Recommending statutory limits
For drug concentrations relating to impaired driving

April 2021
Independent Expert Panel on Drug Driving

Final Report
Recommending Statutory Limits for Drug Concentrations Relating to Impaired Driving

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Contents

1 Background ................................................................................................................................. 5
2 Executive summary ...................................................................................................................... 5
3 Limitations .................................................................................................................................. 6
4 Introduction .................................................................................................................................. 8
5 Terms of Reference ...................................................................................................................... 10
  5.1 Setting criminal drug limits based on impairment equivalence to 80 mg/100 mL blood alcohol ---- 12
  5.2 Blood drug limits set by other jurisdictions ........................................................................ 13
  5.3 International approaches to setting concentration thresholds for drug driving. .................. 13
  5.4 Setting per se thresholds ...................................................................................................... 14
  5.5 Applying low-level threshold testing to blood and oral fluids – the use of fluid testing devices ---- 15
6 Drugs that can impair driving .................................................................................................... 16
  6.1 ‘Recreational’ Drugs ............................................................................................................ 17
  6.2 Prescription Drugs ................................................................................................................ 18
7 Overview of drug use by NZ drivers .......................................................................................... 20
  7.1 Deceased drivers ................................................................................................................... 21
  7.2 Drug impaired (failed CIT) drivers ....................................................................................... 22
  7.3 Hospitalised drivers .............................................................................................................. 22
  7.4 Drunk drivers ...................................................................................................................... 23
  7.5 Multiple drug use by drivers ............................................................................................... 23
8 Detection of drugs in oral fluid .................................................................................................. 26
  8.1 Oral fluid detection devices ................................................................................................... 28
  8.2 Consideration of oral fluid on-site cut-offs compared with recommended per se blood drug concentrations ................................................................................................................................. 32
9 Individual Drugs and Drug Classes .......................................................................................... 34
  9.1 Cannabis ................................................................................................................................ 34
    9.1.1 Blood THC concentrations following inhalation ............................................................. 35
    9.1.2 Blood THC concentrations and impairment .................................................................. 37
    9.1.3 Oral fluid and blood THC concentrations from various exposure routes ................…… 40
Inhalation ....................................................................................................................................... 40
Passive exposure .......................................................................................................................... 41
Ingestion of cannabis edibles ........................................................................................................ 42
Hemp-based food ingestion ......................................................................................................... 43
Cannabis as a medicine ................................................................................................................ 44
Chronic cannabis use .................................................................................................................. 45
  9.1.4 Duration of detection of cannabis use in biological samples ................................................. 45
  9.1.5 Blood THC concentrations in NZ drivers ........................................................................ 46
List of Tables

Table 1  Recommended blood concentration threshold and criminal limits completed with corresponding limits for other jurisdictions
Table 2  Cut-off drug concentrations in oral fluid as set out in AS/NZS 4760:2019
Table 3  Detection limits of drugs by oral fluid as set out in AS/NZS 4760:2019
Table 4  An assessment of some on-site oral fluid devices
Table 5  Recommended blood concentrations compared with oral fluid on-site devices
Table 6  Oral fluid THC concentrations in following passive exposure in an enclosed room
Table 7  Comparison of oral fluid THC concentrations following oral ingestion and smoking cannabis
Table 8  Occurrence of cannabis taking and THC concentrations in deceased NZ drivers
Table 9  Sedative drugs detected in blood of deceased NZ drivers in descending order of occurrence
Table 10 Statutory limits for sedatives in the UK, Norway and Denmark compared with limits proposed in this report
Table 11 Opioids detected in blood of impaired and hospitalised driver in NZ
Table 12 Statutory limits for opioids in the UK, Norway and Denmark compared with limits proposed in this report

References

Appendices

Appendix 1  Glossary and abbreviations
Appendix 2  Terms of Reference for the Independent Expert Panel on Drug Driving June 2020
Appendix 3  Revised Terms of Reference for the Independent Expert Panel on Drug Driving October 2020
Appendix 4  Per se blood drug limits in international jurisdictions
Appendix 5  Excerpts from reports by international expert panels on drug driving
Appendix 6  International oral fluid drug concentration cut-offs
Appendix 7  Summary of data used during consideration of recommended per se limits
Appendix 8  Analytical Issues
Appendix 9  Consideration of the age of a driver
1 Background

This is the final report from the Independent Expert Panel on Drug Driving (the Panel). It brings together our thinking on alcohol, cannabis and methamphetamine, and other (including prescription) drugs in an impaired driving context. In this report, we offer advice to ministers on what we consider appropriate blood drug concentrations at both infringement and criminal levels – it is anticipated that the latter will be specified in the legislation.

2 Executive summary

The Panel has reviewed the published scientific literature, taken account of New Zealand data from drug-related car crashes, and referred to drug driving limits set by other jurisdictions to recommend statutory limits (infringement and criminal) for New Zealand’s legislation.

Twenty-six drugs were selected for consideration (see Table below) based on NZ data linking road traffic accidents with the presence of the drugs in drivers’ blood samples; they include, recreational drugs (e.g., methamphetamine), opioids (e.g., morphine) and sedatives (predominantly benzodiazepines; e.g., diazepam).

It was hoped to relate the recommended drug statutory limits to a level of driving impairment commensurate with the criminal limit for blood alcohol (80 mg/100 mL). This was not possible because there are insufficient data available to associate the concentration of a particular drug in blood with its effect on driving.

Because it is not possible to set limits for drugs based on an equivalent impairment effect to the alcohol criminal limit, the Panel based its recommendations on the blood drug concentrations in NZ drivers, its interpretation of the scientific literature, and consideration of statutory limits set by other countries.

Commercially available roadside oral fluid testing devices were assessed in terms of their applicability to the limits recommended in this report. Since there is a poor correlation between oral fluid and blood drug concentrations, laboratory confirmation of blood concentrations is recommended following a positive oral fluid screen.

Not all of the 26 drugs considered by the Panel can be detected by commercially available oral fluid testing devices. The panel concluded that it would not be cost-effective to seek bespoke manufacture of devices for application to the drug concentrations recommended in this report.
The table below summarises the Panel’s recommendations for threshold (infringement) and criminal (statutory) limits for drugs.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Criminal (Statutory) Limit</th>
<th>Threshold ng/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>THC (Cannabis)</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>100</td>
<td>20</td>
</tr>
<tr>
<td>MDMA</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>MDA</td>
<td>No limits proposed</td>
<td></td>
</tr>
<tr>
<td>Cocaine</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>GHB</td>
<td>50,000 (50 μg/mL)</td>
<td>10,000 (10 μg/mL)</td>
</tr>
<tr>
<td>Ketamine</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>Alprazolam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Diazepam</td>
<td>200</td>
<td>100</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Midazolam</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>Temazepam</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>Triazolam</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Zopiclone</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Codeine</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Dihydrocodeine</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Methadone</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Morphine</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Tramadol</td>
<td>250</td>
<td>100</td>
</tr>
</tbody>
</table>

3 Limitations

It is well recognised that many drugs can adversely impact the ability of people to drive safely. This is based on empirical evidence derived from drivers who are stopped and undergo a Compulsory Impairment Test (CIT), and drivers who are involved in road traffic accidents, and have these drugs in their biological fluids. There is also very limited evidence in which volunteers in scientific trials have taken drugs of interest and then had their ability to drive assessed (e.g., using a simulator) for impairment.

Numerous prescription and over-the-counter medicines have the potential to impact driving and this is recognised by the inclusion of a warning label. In some situations, these prescription drugs are used recreationally or inappropriately. In other situations, there are drugs that are used recreationally, that have no current therapeutic use, but have ability to impair driving.
In determining concentration thresholds, the panel has used data from the scientific literature, considered statutory limits in overseas jurisdictions and used New Zealand (NZ) data on drug blood concentrations in impaired drivers, to develop its advice. However, it must be noted that despite the available evidence, there is a paucity of studies that have directly addressed the issue of driving impairment after a given dose of a drug, let alone the complex relationship between the dose and the concentration in biological fluids that can be sampled routinely and used as forensic evidence (oral fluid and blood).

In contrast to controlled use, when used recreationally, there are many parameters that influence the predictability of the drug’s effect: the dose of the drug may not be accurately known; drugs may be administered by various routes that can impact the rate and amount of drug absorbed and distributed so that the relative concentrations in oral fluid, blood and brain are not consistent. This is important because the concentration of the drug in the brain determines its activity, and this is not necessarily predicted by oral fluid or blood drug concentrations. In addition, multiple doses at irregular times can also impact the concentrations measured, especially if processes such as drug metabolism and excretion become saturated.

With regard to the effect of any given drug dose on driving impairment, this may be dependent on whether the person is naïve to the drug (as is often the case in scientific studies) or has been exposed to the drug chronically. Not only are some of these processes affected by biological factors such as age, body size, genetics and disease, they may also be affected by concurrent exposure to one or more other drugs (e.g., by competition for metabolic processes).

The drugs covered in this report include stimulants, sedatives and hallucinogenic substances: it is impossible with current knowledge to predict with any certainty the impact that taking several of these drugs would have on driving impairment, although it is generally accepted that taking more than one drug would have a greater detrimental effect.

Terms, abbreviations and units used in this report

The International System of Units (Système International d'Unités ; SI) has been used throughout this report. The units, their symbols and definitions are shown in the table below.

<table>
<thead>
<tr>
<th>Unit</th>
<th>Symbol</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kilogram</td>
<td>kg</td>
<td>1,000 or $10^3$ g</td>
</tr>
<tr>
<td>Gram</td>
<td>g</td>
<td></td>
</tr>
<tr>
<td>Milligram</td>
<td>mg</td>
<td>1/1,000 or $10^{-3}$ g</td>
</tr>
<tr>
<td>Microgram</td>
<td>μg</td>
<td>1/1,000,000 or $10^{-6}$ g</td>
</tr>
<tr>
<td>Nanogram</td>
<td>ng</td>
<td>1/1,000,000,000 or $10^{-9}$ g</td>
</tr>
<tr>
<td>Time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day</td>
<td>d</td>
<td>24 h</td>
</tr>
<tr>
<td>Hour</td>
<td>h</td>
<td></td>
</tr>
<tr>
<td>Minute</td>
<td>min</td>
<td>1/60 h</td>
</tr>
<tr>
<td>Volume</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Litre</td>
<td>L</td>
<td></td>
</tr>
<tr>
<td>Millilitre</td>
<td>mL</td>
<td>1/1,000 or $10^{-3}$ L</td>
</tr>
</tbody>
</table>
4 Introduction

There are two basic types of law for dealing with impaired drivers. The first is an assessment of impairment by a qualified individual whereby a driver suspected of driving under the influence of drugs undergoes a physical test to determine if that driver is safe to drive a vehicle. Such a regime has been in place for drug impaired drivers in NZ since December 2009. The second is known as per se law whereby it is an offence to drive a vehicle with a concentration of alcohol or other identified drugs in the blood above a specified threshold. This has been in place for alcohol in NZ for decades. Per se concentrations for drugs are considered a more effective means of dealing with drug impaired drivers.

There are many drugs that can adversely affect driving ability whether used recreationally or medicinally. The drugs recommended by the Panel for inclusion in per se legislation are those widely acknowledged as having significant risk in relation to safe driving as determined by evidence collected in NZ, overseas studies, and inclusion in per se legislation overseas.

The drugs selected for inclusion in per se legislation in NZ include methamphetamine, amphetamine, methylenedioxyamphetamine (MDMA), cocaine, tetrahydrocannabinol (THC, the main psychoactive constituent of cannabis), gamma-hydroxybutyrate (GHB) and ketamine. Additionally, some opioid and sedative medicines have been included. The opioids are buprenorphine, codeine, dihydrocodeine, fentanyl, methadone, morphine, oxycodone and tramadol. The sedatives are alprazolam, clonazepam, diazepam, lorazepam, midazolam, nitrazepam, oxazepam, temazepam, triazolam and zopiclone.

The Panel has been asked to provide objective advice for the Associate Minister of Transport and the Minister of Police (together termed Joint Ministers) and make non-binding recommendations on the following topics:

- the ‘blood-drug’ limits to be specified in legislation (criminal limits),
- the low-level tolerance thresholds to be applied to the detection of drugs in blood (blood thresholds), and
- the cut-off thresholds to be included in oral fluid testing devices (oral fluid thresholds).

In preparing advice on criminal limits, the Panel has based its recommendations for criminal limits on:

- limits set in other jurisdictions,
- drug concentrations in impaired drivers in NZ, and
- data from the scientific literature.

The Panel has considered whether its recommendations align with the outcomes sought by Ministers whereby the criminal limits and blood and oral fluid thresholds should support the Government’s policy intent and desired outcomes for the regime thus:

- Criminal limits should be such that there is a high level of confidence that the individual is impaired.
• Criminal penalties are only applied where the drug is at a concentration likely to impair driving.
• The blood thresholds should be such that there is a high level of confidence that the individual has recently consumed the drug.
• The blood thresholds should not penalise drivers who have been accidentally or passively exposed to drugs.
• The Ministers expect that drivers taking normal prescription amounts should not be detected.

With regard to the drugs selected for inclusion in the per se legislation the following should be noted:
• The opioids and sedatives will impair driving skills with normal therapeutic use and patients prescribed these drugs should be warned not to drive.
• Not all of the drugs selected can be detected by the oral fluid testing devices currently available.
• The use of any of these drugs in combination with alcohol, or in combination with each other, will likely result in enhanced impairment.
• While there are reports from overseas jurisdictions that abuse of prescription medicines such as fentanyl and ketamine is common, there is no evidence that these are currently being abused by NZ drivers.

The Panel has considered blood concentration limits for the drugs in a number of ways, dependent on the drug:
• Where drugs are typically used for recreational purposes, the limits have been based on data from the scientific literature, the concentrations determined in NZ drivers and the limits set in overseas jurisdictions.
• For prescription medicines, the criminal limits have been determined by considering what dose of the drug is known to impair, the maximum dose of the drug that may typically be prescribed, and the blood concentrations expected from such a dose.

Table 1 gives the Panel’s recommended blood concentrations for criminal and threshold levels. These are compared with the range of values used in other jurisdictions. Many jurisdictions have per se limits for a smaller selection of drugs than proposed by the Panel. Some jurisdictions have graduated blood drug limits that are associated with increasing penalties.
### Table 1  Recommended blood concentration threshold and criminal limits compared with corresponding limits for other jurisdictions.

<table>
<thead>
<tr>
<th>Drug Type/Drug</th>
<th>Recommended Blood Concentration</th>
<th>Blood Concentrations Set by Overseas</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Criminal Limit</td>
<td>Threshold</td>
</tr>
<tr>
<td>Recreational</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amphetamine</td>
<td>100</td>
<td>20</td>
</tr>
<tr>
<td>Cocaine</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>GHB</td>
<td>50000</td>
<td>10000</td>
</tr>
<tr>
<td>Ketamine²</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>MDMA</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>THC</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Opioids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Codeine</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Dihydrocodeine</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Methadone</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Morphine</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Tramadol</td>
<td>250</td>
<td>100</td>
</tr>
<tr>
<td>Sedatives</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alprazolam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Diazepam</td>
<td>200</td>
<td>100</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Midazolam</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>Temazepam</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>Triazolam</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Zopiclone</td>
<td>50</td>
<td>20</td>
</tr>
</tbody>
</table>

¹Data from Norway, Denmark and the UK [1-3]
²Ketamine is frequently administered by medical personnel to drivers injured in a crash
³50 µg/mL
⁴10 µg/mL
⁵10.3-123.6 µg/mL
x = No concentration set

The Panel recommends that the list of drugs in this table should be viewed as a live document with the option of adding drugs or amending levels should new data on the measurement of impairment become available.

## 5 Terms of Reference

The Panel was appointed in May 2020. It was tasked with advising Ministers on blood and oral fluid concentration thresholds associated with driving impairment for an array of drugs, with a view to incorporating the values in legislation for a compulsory random roadside oral fluid testing scheme (Land Transport (Drug Driving) Amendment Bill 2020 (317-1)).
The main points of the Terms of Reference for the Panel, a document provided June 2020 (see Appendix 2) are outlined below.

The Panel’s Terms of Reference reflect important components of the design of the roadside testing scheme, including:

- The ‘blood-drug’ limits to be specified in legislation based on drug concentrations in blood that align with impairment equivalent to a blood-alcohol concentration of 80 mg of alcohol per 100 mL of blood.
- The low-level tolerance thresholds to be applied to the detection of drugs in blood by the Institute of Environmental Science and Research.
- The cut-off thresholds to be included in oral fluid testing devices (noting that this will require alignment with the procurement process by Police and the technical limitations of any device procured).
- Any other matters that may be referred to it by Joint Ministers.

After considerable research in the scientific literature and in-depth discussion, the Panel concluded that it is impossible to equate the dose of the above drugs with a particular blood alcohol concentration and its concomitant pharmacological effect(s).

As a result of these deliberations, the Panel requested a set of realigned Terms of Reference that it considers achievable. A summary of the discussion why the original terms of reference were not achievable is given below. The revised terms of reference can be found in Appendix 3.

The revised Terms of Reference were provided in October 2020 and requested that the Panel prepare objective advice for the Joint Ministers and make non-binding recommendations on the following topics:

- the ‘blood-drug’ limits to be specified in legislation (criminal limits),
- the low-level tolerance thresholds to be applied to the detection of drugs in blood by the Institute of Environmental Science and Research (blood thresholds),
- the cut-off thresholds to be included in oral fluid testing devices (oral fluid thresholds), and
- any other matters that may be referred to it by the Joint Ministers.

A number of jurisdictions have already introduced, or are moving towards introducing, legal ‘blood-drug’ limits for recreational drugs and/or medicines, including the United Kingdom, Norway and several jurisdictions in North America. Like alcohol, these limits have been established as a proxy for impairment, based on scientific research about the impairing effects of different doses of drugs. The Panel has considered the findings of these jurisdictions in its deliberations.

Initially, the Panel considered which drugs should have legislative limits. This decision-making process was based on NZ data linking road traffic accidents with the presence of these drugs in the drivers’ blood samples (impaired, hospitalised and deceased driver samples analysed by ESR). Having determined which drugs to consider, the Panel examined the scientific literature, reports of similar expert panels from overseas, and findings from other jurisdictions in order to consolidate international thinking on the subject.
The drugs the Panel concluded should have legislative limits are: THC, methamphetamine, amphetamine, MDMA, cocaine, GHB, ketamine, a selection of opioids – buprenorphine, codeine, dihydrocodeine, fentanyl, methadone, morphine, oxycodone and tramadol, and a selection of commonly used sedatives – alprazolam, clonazepam, diazepam, lorazepam, midazolam, nitrazepam, oxazepam, temazepam, triazolam and zopiclone.

5.1 Setting criminal drug limits based on impairment equivalence to 80 mg/100 mL blood alcohol

A key facet of the Terms of Reference is that ‘criminal limits [should be] based on drug concentrations in blood that align with drink driving measures of impairment, being equivalent to a blood-alcohol limit of 80 mg of alcohol per 100 mL of blood (above which a driver would commit a criminal offence).’ For the reasons outlined below, the Panel consider that this goal is not achievable.

The degree of impairment for a particular drug, including alcohol, is a combination of pharmacodynamics (effects of a drug at its biological targets) as well as pharmacokinetics (the drug’s concentrations over time in blood, oral fluids or other tissues such as brain, where the drug has its effect). Whilst there will be a relationship between the concentration of a drug at its target and its ability to impair driving skills, as well as a relationship between the exposure to a drug and the concentration achieved in a body fluid, there is no simple relationship between the dose of a drug and the resultant impairment of driving.

The drugs of interest (listed above) do not exert their effects at the same pharmacological targets within the body, and even within the same class of drugs, such as the opioids or the sedatives, different members of the same class have different potencies. Thus, they have different pharmacodynamic properties.

The concentration of a drug in blood and oral fluid at any given time is dependent not just on the dose itself, but also factors such as:

- Route of administration
- Time since the last dose
- Cumulative effect(s) of previous doses
- Ability of an individual to eliminate the drug from their body

For example, intravenous administration leads to instantaneous peak blood concentrations, inhalation to a rapid blood peak, whereas oral administration often results in a relatively slow time to blood peak concentration, which can be several minutes to hours after the dose.

In addition, there is exposure to the complete dose immediately if it is administered intravenously, whereas oral doses are subject to a process termed first pass effect, whereby some of the dose is removed from the body before it can reach the target site. For example, a drug taken orally is absorbed from the intestine into blood vessels which go directly to the liver – the liver may metabolise a fraction of the drug (which might make it less active) before it is released into the general circulatory
system. Once the drug enters the circulatory system, its concentration in blood declines as the drug distributes into tissues (e.g., the brain) and is eliminated from the body (e.g., by further metabolism and excretion in urine). The mechanisms for absorption, distribution, metabolism and elimination for the drugs of interest vary, thus the pharmacokinetics for each drug will also differ.

It is well accepted that alcohol can affect driving performance and that there is a strong correlation between increasing blood alcohol concentration (BAC) and increased crash risk. The determination of risk associated with alcohol use is possible because of the prevalence of use, legality of the drug, and ease of analysis. This has made it possible to carry out large epidemiological studies of alcohol use and crash risk. The legislative BAC limits are set at a level of crash risk deemed to be acceptable to government.

It is possible that a correlation between increasing blood drug concentrations and increasing crash risk could be determined for the drugs of interest. However, to date there is a paucity of studies due to illegality of the drugs, (in)frequency of their use and inconsistency of analytical methodology employed to measure blood concentrations. Some published studies report a dose that caused driving impairment, but not the blood concentration of the drug at the time of impairment. For forensic purposes, it is the blood concentration-dependent impairment at the time of testing that is critical, not the dose that the driver took.

The Panel concludes that in the absence of such evidence, it is not possible to align specific blood drug concentrations to a degree of impairment that equates to a BAC of 80 mg/100 mL.

5.2 Blood drug limits set by other jurisdictions

The Panel agrees that it is possible to recommend blood drug concentration limits based on those set by other jurisdictions. However, it is important to understand the basis upon which other jurisdictions have set their legal limits before accepting them.

5.3 International approaches to setting concentration thresholds for drug driving.

Three approaches to setting a concentration threshold for a psychoactive drug in relation to road traffic legislation have been used in other jurisdictions [4-6]:

1. **‘Zero tolerance’ approach**: this equates to a complete ban on the use of a specified drug whilst driving. The ‘zero tolerance’ approach regards any amount of drug detected in a specified body fluid as unacceptable; the limit of detection (LOD; i.e., the lowest amount detectable) of the analytical method used is important for this approach.

2. **Proof of impairment approach**: this uses impairment testing in conjunction with drug analysis.

3. **Per se approach**: this is based on the detection of a drug above a defined cut-off blood (or other biological fluid) concentration.
5.4 Setting *per se* thresholds

*Per se* thresholds can be analytical and set at the laboratory’s LOD (i.e., akin to ‘zero tolerance’) or the threshold can be technical and based on the laboratory’s limit of quantification (LOQ) – this is the amount of drug the laboratory can reliably quantify in a sample.

On the other hand, the threshold can specifically relate to the effects of a drug and can be set to the biological fluid concentration of the drug at which an effect on driving ability has been shown to occur. A ‘lower effect threshold’ can be set at the lowest concentration where an effect on driving has been observed (this accounts for effect variability (e.g., impairment variability)).

A *per se* threshold can also relate to risk. In this case, a blood drug concentration threshold is set at a level which is associated with an unacceptable crash risk. This is the risk-based approach used in NZ to set blood alcohol limits where a BAC of 50 mg/100 mL is associated with a particular crash risk and 80 mg/100 mL is associated with a greater and unacceptable crash risk.

Using the risk approach to set *per se* thresholds requires good estimates of crash risk versus blood (or other biological fluid) drug concentrations. Estimates of crash risk can be obtained from three main sources:

1. Prevalence (and concentrations) of specific drugs or drug classes in biological fluids from drivers who have crashed compared with those who have not crashed.
2. Culpability analysis, where the proportion of culpable drivers using a particular drug is compared with the proportion of drivers not using the drug.
3. Data obtained from databases and registries.

The Panel assessed reports from other expert panels – the *per se* limits for various drugs are tabulated in Appendix 4. Excerpts from reports written by other expert panels are also included in Appendix 5. These reports reinforce the difficulties associated with determining *per se* limits in a scientifically robust manner. None of the other panels have tried to equate the impairing effects of the drugs at a particular blood concentration to the effects of alcohol at a particular blood concentration.

**In brief:**

The Canadian panel (2017) [4] recommended *per se* limits for cocaine, GHB, methamphetamine and THC. ‘Zero-tolerance’ limits were recommended for other illicit drugs.

The UK panel (2013) [5] determined the feasibility of establishing and making recommendations for thresholds using estimates of traffic risk, epidemiological evidence and experimental studies. Their recommendations were accepted only for benzodiazepines. The legislative limits (2015) for medicinal drugs were set to ensure that patients would not be dissuaded from taking their prescribed medication for fear of exceeding statutory limits. For illicit drugs, a ‘zero-tolerance’ limit was set, while making allowance for possible accidental exposure (e.g., exposure to smoke from illicit users).
The Norwegian panel (2010) [6] assessed the effects seen after consumption of drugs by non-dependent individuals. The maximum blood drug concentration determined for an intoxicating dose of a drug was considered equivalent to a BAC of 100 mg/100 mL. This concentration was considered the criminal limit. The blood drug concentration divided by 5 (the scientific rationale for this is unclear) was considered equivalent to a BAC of 20 mg/100 mL and was termed the prohibition limit. *Per se* limits have been used in Norway since 2012.

Legislative limits have been used in Denmark since 2007 [3]. The blood drug concentrations are based on the lower concentration limits typically associated with pharmacological effects as reported in the scientific literature.

The Panel concludes that it would be possible to set legislative limits for non-medicinal drugs taking into consideration those set in other jurisdictions and, where possible, that blood concentrations previously detected in impaired drivers in New Zealand should be taken into account. Furthermore, the Panel considered that it would be possible to set legislative limits for medicinal drugs since patients prescribed a medicine have a defence under New Zealand legislation, which ensures that *per se* limits will target only illicit users of the drug.

Finally, while exceeding the statutory drug limit will likely incur a penalty akin to that associated with driving with a BAC of 80 mg/100 mL or more, it will not be possible to equate impairment associated with the drug at its statutory limit with impairment due to a BAC of 80 mg/100 mL for that individual.

5.5 Applying low-level threshold testing to blood and oral fluids – the use of fluid testing devices

The Panel’s Terms of Reference state that ‘Both the low-level thresholds [are] to be applied to the detection of drugs in blood and the cut-off thresholds [that are] to be included in oral fluid testing devices are intended to be set at levels that avoid penalising drivers who have:

- accidental or passive exposure to drugs,
- low residual levels of a drug in their blood due to previous use but have not recently used drugs,
- consumed standard prescription doses of some medicines.’

To achieve the above it is important to understand the relationship between blood and oral fluid concentrations of drugs and to set the latter in the context of the testing devices’ capabilities.

At a given time, the concentration of a drug in oral fluid is not necessarily the same as the concentration in blood. The ratio of the drug concentration in oral fluid to blood varies according to the route of administration and relates to the time that the drug was last taken. In short, there is often not a simple relationship between blood and oral fluid concentrations for a particular drug at a particular time.

The cut-off drug concentrations for commercially available testing devices are generally aligned to oral fluid concentrations set in Standards. These Standards are most commonly applied to (and were
developed for) workplace safety, and the recommended cut-offs are accepted as indicative of recent drug use rather than historical use or accidental exposure.

The commercially available oral fluid testing devices that are suitable for roadside testing are currently used in several jurisdictions. Their drug concentration cut-offs cannot be reset by the operator (i.e., they are set by the manufacturers). The Panel considers it unlikely that it would be cost effective to commission a specific testing device for NZ-specific oral fluid levels.

The commercially available oral fluid testing devices that detect opiates and benzodiazepines lack specificity and will detect more than one drug in each of the drug classes. This covers a wide range of concentrations of the individual drugs. This makes them unreliable for the detection of some opiate and benzodiazepine class drugs.

The Panel concludes that the cost of specifying oral fluid testing device drug concentration cut-offs is not offset by the benefit, since there is a poor correlation between oral fluid and blood drug concentrations. However, the Panel will assess the commercially available devices with particular reference to oral fluid cut-off concentrations in relation to recommended blood drug concentrations.

With respect to ‘low level threshold’ drug concentrations of drugs in blood, sophisticated laboratory analytical techniques can detect extremely low drug concentrations that might have no significance in terms of pharmacological activity (e.g., driving impairment) of the tested drug. The analytical methodology used to determine concentrations of drugs in blood will require a reliable quantification limit that is relevant to the drug’s pharmacological activity.

The possibility of accidental and passive exposure to drugs is limited to cannabis (e.g., cannabis side-stream smoke). Other drugs are unlikely to be present in blood samples due to accidental or passive exposure, which means that laboratory testing is unlikely to give false positives in terms of drug use. However, since drug contaminated air might be inhaled through the mouth, it is possible that the drug will be detected in oral fluids. This means that a roadside screening might be positive, but in such a case the confirmatory laboratory test would likely be negative.

### 6 Drugs that can impair driving

Drugs that can affect driving have effects on the body’s central nervous system. The central nervous system (CNS) includes the brain and the nerve system that sends messages to and from the brain.

