IN THE CORONERS COURT

OF VICTORIA

AT MELBOURNE

Court Reference: COR 2019 5437

FINDING INTO DEATH WITHOUT INQUEST

Form 38 Rule 63(2)

Section 67 of the Coroners Act 2008

Findings of:	**AUDREY JAMIESON, CORONER**
Deceased:	**Mr P**[1]
Date of birth:	**31 March 1966**
Date of death:	**6 October 2019**
Cause of death:	**Coronary artery atherosclerosis and cardiomegaly in a man using synthetic cannabinoids**
Place of death:	**On board V/Line Train at Broadmeadows Railway Station, Broadmeadows, Victoria 3047**

[1] The Finding has been de-identified at the request of the deceased's family.

Pursuant to section 67(1) of the **Coroners Act 2008**, I make findings with respect to **the following circumstances**:

1. Mr P was 53 years of age and lived in Killara, Victoria, with his parents. He had two daughters and was not in a relationship.

2. At approximately 6.00pm on 6 October 2019, Mr P boarded a V/Line train bound at Southern Cross Station in Melbourne. The V/Line was bound for Albury, New South Wales. The train left the platform.

3. At approximately 6.00pm, Mr P sat down next to another passenger. He appeared unwell and then slumped to one side, struggling to breathe. An off-duty paramedic, Sandon Allen, was on board the train. The paramedic immediately commenced cardio-pulmonary resuscitation and emergency services were contacted.

4. V/Line operators coordinated with Ambulance Victoria and the Metropolitan Fire Brigade, arranging to stop on Platform 3 at Broadmeadows Train Station. Ambulance Victoria paramedics arrived at the platform and attended to Mr P, continuing resuscitative efforts. Mr P was unable to be revived and he was declared deceased by paramedics at 7.12pm.

5. Mr P's death was reportable pursuant to section 4 of the *Coroners Act 2008* (Vic) ('the Act'), because it occurred in Victoria, and was considered unexpected.

INVESTIGATIONS

Forensic pathology investigation

6. Dr Matthew Joseph Lynch, Forensic Pathologist at the Victorian Institute of Forensic Medicine (VIFM), performed an autopsy upon the body of Mr P, reviewed a post mortem computed tomography (CT) scan and referred to the Victoria Police Report of Death, Form 83. Dr Lynch identified a number of significant natural disease processes, including: two vessel coronary artery atherosclerosis and cardiomegaly; he commented that both of these conditions predispose to the development of ventricular tachyarrhythmias. Dr Lynch stated that high blood pressure was a common cause of

cardiomegaly in the community but that he did not have any available evidence of the same in Mr P. Post mortem examination also identified the presence of coronary calcification. There was no evidence of intracranial haemorrhage.

7. Toxicological analysis of post mortem blood detected dozepin[2] and Cumyl-PeGACLONE.[3] The latter is a synthetic cannabinoid belonging to the family of Cumyl derivatives. Dr Lynch stated that synthetic cannabinoids are known to be associated with sudden death, however, the exact physiological mechanism remains unclear; they can induce cardiac arrhythmias.

8. Dr Lynch formulated the medical cause of Mr P's death as coronary artery atherosclerosis and cardiomegaly in a man using synthetic cannabinoids.

COMMENTS

Pursuant to section 67(3) of the *Coroners Act 2008* (Vic), I make the following comments connected with the death:

1. On 26 May 2020, Coroners Prevention Unit Manager[4] of Research and Data Dr Jeremy Dwyer completed a background briefing on Cumyl-PeGACLONE involved deaths. Dr Dwyer completed this briefing at the behest of multiple Coroners investigating deaths that involve synthetic cannabinoids and specifically Cumyl-PeGACLONE. In light of its broad application, I have attached the entire background briefing as an annexure to my findings.

2. Mr P's death was caused from naturally occurring disease in the context of illicit drug use; synthetic cannabinoids. The investigation has not identified whether he knew that smoking synthetic cannabinoids could be more dangerous than smoking cannabis. There is so little definitive information about the effects of novel synthetic substances and

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[2] Dozepin is a dibenzoxepin analogue of amitriptyline and is a tricyclic antidepressant.

[3] The adverse and toxic effects of Cumyl- PeGACLONE have not been reported. Transdermal ("through the skin") absorption of other cumyl-derivatives have cause: ataxia, balance disorder, blurred vision, confusion, dizziness, dry mouth, lethargy, mydriasis, nausea, numbness, orthostatic, hypotension, palpitations, somnolence, tachycardia, vomiting and weakness.

