Coalition of Alcohol and Drug Educators

Dalgarno INSTITUTE

Western Australian Parliament – Inquiry into Cannabis and Hemp

Dalgarno Institute Submission

https://www.parliament.wa.gov.au/Parliament/commit.nsf/(EvidenceOnly)/78055DF4E73607E74825876F001F67DD?opendocument#ToR

Terms of Reference:

(1) A Select Committee to be known as the Cannabis and Hemp Select Committee, is established.

Member Conducting Enquiry

Hon. Dr Brian Walker MLC
Hon. Matthew Swinbourn MLC
Hon. Jackie Jarvis MLC
Hon. Lorna Harper MLC
Hon. James Hayward MLC

(2) The Select Committee is to inquire into and report on the **potential to amend the current legislation** and regulations which apply to **cannabis** and h**emp in Western Australia**, with particular reference to —

(a) the current barriers to **pharmaceutical nutraceutical** use of **cannabinoid products;**
(b) **medicinal cannabis**, its **prescription**, availability and **affordability**; and
(c) the **potential benefits** and **risks** of **permitting industrial hemp** for **human consumption.**

Parliament of Western Australia

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Dear Committee Members,

Thank you for the opportunity to make a submission on an extremely important issue. The regulation of Cannabis and its derivatives is an incredibly important public health and safety issue. Political and policy decisions have the capacity to shape public health and well-being policy for either the better of the worse. When it comes to Cannabis regulation, and more importantly any further 'liberalization' of it, extreme caution must be exercised – not merely for the present society but for generations to come.

Dalgarno Background

Dalgarno Institute has well over 150 years' experience with alcohol and other drug issues, including counselling, educating and researching around the subject of cannabis.

The organisation has been at the forefront of demand reduction and primary prevention strategies. It has engaged with clients from a wide cross section of demographic and socio-economic communities who have shared their experiences, narratives and outcomes of drug abuse. The majority either started with or remain chronically dependent on cannabis.

Introduction

There is very real evidence-based research and best-practice health for the following *strong reluctance to prescribe* a product with very limited clinically proven therapeutic capacity.

Even four more years on, and with a veritable tsunami of anecdotal frenzy, science still has not come close to affirming even some of the therapeutic claims, of this very limited and now highly engineered 'plant'.

This is not helped by simply changing TGA protocols to *'remove unnecessary bureaucracy'* and poor evidence-based attempts at the coercive promotion by the pro-cannabis industry and again, it's a highly propagandized anecdotal evidence around this still unproven and pharmaceutically *under-trialled* and unpredictable substance.

> ***Medicinal cannabis access a challenge for WA patients despite legalisation last year***
>
> *West Australian patients are finding it almost impossible to obtain medicinal cannabis more than eight months after it was legalised, advocates say.*
>
> *By May, no health professional had applied to prescribe the drug, despite it being made legal in November.*
>
> *Ms Neville said some doctors seemed reluctant to try to prescribe the drug.*
>
> *Health Minister Roger Cook said the drug companies behind the products needed to educate doctors more on how the products could be used, and their benefits.*
>
> *"I have called on the industry to step up to that roll and to consider why — when clearly the WA community expressed a desire to see these products made available — they are not being accessed," he said.*

AMA cautious about medicinal cannabis

But the Australian Medical Association of WA said it remained cautious about the use of medicinal cannabis.

AMA WA president Omar Khorshid said it was important rules around the use of medicinal cannabis remained strict, as its efficacy was still being tested.

"The AMA is certainly not supportive of shortcuts, and instead of avoiding all the regulatory steps, we should be investigating cannabis-based products, how good they are, how safe they are, and once that's been done, they should available just like any other drug," he said.

"The AMA is calling for more research on cannabis-based drugs so that we know what's in them, how well they work, and how safe they are, and once that's done, we'll be able to prescribe to prescribe better drugs for patients to manage these conditions."

Ms Neville said there was international research to show cannabinoid-based products were safe and efficient.

The Department of Health said an application was yet to be received from Ms Neville's doctor, and the department had contacted this doctor to provide information and regulatory assistance.

By Georgia Loney Updated 7 Jul 2017 https://www.abc.net.au/news/2017-07-07/families-struggling-to-access-to-medicinal-cannabis/8685712

Any legislative agendas and mechanisms that seek to further negate best practice process of thorough and exhaustive clinical trials would be at least concerning, at best – and time will confirm this with growing **genotoxic evidence** – that hasty short-cuts in the unleashing of this psychotropic toxin into our communities will border of negligence, if not culpability.

Short and long term harms that already surround this substance, should be prompt enough for those considering legislative 'laxity' to pause and thoroughly consider such moves, as those who do decide on measures that only add to the public harm will, or should be, liable for those very negative outcomes.

This submission is just an overview of the growing body of evidence unfurling from those researchers and scientists, who have not succumbed to the fiscal draw, or the anecdotal spin.

Coalition of Alcohol and Drug Educators

Dalgarno INSTITUTE

CBD, Non-THC and THC Analog Cannabinoid Extracts & Potential Harms From 'Anecdotal Evidence' Based Policy Making.

www.dalgarnoinstitute.org.au | 1300 975 002 | PO Box 7005, Dandenong, Vic 3175

CBD, Non-THC and THC Analog Cannabinoid Extracts & Potential Harms From 'Anecdotal Evidence' Based Policy Making.

Medical Marijuana Is Not Regulated as Most Medicines Are

The industry lacks randomized controlled clinical trials that can clearly establish benefits and risks of the same health concerns raised decades ago about using marijuana therapeutically are still unresolved, even as the potency of the plant's intoxicating ingredient, Delta 9 -Tetrahydrocannabinol, best known as THC, has increased at least fivefold. Furthermore, exclusive medical use is uncommon; in a Canadian study of 709 medical users, 80.6 percent also reported using marijuana recreationally.

"People are using a medical excuse for their recreational marijuana habit," said Dr. Kenneth Finn, a pain management specialist in Colorado Springs and editor of a new, 554-page professional book on the subject, "Cannabis in Medicine: An Evidence-Based Approach."

The evidence — or lack thereof — of health benefits that can be reliably attributed to smoking, vaping or ingesting marijuana, even in its purest form, is described in great detail in Dr. Finn's book. "Components of the cannabis plant can help in various conditions, but that's not what people are buying in stores," he said in an interview. "Let's do the research on purified, natural, noncontaminated cannabinoids," as the various potentially therapeutic chemicals in marijuana are called.

Three such substances have been approved by the Food and Drug Administration. One, Epidiolex, a cannabidiol-based liquid medication, is approved to treat two forms of severe childhood epilepsy. The others, dronabinol (Marinol, Syndros) and nabilone (Cesamet), are pills used to curb nausea in cancer patients undergoing chemotherapy and to stimulate appetite in AIDS patients with wasting syndrome.

Another marijuana-based drug, nabiximols (Sativex), is available in Canada and several European countries to treat spasticity and nerve pain in patients with multiple sclerosis.

Medicinal cannabis is hardly a new therapeutic agent. It was widely used as a patent medicine in the United States during the 19th and early 20th centuries and was listed in the United States Pharmacopoeia until passage of the Marijuana Tax Act in 1937 rendered it illegal.

Then a federal law in 1970 made it a Schedule 1 controlled substance, which greatly restricted access to marijuana for legitimate research. Also complicating attempts to establish medical usefulness is that plants like marijuana contain hundreds of active chemicals, the amounts of which can vary greatly from batch to batch. Unless researchers can study purified substances in known quantities, conclusions about benefits and risks are highly unreliable.

That said, as recounted in Dr. Finn's book, here are some conclusions reached by experts about the role of medical marijuana in their respective fields:

Pain Management:
People using marijuana for pain relief do not reduce their dependence on opioids. In fact, Dr. Finn said, "patients on narcotics who also use marijuana for pain still report their pain level to be 10 on a scale of 1 to 10." Authors of the chapter on pain, Dr. Peter R. Wilson, pain specialist at the Mayo Clinic in Rochester, Minn., and Dr. Sanjog Pangarkar of the Greater Los Angeles V.A. Healthcare Service, concluded, "Cannabis itself does not produce analgesia and paradoxically might interfere with opioid analgesia." A 2019 study of 450 adults in the Journal of Addiction Medicine found that medical marijuana not only failed to relieve patients' pain, it increased their risk of anxiety, depression and substance abuse.

Multiple Sclerosis:
Dr. Allen C. Bowling, neurologist at the NeuroHealth Institute in Englewood, Colo., noted that while marijuana has been extensively studied as a treatment for multiple sclerosis, the results of randomized clinical trials have been inconsistent. The trials overall showed some but limited effectiveness, and in one of the largest and longest trials, the placebo performed better in treating spasticity, pain and bladder dysfunction, Dr. Bowling wrote. Most trials used pharmaceutical-grade cannabis that is not available in dispensaries.

Glaucoma:
The study suggesting marijuana could reduce the risk of glaucoma dates back to 1970. Indeed, THC does lower damaging pressure inside the eye, but as Drs. Finny T. John and Jean R. Hausheer, ophthalmologists at the University of Oklahoma Health Sciences Center, wrote, "to achieve therapeutic levels of marijuana in the bloodstream to treat glaucoma, an individual would need to smoke approximately six to eight times a day," at which point the person "would likely be physically and mentally unable to perform tasks requiring attention and focus," like working and driving. The major eye care medical societies have put thumbs down on marijuana to treat glaucoma.

Mental Health:
Allison Karst, a psychiatric pharmacy specialist at the V.A. Tennessee Valley Healthcare System, who reviewed the benefits and risks of medical marijuana, concluded that marijuana can have "a negative effect on mental health and neurological function," including worsening symptoms of PTSD and bipolar disorder.

Dr. Karst also cited one study showing that only 17 percent of edible cannabis products were accurately labeled. In an email she wrote that the lack of regulation "leads to difficulty extrapolating available evidence to various products on the consumer market given the differences in chemical composition and purity." She cautioned the public to weigh "both potential benefits and risks," to which I would add caveat emptor — buyer beware.

(Source: 8/3/2021 https://www.nytimes.com/2021/03/08/well/live/medical-marijuana.html)

Further Problems with DELTA – 8 THC & Other THC Analogs

Why is the United States passively allowing the manufacture, sale, and use of Delta 8-THC, Delta 10-THC, and THC-O, all of which are harmful chemicals that may be even more damaging than nicotine/tobacco? Why is this happening in a supposedly science-based society? Could this be occurring because of loopholes in the 2018 Agriculture Bill?

Currently, hemp-derived CBD is being synthesized and converted into these chemicals, which are then made into different products for retail sale.[1] [2] [3] We can see many of these products being advertised for sale online, in vape & smoke shops, gas stations, and convenience stores in almost every state. These items are even being marketed to children through the use of very inviting packaging and products, such as the all popular gummy bears. Luckily, some state authorities have decided to stop the sale of these products because health officials have identified that there is a potential for serious harm when using these chemicals.

The FDA even recently issued a warning about one of these synthetically produced chemicals, namely Delta 8-THC: https://www.fda.gov/consumers/consumer-updates/5-things-know-about-delta-8-tetrahydrocannabinol-delta-8-thc.

Currently, because CBD is very economical to procure, it is being used as a feedstock to synthesize these mind-altering substances using dangerous chemicals.

CBD turned into dangerous THC analogs

1. Delta 8-THC[1] – This chemical is being synthesized using CBD, an acid, and an organic solvent, such as toluene, which is usually used in the manufacture of paint products. This chemical exists naturally in cannabis plants but at very low levels; therefore, it must be synthesized for mass production.

2. Delta 10-THC[2] – This chemical was discovered by a random accident and resulted from the extraction and distillation of THC from marijuana plants that were exposed to a fire retardant used to combat a nearby forest fire. It too can be synthesized from CBD. This chemical does not occur naturally.

3. THC-O[3] – This chemical is also being synthesized using not only CBD, an acid, and an organic solvent but by also using a very toxic chemical needed to complete the process. If synthesized from its nearest analog molecule, specifically Delta 9-THC, it can be made by using only the last step, specifically through the use of a very toxic chemical. It is three times more potent when compared to Delta 9-THC. This chemical also does not occur naturally.

The Basic Chemistry

CBD is considered a hydrocarbon molecule comprised of twenty-one carbon atoms, thirty hydrogen atoms, and two oxygen atoms. It can therefore be denoted as $C_{21}H_{30}O_2$. There are other molecules that have the exact same number of carbon, hydrogen, and oxygen atoms, and they are known as isomers.[4]

Both the Delta 8-THC and Delta 10-THC variants are isomers of CBD. Also, the naturally occurring and psychoactive Delta 9-THC in cannabis plants (hemp and

marijuana) is an isomer of CBD. All of these variants thus have the same molecular formula, namely $C_{21}H_{30}O_2$. However, they are different structurally.[5]

A simple comparison to consider would be through the use of a chemical Lego® block model.[6] For an example, a carbon atom could be represented by a red block, a hydrogen atom could be represented by a white block, and an oxygen atom could be represented by a blue block. For the CBD molecule, one could symbolically represent it by connecting twenty-one red blocks (C_{21}), thirty white blocks (H_{30}), and two blue blocks (O_2) all together in a manner to represent its structural arrangement. Using the exact same number of blocks, differently shaped models could be made to represent Delta 8-THC, Delta 9-THC, and Delta 10-THC.

Chemistry allows for the alteration of CBD

In the example above, the CBD building block model would be altered structurally by one's hands. In the world of chemistry, structural alterations to CBD can be facilitated through the use of different levels of temperature, various catalysts (in the form of an acid), and various organic solvents (such as toluene) into other molecules such as Delta-8, Delta-9, and Delta-10 THC.[1] [2] There are also by-products formed after the structural transformation along with some residual acid and organic solvent.

In the FDA-regulated pharmaceutical world, the by-products and solvents are removed, and the acid is neutralized. However, few, if any, safeguards exist in the cannabis world because there is no regulatory oversight by the FDA.[7]

Keep in mind that the manner in which a molecule interacts and affects the human body and mind is dependent upon its atomic makeup, specifically the number and type of atoms present, and its structural arrangement.[8] In the case of CBD and its isomers, the way that they can affect the body and mind can be traced back to how they interact with the receptors where they connect. A CBD molecule is molecularly the same but structurally different from the other variants of the THC molecule, hence they can each affect the body and mind in similar and/or completely different ways.

Delta 8-THC – Part 1

Delta 8-THC was first synthesized in 1967 by Israeli chemist Raphael Mechoulam.[5] It exists naturally in cannabis plants, but only in very small (normally ~0.1%)[1] or in trace amounts.[5] There was therefore a need to synthesize the molecule so that it could be studied in depth because of its rarity in cannabis plants.