Of the 25 drugs for which per se limits have been recommended, seven are generally considered as recreational, although most also have a legitimate medical use. They are:

- THC, the main psychoactive component in cannabis. The major cannabinoids found in cannabis plant, THC and cannabidiol (CBD) may be prescribed for a range of disorders. THC is currently only available in imported products, typically for control of muscle spasticity. CBD is not psychoactive and will not impair driving.
- Methamphetamine has no legitimate use in NZ but is used in some countries to treat narcolepsy, attention deficit hyperactivity disorder (ADHD) and attention deficit disorder (ADD).
• Amphetamine is prescribed in NZ (as dexamphetamine) to treat narcolepsy.
• MDMA has no legitimate use in NZ but research is ongoing for its use as an antidepressant.
• Cocaine is used as a local anaesthetic for eye surgery.
• GHB is used to treat narcolepsy and can suppress the symptoms associated with alcohol or opioid withdrawal.
• Ketamine currently is mainly used by emergency services as an anaesthetic; however, there is ongoing research (including in NZ) for its use for severe depression and acute treatment for suicidal tendencies.

The remaining 18 drugs are sedatives, hypnotics or analgesics and are all prescribed in NZ.

6.1 ‘Recreational’ Drugs

The Panel has recommended *per se* blood drug limits for seven drugs that can be considered recreational. There are many recreational drugs currently available in NZ that the Panel has not recommended *per se* blood concentrations. These include the synthetic cannabinoids, the synthetic cathinones and the hallucinogens such as the N-methoxybenzyl (NBoME) derivatives. The use of these drugs by NZ drivers is largely unknown, but all are considered to have effects incompatible with safe driving. Most of these drugs are not detected by the routine analyses currently carried out at ESR on samples taken under the Land Transport Act (LTA) or from deceased drivers. Specific analyses are often required to detect these drugs: such analytical techniques require access to authentic reference drug material and the difficulty of the situation is compounded by the frequency at which illicit manufacturers develop structural analogues of the drugs in an attempt to keep ahead of legislation.

For example, the original synthetic cathinones sold in ‘party pills’ contained methylone or mephedrone, which was superseded initially by butylone and most recently (2020) by eutylone. Each of these analogues has a different and unknown potency, with no clearly defined concentration for activity or the dose required to achieve such a concentration.

The first synthetic cannabinoids, JWH18 and JWH73, were sold in NZ dairies in 2009. Since then, there have been dozens of different synthetic cannabinoids available on the illicit market, each with unknown potency and unpredictable effects.

Because the detection of the synthetic cannabinoids requires specific methodology which is not routinely carried out on LTA blood samples, the use of such drugs by NZ drivers is largely unknown. However, there is evidence these drugs are being used by NZ drivers.

Blood samples from 65 impaired drivers, from the period 2017 to 2018, had been analysed by ESR’s routine methodology and no drugs were detected. These blood samples were later retrospectively analysed for evidence of the use of a range of synthetic cannabinoids. Evidence of synthetic cannabinoid use was detected in 39 of 65 (60%) samples analysed.

In 2019 the synthetic cannabinoids AMB-FUBINACA and 5F-ABD were classified as Class A controlled drugs under the Misuse of Drugs Act 1975, following a number of drug related fatalities in 2017. These compounds are now very rarely detected by laboratory analyses because different synthetic cannabinoids are available on the illicit market.

None of these drugs are detected by the currently available roadside oral fluid testing devices. Furthermore, there is insufficient knowledge of their pharmacology for it to be possible to define *per se* limits for them. However, drivers taking these drugs will likely be detected by the compulsory impairment test (CIT).
6.2 Prescription Drugs

As stated earlier, 18 of the drugs for which per se limits are recommended are commonly prescribed sedatives, hypnotics and analgesics. These prescription medications are those that are associated with abuse and/or recreational use. While many people take them for legitimate purposes, their effects have led to misuse. It is these effects that can impair driving.

More recognition is required of possible driving impairment that might be caused by all types of prescription medication, not just those classified as narcotic analgesics and sedatives. Importantly, it is not necessarily the primary action (effect) of the medicine that is responsible for impairment. Many medicines have side effects that could cause impairment. Indeed, there are a number of medicines with legitimate medical use; that have been shown to impair driving skills in laboratory tests and driver simulation studies [7].

In a list of medicines and preparations prescribed in NZ (as reported on the Ministry of Health web site) there were about 1,966 different medicines, drugs and preparations. These 1,966 medicines were divided into 13 groups depending on their purpose [8].

Eight of these groups are unlikely (because of their pharmacology) to be of concern in relation to impairing driving. These medicines were used for treating:

- The alimentary tract - medication for the treatment of ulcers and diarrhoea, laxatives, minerals and vitamins.
- Blood forming organs - medication for the treatment of low or excessive iron, lowering cholesterol and agents that prevent blood clotting.
- Dermatological diseases - medication to treat skin conditions, anti-acne and anti-fungal creams.
- Hormone anomalies - hormone replacement therapy, thyroid treatment and some steroids.
- Infections - antibiotics, antifungal and antiviral medications.
- Cancer - immune-suppressants, chemotherapeutic drugs.
- Sensory organs - preparations to treat eyes and ears.
- Dietary disorders – dietary supplements.

These eight groups accounted for about 1,135 of the 1,966 (approx. 58%) medicines or preparations listed by the Ministry of Health.

The medicines in four of the remaining groups were present in medication for the treatment of ailments relating to the following systems:

- Genito-urinary
- Musculoskeletal
- Respiratory
- Cardiovascular

While they are not medicines that directly affect the CNS, some have the potential to impair driving due to known side effects.

Genito-urinary medication includes contraceptives, anti-infectives and erectile dysfunction medications. The majority of drugs under this heading are unlikely to cause impairment. However, the on-line patient information for one of these medications, sildenafil (the active ingredient in Viagra) reads – May cause visual disturbance and dizziness, even temporary blindness, do not drive while affected.
Musculoskeletal medications are used to treat gout, rheumatism and bone metabolism disorders. Some of these medications are muscle relaxants, which may affect the ability to drive. The patient information associated with some of the non-steroidal anti-inflammatories in this category, included the warning – May cause dizziness, do not drive if affected.

Medications for the treatment of respiratory disorders include antihistamines for treatment of allergies, bronchodilators for asthma sufferers, and cough medication. There were 78 such medicines listed by the Ministry of Health. The patient information for a selection of these medicines is as follows:

- Chlorpheniramine (an antihistamine) - May cause drowsiness, do not drive or operate machinery if affected.
- Promethazine (an antihistamine) - May cause drowsiness, do not drive or operate machinery if affected, avoid alcohol.
- Salbutamol (asthma medication) - May cause dizziness, visual disturbances.
- Pholcodine (cough medication) can be purchased without prescription from a pharmacy) - May cause drowsiness, avoid alcohol, do not drive or operate machinery if affected.

There were 78 cardiovascular medicines listed by the Ministry of Health. These are included in anti-arrhythmic medication, beta-blockers (to control blood pressure), anti-hypertensives (used to lower blood pressure) and diuretics (used to reduce fluid retention by tissues). The on-line patient information for many of these medicines includes the warning – May cause dizziness, do not drive if affected.

Although these medicines do not act directly on the CNS, patients should be made aware that their side effects could affect their ability to drive safely.

There were 127 medicines listed by the Ministry of Health as used in nervous system medication; these include:

- Medication for movement disorders (e.g., Parkinsonism)
- Anaesthetics – usually only used by doctors or dentists
- Analgesics
- Antidepressants
- Medications for epilepsy and migraines
- Antinausea medications
- Antipsychotics
- Anxiolytics (used to treat anxiety)

All of these medicines would be expected to have the same warning in the patient information section – Do not drive or operate machinery if affected, may cause drowsiness. For many of these medicines there is also the advice to either restrict or avoid alcohol.

The Panel has recommended per se limits for only 18 of these 127 (14%) medicines. This does not mean that a driver will not be impaired by the other prescribed medicines, particularly when they are first prescribed.

It is important that doctors and pharmacists prescribing and dispensing these medicines advise patients of their potential driving impairment effects – particularly if taken either in conjunction with alcohol or other medications. Professional development courses have been developed by the NZ College of General Practitioners (GPs) and the Pharmaceutical Society of NZ (PSNZ) in conjunction with
the NZ Transport Agency (NZTA, Waka Kotahi). These courses should be promoted by the professional bodies since the current uptake of training in this important area is not known.

Currently, it is not possible to randomly sample the NZ driving population to get an indication of what medications are being used by drivers. Samples available for analysis are limited to those taken from drivers who have blood samples taken under the LTA and from deceased drivers.

Prescription medicines that have the potential to impair driving should not be taken with alcohol as it is likely to enhance impairment [9]. An ESR/NZTA study (discussed below) analysed blood samples of 3,050 non-hospitalised drunk drivers, 460 (15%) had used medication that would be classified as ‘Likely to impair or Avoid alcohol’.

The Panel recommends that the Ministry of Health conduct a review on the processes and procedures for prescribers and dispensers (pharmacists) of prescription medicines and what information should be provided to the patient about driving impairment (and effects on operating machinery). It may also be advisable for the Ministry of Health and/or PHARMAC to provide manufacturers with updated medicine packaging and labelling requirements to satisfy or facilitate the implementation of any new procedures.

7 Overview of drug use by NZ drivers

ESR is the main provider of forensic services for the NZ Police. This includes all of the toxicological analyses in relation to criminal, coronial and LTA samples. Through these analyses, data on drug use by NZ drivers has been collected for many years. The data include the prevalence of alcohol use and other drug use obtained from analyses carried out since 2004.

Over this time there have been changes to the LTA, including changes to the blood alcohol limits, and the introduction of drugged driving legislation. In addition, there have been changes to the analytical techniques used by ESR that has increased the range of drugs that can be detected.

In 2009, changes to the LTA enabled Police to stop and test suspected drug-impaired drivers; i.e., those observed to be driving poorly. The driver is first breath tested for alcohol. If the breath test result is negative, the driver undergoes a compulsory impairment test (CIT) conducted by a trained police officer. If the driver fails to satisfactorily complete the CIT, a blood sample can be taken for drug analysis.

The ESR Toxicology Laboratory carries out analyses for alcohol and a range of other drugs in samples taken from four categories of drivers (listed below); the results of these analyses may be used to provide evidence of drug use by the NZ driving population in the four categories:

1. **Deceased drivers** – drivers who have died as a result of a motor vehicle crash. Blood samples from most drivers are sent to ESR for analysis. It is important to recognise that some drivers may have suffered a medical event that precipitated the crash and thus drugs might not be involved.
2. Drug impaired drivers – drivers stopped by Police due to observed poor driving who have not used alcohol as determined by a breath alcohol test. These drivers have failed a CIT and have provided a blood sample for drug analysis.

3. Hospitalised drivers – drivers in hospital following a motor vehicle crash are obliged to provide a blood sample for analysis. The blood is initially analysed for alcohol. Analysis for evidence of drug use by these drivers is carried out at the specific request of the Police. However, if the concentration of alcohol in the blood is greater than the legal limit, analyses for drugs are not generally carried out. There are numerous reasons why blood samples might not be sent for analysis.

4. Drunk drivers – drivers who fail the evidential breath test are not obliged to provide a blood sample for analysis, as they may accept the evidence of the breath test. However, some drivers elect to have a blood sample taken for analysis. Samples taken from these drivers are analysed to determine the alcohol concentration, but are rarely analysed for drug use.

7.1 Deceased drivers

2004 to 2009
From July 2004 to June 2009, 1,177 drivers died on the road in NZ. Blood samples from 1,046 (89%) of the deceased drivers were analysed for the presence of a wide range of drugs.

Analysis of these samples showed that:

- 48% had not used alcohol or other drugs (analytical techniques cannot detect all potentially impairing drugs),
- 32% were positive for alcohol,
- 30% had used cannabis,
- 13% had used both alcohol and cannabis, but no other drugs,
- 4% had used methamphetamine,
- 4% had used opioid type drugs, and
- 4% had used sedative-type drugs.

2013 to 2018
During this five-year period, there were 1,342 identified driver fatalities. Analyses were carried out on blood samples received at ESR from 1,069 (80%) of this group.

Analyses of these samples showed that:

- 41% had not used alcohol or other drugs (analytical techniques cannot detect all potentially impairing drugs),
- 27% were positive for alcohol,
- 25% had used cannabis,
- 7% had used both alcohol and cannabis, but no other drugs,
- 8% had used methamphetamine,
8% had used opioid type drugs, and
7% had used sedative-type drugs.

7.2 Drug impaired (failed CIT) drivers

From 2013 to 2018, 1,899 samples from impaired drivers were submitted for analysis; of these:
- 9% had no detectable drugs (analytical techniques cannot detect all potentially impairing drugs),
- 59% had used cannabis,
- 37% had used methamphetamine,
- 22% had used opioid type drugs,
- 16% had used sedative type drugs, and
- multiple drug use was common with 36% of the drivers using more than one drug.

Since 2017, the analyses routinely carried out by ESR on blood samples from impaired drivers are able to detect the 18 prescription drugs with recommended per se limits. However, the routine analyses do not detect the many other potentially impairing prescription medicines discussed above. Only if no drugs are detected by the routine tests, will further analyses for a wider range of drugs be carried out. As a consequence, the use of other prescription drugs that can affect the CNS by impaired NZ drivers is largely unknown.

For the period 2013 to 2016, all samples from impaired drivers were analysed by ESR using an analytical method that could detect most of these 127 plus potentially impairing prescription medicines. Samples from 961 impaired drivers were received over this period. 157 (16%) drivers had used a prescription medicine that does not have a recommended per se limit. However, 148 of these 157 drivers had also used a medicine that does have a recommended per se limit.

7.3 Hospitalised drivers

Drivers hospitalised following a crash are unable to undergo an impairment test. However, they may be prosecuted for drug impaired driving, without proof of actual impairment, if a Class A controlled drug (as defined in the Misuse of Drugs Act 1975) is detected in their blood. The Class A drugs most likely to be encountered in NZ are methamphetamine, LSD, cocaine or heroin.

ESR does not receive blood samples from all drivers hospitalised following a crash. However, all samples from hospitalised drivers are analysed for alcohol; indeed, in many cases alcohol is the only analysis requested. Analysis for drugs must be requested by the Police. Further to this, if a drugs analysis is requested and the BAC is above the legal limit, drugs analyses will not be carried out without an additional request by Police.

From 2013 to 2018, 1,939 blood samples from drivers hospitalised following a crash have been analysed for evidence of drug use, of these:
- No drugs were detected in 33% of the samples,
- 37% had used cannabis,
- 28% had used methamphetamine,
15% had used opioid type drugs,
14% had used sedative-type drugs,
Two drivers had used cocaine, and
One driver had used LSD, but
No drivers had used heroin.

7.4 Drunk drivers

Generally, samples from drivers who have blood alcohol concentrations greater than the legal limit are not analysed for evidence of additional drug use. In 2011, a study was commissioned by the NZTA (Waka Kotahi) to look at drug use by this portion of the driving population. 3,050 samples received by ESR between 2011 and 2015 were analysed for a wide range of drugs. These blood samples were taken from drivers who were not in hospital but had failed a breath alcohol test and elected to have a blood sample taken.

All of the samples analysed had BACs greater than the legal limit. Fifty-nine percent of the drivers had used alcohol only (i.e., no other drugs were detected) and 41% of the drivers had used other drugs in addition to alcohol. Of the 3,050 driver samples analysed:

- 27% had used cannabis,
- 3.5% had used sedative type drugs,
- 2.3% had used opioid-type drugs, and
- 1.6% had used methamphetamine.

Drivers who have breath alcohol concentrations (BrAC) equivalent to BACs between 50 and 80 milligrams per 100 millilitres (mg/100 mL) are not given the option to provide a blood sample. This breath alcohol concentration automatically incurs an infringement charge, and consequently possible additional drug use by these drivers is unknown.

Of the 3,050 samples from the non-hospitalised drivers, 1,815 (59.5%) had not used another drug. That is, they had used alcohol, but no other drug was detected. Of the 3,050 drivers, 1,235 (40.5%) had also used another drug. There was evidence of the use of prescription medication by 503 (16%) drivers. Of the 503 drivers using prescription medication, 460 (91.5%) had used a medication that would be classified as ‘Likely to impair’ or ‘Avoid alcohol’.

7.5 Multiple drug use by drivers

The drugs covered in this report include stimulants, anaesthetics, sedatives and hallucinogenic substances. Whilst many are agonists at various biological targets, they each have an individual profile with regard to their target (receptor), their potency at that target and their pharmacokinetic profile.

Taking two or more drugs from the same class (e.g., opioids) may produce a simple additive effect, whereas taking two drugs from different classes (e.g., an opioid and a sedative) may produce a synergistic effect. In contrast, taking a stimulant and a sedative will produce a varying antagonistic response dependent on the relative potencies and blood concentrations over time. In addition, it is
possible that two or more drugs have a pharmacokinetic interaction, often leading to a decreased clearance and thus higher than predicted blood concentrations.

With our current knowledge it is impossible to predict with certainty the total impact of taking several of the drugs of interest on driving impairment. However, it is generally accepted that taking more than one drug would have a greater detrimental effect than taking a single drug.

It is important to consider the relevance of per se limits, both threshold and criminal, when more than one drug has been used because of the possible pharmacological interactions between drugs.

The Norwegian Expert Panel noted that their recommended limits were for a single substance and not for combinations of substances [2,6].

A case-controlled study of road crashes requiring hospitalisation carried out in the Netherlands 2000 to 2001 [7] found a significant increase in crash risk for drivers using combinations of drugs and using alcohol with combinations of drugs.

Multiple drug use is common in NZ drivers. From 2017 to the middle of 2020, blood samples were received at ESR from 1,679 impaired (failed CIT) drivers. Of these drivers, 620 (37%) had used more than one drug.

The most common combination of drugs used by impaired drivers was cannabis and methamphetamine. 350 (56.5%) of the 620 drivers using multiple drugs, were found with this combination of drugs. The other 270 (43.5%) drivers who had used multiple drugs, had used various combinations of cannabis, stimulants, opioids and sedatives, all drugs with recommended per se limits. Of the 620 drivers who had used more than one drug, 132 (21%) had used more than two drugs.

Of the 350 drivers who had used both cannabis and methamphetamine, 262 (75%) had blood THC concentrations that would be below the recommended criminal limit. However, most of the drivers had blood methamphetamine concentrations greater than the recommended criminal limit. Of the 350 impaired drivers, 50 who had used methamphetamine and cannabis, but no other drug, had blood concentrations of both drugs below the recommended criminal limit.

Of the 270 drivers who had used other combinations of drugs, 190 drivers (70%) had at least one drug above the recommended criminal limit while for 80 drivers (30%) all drugs detected were below the recommended criminal limits.

Of the 132 drivers found to be impaired by multiple drugs, 21% had blood concentrations of these drugs below the recommended criminal limit. None of these impaired drivers had used alcohol.

Very little is known about the combined use of other drugs with alcohol by NZ drivers. This is because if a driver is stopped for poor driving, they are first breath tested, and if they are found to be above the legal breath alcohol limit, no CIT or drug testing is carried out. Blood samples from drivers in hospital following a crash are first analysed for alcohol. If alcohol is detected above the legal limit, it is rare for drug testing to be carried out. For these reasons, there is a dearth of information on drug-alcohol combinations.

The little information available about the combined use of alcohol and other drugs by NZ drivers comes from the analysis of blood samples from drivers killed in motor vehicle crashes, and from the ESR/NZTA study data on drunk drivers.
Blood samples from 1,067 deceased drivers were analysed in the period 2013 to 2018. Of these drivers, 293 (27.5%) had used alcohol (240 drivers (22.5%) had BAC greater than 50 mg/100mL, 224 (21%) had BAC greater than 80 mg/100mL). Over half of the drivers using alcohol had also used another drug.

There were 156 deceased drivers who had used alcohol with another drug (126 of the 156 drivers had BAC greater than 50 mg/100mL, 117 of the 156 drivers had BAC greater than 80 mg/100mL).

The ESR/NZTA study of drunk drivers involved the analysis of 3,050 blood samples taken from drivers who failed the breath alcohol test and elected to have a blood sample taken. These drivers were not involved in a crash and had a BAC greater than the legal limit. The time period covered by this study is from November 2011 to April 2015, and blood from drivers from three Police districts was analysed for evidence of use of a wide range of drugs.

For the purposes of this study, the drugs detected were ranked in the following way:

- Recreational drugs
- Drugs likely to impair driving with or without alcohol
- Drugs that may impair driving when first prescribed - advisory labelling: Avoid alcohol
- Drugs that may impair when first prescribed – advisory labelling Limit alcohol.

The drugs classified as recreational have no legitimate purpose. Those drugs classified as ‘Likely to impair with or without alcohol’ include the prescription drugs for which per se limits have been recommended, plus some others such as sedating antidepressants and antihistamines.

Of the 3,050 samples from the non-hospitalised drivers, 1,815 (59%) had not used another drug. That is, they had used alcohol, but no other drug was detected by the analyses carried out.

Of the 3,050 samples from non-hospitalised drivers, 1,235 (40.5%) had also used another drug as follows:

- 816 (27%) evidence of cannabis use,
- 78 (2.6%) other recreational drugs,
- 503 (16%) prescription drugs.

Of the 503 drivers using prescription medicines, 460 (81%) had used a drug or drugs that were classified as ‘Likely to impair driving’ or ‘Avoid alcohol’.

Based on these admittedly biased driver populations, it appears that the use of multiple drugs, both illicit and prescription, by NZ drivers is common.

It is not possible to recommend limits for all recreational drugs. Drugs such as synthetic cannabinoids and cathinones may be used alone or in combination with other recreational drugs such as cannabis and methamphetamine, for which limits are recommended. Similarly, it is not possible to recommend limits for all potentially impairing prescribed medication. Use of legitimately prescribed medicines in combination with recreational drugs or other prescribed medicines may result in a greater degree of impairment.

When more than one drug is detected in the blood of a driver, the recommended criminal limits should no longer be applied, because use of multiple drugs will increase the degree of impairment.
Detection of drugs in oral fluid

Saliva comprises the secretions from the salivary glands, whereas oral fluid is saliva plus other debris in the oral cavity. Oral fluid composition and flow is influenced by many factors and therefore varies between individuals. Furthermore, drug transfer from blood to oral fluid is also dependent on many factors and also varies between individuals.

Smoking, insufflation (e.g., vaping), and sublingual administration of drugs results in high concentrations of drugs in the oral cavity due to direct exposure of the oral mucosa. When drugs are administered by intravenous (IV), intramuscular (IM) injection, or in encapsulated forms, oral mucosal contamination does not occur.

Oral fluid has become popular in many jurisdictions as a means to screen for evidence of drug use. The lack of invasiveness and with little need for privacy while collecting the sample make it an ideal sample for roadside drug testing.

Drug use is often accompanied by reduced saliva production. It should be noted that artificial stimulation of saliva (use of gum or acidic sweets) will result in lowering of drug content in oral fluid due to dilution. Similarly, rinsing the mouth with water will result in significantly lower drug concentrations, although for some drugs this reduction is only temporary.

The AS/NZS 4760:2019 Australian/New Zealand Standard “Procedure for Specimen Collection and the Detection and Quantification of Drugs in Oral Fluid” [10] outlines how to collect specimens and give recommended cut-off concentrations for some drugs in oral fluid. While much of the Standard is procedural and targeted at work-place drug testing environments, the recommended cut-off limits may have relevance for testing drivers at the roadside.

The Standard [10] specifies the expected cut-off concentrations that should be detected by on-site oral fluid screening devices as well as oral fluid laboratory analytical detection cut-offs (Table 2). Cut-off concentrations for on-site screening devices are higher than the laboratory cut-offs because the screening devices are not specific and detect more than one drug of a particular class and/or the metabolites of the drug.

*Table 2 Cut-off drug concentrations in oral fluid as set out in AS/NZS 4760:2019*

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<thead>
<tr>
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<th>On site cut-offs ng/mL</th>
<th>Laboratory cut-offs ng/mL</th>
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<td>Cocaine</td>
<td>50</td>
<td>Cocaine 25</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Benzoylecgonine 25</td>
</tr>
<tr>
<td>Opiates</td>
<td>50</td>
<td>Codeine 25</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Morphine 25</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6-Acetylmorphine 10</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>40</td>
<td>Oxycodone 20</td>
</tr>
</tbody>
</table>

*natural cannabinoïds, not synthetic*
The oral fluid cut-offs for laboratory confirmation were deemed to be those above which the risk of impairment was sufficient to be of concern and below which the risk of potential impairment was not of concern.

An on-site or roadside device with cut-offs as set out in Table 2 should mean the oral fluid samples analysed at the laboratory will have oral fluid drug concentrations above the specified cut-offs.

It is important to note that on-site oral fluid testing devices are screening tools. They do not prove use of a drug and for many drugs they do not even indicate the use of a specific drug, but rather a class of drugs. For example, the methamphetamine channel also detects MDMA. This means a positive result from this channel could mean the possible use of either methamphetamine or MDMA or both.

Amphetamine is not detected by the methamphetamine channel and oral fluid devices have a separate channel for this drug if its detection is required. Similarly, the opiate channel detects possible use of morphine, codeine, dihydrocodeine and acetylmorphine, but not other opioids. The benzodiazepine channel can detect possible use of several drugs in that class. The cannabinoid channel is designed to detect THC and its metabolite THC-acid. Analysis at the laboratory is required to prove use of a specific drug.

Other international agencies have set similar laboratory oral fluid drug concentration cut-offs (Appendix 6) [11-13] to those in AS/NZS 4760:2019.

It is not possible to predict a blood drug concentration based on an oral fluid drug concentration. Simultaneous sampling of oral fluid and blood has shown that most drugs are present at higher concentrations in the oral fluid than in the blood, when drug use is recent. The exception is the benzodiazepine type medicines which are always present at lower levels in oral fluid than in blood.

Cannabis may be smoked, vaporised, or ingested as various forms of edible products. Following all of these methods of use, oral fluid THC concentrations are high - initial concentrations can range from 400 to 10,000 ng/mL. These initial high concentrations drop rapidly to about 10% of the peak concentration within an hour. Rinsing the mouth will have a significant effect on oral fluid THC concentrations [14].

With an oral fluid THC cut-off of 2 ng/mL, THC can be detected up to 24 h after use for occasional users and after up to 72 h for frequent users. Following non-extreme passive exposure oral fluid THC concentrations are less than 5 ng/mL at peak with a rapid decrease when the individual is no longer exposed [14].

Unlike other drugs THC does not transfer from blood to oral fluid. However, its metabolite THC-acid may be detected in the oral fluid of heavy cannabis users. THC-acid concentrations in blood exceed those of THC within 0.5 h after smoking cannabis, and the metabolite can be detected in the blood for a longer period than THC itself. THC-acid can be detected in oral fluid for a similar time as for blood, but it may not be at detectable concentrations for the occasional user of cannabis. Oral fluid testing devices are more sensitive to THC-acid than to THC; this might result in false positive oral fluid screen results for heavy cannabis users.

Methamphetamine and MDMA concentrations are greater in oral fluid than in blood. While there have been few controlled methamphetamine smoking studies, it is expected that long-term users are likely to have detectable methamphetamine in oral fluid for several days after its last use [14]. MDMA may be detected in oral fluid for 12 to 48 h after use.

Cocaine may be detected in oral fluid up to 6 h after smoking and up to 12 h after snorting [14].
How long codeine and morphine might be detected in oral fluid depends on the cut-off applied. Codeine taken as a medicine is detected up to 20 h after dosing. A similar time range would be expected for morphine and oxycodone, depending on the size of the dose. Morphine and codeine may also be detected in oral fluid after consumption of poppy seed-containing foods.

All of the benzodiazepines are present in oral fluid at lower concentrations than those detected in blood, although concentrations are dependent on dose [14]. In contrast, oral fluid zopiclone concentrations are higher than in blood, even when the drug is taken as a capsule so there can be no direct oromucosal contamination [15].

While there is a correlation between oral fluid and plasma/blood drug concentrations over time, there is too much intra-individual and inter-individual variability to be able to predict blood drug concentrations from oral fluid drug concentrations [14].

A number of studies have looked at the relationship between oral fluid and blood drug concentrations. Some have compared the analytical results of blood and oral fluid samples taken at the same time from drivers [16-20]. Others have carried out statistical analyses of aggregated data [21-23].

Paired oral fluid and blood samples were taken as part of the DRUID project. Statistical analysis of 311 THC positive samples and 197 amphetamine positive samples was carried out. The ratio of the oral fluid THC concentration to the blood THC concentration ranged from 0.006 to 569 (mean 34.1, median 15.4). The ratio of oral fluid amphetamine concentration to blood amphetamine concentration ranged from 0.27 to 210 (mean 19, median 12) [17]. This wide range of oral fluid and blood drug concentration ratios shows why it is not possible to predict a blood drug concentration based on an oral fluid drug concentration.

In a 2012 study involving the simultaneous collection of blood, urine and oral fluid from drivers, it was found that all drugs detected in blood were also detected in oral fluid. This shows that oral fluid is a valid screening tool for evidence of drug use [18]. Amphetamines, opiates, cocaine and THC all had higher levels in oral fluid than in blood. A comparison of oral fluid and blood concentrations for alprazolam, clonazepam, diazepam and nitrazepam found these benzodiazepines had lower levels in oral fluid than blood with a ratio ranging from 0.5 to 0.1 [18].