[4] The Coroners Prevention Unit (CPU) was established in 2008 to strengthen the prevention role of the coroner. The unit assists the coroner with research in matters related to public health and safety and in relation to the formulation of prevention recommendations, as well as assisting in monitoring and evaluating the effectiveness of the recommendations. The CPU comprises a team with training in medicine, nursing, law, public health and the social sciences.

there is no practicable way for a user to know precisely which illicit synthetic drugs are being consumed. It thus seems unlikely that Mr P and other users of illicit synthetic drugs would or could anticipate any potential increased risk to health and life.

3. The combination of limited information and general ignorance about the potential risks of synthetic illicit drugs presents a unique challenge to health and governance policymakers. A pertinent recommendation will follow.

RECOMMENDATIONS

Pursuant to section 72(2) of the *Coroners Act 2008* (Vic), I make the following recommendations:

1. With the aim of promoting public health and safety and preventing like deaths, I recommend that the Victorian Department of Health and Human Services review how education regarding synthetic cannabinoids is disseminated to health services and, if deemed appropriate and necessary, develop a training package or similar resource for clinicians to equip them to have conversations with patients about synthetic cannabinoid risks and harm reduction.

FINDINGS

1. I find that Mr P, born 31 March 1966, died on 6 October 2019 at Broadmeadows Train Station, Broadmeadows, Victoria 3047.

2. I find that Mr P boarded an Albury bound V/Line train at Southern Cross Station in Melbourne.

3. I find that the train left the platform and shortly thereafter Mr P became unwell and unconscious.

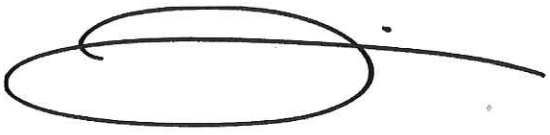
4. The investigation did not identify how Mr P had obtained and ingested the synthetic cannabinoid.

5. I accept and adopt the cause of death formulated by Dr Mathew Joseph Lynch and I find that the cause of Mr P's death was coronary artery atherosclerosis and cardiomegaly in a man using synthetic cannabinoids.

Pursuant to section 73(1A) of the *Coroners Act 2008* (Vic), I order that this Finding be published on the internet.

I direct that a copy of this finding be provided to the following:
Senior Next of Kin
Victorian Department of Health and Human Services
Leading Senior Constable Mark McVea

Signature:



AUDREY JAMIESON
CORONER
Date: **22 July 2020**

Coroners Court of Victoria

COR 2019 5437

CORONIAL INVESTIGATION INTO THE DEATH OF

Mr P

Annexure 1:

Victorian deaths involving the synthetic cannabinoid Cumyl-PeGACLONE

Coroners Court of Victoria
65 Kavanagh Street
SOUTHBANK VIC 3006
Tel: (03) 8688 0700
Email: courtadmin@coronerscourt.vic.gov.au

Coroners Prevention Unit
Background Briefing

Coroners Court of Victoria

To:	Coroner Audrey Jamieson	**Date:**	10 July 2020
From:	Jeremy Dwyer		
Subject:	Victorian deaths involving the synthetic cannabinoid Cumyl-PeGACLONE		

1. Purpose

The Coroners Prevention Unit (CPU) collated this background briefing on Cumyl-PeGACLONE involved deaths at Coroner Jamieson's request, to inform Coroner Jamieson's investigation into a relevant death.

2. Introduction to synthetic cannabinoids

Synthetic cannabinoids are drugs that bind to and activate human cannabinoid receptors to produce subjective effects similar to cannabis, but which are chemically dissimilar to THC (the main psychoactive chemical in cannabis). Synthetic cannabinoids are typically sprayed onto plant matter such as herbs, which are then smoked.

The emergence of synthetic cannabinoids is well documented, with recreational use first being reported in 2004 in Europe before spreading to other regions including the United States, Australia and New Zealand.[1] This occurred as part of a broader trend in new psychoactive substances (NPS): drugs that have not historically featured in drug markets.[2] Underground chemists working in clandestine home laboratories appear to have been the first to produce NPS for drug markets. They were subsequently joined by large-scale commercial laboratories, particularly in China (and to a lesser extent India) where many NPS precursor chemicals are manufactured.[3]

2.1 Types and use

The United Nations Office on Drugs and Crime[4] and the European Monitoring Centre for Drugs and Drug Addiction[5] have documented the existence of hundreds of synthetic cannabinoids in drug markets in Europe and elsewhere, with new compounds appearing regularly. The rapid evolution of synthetic cannabinoids (and NPS more broadly) has been driven at least in part by the legal context. When they first emerged, in most countries they were legal to import, manufacture, possess, sell and use.[6] If concern arose regarding an individual NPS and it was regulated, importers and

1 Winstock A and Barratt M, "Synthetic cannabis: a comparison of patterns of use and effect profile with natural cannabis in a large global sample", *Drug and Alcohol Dependence*, vol 131, 2013: 106-111.