The passage of the Hemp Bill and subsequent rush to cash in on this crop via the production of CBD has resulted in a glut of this chemical in the marketplace.[10] However, it has been known for decades that CBD can be synthesized into other molecules, and one of those molecules is Delta 8-THC[5], which can have reportedly similar but milder mind-altering effects as Delta 9-THC.[11]

Multiple concerns arise when synthesizing Delta 8-THC from CBD. What is especially concerning is that it may not be processed in a manner that guarantees the safety of the consumer. Since a strong acid and an organic solvent such as toluene, which is normally used in paint products, can be used, the resulting product needs to be "washed" in a base, in order to neutralize any residual acid and any remaining organic solvent needs to be removed. There are also other by-products that may need to be removed, some of which are unknown. The reaction by-products can also include traces of Delta 9-THC. Given that there are few, if any, testing protocols

and almost no regulatory oversight for this product, it is basically the "Wild West" for the consumer.[7 9 12]

Delta -8 compared to Delta -9 THC

Delta-8 THC is reportedly weaker in its effects when compared to Delta-9 THC, which has many unwanted side effects including cyclic nausea and vomiting, anxiety, and paranoia.[13 14] Delta 9-THC can also trigger mental illnesses such as psychosis and schizophrenia, especially in teens.[15] Could these same side-effects, even if slightly milder, also apply to Delta-8 THC? Only time will tell, but anecdotally, the answer appears to be "yes".

There is currently no scientific research about the long-term effects of Delta 8-THC use. Therefore, anyone who uses this product becomes an unwitting participant in a science experiment, an experiment that could result in addiction, mental illnesses, and/or bodily harms.

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Cannabis sativa (Hemp) Seeds, D9-Tetrahydrocannabinol, and Potential Overdose

Abstract - Introduction:

Cannabis sativa (hemp) seeds are popular for their high nutrient content, and strict regulations are in place to limit the amount of potentially harmful phytocannabinoids, especially D9-tetrahydrocannabinol (D9-THC). In Canada, this limit is 10 lg of D9-THC per gram of hemp seeds (10 ppm), and other jurisdictions in the world follow similar guidelines.

Discussion:

We discovered that D9-THC concentrations in these hemp seeds could be as high as 1250% of the legal limit, and the amount of phytocannabinoids depended on the extraction procedure employed, Soxhlet extraction being the most efficient across all three brands of seeds. D9-THC and CBD exhibited significant variations in their estimated concentrations even from the same brand, reflecting the

inhomogeneous nature of seeds and variability due to the extraction method, but almost in all cases, D9-THC concentrations were higher than the legal limit. These quantities of total D9-THC may reach as high as 3.8mg per gram of hemp seeds, if one were consuming a 30-g daily recommended amount of hemp seeds, and is a cause for concern for potential toxicity. It is not clear if these high quantities of D9-THC are due to contamination of the seeds, or any other reason.

(Source: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5665515/ 06 May 2019)

EU drug agency flags CBD market issues in report on low-THC cannabis products

The European Union's drug agency has signaled that it is mulling the legal and commercial status of CBD and cannabis-based products in Europe in a new report.

The Lisbon-based European Monitoring Centre for Drugs and Drug Addiction published the report on Monday, detailing the results of a study that kicked off in 2018 and aimed to provide insight into the open sale of low-THC products in Europe.

"The specific objectives were to identify and further explore the types of product available and the range of sales outlets, user profiles, associated harms and responses taken in different EU countries," the authors wrote.

The Centre took note of several glaring issues with the marketing and promotion of some CBD products across Europe, including:

(Source: https://hempindustrydaily.com/eu-drug-agency-flags-cbd-market-issues-in-report-on-low-thc-cannabis-products/ February 2021)

Growing Research Finds CBD not 'harmless' and Can Cause Serious Harm.

Cannabidiol Adverse Effects and Toxicity – Research

Abstract

Background:

Currently, there is a great interest in the potential medical use of cannabidiol (CBD), a non-intoxicating cannabinoid. Productive pharmacological research on CBD occurred in the 1970s and intensified recently with many discoveries about the endocannabinoid system. Multiple preclinical and clinical studies led to FDA-approval of Epidio-lex®, a purified CBD medicine formulated for oral administration for the treatment of infantile refractory epileptic syndromes, by the US Food and Drug Administration in 2018.

The World Health Organization considers rescheduling cannabis and cannabinoids. CBD use around the world is expanding for diseases that lack scientific evidence of the drug's efficacy. Preclinical and clinical studies also report adverse effects (AEs) and toxicity following CBD intake.

Methods:

Relevant studies reporting CBD's AEs or toxicity were identified from PubMed, Cochrane Central, and EMBASE through

January 2019. Studies defining CBD's beneficial effects were included to provide balance in estimating risk/benefit.

Results:

CBD is not risk-free. In animals, CBD AEs included developmental toxicity, embryo-fetal mortality, central nervous system inhibition and neurotoxicity, hepatocellular injuries, spermatogenesis reduction, organ weight alterations, male reproductive system alterations, and hypotension, although at doses higher than recommended for human pharmacotherapies. Human CBD studies for epilepsy and psychiatric disorders reported CBD-induced drug interactions, hepatic abnormalities, diarrhea, fatigue, vomiting, and somnolence.

Conclusion:

CBD has proven therapeutic efficacy for serious conditions such as Dravet and Lennox-Gastaut syndromes and is likely to be recommended off label by physicians for other conditions. However, AEs and potential drug interactions must be taken into consideration by clinicians prior to recommending off-label CBD.

(Source: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7052834/ 2019)

FDA Drug Trials Snapshots: Epidiolex (CBD)

EPIDIOLEX (cannabidiol) EH-peh-DYE-oh-lex Greenwich Research Ltd Approval date: June 25, 2018

DRUG TRIALS SNAPSHOT SUMMARY:

What is the drug for?

EPIDIOLEX is a drug for the treatment of seizures in two rare and severe forms of epilepsy, Lennox-Gastaut syndrome and Dravet syndrome, in patients two years of age and older.

Lennox-Gastaut and Dravet syndromes start during early childhood. They are associated with difficult to control seizures and various degrees of development disability.

How is this drug used?

EPIDIOLEX is a liquid solution that is taken twice daily by mouth to control seizures. It is usually taken with other drugs. The dose of EPIDIOLEX is based on the patient's weight. EPIDIOLEX is started at a low dose. After one week, the dose can be increased weekly based on the patient's response and ability to tolerate the drug.

What are the possible side effects?

EPIDIOLEX may cause serious side effects including increase in liver enzymes, sleepiness, *thoughts about suicide or dying*, and severe allergic reactions.

The most common side effects of EPIDIOLEX are sleepiness, decreased appetite, diarrhea, increase in liver enzymes, lack of energy, and rash.

(Source: https://www.fda.gov/drugs/drug-approvals-and-databases/drug-trials-snapshots-epidiolex 2018)

The Impact of Cannabidiol on Psychiatric and Medical Conditions

Conclusion

More studies need to be done in humans in a controlled setting to determine the medicinal value of CBD for various diagnoses in order to be able to make clear recommendations.

Preclinical studies have shown some promising data regarding the medicinal value of CBD but studies in human are not consistent in outcome and controversial in their design. More studies need to be performed in human with larger sample sizes and longer follow-up periods.

Dosing guidelines for CBD need to be established for different indications and follow-up with a physician. The current situation, where the user does not know what they are actually getting in the product, makes CBD unsafe and the risk outweighs the benefit of recommending or using that substance. Especially the content of CBD needs to be under stricter regulations with lab monitoring to determine and guarantee a particular dose or content or CBD and to exclude a higher content of THC than the 0.3% that are allowed by law. Currently there is no consistency in the content of the CBD product.

It is dangerous to assume that the CBD is a "miracle drug" without any safety concerns given the list of potential toxic reactions. Particularly the sedating effect appears to be concerning and limiting in its use.

Cross-interactions with other medications need to be investigated to rule out toxicity in co-morbid patients taken numerous prescribed medications.

A possible development of an addictive disorder to CBD can, from the current knowledge, not be excluded and further data on long-term administration, the effects of tolerance and toxicity with administration of higher doses need to be investigated. From experience, if a substance is used over a prolonged period of time, there is a process of habituation involved and consecutively an increase in consumption of the substance.

Also, the claimed absence of psycho-active effects and absence of withdrawal symptoms upon discontinuation of CBD is from current point of view, subject to speculation.

The current trend of decriminalization of marijuana and its products bear the risk to further increase the CBD consumption with associated increase in health problems, violence, criminality and lethality.

(Source: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7331870/ 2020)

CBD and Liver Damage

Researchers at the University of Arkansas for Medical Science recently rolled up their sleeves to investigate CBD hepatotoxicity in mice. What they found was while this cannabis derivative is gaining significant recognition as of late in the world of wellness, people that use CBD are at an elevated risk for liver toxicity.

The findings, which were published earlier this year in the journal *Molecules*, suggest that while people may be using CBD as a safer alternative to conventional pain relievers, like acetaminophen, the compound may actually be just as harmful to their livers.

(Source: https://www.forbes.com/sites/mikeadams/2019/06/18/marijuana-study-finds-cbd-can-cause-liver-damage/?sh=2391e38843ff June 2019

(Also see **Hepatotoxicity of a Cannabidiol-Rich Cannabis Extract in the Mouse Model** 2019)

Medicinal cannabis blacklisted by Australian pain specialists

Doctors are being told not to use medicinal cannabis to treat patients with chronic pain, warning there is no solid evidence it is effective, as Australia's medical regulator approves its 100,000th cannabinoid script.

The recommendation from the country's peak pain advisory body to doctors is:

"Do not prescribe currently available cannabinoid products to treat chronic non-cancer pain unless part of a registered clinical trial."

The Faculty of Pain Medicine at the Australian and New Zealand College of Anaesthetists (ANZCA) says there is no robust evidence

from gold-standard studies that proves cannabinoid products effectively treat these patients' suffering. Cannabinoids are the active chemicals in cannabis.

But the Therapeutic Goods Administration (TGA) is allowing doctors to apply for special access to prescribe medicinal cannabis products. Proponents argue the substances should be given the benefit of the doubt and offered to patients on compassionate grounds.

Dean of ANZCA's pain medicine faculty Professor Michael Vagg said medicinal cannabis products on the market "are not even close" to showing they are effective in the management of patients with complex chronic pain.

"The research available is either unsupportive of using cannabinoid products in chronic non-cancer pain or is of such low quality that no valid scientific conclusion can be drawn," the pain specialist and physician said.

(Source: https://www.smh.com.au/national/medicinal-cannabis-blacklisted-by-australian-pain-specialists-20210322-p57cyw.html March 2021)

Cannabis Products Use in Pregnancy
(Appendix with Further extensive data)

> *"Medical marijuana is not safer than recreational marijuana."*
>
> (American College of Obstetricians & Gynaecologists)

Cannabis in Pregnancy – Rejoinder, Exposition and Cautionary Tales

Cannabis use during pregnancy is associated with a host of negative outcomes.

The recent paper by Stanciu discussing cannabis use in pregnancy[1] makes several useful and highly salient points. With a more complete understanding of the published literature further important patterns in the data emerge. They aid our understanding of the pathobiology of *in utero* cannabis exposure and thereby powerfully inform the community on the most appropriate manner in which to regulate cannabis and cannabinoids from an improved evidence base.

It is well known that cannabis use has been liberalized across the United States as a result of well-financed and orchestrated campaigns.[2] Stanciu is correct that most epidemiological studies point towards harmful associations, that cannabis use in pregnancy is becoming more common, that it is widely recommended in pregnancy by cannabis dispensaries, and that increased rates of low birth weight, premature and stillbirths, and increased neonatal intensive care admission are well recognized associations. It is correct that all 4 longitudinal studies of children born after prenatal cannabis exposure (PCE) show increased adverse neurodevelopmental outcomes including impaired executive function, visuomotor processing deficits, heightened startle responses, impulse control, heightened susceptibility to addiction in later life, emotional behaviors, and motor defects.[3-5]

Well-documented impacts on the glutamatergic, GABAergic and dopaminergic signaling in the brain are of concern as they represents major neurotransmitters in the central nervous system [CNS https://pubmed.ncbi.nlm.nih.gov/17162495/]. A large Hawaiian study found an increased incidence of microcephaly (R.R. = 12.80, 95%C.I. 4.13-36.17)[8] and the CDC have twice reported elevated rates of anencephalus (adjusted O.R. 1.7, C.I. 0.9-3.4) and (posterior O.R. 1.9 (C.I. 1.1, 3.2).[9,10] This sets up a clear spectrum of severity from mild neurodevelopmental impairment, to microcephaly, to anencephalus and then fetal death. In the context of

dose-response relationships and strong geotemporospatial associations issues of causality necessarily arise.

Stanciu's observation that preclinical studies in experimental animals are important to understand the likely effects of PCE in individuals, not least due to the problem of the frequent exposure to multiple substances clinically, is also correct. This issue was studied in detail long ago in the 1960s and 1970s, and succinctly summarized by Graham's telling observation: "oedema, phocomelia, omphalocoele, spina bifida, exencephaly, multiple malformations including myelocoele. This is a formidable list."[11]

However, a reasonable question might be: "Why don't we see such a broad teratological spectrum clinically?"

Stanciu's remark that there are "no overt birth defects" is an oft-repeated myth and is in error, as well as obviously being at odds with several preclinical studies, especially in the most predictive species for human teratology (ie, hamsters and white rabbits).[12,13]

A recent paper from the Centers for Disease Control (CDC) noted that 4 defects, anencephalus, gastroschisis, diaphragmatic hernia and esophageal atresia were more common following PCE.9 The American Academy of Pediatrics (AAP) and the American Heart Association (AHA) issued a joint position statement that both ventricular septal defect (VSD) and Ebsteins anomaly were also elevated by PCE.[14]

The review of 17 years of birth defects from Hawaii found 21 defects to be elevated after PCE and featured prominently cardiovascular defects (atrial septal defect (ASD), VSD, hypoplastic left heart syndrome, tetralogy of Fallot (ToF) and pulmonary valve atresia or stenosis), chromosomal defects such as Downs syndrome, body wall defects such as gastroschisis, limb defects including syndactyly and upper limb reduction defects, facial, bowel and genitourinary system defects with calculated rate ratios ranging from 5.26 (C.I. 1.08-15.46) to 39.98 (C.I. 9.03-122.29).[8]

In September and October 2018 Colorado released 2 datasets of congenital anomalies across the period of its cannabis legalization program from 2000 to 2013 and 2000 to 2014 and reported 87,772 and 64,463 major defects respectively (which are obviously contradictory).[15] Based on 4830 and 4026 major anomalies in the year 2000 this represents a case excess of 20,152 (29.80%) or 11,753 anomalies (22.30%) respectively. During this period the use of tobacco and alcohol was declining and other drug use was not rising. Only cannabis use rose. Importantly, models quartic in time indicated a non-linear response of total birth defects to rising cannabinoid exposure. Estimated exposure to several cannabinoids including cannabinol, THC, and tetrahydrocannabivarin was shown to be positively associated with major defect rates and to be robust to adjustment for other drug use. CNS defects (microcephalus, neural tube defects), cardiovascular defects (ASD, VSD, patent ductus arteriosus (PDA)), total chromosomal anomalies including Downs syndrome, musculoskeletal, respiratory and genitourinary anomalies all rose dramatically.

