a\)THC or Tetrahydrocannabinol is the principal psychoactive constituent of cannabis.

Another Norwegian case control study (Gjerde et al 2013) compared 508 drivers killed between 2003 and 2010 and 9,261 randomly chosen drivers in normal traffic. For the controls, saliva was tested and for the cases blood. The OR for death in a crash with blood alcohol content (BAC) above 0.02 g/dl and no drugs detected was 124.6 (95% CI 69.1-224.9); the OR for alcohol combined with drugs 602.2 (95% CI 76.1-4764.0). The very high ORs for alcohol alone and in combination, were influenced by study design features and confounding factors and are to be treated with caution. An important factor is that the Norwegians have been so successful at deterring drink driving. Gjerde (2016) reported only 0.2% of drivers at all times of day over 0.02 mg/100 ml), so that those left drinking and driving were at the worst end of the spectrum. As a comparison McSaveney (2009), using travel survey data, reported that in New
Zealand, 2% of vehicle trips in high alcohol hours\(^5\) had a driver with a BAC of 0.05 mg/100 ml or over. The corresponding figure for trips at all times of the day was 0.23%.

The ORs and CIs for cannabis and amphetamines are depicted in table 3.2, with the OR for cases who had tested positive for THC by itself not significantly different from 1. Noteworthy is the high value for amphetamines and the finding that both cannabis and amphetamines form more dangerous combinations with other substances than by themselves. It is also worth noting that this study related to fatally injured drivers and did not have a culpability component. Therefore, the cases include drivers who may have been impaired but not enough to be culpable for the crash in which they were involved. However, their impairment may, notwithstanding, have made them more at risk of non-culpable involvement.

<table>
<thead>
<tr>
<th>Substance(s)</th>
<th>No. of positive cases/controls</th>
<th>Adjusted OR(^6)</th>
<th>95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>THC (may be with other drugs)</td>
<td>31/54</td>
<td>8.9</td>
<td>5.2–15.4</td>
</tr>
<tr>
<td>THC only</td>
<td>7/48</td>
<td>1.9</td>
<td>0.8–4.6</td>
</tr>
<tr>
<td>Amphetamines</td>
<td>45/17</td>
<td>76.9</td>
<td>38.7–152.9</td>
</tr>
<tr>
<td>Only amphetamines</td>
<td>10/8</td>
<td>41.6</td>
<td>12.6–137.1</td>
</tr>
</tbody>
</table>

Three case-control studies regarding alcohol and drugs were carried out as part of the European Commission Immortal project, but only one from the Netherlands was published. SWOV in the Netherlands (Mathijssen and Houwing 2005) compared substances among a hospital sample of 184 seriously injured drivers with the prevalence in a random roadside sample of 3,799 drivers from the catchment area of the hospital. Results are depicted in table 3.3. Strongly increased injury risks were associated with the combined use of several drugs, and with the combination of drugs and a BAC between 0.2 and 0.8 g/l. The OR for cannabis only was a non-significant 1.45, a not surprising result given that a greater proportion of the controls (3.9%) had only cannabis detected compared with the cases (3.4%).

<table>
<thead>
<tr>
<th>Psychoactive substances</th>
<th>Weighted distribution among cases and controls</th>
<th>Odds ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases (N=184)</td>
<td>Controls (N=3,799)</td>
<td></td>
</tr>
<tr>
<td>None detected</td>
<td>55.4%</td>
<td>90.1%</td>
<td>1.00</td>
</tr>
<tr>
<td>Cannabis only</td>
<td>3.4%</td>
<td>3.9%</td>
<td>1.45 (NS)</td>
</tr>
<tr>
<td>Combination of drugs</td>
<td>7.2%</td>
<td>0.5%</td>
<td>24.0</td>
</tr>
<tr>
<td>Alcohol only 0.2–0.5 BAC</td>
<td>1.2%</td>
<td>0.5%</td>
<td>2.12 (NS)</td>
</tr>
<tr>
<td>Alcohol only 0.5–0.8 BAC</td>
<td>2.2%</td>
<td>0.4%</td>
<td>8.28</td>
</tr>
<tr>
<td>Alcohol only 0.8–1.3 BAC</td>
<td>2.5%</td>
<td>0.2%</td>
<td>17.6</td>
</tr>
<tr>
<td>Alcohol only ≥ 1.3 BAC</td>
<td>12.7%</td>
<td>0.2%</td>
<td>87.2</td>
</tr>
<tr>
<td>Alcohol &lt; 0.8+drug(s)</td>
<td>2.0%</td>
<td>0.2%</td>
<td>12.9</td>
</tr>
<tr>
<td>Alcohol ≥ 0.8+drug(s)</td>
<td>8.3%</td>
<td>0.08%</td>
<td>179</td>
</tr>
</tbody>
</table>

\(^5\) High alcohol hours: 10 pm to 4 am daily, plus 4 am to 6 am on Fridays, Saturdays and Sundays.
\(^6\) Adjusted for time period, region, season, road type gender and age group.
After the IMMORTAL project another more comprehensive project was carried out by the EU. This was the DRUID (Driving under the Influence of Drugs, Alcohol and Medicines) project. This project made estimates of the injury risk associated with driving after consuming alcohol and various illegal drugs and medicines. Roadside surveys were conducted in 13 European countries to estimate prevalence and to provide controls for the roadside surveys. The cases were from killed and seriously injured drivers in nine European countries. Blood and/or saliva samples were collected from the cases and the controls. The estimates made related to concentrations at and above cut-off points where both blood and saliva measures could be harmonised. ORs were adjusted for age. All country ORs for serious injury and fatality are depicted in tables 3.4 and 3.5 respectively.

### Table 3.4  Crude and adjusted odds ratios including confidence intervals of getting seriously injured when driving with various substances, based on data from all countries

<table>
<thead>
<tr>
<th>Substance</th>
<th>Crude OR</th>
<th>C.I.</th>
<th>Adjusted OR</th>
<th>C.I.</th>
</tr>
</thead>
<tbody>
<tr>
<td>No substances detected</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>All alcohol concentrations</td>
<td>7.55</td>
<td>6.47–8.80</td>
<td>8.27</td>
<td>7.03–9.74</td>
</tr>
<tr>
<td>0.1 g/L ≤ alcohol &lt; 0.5 g/L</td>
<td>1.05</td>
<td>0.73–1.53</td>
<td>1.18</td>
<td>0.81–1.73</td>
</tr>
<tr>
<td>0.5 g/L ≤ alcohol &lt; 0.8 g/L</td>
<td>3.8</td>
<td>2.48–5.82</td>
<td>3.64</td>
<td>2.31–5.72</td>
</tr>
<tr>
<td>0.8 g/L ≤ alcohol &lt; 1.2 g/L</td>
<td>13.97</td>
<td>8.75–22.29</td>
<td>13.35</td>
<td>8.15–21.88</td>
</tr>
<tr>
<td>Alcohol ≥ 1.2 g/L</td>
<td>55.27</td>
<td>39.52–77.31</td>
<td>62.79</td>
<td>44.51–88.58</td>
</tr>
<tr>
<td>All illicit drugs</td>
<td>2.87</td>
<td>2.12–3.89</td>
<td>2.35</td>
<td>1.72–3.21</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>9.66</td>
<td>4.80–19.46</td>
<td>8.35</td>
<td>3.91–17.83</td>
</tr>
<tr>
<td>Cannabis</td>
<td>1.86</td>
<td>1.20–2.88</td>
<td>1.38</td>
<td>0.88–2.17</td>
</tr>
<tr>
<td>All alcohol-drug combinations</td>
<td>31.97</td>
<td>20.76–49.25</td>
<td>28.82</td>
<td>18.41–45.11</td>
</tr>
<tr>
<td>All multiple drug combinations</td>
<td>8.64</td>
<td>5.85–12.75</td>
<td>8.01</td>
<td>5.34–12.01</td>
</tr>
</tbody>
</table>