False positive results can be considered those where the oral fluid result for a drug is positive but the result cannot be confirmed by laboratory analysis of blood. The proportion of false positive results depends on the cut-off levels used for the oral fluid devices and the blood analyses. For example, in a 2012 study of blood and oral fluid samples taken from 2,750 drivers, drugs were detected above the oral fluid cut-off in 71 (approx. 3%) drivers but only 28 (39%) of these drivers had blood drug concentrations higher than the legal limit [19]. Similarly, a 2015 study found a proportion of positive oral fluid on-site screen results were not confirmed by blood concentrations greater than the legal limits. The proportion of these false positive results were THC 9%, cocaine 36%, amphetamine 26% and opiates 53% [24].

Although it depends on the legal limits set for confirmation analyses, some of the oral fluid testing devices might now be too sensitive. The high sensitivity (i.e., low LoDs) of the oral fluid devices currently available might mean that drug concentrations (i.e., by laboratory analysis) in blood samples taken at the same time will not be above per se limits.

8.1 Oral fluid detection devices

The Oral Fluid Standard AS/NZS 4760:2019 [10] recommends detection cut-offs for on-site devices and laboratory analysis. There should be no requirement to follow strictly the detection limits in the
Standard for devices used by NZ police because confirmatory analyses will be carried out on blood samples.

If a drug has been used recently, blood drug concentrations are generally lower than oral fluid concentrations. Furthermore, there is a very poor correlation between oral fluid and blood drug concentrations. Therefore, oral fluid drug concentrations cannot be used to predict blood drug concentrations.

As of 2020, there are five companies providing oral fluid testing kits in NZ. There are five devices that are hand-held devices and can be used for on-site testing. One device (Dräger DT5000) is not a handheld device but could be used in a ‘booze bus’ environment. The practicality of these devices for roadside use needs to be considered by the police.

The handheld devices currently available in NZ are Oratech III, ToxWipe, DrugCheck3000, OralCube 7+ and DrugWipe 6S. The drug detection limits for some these devices are given in Table 3, and are compared with the limits required by the AS/NZS 4760:2019 Standard [10]. On-site oral fluid testing devices are undergoing continual improvements with the demand for detection of different drug types and faster response times.

Table 3 Detection limits of drugs by oral fluid roadside devices compared to cut-off levels specified in the Standard AS/NZS 4760:2019

<table>
<thead>
<tr>
<th>Drug/class</th>
<th>AS/NZS 4760:2019</th>
<th>Oratect III</th>
<th>DrugCheck 3000</th>
<th>OralCube 7+</th>
<th>DrugWipe 6S</th>
<th>Dräger</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amphetamine</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td>60</td>
<td>50</td>
</tr>
<tr>
<td>Benzodiazepine</td>
<td>-</td>
<td>5</td>
<td>15</td>
<td>50</td>
<td>-</td>
<td>15</td>
</tr>
<tr>
<td>Cocaine</td>
<td>50</td>
<td>20</td>
<td>20</td>
<td>50</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td>60</td>
<td>35 (MDMA)</td>
</tr>
<tr>
<td>Methadone</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>20</td>
</tr>
<tr>
<td>Opiates</td>
<td>50</td>
<td>40</td>
<td>20</td>
<td>50</td>
<td>-</td>
<td>20</td>
</tr>
<tr>
<td>THC</td>
<td>15</td>
<td>40</td>
<td>15 or 25</td>
<td>12 or 25</td>
<td>10</td>
<td>5</td>
</tr>
</tbody>
</table>

Points to note:
- The detection limits for on-site devices were provided by MOT from on-line sources or obtained from the scientific literature [25].
- ToxWipe detection limits were not supplied, but claim to meet the Standard.
- OralCube 7+ has a channel to detect synthetic cannabinoids. However, the synthetic cannabinoids listed as detected by the device have a chemical structure that has not been seen in NZ for several years. This device is very unlikely to be able to detect the synthetic cannabinoids currently being sold in NZ.

As stated previously, these devices are not confirmatory devices. They do not prove the use of a drug. False positives and false negatives are possible.

In the drug driving context, a false positive is when a drug is detected by the oral fluid device but is not detected by confirmatory analysis. The inability to confirm a finding may be because, although the
drug is present, the concentration is not above the legislative limit. A false positive may also arise from cross-reactivity, where a different drug causes the positive reaction.

A false negative result occurs when there is no reaction to the presence of a drug by the oral fluid device, even though the drug is present. Such occurrences are more difficult to determine by confirmatory analyses because, of course, a confirmatory analysis would not usually be carried out on a negative screen test. Some devices are known to give false negative results for high THC concentrations.

Further to this, it is very important to understand that few of the channels on these devices react to a single drug. This is known as cross reactivity because, due to similarities between the chemical structures of the drugs (Fig. 1), the device cannot distinguish between them. The degree of cross-reactivity, or the sensitivity of a particular device to drugs not being specifically targeted depends on the specific technology used. This can differ for the different devices available.

![Molecular structures of CBN, THC, THC-OH and THC-COOH showing their similarities, which is why they cross react (i.e., cannot be distinguished) in oral fluid detection devices.](image)

**Figure 1** Molecular structures of CBN, THC, THC-OH and THC-COOH showing their similarities, which is why they cross react (i.e., cannot be distinguished) in oral fluid detection devices.
The THC channel has cross reactivity with cannabinol (CBN), THC-COOH and THC-OH (Fig. 1). CBN is generally only present in significant quantities in old plant material. THC-COOH and THC-OH are metabolites of THC. Although THC-COOH is detected in blood at higher concentrations than THC within 0.5 h of smoking, it is not immediately present in oral fluid as a result of smoking. THC-COOH oral fluid concentrations are unlikely to be detectable for infrequent smokers, but with heavy or chronic use of the drug this metabolite will be detected in oral fluid for a longer period than THC itself [26].

The methamphetamine channel has strong cross reactivity with MDMA and may also have cross reactivity with 3,4-methylenedioxy-N-ethylamphetamine (MDEA), MDMB, mephedrone, paramethoxyamphetamine (PMA), 1,3-dimethylamylamine (DMAA), phentermine, phenethylamine, ephedrine and pseudoephedrine. The amount of drug required to give a positive result varies depending on the device. The devices are most sensitive to methamphetamine and MDMA, and higher concentrations of the other drugs are needed for a positive response. Furthermore, a number of the other drugs listed are now infrequently used. Pseudoephedrine is no longer available as a component in cold and flu medication. However, phentermine is a prescription weight loss medication, and phenethylamine is a common component in sports supplements.

The amphetamine channel will also react to the presence of MDA. MDA is a metabolite of MDMA but it is also a drug itself.

The cocaine channel will also detect its metabolite benzoylecgonine.

The Dräger opiate screen will react with a number of opiates at or near its detection limit: morphine, codeine, dihydrocodeine, morphine-glucuronide (a metabolite of morphine), 6-monoacetylmorphine (6MAM; a metabolite of heroin), nalorphine and hydrocodone (not prescribed in NZ). Oxycodone and naloxone are only detected at significantly higher concentrations. The DrugWipe opiate screen has cross reactivity with 6MAM, codeine, ethylmorphine, heroin, hydrocodone, oxycodone and morphine.

The Dräger benzodiazepine screen will react to a range of benzodiazepines at similar oral fluid concentrations (10 to 40 ng/mL). Of the benzodiazepines commonly detected in NZ drivers, this device will detect use of diazepam, alprazolam, clonazepam, midazolam, nitrazepam, oxazepam, temazepam and triazolam. The device reacts poorly to lorazepam. The DrugWipe benzodiazepine screen claims to be able to detect alprazolam, clobazam, clonazepam, diazepam, lorazepam, midazolam, nitrazepam, oxazepam, temazepam and triazolam.

Whichever oral fluid devices are selected for roadside testing, it is important to clearly ascertain which drugs the devices are capable of detecting and at what concentrations in the oral fluid. The oral fluid drug concentrations that the devices claim to detect are calibrated to a specific drug.

For example, the Dräger benzodiazepine screen detects diazepam at an oral fluid concentration of 15 ng/mL. The other benzodiazepines may be detected at higher or lower concentrations than this. It is not uncommon for NZ drivers to have used more than one benzodiazepine.

There have been a number of reports in the scientific literature assessing roadside devices in ‘real life’ situations [25, 27-31]. However, with improvement in technology of these devices some of the older reports are now of little value.

A 2017 Canadian study [25] assessed three devices, Alere DDS, Securetec Drugwipe-5+ and Dräger Drug Test 5000. The study considered:

- Sensitivity – proportion of true positive cases correctly identified
- Specificity – proportion of drug negative cases correctly identified
- Miss rate – proportion of drug positive cases not identified
• False alarm – proportion of positive screen results not confirmed

The results of this study were pooled (Table 4); therefore, it is not possible to distinguish between the performance of individual devices. In addition, confirmation analyses were carried out in oral fluid, not blood.

Table 4 An assessment of some on-site oral fluid devices [from ref 25]

<table>
<thead>
<tr>
<th>Drug</th>
<th>Number</th>
<th>Sensitivity</th>
<th>Miss rate</th>
<th>Specificity</th>
<th>False alarm</th>
</tr>
</thead>
<tbody>
<tr>
<td>THC</td>
<td>323</td>
<td>0.86</td>
<td>0.13</td>
<td>0.96</td>
<td>0.045</td>
</tr>
<tr>
<td>Cocaine</td>
<td>256</td>
<td>0.85</td>
<td>0.15</td>
<td>0.99</td>
<td>0.007</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>306</td>
<td>0.77</td>
<td>0.23</td>
<td>0.96</td>
<td>0.036</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>306</td>
<td>0.84</td>
<td>0.16</td>
<td>0.97</td>
<td>0.035</td>
</tr>
<tr>
<td>Opiates</td>
<td>301</td>
<td>0.9</td>
<td>0.1</td>
<td>0.93</td>
<td>0.069</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>241</td>
<td>0.59</td>
<td>0.41</td>
<td>0.93</td>
<td>0.024</td>
</tr>
</tbody>
</table>

However, this study shows that the devices studied are not perfect even when used to compare oral fluid screening with oral fluid confirmation. As blood drug concentrations are not the same as oral fluid drug concentrations, and the relationship between oral fluid and blood drug concentrations is dependent on a number of factors previously discussed, it will be necessary to carefully collate measurement data once the testing regime is in place in order to assess the reliability of oral fluid detection and blood detection at the recommended per se limits.

8.2 Consideration of oral fluid on-site cut-offs compared with recommended per se blood drug concentrations

One of the terms of reference for the Panel was to consider the cut-off limits to be included in the on-site oral fluid devices. It was required that cut-off thresholds for oral fluid testing devices needed to be selected at a level that accounted for the amount of a drug that would indicate recent use rather than past or passive exposure to a drug.

The commercially available oral fluid testing devices that are suitable for roadside testing, as described above, are currently used in several jurisdictions. Their drug concentration cut-offs are manufacturer set and cannot be reset by the operator. Therefore, in order to procure custom cut-off set devices, it would be necessary to commission custom manufactured devices to fulfil NZ’s needs.

The Panel considers it unlikely that it would be cost effective to commission a specific test device for New Zealand-specific oral fluid levels. Furthermore, it would be impractical to do so because some channels detect a number of different drugs that are present in oral fluid and blood at widely different concentrations.

As stated previously:

• Oral fluid drug concentrations cannot be used to predict blood drug concentrations.
• When samples are taken at the same time, blood drug concentrations have been found to be lower than oral fluid drug concentrations following recent use of all of the recommended drugs except benzodiazepines.
• The oral fluid on-site devices can detect more than one drug, and the cut-off limit associated with the device is based on the cut-off of one drug in that class.
For some channels on oral fluid on-site devices, several drugs in the same class as the targeted drug might give a positive response at a higher or lower concentration than the targeted drug.

Table 5 gives the recommended criminal blood drug limits and threshold limits for the drugs recommended by the Panel. These can be compared with the range of cut-off limits for the currently available oral fluid on-site devices.

**Table 5 Recommended blood concentrations compared with oral fluid on-site devices**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Recommended Criminal Blood Limit ng/mL</th>
<th>Recommended Threshold Blood Limit ng/mL</th>
<th>Oral fluid on-site detection range ng/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>THC (Cannabis)</td>
<td>3</td>
<td>1</td>
<td>12 to 50</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>50</td>
<td>10</td>
<td>50 to 60</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>100</td>
<td>20</td>
<td>50 to 60</td>
</tr>
<tr>
<td>MDMA</td>
<td>50</td>
<td>10</td>
<td>50 to 75</td>
</tr>
<tr>
<td>Cocaine</td>
<td>20</td>
<td>5</td>
<td>20 to 50</td>
</tr>
<tr>
<td>GHB</td>
<td>50,000 (50 μg/mL)</td>
<td>10,000 (10 μg/mL)</td>
<td>No on-site device</td>
</tr>
<tr>
<td>Ketamine</td>
<td>50</td>
<td>10</td>
<td>No on-site device</td>
</tr>
<tr>
<td>Alprazolam</td>
<td>50</td>
<td>20</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>50</td>
<td>20</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Diazepam</td>
<td>200</td>
<td>100</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>30</td>
<td>10</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Midazolam</td>
<td>30</td>
<td>10</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>50</td>
<td>20</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>800</td>
<td>200</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Temazepam</td>
<td>800</td>
<td>200</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Triazolam</td>
<td>4</td>
<td>4</td>
<td>5 to 50</td>
</tr>
<tr>
<td>Zopiclone</td>
<td>50</td>
<td>20</td>
<td>No on-site device</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>1</td>
<td>1</td>
<td>No on-site device</td>
</tr>
<tr>
<td>Codeine</td>
<td>200</td>
<td>50</td>
<td>20 to 50</td>
</tr>
<tr>
<td>Dihydrocodeine</td>
<td>200</td>
<td>50</td>
<td>20 to 50</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5</td>
<td>0.5</td>
<td>No on-site device</td>
</tr>
<tr>
<td>Methadone</td>
<td>200</td>
<td>50</td>
<td>20*</td>
</tr>
<tr>
<td>Morphine</td>
<td>20</td>
<td>10</td>
<td>20 to 50</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>50</td>
<td>20</td>
<td>40*</td>
</tr>
<tr>
<td>Tramadol</td>
<td>250</td>
<td>100</td>
<td>No on-site device</td>
</tr>
</tbody>
</table>

*Few devices offer channels for methadone and oxycodone

Following recent use of cannabis, oral fluid THC concentrations are higher than blood concentrations. The currently available on-site oral fluid devices detect cannabis use at higher concentrations than the recommended blood criminal limit. Methamphetamine and MDMA, both detected by the same channel, are also expected to have higher oral fluid concentrations than blood concentrations. The currently available on-site oral fluid devices detect these drugs at a similar concentration to the recommended blood criminal limit.
The oral fluid testing devices that detect benzodiazepines lack specificity and will detect more than one drug in this class. This covers a wide range of concentrations of individual drugs. The devices are expected to detect use of alprazolam, clonazepam, diazepam, midazolam, nitrazepam, oxazepam, temazepam and triazolam, but possibly not lorazepam. However, oral fluid concentrations of the benzodiazepines are lower than blood concentrations even following recent use. Some devices may only be able to detect the use of diazepam, oxazepam and temazepam.

The opiates, morphine, codeine and dihydrocodeine are generally found at higher concentrations in oral fluid than in blood at the sampling time. On-site oral fluid devices may detect codeine and dihydrocodeine for a longer period than that associated with recent use.

9 Individual Drugs and Drug Classes

9.1 Cannabis

The potential impairing effects of cannabis on driving include disorientation, altered sense of time and distance, lack of concentration, difficulty in thinking, loss of coordination, increased reaction time, lateral travel and impaired sustained vigilance [32].

Cannabis plants produce a large number of compounds unique to the species, called cannabinoids. Tetrahydrocannabinol (THC; Figure 1), is the primary psychoactive cannabinoid of the cannabis plant itself and any product made from the plant. The other cannabinoids present in the plant are generally at very low concentrations and contribute little or no psychoactive effects.

Maximum blood THC concentrations occur prior to the end of smoking a cannabis cigarette [32]. THC is enzymatically metabolised to hydroxy-THC and then rapidly to THC-acid (Figure 2). Blood hydroxy-THC concentrations reach a maximum soon after smoking has ceased, at much lower concentrations than blood THC concentrations. Hydroxy-THC is psychoactive. Blood THC-acid concentrations are greater than THC soon after smoking has ceased and reach a maximum at about 30 to 100 minutes. THC-acid is not psychoactive.

![Figure 2 Metabolism and excretion of THC showing pharmacological activity.](image)

The amount of THC in cannabis plants and products varies greatly. The concentration of THC in cannabis plants currently being grown in NZ is largely unknown. Determinations of THC concentrations
have been carried out by ESR over many years: the THC concentration in the female heads (cannabis plants have separate sexes: i.e., they are diecious) submitted following police seizures in the period 1994 to 1996 ranged from 0.1 to 8.8% w/w. A smaller study carried out from 2016 to 2017, found the concentration ranged from 0.4 to 7.8% w/w. In 2019, nine plant samples submitted for analysis ranged from 6.5 to 26% w/w THC.

Extracts from the cannabis plant can contain a significantly higher concentrations of THC. Twelve samples of fully extracted cannabis oil analysed by ESR in 2019 had THC concentrations that ranged from 5 to 87% w/w. However, if cannabis plant material or extracts are included in edibles, such as cakes or sweets, the final concentration of THC may be lower than that in the original plant.

Cannabis plant or products may be taken in a variety of ways for recreational purposes, but it is generally inhaled (smoked or vaped) or ingested.

Following changes in legislation since 2018, there is increased availability of cannabis-based products as foods and medicines. Food products made using hemp seeds do not contain enough THC to give a positive oral fluid screening test or detectable THC in blood samples.

Cannabis-based medicines may contain THC and/or CBD. If medicines contain high concentrations of THC, they may cause impairment, positive oral fluid tests and blood THC concentrations. CBD-based medicines have legally restricted, low concentrations of THC that will not result in impairment, positive oral fluid tests or blood THC concentrations.

These differences in products and routes of administration affect blood and oral fluid THC concentrations and their pharmacological effects.

9.1.1 Blood THC concentrations following inhalation

Cannabis plant material is generally smoked in the form of cigarettes. Other forms of cannabis, such as cannabis oil and hashish or extracts of the plant, may be heated and inhaled using a variety of techniques including vaping. Although the following information usually refers to ‘smoking cannabis’, similar findings are expected for all methods of inhalation.

When cannabis is inhaled, maximum blood THC concentrations occur within minutes of dosing and reach the maximum while a cannabis cigarette is still being smoked. Blood THC concentrations then drop rapidly as the THC is distributed around the body, particularly into fatty tissues, at a rate that varies between individuals. THC principally acts at sites in the brain (where it interacts with receptors), consequently blood concentrations correlate poorly with the effects of the drug (because it is the concentration in the brain that determines effect).

Figure 3 shows an amalgamation of blood THC concentrations from nine smoking studies [33-41] in which experienced smokers smoked cannabis cigarettes containing 15 or 30 mg THC. Approximately 10 min was allowed to smoke the cigarette. Blood THC concentrations were measured over an extended time period. Maximum blood THC concentrations of approximately 100 ng/mL occurred during smoking and started declining immediately to below 10 ng/mL within 1 h. At 3 h the blood THC
concentrations of all smokers had decreased to below 3 ng/mL and at 4 h the blood THC concentrations were all below 2 ng/mL. These studies did not consider impairment but showed the rapid decrease and wide variability in blood THC concentrations when smoking cannabis in a standardised environment.

Figure 3 Concentration of THC in blood of volunteers after inhalation (smoking) of cannabis cigarettes containing 15 or 30 mg THC where the time allowed to smoke the cigarette was 10 min (data from references 33-41)
The rapid decrease in blood THC concentrations, as well as the inconsistency of this decrease between individuals, have led some scientists to state that it is not advisable to set a per se blood THC concentration. The delay between the time a driver is stopped at the roadside and the time a blood sample is taken can result in a significant and unpredictable decline in blood THC concentrations [42].

9.1.2 Blood THC concentrations and impairment

An impairing dose of THC is considered to be approximately 5 mg. The bioavailability of THC is variable, but is generally higher when the drug is smoked rather than ingested. A recent study found that vaping cannabis extracts resulted in greater impairment and higher blood THC concentrations compared with smoking the same dose [43].

The greatest impairment to driving occurs 60 to 90 min following inhalation, a time range at which blood THC concentrations would have already declined significantly [32,44]. The effects of cannabis are reported to last about 3 to 4 h.

There have been many studies and reports trying to relate blood THC concentrations to impairment. The results are not consistent.

In 2005 an international expert panel issued a document entitled “Developing Science-based Per Se Limits for Driving Under the Influence of Cannabis’ [45] which was summarised in a peer reviewed journal in 2007 [46].

The study considered more than 120 individual experimental studies evaluating the impact of cannabis on driving performance; their findings are summarised below.

1. Acute use impairs cognition and psychomotor performance.
2. Impairment increases with dose.
3. The effect of a given THC dose varies considerably between individuals.
4. Impairment is greatest in the acute phase 30 to 60 min after smoking; the post-acute phase lasts 60 to 150 min.
5. Most acute effects subside within 3 to 4 h.
6. Following oral consumption, peak effects occur at 2 to 3 h.
7. A THC dose of 15 to 20 mg is considered a medium to high dose.
8. Frequent chronic users of cannabis may have blood THC concentrations over 2ng/mL for up to 48 h.
9. The combined use of alcohol and cannabis has additive effects.
10. Passive exposure to cannabis smoke can result in oral fluid concentrations of several ng/mL.
11. Blood THC concentrations of 3.5 to 5 ng/mL may achieve reasonable separation of impaired from unimpaired drivers.
12. Meta-analysis indicates a THC blood concentration of about 5 ng/mL equates to a BAC of 80 mg/100 mL.
13. THC serum concentrations of 7 to 10 ng/mL (equivalent to 3.5 to 5 ng/mL whole blood) correlated to impairment of BAC 50 mg/100 mL.
14. A study of Australian fatalities indicated crash risk increased at THC concentrations greater than 5 ng/mL.
There is general agreement between studies with the first 10 points and some of these, such as passive exposure and chronic use, will be discussed further later in this report. The last four points require critical assessment in light of studies carried out since the report was written.

In 2005, based on the literature available at the time, the international expert panel found that blood THC concentrations of 3.5 to 5 ng/mL may achieve reasonable separation of impaired from unimpaired drivers [45]. Some later studies are summarised below.

A 2005 study [44] looked at the relationship between sobriety tests, simulated driving, and blood THC concentrations. Subjects smoked cannabis with concentrations of 1.74% w/w or 2.93% w/w (weak by today’s standards). Sobriety tests were carried out at 5, 55 and 105 min post-smoking and simulated driving tests were carried out at 30 and 80 min post-smoking. Driving performance was not significantly impaired at 30 min but was significantly impaired at 80 min for both plant concentrations. Blood THC concentrations were below 2.5 ng/mL at 125 min for both plant concentrations.

A 2006 study [47] looked at the relationship between THC blood concentrations and impairment in apprehended drivers. Those drivers assessed as impaired had blood THC concentrations ranging from 0.3 to 45.3 ng/L (median 2.5 ng/mL). Those drivers assessed as not impaired had blood THC concentrations ranging from 0.32 to 24.8 ng/mL (median 1.9 ng/mL). Although there is a complete overlap in the blood THC concentrations in the impaired and non-impaired drivers, and very similar median values, the authors of this paper reported that if the driver had a blood concentration greater than 3 ng/mL there was a greater likelihood of them being identified as impaired.

A 2013 systematic review of the literature relating cannabis effects on driving found that blood THC concentrations of 2 to 5 ng/mL were associated with substantial impairment [48].

A 2015 study [49] of blood THC concentrations in conjunction with field sobriety testing found no correlation between blood THC concentrations and the ability to perform sobriety tests. The study found that driving behaviour after using cannabis was similar to alcohol; namely, speeding and an inability to maintain the lane.

A 2015 study [50] found blood THC concentrations in arrested drivers had a mean of 5 ng/mL and a median of 3 ng/mL (half the arrested drivers had blood concentrations less than 3 ng/mL).

A paper published in 2016 [51] pointed out that the major concern with per se limits for cannabis was the effect of the time of blood collection on the blood THC concentrations. Typically, blood may be collected 1 to 4 h after a driver is stopped. Depending on how recently the cannabis had been smoked, blood THC concentrations might drop rapidly and erratically. There is broad inter-individual variability in the rate at which THC blood concentrations drop, but it is not possible to back-extrapolate a blood THC concentration. From the time of the highest blood concentration, while the cannabis is being smoked, the blood THC concentration decreases an average of 75% within 0.5 h, with a range of 12% to 90%. The average decrease is 90% within 1.5 h after smoking, with a range of 54% to 100%. In this study only one person had a blood THC concentration greater than 5 ng/mL, 3.3 h after smoking. Acute effects are expected to last 3 to 4 h.
In summary, while higher blood THC concentrations can be associated with recent use and likely impairment, lower blood THC concentrations do not mean lower impairment. There is very little correlation between blood THC concentrations and impairment.

In 2005, based on meta-analysis of the literature available at the time, the international panel determined that a THC blood concentration of about 5 ng/mL equates to a BAC of 80 mg/100mL and THC serum concentrations of 7 to 10 ng/mL (equivalent to 3.5 to 5 ng/mL whole blood) correlated to impairment of BAC 50 mg/100mL [45].

The Panel strongly disagrees with the concept of equating the degree of impairment of a BAC to that of a blood drug concentration.

In general terms, the degree of impairment for a particular drug (e.g., THC), including alcohol, is a combination of pharmacodynamics (effects of a drug at its biological targets) as well as pharmacokinetics (the drug’s concentrations in blood, oral fluids or other tissues such as brain, where the drug has its effect). Whilst there will be a relationship between the concentration of a drug at its target and its ability to impair driving skills, as well as a relationship between the exposure to a drug and the concentration achieved in a body fluid, there is no simple relationship between the dose of a drug and the resultant impairment of driving.

Different drugs do not exert their effects at the same pharmacological targets within the body, and even within the same class of drugs, such as the opioids or the sedatives, different members of the class have different potencies. Thus, they have different pharmacodynamic properties.

The concentration of a drug in blood and oral fluid at any given time is dependent not just on the dose itself, but also factors such as:

- Route of administration
- Time since the last dose
- Cumulative effect(s) of previous doses
- Ability of an individual to eliminate the drug from their body

It is well accepted that alcohol can affect driving performance and that there is a strong correlation between increasing BAC and increased crash risk. However, the degree of impairment displayed by individuals at a BAC of 80 mg/100mL or 50 mg/100mL is not the same. In the same way the degree of impairment displayed by individuals under the influence of other drugs, such as cannabis, will not be the same.

The determination of crash risk associated with alcohol use is possible because of the prevalence of use, legality of the drug, and ease of analysis. This has made it possible to carry out large epidemiological studies of alcohol use and crash risk. The legislative BAC limits are set at a level of crash risk deemed to be acceptable to government.

While the prevalence of cannabis use in the driving population has resulted in many reports assessing crash risk, the results are not consistent.
The 2005 report [45] from the international expert panel based its findings on meta-analysis of data available to that time. They equated a blood THC concentration of about 5 ng/mL to a BAC of 80 mg/100mL, and serum THC concentrations of 7 to 10 ng/mL (equivalent to 3.5 to 5 ng/mL whole blood) to impairment of BAC of 50 mg/100mL.

Literature reviews and meta-analyses carried out more recently have reported:

- Blood THC concentrations of 2 to 5 ng/mL are associated with substantial impairment [48].
- There is no correlation between blood THC concentration and ability to perform sobriety tests [49].
- There is no evidence of increased crash risk with use of cannabis or cannabis with alcohol [52].

Many assessments of crash risk and cannabis use are based on culpability studies and blood THC concentrations found in driver fatalities. Apart from the inherent problem of comparing blood drug concentrations taken from deceased and living individuals, such analyses tend to deal with relatively small populations.

One frequently quoted culpability and crash risk study is an Australian study [53] of driver fatalities that found an increased crash risk at blood THC concentrations greater than 5 ng/mL.

However, a study carried out on NZ driver fatalities [54] used identical methodology to the Australian study [53] and assessed a larger number of deceased drivers who had used cannabis (and no other drug). This study found a greater crash risk at blood THC concentrations less than 2 ng/mL than levels greater than 2 ng/mL.

In conclusion the panel believes that it is not appropriate to correlate blood drug concentrations directly to blood alcohol concentrations. The degree of impairment displayed by individuals at a BAC of 80 mg/100mL or 50 mg/100mL is not the same. The degree of impairment displayed by individual with the same blood THC concentration will not be the same. Furthermore, the variability in the findings in relation to blood THC concentrations and impairment demonstrate the complexities of cannabis and driving impairment.

9.1.3 Oral fluid and blood THC concentrations from various exposure routes

Inhalation

Like blood, maximum oral fluid concentrations occur while cannabis plant is being smoked, however the correlation between oral fluid and blood THC concentrations is poor. Oral fluid concentrations cannot be used to predict a blood THC concentration. After smoking, cannabis concentrations of THC are higher in oral fluid than in blood for several hours [55,56].

There are wide variations in oral fluid/blood THC concentration ratios between individuals, but also for an individual, following smoking or vaping cannabis [55]. One study found that, after smoking cannabis, the oral fluid/blood THC ratio at 1 hour ranged between 0.2 to 350 (median = 6) [26].
Another study found that, while it is not possible to use an oral fluid THC concentration to predict a blood concentration, the optimum oral fluid cut-off was 1.2 ng/mL for a LOQ in blood of 0.5 ng/mL and an oral fluid cut-off of 5 ng/mL was required for THC blood concentrations of 2 ng/mL [57].