2 For an overview of NPS including definitions and a typology see European Monitoring Centre for Drugs and Drug Addiction, *EU Drug Markets Report*, Luxembourg: Publications Office of the European Union, 2019.

3 European Monitoring Centre for Drugs and Drug Addiction, *EU Drug Markets Report*, Luxembourg: Publications Office of the European Union, 2019, p.178.

4 See United Nations Office on Drugs and Crime, *World Drug Report*, New York: United Nations, May 2016, p.60.

5 See European Monitoring Centre for Drugs and Drug Addiction, *EU Drug Markets Report*, Luxembourg: Publications Office of the European Union, 2019, p.176.

6 For this reason, NPS were often referred to as 'legal highs'.

manufacturers and retailers would simply switch to another NPS that had not yet been subjected to regulation.[7] This cycle was described in 2015 as a:

> *[...] cat-and-mouse process [...] wherein unknown chemists and laboratories are producing new, and as yet nonproscribed, compounds for human consumption; and as soon as they are banned, which they inevitably are, slightly modified analogues are produced to circumvent new laws.*[8]

A more recent commentary noted, specifically with respect to synthetic cannabinoids, that:

> *Suppliers care less about a specific substance and more about mimicking, broadly speaking, the pharmacological effects of cannabis. In essence, each synthetic cannabinoid is disposable: as soon as a substance is controlled, or even before, manufacturers can have one or more replacement substances ready for sale, while suppliers and retailers can have legal replacement products on the shelves.*[9]

This churning of synthetic cannabinoids is reflected in user behaviour: users tend to consume whatever synthetic cannabinoid is being sold, rather than seeking out a particular drug. The ever-changing composition of 'Spice' serves to illustrate this point. Spice was the brand name of an early synthetic cannabinoid preparation offered for sale in Europe, the US, New Zealand and Australia. The brand name remained the same for several years, but the active ingredients were regularly changed to stay ahead of regulation.[10]

2.2 Regulation in Australia

As intimated above, a central challenge for law enforcement was that, at the time when NPS were emerging in drug markets around the world, most jurisdictions regulated drugs on an individual basis. This was certainly the case in Victoria (and Australia), where the regulatory mechanism was the schedules in the Standard for the Uniform Scheduling of Medicines and Poisons. This specified for example that drugs listed in Schedule 9 were illegal to possess, import, manufacture, sell or use except for specific medical or scientific research (ie methamphetamine, heroin); drugs in Schedule 8 were legal to use but only if prescribed under strict conditions because of the risk of misuse and dependence (ie morphine, dexamphetamine, oxycodone); and so on.

Listing individual drugs for regulation is not effective in circumstances where a regulated drug could be replaced immediately by another. To address this, in Victoria the *Drugs Poisons and Controlled Substances Act 1981* (Vic) was amended to insert a new definition, "psychoactive substance",[11] and

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7 Winstock A and Barratt M, "Synthetic cannabis: a comparison of patterns of use and effect profile with natural cannabis in a large global sample", *Drug and Alcohol Dependence*, vol 131, 2013: 106-111; Pierre J, "Cannabis, synthetic cannabinoids, and psychosis risk: what the evidence says", *Current Psychiatry*, vol 10, no 9, 2011: 49-58.

8 Baumeister D, et al, "Legal highs: staying on top of the flood of novel psychoactive substances", *Therapeutic Advances in Psychopharmacology*, vol 5, no 2, 2015: 97-132. Regarding the rapid appearance and disappearance of NPS in Scandinavian markets see Simonsen KW, et al, "Fatal poisoning in drug addicts in the Nordic countries in 2012", *Forensic Science International*, vol 248, 2015: 172-180.

9 European Monitoring Centre for Drugs and Drug Addiction, *EU Drug Markets Report*, Luxembourg: Publications Office of the European Union, 2019, p.178.