### Table 3.5  Crude and adjusted odds ratios including confidence intervals of getting killed when driving with various substances, based on data from all countries

<table>
<thead>
<tr>
<th>Substance</th>
<th>Crude OR</th>
<th>C.I.</th>
<th>Adjusted OR</th>
<th>C.I.</th>
</tr>
</thead>
<tbody>
<tr>
<td>No substances detected</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>All alcohol concentrations</td>
<td>37.64</td>
<td>29.36–48.24</td>
<td>34.9</td>
<td>27.00–45.11</td>
</tr>
<tr>
<td>0.1 g/L ≤ alcohol &lt; 0.5 g/L</td>
<td>9.23</td>
<td>6.07–14.05</td>
<td>8.01</td>
<td>5.22–12.29</td>
</tr>
<tr>
<td>0.5 g/L ≤ alcohol &lt; 0.8 g/L</td>
<td>42.94</td>
<td>21.99–83.86</td>
<td>45.93</td>
<td>23.02–91.66</td>
</tr>
<tr>
<td>0.8 g/L ≤ alcohol &lt; 1.2 g/L</td>
<td>34.81</td>
<td>16.02–75.65</td>
<td>35.69</td>
<td>15.68–81.22</td>
</tr>
<tr>
<td>Alcohol ≥ 1.2 g/L</td>
<td>450.37</td>
<td>224.06–905.25</td>
<td>500.04</td>
<td>238.07–inf</td>
</tr>
<tr>
<td>All illicit drugs</td>
<td>3.85</td>
<td>2.17–6.80</td>
<td>3.55</td>
<td>1.97–6.42</td>
</tr>
<tr>
<td>Cannabis</td>
<td>1.8(1)</td>
<td>0.73–4.44</td>
<td>1.33</td>
<td>0.48–3.67</td>
</tr>
<tr>
<td>All alcohol–drug combinations</td>
<td>41.22</td>
<td>22.59–75.24</td>
<td>31.52</td>
<td>16.83–59.05</td>
</tr>
<tr>
<td>All multiple drug combinations</td>
<td>16.77</td>
<td>9.95–28.27</td>
<td>18.51</td>
<td>10.84–31.63</td>
</tr>
</tbody>
</table>

(1) Means that in the case of a zero cell in the 2x2 chi square table, a value of 0.5 was placed in all the cell to allow a calculation to proceed.
It is apparent that for both fatalities and serious injuries, alcohol was the riskiest substance (at levels greater than 0.05 gm/dl) for this study, followed by amphetamine with weak evidence of cannabis following as a slightly increased risk on its own. Alcohol drug combinations were riskier than drugs in combination or drugs or alcohol singly. Overall the fatality risks were considerably higher than the serious injury risks.

3.3 Culpability studies and other similar studies.

Before considering individual culpability studies, it is instructive to look at the findings of Rogeberg (2019). Rogeberg conducted a meta-analysis of the crash risk of cannabis-positive drivers in culpability studies. His major point, a very valid one, was that culpability studies typically reported an OR which reflected the increased risk of a culpable driver, in the case of this report, a drug affected culpable driver. These ORs are frequently reported as if they are estimates of increased crash risk per se. This is called ‘interpretational bias’. The impact of this bias is that culpability studies typically exaggerate the size of risk related to all drivers in crashes, including those who are not culpable, and increase the uncertainty in those risks. Rogeberg provides a correction mechanism for this by applying a model to 13 published culpability studies of the increase in average culpable crash risk associated with cannabis positive drivers. These studies were gleaned from the no-alcohol counts of the studies in the meta-analysis of Rogeberg and Elvik (2016). The model was in terms of the three parameters below:

- The true share of drivers on the road who are positive and negative on some substance or risk factor (ideally in a sample conditioned on scoring negative on other substances).
- The baseline culpability probability amongst negative drivers
- The relative risk of having a culpable accident given that you are positive

The modelling provided table 3.6 which compares culpability study relative risks with crash relative risks, evolved from the culpability studies via modelling. The exaggeration of the crash risks from the culpability studies is obvious.

Table 3.6 Crash relative risks and culpability relative risks derived from various culpability studies via modelling

<table>
<thead>
<tr>
<th>Study</th>
<th>Crash relative risk</th>
<th>Culpability relative risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Terhune (1982) (N = 259)</td>
<td>1.18 (0.91–1.56)</td>
<td>1.48 (0.78–2.46)</td>
</tr>
<tr>
<td>Williams et al (1985) (N = 97)</td>
<td>1.15 (0.74–1.59)</td>
<td>1.23 (0.61–1.92)</td>
</tr>
<tr>
<td>Terhune et al (1992) (N = 818)</td>
<td>1.13 (0.73–1.58)</td>
<td>1.20 (0.6–1.87)</td>
</tr>
<tr>
<td>Longo et al (2000) (N = 1931)</td>
<td>1.08 (0.82–1.36)</td>
<td>1.15 (0.66–1.69)</td>
</tr>
<tr>
<td>Lowenstein Kozial-Mcclain (2001) (N = 250)</td>
<td>1.14 (0.83–1.51)</td>
<td>1.30 (0.65–2.06)</td>
</tr>
<tr>
<td>Drummer et al (2004) (N = 1646)</td>
<td>1.82 (1.12–2.95)</td>
<td>1.82 (1.15–3.04)</td>
</tr>
<tr>
<td>Laumon et al (2005) (N = 8421)</td>
<td>1.49 (1.3–1.72)</td>
<td>1.89 (1.54–2.31)</td>
</tr>
<tr>
<td>Soderstrom et al (2005) (N = 1705)</td>
<td>1.18 (0.96–1.43)</td>
<td>1.28 (0.94–1.67)</td>
</tr>
<tr>
<td>Bédard et al (2007) (N = 32,543)</td>
<td>1.23 (1.15–1.32)</td>
<td>1.96 (1.25–1.55)</td>
</tr>
<tr>
<td>Poulsen et al (2014) (N = 623)</td>
<td>1.33 (0.96–1.8)</td>
<td>1.44 (0.94–2.07)</td>
</tr>
<tr>
<td>Li et al (2017) (N = 23,864)</td>
<td>1.29 (1.21–1.39)</td>
<td>1.98 (1.48–2.62)</td>
</tr>
<tr>
<td>Martin et al (2017) (N = 3262)</td>
<td>1.54 (1.25–1.95)</td>
<td>1.98 (1.46–2.74)</td>
</tr>
</tbody>
</table>
By looking at the confidence limits in table 3.6, it is possible to select out those studies where the bottom
of the confidence range exceeds 1, denoting that the risk is significantly greater than 1 according to the
model. These studies are highlighted in green. It is apparent that these are the studies with the highest
sample size. Some of the other studies may have also found significant differences had the samples
available to them been larger, so their results cannot be interpreted as an absence of heightened risk.

Paulson et al (2014) examined the culpability of drivers killed in New Zealand road crashes and their use
of alcohol and other drugs. The study analysed blood samples taken from 1,046 dead drivers over five
years. Driver culpability was determined for all crashes. The control group was drug- and alcohol-free
drivers whose crashes were 52.2% of the total. Driver culpability with combined alcohol and cannabis use
exhibited an OR of 6.9, 95% CI 3.0–16. The association between cannabis use (with no other drug) and
culpability (OR 1.3, CI 95% 0.8–2.3) was weak and the OR for drivers with blood THC concentrations
greater than 5 ng/mL was lower (OR 1.0, CI 95% 0.4–2.4) than those with blood THC concentrations less
than 2 ng/mL (OR 3.1, CI 95% 0.9–10). Sample sizes were not large enough to give any risk information
for methamphetamine or ecstasy.