Defects described as being cannabis-related (by the Hawaiian, CDC, AAP and AHA investigators) rose more quickly than cannabis-unrelated defects ($P<0.003$). As fetal cardiac tissue and the central great vessels have high numbers of cannabinoid receptors from early in fetal life it is easy to understand why this pattern might emerge. Since ASD, VSD and PDA are the most common cardiovascular congenital anomalies it is understandable that total cardiovascular anomalies increased in Colorado.

A recent review of total congenital anomalies in Canada showed that they were 3 times more common in the northern territories which consume more cannabis, and that these effects were robust to adjustment for other drug exposure and for socioeconomic variables.[16] Total cardiovascular defects, Downs syndrome and gastroschisis were noted prominently in this series. Neural tube defects including anencephalus and spinal bifida and meningomyelocoele were falling

across Canada from 1991 to 2007, although it was not clear whether the decline was due to dietary folate supplementation or increased antenatal early termination of pregnancy for anomalies (ETOPFA).[17] Notwithstanding this it was recently shown that within each of 3 periods (the pre-folate period, the transitional period and the post-folate period) neural tube defects across Canada were becoming more common.[17]

An Australian dataset found greatly elevated relative rates of cardiovascular (PDA, ASD, VSD, ToF, transposition of great vessels), body wall (gastroschisis, exomphalos, diaphragmatic hernia), chromosomal (Downs syndrome, Turners syndrome, Edwards Syndrome (trisomy 18)), genitourinary, hydrocephalus, neural tube defects, and bowel defects with borderline results for anencephalus (ETOPFA data unavailable) in a high cannabis use area in Northern New South Wales compared to Queensland state-wide data.[18]

Transposition of the great vessels was previously linked with paternal cannabis exposure.[19]

The presence of Downs syndrome on the list of cannabis-associated anomalies in Hawaii, Colorado, Canada and Australia is important as it necessarily implies megabase-scale genetic damage.[8,15,16,18] Since cannabis interferes with tubulin metabolism and thus the separation of the chromosomes which occurs in mitotic anaphase it is easy to see how PCE-induced chromosomal mis-segregation errors might occur.[20] Studies of PCE in rodents show that cannabis induces major alterations of gene expression widely with 8% alteration in DNA sperm methylation patterns, changes which are transmissible to subsequent F1 generations.[21]

Stanciu's comment about a so-called "cannabis phenotype" is provocative. It is true that a "fetal cannabis syndrome" (FCS) has not been described in the way that a "fetal alcohol syndrome" (FAS) has. Fetal alcohol syndrome of course is a very diverse and pleomorphic group of clinical presentations and a wide spectrum of presentations is described. Importantly the fetal alcohol has been described as being mediated by the cannabinoid type 1 receptor (CB1R's) and is mediated epigenetically.[22-26] The suggestion that alcohol can work epigenetically via CB1Rs but cannabinoids cannot defies the bounds of credulity. Moreover, as noted above, there is as yet no objective marker of gestational cannabinoid exposure. Once such a biomarker has been derived (say epigenetically and / or glycomically27) then an objective measure will exist to allow genotype-epigenotype-phenotype correlative studies to be performed so that we can usefully investigate if a fetal cannabis syndrome phenotype spectrum might exist. However, if researchers do not believe it might exist then it is clear that one will not be described. It is our view that once an objective biomarker is established it will only be a matter of time before a diverse and highly variable FCS is also defined and enters the clinical diagnostic compendium.

(Source: Cannabis in Pregnancy – Rejoinder, Exposition and Cautionary Tales - Psychiatric Times October 10, 2020)

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Cannabis in Cancer

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Cannabis in Cancer

The genotoxicity of Cannabis has long been suspected, even acknowledged, be it only in part. Research over the last 5 to 10 years has confirmed the case. Much of this important research has been 'buried' in the deluge of 'hopeful' and even spectacular claims of the potential therapeutic capacity of cannabis. Claims and promises that have persisted for well over 20 years, yet with little to nothing to show for it. However, the harms associated with the use of this now heavily engineered plant/product are mounting, and the research is not only monitoring, but discovering these harms. If science and health matter, then all research must be thorough and properly vetted to ensure that health is advanced, not mere 'symptom abated' whilst disease, disorder or other harms grow.

Whilst it is obvious that low birth weight has been noted by many papers looking at the effects of cannabis in pregnancy a much more serious pattern is also emerging which has been replicated now in five jurisdictions namely Hawaii [1], Colorado [2], Canada [3], Australia [4] and USA [5-7].

In fact, in 6 it was shown that cannabinoids are genotoxic for at least 20% of the human genome by way of chromosomal toxicity. Moreover cannabis has been shown to inhibit sonic hedgehog signalling by several mechanisms [8] which has profound implications for foetal development as sonic hedgehog is one of the most important human embryonic morphogens of all [9]. Sonic hedgehog inhibition alone both implies and accounts for elevated rates of the numerous birth defects in which prenatal cannabis exposure is now implicated.

Cannabinoids also have a heavy epigenetic footprint. This has serious and multi-generational impacts. Moreover, cannabinoids have also been shown to inhibit mitochondrial metabolism by many means including direct inhibition through a full complement of endocannabinoid signalling machinery held on their inner and outer mitochondrial membranes and in the intermembrane space. Both the epigenomic and metabolic effects of cannabinoids are critical and are also closely related as metabolic state controls epigenetic state both directly through substrate supply and indirectly through small molecular signalling shuttles which have the effect of coordinating nuclear and mitochondrial genomic expression and signalling mitonuclear stress[10]. That is to say that metabolic state and epigenomic state – and hence multigenerational inheritance – are closely and intimately related.

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Cancer Causing, Not Preventing

Association Between Marijuana Use and Risk of Cancer: *A Systematic Review and Meta-analysis*

Question:
What is the association between marijuana use and cancer development in adults with at least 1 joint-year exposure (equivalent to 1 joint per day for 1 year)? Findings This systematic review and meta-analysis identified 25 English language studies assessing marijuana use and the risk for developing lung, head and neck, urogenital, and other cancers. In meta-analyses, regular marijuana use was associated with development of testicular germ cell tumors, although the strength of evidence was low; evidence regarding other cancers was insufficient.

Meaning Sustained marijuana use may increase the risk for testicular cancer, but overall, the association of marijuana use and cancer development remains unclear.

Conclusions & Relevance:
Low-strength evidence suggests that smoking marijuana is associated with developing TGCT; its association with other cancers and the consequences of higher levels of use are unclear. Long-term studies in marijuana-only smokers would improve understanding of marijuana's association with lung, oral, and other cancers.

(Source: JAMA Network Open. 2019;2(11): doi:10.1001/jamanetworkopen.2019.16318)

Epidemiological Overview of Multidimensional Chromosomal and Genome Toxicity of Cannabis Exposure in Congenital Anomalies and Cancer Development

Cannabis and cannabinoids are implicated in multiple genotoxic, epigenotoxic and chromosomal-toxic mechanisms and interact with several morphogenic pathways, likely underpinning previous reports of links between cannabis and congenital anomalies and heritable tumours. However the effects of cannabinoid genotoxicity have not been assessed on whole populations and formal consideration of effects as a broadly acting genotoxin remain unexplored.

Our study addressed these knowledge gaps in USA datasets. Cancer data from CDC, drug exposure data from National Survey of Drug Use and Health 2003–2017 and congenital anomaly data from National Birth Defects Prevention Network were used. We show that cannabis, THC cannabigerol and cannabichromene exposure fulfill causal criteria towards first Principal Components of both: (A) Down syndrome, Trisomies 18 and 13, Turner syndrome, Deletion 22q11.2, and (B) thyroid, liver, breast and pancreatic cancers and acute myeloid leukaemia, have mostly medium to large effect sizes, are robust to adjustment for ethnicity, other drugs and income in inverse probability-weighted models, show prominent non-linear effects, have 55/56 e-Values>1.25, and are exacerbated by cannabis liberalization ($P = 9.67 \times 10–43$, $2.66 \times 10–15$).

The results confirm experimental studies showing that cannabinoids are an important cause of community-wide genotoxicity impacting both birth defect and cancer epidemiology at the chromosomal hundred-megabase level.

(Source: Reece A.S., Hulse G.K. Scientific Reports. 2021;11(1):13892).

Cannabinoid exposure as a major driver of pediatric acute lymphoid Leukaemia rates across the USA: combined geospatial, multiple imputation and causal inference study.

Acute lymphoid leukaemia (ALL) is the commonest childhood cancer whose incidence is rising in many nations. In the USA, between 1975 and 2016, ALL rates (ALLRs) rose 93.51% from 1.91 to 3.70/100,000< 20 years. ALL is more common in Caucasian-Americans than amongst minorities. The cause of both the rise and the ethnic differential is unclear, however, prenatal cannabis exposure was previously linked with elevated childhood leukaemia rates. We investigated epidemiologically if cannabis use impacted nationally on ALLRs, its ethnic effects, and if the relationship was causal.

Methods:

State data on overall, and ethnic ALLR from the Surveillance Epidemiology and End Results databank of the Centre for Disease Control (CDC) and National Cancer Institute (NCI) were combined with drug (cigarettes, alcoholism, cannabis, analgesics, cocaine) use data from the National Survey of Drug Use and Health; 74.1% response rate. Income and ethnicity data was from the US Census bureau. Cannabinoid concentration was from the Drug Enforcement Agency Data. Data was analyzed in R by robust and spatiotemporal regression.

Conclusions:

Data show that ALLR is associated with cannabis consumption across space-time, is associated with the cannabinoids, THC, cannabigerol, cannabinol, cannabichromene, and cannabidiol, contributes to ethnic differentials, demonstrates prominent quintile effects, satisfies criteria for causality and is exacerbated by cannabis legalization.

(Source: Reece A. S., Hulse G.K. BMC Cancer. 2021;21(1):684).

Causal inference multiple imputation investigation of the impact of cannabinoids and other substances on ethnic differentials in US testicular cancer incidence.

Ethnic differences in testicular cancer rates (TCRs) are recognized internationally. Cannabis is a known risk factor for testicular cancer (TC) in multiple studies with dose-response effects demonstrated, however the interaction between ancestral and environmental mutagenic effects has not been characterized. We examined the effects of this presumed gene-environment interaction across US states.

Methods:

State based TCR was downloaded from the Surveillance Epidemiology and End Results (SEER) website via SEERStat. Drug use data for cigarettes, alcohol use disorder, analgesics, cannabis and cocaine was taken from the National Survey of Drug Use and Health a nationally representative study conducted annually by the Substance Abuse and Mental Health Services Administration (SAMHSA) with a 74.1% response rate. Cannabinoid concentrations derived from Drug Enforcement Agency publications. Median household income and ethnicity data (Caucasian-American, African-American, Hispanic-American, Asian-American, American-Indian-Alaska-Native-American, Native-Hawaiian-Pacific-Islander-American) was from the US Census Bureau. Data were processed in R using instrumental regression, causal inference and multiple imputation.

Conclusion:

Cannabis is shown to be a TC risk factor for all ethnicities including Caucasian-American and African-American ancestries, albeit at different rates. For both ancestries cannabis legalization elevated TCR. Dose-response and causal relationships are demonstrated.

(Source: Reece AS, Hulse GK. BMC Pharmacol Toxicol. 2021;22(1):40).

Therapeutic applications? Very Limited.

There has been even less 'advancement' in potential since the 'panacea' mantras that started aggressively promoting this plants potential over 30 years ago. Science is not 'catching up', is proving what is not there.

Very limited therapeutic application only for limited and temporary alleviation of some neuropathic pain in cancer patients and the increase of appetite in cancer patients going through chemotherapy. These cannabis pharmaceuticals have been on the Australian PBS for over 20 years. New and 'stronger' iterations of cannabis with high THC quotients add nothing more to these therapeutic applications.

Appendix

- **Cancer-causing (carcinogenic) substances in cannabis:** *Cannabis smoke has a higher concentration of certain cancer-causing (carcinogenic) agents than the smoke from tobacco. Evidence suggests that cannabis may cause cancers of the lung and the aerodigestive tract (which includes the respiratory tract and the upper digestive tract) Better Health Victoria.*
- **Cannabis and Cannabinoids (PDQ®)–Health Professional Version** - National Cancer Institute
- **Is marijuana use associated with a higher risk of cancer?** (medicalnewstoday.com)
- **Dalgarno Institute Cannabis Library – Genetic Impact**

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Hemp Industry & Policy in Legalized Cannabis Jurisdictions

Hemp Industry & Policy in Legalized Cannabis Jurisdictions

Court rules CBD is Schedule 1 controlled substance, cannabidiol sales only where Pot legal. (USA)

On Thursday, Feb. 1, 2018, the Cosmic Grind Coffee Shop on Church Street in Burlington, Vermont, started offering CBD hemp oil shots in their drinks. But don't worry, CBD is a 'non-psychoactive' extract, not to be confused with THC in marijuana. RYAN MERCER/FREE PRESS

DENVER (AP) — A federal appeals court sided with the Drug Enforcement Administration and upheld its decision that CBD is a Schedule 1 controlled substance — a major setback for the American hemp industry.

The decision, issued Monday by a three-judge panel of the 9th Circuit in San Francisco, means that hemp producers can only sell cannabidiol where it is allowed under state law.

It also means that states that allow CBD — even if they don't allow the sale of high-THC marijuana - are violating federal law, the same as states that allow recreational cannabis.

CBD producers who brought the case vowed to appeal

"We will be appealing, and we will be funding that appeal," said Michael Brubeck, CEO of Centuria Natural Foods and a plaintiff in the case.

Based in Las Vegas, Centuria grows hemp and produces CBD products for sale in all 50 states. Centuria was joined in its challenge by the Hemp Industries Association.

CBD case history

The case started in 2016, when the DEA issued a "clarifying rule" stating that CBD is an illegal drug, because it is extracted from marijuana flowers.

Hemp producers cried foul, arguing that CBD can also be extracted from legal hemp flowers, and there is no way to tell whether extracted CBD came from marijuana or from hemp.