These studies have considered inhalation of THC by smoking the plant material or vaping an extract of the plant. There are other methods of inhalation which may further affect the relationship between oral fluid and blood concentrations. These include inhaling the fumes from heated cannabis oil, an extremely concentrated form of cannabis product, or use of a bong.

**Passive exposure**

Smoking cannabis can expose other people in the vicinity to THC (passive exposure), thus there are concerns that passive exposure to cannabis smoke may result in a positive oral fluid roadside test. Several studies have addressed this issue directly:

In a 2005 study, four passive and four active smokers were placed in an unventilated eight-seater van. Oral fluid was collected from the passive participants both inside and outside the van [58]. When the oral fluid was collected in the contaminated environment inside the van, the highest oral fluid THC concentration was 7.5 ng/mL. After the passive smokers left the van for the testing process, oral fluid THC concentrations up to 1.2 ng/mL were detected. The oral fluid concentrations declined sufficiently to give a negative result 30 min after leaving the van.

In a 2011 study, participants were exposed to cannabis smoke for 3 hours in a coffee shop in the Netherlands [59]. Oral fluid specimens were collected outside after exposure for 20, 40, 60, 120 and 180 min, then 12 h later. THC was detected in oral fluid but the amount varied, likely depending on the number of cannabis smokers in the coffee shop. Few of the oral fluid samples collected from the passive participants exceeded 5 ng/mL during the three-hour exposure period, and all were below 5 ng/mL 1 h after exposure.

In a 2015 study designed to produce extreme cannabis smoke exposure, six passive and six active smokers were placed in closed chambers [60]. Two sessions were carried out with no ventilation and one session allowed ventilation of the chamber. Oral fluid concentrations greater than 5 ng/mL were detected for up to 2 h after exposure in the unventilated environment. When the chamber was ventilated, results were variable, and only some of the passive participants achieved oral fluid concentrations greater than 5 ng/mL. THC was detected in the blood in the passively exposed participants. In the unventilated environment blood THC concentrations generally decreased to below 1 ng/mL with 1 h. When the chamber was ventilated blood THC concentrations did not reach 1 ng/mL.

In 2009 an ESR study looked at THC concentrations in oral fluid either following smoking a single cannabis cigarette or following passive exposure. While eight subjects each smoked one cannabis cigarette (1.3 g cannabis (3.3% THC)), eight other participants (who did not smoke a cannabis cigarette) were exposed to the cannabis smoke in the same small room (i.e, passive exposure). Oral fluid specimens were collected and analysed.
The oral fluid THC concentrations detected due to passive inhalation in these extreme exposure conditions can be seen in Table 6. Oral fluid THC concentrations in following passive exposure in an enclosed room. It was noted that the passive smoker who had quantifiable oral fluid THC concentrations for the longest, had been seen kissing an active smoker.

Table 6  Oral fluid THC concentrations in following passive exposure in an enclosed room

<table>
<thead>
<tr>
<th>Time (h)</th>
<th>Oral fluid THC concentrations (ng/mL) after passive exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>nd</td>
</tr>
<tr>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>0.25</td>
<td>9.4</td>
</tr>
<tr>
<td>0.5</td>
<td>7.6</td>
</tr>
<tr>
<td>0.75</td>
<td>8.8</td>
</tr>
<tr>
<td>1</td>
<td>7.6</td>
</tr>
<tr>
<td>1.5</td>
<td>2.8</td>
</tr>
<tr>
<td>2</td>
<td>det</td>
</tr>
<tr>
<td>2.5</td>
<td>det</td>
</tr>
</tbody>
</table>

det = >0.2 but <2 ng/mL, nd = not detected, LLOQ (lower limit of quantitation) = 2 ng/mL.

Taken together, these studies indicate that it is possible for a person to test positive for THC after passive smoking, and also to achieve a concentration associated with impaired driving ability, but only under conditions where the person exposed (e.g., a driver) was subjected to extreme and prolonged exposure.

The other methods used for inhalation of cannabis products are unlikely to result in similar environmental exposure to THC when compared to cigarette smoking, but this requires further investigation.

Based on these studies, it is unlikely that passive exposure to cannabis smoke under realistic exposure conditions will result in a positive oral fluid test at the roadside.

**Ingestion of cannabis edibles**

Both blood and oral fluid THC concentrations are lower when food containing cannabis (cannabis edibles) is ingested, when compared with smoking [61,62].

Maximum blood THC concentrations occur 2 to 4 h after ingestion. Psychoactive effects are also delayed, with maximum effects reported 2 to 4 h after ingestion. Effects following ingestion are reported to be more intense than when the drug is smoked. One study found that the concentrations of THC in oral fluid and blood were similar when the psychoactive effects were at the maximum [63].

The 2009 ESR study looked at THC concentrations in oral fluid following oral ingestion and smoking cannabis. Four subjects were provided with a single cannabis-laced Afghan cookie (1.3 g cannabis (3.3% THC) per cookie, approximately 43 mg THC). Eight subjects each smoked one cannabis cigarette (1.3 g cannabis (3.3% THC)). Oral fluid specimens were collected and analysed. The results of part of
this study can be seen in Table 7. Comparison of oral fluid THC concentrations following oral ingestion and smoking cannabis.

**Table 7** Comparison of oral fluid THC concentrations following oral ingestion and smoking cannabis

<table>
<thead>
<tr>
<th>Time (h)</th>
<th>Oral fluid THC concentrations (ng/mL) after eating cookie</th>
<th>Oral fluid THC concentrations (ng/mL) after smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Oral fluid THC concentrations after eating cookie</td>
<td>Oral fluid THC concentrations after smoking</td>
</tr>
<tr>
<td></td>
<td>after eating cookie</td>
<td>after smoking</td>
</tr>
<tr>
<td>Baseline</td>
<td>det</td>
<td>nd</td>
</tr>
<tr>
<td>0</td>
<td>87</td>
<td>133</td>
</tr>
<tr>
<td>0.25</td>
<td>5</td>
<td>59</td>
</tr>
<tr>
<td>0.5</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>0.75</td>
<td>35</td>
<td>5</td>
</tr>
<tr>
<td>1</td>
<td>36</td>
<td>10</td>
</tr>
<tr>
<td>1.5</td>
<td>31</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>det</td>
<td>det</td>
</tr>
<tr>
<td>2.5</td>
<td>det</td>
<td>det</td>
</tr>
</tbody>
</table>

det = >0.2 but < 2 ng/mL, nd = not detected, LLOQ (lower limit if quantitation) = 2 ng/mL

Oral fluid concentrations following ingestion of cannabis in Afghan cookies were significantly lower than following smoking. As stated previously, the maximum psychoactive effects occur 2 to 4h after ingestion of the drug. From Table 7 it can be seen that, for most of the participants eating the cookie, oral fluid concentrations were below detection limits before the maximum psychoactive effects would be felt.

These results are comparable to a similar study in which three participants ate brownies containing 20 to 25 mg THC [64]. One hour after ingestion, oral fluid concentrations were 2.2, 6.9 and 5.2 ng/mL. At 4 h oral fluid concentrations were 0.7, 3.0 and 0.4 ng/mL.

Oral fluid roadside testing devices might not detect ingestion of cannabis-containing food products at the time the effect of the drug is at its maximum.

**Hemp-based food ingestion**

Hemp is the common name for cannabis that has a very low concentration of THC. Hemp has been grown in NZ for over 20 y, but it is only since 2018 that food products made from hemp seeds have been allowed to be sold commercially. Hemp seeds can be used to make a variety of food products: hemp seed oil, hemp milk, hulled hemp seeds, hemp protein powders and hemp seed butter (used like peanut butter).

The inside of the hemp seed contains no cannabinoids [65] but the leaves surrounding the seeds are rich in cannabinoids. Therefore, the concentration of cannabinoids in hemp seed food products will depend on how well the seeds are cleaned prior to processing. In 1997 several articles appeared in the scientific literature about positive urine tests after ingestion of hemp food products [66-69]. In addition, cannabinoids had been detected in blood and urine following ingestion of hemp tea and hemp milk [70].
Since then, the regulations covering the amount of THC allowed in such products have been tightened. Since 1998 more thorough seed drying and cleaning has considerably reduced THC concentrations in hemp seed-based products. Compliance with these tightened regulations, means that urine cannabis tests no longer give a positive response after consumption of such products [71].

Hemp seed oil (i.e., edible oil made from hemp seed kernels) contains the most THC when compared with other hemp seed food products. As in most countries where hemp food products are sold, NZ regulations require THC concentrations in food products to be less than 10 milligrams of THC per kilogram (mg/kg) of oil. Therefore, ingestion of 15 mL (approx. one tablespoon) of hemp seed oil containing the maximum allowed amount of THC is equivalent to ingesting 0.15 mg THC.

The oral fluid THC concentrations detected after ingestion of cookies containing 20 to 40 mg THC/cookie reported in the previous section [64] were low and near to the limit of detection of the currently available roadside oral fluid test devices. Ingestion of hemp seed-based food containing 100 times less THC would not give rise to a positive oral fluid screening test.

A study carried out by Swinburne University, Melbourne, Australia showed that ingestion of 5 mL (approx. one teaspoon) of spiked oil containing 10 or 20 mg/kg THC, did not result in a positive oral fluid test or detectable THC in the blood [72].

Based on these studies it is clear that ingestion of hemp seed-based food will not result in a positive oral fluid screening test or detectable THC in the blood.

**Cannabis as a medicine**

Currently the only medicinal products containing high amounts of THC available in NZ are the imported Sativex and Tilray products, usually administered as oral sprays. Sativex and some Tilray products contain approximately equal amounts of THC and CBD. These and future medicinal products may deliver impairing doses of THC that will be detected in oral fluid and blood.

Concern about driving impairment following use of medicinal cannabis led to a review of the literature which found that most driving experiments are only carried out for 2 to 3 h after cannabis administration; however, the authors of this study recommended that cannabis-treated patients should not drive for 8 h after use [73].

A study compared blood THC concentrations following oromucosal administration and capsulated forms [74] of medicinal Sativex, 5 and 15 mg doses. The higher dose formulations resulted in maximum blood THC concentrations ranging from 1.6 to 19.0 ng/mL at any time from 1.2 to 5.5 h after administration.

A 2014 study [75] reported oral fluid THC concentrations following use of an oral cannabinoid spray. Using the Sativex high dose regime (i.e., 8 sprays delivering 21.6 mg THC) oral fluid THC concentrations measured about 15 min after dosing were of the order of 10,000 ng/mL (10 μg/mL). Two hours after dosing, oral fluid THC concentrations in the five participants were still just over 100 ng/mL. These high oral fluid concentrations do not mean the blood THC concentrations are similarly elevated at the same time.
If cannabis is taken in a way that avoids oral mucosal contamination, such as by capsule, there will be no THC in the oral fluid [26]. However, blood THC concentrations and potential impairment would be expected to be similar to that seen following ingestion of cannabis edibles.

THC used as a medicine has the potential to impair driving. However, detection of medicinal cannabis use by oral fluid roadside testing will depend on the route of administration.

**Chronic cannabis use**

Following chronic use of cannabis, blood THC concentrations can remain elevated. Chronic use may be defined as daily use of the equivalent of several cannabis cigarettes. Chronic use of cannabis will result in elevated blood THC concentrations being detected for a longer time than following a single dose (i.e., steady state concentration; this means that the absorption and blood clearance of THC are in equilibrium).

In one study [75], 30 chronic cannabis smokers were held in a secure research unit and abstained from cannabis for one month. Blood THC concentrations on admission ranged from 0.3 to 6.3 ng/mL (median 1.4 ng/mL). The median blood THC concentration on day 1 was less than 2 ng/mL and the median concentration on day 2 was less than 1 ng/mL. However, it was only after day 10 that blood THC concentrations of all 30 participants fell below 2 ng/mL.

Oral fluid THC concentrations may also remain elevated following heavy use of cannabis. One study [76] found that oral fluid THC concentrations took between 2.5 and 30.0 h to drop below 5 ng/mL and blood THC concentrations took between 2.5 and 150.0 h to drop below 2 ng/mL. Furthermore, THC-acid may be detected in oral fluid of heavy cannabis users [56]. Oral fluid testing devices will also detect the presence of THC-acid which is an inactive metabolite of THC.

A number of studies have also assessed the driving performance of chronic cannabis users, with mixed results. The results of these studies vary, from the finding that heavy use had little effect on critical thinking or divided attention tasks [77], to finding persistent cognitive decrements after weeks of abstinence, including deficits in attention, concentration, decision-making, concept formation and planning [78]. This significant variability makes linking blood THC concentrations to degree of impairment difficult, if not impossible.

A 2018 study [79] found that chronic cannabis users with plasma THC concentrations of 2 to 5 ng/mL (equivalent to blood THC concentrations of 1 to 2.5 ng/mL) exhibited poorer driving performance than non-users. These plasma concentrations were considered to indicate that the chronic users used in the study had not used the drug recently. These findings supported an earlier (2016) study [80] which found that chronic cannabis users have an extended duration of impairment.

**9.1.4 Duration of detection of cannabis use in biological samples**

Concern has been expressed regarding the validity of testing drivers for evidence of cannabis use. It has been claimed that cannabis use can be detected in biological fluids for weeks after its use. This is not true for the detection of THC in oral fluid or blood.
The time after administration for which any drug can be detected in biological fluids depends on which biological sample is analysed. Drugs are detectable for the shortest time in oral fluid and blood, and are detectable for a longer period in urine, and even longer in hair samples.

After smoking cannabis, blood THC concentrations rise and drop rapidly. After smoking a single cannabis cigarette, assuming the blood was clear of any previous cannabinoids, THC may be detected for up to 12 h. For some individuals, blood THC concentrations drop to below detection limits within a few hours of smoking a cannabis cigarette.

Like blood, maximum oral fluid concentrations occur while cannabis is being inhaled. However, after smoking cannabis oral fluid THC concentrations can decline more slowly and might remain higher than blood concentrations for several hours [55].

Chronic use of cannabis results in higher THC blood concentrations that persist for longer than following single or infrequent use. In support of this, one study [80] showed that chronic users also have an extended duration of impairment.

Evidence of cannabis use can be detected for a longer period if urine is the biological sample tested. Urine is analysed for the presence of THC-acid, the metabolite of THC. For the infrequent cannabis user, THC-acid may be detected in urine for two or three days after the use of the equivalent of a single cannabis cigarette. Chronic use (several cigarettes daily for an extended time) results in an extended detection period of the metabolite in the urine. A study involving 22 chronic drug abusers [81] found that it took between 0 and 19 days to obtain a negative urine result after cessation of cannabis smoking.

9.1.5 Blood THC concentrations in NZ drivers

Deceased drivers

Table 8 shows occurrence of cannabis taking and THC concentrations in the blood of NZ drivers killed in road traffic accidents.

Table 8 Occurrence of cannabis taking and THC concentrations in deceased NZ drivers.

<table>
<thead>
<tr>
<th>Years</th>
<th>Number of Drivers</th>
<th>Positive for Cannabis</th>
<th>THC Concentration Range ng/mL</th>
<th>Mean THC Concentration ng/mL</th>
<th>Median THC Concentration ng/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004 to 2009</td>
<td>1,046</td>
<td>314 (30%)</td>
<td>0.2 to 44</td>
<td>5.5</td>
<td>3</td>
</tr>
<tr>
<td>2013 to 2018</td>
<td>1,069</td>
<td>266 (25%)</td>
<td>0.2 to 100</td>
<td>7.2</td>
<td>4</td>
</tr>
</tbody>
</table>

It is interesting to note that:

- Between 2013 to 2018, 72% of deceased drivers had blood THC concentrations greater than 1 ng/mL and that 50% had blood THC concentrations greater than 3 ng/mL.
- Between 2013 to 2018, 38% of drivers using cannabis had also used alcohol.
**Impaired drivers**

For the period 2017 to mid-2020, blood samples from 1,679 impaired drivers were analysed. Of these drivers:

- 1,023 (61%) had used cannabis.
- 523 (31%) had used cannabis and no other drugs. Blood THC concentrations ranged from 0.2 to 40 ng/mL (mean 9.8 ng/mL, median 7 ng/mL).
- 52 (10%) had blood THC concentration less than 1 ng/mL.
- 96 (18%) had blood THC concentration less than 2 ng/mL.
- 132 (25%) had blood THC concentration less than 3 ng/mL.

**Hospitalised drivers**

For the period 2017 to mid-2020, blood samples from 2,796 hospitalised drivers were analysed. Of these drivers:

- 1,036 (37%) had used cannabis.
- 426 (15%) had used cannabis and no other drugs. Blood THC concentrations ranged from 0.2 to 40 ng/mL (mean 4.4 ng/mL, median 2.7 ng/mL).
- 110 (26%) had blood THC concentrations less than 1 ng/mL.
- 173 (41%) had blood THC concentrations less than 2 ng/mL.
- 227 (54%) had blood THC concentration less than 3 ng/mL.

**9.1.6 Use of blood THC concentrations in evidence**

The following points taken from the scientific literature and from blood THC concentrations in NZ drivers are important when considering the use of blood THC concentrations as evidence:

- Blood THC concentrations will likely drop below 3 ng/mL within 3 h of smoking an impairing cannabis dose.
- Impairment following the use of cannabis would be expected to last for 3 to 4 h.
- There will inevitably be a delay between stopping a driver and obtaining a blood sample.
- Of 523 drivers found to be impaired by a police CIT, 25% had THC blood concentrations less than 3 ng/mL, 10% had blood concentrations less than 1 ng/mL.
- Overseas jurisdictions have set *per se* limits for THC ranging from 1 to 9 ng/mL.

**Proposed statutory limit**

Based on the findings discussed in this section, the blood drug concentrations detected in NZ drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood THC limit of 3 ng/mL and a threshold limit of 1 ng/mL.
9.2 Stimulants

Stimulants (psychostimulants) are a class of drugs that increase activity of the CNS via interactions with specific receptors in the brain; as a result of this they can increase nervous stimulation of other parts of the body resulting increased activity and performance. They are prescribed (e.g., methylphenidate for treatment of Attention Deficit Hyperactivity Disorder (ADHD)) and used illicitly (e.g., methamphetamine) as performance enhancing or recreational drugs.

It may be dangerous to drive after using psychostimulants due to:

- overconfidence in driving skill that is not supported by actual improvement in driving ability,
- propensity to take unnecessary risks,
- aggressive and dangerous driving,
- impaired ability to react appropriately, and
- drivers suddenly falling asleep as the stimulant effects wear off [32].

A case-controlled study [82] estimated accident risk for alcohol, medicines and illegal drugs (using data from the Driving Under the Influence of Drugs (DRUID) project). For single drug use, the study attempted to determine the threshold at which a significant increase in risk was observed. In a study on amphetamine-type drugs, no clear threshold could be determined; indeed, there was a similar high risk at all blood drug concentrations.

9.2.1 Methamphetamine

Methamphetamine is a CNS stimulant: low dose administration increases alertness, but with increased dosing and duration of use there are disorienting effects on cognition, reasoning and psychomotor ability. After high-dose or chronic use, delusions and psychotic episodes may also occur [83].

Blood methamphetamine concentrations depend on the route of administration, the dose and the frequency that the drug is taken. Methamphetamine may be smoked, snorted, injected or ingested. The onset of effects is significantly faster for the first three routes of administration compared to the oral route.

Peak blood methamphetamine concentrations occur soon after injection, a few minutes after smoking, and around 3 h after oral dosing [32].

Doses associated with recreational use or abuse of methamphetamine may range from 100 mg/d to over 1,000 mg/d [32]. When 12 young men were given a 30 mg oral dose, an average peak blood methamphetamine concentration of 94 ng/mL was determined, 3 to 5 h after ingestion [84].

When 30 mg methamphetamine hydrochloride was smoked using a glass pipe, oral fluid methamphetamine concentrations were of the order of 10,000 ng/mL (10 μg/mL) immediately after smoking and dropped to below 500 ng/mL within 5 h. Maximum plasma methamphetamine concentrations after smoking this dose were less than 100 ng/mL and were still about 80 ng/mL 10 h after use [85].
**Passive exposure**

Inadvertent ingestion or exposure to methamphetamine may arise by inhalation of airborne methamphetamine or by hand-to-mouth contact, following dermal exposure to contaminated surfaces. Methamphetamine is water soluble (and so dissolves in airway fluids), thus little methamphetamine is lost in the exhaled breath as demonstrated by its high bioavailability [85]. However, some methamphetamine does become airborne as seen by contamination of houses where the drug has been smoked [86].

Studies have shown that methamphetamine can be adsorbed onto the skin by touching contaminated surfaces. The amount transferred depends on the type of surfaces and whether the hands are dry or wet, with transfer to wet hands (26%) being greater than to dry hands (11%) [87].

Many houses in NZ have been tested for methamphetamine where there has been suspicion of methamphetamine use and/or manufacture. In a study of surface contamination carried out by ESR, swabs from an area measuring 10 x 10 cm were analysed [86]. In the houses tested where contamination was through smoking rather than via manufacture, the median methamphetamine contamination was less than 5 micrograms per 10 square centimetres (µg/100 cm²). Fewer than 10% of swabs contained more than 20 µg/100 cm² and contaminations greater than 30 µg/100 cm² were indicative of manufacture [86].

This points to the amount of airborne methamphetamine being greatest when associated with clandestine manufacture of the drug. A 2008 study reported airborne methamphetamine following the manufacture of two batches of the drug [88]. In the kitchen, where the drug was manufactured, airborne methamphetamine was 520 µg/m³ after the first drug batch and 760 µg/m³ after the second batch. The following day, airborne methamphetamine concentrations had dropped to 70 to 210 µg/m³ depending on the amount of activity in the room [88].

In conclusion, environmental or passive exposure to methamphetamine does not result in measurable concentrations of methamphetamine in blood.

Blood methamphetamine concentrations do not decline quickly after dosing. The half-life of the drug ranges from 6 to 15 h [89]. As a consequence, the drug can be detected for 24 h or longer after its use.

The onset of effects following a methamphetamine dose is rapid following intravenous use and smoking, while the effects are slower following oral use. Overall, the stimulatory effects typically last 4 to 8 h, but residual effects can persist for up to 12 h [32].

The effects of methamphetamine are dependent on the dose, pattern of administration, and time elapsed since last use. Single dose use produces CNS excitation characterized by increased energy, euphoria and an elevated sense of confidence; sedation may follow the intense stimulatory experience [32].

Repeated administration of methamphetamine to prolong the drug effects and minimize the withdrawal effects, is a common pattern of use often referred to as ‘binge use’. Binge use may occur over hours or days but is frequently followed by a ‘crash’ phase which is accompanied by anxiety and fatigue. The stimulatory effects of methamphetamine diminish the desire to sleep; this might lead to a user being awake for an extended period. The resulting extreme tiredness...
can lead to the person falling asleep whilst driving even if they still have measurable blood methamphetamine concentrations [32].

As would be expected from the above, methamphetamine is known to affect the ability to drive safely. However, it has been suggested that low dose stimulants (e.g. methamphetamine) might improve performance. Indeed, driving simulation studies have shown increased alertness following low doses of methamphetamine [84]. Importantly, such doses are not typical of recreational methamphetamine use and do not apply to drug abuse situations.

In two studies of methamphetamine-positive drivers, blood concentrations ranged from less than 50 ng/mL to 9,460 ng/mL (~9.5 μg/mL). Common observations in these studies included rapid and confused speech, rapid pulse, agitation, paranoia, and violent/aggressive behaviour. Erratic driving, speeding and weaving were some of the reported driving observations [90,91].

Importantly, studies have found no correlation between blood methamphetamine concentrations and increased risk of crashing because the use of methamphetamine increases crash risk at all blood concentrations determined [82].

Since 2009, the state of Victoria in Australia has had compulsory alcohol and drugs testing of all injured drivers. The crash risk associated with drug use has been assessed using culpability analysis for nearly 5,000 injured drivers. Alcohol increased crash risk with increasing BAC. Methamphetamine also increased crash risk, but there was no correlation with blood methamphetamine concentration [92].

Oral fluid testing devices cannot distinguish between methamphetamine and MDMA; therefore, a positive oral fluid result could be due to use of either drug [25]. The oral fluid testing devices claim to detect methamphetamine at concentrations ranging from 35 to 50 ng/mL.

Methamphetamine is generally detected in oral fluid at higher concentrations than is present in blood. Although there have been no controlled methamphetamine smoking studies, long-term users are likely to have detectable methamphetamine in oral fluid for several days after dosing [56].

In the years from 2017 to mid-2020, blood taken from 1,679 NZ impaired drivers was analysed for evidence of drug use. Of these drivers, 823 (49%) had used methamphetamine. Of these 823 drivers, methamphetamine was the only drug detected in 326 drivers (19% of the total impaired drivers tested). Of the 326 methamphetamine positive drivers who had used methamphetamine alone, 305 (94%) had blood methamphetamine concentrations greater than 50 ng/mL.

In the years from 2017 to mid-2020, blood taken from 2,796 hospitalised drivers was analysed for evidence of drug use. Of these drivers, 771 (28%) had used methamphetamine. The blood methamphetamine concentrations in these 771 drivers ranged from 10 to 2,000 ng/mL (average 240 ng/mL, median 170 ng/mL) and 645 (84%) of the drivers had blood methamphetamine concentrations greater than 50 ng/mL.

The UK, Norway and Denmark governments have set per se limits for methamphetamine at 10, 48 and 32 ng/mL respectively [1-3].
Proposed statutory limit
Based on the blood drug concentrations detected in NZ drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood methamphetamine limit of 50 ng/mL and a threshold limit of 10 ng/mL.

9.2.2 Amphetamine
Amphetamine is a CNS stimulant with effects similar to methamphetamine, albeit with lower potency [93]. Amphetamine is more commonly used than methamphetamine in many European countries but to date, amphetamine is rarely used in NZ.

Amphetamine is also a metabolite of methamphetamine and is usually detected when methamphetamine is present in blood. However, blood concentrations of amphetamine from metabolism of methamphetamine are rarely greater than 10% of the blood concentration of the methamphetamine itself [32], thus it is possible to distinguish the use of amphetamine itself from its presence due to methamphetamine use and consequent metabolism.

Like other stimulants, oral fluid amphetamine concentrations are greater than blood concentrations [56]. That said, amphetamine is not detected by the same immunoassay screen as methamphetamine, so will not be detected by the methamphetamine channel on an oral fluid screening device - a separate channel is used for amphetamine. Oral fluid testing devices claim to detect amphetamine in oral fluid at 50 to 60 ng/mL.

From 2017 to mid-2020, amphetamine use has been detected in three impaired drivers in NZ. Blood amphetamine concentrations were 40, 70 and 170 ng/mL in these three drivers.

The UK, Norway and Denmark have set per se limits for amphetamine of 250, 41 and 32 ng/mL respectively [1-3].

Proposed statutory limit
Based on the blood drug concentrations detected in NZ drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood amphetamine limit of 100 ng/mL and a threshold limit of 20 ng/mL.

9.2.3 Methyleneoxyamphetamine (MDMA)
MDMA is a mild to moderate CNS stimulant with hallucinogenic properties. MDMA toxicity may include hyperthermia, seizures, tachycardia and hypertension [94].

No clear correlation exists between blood MDMA concentrations and effects [32]. Toxicity due to MDMA is variable among individuals and there is a significant overlap between concentrations associated with minimal toxicity, impairment and concentrations associated with fatal overdose [95,96].
An intoxicating dose of MDMA is considered to be approximately 100 mg. Such doses can cause acute changes in cognitive performance and impair information processing, which in turn impair driving ability [32]. An average peak plasma concentration of 200 ng/mL was detected 1.5 to 2.0 h following an oral 100 mg dose of MDMA.

The blood half-life of MDMA ranges from 4 to 12 h [84]. Onset of effects is rapid following snorting, while the effects are felt more slowly, 20 to 30 min, following oral use. Overall, the effects last typically for 2 to 3 h depending on the dose [32]. As a consequence, the drug can be readily detected for longer than its effects.

A simulator study was carried out to determine the effect of methamphetamine and MDMA on driving [97]. Volunteers were dosed with either 100 mg MDMA or 0.42 mg/kg body weight methamphetamine. The mean peak blood MDMA concentration at 3 h was 200 ng/mL, and the mean peak blood methamphetamine concentration was 90 ng/mL. In this study, the drivers using MDMA performed less well than drivers using methamphetamine.

MDMA tablets are not commercially prepared; users/buyers do not know the MDMA content per tablet (i.e., strength of the tablet) or the purchased batch. The UK expert panel [5] reported that MDMA tablets contained 40 to 75 mg. However, in a recent ESR study of 37 tablets seized at the NZ border in 2019, the MDMA content ranged from 44 to 290 mg per tablet (mean 176 mg, median 168 mg).

In general, the oral fluid concentrations of stimulant-type drugs are higher than blood concentrations, but this depends on the route of administration. MDMA is commonly found in tablet form and may be swallowed, therefore resulting in low oral fluid concentrations.

As discussed previously, oral fluid testing devices cannot distinguish between methamphetamine and MDMA: a positive result could be due to use of either drug, although the sensitivity of devices is greater for methamphetamine than for MDMA [25]. Information provided for the Dräger device specifies this difference with detection of methamphetamine at 35 ng/mL and MDMA at 75 ng/mL.