Brubacher et al (2019) of British Columbia conducted a responsibility analysis to determine if drivers
injured in motor vehicle crashes testing positive for THC or other drugs were more likely to have
contributed to the crash than those not testing positive. The driver information was from clinically required
blood tests on 3,005 drivers following a crash gathered by trauma centres in British Columbia from
January 2010 to July 2016. The analysis quantified alcohol and THC levels and provided semi-
quantitative information on other drugs. Information on which drivers contributed to their crash and which
did not were gleaned for 2,318 drivers from Police crash reports. A consent waiver was obtained,
obviating the risk of drug users refusing the test.

Unconditional logistic regression was used to determine the OR of crash responsibility in drivers related
to THC level. Risk estimates were adjusted for age, gender and the presence of other impairing
substances. Alcohol was detected in 14.4% of drivers, THC in 8.3%, other drugs in 8.9% and sedating
medications in 19.8%. For driver THC lower than 5 ng/mL no increased risk of crash responsibility was
found. For drivers with THC 5 ng/mL or more, the adjusted OR of 1.74 was not significant (95% CI=0.59–
6.36; p=0.35). Risk was significantly increased for driver BAC≥0.08%, (OR =6) and when other
recreational drugs or sedatives were detected. Drinking drivers who also used cannabis had a higher risk
(OR=7.3 for 0<THC<2ng/ml; OR=6.8 for THC≥2ng/mL) than those who did not use cannabis (OR=4.2). It
must be noted that all drivers in this study had been involved as a driver in a crash. Therefore, there may
be a bias associated with drivers who did not crash being excluded. The drivers in the comparison group
had also been involved in a crash, so there was likely to be some culpability and these odds would
underestimate relative crash involvement odds.

A culpability study of fatal crashes aimed primarily at cannabis was conducted in France in 2001 by
Laumon et al (2005). There were 6,766 cases where drivers were at fault, and 3,006 controls where
drivers were not at fault. Of these, 681 drivers were positive for cannabis including 8.8% of cases and
2.8% of controls. A dose response relationship found between cannabis use and ORs of at fault drivers is
shown in table 3.7.
Table 3.7  Odds ratios and confidence limits

<table>
<thead>
<tr>
<th>THC concentration (ng/ml)</th>
<th>Odds ratio</th>
<th>Confidence limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not detected</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>1.89</td>
<td>(1.03 to 3.47)</td>
</tr>
<tr>
<td>1 to 2</td>
<td>2.04</td>
<td>(1.47 to 2.84)</td>
</tr>
<tr>
<td>3 to 4</td>
<td>2.78</td>
<td>(1.61 to 4.78)</td>
</tr>
<tr>
<td>≥5</td>
<td>2.13</td>
<td>(1.22 to 3.73)</td>
</tr>
<tr>
<td>Overall</td>
<td>2.37</td>
<td>(1.89 to 2.97)</td>
</tr>
</tbody>
</table>

The results were adjusted for the effects of alcohol, which was present in 285 of the cannabis positive cases. It is important to note that in this case the OR did not approximate to a relative risk, as the number of cases was greater than the number of controls.

A broadly similar dose response relationship was found for alcohol but at a considerably higher level with the overall OR for alcohol being 9.5 compared with 2.37 for cannabis. The ‘overall’ alcohol risk was dependent on the average level of alcohol in their particular sample (which would be very setting specific).

The study did not unearth any synergy-related increased culpability risk of cannabis in combination with alcohol. The adjusted joint overall impact of blood alcohol and THC was 14 (CI 8.00 to 24.7), not far from the product of the adjusted individual effects (1.78×8.51 = 15.1). The study included all at-fault drivers, whether they or others were hurt in the crash. This may be a reason why the study found higher risks than in some studies involving only fatally injured drivers who may or may not have been at fault.

Dubois et al (2015) examined drivers aged 20 and older involved in fatal crashes in the USA between 1991 and 2008 by means of a case control study. The cases had at least one unsafe driving action (UDA) recorded in relation to the crash—the cases had none, making it similar to a culpability study. The study looked at the combined influence of cannabis combined with alcohol doses less than or equal to 0.08milligrams/100ml of blood. They concluded that drivers positive for both alcohol and cannabis had a greater propensity to error than those positive for cannabis or alcohol alone. They presented their results in the form of dose response relationships between alcohol, cannabis, both combined and the applicable ORs. They found that within the range of the data:

> each 0.01 BAC unit increased the odds of an UDA by approximately 9-11%. Drivers who were positive for THC alone had 16% increased odds of an UDA. When alcohol and THC were combined the odds of an UDA increased by approximately 8-10% for each 0.01 BAC unit increase over alcohol or THC alone.

### 3.4 Systematic reviews/meta analyses

No systematic reviews involving methamphetamine or ecstasy contained enough users to calculate significant ORs. A systematic review of studies involving cannabis (Asbridge et al 2012) drilled down to nine case control studies of cannabis (one of which used self-report), with the rest analysing blood samples. Confounding effects of alcohol were removed for the cannabis only findings by excluding cases where alcohol was also found.

The analysis was confined to studies involving only deaths and serious injuries and included culpability studies. Its findings were that case-control studies (2.79 (CI 1.23 to 6.33); P=0.01) yielded larger ORs
than culpability studies (1.65 (CI 1.11 to 2.46); P=0.07), a finding which is in conflict with other studies (Rogeberg 2019).

Fatal crash studies yielded an OR of 2.10 (CI 1.31 to 3.36); P=0.002), which was statistically significant, but non-fatal crash studies did not reach significance (1.74 (CI 0.88 to 3.46); P=0.11).

In all cases, where both cannabis and alcohol were used, the estimated OR for the combination was higher than cannabis use alone. This was described as suggesting a synergistical effect, but it may also be an additive effect, as suggested by some other authors.

It is not clear why the authors included the self-report study. They must have had a high level of confidence in the way this method was used in this case vis-à-vis direct measurement of cannabis.

Another systematic review (Li et al 2012) also looked at nine studies. Estimated OR relating cannabis use to crash risk ranged from 0.85 to 7.16 with a pooled OR estimate of 2.66 (95% CI = 2.07, 3.41), indicating a significantly increased risk of involvement in motor vehicle crashes. However, of the nine studies five were based on self-report, one used metabolites in urine,\(^7\) and the other partially used metabolites in urine. This makes the results of these studies more open to question than studies using THC levels in saliva/blood.

To pull the above diverse figures together and provide some sound overall estimates for cannabis, Rogeberg et al (2018\(^8\) and 2016) replicated the systematic reviews of Li et al (2012) and Asbridge et al (2012) adjusting for what they considered to be deficiencies in methodology and adding results obtained from some more recent work. The improved methodology of the review combined with the greater number of studies included should provide better estimates of risk than the other studies.

They found that overall pooled risks using the standard mixed effects model was 1.32 (95% CI = 1.09, 1.59) and 1.18 (95% CI = 1.07, 1.3) using a meta-regression model (Rogeberg et al 2018\(^9\)). The reason for the smaller estimates in the meta regression model was an endeavour to control for publication bias which should increase robustness. The authors divided the literature they perused into several subgroups based on type of study, quality, confounding effects, data quality and whether fatalities were involved or not (see table 3.8). It is apparent that over all the subgroups, the ORs are low to moderate.