Brubeck and the HIA argued that the DEA was attempting to add a new substance to the Controlled Substances Act, something it cannot do.

The DEA said the extract rule was simply a clarification of existing law and that it "makes no substantive change to the government's control of any substance."

The agency also scoffed at the suggestion that CBD is being made from anything but flowering parts of the cannabis plant because cannabinoids "are found in the parts of the cannabis plant that fall within the definition of marijuana, such as the flowering tops, resin and leaves."

The three-judge panel of the 9th Circuit agreed. Their decision means that the DEA was within its authority to clarify CBD as a "marijuana extract."

(Source: https://www.dalgarnoinstitute.org.au/resources/cannabis-as-medicine/517-court-rules-cbd-is-schedule-1-controlled-substance-cannabidiol-sales-only-where-pot-legal.html Hemp Industry Daily May 4, 2018)

Cannabis, Hemp, THC in the Food-Cosmetic Supply

Fiber Cannabis hemp seed, though containing tetrahydrocannabinol (THC, the main psychoactive ingredient in hemp/marijuana) and other cannabinoid residue, is being heavily marketed and promoted by the hemp industry as a source of food, nutraceuticals, and cosmetics. The harmful effects of THC on humans and other animals is well documented. Hemp advocates, however, mimicking the tactics of tobacco industry apologist, challenge and "call into question" every statement substantiating harm caused by the use of Cannabis sativa L. hemp. (Where used in this paper, the term hemp refers to cannabis sativa, aka marijuana, and not to any of the numerous other plant fibers also commonly referred to as hemp.)

The campaign to use hemp fiber for paper, biomass, textiles, etc. has largely failed because hemp is neither economically viable nor technically feasible. However, because the handling, storage, and processing of hemp seed is more adaptable to present technologies than for hemp fiber, hemp seed production and products are now being aggressively promoted.

Low THC Cannabis sativa hemp that contains less than .3% (w/w) THC became legal to grow in Canada in March, 1998. THC and the other cannabinoids are found in food and other products made from fiber hemp seed. According to Canada's national health department, Health Canada, "In theory the ripened seeds of Cannabis contain no detectable quantity of THC. However, because of the nature of the material it is almost impossible to obtain the seeds free from extraneous THC in the form of residues arising from other parts of the plant which are in close proximity to the seeds. Although it is required for the seeds to be cleaned before any subsequent use, the resinous nature of some of the material makes complete cleaning extremely difficult."[1]

Since THC and the over 60 other cannabinoids are fat-soluble, i.e., store themselves in the fatty tissues of the brain and body, even a very small amount may be damaging, especially if ingested regularly. Fat-soluble substances accumulate in the body.

THC has a half-life of about seven days, meaning that one-half of the THC ingested or inhaled stays in the brain and body tissue for seven days. Traces can stay in body tissues for a month or more. The only important substance that exceeds THC in fat solubility is DDT.[2]

A risk assessment done for Health Canada states that, "New food products and cosmetics made from hemp – the marijuana plant – pose an unacceptable risk to the health of consumers. It also says that hemp products may not be safe because even small amounts of THC may cause developmental problems. "Those most at risk," the study says, "are children exposed in the womb or through breast milk, or teenagers whose reproductive systems are developing."[3]

"Hazards associated with exposure to THC include acute neurological effects and long-term effects on brain development, the reproductive system and the immune system," the study says. "Overall, the data considered for this assessment support the conclusions that inadequate margins of safety exist between potential exposure and adverse effect levels for cannabinoids (the bio-active ingredients) in cosmetics, food and nutraceutical products made from hemp."[3]

The study reviewed the results of existing tests on lab animals. Health Canada may require warning labels or new regulations that could stop some products from being sold. It is considering new animal studies to examine the effects of low-level exposure to THC over several generations.[3]

To cast further doubt about safety, the Journal of Immunology (July 2000) recently reported that THC, the major psychoactive component of marijuana (hemp), "can promote tumor

growth by impairing the body's anti-tumor immunity system."[4]

Another unknown is hemp as forage for animals. According to Stan Blade, a director of crop diversification for Alberta Agriculture, a program that will test hemp over the next year as feed for livestock is being considered in Canada. Forage hemp will be tested on cattle against a more traditional mixture of oats and barley.[5]

Buffalo, the common dairy animal of Pakistan, are allowed to graze on Cannabis sativa (hemp), which, after absorption, is metabolized into a number of psychoactive agents. These agents are ultimately excreted through the urine and milk, making the milk, used by the people of the region, subject to contamination. Depending on the amount of milk ingested and the degree of contamination, the milk could result in a low to moderate level of chronic exposure to THC and other metabolites, especially among the children raised on this milk. Analysis from the urine obtained from children who were being raised on the milk from these animals, indicated that 29% of them had low levels of THC-COOH (THC-carboxylixc acid, which is a major metabolite for THC) in their urine. This study indicates that the passive consumption of marijuana through milk products is a serious problem in this region where wild marijuana grows unrestricted, and that children are likely to be exposed more than adults."[6]

Hemp use could compromise drug testing. In his book, "Fats that Heal, Fats that Kill," Udo Erasmus warns that people whose jobs require mandatory drug screening should avoid the use of hemp products, since THC residues in hemp products can show up in urine tests.[7] THC-positive urine tests from hemp product use were also reported in the August 1997 Journal of Analytical Toxicology.[8] For drug-testing reasons, the U.S. Air Force, the Air Force National Guard, the New York Police Dept., and the U.S. Coast Guard have banned the use of hemp foods and health supplements by their personnel.[8&9]

"Dr. Hugh Davis, Acting Head of Microbiology and Cosmetics at Health Canada, is quoted as saying that he has been looking at studies on hemp and has found research showing hemp (i.e., fat soluble cannabinoids) is accumulative in the body because of its long half-life and has the same adverse physiological (but not hallucinatory) effects that smoking marijuana does. One study states that cannabinoids may postpone puberty. There are 60 known cannabinoids, only three of which have been widely studied. This means that the potential harmful aspects of the remaining 57 cannabinoids, when used in a cream or shampoo, are unknown."[10]

John Bailey, Microbiology and Cosmetics Division, US-FDA, (US-Federal Drug Administration) is concerned as well, stating that there is no definitive information about THC in food and cosmetics.[10]

Dr. Mohmoud ElSohly, Ph.D., Marijuana Project Director, NIDA (National Institute of Drug Abuse), states that "Fiber hemp can have significant potential for narcotic application.... The threshold THC concentration (below which Cannabis would have no significant psychoactive properties) has not been determined."[11] [Emphasis added] Dr. Roy H. Hart, Clinical Psychiatrist and research chemist (ret.), asserts that it is possible to experience chronic intoxication without being high.[12]

In addition to THC, there are other bioactive, but nonpsychoactive, cannabinoids [cannabinol (CBN), cannabidiol (CBD), and cannabigerol (CG)] in Cannabis sativa marijuana(hemp).[13] David West, Ph.D., pro-hemp activist (HI), claims that CBD blocks the effects of THC in the nervous system.[14] However, Dr. Carlton Turner, Director of the Federal NIDA Marijuana Project (1970-1981) and former US Drug Czar (1980s) counters that "CBD is abundant in hashish and if CBD blocked THC's action, why would hashish be so popular? I know of no known definitive study that shows that CBD blocks THC's affects. Fiber cannabis is rich in CBD with little THC. However, naive users can sometimes get high but regular users will not."[15]

The nonpsychoactive cannabinoids may be even more toxic than THC. According to Dr. Roy Hart, "Cannabidiol (CBD) exerts an important effect on the hippocampus which is part of the limbic system of the brain, a collection of interfunctioning units concerned with emotion. CBD produces a depression of hippocampal function...Thus far experimental evidence indicates that CBD is even more toxic to tissues than THC."[16] [Emphasis added] Dr. Gabriel Nahas, Research Professor, New York University, states that cannabionids other than THC (CBN and CBD) also impair dividing cells, and "are even more potent than THC when it comes to inhibiting DNA production."[17]

Dr. Hart further states that "Both the psychoactive and nonpsychoactive cannabinoids occurring in nature interfere with protein synthesis, deoxyribonucleic acid (DNA) synthesis, and ribonucleic acid (RNA) synthesis. This is without doubt the most important statement to be made about marijuana (hemp) and is based upon the burgeoning literature of basic and applied research into cannabis. Cell-tissue-organ damage follows inevitably from these alternations occurring at the molecular level."[18]

Longtime and internationally renowned Cannabis researcher, Dr. Gabriel Nahas says that research has shown that the most serious adverse consequences of consumption of THC and other cannabinoids have been observed at the earliest state of reproductive function, on the "gametes" or germ cells of man. These drugs cause damage to the genetic information contained in DNA, causing apoptosis (programmed cell death and deletion). This threatens future generations before they are conceived.[19]

A 1996 study conducted in the Ukraine (formerly Russia) showed that there are no varieties that completely lack(ed) cannabinoids. A rather high content of these substances (cannabinoids) was found in some varieties. The results obtained have shown that hemp cultivated in more northerly areas is naturally rich in cannabinoids.[20]

European Union (EU) hemp regulations for the year 2000 state that hemp subsidies will be paid on condition the farmer uses certified seed of hemp varieties with a THC content of less than 0.3%. From the years 2001/02, that upper limit will be lowered to 0.2%.[21]

The European Union (EU) too is concerned about any inclusion of hemp products' in food, stating in their regulations, "...Hemp seed has one traditional but limited application as food for fish and birds. The oil from hemp seed can be used for specialist cosmetics applications. The use of hemp seed or the leafed parts of the plant for human consumption would, however, even in the absence of THC, contribute towards making the narcotic use of cannabis acceptable and, in any event, there is no nutritional justification for this. [Emphasis added] None of these products should be encouraged in their own right by Community aid....Moreover, the International Narcotics Control Board (INCB, a United Nations body) states that: 'while illicit cannabis cultivation (sic) have soared, a considerable market for food products and beverages produced with cannabis has developed in the European Union (...). The health effects of these products have not been adequately researched.'(...) [Emphasis added] The wide and unrestricted availability of such products in shops, where cannabis candy bars can be sold to minors without restriction, contribute to the overall benign image of cannabis, a drug under international control." [OICS note of 12.3.1999.][21]

"It is therefore important to remain vigilant and step up controls to ensure that illegal crops do not tarnish the reputation of the sector producing hemp for fibre. To avert such dangers, the cultivation of hemp for fibre must be strictly controlled, which means the area cultivated will have to be restricted, and the uses to which it is put must NOT include human nutrition." [Emphasis added] These EU regulations apply from July 1, 2000. [21]

The findings of the previously mentioned Health Canada THC Assessment are quite alarming from a consumer health and safety

standpoint. Two key areas of health hazards to humans were reviewed, and the potential for risks from consumption of hemp products was characterized.[22]

One health area was neuroendocrine disruption during developmental states (perinatal, prepubertal and pubertal) that leads to permanent adverse effects on the brain and reproductive systems. The second area was neurological impairment manifested as deficits in cognitive and motor skills' performance.[22]

The study could not, due to data gaps, develop definitive conclusions regarding the degree of potential risk from ingesting THC through hemp products. However, even without considering the bioaccumulative hazard potential of THC through repeated or multiple-product use, or the risk from chemicals other than THC in Cannabis sativa hemp, it nevertheless came to the following conclusions:

Characterizations of Risks from THC in Hemp Products for Human Use & Consumption

Health Canada Study (DRAFT of November 23, 1999)

Health Risk/ Product	*Food*	*Cosmetics*	*Nutraceuticals*
Risk Of Neuroendocrine Disruption *	*Likely*	*Possible*	*Likely*
Risk Of Neurologicalimpairment Andpsychoactivity	*Likely, particularly for children (Also Risk of psychoactivity for children)*	*Unlikely, though cannot be excluded entirely due to limitations of study*	*Possible, particularly in children.*

**Developing fetus, nursing infant, and prepubertal/pubertal child are at greatest risk of long-term effects. THC is rapidly transferred from mother to fetus within minutes of exposure. THC accumulates and is transferred via breast-milk.[22]*

The in-depth Health Canada Risk Assessment on THC and Other Cannabinoids (in products) Made with Industrial Hemp (11/23/99) warns "On the basis of currently available data it is concluded that the present Canadian limit of 10ug/g (i.e.,10 ppm) THC in raw materials and products made from industrial hemp (Cannabis sativa cultivars with less than 0.3% THC) would likely not protect the Canadian consumer using industrial hemp-based food, cosmetic and personal care, and nutraceutical products from potential health risks of neurological impairment and neuroendocrine disruption associated with low level exposure to THC and other cannabinoids."[22]

In the United States even salad oils must be examined and certified by the US-FDA as "generally recognized as safe." This has not been done for hemp.

Allowing or introducing toxic chemicals in our food and cosmetic systems through use of THC-containing industrial hemp products is unthinkable. To do so would jeopardize public health and safety. U.S. citizens and government agencies and officials should do everything possible to prevent this from happening, thus protecting future generations from both known and unknown health and genetic hazards.

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(Source: http://www.drugwatch.org/resources/publications/articles/161-cannabis-hemp-thc-in-the-food-cosmetic-supply.html Hemp Committee, Drug Watch Intl. 2000**)**

Italy & Sweden's Supreme Court says CBD oil containing THC is a narcotic preparation

On 18 June 2019, the Supreme Court of Sweden ruled on a case involving possession of 'CBD oil' extracted from industrial hemp. Under Swedish law industrial hemp, defined as any variety of cannabis eligible for EU support, is exempt from the narcotic control laws. However, the oil contained THC (the concentration was not determined). THC and preparations containing it are covered by the narcotic control laws. The offender was charged with a minor case of possession of a controlled drug (a preparation of THC). The court ruled that, while industrial hemp is exempted from coverage, preparations made from it that contain THC are not exempted, and are therefore included in narcotic control laws.

(Source https://www.emcdda.europa.eu/news/2019/italy-and-sweden-court-decisions-low-thc-cannabis-products_en 20 July 2019)

Is your beef 'Grass Fed' or 'Weed Fed'? Industrial Hemp in Food Chain

Harmless Hemp and Passive Toxicity – Not New, but a Growing Concern.

Not unsurprisingly, the Cannabis Industry creates many and varied 'contaminants' to the environment, community and humanity itself, but it also creates its very own irony in the contaminant context.

As far back as 2015, concerns were being raised about cannabis contaminants, but not in perhaps the way we view it now.

In Southern Oregon (USA) marijuana growers wanted to ban industrial hemp production from the region out of fear that hemp may pollinate their cannabis crops and render them worthless!