10 Baumeister D, et al, "Legal highs: staying on top of the flood of novel psychoactive substances", *Therapeutic Advances in Psychopharmacology*, vol 5, no 2, 2015: 97-132; European Monitoring Centre for Drugs and Drug Addiction and Europol, "Understanding the 'Spice' phenomenon", Luxembourg: Publications Office of the European Union, 2009.

11 "Psychoactive substance" was defined in the Act to be a substance that, when consumed by a person, has a psychoactive effect; or a substance that is represented to have a psychoactive effect when

a new Part IIIA created offences for producing, selling, supplying or advertising a psychoactive substance. This meant that synthetic cannabinoids and other NPS did not need to be listed individually in a schedule to be prohibited. The new laws commenced on 1 November 2017.

In the amended Act, section 56C specified that:

> *It is not a defence to a charge for an offence against this Part that any labelling of, or representation made about, a substance indicates that it is not for human consumption.*

This section was specifically included to address another challenge that faced governments in the early years of NPS regulation: that some NPS were imported into Australia and other countries (particularly from manufacturers in China) as products labelled not for human consumption, to conceal their true purpose and protect importers and vendors from prosecution.[12] One common synonym for NPS, 'bath salts', references the early practice of labelling certain NPS (often synthetic cathinones) as bath salts.

2.3 Toxicology

The rapid changes in the chemical composition of synthetic cannabinoids and other NPS mean that when a new substance appears in a drug market, by the time it is identified and described through toxicological analysis it has usually already been superseded by other NPS. Further to this point, with so many NPS circulating in drug markets, it is difficult (and in some cases possibly pointless) to dedicate time and resources to study the metabolism of a particular NPS in the human body, and to establish which metabolites might be suitable analytical targets for drug screening.[13]

These toxicology issues in turn exacerbate the law enforcement challenge: seizing an NPS or prosecuting somebody for possession and use is difficult when the exact substance is not known and available standard drug tests cannot tell you what it is.

2.4 Risk of harm

Documented adverse effects of synthetic cannabinoid use include:

- Tachycardia.
- Induction of acute psychotic symptoms and exacerbation of existing psychosis.
- Agitation.
- Panic and anxiety.
- Nausea and vomiting.
- Seizures.

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consumed. The definition excluded drugs and poisons otherwise defined in the Act, so that existing pharmaceutical drugs approved for therapeutic use would not be captured under "psychoactive substance".

12 See for example Winstock A, "Harm reduction: only collaboration between regulators, manufacturers and consumers will work", *Prevention in Action*, Australian Drug Foundation, April 2013: 3-4; Simonsen KW, et al, "Fatal poisoning in drug addicts in the Nordic countries in 2012", *Forensic Science International*, vol 248, 2015: 172-180.

13 This point is explored in more depth in Trecki J, et al, "Synthetic Cannabinoid–Related Illnesses and Deaths", *New England Journal of Medicine*, vol 373, no 2, 9 July 2015: 103-107.

- Acute kidney injury.
- Dependence and withdrawal following prolonged use.[14]

2.5 Death

While no deaths from acute cannabis (THC) toxicity have ever been documented, the acute toxic effects of synthetic cannabinoids have been identified as a contributing factor to death in several published systematic reviews.[15] Explanations for its fatal effects include:

- Synthetic cannabinoids are generally more potent than THC (that is, they produce subjective effects at doses far lower than THC).
- Different batches of the same product can contain very different concentrations of synthetic cannabinoid compounds, which may increase the risk of toxicity.
- The cardiac effects of synthetic cannabinoids might be fatal to people who have pre-existing cardiac conditions.
- Synthetic cannabinoids may induce seizures because they lack the anti-convulsant effects that THC produces.[16]

However, forensic pathologists and toxicologists who write case reports about fatalities in a setting of synthetic cannabinoid use have tended to be cautious in attributing the cause of death. For example, in 2015 Victorian Institute of Forensic Medicine (VIFM) Forensic Toxicologist Dimitri Gerostamoulos and colleagues reported three Victorian deaths from unascertained causes where the synthetic cannabinoid PB-22 was detected.[17] They concluded:

> *We remain unsure about the contribution of PB-22 to these deaths however all three deaths (ages 15-35) occurred at home in the setting of using this drug with no other competing cause of death identified. We feel it important that practitioners be aware so they can alert patients who may be experimenting with or using designer drugs such as synthetic cannabinoids. The safety of these substances is largely unknown; and those with an existing heart condition may be at risk of sudden death*

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14 Pierre J, "Cannabis, synthetic cannabinoids, and psychosis risk: what the evidence says", *Current Psychiatry*, vol 10, no 9, 2011: 49-58; Winstock A and Barratt M, "Synthetic cannabis: a comparison of patterns of use and effect profile with natural cannabis in a large global sample", *Drug and Alcohol Dependence*, vol 131, 2013: 106-111; Trecki J, et al, "Synthetic Cannabinoid–Related Illnesses and Deaths", *New England Journal of Medicine*, vol 373, no 2, 9 July 2015: 103-107.