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Mixed-effects model</th>
<th></th>
<th>Meta-regression model</th>
<th></th>
<th>Publication bias p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>OR</td>
<td>CI</td>
<td>OR</td>
<td>CI</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>26</td>
<td>1.32 (1.09, 1.59)</td>
<td>1.18 (1.07, 1.3)</td>
<td>0.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case–control</td>
<td>15</td>
<td>1.82 (1.19, 2.79)</td>
<td>1.11 (0.86, 1.43)</td>
<td>0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Culpability</td>
<td>11</td>
<td>1.12 (1.05, 1.2)</td>
<td>1.20 (1.08, 1.35)</td>
<td>0.78</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High quality</td>
<td>8</td>
<td>1.53 (1.11, 2.09)</td>
<td>1.19 (1.05, 1.35)</td>
<td>0.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medium quality</td>
<td>14</td>
<td>1.26 (0.88, 1.81)</td>
<td>1.02 (0.8, 1.29)</td>
<td>0.13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low quality</td>
<td>4</td>
<td>1.20 (0.7, 2.06)</td>
<td>1.58 (0.87, 2.86)</td>
<td>0.46</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^7\) These may persist for up to 10 days after ingestion of cannabis

\(^8\) The 2018 reference corrects some issues which came to light after Rogeberg et al (2016 was published, but which made only very small changes to the final results. This review quotes the final 2018 corrected figures.

\(^9\) Non-peer reviewed letter to the editor
Risks of driving when affected by cannabis, MDMA (ecstasy) and methamphetamine and the deterrence of such behaviour: a literature review

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Mixed-effects model</th>
<th>Meta-regression model</th>
<th>Publication bias p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited or no confounder adjustment</td>
<td>16</td>
<td>1.62 (0.98, 2.67)</td>
<td>0.88 (0.48, 1.59)</td>
<td>0.09</td>
</tr>
<tr>
<td>High confounder adjustment</td>
<td>10</td>
<td>1.20 (1.05, 1.37)</td>
<td>1.19 (1.07, 1.32)</td>
<td>0.43</td>
</tr>
<tr>
<td>Low-quality use data</td>
<td>6</td>
<td>1.12 (0.93, 1.35)</td>
<td>1.11 (0.9, 1.36)</td>
<td>0.85</td>
</tr>
<tr>
<td>Medium-quality use data</td>
<td>9</td>
<td>2.35 (1.1, 5)</td>
<td>1.41 (0.58, 3.45)</td>
<td>0.27</td>
</tr>
<tr>
<td>High-quality use data</td>
<td>11</td>
<td>1.26 (0.97, 1.64)</td>
<td>1.19 (1.04, 1.37)</td>
<td>0.47</td>
</tr>
<tr>
<td>Alcohol controlled</td>
<td>24</td>
<td>1.31 (1.07, 1.6)</td>
<td>1.17 (1.06, 1.3)</td>
<td>0.09</td>
</tr>
<tr>
<td>Alcohol not controlled</td>
<td>2</td>
<td>1.50 (1.05, 2.16)</td>
<td>1.74 (NaN)</td>
<td>NaN</td>
</tr>
<tr>
<td>Fatalities involved</td>
<td>13</td>
<td>1.30 (1.04, 1.61)</td>
<td>1.21 (1.08, 1.35)</td>
<td>0.34</td>
</tr>
<tr>
<td>Fatalities not involved</td>
<td>13</td>
<td>1.52 (0.99, 2.34)</td>
<td>1.00 (0.77, 1.31)</td>
<td>0.09</td>
</tr>
</tbody>
</table>

(a) This means the confidence limits were unable to be defined.

The overall risks quoted are for all injury crashes. Risks for fatal crashes, eg (1.30, CI (1.04, 1.61)) and injury only crashes (1.52, CI (0.99, 2.34)) using the mixed-effects model, can be found in the table above.

In a later publication, Rogeberg et al (2018) look at the risks of cannabis-affected drivers at higher blood levels of the drug. They suggest an average upper bound OR of around 2 for a THC level higher than 5 ng/ml. This is between the adjusted alcohol ORs quoted in table 3.4 related to risk of traffic injury for the ranges (0.1 g/L ≤ alcohol < 0.5 g/L) where the OR is 1.18 and (0.5 g/L ≤ alcohol < 0.8 g/L) where the OR is 3.64. They then stress that these risks are average risks for those considered THC positive according to the criteria of the underlying studies. For every individual, impairment can be expected to rise as their THC levels rise with the susceptibility to impairment varying with the individual. For any single individual, impairment would be expected to rise with higher THC levels, although the relationship between THC levels and impairment across people will vary due, for instance, to different tolerance, experience and ‘baseline’ unimpaired THC blood levels. The authors point out that given cannabis users tend to know they are impaired, users may avoid driving when they feel their impairment is high. If such people became more likely to drive, they could have substantially higher risks than those reflected in the average quoted above.

Hostiuc et al (2018) carried out a meta-analysis of the association of cannabis usage with ‘unfavourable traffic events’ which meant crashes involving injury or death. Cannabis use was assessed by detection of in blood THC, metabolites in urine or saliva, or self-report of pre-crash use. This means that for the non-blood analyses, some of the cannabis may have been ingested days before the crash thus influencing odds ratios downwards. Twenty-four studies survived the chosen inclusion criteria. Publication bias was considered as part of the study. It is not totally clear, but it appears that the ORs obtained refer to the odds of any unfavourable traffic event occurring. Their conclusion after producing ORs for several
subgroups was that overall, they could not find a significant impact of cannabis use on unfavourable traffic events.

### 3.5 Behavioural studies

It is well known that drugs have a variety of impacts which may affect driving behaviour in both negative and positive directions. The overall impact of drugs depends on:

- the context in which they are used and how much they are used
- in what doses and combinations they are used.

There is more behavioural research aimed at the impact of cannabis on driving than related to the impact of methamphetamine and ecstasy on driving. Some research relates to the wider group of amphetamines and this will be included here to give further insight into the impact of stimulants, a group to which both methamphetamine and ecstasy belong.

#### 3.5.1 Cannabis

##### 3.5.1.1 Plant derived cannabis

Smiley (1999) conducted a review of driving simulator and on-road studies of the impact of cannabis on driving. The studies showed that cannabis might impair some driving skills at low smoked doses of THC (6.25 mg). Results were variable between the skills and among studies. The cannabis impairment was subject to partial user mitigation. It appeared that subjects recognised their impairment and, where possible tried to compensate by keeping greater distance from other vehicles, not overtaking, slowing down and focusing their attention before a known response was required. Of course, this cannot happen when an event is unexpected.

Berghaus et al (1995) conducted meta-analyses of experimental studies on the impacts of low doses of alcohol and cannabis. Performance areas including road tracking, psychomotor skills, reaction time, visual functions, attention and encoding/decoding all declined in simulated or real driving experiments. The worst deterioration was in attention, tracking and psychomotor skills. They found that blood THC above 5 ng/ml impaired automatic functions. Performance of tasks requiring cognition was stable up to 10 ng/ml. This supports findings of Smiley (1999) who found that drivers influenced by cannabis might compensate consciously for some of their performance impairment.

Starkey and Charlton (2017) carried out a systematic review including cannabis-related behavioural studies: Their findings included:

- Habitual use was associated with increased reckless driving and speeding in a driving simulator task (Bergeron et al 2014; Bergeron and Paquette 2014).
- More signalling errors and greater driving impairment occurred when drivers were tested under the influence of cannabis (Bergeron et al 2014; Bergeron and Paquette 2014).
- There was impaired control of speed, headway and lateral position in a simulated driving task (Lenne et al 2010; Hartman et al 2015)
- As task demand increased, participants’ car control decreased (Lenne et al 2010).
- There was decreased performance on tracking tasks (Menetrey et al 2005).
- There was an increased likelihood of being classed as impaired on a clinical test of impairment (Bramness et al 2010; Khiabani et al 2006).
Risks of driving when affected by cannabis, MDMA (ecstasy) and methamphetamine and the deterrence of such behaviour: a literature review

More recent information (Hartman et al 2016) indicated that cannabis was associated with slower driving and greater headway on a simulated drive. The authors interpreted this as suggesting awareness of impairment and a desire to compensate, in line with the conclusions of Smiley (1999) and Berghaus (1995). No author appears to consider the possibility that this sort of behaviour could be a direct physiological impact of cannabis ingestion rather than awareness of impairment and a desire to compensate for that impairment.