> *"Allowing industrial hemp in an area known for churning out high-grade marijuana could undermine the industry", growers argue…"It basically makes the medicine worthless,"*

Hmmm, isn't Cannabidiol (CBD) and other Cannabinoids that are supposed to be the 'medicine', not really Delta 9 THC? CBD is seemingly not impacted by Hemp cross-pollination, only the 'recreational quality' product, so why the hysteria? Ah, the cannabis logic is confusing.

The zeal for the addiction for profit sector of the Marijuana market, engendered a paranoia that cross pollination with the all but zero THC content hemp, will weaken and thus render uncommercial their 'recreational' product, which they referred to as 'medicine'.

However, no concerns were being raised back then that the reverse may be true.

In a paper published as far back as August 2000 research-based warnings were already being issued about this blurring of the lines with Hemp and other Cannabis strains. The following excerpt from ***Cannabis, Hemp, THC in the Food-Cosmetic Supply*** gives some insight,

Another unknown is hemp as forage for animals. According to Stan Blade, a director of crop diversification for Alberta Agriculture, a program that will test hemp over the next year as feed for livestock is being considered in Canada. Forage hemp will be tested on cattle against a more traditional mixture of oats and barley.

Buffalo, the common dairy animal of Pakistan, are allowed to graze on Cannabis sativa (hemp), which, after absorption, is metabolized into a number of psychoactive agents. These agents are ultimately excreted through the urine and milk, making the milk, used by the people of the region, subject to contamination. Depending on the amount of milk ingested and the degree of contamination, the milk could result in a low to moderate level of chronic exposure to THC and other metabolites, especially among the children raised on this milk. Analysis from the urine obtained from children who were being raised on the milk from these animals, indicated that 29% of them had low levels of THC-COOH (THC-carboxylixc acid, which is a major metabolite for THC) in their urine. This study indicates that the passive consumption of marijuana through milk products is a serious problem in this region where wild marijuana grows unrestricted, and that children are likely to be exposed more than adults."

The legal requirement for 9-tetrahydrocannabinol (9-THC) content of HEMP is supposed to be 0.3 percent or less, yet from both anecdotal evidence and an ever-decreasing quality control management, one will find it difficult to ensure even basic health and safety issues are monitored, let alone acted upon.

One such issue and now after thought, is that the hemp industry has a lot of waste bi-product and finding ways to deal with it, apart from burning it (and all the attending concerns around that) is determining its suitability as fodder.

In 2013 a Washington State pig farmer thought he would experiment with his hobby hogs and see if Pot waste would change the *flavour* of his pork products. Thinking as many in the industry do now, that the waste from cannabis grows must be useful, he tried the experiment. Anecdotally, it was a 'success', his retailers declaring it better tasting.

However, John P. McNamara, a professor at Washington State University's Department of Animal Sciences, did not find the experiment amusing, nor should he.

"Of all the crazy things I've seen in my 37-plus years, this is the dumbest things I've ever seen in my life," McNamara said in order to introduce a drug or medicine to feed, that's being given to animals that make part of the food supply, the federal government must sign off on it after extensive review. He adds that research has shown that cannabis ingested can be transferred onto tissues.

What of the pigs? Well, according to the producer, no real difference as *'pigs just eat and sleep anyway'*, though the manager noticed one of the more salty sows was calmer after feeding...Hmmm? Again, all anecdote, no data – yet that seems to be a key driver for policy making around this increasingly complex and far from benign product.

In a, Hemp advocate Hunter Buffington was interviewed on this complex Hemp issue.

The interview revealed some of those complexities and the current attempts to (if not overcome) negate them. The interview confirmed the real need to ensure not only any feed potential of this substantial and growing bio-waste, but also determine any contamination *of it*, or *in it*. The imperative of ensuring that what is 'fed into' the human food chain is safe should not be understated, but it may well be if pro-cannabis advocates are in-charge of the scrutiny process. Any potential toxicity acquired by the growing environment, (i.e. soils, horticultural practices and/or pesticides) or from the plants own innate compound toxicities, need to be understood and guarded against.

Alongside these stringent safety protocols, clinical feed trials must also be conducted of the by-products being offered as fodder. Each product must be tested against each of the animal breeds it is going to be fed to, ensuring no further harms are done to the animal being fed, or to those further up the food chain.

Kansas State University are undertaking some studies to that end, with the following outcomes reported in part in the following,

> *While there is interest in the use of hemp for cattle feeds, there are questions about whether the feed can be used safely because of concerns about tetrahydrocannabinol, or THC, intoxication and the presence of other bioactive cannabinoids. Kleinhenz noticed that most research was focused on humans, mice and swine, but not on cattle.*
>
> *"This is surprising because cattle can readily utilize industrial hemp byproducts as they can digest cellulose plant materials in their rumens," Kleinhenz said.*
>
> *"We observed that the acidic cannabinoids, such as CBDA and THCA, are more readily absorbed from the rumen than other non-acid cannabinoid forms, such as CBD and CBG," Kleinhenz said. "Now that we have found that some cannabinoids are readily absorbed from the rumen, the next steps are to study the tissue and milk residue udepletion profiles of these compounds after animal feeding experiments. The effects of cannabinoids on cattle are also unknown."*
>
> KSU News & Communication Services

Whilst Kansas State University were conducting their review another long promised study on the use of Hemp crop residue (an environmental concern in its own right) revealed what had been suspected 20 years earlier.

Published in Nature – Scientific Reports Plasma concentrations of eleven cannabinoids in cattle following oral administration of industrial hemp (Cannabis sativa) (nih.gov) uncovered the following,

From the "Discussion" section,

- *Moreover, the impact of the rumen on the fate of oral cannabinoids requires further investigation. Rumen microbes could potentially degrade or metabolize cannabinoids causing alterations in the cannabinoids available for absorption. Merrick et al., reported the in vitro conversion of cannabidiol (CBD) to 9-tetrahydrocannabinol (9-THC) in simulated gastric fluid. Although these findings were not supported in vivo; there is still potential for rumen microbes to play a significant role in the conversion of fatty-acids through biohydrogenation.*

- *The results of the finding of this study have implications for IH (Industrial Hemp) as an agriculture commodity. In the short-term, these findings can be used to develop strategies for cattle accidently exposed to IH and hemp by-products, as the U.S. Food and Drug Administration (FDA) has explicitly stated cannabinoids are considered adulterants in food production species. However, cattle and other ruminants are ideally suited to utilize IH and the byproducts of cannabinoid production from IH as a novel source of nutrition.*
- *Understanding of plasma half-lives for cannabinoids will allow veterinarians to work with cattle producers to establish withdrawal intervals to ensure exposed cattle can enter the food supply.*
- *Additionally, understanding of cannabinoid pharmacology is needed if IH and hemp byproducts are to be considered by the US FDA and the Association of American Feed Control Officials (AAFCO) for inclusion into animal diet.*

On interrogating this research, one commentator also made the following remarks, which bear further consideration.

> *And of course, they need more research to answer the additional questions that they bring up. They now need to "understand plasma half-lives for cannabinoids" and need an "understanding of cannabinoid pharmacology".*
>
> *The establishment of a "withdrawal" period is what I find really interesting and would be a challenge. Basically, the cows would need a drug test before they can be sent to slaughter. If such a protocol was established, I think that we all know what could happen; falsification of test results is a strong possibility because it is basically about the money, which always depends on speed to market.*
>
> *What I also find especially troubling is that in the discussion session, the authors state that their findings can be used to develop strategies* ***for cattle accidently exposed*** *to IH and hemp by-products.*

The need to monitor this industry and the management of its growing waste products is imperative if the health and well-being of people, as well as animals, is a priority. The rush to market of cannabis by 'voting for medicine' initiatives is a staggering backward step for a so-called evidence-based scientifically anchored culture.

If indeed, this bio-waste can be proven utterly safe and beneficial as animal feed, with absolutely no potential harms permitted, this may be one positive for an industry that has proven in past decades that salesmanship trumps science every time.

More thorough and robust research is needed at all levels and the tightening of regulations around 'supplements', of which there is currently little to none in the United States, Australia, and other lax jurisdictions. The time for 'free pass' on these untrailed or clinically untested products must end for the sake of community and animal well-being.

(Source: https://www.dalgarnoinstitute.org.au/resources/next-phase-blog/1501-is-your-beef-grass-fed-or-weed-fed-industrial-hemp-in-food-chain.html Dalgarno Institute Research 2021)

Appendix

Cannabis sativa (Hemp) Seeds, Δ9-Tetrahydrocannabinol, and Potential Overdose

We discovered that D9-THC concentrations in these hemp seeds could be as high as 1250% of the legal limit, and the amount of phytocannabinoids depended on the extraction procedure employed, Soxhlet extraction being the most efficient across all three brands of seeds. D9-THC and CBD exhibited significant variations in their estimated concentrations even from the same brand, reflecting the inhomogeneous nature of seeds and variability due to the extraction method, but almost in all cases, D9-THC concentrations were higher than the legal limit. These quantities of total D9-THC may reach as high as 3.8mg per gram of hemp seeds, if one were consuming a 30-g daily recommended amount of hemp seeds, and is a cause for concern for potential toxicity. It is not clear if these high quantities of D9-THC are due to contamination of the seeds, or any other reason.

Conclusion:

Careful consideration of the extraction method is very important for the measurement of cannabinoids in hemp seeds.

(Source: Cannabis and Cannabinoid Research Volume 2.1, 2017)

Drug Watch International OPPOSEs Legalization of Industrial Hemp

Published: 30 October 2013

WE, US drug preventionists, OPPOSE the legalization of industrial hemp (low-grade marijuana) as an agricultural crop; RESIST all efforts to change Federal or state laws to allow industrial hemp to be defined as a legal crop; ACKNOWLEDGE that the US government is responsible for regulating controlled substances and ensuring food safety, and to INFORM US farmers, the general public, and government agencies about the long-standing drug culture ties and questionable economics of Cannabis hemp/marijuana as a crop.

Wheras:

both fiber-hemp/marijuana and drug-hemp/marijuana are Cannabis sativa L. plants, contain a mind altering drug called THC (tetrahydrocannabinol), and are prohibited by Federal law;

both official US drug control agencies, the Office of National Drug Control Policy (ONDCP) and the US Drug Enforcement Administration (DEA), believe and have publicly stated that it would not be in the public interest to change the current status of Cannabis sativa L. (hemp/marijuana), determining that;

> *"the threat of diversion into the illicit drug trade associated with the cultivation of hemp/marijuana would not be in the public interest. Marijuana drug dealers will pay many times higher for hemp as a mix with higher grade marijuana to increase their profit than the hemp market could offer. There is no reliable field test to distinguish fiber-hemp from other varieties, therefore, law enforcement would be unable to arrest cannabis violators based on the required "probable cause" standard;"*

- the DEA ban on THC in hemp food products, though characterized as a drug war issue, is, in fact, a food safety issue. Despite the fact that NO state or country in the world has scientifically established the safety of food products made from hemp, the Ninth Circuit Court struck down the DEA ban in 2004;
- the present hemp movement in the U.S. and internationally was initiated by marijuana activists as officially reported by the U.S. Congress' research arm (CRS 92-510) which states that the "legalize marihuana (Hemp) movement" was "largely spurred by...Jack Herer,...."an internationally known marijuana activist dubbed the "godfather of hemp." Herer was quoted as saying he dreamed up the hemp movement one night while high on LSD.;
- "Vote Hemp"(chief lobby organization) & Hemp Industries Association – HIA (chief trade group) are orchestrating and funding hemp legalization efforts in the US; (Who funds them? George Soros" his co-funder, Peter Lewis?)
- Vote Hemp & HIA are headed by a former NORML employee, who, with High Times, co-produced two pro-marijuana/hemp CD albums. One, entitled Hempilation: Freedom is Norml, features pro-pot bands performing their favorite weed classics such as, "I Wanna Get High," "I Like Marijuana," and "Legalize It." Vote Hemp's President included in his closing remarks on the pro-pot CD liner notes, "Isn't it time we reconsider marijuana prohibition? ... We all need to ...demand the end of hemp prohibition now."; (Emphasis added)
- Vote Hemp helped to write federal bill, H.R. 1009 (now H.R. 831), which was introduced to Congress in 2007 by Ron Paul. That Bill would legalize hemp as an agricultural crop, and would take authority to regulate Cannabis hemp from the federal government and, assign authority over it to the states;
- H.R. 831 would make federal law enforcement subservient to the state legislative process;

Therefore, US Drug Preventionists, URGE that citizens, lawmakers, and other officials OPPOSE & PREVENT the legalization of Industrial Hemp (Cannabis sativa L.).

Additional Reasons to OPPOSE Legalization of Hemp (low-grade marijuana)

Drug abuse threatens our democratic institutions, national security, and Nation's future.

Pro-drug advocates use industrial hemp/marijuana as a symbol to promote the acceptance and legalization of marijuana.

The drug-driven Hemp Movement predated and created farmer and business involvement.

Vote Hemp drafted hemp bills & recruited farmers and public officials to introduce and spearhead their passage.

Vote Hemp recruited and funded legitimate US farmers to bring lawsuits against the DEA to change the legal status of hemp.

Claims of economic advantages to the agricultural community from growing industrial hemp-marijuana are significantly exaggerated. On an international level hemp/marijuana is not profitable for farmers who are growing it without government subsidies (the EU). In 2011, Canada's hemp market supported fewer than 40,000 acres of hemp.

Claims of environmental advantages from growing industrial hemp/marijuana are also significantly exaggerated.

Law enforcement at all levels – federal, state, and local – oppose the legalization of hemp/marijuana for industrial purposes, knowing that industrial hemp/marijuana can promote the illicit drug trade:

- Through increasing potency by harvesting selected parts of the plant.
- Through manufacturing into a higher-potency drug product using accessible recipes and ingredients, and
- Through using low-potency marijuana as a filler to increase the bulk of higher-potency marijuana sold in illicit markets.

The US Military and many city police departments (NYPD for one) prohibit their personnel from ingesting (eating/drinking) hemp products, which could jeopardize drug testing results.

THC accumulates in the fatty cells of the body.

A threshold THC concentration – below which industrial hemp/marijuana would have no significant psychoactive properties – has not been determined, such level being dependent upon the personal characteristics of each user. Inexperienced users, (for example, children) are especially vulnerable.

Smoking hemp/marijuana with a low THC level of 0.25 percent could result in psychological effects on inexperienced users (children, for example), or in individuals with a high decree of sensitivity to THC.

Supporting industrial hemp/marijuana sends an ambivalent and harmful message to youth and others regarding marijuana.

Marijuana use among our youth in the United States accounts for the highest percentage of admissions for drug treatment.