The use of MDMA is often combined with alcohol and other drugs. Of the 44 NZ impaired drivers found with MDMA in their blood from 2017 to mid-2020, only two (4.5%) had not used other drugs. Similarly, of the 73 hospitalised drivers over the same time period, only nine (12%) were found with only MDMA in their blood.

Blood MDMA concentrations for the 44 impaired NZ drivers ranged from 10 to 1,700 ng/mL (mean 250 ng/mL, median 80 ng/mL). Blood MDMA concentrations from the 73 hospitalised drivers ranged from 10 to 7,300 ng/mL (mean 260 ng/mL, median 70 ng/mL).

The UK, Norway and Denmark governments have set per se limits for MDMA of 10, 48 and 21 ng/mL respectively [1-3].
**Proposed statutory limit**

Based on the blood drug concentrations detected in NZ drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood MDMA limit of 50 ng/mL and a threshold limit of 10 ng/mL.

9.2.4  *Methylenedioxyamphetamine (MDA)*

MDA is a metabolite of MDMA, but is also a drug in its own right. It is a CNS stimulant with considerably greater potency than MDMA [32].

When MDMA is detected in blood, MDA is usually also detected. The finding of a metabolite in blood is not generally reported in LTA certificates, so when MDA is detected in the blood with MDMA, only MDMA is reported (this is to avoid ‘double counting’ of drug use). Use of the drug MDA itself can be distinguished readily from its presence due to MDMA use because the blood concentration of MDA when present as a MDMA metabolite is rarely greater than 10% of the concentration of MDMA.

In NZ, MDA itself is not a commonly used drug. It has been detected in a driver only once in the years from 2017 to mid-2020, at a concentration of 410 ng/mL.

Overseas jurisdictions have not set *per se* limits for MDA.

**Because of the paucity of data for MDA, its very low use in NZ, and because other jurisdictions have not set per se limits, the Panel does not propose a per se limit for MDA.**

9.2.5  *Cocaine*

Cocaine is a potent CNS stimulant. The routes of administration for cocaine are dependent on its form and include snorting, smoking or injection. The onset of effects is rapid, from seconds to minutes after dosing, irrespective of the route of administration [32].

Cocaine is rapidly metabolized and has a short half-life in the body (0.6 to 1.0 h) [32]. Any time delay between a driver being stopped and a blood sample being taken will result in a significant reduction in the concentration of cocaine in the blood. Benzoylecgonine is an inactive metabolite and breakdown product of cocaine and its presence in blood indicates cocaine use.

Cocaine generally has a higher concentration in oral fluid than blood, and may be detected in oral fluid up to 6 h after smoking and up to 12 h after snorting [56]. Manufacturers of oral fluid detection devices claim to detect cocaine in oral fluid at concentrations between 10 and 30 ng/mL with one device also reported as detecting benzoylecgonine at 70 ng/mL.

Cocaine is rarely detected in NZ drivers. During the period 2017 to 2019 only one cocaine-impaired driver was detected – the person had a blood cocaine concentration of 30 ng/mL. Benzoylecgonine has been detected in the blood of some drivers but this is not generally reported as it is a metabolite, not a drug listed in the Misuse of Drugs Act 1975.
Some countries have set a *per se* limit for cocaine and benzoylecgonine. *Per se* limits for cocaine of 10, 24 and 21 ng/mL have been implemented in the UK [1], Norway [2] and Denmark [3] respectively. The UK also set a *per se* limit for benzoylecgonine of 50 ng/mL.

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood cocaine limit of 20 ng/mL and a threshold limit of 5 ng/mL.

9.3 Gamma-hydroxybutyrate (GHB)

GHB is used therapeutically as an alcohol withdrawal treatment due to the similarities in its effects, and is also used for the treatment of narcolepsy. It is also a drug of abuse in various settings and may be abused/used by body-builders and in social settings such as nightclubs.

Gamma-butyrolactone (GBL) and 1,4-butanediol are chemically related to GHB and are used as GHB substitutes. These compounds are commercially available as industrial solvents and are used as ingredients in cleaners, solvents, paint removers, and engine degreasers. After ingestion, GBL and 1,4-butanediol are rapidly converted to GHB by enzymes in the blood.

The presence of GHB in blood may be due to the ingestion of GHB itself, GBL or 1,4-butanediol.

GHB is a powerful CNS depressant. At low doses, its effects are similar to alcohol’s [32]. The signs of behavioural effects and impaired performance when under the influence of GHB include erratic driving, ignoring road signs and near-collisions [32]. The onset of effects occurs within 10 to 20 min of dosing and the effects generally last for 2 to 5 h.

Following oral administration, peak plasma concentrations are achieved within 20 to 45 min. The half-life of GHB in blood is in the region of 20 to 40 min [98]. This means that even after high doses, GHB is undetectable in blood within 4 to 6 h. An hour delay between the time of an infringement to the time a blood sample is taken, would mean a blood GHB concentration of 100,000 ng/mL (100 μg/mL) could drop to about 10,000 ng/mL (10 μg/mL: i.e., 90% decline).

A Swedish study of GHB concentrations in blood taken from 473 impaired drivers over a 10-year period reported an average blood concentration of 90,000 ng/mL (maximum 340,000 ng/mL; 90 μg/mL, maximum 340 μg/mL) [99].

GHB is not detected by oral fluid screening tests. Use of the drug is generally inferred by observed poor driving and a failed CIT.

Laboratory drug analysis procedures usually detect a range (suite) of drugs in a single analysis; however, it is not possible for all drugs to be included in a single drug suite. GHB is not detected by the analytical methods usually applied to LTA samples; therefore, GHB analysis needs to be requested specifically by the police so the appropriate analysis can be carried out.
The blood GHB concentrations found in samples from three NZ impaired drivers since 2017 were 45,000, 70,000 and 90,000 ng/mL (45, 70, 90 μg/mL respectively). In each case, use of the drug was suspected and the analysis was specifically requested by the Officer in Charge.

Anecdotal information from Australia indicating that GHB was commonly used with methamphetamine led to an ESR study of 290 drivers who had used methamphetamine. This study found 42 of the drivers (14%) had also used GHB. Blood GHB concentrations in these drivers ranged from 15,000 to 200,000 ng/mL (mean 80,000 ng/mL; 15-200 μg/mL, mean 80 μg/mL). It is clear from this study that GHB use by drivers in NZ is more prevalent than previously thought.

Norway has specified a graduated per se limit for GHB of 10,300 to 123,600 ng/mL (10.3-123.6 μg/mL) [2]. A per se limit for GHB has not been specified in the UK or Denmark.

**Proposed statutory limit**

Based on the blood drug concentrations detected in NZ drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood GHB limit of 50,000 ng/mL and a threshold limit of 10,000 ng/mL.

9.4 **Ketamine**

Ketamine is classified as a dissociative anaesthetic (i.e., involves catalepsy, catatonia, analgesia and amnesia, but not necessarily loss of consciousness). Patients should be cautioned against operating hazardous machinery for at least 24 h after ketamine administration [84] because of its pharmacological effects.

Ketamine is being researched in New Zealand (and other countries) for the treatment of drug-resistant and severe depression as well as for the treatment of acute suicidal ideation. It is available in both intranasal and oral forms. A recent review [100] outlines the efficacy of ketamine for these conditions. According to the authors “Ketamine bioavailability by oral route varies from 17% with 0.5 mg/kg to 30% with 50 mg of racemic ketamine, because of an extensive first-pass metabolism”. The authors also report that “Racemic ketamine bioavailability by intranasal route is higher than oral route and reaches 45% with a dose of 25 mg”. Ketamine pharmacokinetics reveal that the elimination half-life is 2 to 4 h with the distribution half-life in the range of 2 to 4 min.

Ketamine is also abused recreationally, when it may be administered by injection, smoking, snorting/intranasally or orally [32].

Recreational doses are variable and dependent on the route of administration. Commonly used doses are reported to be 25 to 50 mg when taken intramuscularly, 30 to 75 mg when snorted and 75 to 300 mg when taken orally [32]. Resulting blood concentrations depend on the dose taken and the route of administration. For example, three healthy men given a single oral 50 mg dose achieved peak plasma concentrations averaging 80 ng/mL [89].
There is no direct correlation between blood ketamine concentrations and behaviour. Drowsiness, perceptual distortions and intoxication may be dose-related in a concentration range of 50 to 200 ng/mL, and analgesia begins at plasma concentrations of about 100 ng/mL [32].

The half-life of ketamine is 2 to 4 h. This means that the drug is unlikely to be detected 12 to 15 h after a dose.

Currently (2021) there is little evidence that ketamine is commonly used recreationally in NZ. However, with the indication of efficacy for severe depression and the availability of both oral and intranasal forms, it is likely that it will become more commonly available and perhaps abused. While ketamine is frequently detected in blood taken under the LTA, these samples have all come from drivers who have been hospitalised following a crash and it is most likely that the drug has been administered by medical personnel as part of treatment.

Ketamine is not detected by oral fluid screening tests. Use of the drug will generally be inferred by observed poor driving and a failed CIT.

Some countries have per se limits for ketamine; for example, the UK has set a limit of 20 ng/mL [1]. Norway introduced a graduated system with concentrations ranging from 55 to 300 ng/mL [2].

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood ketamine limit of 50 ng/mL and a threshold limit of 10 ng/mL.

### 9.5 Sedatives

The drugs under consideration in this section are prescribed medicines used as relaxants, sleep aids, anti-anxiety agents and anti-spasmodics. These include benzodiazepine-type drugs and zopiclone. Benzodiazepines are also frequently used recreationally particularly in combination with stimulants and opioids.

Reviews of pharmacodynamic studies with healthy volunteers have generally shown that sedatives can cause severe impairment in tests designed to measure psychomotor and driving performance. Sedatives cause impairment following administration of normal therapeutic doses. In addition, performance may be adversely affected the morning after drug ingestion: this is known as the ‘hangover’ or ‘residual’ effects of benzodiazepines [5].

The magnitude of impairment is dependent on various factors, including dose, pattern of use and time of administration/intake. However, overall, the significant issues for drivers relate to the sedative effects of these drugs.

The drugs under consideration are those commonly prescribed in NZ and/or those detected in the NZ driving population: namely, alprazolam, clonazepam, diazepam, lorazepam, midazolam, nitrazepam, oxazepam, temazepam, triazolam and zopiclone.
A combination of analytical results for blood samples taken from deceased, impaired and hospitalised drivers give an indication of the prevalence of use of these drugs by NZ drivers (Table 1). It should be noted that when sedatives are detected in drivers’ blood, they are rarely the only drug detected.

Table 1  Sedative drugs detected in blood of deceased NZ drivers (n = 966) in descending order of occurrence.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Number of Detections in NZ Drivers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diazepam</td>
<td>298</td>
</tr>
<tr>
<td>Zopiclone</td>
<td>210</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>198</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>102</td>
</tr>
<tr>
<td>Midazolam</td>
<td>62</td>
</tr>
<tr>
<td>Triazolam</td>
<td>49</td>
</tr>
<tr>
<td>Temazepam</td>
<td>24</td>
</tr>
<tr>
<td>Alprazolam</td>
<td>13</td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>8</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>2</td>
</tr>
</tbody>
</table>

The intended use (indication) of a particular sedative determines when, why and how often it is dosed. Those used to induce sleep (hypnotics), such as nitrazepam, temazepam, triazolam and zopiclone, are generally only prescribed to be taken at night before sleep. This means that drivers’ blood concentrations should be lower than the maximum concentration achieved following a recent dose, because clearance from the blood would occur overnight. Those generally prescribed to reduce anxiety (anxiolytics) will be taken during the daytime. The anxiolytics comprise alprazolam, clonazepam, diazepam, lorazepam and oxazepam. Clonazepam is also prescribed to treat epilepsy. Midazolam is most commonly administered prior to surgery as a sedative.

These drugs all have different dosage strengths, the frequency of their administration varies, they are eliminated from the body at different rates and will therefore be present at different concentrations in blood after their use.

There is not a great deal of detailed information about the oral fluid:blood ratios for these drugs other than that the ratio is variable and can range from about 1:2 to over 1:10 [18]. In general, oral fluid concentrations of benzodiazepines are predicted to be lower than the blood concentrations at any given time.

It is important to note that a positive response to the oral fluid screen benzodiazepine channel does not determine which specific benzodiazepine(s) is(are) present.

Information provided about the Dräger oral fluid device lists benzodiazepine oral fluid screen cut-off concentrations as follows: alprazolam (10 ng/mL), clonazepam (15 ng/mL), diazepam (15 ng/mL), midazolam (40 ng/mL), nitrazepam (30 ng/mL), oxazepam (40 ng/mL), temazepam (20 ng/mL) and triazolam (40 ng/mL). This means that based on an oral fluid:blood ratio of 1:10, the oral fluid device will detect recent use of diazepam, oxazepam and temazepam, but not the other benzodiazepines.
Even with a lower oral fluid:blood ratio the device is unlikely to detect triazolam. Furthermore, the screening panel does not detect lorazepam or zopiclone. The latter is not a benzodiazepine, but is used for similar indications and so is included in this section.

All of these drugs have the potential to significantly impair the ability to drive safely when taken at normal therapeutic doses. Patients should be advised not to drive after taking these drugs and not to combine their use with alcohol, but we have no data to indicate whether doctors and/or pharmacists routinely give such advice.

The recommended criminal limit and threshold limit for each of these drugs is based on consideration of the blood concentrations expected following recommended doses, knowledge of the pharmacodynamic properties of the drug, doses that have been shown to cause impairment, the concentrations detected in NZ impaired drivers, and limits set by overseas jurisdictions. For most sedatives, the half-life of the drug is also an important consideration. Drugs that are prescribed to be taken before sleep should not be detected at peak concentrations in the blood of drivers because the drug will be cleared from the blood overnight.

It is advised that a criminal penalty should be considered for these drugs when detected at the threshold concentration in the presence of alcohol or in combination with other sedative/impairing drugs.

Norway, Denmark and the UK have set per se limits for a number of sedative drugs [1-3] (Table 20). The difference in per se limits for these drugs across these three jurisdictions is significant. Norway has a three-tier system with increasing blood concentrations resulting in increased penalty. The concentrations given in Table 10 are those for the maximum penalty, with the exception of midazolam which has a single statutory concentration.

**Table 20 Statutory limits for sedatives in the UK, Norway and Denmark compared with limits proposed in this report.**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Limits Proposed in this Report</th>
<th>Limits in Other Jurisdictions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ng/mL</td>
<td>ng/mL</td>
</tr>
<tr>
<td></td>
<td>Criminal</td>
<td>Threshold</td>
</tr>
<tr>
<td>Alprazolam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Diazepam</td>
<td>200</td>
<td>100</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Midazolam</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>Temazepam</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>Triazolam</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Zopiclone</td>
<td>50</td>
<td>20</td>
</tr>
</tbody>
</table>

x No limit set

*Single statutory concentration
9.5.1 Alprazolam

Alprazolam is prescribed for the treatment of anxiety; it is approximately 10 times more potent than diazepam [101,102]. Alprazolam is available in doses of 0.25 to 0.5 mg dosed 3 times daily with a maximum daily dose of 4.5 mg/d [101,102].

Studies have shown that a single 0.5 mg dose of alprazolam is sufficient to cause impairment [84].

Following a single 1 mg oral dose to six male patients, peak plasma concentrations averaged 19 ng/mL at 1.3 h after dosing [89]. With regular repeat dosing of a drug, a steady state blood drug concentration will develop, although individuals will develop different steady state concentrations with the same dose. For alprazolam, serum (blood minus blood cells) concentrations of 25 to 55 ng/mL have been reported in six patients taking daily oral doses of 1.5 to 6 mg [89].

Alprazolam has a half-life of between 6 to 27 h [89]. Such a wide range indicates that in some people the drug is eliminated much more rapidly than others.

Alprazolam is not commonly found in NZ driver samples. The blood concentrations found in four impaired drivers were 70, 100, 200 and 200 ng/mL.

Norway has three per se limits representing increasing penalties with increasing blood concentrations - 3, 6 and 15 ng/mL [2]. Denmark has a per se limit of 5.3 ng/mL [3].

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood alprazolam limit of 50 ng/mL and a threshold limit of 20 ng/mL.

9.5.2 Clonazepam

Clonazepam may be prescribed as an anxiolytic or anticonvulsant drug. It is approximately 20 times more potent than diazepam [101,102]. In NZ, clonazepam is available as tablets containing 0.5 or 2 mg, oral liquid at 2.5 mg/mL and an injectable formulation at 1 mg/mL.

The standard dose of clonazepam is 0.5 to 2 mg as a single dose up to a maximum of 8 mg/d depending on the indication. Clonazepam has multiple uses, including epilepsy treatment, anxiety, panic and in combination with opiate substitution treatments. Doses above 2 mg require split dosing with dose intervals of at least 4 h [101,102].

Studies have determined that a single dose of 1 to 2 mg clonazepam is sufficient to cause impairment [84].

Therapeutic plasma concentrations of clonazepam observed in 25 patients, undergoing continuous treatment with 6 mg of clonazepam daily for 15 to 26 days, were found to range between 29 and 75 ng/mL [103]. On average, 55 ng/mL is considered necessary to achieve an optimum therapeutic effect [102].
Clonazepam has a half-life of between 19 to 60 h [89]. Such a wide range indicates that in some people the drug is eliminated much more slowly than in others.

Clonazepam is one of the more common sedatives found in NZ driver samples. The blood concentrations found in 47 impaired drivers ranged from 10 to 140 ng/mL (mean 30 ng/mL, median 20 ng/mL).

Norway has three per se limits representing increasing penalties with increasing blood concentrations of 1.3, 3 and 8 ng/mL [2]. Denmark has a per se limit of 5.3 ng/mL [3].

**Proposed statutory limit**

Based on the concentrations detected in NZ impaired drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood clonazepam limit of 50 ng/mL and a threshold limit of 20 ng/mL.

9.5.3  Diazepam

Diazepam is prescribed for the short-term relief of anxiety, acute alcohol withdrawal, epilepsy, convulsions and for the control of muscle spasms. In NZ, diazepam is available in tablets containing 2 or 5 mg, an oral liquid at 10 mg/mL an injection at 10 mg/2 mL and a pessary containing 5 or 10 mg.

The standard dose of diazepam is 2 to 10 mg three times daily, with a maximum recommended dose of 30 mg daily (although some severe conditions require 60 mg) [101,102].

Studies have determined that doses greater than 5 mg diazepam are sufficient to cause impairment [84].

For 48 healthy male volunteers receiving a single 10 mg diazepam dose, the peak plasma concentrations ranged from 250 to 590 ng/mL (mean 400 ng/mL) [104].

Diazepam has a half-life of between 12 to 24 h [89]. Such a wide range indicates that for some people the drug is eliminated more slowly than others.

Diazepam is the most common sedative found in NZ driver samples. The blood concentrations found in 93 impaired drivers ranged from 10 to 1,430 ng/mL (mean 140 ng/mL, median 60 ng/mL).

Norway has three per se limits representing increasing penalties with increasing blood concentrations of 57, 143 and 342 ng/mL [2]. Denmark has a per se limit of 110 ng/mL [3]. The UK per se limit is 550 ng/mL [1].

**Proposed statutory limit**

Based on the concentrations detected in NZ impaired drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood diazepam limit of 200 ng/mL and a threshold limit of 100 ng/mL.
9.5.4 Lorazepam

Lorazepam is prescribed for the treatment of moderate-to-severe anxiety, insomnia and peri-operative use. It is approximately five times more potent than diazepam [101,102]. In NZ, lorazepam is available in tablets containing 1 or 2.5 mg, and injections of 2 or 4 mg.

The standard dose is 0.5 to 2.5 mg as a single dose, with a maximum dose of 4 mg daily, although in some cases a daily dose up to 8 mg (as divided doses for perioperative use) is prescribed [101,102].

Studies have determined that doses of 1 to 2 mg lorazepam are sufficient to cause impairment [84].

The peak plasma concentration of lorazepam was in the range 22 to 36 ng/mL (mean 28 ng/mL) for six healthy male volunteers given a single oral 2 mg dose [105].

Lorazepam has a half-life of between 9 to 16 h [89]. Such a wide range indicates that in some people the drug is eliminated more slowly than in others.

Lorazepam is one of the more common sedatives found in NZ driver samples. The blood concentrations found in 34 impaired drivers ranged from 10 to 230 ng/mL (mean 50 ng/mL, median 30 ng/mL).

Denmark has a per se limit of 21 ng/mL [3]. The UK per se limit is 100 ng/mL [1].

Proposed statutory limit

Based on the concentrations detected in NZ impaired drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood lorazepam limit of 30 ng/mL and a threshold limit of 10 ng/mL.

9.5.5 Midazolam

Midazolam is a sedative, often used in preoperative medication for anaesthetic induction. In NZ, midazolam is available as tablets containing 7.5 mg, as injections containing 5, 15 or 50 mg, and a nasal spray containing 5 mg/mL [101,102].

The standard dose of midazolam is 7.5 mg (oral) 1 h prior to a procedure, with a maximum daily oral dose of 15 mg [101,102].

Patients should not drive for at least 12 h following a 7.5 mg dose [106].

Following an oral dose of 10 mg given to 20 healthy young adults, peak plasma concentrations averaged 69 ng/mL for males and 53 ng/mL for females [89]. Peak plasma concentrations averaged 90 ng/mL when 20 healthy elderly patients were administered an oral dose of 10 mg [89].

Midazolam has a half-life of between 1 to 4 h [89]. It is eliminated quickly compared with other benzodiazepines.

Midazolam is commonly detected in NZ driver samples when the driver has received emergency treatment following a car crash.
Blood concentrations of midazolam found in two impaired drivers were 30 and 150 ng/mL.

Norway has a *per se* limit for midazolam of 33 ng/mL [2].

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood midazolam limit of 30 ng/mL and a threshold limit of 10 ng/mL.

### 9.5.6 Nitrazepam

Nitrazepam is usually prescribed as a hypnotic for the treatment of insomnia. It is approximately two times more potent than diazepam. In NZ, nitrazepam is available as tablets containing 5 mg [101,102].

The standard dose of nitrazepam is 2.5 to 10 mg, with a maximum daily dose of 10 mg [101,102].

Studies have determined that doses of 5 mg nitrazepam are sufficient to cause impairment [84].

Following administration of a single 5 mg dose to 15 healthy adults, peak serum concentrations ranged from 25 to 50 ng/mL (mean 35 ng/mL) [89].

Nitrazepam has a half-life of between 17 to 48 h [89]. Such a wide range indicates that in some people the drug is eliminated much more slowly than in others.

Nitrazepam is not often found in NZ driver samples. The blood concentration found in one impaired driver was 40 ng/mL.

Norway has three *per se* limits representing increasing penalties with increasing blood concentrations of 17, 42 and 98 ng/mL [2]. Denmark has a *per se* limit of 21 ng/mL [3].

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood nitrazepam limit of 50 ng/mL and a threshold limit of 20 ng/mL.

### 9.5.7 Oxazepam

Oxazepam is prescribed for the treatment of anxiety (especially anxiety associated with depression) and for the relief of acute alcohol withdrawal symptoms [101,102]. It is approximately 3 times less potent than diazepam. In NZ, oxazepam is available in tablets containing 10 or 15 mg [101,102].

The standard dose of oxazepam is 10 to 30 mg three or four times daily, with no maximum dose specified [101,102].

Studies have determined that a dose of 15 mg oxazepam is sufficient to cause impairment [84].
The peak serum concentration found in eight volunteers given a single 45 mg oral dose ranged from 880 to 1,440 ng/mL (mean 1,090 ng/mL) [107]. The trough serum concentration found in six volunteers given four doses of 15 mg oxazepam at 4 h intervals, ranged from 80 to 780 ng/mL (mean 300 ng/mL). The samples tested were obtained just before the start of daily dosing on day 5 [107].

Oxazepam has a half-life between 4 to 16 h [107]. Such a wide range indicates that in some people the drug is eliminated more rapidly than in others.

Oxazepam is frequently detected in NZ drivers, but this is because it is present as a metabolite of diazepam, rather than it being from dosing oxazepam itself. The presence of oxazepam due to the use of diazepam is easily determined by the presence of diazepam and its other (i.e., non-oxazepam) metabolites.

Norway has three per se limits representing increasing penalties with increasing blood concentrations of 172, 430, 860 ng/mL [2]. Denmark has a per se limit of 110 ng/mL [3] and in the UK it is 300 ng/mL [1].

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood oxazepam limit of 800 ng/mL and a threshold limit of 200 ng/mL.

9.5.8  **Temazepam**

Temazepam is prescribed for the short-term management of insomnia and as a preoperative medication. It has approximately half the potency of diazepam. In NZ, temazepam is available in tablets containing 10 mg [101,102].

The standard dose is 10 to 20 mg daily, with a maximum dose of 30 mg (as either a single or daily dose) [101,102].

Studies have shown that doses of 10 to 20 mg temazepam are sufficient to cause impairment [84].

A single 30 mg oral dose of temazepam given to 24 subjects produced a mean peak plasma concentration of 870 ng/mL [108]. Ten elderly adults given a single 10 mg dose of temazepam developed peak plasma concentrations of temazepam ranging from 205 to 430 ng/mL (average 305 ng/mL) [89].

Temazepam has a half-life of between 3 to 13 h [89]. Such a wide range indicates that in some people the drug is eliminated more rapidly than in others.

Temazepam is frequently detected in NZ drivers but this is because it is present as a metabolite of diazepam, rather than it being from a dose of temazepam itself. The presence of temazepam due to the use of diazepam is easily determined by the presence of diazepam and its other (i.e., non-temazepam) metabolites.
The blood concentrations found in three impaired drivers were 80, 90 and 350 ng/mL.

The UK has a *per se* limit of 1,000 ng/mL [1].

**Proposed statutory limit**

Based on our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood temazepam limit of 800 ng/mL and a threshold limit of 200 ng/mL.

### 9.5.9 Triazolam

Triazolam is used for the treatment of severe or disabling insomnia. It is approximately 20 times more potent than diazepam. In NZ, triazolam is available in tablets containing 0.125 or 0.25 mg [101,102].

The standard dose is 0.125 or 0.25 mg at bedtime, with a maximum of 0.25 mg as a single or daily dose [101,102].

Studies have determined that doses less than 0.5 mg triazolam are sufficient to cause impairment [84].

Oral administration of a single 0.25 mg dose of triazolam to six healthy adults resulted in peak plasma concentrations ranging from 2.3 to 3.7 ng/mL (mean 3.0 ng/mL) [109]. In a study of eight geriatric patients administered 0.25 mg triazolam as a daytime sedative, the mean peak serum triazolam concentration was 2.0 ng/mL [89].

Triazolam has a half-life of between 2 to 4 h [109], indicating the drug is eliminated rapidly.

The blood concentrations found in nine impaired drivers ranged from 4 to 14 ng/mL (mean 7 ng/mL, median 4 ng/mL).

Other countries have not set *per se* limits for triazolam.

**Proposed statutory limit**

Based on the concentrations detected in NZ impaired drivers and our interpretation of the scientific literature, the Panel recommends a statutory blood triazolam limit and a threshold limit of 4 ng/mL.

### 9.5.10 Zopiclone

Zopiclone is a hypnotic and sedative used to treat transient, short-term and chronic insomnia. In NZ, zopiclone is available in tablets containing 3.75 or 7.5 mg [101].

The standard dose is 7.5 mg at bedtime [101]. Although this is not recommended for long-term use, this is NZ’s most continuously prescribed sleeping tablet. While the recommended maximum dose is 7.5 mg [101,102], anecdotally some patients are prescribed 15 mg per night long-term.

Studies have shown that a dose of 7.5 mg zopiclone is sufficient to cause impairment [84].
In a study of 12 subjects who received 15 mg of zopiclone (i.e., twice the recommended therapeutic dose), a mean peak plasma concentration of 130 ng/mL was found [110]. Five subjects receiving a single 7.5 mg dose of zopiclone had plasma levels ranging from 13 to 35 ng/mL at 9 h post dosing [111].

Zopiclone has a half-life of between 3.5 to 6.5 h [89], indicating the drug is eliminated quickly.

A study investigating concentrations of zopiclone detected in impaired drivers reported a mean blood concentration of 100 ng/mL (maximum 410 ng/mL) [112].

Zopiclone is one of the more common sedatives found in NZ driver samples (see Table 9). The blood concentrations found in 52 impaired drivers ranged from 10 to 600 ng/mL (mean 100 ng/mL, median 54 ng/mL).

The use of zopiclone will not be detected by current roadside oral fluid tests. Norway has three per se limits representing increasing penalties with increasing blood concentrations of 12, 23 and 58 ng/mL [2]. Denmark has a per se limit of 11 ng/mL [3].

**Proposed statutory limit**

Based on the concentrations detected in NZ impaired drivers, our interpretation of the scientific literature and consideration of statutory limits in overseas jurisdictions, the Panel recommends a statutory blood zopiclone limit of 50 ng/mL and a threshold limit of 20 ng/mL.

### 9.6 Opioids

Opioid drugs can be broadly classified as natural opiates (morphine and codeine), semi-synthetic opioids (heroin, oxycodone and dihydrocodeine) or synthetic opioids (methadone, buprenorphine, tramadol and fentanyl). Although opioid drugs are used globally for analgesia, many have a significant potential for misuse. Methadone is commonly used to treat opioid addiction.

All opioids are CNS depressants and universally cause drowsiness and lethargy.