Battistella et al (2013) investigated the impact of cannabis on the driving ability of occasional cannabis smokers, by investigating a tracking task and changes in the brain network involved in the task. Thirty-one male volunteers participated in the experiment including clinical and toxicological aspects with MRI brain scans and measurements of psychomotor skills. Cannabis smoking, even at low THC levels, decreases psychomotor skills and alters the activity of the brain networks involved in cognition. Subjects are more attracted by intrapersonal stimuli (‘self’) and neglect task performance, thus reducing overall performance. These effects correlate with a subjective feeling of confusion rather than THC level.

Chow et al (2018) reviewed several studies related to cannabis and driving and came to broadly similar conclusions to authors already mentioned.

3.5.1.1 Synthetic cannabis

Synthetic cannabis is worth mentioning because it is normally an unpredictable and dangerous cocktail of chemicals of which THC may, or may not, be one (Cohen and Weinstein, 2018). Therefore, it should not be confused with plant derived cannabis and generally has a greater impact. There may often be intention among the manufacturers to use hard to detect compounds to reduce users’ probability of being caught by body fluid testing (Cohen and Weinstein, 2018). It is not clear to what extent these potentially diverse, and possibly unique, ingredients are able to be picked up by a saliva test. Chase et al, 2015 reported that drivers under its influence suffered more from confusion, disorientation, and incoherent, slurred speech than drivers under the influence of cannabis in a group of drugged drivers evaluated by Drug Recognition Experts. There have been several deaths from this lately in different setting, including motor vehicle crash deaths. There have been a concerning number of deaths from this lately in different settings including on the road, with ESR reporting that synthetic cannabinoids were linked to 90 deaths referred to the coroner between May 2017 and May 2019.

3.5.2 Stimulants

3.5.2.1 Ecstasy

Ecstasy is a drug which is used both as a stimulant, and to improve mood (Bosker et al 2010; Kuypers et al 2008), particularly at nightclubs and ‘raves’. It is to be expected that some attendees will drive home after taking ecstasy at an event. Studies from Australia (Duff and Rowland 2006) and Scotland (Riley et al 2001) report a likelihood that after such events some people will drive home under the influence of ecstasy. 8–33% of the ecstasy users reported that they would either drive themselves while under the influence of drugs or were passengers of drugged drivers.

In another study, Kuypers et al (2006) indicated that ecstasy’s stimulating properties were not strong enough to counteract alcohol impairment when taken with alcohol.

This finding was replicated by Brookhuis et al (2004). Rave patrons were subjected to testing in a simulator before and after a rave. All subjects used multiple drugs including ecstasy and all subjects took

11 www.esr.cri.nz/assets/Low-res-Crime-Scene-Intelligence-newsletter-web.pdf
more risks early in the morning than drug free controls. However, the impact of ecstasy on this was confounded by the impact of other drugs and lack of sleep. Using ecstasy alone the subjects had twice the number of simulated crashes than when drug free, accepted smaller gaps, and drove faster. The authors discuss how the mode of operation of ecstasy makes prosecution for driving under its influence problematic. This is because the impairment in driving may not always be directly related to the ecstasy, but more related to lack of sleep, which likely would not have occurred if the person had not consumed ecstasy.

Stough et al (2011) investigated car driving skills and cognitive abilities after ingesting ecstasy. Sixty-one healthy, recreational ecstasy users with full driving licences (29 female and 31 male) were tested on a driving simulator after ingesting ecstasy and with a placebo. Simulated performance related to signal changes, dangerous action skidding, inappropriate braking, safe following distance, and appropriate urban speed was significantly worse compared to the placebo.

Dastrup et al (2010) looked at the driving skills of ecstasy users, who were currently abstaining, using a simulator. The controls were abstinent THC users, abstinent alcohol users, and non-drug users. The task was to stay a set minimum distance (2 car lengths) behind a lead vehicle, the speed of which was changing unpredictably. The ecstasy group performed as well as the controls in vigilance and visuomotor control. Drivers could mitigate risk by increasing following distance from the lead vehicle. All the subjects travelled at approximately 89 km/h, but the ecstasy drivers followed 64 m closer to the lead vehicle and demonstrated 1.04 s shorter delays to lead vehicle speed changes than other driver groups. This meant they were taking more risks and had to compensate for this by reacting faster. If distractions had happened, they may not have been able to achieve such a reaction time.

3.5.2.2 Methamphetamine

Methamphetamine is a powerful, highly addictive stimulant that affects the central nervous system. Methamphetamine and driving has been a little researched area of road safety according to Silber et al (2012). The authors could only quote one prior published study specifically on driving under the influence of Methamphetamine (Mitler et al 1993). That study observed a dose related improvement for narcolepsy patients and matched controls on a simple driving task.

Silber et al (2012) quote Lemos (2009); Logan (2001); Ogden and Moskowitz (2004); Silber et al (2006) and Walsh et al (2004) as epidemiological studies which collectively have suggested that methamphetamine affected drivers are subject to inattention, erratic driving, drift in-between lanes, impatient, inappropriate speeds, and general displays of increased risk-taking behaviours.

Silber et al (2012) took 20 healthy recreational illicit stimulant users (10 males, 10 females), aged between 21 and 34 years and subjected them to performance testing sessions on a driving simulator. One featured a placebo and the other was carried out after methamphetamine ingestion. No significant improvement or decrement in the driving performance of the subjects was detected two to three hours after administration. However, when compared with the placebo condition, participants who ingested methamphetamine tended to:

- release the brakes inappropriately when stopping
- drive too fast for the traffic conditions
- travel slower on the freeway in an emergency.

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12 www.drugabuse.gov/publications/drugfacts/methamphetamine
The levels of methamphetamine ingested for this study were relatively low for ethical reasons. It is possible that higher levels may exist in the driving population resulting in greater impairment.

Bosanquet et al (2012) looked at the performance of a group of current methamphetamine users with a group of non-users on a driving simulator. The groups were matched by age, gender and driving experience. The users, most of whom were meth dependent, were significantly more likely to speed and to weave from side to side than the controls. They also performed right turn against movements with a smaller gap than the controls. This type of risky behaviour occurred irrespective of whether the users currently had heightened blood levels of methamphetamine or its metabolite, amphetamine. Blood levels varied widely in the group. This indicated that risky behaviour may sometimes be a characteristic of being a methamphetamine user, rather than recent ingestion of methamphetamine.

Stough et al (2011) tested subjects in a simulator after they had ingested methamphetamine. Compared to a placebo, performance on the stopping brake measure was significantly decreased from that observed with the placebo, three hours post drug. The other decrements related to ecstasy which Stough et al (2011) observed (See 3.5.2.1) were not observed. The authors attributed this to the greater stimulant effects of small doses of methamphetamine.

Ramaekers et al 2006 reported that a dose of 75mg MDMA improved road tracking accuracy, but impaired speed adaptation during car following in a simulator, as measured by an increase in “overshoot” when responding to the lead vehicle’s decelerations.

### 3.6 Conclusions and observations

- Evidence for risks associated with drugged driving comes primarily from case-control studies and, culpability studies (which provide estimates of the risk of case group of drugged drivers compared with a control group) and behavioural studies using driving simulators.