(Source: http://www.drugwatch.org/legislation/suggested-talking-points/209-opposes-legalization-of-industrial-hemp.html)

Coalition of Alcohol and Drug Educators

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Cannabis Regulation and Mental Health Policy
Cannabis – Violence & Psychosis

Cannabis Induced Psychosis (CIP) - Prominent, Growing & Devastating

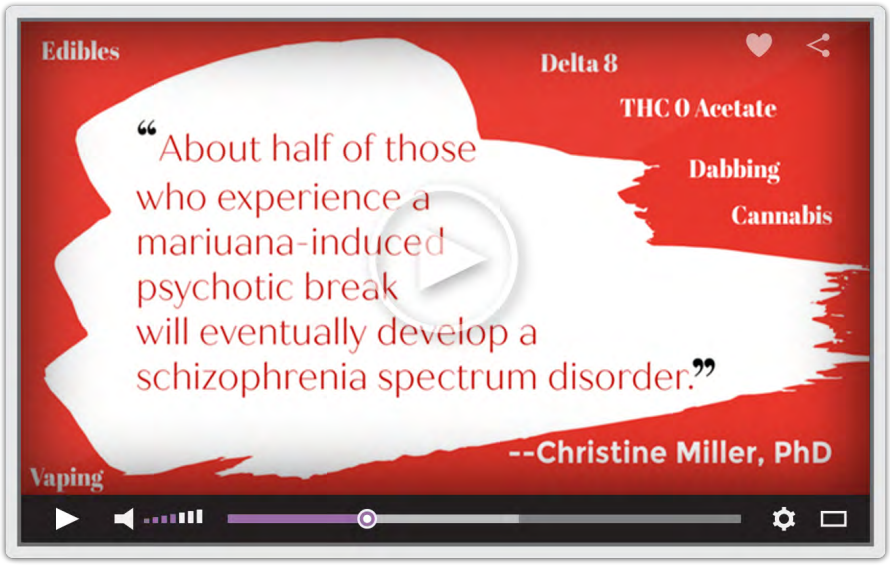
The THC in cannabis can destroy critical neuronal pathways in the developing brain, which can result in permanent brain changes. The worst-case scenario is psychosis that becomes permanent and is then considered schizophrenia, a life-long, debilitating disease. No one can predict in advance who will be susceptible, as some can experience symptoms after a few times of use.

The mental health harms of cannabis are well known to scientific researchers.

Professionals say the evidence found in peer-reviewed studies is undeniable: THC in cannabis, even in low concentrations, can cause psychosis. And out of the drugs that can cause a temporary episode of psychosis, marijuana/cannabis has the highest conversion rate to chronic psychotic disorders like bipolar and schizophrenia.

Dr. Christine Miller is a Molecular Neuroscientist with a PhD. Pharmacology. She researched the causes and nature of psychosis for thirty years of her career.

> *"The causal link between marijuana use and the development of psychosis is quite simply the most well-replicated, high-impact finding in schizophrenia research today. Given current use rates and the strong potency of the drug available, it stands to be responsible for a larger proportion of schizophrenia cases than any other established factor. Who may be at risk cannot be reliably predicted. The time is long overdue for the surgeon general and American neuroscientists and psychiatrists, along with their universities and professional societies, to inform the public and for journalists to pay heed."*
>
> Dr. C Miller



CLICK IMAGE TO PLAY

There are hundreds of peer-reviewed, scientific articles that prove the causal links between marijuana use and psychotic outcomes such as schizophrenia.

- Marijuana use generally comes before the psychosis, not vice-versa, so self- medication is not likely the cause. Continued cannabis use and risk of incidence and persistence of psychotic symptoms.
- The consensus is that use of marijuana with a THC content over 10% increases the risk of a psychotic disorder by 4-fold: Meta-analysis of the Association Between the Level of Cannabis Use and Risk of Psychosis.
- Frequent use of more potent products results in more cases of schizophrenia. Proportion of patients in south London with first-episode psychosis attributable to use of high potency cannabis.
- Cannabis intoxication becomes Cannabis-Induced Psychotic Disorder once certain severity and duration criteria are met and CIP is heavily associated with future schizophrenia diagnoses: Cannabis and Psychosis Through the Lens of DSM-5.
- A person suffering from marijuana-induced psychosis is over 18-times more likely to lash out violently. But individuals with psychosis from non-drug causes and who are medicated with antipsychotics but not using marijuana or other recreational drugs, do not pose a great risk for violence.
- The causal relationship of psychosis with marijuana is outlined in a paper on the International Academy on the Science and Impact of Cannabis website: Applying the Bradford Hill Elements of Causation to Cannabis Causing Psychosis.

CLICK IMAGE TO PLAY

(taken from Mom's Strong)

The egregious 'partnership' of marijuana and mass murder

Marijuana use and psychotic violence go hand-in-hand.

- In 2007 the prestigious medical journal Lancet recanted its previous benign view of marijuana, citing studies showing "an increase in the risk of psychosis of about 40 percent."
- A seminal long-term study of 50,465 Swedish army conscripts found those who had tried marijuana by age 18 had 2.4 times the risk of being diagnosed with schizophrenia in the following 15 years than those who had never used the drug. Heavy users were 6.7 times more likely to be admitted to a hospital for schizophrenia.
- Another study, of 1,037 people in New Zealand, found those who used cannabis at ages 15 and 18 had higher rates of psychotic symptoms at age 26 than non-users.
- A 2011 study in the British Medical Journal (BMJ) of 2,000 teenagers found those who smoked marijuana were twice as likely to develop psychosis as those who didn't. Another BMJ study estimated that "13 percent of cases of schizophrenia could be averted if all cannabis use were prevented."
- In 2014, people who had cannabis use disorder made up about 1.5 percent of Americans. But they accounted for eleven percent of all the psychosis cases in emergency rooms—90,000 cases, 250 a day, triple the number in 2006.
- The National Academy of Medicine found in 2017 that "cannabis use is likely to increase the risk of developing schizophrenia and other psychoses; the higher the use, the greater the risk." Also, that "regular cannabis use is likely to increase the risk for developing a social anxiety disorder."
- Study showing cases in which marijuana led to unnecessary violence, health risks, and, in many cases, both.
- About half of the attackers had a history of illicit drug use and/or substance abuse. This abuse, which included alcohol and marijuana, was evidenced by such factors as the attacker receiving treatment for the abuse, suffer legal consequences, or having significant problems in their personal lives stemming from the abuse.

Further research go to...

- All Young Cannabis Users Face Psychosis Risk
- Cannabis & Psychosis – irrefutable
- Cannabis & Mental Health – Professor Copeland
- Cannabis & Psychosis: Understanding risk is of 'vital importance'
- Cannabis Library – Mental Health Impact
- Marijuana and Psychosis - AALM

(Source https://momsstrong.org, Americans Against Legalizing Marijuana and Dalgarno Institute)

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Cannabis and Pregnancy (Short and Long Term Harms)

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Cannabis and Pregnancy (Short and Long Term Harms)

As early as 1983, when cannabis was closer to it's natural state, concerns were being raised about the prenatal impact of cannabis, but was not pursued at any length, as it appeared 'negligible' . However, the warning signs were in play.

The US. National Institute of Drug Abuse (NIDA) produced a monograph on *Marijuana Effects on the Endocrine and Reproductive Systems.*

This, arguably, seminal research with its heavy restrictions and many obstructive variables, still presented the researchers with a clear warning that this substance, as 'mild' as it was by today's standards was very concerning and that policy and law makers need to be incredibly careful moving forward on this substance.

These warnings were headed up until the mid 90's when a new way of propaganda was launched by pro-cannabis groups, in claiming a vast array of therapeutic potentials. Cannabis use in the early 90's in the US was it's lowest in recent history, largely because of the strong demand and supply reduction efforts of successive governments working to deal with an 'out of control' drug problem throughout the late 70's and 80's. This push is now it is 26th year and has got traction – despite the failure of some many therapeutic promises, and a growing list of serious short and long term harms to children and the damaging of the epigenome that have been buried by pro-pot propaganda.

It is time now for responsible community leaders to, at the very least, look after the emerging and coming generation around this increasing harmful substance.

> *The rate of marijuana use by pregnant women is sufficient to represent a health hazard if marijuana is found to have a deleterious effect upon maternal well-being or fetal development. The reported use among pregnant women, varying in the localities sampled from 10% to 37%, is comparable to an estimated 26% of women between ages 18 and 25 surveyed in the general population (Fishbume et al. 1980). Inconclusive evidence from the first exploratory studies in the human suggest that marijuana use may alter the delivery process, reduce intrauterine weight gain by the fetus or affect visual and neurological excitatory responses. Confirmation of these findings among the studies is lacking as is evidence for the unconfounded or direct action of marijuana. Nevertheless, the studies serve an important function in providing directions for new and ongoing investigations. Finally, three issues might be considered of importance for future investigations of the effects of marijuana as used by pregnant women. First, the degree of placental transfer of -9-THC or its metabolites, including the possibility that rates are variable over the course of pregnancy, has not been determined in the human (Abel 1980). Second, the synergistic or interactive effects of illicit drugs or medications with marijuana as taken by pregnant women has been given little attention. Third, the immediate clinical relevance of findings as well as the long-term effects need to be assessed in evaluating the impact of the drug.*
>
> (Source: https://archives.drugabuse.gov/sites/default/files/monograph44.pdf 1983)

In the ensuing couple of years, small and tentative research was continued, and again, within the context of trial restrictions.

Prenatal Marijuana Use: Epidemiology, Methodology Issues, and Infant Outcome

Marijuana use is prevalent among women of childbearing age, and although most women decrease their use during pregnancy, some continue to use marijuana throughout pregnancy. Reports of the effects of prenatal marijuana use on the newborn and developing child are equivocal and have not been replicated consistently across studies. Important methodologic considerations in the study of prenatal exposure to marijuana include the method and timing of assessment and the use of covariates in the statistical analyses.

(Source: https://www.sciencedirect.com/science/article/abs/pii/S0095510818305359 1985)

Around 1985, studies looking a little more closely at the neo-natal state of babies revealed a clear correlate with cannabis use and emerging harms to the baby, not dissimilar to the impact of other and legal substances of nicotine and alcohol.

Neonatal behavioural correlates of prenatal exposure to marihuana, cigarettes and alcohol in a low risk population

Infant neonatal behaviour is significantly and differentially related to maternal marihuana, cigarette and alcohol use during pregnancy. Data on 250 babies born to healthy, predominantly middle-class women were analyzed using canonical analysis and multiple regression adjusting for potentially confounding variables. Prenatal marihuana exposure was associated with increased tremors and startles and poorer habituation to visual stimuli, prenatal cigarette exposure with increased tremors and poorer auditory habituation, whereas a relatively low level of alcohol consumption was marginally related to increased neonatal irritability.

(Source: https://www.sciencedirect.com/science/article/abs/pii/0892036287900626 1987)

Nearly 40 years on and with greater population use and thus sample size, the evidence of multiple concerns has both confirmed earlier concerns and given even greater insights into the harms of cannabis on the developing foetus.

Cannabis Consumption Patterns Explain the East-West Gradient in Canadian Neural Tube Defect Incidence: An Ecological Study

The epidemiological relationship that we have demonstrated between cannabis use and NTD (Neural Tube Defects) incidence within the context of falling overall NTD rates is interesting, provocative, and intriguing. The ecological association is seen in both live born statistics and also in estimates of the complete dataset including ETOPFA data, which are important to complete the holistic picture of the true epidemiological incidence of NTDs; it is seen with 2 metrics of cannabis use and with 2 categorization algorithms for classifying the provinces.

(Source: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6906350/ 2019)

Cannabis Teratology Explains Current Patterns of Coloradan Congenital Defects: The Contribution of Increased Cannabinoid Exposure to Rising Teratological Trends

Rising Δ9-tetrahydrocannabinol concentrations in modern cannabis invites investigation of the teratological implications of prenatal cannabis exposure. Data from Colorado Responds to Children with Special Needs (CRCSN), National Survey of Drug Use and Health, and Drug Enforcement Agency was analyzed. Seven, 40, and 2 defects were rising, flat, and falling, respectively, and 10/12 summary indices rose. Atrial septal defect, spina bifida, microcephalus, Down's syndrome, ventricular septal defect, and patent ductus arteriosus rose, and along with central nervous system, cardiovascular, genitourinary, respiratory, chromosomal, and musculoskeletal defects rose 5 to 37 times faster than the birth rate (3.3%) to generate an excess of 11 753 (22%) major anomalies. Cannabis was the only drug whose use grew from 2000 to 2014 while pain relievers, cocaine, alcohol, and tobacco did not. The correlation of cannabis use with major defects in 2014 (2019 dataset) was R = .77, P = .0011. Multiple cannabinoids were linked with summary measures of congenital anomalies and were robust to multivariate adjustment.

(Source: https://pubmed.ncbi.nlm.nih.gov/31288542/ 2019)

Maternal cannabis use is associated with suppression of immune gene networks in placenta and increased anxiety phenotypes in offspring

Cannabis use is becoming more prevalent, including during developmentally sensitive periods such as pregnancy. Here we find that maternal cannabis use is associated with increased cortisol, anxiety, aggression, and hyperactivity in young children. This corresponded with widespread reductions in immune-related gene expression in the placenta which correlated with anxiety and hyperactivity. Future studies are needed to examine the effects of cannabis on immune function during pregnancy as a potential regulatory mechanism shaping neurobehavioral development.

(Source: https://www.pnas.org/content/118/47/e2106115118 Nov 2021)

Perinatal THC exposure via lactation induces lasting alterations to social behavior and prefrontal cortex function in rats at adulthood

Abstract:

Cannabis is the world's most widely abused illicit drug and consumption amongst women during and surrounding the period of pregnancy is increasing. Previously, we have shown that cannabinoid exposure via lactation during the early postnatal period disrupts early developmental trajectories of prefrontal cortex maturation and induces behavioral abnormalities during the first weeks of life in male and female rat progeny. Here, we investigated the lasting consequences of this postnatal

cannabinoid exposure on synaptic and behavioral parameters in the adult offspring of Δ9-tetrahydrocannabinol (THC)-treated dams. At adulthood, these perinatally THC-exposed rats exhibits deficits in social discrimination accompanied by an overall augmentation of social exploratory behavior. These behavioral alterations were further correlated with multiple abnormalities in synaptic plasticity in the prefrontal cortex, including lost endocannabinoid-mediated long-term depression (LTD), lost long-term potentiation and augmented mGlu2/3-LTD. Finally, basic parameters of intrinsic excitability at prefrontal cortex pyramidal neurons were similarly altered by the perinatal THC exposure. Thus, perinatal THC exposure via lactation induces lasting deficits in behavior and synaptic function which persist into adulthood life in male and female progeny.