15 For example Trecki J, et al, "Synthetic Cannabinoid–Related Illnesses and Deaths", *New England Journal of Medicine*, vol 373, no 2, 9 July 2015: 103-107; Tait RJ, et al, "A systematic review of adverse events arising from the use of synthetic cannabinoids and their associated treatment", *Clinical Toxicology*, vol 54, no 1, 2016: 1-13; Hvozdovich JA, et al, " Case Report: Synthetic Cannabinoid Deaths in State of Florida Prisoners", *Journal of Analytical Toxicology*, 2020: doi 10.1093/jat/bkz092; Darke S, et al, "Characteristics and circumstances of synthetic cannabinoid-related death", *Clinical Toxicology*, vol 58, no 5, 2020: 368-374.

16 Auwarter V, et al, "'Spice' and other herbal blends: harmless incense or cannabinoid designer drugs?", *Journal of Mass Spectrometry*, vol 44, 2009: 832-837; Winstock A and Barratt M, "Synthetic cannabis: a comparison of patterns of use and effect profile with natural cannabis in a large global sample", *Drug and Alcohol Dependence*, vol 131, 2013: 106-111; Darke S, et al, " Characteristics and circumstances of synthetic cannabinoid-related death", *Clinical Toxicology*, vol 58, no 5, 2020: 368-374.

17 Gerostamoulos D, et al, "Deaths linked to synthetic cannabinoids", *Forensic Science Medicine and Pathology*, vol 11, 2015: 478.

when using drugs which can produce unwanted increases in heart rate and blood pressure.

Similarly, in a 2014 US case series of four deaths where the synthetic cannabinoid 5F-PB-22 was detected in post-mortem blood, the authors concluded:

> *Although synthetic cannabinoids have been associated with sudden death in the USA and abroad, the exact physiological mechanisms for causation, or initiators to contributing factors, remain unclear. […] Clearly, further investigation is required with respect to the pharmacokinetics of 5F-PB-22 and other synthetic cannabinoids, their role in human toxidromes and their relevance to detection in postmortem casework.*[18]

The authors of a 2019 German case series of four deaths where 5F-Cumyl-PeGACLONE was detected, described the circumstances of the deaths in depth to illustrate why the detection of a synthetic cannabinoid does not necessarily mean the synthetic cannabinoid caused the death. The authors commented:

> *Several issues concerning postmortem redistribution, toxic concentration ranges, and possible mechanisms of death remain unsolved and demand increased research efforts and further studies.*[19]

In none of these case studies did the authors dispute that synthetic cannabinoid consumption may cause death. Instead, they pointed out that the scientific understanding of the link between synthetic cannabinoid use and death is still not well understood, and that further research is required. This point was made also in the following conclusion from a 2016 case series study of 25 deaths where synthetic opioids were detected in the US:

> *The cases detailed in this study further demonstrate that synthetic cannabinoid use has been associated with lethal outcomes. Their role, however, in regard to cause and manner of death has not been clearly defined. In part, this is due to the lack of controlled studies and the inability to correlate the concentration of a synthetic cannabinoid to effect especially perhaps, in the presence of other drugs.*[20]

3. Victorian deaths involving Cumyl-PeGACLONE

The first reports of Cumyl-PeGACLONE detection came from Germany in late 2016.[21] The CPU has been unable to find any literature documenting the appearance of Cumyl-PeGACLONE in Australian or Victorian drug markets.

In April 2020 the CPU used the National Coronial Information System (NCIS) to search the text of Victorian autopsy and toxicology reports for the term "pegaclone". The autopsy and toxicology report

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18 Behonick G, et al, "Four Postmortem Case Reports with Quantitative Detection of the Synthetic Cannabinoid, 5F-PB-22", *Journal of Analytical Toxicology*, vol 38, 2014: 559-562.

19 Giorgetti A, et al, "Four cases of death involving the novel synthetic cannabinoid 5F-Cumyl-PeGACLONE", *Forensic Toxicology*, 2019, doi 10.1007/s11419-019-00514-w.