All opioids are characterised by the onset of tolerance with regular dosing and a well-defined withdrawal syndrome upon cessation of dosing. The development of tolerance results in the need for administration of larger doses to achieve the required result. The degree of tolerance that an individual achieves following daily dosing with opioid drugs is quickly lost if dosing is interrupted.

There is mounting epidemiological evidence linking the therapeutic use of opioids to increased crash risk, but there is inconsistency in the literature [5, 113-117].

Most available oral fluid testing devices have an opiate channel. This channel detects 6MAM (Figure 1) a major metabolite of heroin. It also detects codeine, morphine and dihydrocodeine, but cannot distinguish between these drugs. None of the other opioids (buprenorphine, fentanyl, oxycodone or tramadol) are detected by current oral fluid testing devices. Some oral fluid testing device manufacturers include a separate methadone testing channel.
Figure 1 Simplified metabolic pathways of the natural opiates showing their interrelationships and major route to excretion.

9.6.1 Opioid use by NZ drivers

Detection of opioids in the blood of deceased drivers in NZ has increased in recent years. For the period 2004 to 2009, opioids were detected in 4% of deceased drivers: this increased to 9% for the period 2013 to 2018.

Opioid use in impaired and hospitalised drivers for the years 2017 to mid-2020, as determined by analysis of blood samples submitted to ESR, is shown in Table 11. Opioids detected in blood of impaired and hospitalised drivers in NZ. The prevalence of morphine and fentanyl in hospitalised drivers is not given because these drugs are frequently administered for pain relief by medical personnel either en route to the hospital or during admission.

Table 11 Opioids detected in blood of impaired and hospitalised drivers in NZ.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Number of impaired drivers</th>
<th>Number of hospitalised drivers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methadone</td>
<td>69</td>
<td>50</td>
</tr>
<tr>
<td>Tramadol</td>
<td>40</td>
<td>164</td>
</tr>
<tr>
<td>Morphine</td>
<td>23</td>
<td>x</td>
</tr>
<tr>
<td>Codeine</td>
<td>19</td>
<td>66</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Dihydrocodeine</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>2</td>
<td>x</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

x Data not included because these drugs are used for pain relief following traffic crash injury.
It should be noted that when opioids are detected in impaired drivers, it is common to find evidence of other drug use as well. Combinations including cannabis, methamphetamine and/or sedatives were detected in the blood of most of the drivers using opioids.

The recommended criminal limit and threshold limit for each of the opioids is based on the concentrations expected in the blood following ingestion of the recommended doses, knowledge of the pharmacodynamic properties of the drug, doses that are known to cause driving impairment, the concentrations detected in NZ impaired drivers and limits set in overseas jurisdictions. Recommending limits for these drugs is complex due to the wide range of doses that may be prescribed and the variety of methods of administration.

It is advised that the criminal blood limit should only apply if the drug is the only drug detected in the blood. A criminal penalty should be considered for these drugs when detected at the threshold concentration in the presence of alcohol or in combination with other impairing drugs.

Norway, Denmark and the UK have specified per se limits for some of the opioid drugs [1-3]. The difference in per se limits for these drugs across these three countries is significant. Norway has a three-tier system with increasing blood concentrations resulting in increased penalty, but this has only been applied to morphine.

The criminal and threshold limits proposed in this report together with the per se limits used by Norway, Denmark and the UK are shown in Table 32.

Table 32  Statutory limits for opioids in the UK, Norway and Denmark compared with limits proposed in this report.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Limits proposed in this report ng/mL</th>
<th>Limits in other jurisdictions ng/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Criminal limit</td>
<td>Threshold limit</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Codeine</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Dihydrocodeine</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Methadone</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>Morphine</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>Tramadol</td>
<td>250</td>
<td>100</td>
</tr>
</tbody>
</table>

x No limit set

9.6.2  Heroin

Heroin is a powerful euphoriant. It decreases alertness and motor activity regardless of the route of administration (intravenous or snorting/intranasal) [5]. It is approximately five times more potent than morphine.
Heroin itself is not detected in blood samples due to its extremely rapid metabolism to 6MAM (Fig. 3), which is then further metabolised to morphine. 6MAM can be used to provide evidence that heroin has been used but due to its short half-life (6 to 25 min), it may not be detected in blood even as little as 2 h after heroin use.

In NZ, 6MAM has been detected in one impaired and one hospitalised driver since 2017.

The Panel does not recommend a per se limit for 6MAM as the use of heroin will be covered by the per se limit for morphine.

9.6.3 Buprenorphine

In recent years, buprenorphine has become an increasingly popular choice in clinical practice as an alternative to methadone for the treatment of opioid dependence. It is less commonly used in the treatment of moderate to severe pain.

In NZ, buprenorphine is dispensed in combination with naloxone as sublingual tablets that contain 2 mg buprenorphine and 0.5 mg naloxone, or 8 mg buprenorphine and 2 mg naloxone for opioid dependence. For the treatment of pain, it is available as transdermal patches, delivering 5, 10, or 20 μg/h, or as a 300 μg/mL injection [101,102].

The standard opioid substitution maintenance dose is 4 to 24 mg buprenorphine daily as a single sublingual dose, with a maximum dose of 32 mg daily. For treatment of moderate to severe pain, doses of 0.3 to 0.6 mg may be administered by intramuscular injection or slow intravenous injection every 6 to 8 h. Alternatively, transdermal patches delivering 5 μg/h increasing up to a maximum of 40 μg/h may be used [101,102].

Sublingual or intravenous administration of 0.4 mg or less of buprenorphine has been found to impair driving skills [84].

A single 4 mg sublingual dose of buprenorphine resulted in a mean peak plasma concentration of 3.3 ng/mL [89]. Eight milligram maintenance doses resulted in levels ranging from 1 to 8 ng/mL [89].

The half-life of buprenorphine is short, 2 to 4 h [89]. It is eliminated quickly compared with some other opioids.

Buprenorphine is not detected by currently available roadside oral fluid testing devices.

Buprenorphine is not often found in NZ driver samples, possibly due to the low concentrations of the drug in blood and its rapid half-life. The blood concentration found in one impaired driver was 3 ng/mL.

Norway has a per se limit of 0.9 ng/mL [2] and Denmark has a per se limit of 0.53 ng/mL [3]. These concentrations are below the detection limit of the current ESR technology.
Proposed statutory limit

Based on our interpretation of the scientific literature, with consideration of statutory limits in overseas jurisdictions, and acknowledging the current technological limitations, the Panel recommends a statutory blood buprenorphine limit and a threshold limit of 1 ng/mL.

9.6.4 Codeine

Codeine is indicated for treatment of mild to moderate pain (and occasionally for diarrhoea or suppression of non-productive cough). It can cause sedation and drowsiness, and can depress breathing. It has approximately one-tenth the potency of morphine. In NZ codeine is available in tablets containing 15, 30 or 60 mg (it is also available as a paracetamol 500 mg + codeine 8 mg combination).

The standard dose of codeine is 15 to 60 mg up to four times a day, with a maximum daily dose of 240 mg [101,102].

Oral administration of 60 mg of codeine per 70 kg body weight (i.e., ‘average’ human body weight) resulted in peak plasma concentrations ranging from 66 to 413 ng/mL (mean 214 ng/mL) [118]. Similarly, administration of a 120 mg dose resulted in blood concentrations ranging from 184 to 1,158 ng/mL (mean 474 ng/mL) [118].

The half-life of codeine ranges from 1.9 to 3.9 h (mean 2.2 h) [89]. It is eliminated quickly compared with some other opioids.

Oral fluid concentrations of codeine are generally higher than blood concentrations at the same time. The cut-offs for the currently available oral fluid devices are between 20 to 40 ng/mL for the opiate channel. With blood concentrations generally being greater than 50 ng/mL following a normal dose (and a recommended criminal limit of 200 ng/mL), and oral fluid codeine concentrations expected to be higher than blood concentrations, these device cut-offs appear too low.

Since 2017, codeine has been detected in the blood of 19 impaired drivers at concentrations ranging from 10 to 330 ng/mL (mean 98 ng/mL, median 80 ng/mL). Very few of these drivers had used codeine alone. Sedatives, cannabis and/or methamphetamine were commonly detected in the blood of drivers using codeine. The blood codeine concentrations found in 66 hospitalised drivers ranged from 10 to 1,200 ng/mL (mean 100 ng/mL, median 50 ng/mL).

Norway has a per se limit of 9 ng/mL [2]. Denmark and the UK have not set per se limits for codeine.

Proposed statutory limit

Based on our interpretation of the scientific literature and the concentrations found in NZ impaired drivers, the Panel recommends a statutory blood codeine limit of 200 ng/mL and a threshold limit of 50 ng/mL.
9.6.5 Dihydrocodeine

Dihydrocodeine is chemically similar to codeine and has approximately one-tenth the potency of morphine. In NZ, dihydrocodeine is prescribed for the treatment of mild-moderate pain. It is available as modified release tablets containing 60 mg dihydrocodeine [101,102].

The standard dose is 60 to 120 mg every 12 h, with a maximum daily dose of 240 mg [101,102].

Following a single oral dose of 60 mg in 12 adult volunteers, peak plasma dihydrocodeine concentrations ranged from 90 to 110 ng/mL (mean 100 ng/mL). Twice daily oral doses of 60, 90 and 120 mg given to the same volunteers resulted in mean peak plasma concentrations of 150, 220 and 280 ng/mL respectively [119].

The half-life of dihydrocodeine ranges from 3.5 to 4.5 h [89]. It is eliminated quickly compared with some other opioids.

Oral fluid concentrations of dihydrocodeine are generally higher than blood concentrations at the same time. The cut-offs for the currently available oral fluid devices are between 20 to 40 ng/mL for the opiate channel. With blood concentrations generally being greater than 100 ng/mL following a normal dose (and a recommended criminal limit of 200 ng/mL), and oral fluid dihydrocodeine concentrations expected to be higher than blood concentrations, these device cut-offs appear too low.

Since 2017, dihydrocodeine has been detected in the blood of 7 impaired drivers at concentrations ranging from 10 to 420 mg/mL (mean 160 ng/mL, median 130 ng/mL). Very few of these drivers had used dihydrocodeine alone. Sedatives, cannabis and/or methamphetamine were commonly detected in the blood of drivers using dihydrocodeine. The blood dihydrocodeine concentrations found in 13 hospitalised drivers ranged from 10 to 2,000 ng/mL (mean 330 ng/mL, median 120 ng/mL).

Other countries have not set per se limits for dihydrocodeine. This could relate to low prescribing frequency for the drug in those countries.

Proposed statutory limit
Based on our interpretation of the scientific literature and the concentrations found in NZ impaired drivers, the Panel recommends a statutory blood dihydrocodeine limit of 200 ng/mL and a threshold limit of 50 ng/mL.

9.6.6 Fentanyl

Fentanyl is a powerful opioid estimated to be 80 times more potent than morphine. Fentanyl is generally used in medical settings as an anaesthetic agent or for postoperative pain [101,102]. In NZ, fentanyl is available as transdermal patches containing 0.0125, 0.025, 0.05 or 0.1 mg and as injections containing 0.01, 0.02, 0.1, 0.5 or 1 mg.

The standard dose range of fentanyl is 0.0125 to 0.025 mg per hour released transdermally or by injection, with a maximum dose of 0.3 mg/h. Fentanyl is also used off-label as a nasal spray for palliative care [101,102].
Studies have determined that intravenous doses of 0.1 mg fentanyl are sufficient to cause impairment [84].

For a fentanyl transdermal patch (delivery 0.025 mg/h), serum concentrations ranged from 0.3 to 1.2 ng/mL within 24 h of patch application [89]. Buccal fentanyl tablets containing 0.4 mg fentanyl taken by healthy volunteers for six days produced a mean serum concentration of 1.8 ng/mL following the final dose [89].

The half-life of fentanyl in the blood ranges from 3 to 12 h [89]. Such a wide range indicates that in some people the drug is eliminated more rapidly than in others.

Fentanyl is not able to be detected by current roadside oral fluid testing devices.

At this time there is no evidence to suggest that fentanyl is commonly used recreationally in NZ. While it is frequently detected in blood taken under the LTA, these samples have all come from drivers who have been hospitalised following a crash and it is most likely that the drug has been administered by medical personnel as part of treatment.

Fentanyl is not often found in NZ impaired driver samples. The blood concentrations found in two impaired drivers were 3 and 8 ng/mL.

Norway has a per se limit of 0.34 ng/mL [2], which is below the detection limit of the current ESR technology.

**Proposed statutory limit**

Based on our interpretation of the scientific literature, with consideration of statutory limits in overseas jurisdictions, and acknowledging the current technological limitations, the Panel recommends a statutory blood fentanyl limit and a threshold limit of 0.5 ng/mL.

9.6.7 Methadone

Methadone is a synthetic opioid used in the treatment of opioid dependence, but is also used as an analgesic and antitussive. Adverse effects include sedation, cognitive impairment and respiratory depression. Some tolerance to sedation and respiratory depression develops following chronic use [89]. In NZ, methadone is available as tablets containing 5 mg, injection solutions at 10 mg/mL and oral liquids at 2, 5 or 10 mg/mL.

The normal oral dose of methadone is up to 20 mg daily for naïve patients, with a maintenance dose of 60 to 120 mg daily [101]. Higher doses are used by methadone tolerant patients.

Studies have determined that 10 mg doses of methadone are sufficient to cause impairment in naïve users of the drug, but tolerance (and thus reduced impairment at a particular dose) develops with long-term use [84].
A single 10 mg dose to eight healthy adults resulted in a mean peak blood concentration of 43 ng/mL [89]. Daily administration of 100 to 120 mg of methadone to tolerant subjects resulted in peak blood concentrations ranging from 440 to 820 ng/mL [120].

The half-life of methadone ranges from 15 to 55 h [89]. Such a wide range indicates that in some people the drug is eliminated more slowly than in others.

Methadone is not detected by the oral fluid opiate channel. The cut-offs for the methadone specific channels are between of 15 to 20 ng/mL. With blood concentrations potentially being greater than 100 ng/mL following a normal dose (and a recommended criminal limit of 200 ng/mL), and oral fluid methadone concentrations expected to be higher than blood concentrations, these device cut-offs appear too low.

Since 2017, methadone has been detected in the blood of 69 impaired drivers at concentrations ranging from 10 to 1,200 ng/mL (mean 260 ng/mL, median 200 ng/mL). Very few of these drivers had used methadone alone. Sedatives, cannabis and/or methamphetamine were commonly detected in the blood of drivers using methadone. The blood methadone concentrations found in 50 hospitalised drivers ranged from 10 to 800 ng/mL (mean 280 ng/mL, median 280 ng/mL).

Norway has a per se limit of 25 ng/mL [2] and Denmark has a per se limit of 53 ng/mL [3]. The UK’s per se limit for methadone is 500 ng/mL [1].

**Proposed statutory limit**

Based on our interpretation of the scientific literature, with consideration of statutory limits in overseas jurisdictions, and the concentrations found in NZ impaired drivers, the Panel recommends a statutory blood methadone limit of 200 ng/mL and a threshold limit of 50 ng/mL.

### 9.6.8 Morphine

Morphine is used for the relief of severe and chronic pain. Chronic intake of morphine may lead to physical and psychological dependence.

In NZ, morphine is available as immediate release tablets containing 10 or 20 mg, as modified release tablets and capsules containing 10, 30, 60 or 100 mg, as oral liquid at 1, 2, 5 or 10 mg/mL and as injections at a broad range of concentrations [101,102].

The standard doses of morphine are:
- 5 to 20 mg every 4 to 6 h (immediate release),
- 10 to 20 mg twice daily (modified release),
- 1 to 5 mg every 4 h (oral liquid), and
- 5 to 10 mg injected at 1 to 2 mg/min (intravenous) [101].

Maximum doses are difficult to define due to the development of tolerance and extenuating circumstances [101].

Studies have determined that 10 mg doses of morphine are sufficient to cause impairment in naïve users of the drug, but tolerance develops with long-term use [84].
Blood morphine concentrations are difficult to interpret with respect to dose because concentrations achieved after morphine administration depend on the route of administration (i.e., intravenous or oral) and the formulation of the medication (i.e., immediate or controlled release) [89].

A single oral dose of 30 mg of morphine in an immediate release tablet gave rise to an average peak plasma concentration of 24 ng/mL after 0.8 h [121]. A single oral dose of 60 mg morphine in a controlled release capsule gave an average peak plasma concentration of approximately 10 ng/mL at 7.9 h [121].

The half-life of morphine is generally short, ranging from 1 to 8 h (mean 2 h) [122], however this half-life can be significantly longer (up to 16 h) with some controlled release formulations [123].

Oral fluid concentrations of morphine are generally higher than blood concentrations at the same time. The cut-offs for the currently available oral fluid devices are between of 20 to 40 ng/mL for the opiate channel. With blood concentrations expected to be about 10 to 20 ng/mL following a normal dose (and a recommended criminal limit of 20 ng/mL), and oral fluid morphine concentrations expected to be higher than blood concentrations, these device cut-offs appear to be appropriate.

Since 2017, morphine has been detected in the blood of 23 impaired drivers at concentrations ranging from 10 to 110 ng/mL (mean 40 ng/mL, median 30 ng/mL). Very few of these drivers had used morphine alone. Sedatives, cannabis and/or methamphetamine were commonly detected in the blood of drivers using morphine. Morphine is frequently detected in hospitalised drivers, likely due to administration by medical personnel, before or during the drivers’ hospital admission.

Norway has a three tiered per se limit range for morphine of 9, 24 and 61 ng/mL [2]. Denmark has a per se limit of 10 ng/mL [3], and the UK’s per se limit for morphine is 80 ng/mL [1].

**Proposed statutory limit**

Based on our interpretation of the scientific literature, consideration of statutory limits in overseas jurisdictions, and the concentrations found in NZ impaired drivers, the Panel recommends a statutory blood morphine limit of 20 ng/mL and a threshold limit of 10 ng/mL.

### 9.6.9 Oxycodone

Oxycodone is a narcotic analgesic approximately equipotent to morphine [89]. In NZ, oxycodone is available as immediate release capsules containing 5, 10 or 20 mg, as modified release tablets containing 5, 10, 15, 20, 30, 40, 60, or 80 mg, as oral liquid at 5 mg/5 mL and as injection formulations at a range of concentrations. [101].

The standard doses of oxycodone are:

- 5 to 20 mg every 4 to 6 h (immediate release),
- 10 to 40 mg every 12 h with a maximum of 200 mg every 12 h (modified release),
- 2.5 to 20 mg every 4 to 6 h (oral liquid) and initially 5 mg every 4 h (injection),
The maximum daily dose is 400 mg. A 2 mg oral oxycodone dose is approximately equivalent to 1 mg parenteral (administered other than by mouth) oxycodone [101].

Studies have determined that a 20 mg dose of oxycodone is sufficient to cause impairment [84].

Peak plasma concentrations in 12 adult surgery patients receiving a 10 mg immediate release oral dose ranged from 13 to 46 ng/mL (mean 30 ng/mL), whereas subjects receiving 40 or 80 mg modified release tablets attained mean peak plasma concentrations of 30 and 99 ng/mL respectively [89].

The half-life of oxycodone in the blood ranges from 3 to 6 h [89]. It is eliminated quickly compared with some other opioids.

Oxycodone is not detected by current roadside oral fluid testing devices.

Oxycodone is not often found in NZ driver samples. The blood concentrations found in seven impaired drivers ranged from 10 to 140 ng/mL (mean 80 ng/mL, median 50 ng/mL). Blood oxycodone concentrations detected in six hospitalised drivers ranged from 10 to 300 ng/mL (mean 90 ng/mL, median 50 ng/mL).

Norway has a per se limit of 16 ng/mL [2]. Neither Denmark nor the UK have per se limits for oxycodone.

**Proposed statutory limit**

Based on our interpretation of the scientific literature, consideration of statutory limits in overseas jurisdictions, and concentrations detected in NZ impaired drivers, the Panel recommends a statutory blood oxycodone limit of 50 ng/mL and a threshold limit of 20 ng/mL.

9.6.10 Tramadol

Tramadol is a narcotic analgesic that is approximately one-tenth the potency of morphine. In NZ, tramadol is available as immediate release capsules containing 50 mg, as modified release tablets containing 50, 100, 150 or 200 mg and as oral liquids and injections at a range of concentrations [101,102]

The standard doses of tramadol are:
- 50 to 100 mg every 4 to 6 h (immediate release capsules),
- 50 to 200 mg every 12 h (modified release),
- 12.5 mg equivalent to 5 drops or 1 press of a delivery pump up to 100 mg every 4 to 6 h (oral drops or pump spray),

with a maximum daily dose of 400 mg (reduced to 300 mg in patients 75 years of age and over) [101].

There is evidence that tramadol might be associated with increased risk of traffic accidents for at least seven days and up to four weeks following the initiation of treatment [5,124].
A single 50 mg normal release oral dose given to 24 healthy adults resulted in a mean peak plasma concentration of 107 ng/mL [89], and a single 100 mg normal release oral dose given to 10 healthy adults resulted in a mean peak plasma concentration of 280 ng/mL [89].

The half-life of tramadol in the blood ranges from 4 to 8 h [89].

Tramadol is not detected by currently available roadside oral fluid testing devices.

Since 2017, tramadol has been detected in the blood of 40 impaired drivers at concentrations ranging from 10 to 1,000 ng/mL (mean 280 ng/mL, median 220 ng/ml). Very few of these drivers had used tramadol alone - sedatives, cannabis and/or methamphetamine were commonly detected in the blood of drivers using tramadol. The blood tramadol concentrations found in 164 hospitalised drivers ranged from 10 to 3,000 ng/mL (mean 210 ng/mL, median 130 ng/mL).

Norway has a per se limit of 53 ng/mL for tramadol [2]. Neither Denmark nor the UK has a per se limits.

Proposed statutory limit
Based on our interpretation of the scientific literature, consideration of statutory limits in overseas jurisdictions, and the concentrations found in NZ impaired drivers, the Panel recommends a statutory blood tramadol limit of 250 ng/mL and a threshold limit of 100 ng/mL.
Qualifications and affiliations of the Panel members

Dr Helen Poulsen (Chair), BSc (Hons.), PhD, Science Leader Forensic Toxicology Group, ESR

Dr Sharon Kletchko MD, FRCPC, FRACP; Director Quality, Governance, Risk & Compliance, Lakes District Health Board

Andrew McGlashen BPharm, MPS, CMIInstD; Pharmacist and Director of McGlashen, Fry's and Wakefield Pharmacy

Professor Ian Shaw BSc (Hons.), PhD, DSc, FRSC, CChem, FRCPATH; Professor of Toxicology, University of Canterbury

Associate Professor Malcolm Tingle, BSc (Hons.), PhD; Head of the Department of Pharmacology & Clinical Pharmacology, University of Auckland
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Appendices

Appendix 1

Glossary and abbreviations

Absorption: The physical processes of diffusion and penetration across and through membranes and barriers. It is heavily influenced by the route of administration, such as oral, inhalation or sub-lingual. There is no barrier to absorption with intravenous administration.

Adsorption (adsorbed): The adhesion of molecules to the surface of a structure (e.g., skin).

Accuracy: The degree to which the result of a measurement, calculation, or specification conforms to the correct value or a standard.

Additive: the joint effect of two drugs is equal to the sum of their individual effects (cf. synergistic or antagonistic).

ADD: Attention deficit disorder.

ADHD: This refers to Attention Deficit and Hyperactivity Disorder, classified as a disruptive behaviour disorder.

Agonist: A drug capable of binding and activating a receptor, leading to a pharmacological response that may mimic that of a naturally occurring substance.

Antagonist: Chemical that does not produce a biological response on binding to a receptor but instead blocks or reduces the effect of an agonist. It may be competitive or non-competitive.

Analgesic: A drug that dulls the sense of pain. It differs from an anaesthetic agent in that it relieves pain without loss of consciousness.

Anaesthetic: Literally: an – without + aisthesis – perception by the senses (Gr.) A drug that causes loss of sensation. General anaesthetics cause not only loss of sensation, but also loss of consciousness. Local anaesthetics cause loss of sensation by blocking nerve conduction only in the particular area where they are applied.

Antagonism: The joint effect of two or more drugs such that the combined effect is less than the sum of the effects produced by each agent separately. The agonist is the agent producing the effect that is diminished by the administration of the antagonist. Cf. additivity and synergism.

AS/NZS 4760:2019: Australian/New Zealand Standard “Procedure for specimen collection and the detection and quantification of drugs in oral fluid”.

BAC: blood alcohol concentration.
Bioavailability: The fraction of dose entering the blood stream after administration of a given dosage form relative to an intravenous administration of the same dose. It is markedly influenced by the route of administration (oral, sub-lingual, inhalation etc.).

Biological fluid: Any fluid found in the body and including blood, saliva, sweat or urine, which may be used to detect the presence of drugs in the body.

BrAC: Breath alcohol concentration.

CBN: Cannabinol.

CIT: Compulsory impairment test.

Clearance: This describes the efficiency of irreversible elimination of a drug from the blood stream.

CNS: Central nervous system.

Distribution: The processes by which drugs move around the body, which is dependent on the chemical nature of the drug itself, the blood flow to the tissues, whether the drug is bound to proteins and/or dependent on transporter proteins.

Dose: The quantity of drug, or dosage form, administered to a subject at a given time. Dose may be expressed in terms appropriate to a specific dosage form, i.e., one teaspoonful of a liquid medication, rather than the weight of drug in the teaspoonful. Dose may be described as an absolute dose (the total amount administered to a subject) or as a relative dose (relative to some property of the subject as body weight or surface area, mg/kg, or mg/m²).

DRUID: This refers to the Integrated Project DRUID (Driving under the Influence of Drugs, Alcohol and Medicines) and seeks to find answers to questions concerning the use of drugs or medicines that affect people’s ability to drive safely. The European Integrated Project DRUID is a part of the 6th Framework Programme. It brings together 36 institutes from 18 European countries.

Drug: A chemical used in the diagnosis, treatment, or prevention of disease. More generally, a chemical, which, in a solution of sufficient concentration, will modify the behaviour of cells exposed to the solution. Drugs produce only quantitative changes in the behaviour of cells; i.e., drugs increase or decrease the magnitude, frequency, of duration of the normal activities of cells.

Elimination: The irreversible loss of drug from the body. The rate of elimination is dependent on two processes: metabolism and excretion.

ESR: The Institute of Environmental Science and Research Limited.

Excretion: The removal of unchanged drug or its metabolites from the body. The main routes of excretion are through the kidney (with the drug ending up in urine) and biliary system of the liver (with
the drug ending up in faeces), although volatile drugs such as inhalation anaesthetics are also excreted through the lung (with the drug ending up in the breath).

**First Pass Effect:** The metabolism and/or excretion of a drug by intestinal and hepatic, including biliary, mechanisms following absorption of the drug from the gastrointestinal tract, before drug gains access to the systemic circulation.

**Half-Life (t½):** The period of time required for the concentration or amount of drug in the body to be reduced to exactly one-half of a given concentration or amount. Half-life refers to the duration of action of a drug and depends upon how quickly the drug is eliminated from the blood/plasma. Clearance and distribution of a drug from the blood/plasma are important parameters for half-life determination.

**Hypnotic:** A drug that produces a state clinically identical to sleep by means of action in the central nervous system.

**Immunoassay:** This is a type of biochemical test which measures the presence or concentration of a substance in biological solutions such as blood or urine.

**Impaired drivers:** NZ drivers who have been stopped by Police for poor driving and have failed to satisfactorily complete the CIT.

**LOD:** This refers to a laboratory’s limit-of-detection. This is the lowest concentration of a drug that the analytical procedure can reliably differentiate from a concentration of zero and can be positively identified according to predetermined criteria and/or levels of statistical confidence.

**LOQ:** This refers to a laboratory limit-of-quantification. This is defined as the lowest measurable quantity of a drug that can be detected according to the technological limits of the equipment with an acceptable level of accuracy and precision.

**LTA:** Land Transport Act 1998.

**Metabolite:** A compound formed from the drug consumed by the process of metabolism. A metabolite often has little or no pharmacological activity, but in some circumstances it may have similar or the same activity as the parent drug itself.

**Metabolism:** The enzymatic conversion of a drug from one form to another to contribute to the elimination of the drug. The change to the chemical structure through metabolism often abolishes or reduces the pharmacological activity of a drug and changes the rate and perhaps route of excretion. Metabolism alters the water-solubility of the drug and will often influence the rate and route of excretion.

**Narcotic:** Formerly, an agent capable of producing coma or stupor (from Greek *narke*: torpor, numbness). Now, usually, any drug which produces analgesia and is capable of producing stupor: pain is relieved by a dose or narcotic before the occurrence of sleep or unconsciousness.
NZTA: New Zealand Transport Agency (Waka Kotahi).

Opiates: Any of the opioid alkaloids found as natural products in the opium poppy.

Opioids: This term refers to drugs that include natural or synthetic substances, which relieve pain by binding to opioid receptors in the brain.

Parenterally: Administered or taken not through oral consumption and the digestive tract, but for example through injection.

Pharmacodynamics: The study of how drugs produce their biological effects.

Pharmacokinetics: The science of the factors which relates the concentration of drug at their sites of biological effect over time after the dose of a drug. It includes study of drug absorption, distribution, metabolism and excretion.

Potency: An expression of the activity of a drug, in terms of the concentration or amount needed to produce a defined effect; an imprecise term that should always be further defined.

Precision: The closeness of agreement between independent test results obtained under stipulated conditions.