- The case control studies and culpability studies differ in the risks they portray depending on whether the controls are a sober group or a group like all drivers from a roadside survey, which already contains a proportion of drugged drivers. Similarly, the behavioural studies differ in the drug use, outside the study, of their sober control groups. Some behavioural studies use control groups of sober drug users, who may have risk-taking behaviours related to their drug use, even when temporarily sober. Also, sober drug users may be a different group from the driving population.

- The three drugs under consideration do not have well-defined dose response relationships with crashes, such as that of alcohol.
  - Looking at the studies yields a conclusion that the relative crash risk of cannabis with respect to sober driver is around 1.5 for lower doses of cannabis and around 2 for higher doses of cannabis. This is based primarily on the conclusions of Røgeberg and Elvik (2017)
  - There are no specific estimates of the increased risk related to ecstasy and methamphetamine available at the time of writing. Studies of the larger groupings of stimulants and amphetamines have indicated that stimulants as a group and amphetamines as group have a greater risk than cannabis.

For both fatalities and serious injuries alcohol is the riskiest substance (at levels greater than 0.05 gm/dl), followed by stimulants, with cannabis following as moderately risky on its own. Alcohol drug combinations were riskier than drugs in combination or drugs or alcohol singly. All three drugs (cannabis, ecstasy and methamphetamine) form more dangerous combinations with alcohol and other drugs than on their own. There is compelling evidence from behavioural studies and some case-control and culpability studies that
combinations of cannabis and alcohol have a greater risk than either separately. ORs for cannabis and alcohol together have been estimated by a small number of studies. For instance, Laumon et al (2005) estimated an overall O.R. of 14 for the combination. The Druid project (Hels et al 2011; Li et al 2013) estimated O.R.s of over 20 for alcohol combined with drugs generally but did not further disaggregate. As mentioned earlier ‘overall’ alcohol risk is dependent on the average level of alcohol in particular sample which will be very setting-specific. However, the Druid project did have the virtue of producing its overall using data from studies carried out in several countries.

Overall ORs from case control studies indicate that, in general, for combinations of the relevant drugs and alcohol, the odds of being fatally injured, compared to an unaffected control, are considerably higher than the analogous odds for being seriously injured. This does not mean that users of drugs and alcohol are more likely to die than be injured, as the absolute probability of death in the control groups is much lower than the absolute probability of injury in the control groups.

All three drugs impact on a wide range of behaviours in simulated driving. Those discovered from the literature are detailed in table 3.9.

<table>
<thead>
<tr>
<th>Cannabis/synthetic cannabis(^{13})</th>
<th>Methamphetamine</th>
<th>Ecstasy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased reckless driving</td>
<td>Release the brakes inappropriately when stopping</td>
<td>Higher urban speed</td>
</tr>
<tr>
<td>Slower driving, larger headways</td>
<td>Drive too fast for the traffic conditions</td>
<td>Acceptance of smaller gaps</td>
</tr>
<tr>
<td>More signalling errors</td>
<td>Travel slower on the freeway in an emergency</td>
<td>More simulated crashes</td>
</tr>
<tr>
<td>Impaired control of speed, headway</td>
<td>Impaired control of lateral position</td>
<td>More signalling errors</td>
</tr>
<tr>
<td>and lateral position</td>
<td>Execute right turn against movements with a smaller gap</td>
<td>More skidding</td>
</tr>
<tr>
<td>Decreased car control as task demand increases</td>
<td>More inappropriate braking,</td>
<td>More inappropiate braking,</td>
</tr>
<tr>
<td>Decreased performance on road tracking tasks</td>
<td>Less safe following distances</td>
<td>Less safe following distances</td>
</tr>
<tr>
<td>Decreased psychomotor skills, reaction time, visual functions, attention and encoding</td>
<td>More aggressive driving</td>
<td>More aggressive driving</td>
</tr>
</tbody>
</table>

- Cannabis users may try to mitigate their impairment by not overtaking, slowing down and focusing their attention in anticipating an expected event for which a known response is required. Of course, this cannot happen when an event is unexpected.

- Ecstasy and methamphetamine are both stimulants and can have indirect impacts by assisting people to become exhausted. If such people then drive, they are more susceptible to fatigue related crashes. These indirect impacts on fatigue are separate from the drugs’ direct impacts.

- In terms of direct impacts, although as stimulants, the drugs may decrease fatigue related driving errors, generally, users of both ecstasy and methamphetamine exhibit overall decremented driving ability, although studies differ on the size and nature of the decrements.

- The drugs appear to increase driving aggression, but the aggression may be, at least partly, an attribute of the user group rather than just drug ingestion.

\(^{13}\) No simulations have been found using synthetic cannabis. However, synthetic cannabis is generally stronger than plant derived cannabis, and may contain an unpredictable mix of ingredients with unpredictable impacts. Therefore, its impact on simulated driving can be expected to be similar, but more severe than that of plant derived cannabis.
Deterrence takes two forms, general deterrence and specific deterrence. General deterrence refers to the impact of enforcement on those not directly affected by the enforcement, such as those who hear about a campaign by word of mouth, through public education campaigns, the media etc. Specific deterrence refers to those directly affected and includes drivers who have been tested, their passengers and those who go past a checkpoint without being tested. There is a place for both types of deterrence in Road Policing, but widespread societal change is unlikely without large-scale sustained general deterrence operations accompanied by wide supportive public education (Homel and Berger 1987; Davey and Freeman 2011).

Classical deterrence theory is based on the notion that law breaking is inversely related to the perceived certainty, severity and swiftness of punishment (Davey and Freeman 2011). Note that this refers to punishment rather than just apprehension so to achieve deterrence apprehension must be perceived as both certain and resulting in punishment. More recently this concept has been expanded to include non-legal sanctions like social disapproval, feelings of guilt, fear of physical injury. These considerations may be included in enforcement support advertising campaigns (Davey and Freeman 2011).

There are also differences of opinion regarding the necessity for a high severity of punishment. The American Sociologist, Laurence Ross (Ross 1994) contends that:

\[\text{well-publicized law-enforcement campaigns aimed at increasing the certainty and swiftness of punishment are more successful than highly punitive laws that the target population does not expect to be applied.}\]

This approach has a contemporary application in the wider criminal justice system in the Swift, Certain and Fair Sanctions programme which has been implemented in the UK and Australia and is under consideration by Corrections in New Zealand\(^{14}\). Under this approach, sanctions should be severe but also fair.

General deterrence, in the context of Road Policing, reached Australasia with the advent of random breath testing (RBT) in Australia in the 1980s and became used in New Zealand with the advent of compulsory breath testing (CBT) in 1993. The principle of general deterrence is enunciated in Homel (2004, 1993, pp 244–245) as:

\[\text{influencing the behaviours of large numbers of motorists by increasing their fear of the legal consequences of violating the law.}\]

Roadside drug enforcement may be aimed only at a specific target or at a wider target with general deterrence achieved through public education and conspicuousness of relevant Police operations. This will also need to be continuously reinforced through supporting publicity. Otherwise its effectiveness will degrade over time. This is well described in the following statement from Homel (1998), quoted in Homel (2004, 1993). The quote is about RBT for alcohol but would also apply to saliva testing for drug driving.

\(^{14}\text{www.corrections.govt.nz/resources/newsletters_and_brochures/journal/volume_4_issue_1_august_2016/swift%2C\_certain\_and\_fair\_sanctions\_an\_innovative\_new\_programme\_or\_false\_hope.html}\)
4 Deterrence

RBT is always in the process of losing its effectiveness among drivers who, because they feel under pressure to drink or because they have not seen RBT in operation for some time, take the risk of driving after drinking.