20 Labay L, et al, "Synthetic cannabinoid drug use as a cause or contributory cause of death", *Forensic Science International*, vol 260, 2016: 31-39.

21 See Halter S, et al, "Quantification of Herbal Mixtures Containing Cumyl-PeGACLONE—Is Inhomogeneity Still an Issue?", *Journal of Analytical Toxicology*, vol 44, 2020:81–85; Halter S, et al, "Cumyl-PeGACLONE: a comparatively safe new synthetic cannabinoid receptor agonist entering the NPS market?", *Drug Testing and Analysis*, vol 11, 2019:347-349; Mogler L, et al, "Human phase I metabolism of the novel synthetic cannabinoid 5F-CUMYL- PeGACLONE", *Forensic Toxicology*, vol 37, 2019: 154-163.

for every death identified by the search were reviewed to establish whether it occurred in a context of Cumyl-PeGACLONE use. A death was considered relevant if Cumyl-PeGACLONE was detected in blood or urine.

3.1 Cumyl-PeGACLONE involvement

The CPU identified 12 Victorian deaths for which there was evidence the deceased had used Cumyl-PeGACLONE. They included:

- Four deaths in which the forensic pathologist advised that Cumyl-PeGACLONE played a causal or contributory role. Three of these deaths were overdoses where Cumyl-PeGACLONE was nominated as a contributing drug; the fourth was a death from synthetic cannabinoid use in a setting of heart disease.
- Four deaths in which the forensic pathologist was unable to ascertain whether Cumyl-PeGACLONE played a causal or contributory role. Three of these deaths were from unascertained causes. The fourth was an overdose where the forensic pathologist could not determine what role (if any) Cumyl-PeGACLONE played.
- Three deaths in which the forensic pathologist did not explicitly address whether Cumyl-PeGACLONE played a causal or contributory role. In two of these deaths the forensic pathologist mentioned synthetic cannabinoid use in the cause of death but hedged this by describing it as a "setting" rather than playing a role. The third death was a hanging where Cumyl-PeGACLONE was not mentioned in the autopsy report at all.
- One death for which the autopsy report has not yet been finalised.

3.2 Other potentially relevant deaths

Cumyl-PeGACLONE was not detected in blood or urine during the forensic medical investigation into a recent death (20190210), however was detected in a hair sample. Forensic Pathologist Dr Joanna Glengarry commented as follows:

> *A negative finding [in blood] with regards to synthetic cannabinoids does not necessarily negate their use (although it cannot indicate use). Synthetic cannabinoids may be metabolised rapidly (cleared from the blood). It is also possible that despite a large database of synthetic cannabinoid compounds tested for, a novel (new) compound was present in the drug allegedly smoked by the deceased.*

This raises the possibility that there may be relevant deaths in Victoria involving Cumyl-PeGACLONE or other synthetic cannabinoids, which are being missed in forensic medical investigation.

4. Prevention

Given what is known about synthetic cannabinoids, it appears unlikely that Victorians who died in a setting of Cumyl-PeGACLONE use had specifically sought out Cumyl-PeGACLONE. Instead they were probably synthetic cannabinoid users who, during the fatal incident, happened to be using a product that contained Cumyl-PeGACLONE. On a related point, with the continuing evolution of synthetic cannabinoids it is possible that Cumyl-PeGACLONE may be present only temporarily in Australian drug markets before being replaced by other synthetic cannabinoids. Therefore, any approach to death prevention in this area should target synthetic cannabinoids generally rather than Cumyl-PeGACLONE specifically.

The three domains of drug harm minimisation are supply reduction, demand reduction and harm reduction.[22] The following is a brief literature review of opportunities identified in these three domains to address synthetic cannabinoid related harms, and how these opportunities might be relevant to the Victorian context.

4.1 Supply reduction

Supply reduction refers to interventions that prevent people accessing illegal drugs and regulate their access to legal drugs.