Reliability: The extent to which an instrument or assay consistently has the same results if it is used in the same situation on repeated occasions.

Sensitivity: The proportion of positives that are correctly identified (i.e., the proportion of those who have some condition (affected) who are correctly identified as having the condition).

Steady state: With regular repeat dosing of a drug, a steady state blood drug concentration will develop. Such a steady state means that the blood drug concentration is relatively constant with time. Blood drug concentrations will increase shortly after the drug is taken and drop until the next dose is taken, but the drug is not fully eliminated between doses.

Subcutaneous injection: This refers to injection into the lower layers of the skin.

Sublingual: Under the tongue.

Synergy: A mutually reinforcing drug interaction such that the joint effect of two drugs administered simultaneously is greater than the sum of their individual effects. Synergism is distinguished from additivity, in which the joint effect of two drugs is equal to the sum of their individual effects. If the joint effect is less than the sum of the two drugs’ independent effects, the interaction is said to be antagonistic.

THC: Tetrahydrocannabinol, the main psychoactive compound in cannabis.
**UK**: The United Kingdom of Great Britain and Northern Ireland.

**Validity**: The extent to which a concept is accurately measured in a quantitative study.
Appendix 2

Terms of Reference for the Independent Expert Panel on Drug Driving June 2020

Context

1. In December 2019, the Government agreed to introduce a compulsory random roadside oral fluid testing regime for drug driving in New Zealand. The design of this oral fluid testing regime requires the following to be developed:

   a. cut-off thresholds for oral fluid testing devices (above which a driver would show a positive result)

   b. low-level tolerances for blood-drug levels (above which a driver would show a positive result)

   c. criminal limits based on drug concentrations in blood that align with drink driving measures of impairment, being equivalent to a blood-alcohol limit of 80mg of alcohol per 100ml of blood (above which a driver would commit a criminal offence).

2. A number of jurisdictions have introduced, or are moving towards, the introduction of legal ‘blood-drug’ limits for illicit drugs and/or medicines, including the United Kingdom, Norway, and several jurisdictions in North America. Like alcohol, these limits have been established as a proxy for impairment, based on scientific research about the impairing effects of different dosages of drugs.

3. The challenge with setting legal ‘blood-drug’ limits is that, unlike alcohol in which the relationship between dose and impairment is very well understood, other drugs behave differently (in terms of effects, absorption and elimination from the system). This means there is scientific debate about the levels at which impairment occurs.

4. Cut-off thresholds for oral fluid testing devices and blood tests need to be selected at a level that accounts for the accuracy of the testing process, and the amount of a drug that would be associated with recent use rather than past or passive exposure to a drug.

5. Given the highly technical nature of determining the thresholds and limits, Cabinet agreed to commission an independent panel of medical and science professionals to provide advice to the Government on these matters. This approach is similar to that of other jurisdictions that have faced these decisions.

6. Cabinet agreed that the independent expert panel of medical and science experts would be appointed by the Associate Minister of Transport (Hon Julie Anne Genter), the Minister of Police (Hon Stuart Nash) and the Minister of Research, Science and Innovation (Hon Megan Woods) (together, the Appointing Ministers) in accordance with the Cabinet Fees Framework for advisory bodies.

Scope and approach

7. The Expert Panel will prepare objective advice for the Associate Minister of Transport (Hon Julie Anne Genter) and the Minister of Police (together Joint Ministers) and make non-binding recommendations on the following topics:

   • the ‘blood-drug’ limits to be specified in legislation based on drug concentrations in blood that align with impairment equivalent to a blood-alcohol concentration of 80mg of alcohol per 100ml of blood
• the low-level tolerance thresholds to be applied to the detection of drugs in blood by the Institute of Environmental Science and Research
• the cut-off thresholds to be included in oral fluid testing devices (noting that this will require alignment with the procurement process by Police and the technical limitations of any device procured).
• any other matters that may be referred to it by Joint Ministers.

8. Both the low-level thresholds to be applied to the detection of drugs in blood and the cut-off thresholds to be included in oral fluid testing devices are intended to be set at levels that avoid penalising drivers who have:
• accidental or passive exposure to drugs
• low residual levels of a drug in their blood due to previous use but have not recently used drugs
• consumed standard prescription doses of some medicines.

9. Other drug-related policy matters are out of scope of the Expert Panel’s mandate.

10. It is expected that the Expert Panel will consider the following, among other things, in preparing advice:
• relevant scientific literature
• international examples
• advice of other experts outside of the Panel
• advice from oral fluid device manufacturers.

11. The Expert Panel will be supported by a Secretariat based at the Ministry of Transport.

12. Advice and recommendations from the Expert Panel will be provided to Joint Ministers in a written report(s), which will cover the points raised in paragraph 10.

13. The Government will make final decisions on the matters above, taking into account the technical advice of the Expert Panel alongside advice from officials on other public policy considerations.

14. The Chair and members of the Expert Panel will report to the Associate Minister of Transport about matters outside of the recommendations, such as resignations or no surprises reporting.

**Deliverables and milestones**

15. The Expert Panel will report to Joint Ministers as follows:
• final advice on criminal limits to be provided by 15 October 2020 so that it can inform legislation at the Select Committee stage\(^1\)

\(^1\) It is anticipated that Legislation will be introduced without the criminal limits in July 2020. Final advice on the criminal limits will be need to be provided to the Government following the September 2020 General Election so that it can inform the legislation before or while it is being considered at Select Committee. The actual date that
• written and/or verbal interim advice on the Panel’s work to be provided to Joint Ministers between July and September 2020
• final advice on low-level tolerance thresholds for the detection of drugs in blood to be applied by the Institute of Environmental Science and Research to be provided by early 2021
• final advice on the cut-off thresholds for oral fluid testing devices to be provided by early 2021 (noting that this will require alignment with the procurement process by Police and the technical limitations of any device procured).

16. The dates specified for the deliverables above are approximate and subject to change by agreement between the Expert Panel, the Ministry of Transport and Joint Ministers.

17. The Expert Panel may be asked by Joint Ministers to consider further relevant issues.

18. The Expert Panel may be asked to provide follow-up advice on their recommendations, for example at the Select Committee stage.

Responsibilities and Appointment of Chairperson

19. The Chair is appointed by the Appointing Ministers.

20. The Chair is responsible for:

• setting the agenda for Expert Panel meetings, taking into account matters referred by Appointing Ministers and officials for advice
• presiding at each Expert Panel meeting
• casting a deciding vote in the event of a tied vote on the recommendation to be made on a particular issue
• signing off the final version of the Minute of each meeting
• advising Appointing Ministers and officials on appointments to the Expert Panel
• representing the views and interests of the Expert Panel to Government Ministers, Select Committee, officials and departments, and to the media, having first obtained the consent of the Ministry of Transport and, if necessary, Joint Ministers to the act and content of representation
• providing advice to Joint Ministers and officials on the content of relevant external communications.

21. Should the Chair be unable to exercise their functions because they are either unavailable or interested in a matter, the Chair may delegate their responsibilities to a temporary Chair to exercise the power and functions of the Chair. This is to be noted in the Minutes where relevant.

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this advice will be needed by will depend on the time taken for the Government to be formed following the election.

2 Exact dates for the interim reporting have not been specified, but it should be noted that Ministers may request updates in the Panel’s work in this timeframe. It is expected the Panel will provide an initial interim report containing advice on the criminal limits for cannabis and methamphetamine.
Responsibilities of all members

Meetings

22. It is anticipated that the majority of Expert Panel meetings will be held by video conference, and members of the Expert Panel are expected to be able to participate in meetings remotely.

23. If meetings are held in person, they will be held in Wellington. Members may attend the meeting in person or by videoconference.

24. The timing and frequency of meetings is to be coordinated with the Secretariat to fit within the allocated budget.

25. Meetings will be up to one working day long, and generally require up to two working days preparation time, however this is likely to vary based on the agreed agenda. Four to six meetings over a 12 month period are expected, although more may be required dependent on the complexity of the issues that emerge, and the level of agreement between the Expert Panel members.

26. Members are expected, prior to each meeting, to have:
   - critically appraised all papers and information provided to the Expert Panel to be considered at the meeting
   - undertaken independent research on the topics to be discussed at the meeting
   - analysed the subject forming an initial professional view for discussion at the meeting.

27. All members are required to provide their view on each subject under consideration at the meeting based on the available scientific evidence and their professional experience. They are required to be prepared to discuss issues related to these subjects with other members in a professional and constructive manner.

28. If one or more members cannot attend a meeting, the Chair may agree to hold the meeting in the members’ absence and forward background papers and notes of the Expert Panel’s preliminary deliberations to the absent member(s).

29. Members who are unable to attend a meeting of the Expert Panel cannot be represented by a substitute or proxy.

30. The Expert Panel members are expected to reach a consensus on recommendations. If a consensus cannot be reached, a recommendation may be advanced based on a majority view at the discretion of the Chair. Any minority views can be recorded in the minutes.

31. Following the meeting, all members are expected to contribute to the finalisation of the meeting minute in a timely manner.

Interest reporting

32. Expert Panel members must declare any interests prior to each meeting.

33. The Chair will determine, in consultation with the Ministry of Transport, the appropriate mitigation steps required for managing each interest that arises and these steps shall be recorded on the conflicts of interest register that is maintained by the Secretariat. Mitigation steps include a member recusing themselves from discussions pertaining to matters they have an interest in, and also not voting on decisions relating to those matters.
34. If members of the Expert Panel develop new, relevant conflicts of interest, whether real, potential or perceived they will inform the Secretariat as soon as is reasonably practicable.

Media

35. Only the Chair is authorised to comment publicly on the affairs of the Expert Panel, and where appropriate, the Chair will advise the Secretariat and the Associate Minister of Transport in advance.

36. The Chair, members and Secretariat will not make or support any action or public statement that is derogatory of or in any way damaging to the Expert Panel.

Conduct

37. Members must perform their functions in good faith, honestly and impartially, and avoid situations that might compromise their integrity or otherwise lead to conflicts of interest. Proper observation of these principles will protect the Expert Panel and its members and will ensure that it retains public confidence.

38. Members must conduct themselves in accordance with the Expert Panel Terms of Reference at all times.

Membership

Appointment of Expert Panel members

39. Members of the Expert Panel are appointed by the Appointing Ministers.

40. In general, the Expert Panel will comprise of technical experts and health practitioners from multiple specialties selected for their expertise in their specialist areas.

41. Members are not appointed as representatives of their primary employer or any other organisation.

42. Membership of the Expert Panel will be listed on the drug driving section of the Ministry of Transport’s website.

Reappointment, removal and resignation

43. Any member of the Expert Panel (including the Chair) continues in their role despite the expiry of his or her term as specified in their letter of appointment until the first of the following events to occur:
   - they are reappointed
   - their successor is appointed
   - Appointing Ministers inform the member by written notice that the member is not to be reappointed and no successor is to be appointed at that time.

44. Any member may be reappointed at the discretion of Appointing Ministers.

45. Any member of the Expert Panel will cease to hold office if he or she resigns, is removed from office, or becomes disqualified for appointment through a conflict of interest or any other matter as identified in their disclosure and consent letter.
46. A member of the Expert Panel may resign from office by written notice to the Associate Minister of Transport (with a copy to the Secretariat) signed by the member. The resignation is effective on receipt by the Associate Minister of Transport of the notice, or at any later time specified in the notice.

47. Appointing Ministers may, at any time and entirely at their discretion, remove a member or cancel an appointment if they consider the member to be no longer fit to fulfil the role as a Panel member. This removal will be made by written notice and will state the date of removal.

48. Members are not entitled for any reason to any compensation or other payment of benefit if they are removed, resign or are not reappointed.

Remuneration of members

49. Expert Panel members will be remunerated in recognition of the services they provide to the Government on the matters outlined in this document. This includes attendance at meetings, time spent preparing for meetings, and for performing any other work as requested by Joint Ministers or officials.

50. The State Services Commission Fees Framework determines the level of fees paid. The fees for the Expert Panel have been set at $650 per day for the Chair, and $500 per day for the members.

51. The Ministry of Transport will cover reasonable travel and accommodation expenses for members to attend meetings.

52. One to two days preparation is expected for each meeting. The Expert Panel is expected to keep the Secretariat informed of the number of days worked.

Relationship with Ministers and officials

53. The Appointing Ministers, or their delegates, may attend each Expert Panel meeting and participate in the discussions.

54. Officials from the Ministry of Transport, NZ Police and the Ministry of Business, Innovation and Employment may also attend and participate in meetings of the Expert Panel. In general, officials may respond to questions from the Expert Panel and clarify understanding of discussion and recommendations as necessary.

55. Officials from the Ministry of Transport will provide administrative and support services for the Expert Panel (see below).

Secretariat support

56. A Secretariat will be established by the Ministry of Transport to support the Expert Panel and assist the Chair in performing their role.

57. The Secretariat will liaise with the panel members to arrange meetings at a suitable time and frequency. The Members of the Expert Panel will keep the Secretariat updated on the number of days worked.

58. The Secretariat is responsible for ensuring a minute of each meeting of the Expert Panel (including by teleconference or other means of communications) is kept and for liaising with the Expert Panel to agree the final version of the minute. The Secretariat will ensure the finalised minute is published at an appropriate time.
59. The Secretariat will maintain a conflicts of interest register.

60. The Secretariat will support the preparation of any requests for information regarding the Expert Panel, and will arrange publication or release of any necessary information.

61. The Secretariat will be available to provide support by sending the agenda and related papers to the Expert Panel members, and managing correspondence between Expert Panel members and third parties.

62. The Secretariat is not a member of the Expert Panel and does not have voting rights at any Expert Panel meeting.

Confidentiality

63. The recommendations of the Expert Panel regarding criminal limits are confidential, until Ministers have considered and made decisions on the final limits.

64. The recommendations of the Expert Panel regarding thresholds for oral fluid devices and detection limits for blood tests are confidential and will not be disclosed publicly.

Official Information Act requests

65. The Secretariat will arrange for publishing of the meeting minutes and any formal written advice prepared by the Expert Panel at an appropriate time on the Ministry of Transport website. Certain information may be withheld in accordance with the Official Information Act.

66. Communications and advice of the Expert Panel will be subject to Official Information Act requests, which will be compiled by the Ministry of Transport.

Disestablishment

67. The Expert Panel will be disestablished when the purpose and functions of the Panel have been completed, as determined by the Appointing Ministers.
Appendix 3

Revised Terms of Reference for the Independent Expert Panel on Drug Driving October 2020

Background to September 2020 update to Terms of Reference

1. The Panel prepared two interim reports for joint Ministers’ consideration:

2. The Panel finalised Report 1 in mid-July 2020, which provides initial advice on criminal limits for THC and methamphetamine using an alternative approach to that originally set out in this Terms of Reference. The Panel prioritised these two impairing drugs due to their prevalence in deceased, hospitalised and impaired drivers in New Zealand.

3. The Panel finalised Report 2 in September 2020, which explains the difficulty the Panel had in meeting its Terms of Reference and recommends an alternative approach to advising on criminal limits and thresholds.

4. The Panel advises that it cannot recommend criminal limits based on drug concentrations in blood that align with drink driving measures of impairment, being equivalent to a blood-alcohol limit of 80mg of alcohol per 100ml of blood.

5. The amendments to this Terms of Reference clarify the approach the Panel is expected to take in establishing the criminal limits for drugs. This change in approach is based on the Panel’s interim advice and does not change the policy intent agreed to by Cabinet.

6. Ministers agreed on 3 October 2020 to amend this Terms of Reference to enable the Panel to provide recommendations on criminal limits and blood and oral fluid thresholds that align with the Government’s desired outcomes. These agreed amendments are reflected in this document.

Context

7. In December 2019, the Government agreed to introduce a compulsory random roadside oral fluid testing regime for drug driving in New Zealand. The design of this oral fluid testing regime requires the following to be developed (refer Figure 1 below):
   a. cut-off thresholds for oral fluid testing devices (above which a driver would show a positive result)
   b. low-level tolerances for blood-drug levels (above which a driver would show a positive result)
   c. criminal limits for drug concentrations in blood (above which a driver would commit a criminal offence).
8. A number of jurisdictions have introduced, or are moving towards, the introduction of legal ‘blood-drug’ limits for illicit drugs and/or medicines, including the United Kingdom, Norway, and several jurisdictions in North America. Like alcohol, these limits have been established as a proxy for impairment, based on scientific research about the impairing effects of different dosages of drugs.

9. The challenge with setting legal ‘blood-drug’ limits is that, unlike alcohol in which the relationship between increasing blood concentration and increasing impairment is very well understood, other drugs behave less consistently (in terms of effects, absorption and elimination from the system). There is little robust scientific data about the effect of drug-blood levels on impairment.

10. Criminal limits and blood and oral fluid thresholds should align with the Government’s policy intent and desired outcomes for the regime, reflected in the table below:

<table>
<thead>
<tr>
<th>Outcome sought</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Criminal limits</strong></td>
</tr>
<tr>
<td>• a high level of confidence that the individual is impaired criminal penalties are only applied where the drug is at a level likely to impair driving</td>
</tr>
<tr>
<td><strong>Thresholds</strong></td>
</tr>
<tr>
<td>• a high level of confidence that the individual has recently consumed the drug</td>
</tr>
<tr>
<td>• drivers taking normal prescription amounts are not detected and able to rely on a medical defence (due to time and cost for the individual and the system)</td>
</tr>
<tr>
<td>• should avoid penalising drivers who have accidental or passive exposure to drugs</td>
</tr>
</tbody>
</table>

11. Given the highly technical nature of determining the thresholds and limits, Cabinet agreed to commission an independent panel of medical and science professionals to provide advice to the Government on these matters. This approach is similar to that of other jurisdictions that have faced these decisions.
12. Cabinet agreed that the independent expert panel of medical and science experts would be appointed by the Associate Minister of Transport, the Minister of Police and the Minister of Research, Science and Innovation (together, the Appointing Ministers) in accordance with the Cabinet Fees Framework for advisory bodies.

**Scope and approach**

13. The Expert Panel will prepare objective advice for the Associate Minister of Transport and the Minister of Police (together Joint Ministers) and make non-binding recommendations on the following topics:
   - the ‘blood-drug’ limits to be specified in legislation (criminal limits)
   - the low-level tolerance thresholds to be applied to the detection of drugs in blood by the Institute of Environmental Science and Research (blood thresholds)
   - the cut-off thresholds to be included in oral fluid testing devices (oral fluid thresholds).
   - any other matters that may be referred to it by Joint Ministers.

14. In preparing advice on criminal limits, it is expected that the Panel will base its recommendations for criminal limits, where possible, on:
   a. limits set in other jurisdictions
   b. drug concentrations in impaired drivers in New Zealand
   c. data from the scientific literature.

15. The Panel is also expected to consider whether its recommendations for criminal limits align with the following outcomes sought by Ministers:
   a. achieve a high level of confidence that the individual is impaired
   b. criminal penalties are only applied where the drug is at a level likely to impair driving.

16. The Panel should reassess its recommendations for THC and methamphetamine to ensure these align with the outcomes sought.

17. In preparing advice on blood and oral fluid thresholds, the Panel is expected to consider whether its recommendations for thresholds align with the following outcomes sought by Ministers:
   a. achieve a high level of confidence that the individual has recently consumed the drug
   b. drivers taking normal prescription amounts are not detected and do not have to rely on a medical defence (due to time and cost for the individual and the system)
   c. avoid penalising drivers who have accidental or passive exposure to drugs.

18. It is expected that the Panel’s final report would specify how each of its recommendations aligns with the outcomes sought.

19. The Panel’s advice on oral fluid thresholds should focus on the thresholds in available testing devices, rather than recommending oral fluid thresholds in isolation. In the next few months Police will be collecting information about available oral fluid devices through its Request for Information process that may be able to inform the Panel’s deliberation on this issue.

20. In addition to the expectations outlined in paragraphs 14 to 19, the Panel may, in preparing advice on any matter within scope of this Terms of Reference, choose to consider relevant scientific literature or data sources and advice of other experts outside of the Panel.

21. Other drug-related policy matters are out of scope of the Expert Panel’s mandate.
22. The Expert Panel will be supported by a Secretariat based at the Ministry of Transport.

23. Advice and recommendations from the Expert Panel will be provided to Joint Ministers in a written report(s), which will cover the points raised in paragraphs 14 to 19.

24. The Government will make final decisions on the matters above, taking into account the technical advice of the Expert Panel alongside advice from officials on other public policy considerations.

25. The Chair and members of the Expert Panel will report to the Associate Minister of Transport about matters outside of the recommendations, such as resignations or no surprises reporting.

**Deliverables and milestones**

26. The Expert Panel will report to Joint Ministers as follows:
   - final advice on criminal limits and blood thresholds to be provided by 20 November 2020 so that it can inform legislation at the Select Committee stage;
   - if the Panel cannot provide advice on all drugs it recommends setting criminal limits for by this time, it should prioritise recommending criminal limits and blood thresholds for drugs that will be tested for in oral fluid testing devices, and then prioritise based on prevalence in New Zealand drivers;
   - final advice on criminal limits and blood thresholds for drugs not included in the 20 November 2020 advice, to be provided by early 2021;
   - final advice on the cut-off thresholds for oral fluid testing devices to be provided by early 2021.

   The dates specified for the deliverables above are approximate and subject to change by agreement between the Expert Panel, the Ministry of Transport and Joint Ministers.

27. The Expert Panel may be asked by Joint Ministers to consider further relevant issues.

28. The Expert Panel may be asked to provide follow-up advice on their recommendations, for example at the Select Committee stage.

**Responsibilities and Appointment of Chairperson**

29. The Chair is appointed by the Appointing Ministers.

30. The Chair is responsible for:
   - setting the agenda for Expert Panel meetings, taking into account matters referred by Appointing Ministers and officials for advice;
   - presiding at each Expert Panel meeting;
   - casting a deciding vote in the event of a tied vote on the recommendation to be made on a particular issue;
   - signing off the final version of the Minute of each meeting;
   - advising Appointing Ministers and officials on appointments to the Expert Panel.

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3 The Land Transport (Drug Driving) Amendment Bill was introduced in July 2020. The Bill is currently before Select Committee, however the Committee is not expected to meet to consider the Bill until November 2020 at the earliest. Final advice on the criminal limits will need to be provided to the Government following the October 2020 General Election so that it can inform the legislation while it is being considered at Select Committee.
• representing the views and interests of the Expert Panel to Government Ministers, Select Committee, officials and departments, and to the media, having first obtained the consent of the Ministry of Transport and, if necessary, Joint Ministers to the act and content of representation
• providing advice to Joint Ministers and officials on the content of relevant external communications.

31. Should the Chair be unable to exercise their functions because they are either unavailable or interested in a matter, the Chair may delegate their responsibilities to a temporary Chair to exercise the power and functions of the Chair. This is to be noted in the Minutes where relevant.

Responsibilities of all members

Meetings

32. It is anticipated that the majority of Expert Panel meetings will be held by video conference, and members of the Expert Panel are expected to be able to participate in meetings remotely.

33. If meetings are held in person, they will be held in Wellington. Members may attend the meeting in person or by videoconference.

34. The timing and frequency of meetings is to be coordinated with the Secretariat to fit within the allocated budget.

35. Meetings will be up to one working day long, and generally require up to two working days preparation time, however this is likely to vary based on the agreed agenda. Four to six meetings over a 12 month period are expected, although more may be required dependent on the complexity of the issues that emerge, and the level of agreement between the Expert Panel members.

36. Members are expected, prior to each meeting, to have:
   • critically appraised all papers and information provided to the Expert Panel to be considered at the meeting
   • undertaken independent research on the topics to be discussed at the meeting
   • analysed the subject forming an initial professional view for discussion at the meeting.

37. All members are required to provide their view on each subject under consideration at the meeting based on the available scientific evidence and their professional experience. They are required to be prepared to discuss issues related to these subjects with other members in a professional and constructive manner.

38. If one or more members cannot attend a meeting, the Chair may agree to hold the meeting in the members’ absence and forward background papers and notes of the Expert Panel’s preliminary deliberations to the absent member(s).

39. Members who are unable to attend a meeting of the Expert Panel cannot be represented by a substitute or proxy.

40. The Expert Panel members are expected to reach a consensus on recommendations. If a consensus cannot be reached, a recommendation may be advanced based on a majority view at the discretion of the Chair. Any minority views can be recorded in the minutes.

41. Following the meeting, all members are expected to contribute to the finalisation of the meeting minute in a timely manner.
**Interest reporting**

42. Expert Panel members must declare any interests prior to each meeting.

43. The Chair will determine, in consultation with the Ministry of Transport, the appropriate mitigation steps required for managing each interest that arises and these steps shall be recorded on the conflicts of interest register that is maintained by the Secretariat. Mitigation steps include a member recusing themselves from discussions pertaining to matters they have an interest in, and also not voting on decisions relating to those matters.

44. If members of the Expert Panel develop new, relevant conflicts of interest, whether real, potential or perceived they will inform the Secretariat as soon as is reasonably practicable.

**Media**

45. Only the Chair is authorised to comment publicly on the affairs of the Expert Panel, and where appropriate, the Chair will advise the Secretariat and the Associate Minister of Transport in advance.

46. The Chair, members and Secretariat will not make or support any action or public statement that is derogatory of or in any way damaging to the Expert Panel.

**Conduct**

47. Members must perform their functions in good faith, honestly and impartially, and avoid situations that might compromise their integrity or otherwise lead to conflicts of interest. Proper observation of these principles will protect the Expert Panel and its members and will ensure that it retains public confidence.

48. Members must conduct themselves in accordance with the Expert Panel Terms of Reference at all times.

**Membership**

**Appointment of Expert Panel members**

49. Members of the Expert Panel are appointed by the Appointing Ministers.

50. In general, the Expert Panel will comprise of technical experts and health practitioners from multiple specialties selected for their expertise in their specialist areas.

51. Members are not appointed as representatives of their primary employer or any other organisation.

52. Membership of the Expert Panel will be listed on the drug driving section of the Ministry of Transport’s website.
Reappointment, removal and resignation

53. Any member of the Expert Panel (including the Chair) continues in their role despite the expiry of his or her term as specified in their letter of appointment until the first of the following events to occur:
   - they are reappointed
   - their successor is appointed
   - Appointing Ministers inform the member by written notice that the member is not to be reappointed and no successor is to be appointed at that time.

54. Any member may be reappointed at the discretion of Appointing Ministers.

55. Any member of the Expert Panel will cease to hold office if he or she resigns, is removed from office, or becomes disqualified for appointment through a conflict of interest or any other matter as identified in their disclosure and consent letter.

56. A member of the Expert Panel may resign from office by written notice to the Associate Minister of Transport (with a copy to the Secretariat) signed by the member. The resignation is effective on receipt by the Associate Minister of Transport of the notice, or at any later time specified in the notice.

57. Appointing Ministers may, at any time and entirely at their discretion, remove a member or cancel an appointment if they consider the member to be no longer fit to fulfil the role as a Panel member. This removal will be made by written notice and will state the date of removal.

58. Members are not entitled for any reason to any compensation or other payment of benefit if they are removed, resign or are not reappointed.

Remuneration of members

59. Expert Panel members will be remunerated in recognition of the services they provide to the Government on the matters outlined in this document. This includes attendance at meetings, time spent preparing for meetings, and for performing any other work as requested by Joint Ministers or officials.

60. The State Services Commission Fees Framework determines the level of fees paid. The fees for the Expert Panel have been set at $650 per day for the Chair, and $500 per day for the members.

61. The Ministry of Transport will cover reasonable travel and accommodation expenses for members to attend meetings.

62. One to two days preparation is expected for each meeting. The Expert Panel is expected to keep the Secretariat informed of the number of days worked.

Relationship with Ministers and officials

63. The Appointing Ministers, or their delegates, may attend each Expert Panel meeting and participate in the discussions.

64. Officials from the Ministry of Transport, NZ Police and the Ministry of Business, Innovation and Employment may also attend and participate in meetings of the Expert Panel. In general, officials may respond to questions from the Expert Panel and clarify understanding of discussion and recommendations as necessary.
65. Officials from the Ministry of Transport will provide administrative and support services for the Expert Panel (see below).

**Secretariat support**

66. A Secretariat will be established by the Ministry of Transport to support the Expert Panel and assist the Chair in performing their role.

67. The Secretariat will liaise with the panel members to arrange meetings at a suitable time and frequency. The Members of the Expert Panel will keep the Secretariat updated on the number of days worked.

68. The Secretariat is responsible for ensuring a minute of each meeting of the Expert Panel (including by teleconference or other means of communications) is kept and for liaising with the Expert Panel to agree the final version of the minute. The Secretariat will ensure the finalised minute is published at an appropriate time.

69. The Secretariat will maintain a conflicts of interest register.

70. The Secretariat will support the preparation of any requests for information regarding the Expert Panel, and will arrange publication or release of any necessary information.

71. The Secretariat will be available to provide support by sending the agenda and related papers to the Expert Panel members, and managing correspondence between Expert Panel members and third parties.

72. The Secretariat is not a member of the Expert Panel and does not have voting rights at any Expert Panel meeting.

**Confidentiality**

73. The recommendations of the Expert Panel regarding criminal limits are confidential, until Ministers have considered and made decisions on the final limits.

74. The recommendations of the Expert Panel regarding thresholds for oral fluid devices and detection limits for blood tests are confidential and will not be disclosed publically.