Discussion:

Here, we have discovered that perinatal exposure to THC via lactation induces significant behavioral and electrophysiological alterations lasting into adulthood. Specifically, we found that this THC exposure alters social behavior, as well as synaptic plasticity and basic parameters of cell-excitability in the PFC of adult male and female offspring of dams given THC during the first 10 days of postnatal life.

…Our results indicate that perinatal THC exposure via lactation induces lasting deficits at multiple scales which persist into adulthood. THC-exposed offspring exhibit increased social exploration at the cost of discrimination, coupled with significant alterations to multiple forms of plasticity in the PFC which are normalized via enhanced basal 2-AG ex vivo. Increased excitability of principal neurons of the PFC may underlie or accompany these issues, and further investigations are required to further characterize the extent to which basic synaptic transmission may be impacted by this early life exposure. Further, both an increased breadth of behavioral investigations as well as extended characterizations of plasticity and synaptic functions in these animals in other brain regions are necessary to provide a more thorough picture of the extent to which perinatal cannabis exposure induces lasting deficits in brain function into adulthood.

(Source: Neuropsychopharmacology (2020) https://www.nature.com/articles/s41386-020-0716-x)

Psychoactive ingredient in marijuana may impair ability to produce viable embryos

Female eggs exposed to THC, the psychoactive ingredient in marijuana, have an impaired ability to produce viable embryos, and are significantly less likely to result in a viable pregnancy, according to an animal study accepted for presentation at ENDO 2020, the Endocrine Society's annual meeting. The abstract will be published in the Journal of the Endocrine Society.

THC exposure led to a significant decrease in the expression of genes called connexins, which are present at increased levels in higher quality oocytes. Poorer

quality oocytes, with lower connexin expression levels, have been shown to lead to a poorer embryo development. "This embryo would be less likely to proceed past the first week of development, and thus lead to infertility," Misner said.

(Source: Endocrine Society https://www.news-medical.net/news/20200402/Psychoactive-ingredient-in-marijuana-may-impair-ability-to-produce-viable-embryos.aspx April 2020)

Appendix

- **Marijuana Use in Pregnancy May Lead to a More Anxious, Aggressive Child - The New York Times (nytimes.com)**
- **Cannabis & Pregnancy - Real Caution Needed, NOW!**
- **Prenatal cannabis exposure associated with adverse outcomes during middle childhood**
- **Self-reported Daily, Weekly, and Monthly Cannabis Use Among Women Before and During Pregnancy**
- **Cannabis Use During Pregnancy May Affect Brain Development in Offspring**
- **Pregnant women who use marijuana heavily to treat morning sickness affect part of the baby's brain associated with memory**
- Agence France-Presse in Paris. ***France to investigate cause of upper limb defects in babies.*** The Guardian [Internet]. 2018 3rd November 2018 [cited 2018 3rd November 2018]. Available from: https://www.theguardian.com/world/2018/oct/21/france-to-investigate-cause-of-upper-limb-defects-in-babies.
- Gant J. ***Scientists are baffled by spatter of babies born without hands or arms in France, as investigation fails to discover a cause.*** Daily Mail [Internet]. 2019 14th July 2019 [cited 2019 14th July 2019]; Sunday 14th July Available from: https://www.dailymail.co.uk/news/article-7242059/Scientists-baffled-babies-born-without-hands-arms-France-probe-fails-discover-cause.html.
- Willsher K. ***Baby arm defects prompt nationwide investigation in France.*** Guardian [Internet]. 2018 3rd November 2018 [cited 2018. Available from: https://www.theguardian.com/world/2018/oct/31/baby-arm-defects-prompt-nationwide-investigation-france
- **Dalgarno Institute Cannabis Library** – Genetic Impact

Coalition of Alcohol and Drug Educators

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“Medicinal” Cannabis and Driving – is it an Issue?

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“Medicinal” Cannabis and Driving – is it an Issue?

With several global jurisdictions legalizing cannabis for either ‘medicinal’ or recreational use, the issue of its influence on public safety, particularly in motor vehicle crashes and the subsequent injuries and deaths, a more robust understand of harms must be established.

The presence of THC, specifically Delta 9 Tetrahydrocannabinol (potentially other THC variations) as the psychotropic constituent of some cannabis-based medicines does interfere with driving competencies.

As we are aware, may properly vetted and approved prescribed pharmaceutical grade/ manufactured medicines of various origins can create impairment via drowsiness, and the slower reaction times this diminished state can bring. Consequently, these prescriptions come with clear warnings that driving whilst on this medicine is ‘warned’ against.

However, intoxication is a different state, and one that ensures, for example, that the intoxicated is prohibited from driving under current drink driving laws.

What is important to note is that whilst drowsiness can be one symptom of intoxication, it is not the only one. Intoxication brings another level of diminished capacity to the driver, and along with the idiosyncratic nature of intoxicants – not least THC – the potential for multi-level public harms is markedly increased.

One of the big ‘pushes’ from one sector is to have THC based ‘medicinal’ cannabis, as it is promoted, added to the list of medications and removed from the list of prohibited substances for driving. This lobby group site an ‘unfairness’ in the legislation that states their ‘medicine’ is treated differently from other prescriptions and those using such formulations are unfairly penalized.

Under the current Pharmaceutical Benefits Scheme, there are only two THC based preparations that are certified by the TGA as medicines, these are Sativex® and Marinol®. Other proposed formulations that have not been fully clinically double blind, placebo accounted for trailed, and have not been given that pharmaceutical status, and in scientific terms are not medicine.

However, now that the Australian Therapeutic Goods Administration (TGA) have allowed and now actively promoting a ‘new’ category for ‘medicinal cannabis’, the number of THC contained ‘medicines’ are exponentially increased. Making the now Category 4 & 5 (non-clinically trialled) products easier to access for ‘prescribing’ purposes.

The potential for abuse of this *new opportunity to access cannabis* ‘legally’ has grown substantially, and to state the obvious, how will law enforcement know from which source the THC came? Supplementing and misuse of this substance will now be made easier, and the potential for intoxicated driving be given a free pass on the basis of ‘it’s my medicine’ and exempted from penalty.

That very credible hypothetical aside, it is important for everyone’s public safety, not least the THC user, that clear boundaries be set, and that *no driving be permitted at all* for a prolonged period of time for those using this psychotropic substance.

For example, if a base line is to be drawn to maximize safety at say 24 hours, then it

would become clear that someone using this psychotropic substance daily, will not be permitted to drive with any degree of assured safety. Even if it is 12 hours, clear issues present.

The following information and data makes clear that excising Cannabis use (THC) from the prohibition from driving a vehicle legislation would be a public safety mistake.

* * * * *

The jurisdictions with the most experience of this issue are the ones with laws allowing either 'medicinal' or recreational use of cannabis – Two significant jurisdictions are the United States and Canada. There records and research will feature strongly in this work.

The following advice one such example from the **Prevention Policy Alliance** in Ohio, USA:

> *Marijuana use is not without risks and has potentially dangerous consequences - especially for drivers on the road. Since medical marijuana is now legal in Ohio, it's important to understand the risks of marijuana use and driving.*
>
> *While we all know that impaired driving is problematic, driving while high on marijuana carries unique risks. According to the National Highway Traffic Safety Administration, there has been a 48% increase in nighttime drivers who tested positive for THC - the chemical responsible for marijuana's psychological affects. Marijuana can slow reaction time and the ability to make decisions. Driver's high on marijuana hit more pedestrians, exceed the speed limit more often, make fewer stops at red lights and make more center line crossings.*
>
> *Drivers who consume both marijuana and alcohol and then drive experience impaired judgement that leads to some of the most dangerous driving on the road. According to the Traffic Safety Culture Index, drivers who use both marijuana and alcohol were significantly more prone to driving under the influence of alcohol. They are more likely to speed, text, intentionally run red lights and drive aggressively.*
>
> *Prevention professionals understand that legalization of substances lowers an individual's perception of risk, altering an individual's judgement about the likelihood of negative occurrences related to that substance. As Ohio considers expanding marijuana legalization, it is important to understand the dangers it will pose to traffic safety.*

However, this state is only recently coming to grips with this growing public safety problem, where as other jurisdictions, not least the Sate of Colorado, have seen the devastating impact that THC driving has had on both road and public safety.

Colorado's Department of Public Health and Environment have made definitive recommendations around marijuana use and driving. In the 2018 summary, the following evidence-based realities were presented.

Marijuana use and driving

The committee reviewed driving impairment and motor vehicle crash risk relative to marijuana use, as well as evidence indicating how long it takes for impairment to resolve after marijuana use. The risk of a motor vehicle crash increases among drivers with recent marijuana use. In addition, using alcohol and marijuana together increases impairment and the risk of a motor vehicle crash more than using either substance alone. For less-than-weekly marijuana users, using marijuana containing 10 milligrams or more of THC is likely to impair the ability to safely drive, bike or perform other safety-sensitive activities. Less-than-weekly users should wait at least six hours after smoking or eight hours after eating or drinking marijuana to allow time for impairment to resolve. Research is lacking on marijuana and impairment in frequent marijuana users.

Monitoring Health Concerns Related to Marijuana in Colorado: 2018 Summary, Colorado Dept of Public Health & Environment. Detailed findings and data available at colorado.gov/marijuanahealthinfo

This evidence has been affirmed in other arenas, as the video presentation below will confirm, and any 'medicines' with THC preparations involved are going to cause impair, regardless of the perceived impact on the marijuana user https://youtu.be/ToOy2imdYOY

Marijuana

Cannabinoid screens were conducted for 5,032 case filings, representing one-fifth of all case filings (see Table 16). Of these, 34% indicated that no cannabinoids were detected.32 Cases with a positive cannabinoid screen (66%, n=3,335) were further confirmed for Delta 9-THC and other cannabis metabolites.33 The testing positivity rate in 2018 was nearly identical to the 2017 rate, and both years' rates represent a decline from 2016's. Furthermore, among all case filings screened for cannabinoids (n=5,032), 57% tested positive for Delta 9-THC. The presence of Delta 9-THC recorded in a linked toxicology report might indicate the driver's recent use of cannabis preceding the offense. The median value of Delta 9-THC among individuals screened was 5.2 and the mean was 8.2 ng/mL, both of which are over the permissible inference level.

Table 16. Cannabinoid screen results among DUI case filings, 2016-2018

Screen Result n (%)	*2016*	*2017*	*2018*
Cannabinoids Not Present	*1,061 (26.9%)*	*1,622 (33.8%)*	*1,697 (33%)*
Cannabinoids Present	*2,885 (73.1)*	*3,170 (66.2)*	*3,336 (66.3)*
Total N	*3,946*	*4,792*	*5,032*

Data source: State Judicial Department, Denver County Court, CBI, and ChemaTox. Analyzed by the Office of Research and Statistics, Division of Criminal Justice, Colorado Department of Public Safety.

Table 17 compares the various levels of Delta 9-THC detected among case filings undergoing confirmatory testing (n=3,335 in 2018). About a sixth of these case filings had no Delta 9-THC detected or levels that were less than one ng/mL, approximately one-third had levels between one and the permissible inference level of five ng/mL, and about half had a level at or above the permissible inference level.

Table 17. Delta 9-THC levels for case filings with Delta 9-THC confirmation test, 2016-2018

	2016	2017	2018
N	*2,885*	*3,170*	*3,335*
Delta 9-THC level n (%)			
None Detected	*396 (13.7%)*	*431 (13.6%)*	*459 (13.8%)*
Present but <1.0	*90 (3.1)*	*63 (2.0)*	*88 (2.6)*
1.0-4.9	*1,030 (35.7)*	*1,069 (33.7)*	*1,134 (34.0)*
5.0+	*1,369 (47.5)*	*1,607 (50.7)*	*1,654 (49.6)*
Median level (ng/mL)	*5.9*	*5.4*	*5.2*
Mean level (ng/mL)	*8.7*	*8.2*	*8.2*

Data source: State Judicial Department, Denver County Court, CBI, and ChemaTox. Analyzed by the Office of Research and Statistics, Division of Criminal Justice, Colorado Department of Public Safety.

Common Charges Associated with Marijuana

A total of 6,303 final non-DUI charges were associated with the presence of Delta 9-THC; see Appendix I for the top 20 charges. Similar to alcohol, the top four charges were for careless driving (n=665), failure to display proof of insurance (n=437), lane usage violation (n=434), and speeding (n = 208).

Time to Blood Test

Time to blood test data is difficult to capture because it requires manual data entry from CBI's Requests for Laboratory Exam forms. This data entry was completed in 2017 but time constraints precluded this undertaking for the 2018 data. For the current analysis, instead, 2,012 ChemaTox records with draw time data were analyzed, although this represents only 12% of all DUI case filings with toxicology matches. Due to the lower number of cases available, the data from 2016 to 2018 were combined and the aggregate results are presented in Table 18. The higher mean time and lower median time in 2018 compared to 2017 and 2016 data may reflect the increased variability in the data due to the lower sample size.

Table 18. Descriptive statistics and toxicology source for time-to-test analyses by year, 2016-2018

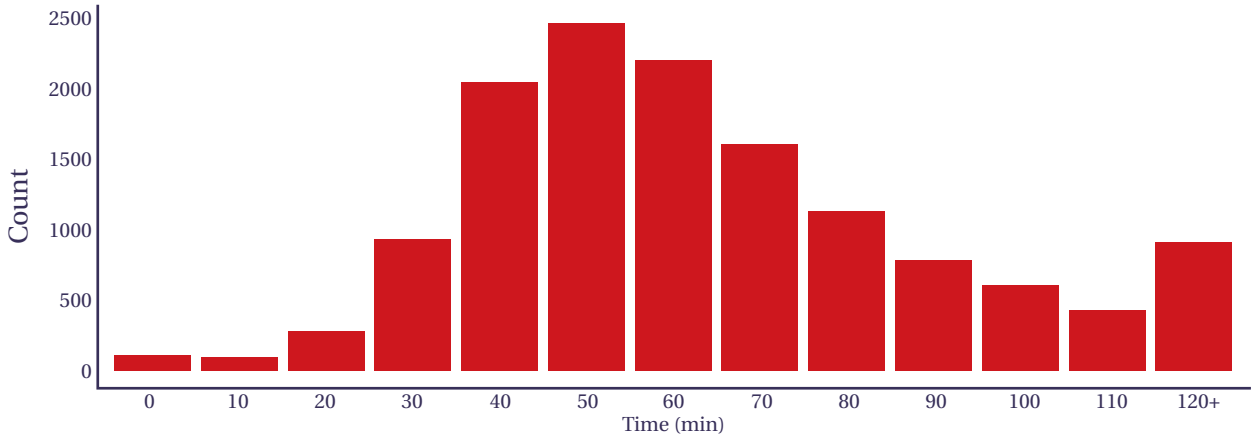
Year	Mean (min)	Median (min)	No. of Case Filings	Toxicology Source
2016	*72.5*	*64*	*4,154*	*ChemaTox*
2017	*75.7*	*64*	*7,667*	*ChemaTox & Colorado Bureau of Investigation*
2018	*88.5*	*60.5*	*2,012*	*ChemaTox*
All	*76.6*	*64*	*13,833*	

Data source: State Judicial Department, Denver County Court, CBI, and ChemaTox. Analyzed by the Office of Research and Statistics, Division of Criminal Justice, Colorado Department of Public Safety.