When synthetic cannabinoids and other NPS were first emerging in drug markets and were for the most part legal, there was discussion in Australia regarding how they could be regulated, including the possible adoption of supply reduction strategies already used for alcohol and tobacco:

> *We know that price, age and promotion restrictions work. We could consider the integration of familiar regulations for other consumables such as consumer protection, quality control and trading standards. Mandating producers to include dosing advice, contraindications, side effects and what to do in an emergency, would be an interesting approach. For example, including a warning such as, 'May cause paranoia, psychosis and extreme mood swings'.*[23]

A supply reduction strategy incorporating some of these elements was tested in New Zealand in 2014, allowing NPS products to be approved for legal manufacture and sale if clinical trial data could be produced to show they pose a low risk of harm. The hope was that users would avoid illegal high-risk NPS, and instead seek out lower-risk NPS which could be accessed legally in a controlled and regulated market; however the trial was cut short and no NPS were approved and, by general consensus, it did not achieve its stated goals.[24]

A more recent related suggestion has been to to identify less potent and less harmful synthetic cannabinoids, and allow these to be sold while heavily regulating (or outlawing) their more potent and harmful counterparts.[25]

In Victoria (as in the rest of Australia) synthetic cannabinoids are illegal to import, sell, possess and use, so there is effectively no scope for consideration of any supply reduction measures beyond those that increase the effectiveness of law enforcement. To this end, the literature suggests that improving methods for quickly identifying synthetic cannabinoids, and for detecting use in urine, should assist in enforcement. The latter proposal would address the finding in the literature that a number of consumers were first attracted to synthetic cannabinoids because they could be used without risk of subsequently showing up in urine drug screening.[26]

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22 Commonwealth of Australia, *National Drug Strategy 2017-2026*, Canberra: Department of Health, 2017.

23 See for example Winstock A, "Harm reduction: only collaboration between regulators, manufacturers and consumers will work", *Prevention in Action*, Australian Drug Foundation, April 2013: 3-4.

24 See for example Rychert M and Wilkins C, "New Zealand's review of the Psychoactive Substances Act 2013: a missed learning opportunity ahead of the national cannabis law reform referendum?", *Addiction*, vol 114, no 6, 2019: 1129-1130.

25 Halter S, et al, "Cumyl-PeGACLONE: A comparatively safe new synthetic cannabinoid receptor agonist entering the NPS market?", *Drug Testing and Analysis*, vol 11, no 2, February 2019: 347-349.

26 Trecki J, et al, "Synthetic Cannabinoid–Related Illnesses and Deaths", *New England Journal of Medicine*, vol 373, no 2, 9 July 2015: 103-107; Barrett M, Cakic V, Lenton S, " Patterns of synthetic cannabinoid use in Australia", *Drug and Alcohol Review*, vol 32: 141-146; Diao X and Huestos M, "New Synthetic Cannabinoids: Metabolism and Strategies to Best Identify Optimal Marker Metabolites", *Frontiers in Chemistry*, vol 7, March 2019: 1-15.

4.2 Demand reduction

Demand reduction encompasses strategies to prevent drug use from occurring and to support recovery from drug dependence. The main demand reduction strategy proposed in the Australian literature is a public education campaign focused on the dangers and negative effects of synthetic cannabinoids:

> *Perhaps the most effective public health approach to addressing a class of compounds which are likely to remain widely available is publicity across the less desirable effect profile and risks of increased anxiety and agitation associated with synthetic cannabinoid use.*[27]

A related demand reduction strategy discussed in the literature is to encourage users away from synthetic cannabinoids and towards natural cannabis. Natural cannabis use appears to be less harmful than synthetic cannabis use, and studies have found that drug users strongly prefer natural cannabis over synthetic cannabis if given a choice,[28] so if cannabis was taxed and regulated less stringently than synthetic cannabinoids then consumers would have less incentive to use it.[29] Of course, this strategy is not applicable in the current Victorian context given the legal status of both cannabis and synthetic cannabinoids.

4.3 Harm reduction

Harm reduction encompasses strategies to reduce harmful effects when people use drugs. According to the widely cited Harm Reduction International definition:

> *'Harm Reduction' refers to policies, programmes and practices that aim primarily to reduce the adverse health, social and economic consequences of the use of legal and illegal psychoactive drugs without necessarily reducing drug consumption.*[30]

Harm reduction is underpinned by a recognition that people use psychoactive drugs for a range of reasons and will commence and continue drug use despite any efforts (legal, medical or otherwise) to prevent them from doing so. Accepting this, harm reduction approaches focus on identifying the specific risks and harms that are associated with different types of drug use, and strategies that can be used to reduce these harms when drug use occurs.