**Official Information Act requests**

75. The Secretariat will arrange for publishing of the meeting minutes and any formal written advice prepared by the Expert Panel at an appropriate time on the Ministry of Transport website. Certain information may be withheld in accordance with the Official Information Act.

76. Communications and advice of the Expert Panel will be subject to Official Information Act requests, which will be compiled by the Ministry of Transport.

**Disestablishment**

77. The Expert Panel will be disestablished when the purpose and functions of the Panel have been completed, as determined by the Appointing Ministers.
### Appendix 4

**Per se blood drug limits in international jurisdictions**

<table>
<thead>
<tr>
<th>Drug</th>
<th>UK (legislative)</th>
<th>UK (recommended by Expert Panel)</th>
<th>Norway (equivalent to BAC 20 mg/100 mL)</th>
<th>Norway (equivalent to BAC 50 mg/100 mL)</th>
<th>Norway (equivalent to BAC 120 mg/100 mL)</th>
<th>Denmark</th>
<th>Netherlands</th>
<th>Canada (recommended by Committee)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood concentration</td>
<td>ng/mL</td>
<td>ng/mL</td>
<td>ng/mL</td>
<td>ng/mL</td>
<td>ng/mL</td>
<td>ng/mL</td>
<td>ng/mL</td>
<td>ng/mL</td>
</tr>
<tr>
<td>THC</td>
<td>2</td>
<td>5</td>
<td>1.3</td>
<td>3</td>
<td>9</td>
<td>1.1&lt;sup&gt;4&lt;/sup&gt;</td>
<td>3</td>
<td>2 to 5</td>
</tr>
<tr>
<td>methamphetamine</td>
<td>10</td>
<td>200</td>
<td>48</td>
<td>x</td>
<td>x</td>
<td>21</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>amphetamine</td>
<td>250</td>
<td>600</td>
<td>41</td>
<td>x</td>
<td>x</td>
<td>21</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>MDMA</td>
<td>10</td>
<td>300</td>
<td>48</td>
<td>x</td>
<td>x</td>
<td>21</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>cocaine</td>
<td>10</td>
<td>80</td>
<td>24</td>
<td>x</td>
<td>x</td>
<td>21</td>
<td>50</td>
<td>30</td>
</tr>
<tr>
<td>GHB</td>
<td>x</td>
<td>x</td>
<td>10300</td>
<td>30900</td>
<td>123600</td>
<td>x</td>
<td>x</td>
<td>10000</td>
</tr>
<tr>
<td>ketamine</td>
<td>20</td>
<td>200</td>
<td>55</td>
<td>137</td>
<td>329</td>
<td>x</td>
<td>x</td>
<td>zero</td>
</tr>
<tr>
<td>alprazolam</td>
<td>x</td>
<td>x</td>
<td>3</td>
<td>6</td>
<td>15</td>
<td>5.3</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>clonazepam</td>
<td>50</td>
<td>50</td>
<td>1.3</td>
<td>3</td>
<td>8</td>
<td>5.3</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>diazepam</td>
<td>550</td>
<td>550</td>
<td>57</td>
<td>143</td>
<td>342</td>
<td>110</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>lorazepam</td>
<td>100</td>
<td>100</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>21</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>midazolam</td>
<td>x</td>
<td>x</td>
<td>33</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>nitrazepam</td>
<td>x</td>
<td>x</td>
<td>17</td>
<td>42</td>
<td>98</td>
<td>21</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>oxazepam</td>
<td>300</td>
<td>300</td>
<td>172</td>
<td>430</td>
<td>860</td>
<td>110</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>temazepam</td>
<td>1000</td>
<td>1000</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>triazolam</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>zopiclone</td>
<td>x</td>
<td>x</td>
<td>12</td>
<td>23</td>
<td>58</td>
<td>11</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>buprenorphine</td>
<td>x</td>
<td>x</td>
<td>0.9</td>
<td>x</td>
<td>x</td>
<td>0.53</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>codeine</td>
<td>x</td>
<td>x</td>
<td>9</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>fentanyl</td>
<td>x</td>
<td>x</td>
<td>0.34</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>methadone</td>
<td>500</td>
<td>500</td>
<td>25</td>
<td>x</td>
<td>x</td>
<td>53</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>morphine</td>
<td>80</td>
<td>80</td>
<td>9</td>
<td>24</td>
<td>61</td>
<td>10</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>oxycodone</td>
<td>x</td>
<td>x</td>
<td>16</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>tramadol</td>
<td>x</td>
<td>x</td>
<td>53</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

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<sup>4</sup> In 2017 Denmark introduced a progressive sanctioning scale for THC similar to Norway. Fines or prison time increase based on the level of THC identified (low = 1 ng/mL, medium = 3 ng/mL and high = 9 ng/mL), and the number of previous offences.
Appendix 5

Excerpts from reports by international expert panels on drug driving

Excerpt of report from Drugs and Driving Committee (2017) – Canada [4]

“While many jurisdictions have introduced per se limits to help with drug-impaired driving enforcement, to date there has not been a consistent approach used in the development of this type of legislation. Per se limits specify the concentration of a particular drug in the blood or other bodily fluid at or above which it is an offence to operate a motor vehicle, irrespective of any observed driving impairment. As such, the court needs only to determine if the individual’s drug concentration was at or above the specified threshold to determine guilt.

In Canada, a per se limit of over 80 mg of alcohol in 100 mL of blood has been in place since 1969. This per se limit is supported by the epidemiological relationship between blood alcohol concentration (BAC) and crash risk, experimental closed-course driving studies, and laboratory studies of alcohol-induced impairment on specific driving-related tasks and functions. Unlike alcohol, one of the challenges for many potentially impairing drugs is that there is not currently substantive and consistent scientific evidence upon which to base per se limits.

The interest in utilizing a per se approach is an attempt to simplify the adjudication process, facilitate enforcement, and enhance deterrence. Together, these factors can have a positive impact on traffic safety. Research has determined that alcohol per se laws are associated with a 14%-15% reduction in alcohol-related fatal crashes. The relative simplicity of per se laws, their widespread acceptance, and the demonstrated effectiveness of alcohol per se laws, have bolstered the call that similar limits be established for other drugs in Canada.

1985, a National Institute on Drug Abuse (NIDA) sponsored consensus development panel (Consensus Report, 1985) stated “In order to establish that use of a drug results in impairment of driving skills and to justify a testing program to respond to this hazard, certain facts must be available.”

1. The drug can be demonstrated in laboratory studies to produce a dose-related impairment of skills associated either with driving or with related psychomotor functions.
2. Concentrations of the drug and/or its metabolites in body fluids can be accurately and quantitatively measured and related to the degree of impairment produced.
3. Such impairment is confirmed by actual highway experience.
4. Simple behavioural tests, such as can be done at the roadside by police officers with modest training, can indicate the presence of such impairment to the satisfaction of courts.
5. A range of concentrations of the drug can be incorporated in laws relating to impaired driving as ipso facto evidence.

These criteria have been met for ethanol. It is not certain that they can be met for other drugs that are now of concern to highway safety.

It remains challenging to fulfil all five aforementioned criteria for many drugs for several reasons: relevant laboratory studies are limited in part due to the medical and ethical issues with
administering illicit drugs and/or prescription drugs to subjects at the elevated levels detected in impaired driving populations; interpretation of crash and fatality data is complicated by the prevalence of poly-drug use in such cases. Further complications with these data include: the potential for drug concentrations to alter due to variable timeliness of sample collection and, for fatalities, postmortem redistribution, choice of sample collection area, and/or putrefactive changes may result in altered drug concentrations between the time of death and the time of sample collection.”

Excerpt of translated report from Professional Advisory Group – Norway (2010) [6]“The limits are based on scientific assessments of impairment after single doses of the drug in naive individuals. No consideration is given to tolerance phenomena or aberrant handling, including metabolism, of the evaluated compounds.

For most of the 20 substances where fixed limits are proposed, there are epidemiological studies showing that use is associated with increased accident risk.

For alcohol, clear influence / intoxication is usually considered to be present at about 1% [100mg/100mL], and the fixed 0.2% [20 mg/100mL] limit in the Road Traffic Act is 1/5 of the “impact concentration”. For substances other than alcohol, limits are suggested which are also about 1/5 of the concentration seen in blood after taking a regular “intoxication / exposure dose”. This limit is called a ban limit and should represent concentrations in blood where the effect will be of the same magnitude as blood alcohol concentrations of 0.2%.

Punishment metric limits are the limits at which exposure can be compared to alcoholic effects corresponding to 0.5% and 1.2%. Such sentencing limits are set for those substances where there is scientific literature showing dose- and / or concentration-dependent effects comparable to given alcohol concentrations in relevant experimental tests. Such limits are used in the Norwegian judicial system today to determine sanctions in criminal cases that deal with driving in an affected state, cf. the Road Traffic Act.”

Excerpt of report from Expert Panel on Drug Driving (2013) – United Kingdom [5]“The main challenge in establishing recommendations for driving under the influence of psychoactive drugs is the need to provide an easily-understood and justifiable scientific rationale for particular drugs being covered by the offence of drug-driving, whilst recognising that the evidence base is dynamic and will develop as our knowledge and understanding increases. The Panel aimed to establish whether there was sufficient evidence in the scientific literature to be able to determine a relationship between the use of psychoactive drugs and an effect on driving performance in average members of the general public.

Setting a concentration or “limit” for a psychoactive drug, for the new drug driving offence, means that if a driver exceeds this threshold the driver can be prosecuted without the requirement to prove that he or she was impaired and that this impairment was caused by the drug in his body. The implications of setting such a limit in law are therefore far-reaching, and the Panel members accept that their task in advising Government on such limits is crucial. Before recommending drug thresholds the Panel have therefore properly considered both the empirical
(epidemiological) and experimental evidence, in relation to blood drug concentrations and driving behaviour.

Therefore the Panel has not sought to define and measure or proportion a concentration of a drug in a person’s body to a certain degree of impairment. There are two main reasons for this decision. Firstly, there is no universal agreement on how to objectively measure impairment for psychoactive drugs and driving. Secondly, the Panel considered that defining impairment for several different classes of drugs would prove too complicated and not sufficiently robust to inform drug-driving legislation, if such a task could be completed at all.”
Appendix 6

*International oral fluid drug concentration cut-offs*

Recommended confirmation cut-offs from these reports/jurisdictions [14] (units, ng/mL)

<table>
<thead>
<tr>
<th>Drug</th>
<th>AS/NZS 2019</th>
<th>SAMHSA 2015</th>
<th>EWDTS</th>
<th>DRUID</th>
<th>Talloires</th>
</tr>
</thead>
<tbody>
<tr>
<td>THC</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Cocaine</td>
<td>25</td>
<td>8</td>
<td>8</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Benzoylecgonine</td>
<td>25</td>
<td>8</td>
<td>8</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Codeine</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Morphine</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Dihydrocodeine</td>
<td></td>
<td>15</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxycodone</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-acetylmorphine</td>
<td>10</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Methadone</td>
<td>20</td>
<td></td>
<td></td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Tramadol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>Amphetamine</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>25</td>
<td>220</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>25</td>
<td>20</td>
</tr>
<tr>
<td>MDMA</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>25</td>
<td>20</td>
</tr>
<tr>
<td>MDA</td>
<td>25</td>
<td>15</td>
<td>15</td>
<td>25</td>
<td>20</td>
</tr>
<tr>
<td>Alprazolam*</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clonazepam</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diazepam</td>
<td>3</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrazepam</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxazepam</td>
<td>3</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temazepam</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zopiclone</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10</td>
</tr>
</tbody>
</table>

*Not all benzodiazepines specified in these reports are included above, just those prescribed in NZ
Appendix 7

Summary of data used during consideration of recommended per se limits

<table>
<thead>
<tr>
<th>Recreational drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
</tr>
<tr>
<td>THC</td>
</tr>
<tr>
<td>methamphetamine</td>
</tr>
<tr>
<td>amphetamine</td>
</tr>
<tr>
<td>MDMA</td>
</tr>
<tr>
<td>cocaine</td>
</tr>
<tr>
<td>GHB</td>
</tr>
<tr>
<td>ketamine</td>
</tr>
</tbody>
</table>

In determining the recommended criminal and threshold limits for drugs that are considered recreational, a number of aspects were considered. The data used to inform the decisions is presented in the table above and was obtained from a number of different literature sources. Aspects that were considered were, how long the effects of the drug lasted and how quickly the drug was eliminated from the body. Recreational doses can vary widely and will be dependent on the user of the drug. Recreational doses are considered to be doses that will impair a person’s ability to drive safely. For some recreational drugs, studies had been carried out measuring blood drug concentrations after use of a known amount of the drug.

In determining the recommended criminal and threshold limits for medicinal drugs, a number of aspects were considered. The data used to inform the decisions is presented in the table below and was obtained from a number of different literature sources. Aspects that were considered were, how long the effects of the drug lasted and how quickly the drug was eliminated from the body. Also considered was the dose of the drug that could cause impairment and how this impairing dose compared with normal therapeutic dosing. Generally, for medicinal drugs there is good data about therapeutic doses and blood drug concentrations resulting from therapeutic use.
<table>
<thead>
<tr>
<th>Drug</th>
<th>1. Duration (hours)</th>
<th>Half-life (hours)</th>
<th>2. Impairing dose (mg)</th>
<th>3. Standard dose (mg)</th>
<th>4. Dose given (mg)</th>
<th>5. Resulting blood concentration (ng/mL)</th>
<th>6. Expected blood concentration from prescribed dose (ng/mL)</th>
<th>Recommended criminal limit</th>
<th>Recommended threshold limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>alprazolam</td>
<td>4 to 6</td>
<td>6 to 27</td>
<td>&gt;0.5 [1]</td>
<td>0.25 to 0.5</td>
<td>1</td>
<td>30</td>
<td>15</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>clonazepam</td>
<td>4 to 6</td>
<td>19 to 60</td>
<td>1 to 2 [1.5]</td>
<td>0.5 to 2</td>
<td>2</td>
<td>20</td>
<td>20</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>diazepam</td>
<td>12 to 24</td>
<td>21 to 37</td>
<td>&gt; 5 [15]</td>
<td>2 to 10</td>
<td>10</td>
<td>150</td>
<td>150</td>
<td>200</td>
<td>100</td>
</tr>
<tr>
<td>lorazepam</td>
<td>10</td>
<td>9 to 16</td>
<td>1 to 2</td>
<td>0.5 to 2.5</td>
<td>2</td>
<td>20</td>
<td>25</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>midazolam</td>
<td>8</td>
<td>1 to 4</td>
<td>10 to 20</td>
<td>7.5</td>
<td>10</td>
<td>70</td>
<td>50</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>nitrazepam</td>
<td>8</td>
<td>17 to 48</td>
<td>5 to 10 [10]</td>
<td>2.5 to 10</td>
<td>5</td>
<td>50</td>
<td>100</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>oxazepam</td>
<td>4</td>
<td>4 to 16</td>
<td>15 [45]</td>
<td>10 to 30</td>
<td>30</td>
<td>500</td>
<td>500</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>temazepam</td>
<td>12</td>
<td>3 to 13</td>
<td>10 to 20</td>
<td>10 to 30</td>
<td>10</td>
<td>400</td>
<td>1200</td>
<td>800</td>
<td>200</td>
</tr>
<tr>
<td>triazolam</td>
<td>6 to 8</td>
<td>1.8 to 3.9</td>
<td>&lt; 2</td>
<td>0.125 to 0.25</td>
<td>0.5</td>
<td>10</td>
<td>5</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>zopiclone</td>
<td>5</td>
<td>3.5 to 6.5</td>
<td>7.5</td>
<td>7.5 to 15</td>
<td>7.5</td>
<td>80</td>
<td>160</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>buprenorphine</td>
<td>8</td>
<td>2 to 4</td>
<td>0.4 (subling) [0.6]</td>
<td>4 to 24 (subling)</td>
<td>2 (subling)</td>
<td>24</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>codeine</td>
<td>x</td>
<td>1.2 to 3.9</td>
<td>25 to 120</td>
<td>15 to 60</td>
<td>30</td>
<td>70</td>
<td>140</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>dihydrocodeine</td>
<td>x</td>
<td>3.4 to 4.5</td>
<td>60 to 120</td>
<td>20</td>
<td>60</td>
<td>360</td>
<td>200</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>fentanyl</td>
<td>3</td>
<td>2</td>
<td>0.1 mg IV</td>
<td>0.3 mg/hr transdermal</td>
<td></td>
<td>1.0 mg/h</td>
<td>12</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>methadone</td>
<td>x</td>
<td>15 to 55</td>
<td>5 to 10 naive [30]</td>
<td>up to 20 for naive, up to 200 tolerant</td>
<td>15, 100 to 200</td>
<td>75, 830</td>
<td>100, 850</td>
<td>200</td>
<td>50</td>
</tr>
<tr>
<td>morphine</td>
<td>4</td>
<td>1.3 to 6.7</td>
<td>10 to 20 [15]</td>
<td>5 to 20</td>
<td>30</td>
<td>25</td>
<td>20</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>oxycodone</td>
<td>5 to 8</td>
<td>3 to 6</td>
<td>20</td>
<td>5 to 20</td>
<td>10</td>
<td>45</td>
<td>90</td>
<td>50</td>
<td>20</td>
</tr>
<tr>
<td>tramadol</td>
<td>x</td>
<td>4.3 to 8.3</td>
<td>100 to 150</td>
<td>50 to 100</td>
<td>100</td>
<td>300</td>
<td>300</td>
<td>250</td>
<td>100</td>
</tr>
</tbody>
</table>

1. Possible duration of impairment (hours), 2. Considered an impairing dose (mg), 3. Standard prescribed dose (mg), 4. Dose of medicine administered to determine blood concentration (mg), 5. Resulting maximum blood concentration from known administered dose (ng/mL), 6. Maximum blood concentration expected following maximum recommended prescribed dose (ng/mL)
Appendix 8

Analytical issues

There are many drugs, both drugs of abuse and medicinal drugs that may affect the ability to drive safely. All drugs cannot be detected by a single analytical technique. Due to the different chemical natures of the drugs, different extraction techniques are required to separate the drug from the blood before it can be analysed by liquid chromatography with mass spectrometric detection (LC-MSMS).

The current situation (2021) for analysis of LTA blood samples is as follows:

- The current legislation requires only the detection of the drug, not the concentration of the drug in the blood.
- LTA samples are analysed by two LC-MSMS techniques, one for the presence of THC by an analysis specific for that purpose and a separate technique that can detect a limited range of opioids, sedatives and amphetamine type drugs.
- Of the 25 drugs that the Panel has recommended *per se* limits, all but two can be analysed by this second analytical technique.
- THC and GHB require different separate techniques.
- All of the techniques used for the detection of these drugs can also be used to determine concentrations of the drugs in the blood.
- If no drugs are detected by these two techniques, in the blood from a driver who has failed the CIT, further analyses are carried out. The technique used (liquid chromatography with time-of-flight mass spectrometry, LC-TOFMS) can detect a very wide range of medicinal drugs and drugs of abuse but cannot be used to determine the concentration of the drug in the blood.
- Currently analysis for the use of synthetic cannabinoids also requires a separate LC-MSMS technique.

Analytical techniques can change with the availability of new instruments.

Many of the blood drug concentrations determined in NZ drivers and used in this report do not meet the current requirements for forensic reporting. In ESR’s toxicology laboratory the determination of drug concentrations for forensic purposes requires extraction of a minimum of duplicate portions of the blood sample. Since the introduction of the impaired driver legislation in 2009, a blood drug concentration has not been required and in many cases only a single blood sample was extracted. **Therefore, the concentrations of the drugs as determined in NZ drivers in this report should be considered approximate.**

Uncertainty of measurement

Any numerical value determined by any type of measurement has associated with it a degree of uncertainty. For example, measurements of the same two metre length of wood may result in slightly different results (e.g. 2.0, 2.1, 2.05, 2.03 m) depending on the person doing the measurement and the tools used.

The uncertainty associated with a measurement can come from many possible sources, depending on what is being measured, and may include such things as sampling, environmental conditions and volumetric equipment.
Uncertainty of measurement does not imply doubt about the validity of the measurement; on the contrary, knowledge of uncertainty implies increased confidence in the validity of the measurement.

In relation to the concentration of a drug in the blood, the different nature of all blood samples, as in viscosity or matrix effects, can affect a measurement.

Uncertainty associated with the analytical technique used to measure the concentration of a particular drug is determined during the validation of the analytical method. Such validation studies determine:

- precision - the results obtained by repeated analysis of the same sample, that is the same drug at the same concentration in the same blood sample,
- matrix effects - the same drug at the same concentration in different blood samples
- linearity - consistency of results at different drug concentrations
- detection limits - the lowest concentration at which a drug can be consistently identified (limit of detection, LOD) or quantitated (limit of quantitation, LOQ)
- accuracy - the results from analysis of externally sourced, independently produced blood samples spiked with a drug at a known concentration

Analysis of drugs in blood requires access to drug reference standards of known concentration, both in a solution that can be added to blank blood to provide calibration for the instrument and in a blood matrix. Most drugs are stable in an organic solution, that is they do not breakdown over time if kept in dark storage conditions and refrigerated at minus 20°C. However, a number of drugs are not stable in a blood matrix, in particular THC, clonazepam and zopiclone. This makes determination of accuracy of the analysis for these drugs more difficult.

When determining the concentration of a drug in the blood sample provided, the sample is generally analysed at least in duplicate, that is, two measured portions of the blood undergo the analysis.

The analytical technique used to determine the concentration of ethanol (alcohol) in blood samples is called head space gas chromatography with flame ionisation detection. This type of analysis, together with the nature of alcohol, results in low measurements of uncertainty.

For example, at a BAC of around 80 mg/100 mL, the measurement of uncertainty has been determined to be ± 4 mg/100 mL.

Therefore a BAC of 85 mg/100 mL can be reported as follows:

*On analysis of the blood specimen by XX, analyst, a proportion of 85 ± 4 milligrams of alcohol per 100 millilitres of blood was found in the specimen.*

*There is a greater than 99.9% probability that the proportion of alcohol in the blood specimen is greater than 80 milligrams per 100 millilitres.*

The value in this second sentence can change depending on the concentration of alcohol determined. In this case 80 mg/100 mL is the relevant legislative limit to the concentration determined.

This is saying that the most likely result for this analysis is 85 mg/100 mL, and there is only a 0.1% chance that the actual value falls outside the range of 81 to 89 mg/100 mL.

This 5% uncertainty of measurement (UOM) cannot be achieved for other drugs, due to the nature of the drugs, the techniques required to extract the drugs from the blood and the natural variation in blood samples.
During the method validation for the drugs for which per se limits have been recommended, the UOM was found to range from 20% to 33% using the confidence factor (probability) of 94.5%.

In order to report that a blood drug concentration is greater than a legislative limit with 99% probability, it is recommended that a UOM of 50% be applied to all blood drug concentrations determined.

Therefore a certificate might read as follows:

*On analysis of the blood specimen by XX, analyst, a proportion of 5 ± 50% nanograms of tetrahydrocannabinol (THC) per millilitre of blood was found in the specimen.*

*There is a greater than 99% probability that the proportion of THC in the blood specimen is greater than 3 nanograms per millilitre.*

Again, the value in this second sentence can change depending on the drug detected and the concentration of the drug determined and how it relates to any legislative limit.

This recommended UOM means that to be 99% certain that the blood concentration reported is above a per se limit, the calculated concentration must be at least 50% higher than the legislative limit. A safety margin of 99% means that it is possible that one person in 100 may have a lower blood drug concentration. However, a blood drug concentration close to the value determined is most likely to be correct.

This seemingly high value for UOM is not unusual. The Norwegian laboratories have determined the UOM for each drug at the blood drug concentrations that are considered in the low and high blood ranges for their legislation. UOM is higher for higher blood concentrations. The UOM ranged from 19.5% for low concentrations of diazepam to 40% for cocaine [125]. Demark has used the approach recommended above of applying uncertainty of 50% to all blood drug concentrations [3].

**Back calculation of blood drug concentrations**

It is not recommended that back calculations of blood drug concentrations be carried out.

It is not uncommon for there to be a delay between the time that a driver is stopped, or involved in a crash, and the time that a blood sample is taken. A time delay can result in decreasing concentrations of drugs in the blood.

Sometimes requests are made to estimate a blood alcohol concentration at the time of an incident, to determine if the person was above the limit at the time. Such requests may come from Police for prosecution purposes or from insurance investigators. This back calculation can be done for alcohol, due to the nature of the drug and the vast amount of knowledge about how alcohol behaves in the body. Such alcohol back calculations always involve a number of caveats and assumptions and depending on the time delay can result in an estimated BAC that covers a very wide range.

There are too many variables that can affect a blood drug concentration to attempt a back calculation. This includes how much was taken, how often and when, the route of administration and formulation of the drug, but the greatest unknown is each individual’s absorption, metabolism and elimination rates.
Appendix 9

Consideration of the age of a driver

The Panel considers that there are two age groups of drivers that may require a specific focus, drivers aged under 20 years and those aged over 75 years.

The current LTA has different legislative blood and breath alcohol limits for drivers aged under 20 years. Should lower blood drug limits also be applied to the younger drivers?

The older driver, those over 75 require more frequent licencing renewals. At what age a driver can be considered an older driver is open to debate and may be related more to relevant health issues and associated medication(s) that can affect driving.

In order to assess the need to consider these different aged driver populations, data has been collated from analysis of blood samples from deceased, impaired and hospitalised drivers. The proportion of these age groups can be compared with all driver samples received at ESR and drug use by the younger and older drivers can be compared with that of all drivers over the same time period.

The tables below consider the results of the analyses carried out on blood samples received over the period 2013 to 2018: Table A – deceased drivers, Table B- impaired drivers, Table C- hospitalised drivers.

The younger driver accounted for 8% of deceased drivers over the period 2013 to 2018. The drivers aged over 75 accounted for 7% of the deceased drivers.

Table A    Deceased drivers 2013-2018

<table>
<thead>
<tr>
<th></th>
<th>All drivers</th>
<th>Under 20 years</th>
<th>Over 75 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1069</td>
<td>85 (8%)</td>
<td>74 (7%)</td>
</tr>
<tr>
<td>No drugs</td>
<td>438 (41%)</td>
<td>35</td>
<td>40</td>
</tr>
<tr>
<td>Alcohol</td>
<td>289 (27%)</td>
<td>29</td>
<td>1</td>
</tr>
<tr>
<td>Cannabis</td>
<td>267 (25%)</td>
<td>29</td>
<td>-</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>86 (8%)</td>
<td>4</td>
<td>-</td>
</tr>
<tr>
<td>MDMA</td>
<td>4 (0.4%)</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Opioids</td>
<td>86 (8%)</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Sedatives</td>
<td>75 (7%)</td>
<td>-</td>
<td>5</td>
</tr>
</tbody>
</table>

50 (59%) of these young drivers had used a potentially impairing drug, with most using alcohol or cannabis or both.

Although drugs were detected in 34 (46%) of the older drivers, only one had used alcohol and none had used a recreational drug. Most of the drugs detected were not those recommended for legislative limits and were more commonly medicines used to treat heart conditions.
The younger driver accounted for 12% of the impaired driver samples submitted by the police over the six-year period. Very few samples from older drivers were submitted following observed impaired driving.

Table B Impaired drivers 2013-2018

<table>
<thead>
<tr>
<th></th>
<th>All drivers</th>
<th>Under 20 years</th>
<th>Over 75 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cannabis</td>
<td>1120 (59%)</td>
<td>199</td>
<td>-</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>703 (37%)</td>
<td>8</td>
<td>-</td>
</tr>
<tr>
<td>MDMA</td>
<td>20 (1%)</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Opioids</td>
<td>418 (22%)</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Sedatives</td>
<td>304 (16%)</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

Although 12% of the impaired driver samples submitted by the police were the drivers aged under 20, this does not account for young drivers who may have used alcohol. If a driver had a BrAC greater than the limit they rarely have a blood sample taken for drugs analysis. The majority (91%) of the younger drivers had used cannabis.

The younger driver accounted for 10% of the hospitalised driver samples submitted by the police for drug testing. A smaller proportion of samples from older driver were submitted for analysis.

Table C Hospitalised drivers 2013-2018

<table>
<thead>
<tr>
<th></th>
<th>All drivers</th>
<th>Under 20 years</th>
<th>Over 75 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cannabis</td>
<td>717 (37%)</td>
<td>90</td>
<td>-</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>543 (28%)</td>
<td>14</td>
<td>-</td>
</tr>
<tr>
<td>MDMA</td>
<td>28 (1%)</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>Opioids*</td>
<td>291 (15%)</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>Sedatives*</td>
<td>271 (14%)</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

*Have tried to exclude drugs administered by emergency services

Determination of drug use by drivers hospitalised following a crash is made more difficult by the drugs that may be administered by medical personnel either in the ambulance or at hospital prior to an LTA blood sample being taken. Fentanyl, ketamine, midazolam and morphine can be administered in such emergency situations and are often detected in these samples.

Although the younger driver accounted for 10% of the hospitalised driver samples submitted by the police for drug testing, this data does not account for young drivers who may have used alcohol because if a hospitalised driver has a BAC greater than the legal limit for their age, further drugs analyses are rarely carried out.

A smaller proportion of older driver samples were submitted for analysis. It is unlikely that this lower proportion is representative of the older drivers who are hospitalised following a crash. It is possible that drug impaired driving may be thought to be of less concern for the older drivers.

It is clear from the number of samples received at ESR laboratories each year that samples from all hospitalised drivers are not submitted for alcohol or other drug testing.