The sorts of influences which can degrade the effectiveness of attempts at general deterrence were found in a survey of 516 Queensland motorists perceptions of legal (certainty, severity and swiftness) and non-legal (social, internal and physical) sanctions associated with drug driving, (Davey et al 2008) The survey also looked at the impact of random roadside drug testing and non-legal sanctions on intentions to drug drive. Respondents considered legal sanctions to be certain and severe but not swift. For non-legal sanctions, most reported concerns like ‘losing friends’ respect’ and physical injury. Low certainty of apprehension was significantly associated with self-reported intention to offend in the future. A group of 45 convicted drug offenders was also found to be significantly more likely to drug drive in the future than a matched group of ordinary motorists. The authors recommended measures to increase perceptions of the certainty of detection within a deterrence context with supportive public education campaigns, and suggested thought be given to emphasising the non-legal consequences of drug driving like personal injury or peer loss.

It is an important part of Police tactics to balance enforcement effort between general deterrence, specific deterrence and the various means of carrying out drug enforcement within general and specific deterrence. Evidence from Waikato University (Starkey and Charlton 2017) indicates that levels of enforcement in New Zealand are insufficient to produce widespread general deterrence. The authors found that 60% of drivers in a survey considered apprehension for drink driving by Police was likely but only 26% apprehension was likely for drug driving. Therefore, we can take present levels as a base which requires improvement, and if there is to be a greater deterrent effect, the perceived apprehension risk needs to be higher.

Apart from the Victorian information outlined later in this report there is little information available on the effectiveness of real deterrence campaigns. There is however information from Australia and New Zealand on the deterrent impact of campaigns in other realms of road safety.

In December 1982, RBT similar to the well-known New Zealand CBT was introduced in New South Wales (Homel 2004, 1993). The initiative supported by extensive, high-profile advertising and the enforcement was vigorous, with a ratio of one test per three licensed drivers per year. In later years this ratio of one to three was further improved. As recounted by Homel (2004, 1993) there was an immediate 22% reduction in all fatal crashes and a 36% drop in alcohol-related fatal crashes, compared with the previous three years. The impact was sustained for almost 10 years with only occasional temporary hints of diminished impact.

Prior deterrence experience in New Zealand includes a Supplementary Road Safety Package (SRSP) introduced in 1995. The SRSP increased existing CBT and speed camera programmes introduced in 1993 and introduced high-profile enforcement support advertising aimed at improving speeding behaviour, drink driving behaviour and safety belt use. In 1995/1996, annual road deaths for the two years before the SRSP were stable (at around 580). However, post SRSP, by the year 2000, annual road deaths had reduced to 462. Analyses (Guria and Leung 2004) indicated that over this period 285 lives were directly saved by the package.

4.2 Targeting to risk

4.2.1 Its application in joint operations with CBT

It is clear from earlier sections that drugs increase drivers’ risks relative to other drivers and that combinations of drugs carry more risk than drugs alone. The most important combination is cannabis and
alcohol, mainly because of the use of high doses of alcohol which has a relatively steep dose response curve and provides a springboard for the added impact of other drugs, notably cannabis. For cannabis and alcohol, the risks appear to be additive as indicated earlier from case control studies and culpability studies. Therefore, it is safe to assume that people who drive after using both alcohol and cannabis are high-risk individuals and should be targeted similarly to the way common road deficiencies (eg out of context curves) are targeted by road safety engineers.

This means that some method of implementing this in conjunction with random breath testing (RBT) is required. One such method is that used in Victoria between 2005 and 2009.

Cameron (2013) discusses the impact of the Victorian roadside oral fluid testing programme (ROFT) as it was between 2005 and 2009. According to Cameron (2013) the type of drug testing regime in place in Victoria at the time was as follows:

- Most ROFTs in were carried out at bus-based testing stations (drug/booze buses) in conjunction with RBT and usually following an initial preliminary breath alcohol test.
- Drivers were initially tested at the roadside by a tongue swipe.
- If this test was positive to one or more of the proscribed drugs, oral fluid was collected from the driver and tested using a second device.
- If this test was also positive, the driver would be banned from driving for 24 hours and an oral fluid sample was sent for confirmation by laboratory analysis.
- Where the driver was unable to provide sufficient oral fluid, he or she was required to provide a blood sample for laboratory analysis.

It is not clear if only those positive to alcohol in the screening test were tested. If this was the protocol, then random drug testing in Victoria amounted to testing those who were alcohol positive at the bus-equipped RBT checkpoints with the ability to process the drug tests.

Since then, Victoria has introduced a higher penalty for those caught with both alcohol and cannabis in their system to allow for their greater risk. An element of targeting to risk could be used at random testing sites in New Zealand by:

- having a higher penalty for multidrug use including alcohol
- as in Victoria, carrying out drug screening at a subset of alcohol screening sites specially equipped for drug screening. This includes having a booze bus in Victoria
- screening for alcohol first and imposing the higher penalty on offenders also caught with both alcohol and drugs.

This could be carried out in parallel with targeted testing where appropriate. Victoria has a history of testing target groups near places where they congregate. Haworth and Lenné (2007) describe targeted testing of truck drivers and nightclub patrons.

4.2.2 When should it be carried out?

Logically it should be carried out to maximise general deterrence and to target risk. This means locations and times should be as widely spread and as unpredictable as possible. This will increase the perception in the public mind that the Police are “out there” taking drug deterrence seriously. The times should also

15 Personal communication, Professor Maxwell Cameron, 21/6/2019
16 Personal communication, Professor Maxwell Cameron, 21/6/2019
Deterrence

be biased towards times of day when drugged driving is most likely to take place and when other factors (primarily high alcohol use) makes the use of drugs riskier. Not much information on times of high drugged driving information is available in New Zealand. However, a useful source of overseas information is the US National Roadside Survey (NRS)\textsuperscript{17}. This samples the blood and saliva of a structured sample of American drivers for drugs and alcohol.

Table 4.1 shows the percentage of drivers with drugs and alcohol detected in the survey of 2013–2014, during day and night collection periods.

Table 4.1 The percentage of drivers with drugs and alcohol detected in the US NRS of 2013–2014, during day and night collection periods.

<table>
<thead>
<tr>
<th></th>
<th>Illegal drug percentage</th>
<th>Alcohol percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weekday daytime</td>
<td>9.3</td>
<td>1.1</td>
</tr>
<tr>
<td>Weekend night -time</td>
<td>13.2</td>
<td>8.3</td>
</tr>
</tbody>
</table>

This indicates that drug enforcement should concentrate most on night-time when the greater part of drugged driving takes place and when alcohol, which forms a dangerous combination with most drugs, is consumed heavily. There should also be an enforcement presence in the day when alcohol use is relatively low, but illegal drug use still 9.3% in the US. There should also be a high weighting for weekend nights when both alcohol driving and drug driving peak.

4.3 Quantification of the impact of deterrence

4.3.1 Cameron’s method

The impact of deterrence is obviously very situation specific, and Police have many tactics for achieving deterrence. Under the 2005–2009 Victorian regime described above drivers screened increased each year from 13,158 in 2005 to 27,883 in 2009 with the detection rate of proscribed drugs falling from 2.3% to 1.0% over the same period (Boorman 2010), after having increased from 2001 to 2005. This meant a trend reversal associated with random drug testing (RDT)\textsuperscript{18}. Figure 4.1 from Cameron (2013) illustrates relationships between drivers killed with drugs in their system and the numbers of drivers screened for drugs.