One harm reduction opportunity identified in the literature is the need to support basic science in this area. Drugs such as heroin, cocaine, methamphetamine and THC have been extensively studied over decades: their pharmacokinetics and pharmacodynamics are well understood, as are their subjective effects, drug interactions, toxicity in overdose, immediate and long-term consequence of use, potential for dependence and misuse, and so on. By comparison there is far less understanding of synthetic cannabinoids and how they might produce harms; this needs to be addressed to identify harm reduction strategies.[31]

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27 Winstock A and Barratt M, "Synthetic cannabis: a comparison of patterns of use and effect profile with natural cannabis in a large global sample", *Drug and Alcohol Dependence*, vol 131, 2013: 106-111.

28 Winstock A and Barratt M, "Synthetic cannabis: a comparison of patterns of use and effect profile with natural cannabis in a large global sample", *Drug and Alcohol Dependence*, vol 131, 2013: 106-111.

29 Bright S, "The Kronic Chronicles", *Prevention in Action*, Australian Drug Foundation, April 2013: 4-5.

30 Harm Reduction International, "What is harm reduction?", <https://www.hri.global/what -is-harm-reduction>, accessed 6 October 2016.

31 See for example Wiley J, et al, "Hijacking of Basic Research: The Case of Synthetic Cannabinoids", *Methods Reports RTI Press*, November 2011, doi:10.3768/rtipress.2011.op.0007.1111; Behonick G, et al, "Four Postmortem Case Reports with Quantitative Detection of the Synthetic Cannabinoid, 5F-PB-22", *Journal of Analytical Toxicology*, vol 38, 2014: 559-562; Labay L, et al, "Synthetic cannabinoid drug use as

The other main harm reduction strategy discussed in the literature is education for those who use synthetic cannabinoids: for example, educating users who have existing heart conditions about the cardiac effects of the drugs,[32] and advising pregnant women to avoid synthetic cannabinoids because of their potential to interfere with embryo neural development.[33] The finding in Australia that most synthetic cannabinoid and other NPS users are users of other drugs also, suggests that:

> *[...] there may not need to be specialised NPS interventions or harm reduction messages; rather, they could be built into existing responses to drug use and targeted towards illicit drug consumers more generally.*[34]

A harm reduction strategy touched on in the literature but which would not be relevant to the Victorian legal context, is better regulating the manufacture of synthetic cannabinoid containing products to ensure that they contain standard concentrations of active ingredients, rather than the highly variable concentrations that presently occur and which may increase the risk of overdose.[35] A related strategy is labelling of the active ingredients in synthetic cannabinoid containing products, so that consumers know what they are using.

5. Conclusion

While a range of strategies were identified in the literature to reduce harms associated with synthetic cannabinoid use, the applicability of many of these strategies to the Victorian context may require further investigation. Some strategies could not be implemented given the current legal status of both natural cannabis and synthetic cannabinoids; others may be too broad in scope to pursue at a state level.

One strategy that could be investigated further is harm reduction focused education for users. To this end, it should be recognised that there are already education resources for synthetic cannabinoid users available on the Alcohol and Drug Foundation website[36] and the Harm Reduction Victoria website.[37]

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a cause or contributory cause of death", *Forensic Science International*, vol 260, 2016: 31-39; Giorgetti A, et al, "Four cases of death involving the novel synthetic cannabinoid 5F-Cumyl-PeGACLONE", *Forensic Toxicology*, December 2019, doi: 10.1007/s11419-019-00514-w.

32 Gerostamoulos D, et al, "Deaths linked to synthetic cannabinoids", *Forensic Science Medicine and Pathology*, vol 11, 2015: 478.

33 Baumeister D, et al, "Legal highs: staying on top of the flood of novel psychoactive substances", *Therapeutic Advances in Psychopharmacology*, vol 5, no 2, 2015: 97-132.

34 Sutherland R, "New psychoactive substances in Australia: Who uses them, how prevalent are they, and where are they sourced?", *Drug and Alcohol Research Connections*, June 2019, <http://connections.edu.au/researchfocus/new-psychoactive-substances-australia-who-uses-them-how-prevalent-are-they-and-where>, accessed 20 April 2020.

35 Halter S, et al, "Quantification of Herbal Mixtures Containing Cumyl-PeGACLONE: Is Inhomogeneity Still an Issue?", *Journal of Analytical Toxicology*, vol 44, 2020:81–85.

36 Alcohol and Drug Foundation, "Synthetic cannabis", 26 June 2019, <https://adf.org.au/drug-facts/synthetic-cannabis/>, accessed 26 May 2020.

37 Harm Reduction Victoria, "Resources", undated, <https://www.hrvic.org.au/resources>, accessed 26 May 2020.