\textsuperscript{18} Since then Victoria Police have moved to around 150,000 tests per year, backed up by stronger publicity in order to gain a greater general deterrence impact (personal communication, Professor Maxwell Cameron, 21/6/2019.)
Figure 4.1 suggests a positive impact with diminishing returns, a relationship also found for RBT, for which much more data is available. From figure 4.1, Cameron concluded that by testing 1.24% of licensed drivers per year 15.2% percent of current driver fatalities could be avoided. If applied to New Zealand this would mean in round figures 40,000 tests to save 15.2% of driver road deaths (or 32 deaths) in 2018. That corresponds to around one driver death saved per 1,000 tests. There would be additional vehicle occupant deaths saved as well. As made clear by Cameron (2013) the reader should note that this figure is based on limited Victorian data. For it to apply in New Zealand, similar roadside testing procedures to those used by the Victorian Police at the time would need to be followed here. It also assumes that if the driver who was killed had drugs in their system, the drugs were responsible for the crash. In actuality, crashes normally have several contributory factors and the drugs may have been or may not have been crucial. Therefore, this estimate may be optimistic.

4.3.2 A method based on relative risks

Another way of attacking this problem is to look at relative risks. As mentioned earlier, Starkey and Charlton (2017) found that 2.55% of respondents reported using cannabis on its own within three hours of driving (telephone survey) as did 14.2% from a younger sample via an internet survey. Of those who reported taking cannabis within three hours of driving, for both surveys, around 40% reported doing so once a week or more often during the year.

As an illustration, 8.4% (the arithmetic mean of the two results above) could be taken as an estimate of the percentage of drivers who have taken cannabis on its own within three hours of driving, and further that these drivers are impaired in some way. Let us further assume that their relative risk of serious crash is 1.5 when they have taken cannabis on its own within three hours of driving, ie their risk of serious crash in a year is 50% more than the average New Zealand driver. Also assume that 50% of their driving is within three hours of taking cannabis. This brings their relative risk down to 1.25.
This makes the average risk of the driving population, compared with a population not using cannabis within three hours of driving to be as shown below:

Driving Pop\(n\) risk \(=1^*\left(1-0.084\right) + 1.25^*.084= 1.02\)

This would mean that these cannabis-affected drivers would increase the national risk of fatal or serious crashes by approximately 2%. Therefore, if they all became sober via deterrence, the 2,452 fatal and serious crashes that occurred in 2018 would reduce to \((2,420/102) \times 100=2,373\), a saving of 47 fatal and serious crashes.

If we assume the deterrence efforts convince, say, 30% to become sober\(^{19}\), then we would save around 15 fatal or serious crashes.

The Victorians target the more dangerous combination of alcohol and cannabis by testing for cannabis those positive on an alcohol screen. Starkey and Charlton (2017) found that 15.38% reported cannabis/alcohol use within three hours of driving (telephone survey), as did 36.84% from a younger sample via an internet survey.

As an illustration, 26% (the arithmetic means of the two results above) could be taken as an estimate of the percentage of drivers who have taken cannabis with alcohol within three hours of driving, and further that these drivers are impaired in some way. Let us further assume that their relative risk of serious crash is 14 (Laumon et al 2005) when they have taken cannabis/alcohol within three hours of driving, ie their risk of serious crash in a year is 14 times more than that of the average New Zealand driver. Also assume that 50% of their driving is within three hours of taking cannabis in combination with alcohol. This brings their relative risk down to 7.

This makes the average risk of the driving population, compared with a population not using cannabis/alcohol within three hours of driving to be as shown below:

Driving Pop\(n\) risk \(=1^*\left(1-0.26\right) + 7^*.0.26= 2.56\)

This would mean that these cannabis/alcohol-affected drivers would increase the national risk of fatal or serious crashes by approximately 160%. Therefore, if they all became sober via deterrence, we would reduce the 2,452 fatal and serious crashes that occurred in 2018 to \((2,420/2.56) \times 100=945\), a saving of 1,475 fatal and serious crashes.

If we assume the deterrence efforts convince, say, 30% to become sober, then we would avoid 442 fatal or serious crashes. Table 4.2 depicts the impact at a range of sobriety levels which would correspond to varying levels of deterrence.

\(^{19}\) There is little information on which to base such an assumption. Cameron (2013) reports that from 2005 to 2009 an expanding RDT programme in Victoria coincided with a reduction in the detection rate of proscribed drugs from 2.3% to 1.0% – a 56% reduction. The 30% reduction assumed here is based on this figure arbitrarily discounted in the interests of conservatism.
Table 4.2 The impact at a range of sobriety levels which would correspond to varying levels of deterrence

<table>
<thead>
<tr>
<th>Percentage becoming sober</th>
<th>Fatal and serious crash reduction for cannabis only</th>
<th>Fatal and serious crash reduction for cannabis/alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>47</td>
<td>1,185</td>
</tr>
<tr>
<td>80</td>
<td>38</td>
<td>984</td>
</tr>
<tr>
<td>60</td>
<td>28</td>
<td>711</td>
</tr>
<tr>
<td>40</td>
<td>19</td>
<td>492</td>
</tr>
<tr>
<td>20</td>
<td>9</td>
<td>246</td>
</tr>
</tbody>
</table>

This indicates that random drug testing, based on testing those who test positive for alcohol in the screening test at CBT sites could be an effective strategy in conjunction with some testing of those who test negative for alcohol. The above illustrative analysis is for cannabis, but the basis of it would also hold for ecstasy and methamphetamine.

4.3.3 Optimal testing levels

Several different testing regimes and levels have been carried out in Victoria and other jurisdictions over time, with little consensus about where diminishing returns occur. There is no acceptably accurate method to work out a crash saved per test ratio or a crash saved per positive test ratio. Of course, the number of crashes saved is not just dependent on the number of tests but also the publicity back up unless the only interest is in specific deterrence, which is a very sub-optimal use of the road safety dollar.

4.4 Conclusions and observations

- Effective deterrence requires a highly visible general deterrence component, backed up with supportive public education along with specific deterrence when the risk of a subgroup of the population is supportive of such action. The impact of non-legal consequences should be a component of supporting public education.

- The results of being caught should be perceived as swift, certain and severe but should not be perceived as unfair. The impact of non-legal consequences should be a component of supporting public education.

- Owing to the extra risk associated with combinations of drugs with alcohol, a significant component of drug testing should be aimed at those who test positive for alcohol. This does not need to be at the legal limit for alcohol, as any combination has the potential for a concerning increase in risk although, of course higher doses of alcohol will always lead to greater impairment.

- The impact of deterrence is hard to predict as it depends among other factors on the minutiae of Police tactics and the resources available to them. There would be an appropriate blend of general and specific deterrence applicable uniquely to each situation, under the overarching deterrence principles enunciated above.

- Similarly, optimal testing levels are hard to quantify. However, it can be said that any successful campaign will include a substantial general deterrence component.
5 Overall conclusions

All three drugs have a deleterious impact on the driving of users. This may be an acute impact from recent ingestion of the drug or a long-term impact from being a regular user of the drug or both.

For both fatalities and serious injuries alcohol is the riskiest substance (at levels greater than 0.05 gm/dl), followed by stimulants, with cannabis following as moderately risky on its own. Alcohol drug combinations are riskier than drugs in combination or drugs or alcohol singly.

The combination with alcohol is the most important because of the relative common nature of drink driving and the fact that alcohol combinations are generally more potent than those with other drugs.

Ecstasy and methamphetamine may indirectly impact on road safety by assisting users to become exhausted after which they may crash through driving in an exhausted state. Potential examples are ‘rave’ goers and commercial drivers.

Synthetic cannabis is an emerging threat because it is normally an unpredictable and dangerous cocktail of chemicals, of which THC may or may not be one. It should not be confused with plant-derived cannabis and generally has a greater impact. There have been several deaths from this lately in different settings. It is not clear to what extent these potentially diverse, and possibly unique, ingredients are able to be picked up by a saliva test.

Deterrence of driving after drug ingestion is a crucial tool in combatting the harmful impact of this practice on road safety. To be successful this should include general deterrence operations to deter the practice at a societal level in addition to targeted operations aimed at groups known to be at high risk of offending. To achieve a societal impact the deterrence operations should be backed up with supportive public education.
6 References


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