For the combined 2016 through 2018 data, 310 records reporting test times of over 200 minutes were excluded in an attempt to analyze measurements that might be more associated with impairment. This sample of case filings (n=13,539) was used in the analyses below.

The frequency for time-to-test is depicted in Figure 9. The time interval of 50–59 minutes (category 50 in Figure 9) had the greatest number of blood draws (n=2,469), accounting for 21% of the time categories. Nine percent (n=910) of records exceeded an elapsed time of 120 minutes from time of offense to time of blood draw.

Figure 9. Time-to-test for DUI case filings, 2016-2018 (n=13, 539)

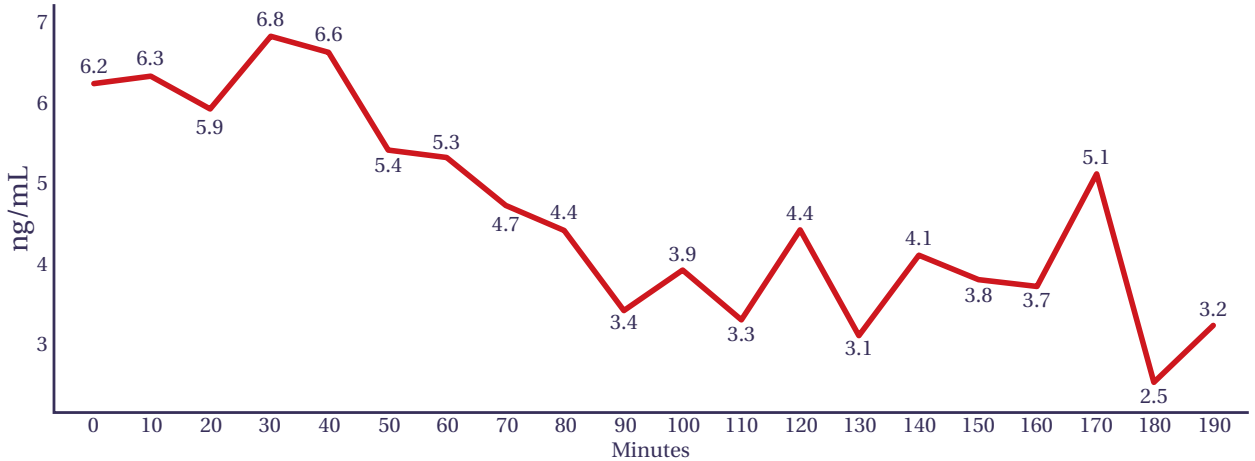


Data source: State Judicial Department, Denver County Court, CBI, and ChemaTox. Analyzed by the Office of Research and Statistics, Division of Criminal Justice, Colorado Department of Public Safety.

Marijuana and Time-to-Test

A comparison of time to blood test by median Delta 9-THC value for 2016 to 2018 can be seen in Figure 10. Median Delta 9-THC values peaked between 30-39 minutes for the time of the offense to blood draw and then gradually fell for blood draws collected between 40-99 minutes. The changes in the slope in the Delta 9-THC levels for blood draws collected after 100 minutes might highlight the fragility of this relationship, and/or the presence of a threshold where time to draw may be more reflective of residual Delta 9-THC in the driver.

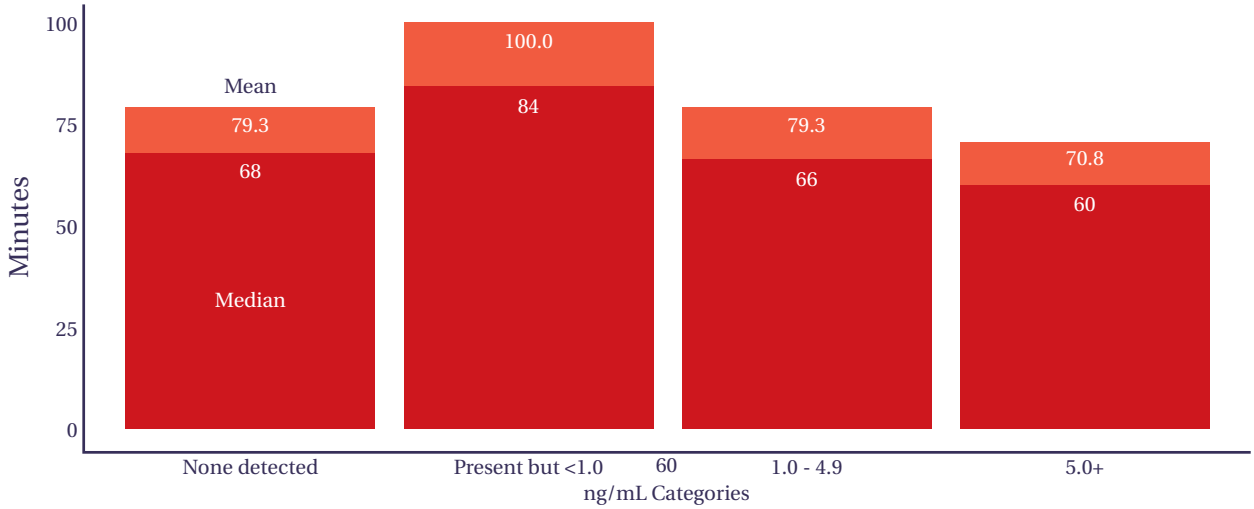
Figure 10. Median Delta 9-THC value by time-to-test and number of cases, 2016-2018 (n=13, 539)



Data source: State Judicial Department, Denver County Court, CBI, and ChemaTox. Analyzed by the Office of Research and Statistics, Division of Criminal Justice, Colorado Department of Public Safety.

In addition, we also compared the mean and median time to draw for each of the Delta 9-THC categories for case filings with positive cannabinoid screenings, as shown in Figure 11. The median and mean of the elapsed draw time for the quantified Delta 9-THC category decreased as the Delta 9-THC values increased. This trend aligns with evidence in the research literature that Delta 9-THC levels peak early and then quickly dissipate.

Figure 11. Mean and median Delta 9-THC value by time-to-test, 2016-2018 (n=13, 539)



Data source: State Judicial Department, Denver County Court, CBI, and ChemaTox. Analyzed by the Office of Research and Statistics, Division of Criminal Justice, Colorado Department of Public Safety.

(Source: Driving Under the Influence of Drugs and Alcohol: A Report Pursuant to House Bill 17-1315 (state.co.us)**)**

Cannabis Legalization and potential Associations with an Increase in Cannabis-related Motor Vehicle Crash Fatalities.

Cannabis use is a risk factor for motor vehicle crash (MVC) fatalities, but the degree of a driver's intoxication varies by tetrahydrocannabinol (THC) level. However, cannabis testing does not assess THC levels in most US states, and testing rates among MVC decedents vary among states and over time, which may bias estimates of cannabis involvement. Researchers assessed cannabis involvement and THC levels among fatally injured drivers in Washington State before and after the legalization of non-medical ("recreational") cannabis use, with and without imputation of missing cannabis testing data among the roughly half of decedents who were not tested.

- Using data from all MVC decedent drivers based on observed and imputed values, the prevalence of cannabis involvement in MVC fatalities was 9% prior to legalization and 19% after.
- In adjusted analyses, the proportion of decedent drivers with high THC levels (>10 ng/mL) increased nearly 5-fold after legalization.
- Although cannabis testing rates increased during the study period, findings were generally similar when restricted to those with completed cannabis testing.

Comments: This study is one of the first to impute cannabis involvement in MVC fatalities among decedents without testing, and to measure and impute THC levels (rather than simply the presence or absence of THC). Legalization of non-medical cannabis use in Washington State was associated with increases in cannabis involvement in MVC fatalities, including at levels clearly associated with impairment. These results add to literature suggesting that legalizing cannabis may increase MVC fatalities, and highlights the need to better characterize and mitigate those risks.

(Source: *Is Cannabis Legalization Associated with an Increase in Cannabis-related Motor Vehicle Crash Fatalities? | Alcohol, Other Drugs, and Health: Current Evidence (bu.edu)***)**

The Canadian Perspective

Marijuana impairment. In comparison to alcohol, less is known about marijuana and driving in terms of how marijuana specifically impairs driving skills. Marijuana studies have shown the psychoactive chemical delta-9-tetrahydrocannabinol (or THC) enters the user's bloodstream and brain immediately after smoking or consuming it. Since marijuana is very soluble in fat tissue, the absorption, distribution, and elimination of marijuana does not occur at a steady rate. Instead, it varies based on biological processes according to several factors, including route and frequency of intake; THC dose; titration of dose when smoked or vaporized; and, user characteristics. Not only do these factors affect the amount of marijuana intake and metabolism, they also affect the degree of behavioural impairment exhibited by users. For example, if marijuana is ingested, the onset of the impairing effects of edible marijuana products occurs more slowly and last longer as compared to smoking.

Furthermore, marijuana does not display a dose-response (in this case concentration) relationship, as is the case with alcohol. Unlike BACs, peak THC concentrations do not correlate well with the degree of behavioural impairment (Huestis 2007; Compton 2017). For example, studies of marijuana use and driving impairment have shown the level of THC measured in blood or oral fluid and the degree of impairment are not closely related; peak THC levels can occur when low levels of impairment are measured, and high levels of impairment can be measured when THC levels are low (Compton, 2017; Marcotte, 2020). The lack of definitive knowledge to quantify a concentration-response relationship for marijuana may be in part due to typical differences in research methods, tasks, subjects and dosing that have been used to date (Compton, 2017). Additionally, some studies have reported a wide variability in THC levels in the blood which are affected by the means of ingestion (smoking, oil, and edibles), potency, and user characteristics (Compton, 2017). This may indicate the concentration-response relationship can vary according to specific types of marijuana products consumed and individual biology. The lack of a concentration-response relationship for marijuana has important implications. Notably, there is much debate concerning the validity of a per se limit for marijuana due to the lack of strong scientific consensus regarding THC concentration in blood that constitutes driving impairment (Grotenhermen et al. 2007; Newmeyer et al. 2017). However, generally speaking, studies on marijuana showed:

Low doses of marijuana produce mild to moderate impairment in cognitive and psychomotor abilities; and larger doses showed significant impairment in cognitive, psychomotor and driving performance.

Laboratory studies of the impairment effects of marijuana use on psychomotor and cognitive functions suggested marijuana consumption can impair driving task-related abilities such as motor control, executive function, visual processing, short-term memory, and working memory in a dose-dependent fashion (Broyd et al. 2016; Ramaekers et al. 2004; Ramaekers et al. 2006). Reviews of studies on the effects of marijuana on driving skills demonstrated marijuana can specifically impair certain skills necessary for safe driving (Hartman et al., 2012; Compton 2017; Battistella et al., 2013), such as:

- controlling speed variability;
- lane positioning;
- reaction time;
- divided attention;
- attention maintenance;
- route planning;
- decision-making; and,
- risk-taking.

In some driving simulator studies, marijuana use was shown to increase driver reaction time and the number of incorrect responses to emergencies. In addition, drivers crashed more frequently into a sudden obstacle on a high dose of THC, although this was not seen at low doses (Sewell et al., 2009: citing Smiley, 1986; Smiley et al., 1981). Starkey and Charlton (2017) conducted a systematic review of marijuana-related behavioural studies and found that marijuana use was associated with reckless driving and speeding, signaling errors and decreased ability on tracking tasks.

A recent study involving participants who smoked marijuana and used a driving simulator demonstrated a moderate effect of THC on driver performance. Some subjects showed reduced performance compared to a placebo group, while other subjects showed little difference (Marcotte, 2020). Driving performance was assessed in terms of ability to maintain lateral position while undertaking a distracting task as well as maintaining the distance from a leading vehicle. Furthermore, the effects were seen to be most pronounced in the first two hours after use, with some recovery seen after three and a half hours.

Marijuana use has been associated with a significantly increased risk of fatal crash involvement. Drivers using marijuana are at an increased risk of injury anywhere from 1.8 to 2.8 times higher. Furthermore, the odds of drivers being found responsible for a crash increased with rising marijuana concentrations in the blood (Li et al., 2013; Asbridge et al., 2012; Starkey and Charlton 2017; Els et al., 2019; Drummer et al., 2003; Drummer et al. 2004). In fact, research on drivers in fatal crashes has shown THC-positive drivers were more than twice as likely to crash as drivers without THC (Grondel 2016).

However, while marijuana use has been shown to have impairing effects on skills required for driving, simulator studies investigating behavioural changes driving under the influence of marijuana have concluded marijuana use by drivers may result in compensatory behaviours, such as:

- ✓ decreased speeds;
- ✓ fewer attempts to overtake; and,
- ✓ an increased following distance to the vehicle in front.

These findings are in sharp contrast to studies investigating the effects of alcohol use (Hartman et al., 2016; Sewell et al. 2009). Other studies have demonstrated no adverse effects of marijuana use on sign detection, a sudden lane-changing task, or the detection of and response to hazardous events. (Sewell et al., 2009: citing Sexton et al., 2000; Smiley, 1986; Stein et al., 1983). It has been hypothesized that despite the impairing effects of marijuana, drivers using marijuana alone tend to overestimate their level of impairment and rely on compensatory behaviours to reduce crash risk. In one study, following a 7 ng dose of THC, drivers rated themselves as impaired even though their driving performance was not. Conversely,

alcohol at a relatively low BAC of .04 resulted in impaired driving performance although drivers rated themselves as unimpaired (Sewell et al.,2009: citing Robbe and O'Hanlon, 1993). In other words, drivers using marijuana may be more aware of their level of impairment whereas drivers using alcohol under-estimate their impairment. However, this may not always be the case. One study (Marcotte, 2020) measuring driver performance in a simulator showed subjects perceived the impairing effects of THC to be eliminated before a measurable improvement in driving performance was seen.

(Source: *ALCOHOL, MARIJUANA & DRIVING RISK December 2020 By Craig Lyon & Robyn D. Robertson (Traffic Injury Research Foundation of Canada - SoberSmartDriving.tirf.ca)***)**

The recently released Australian research Determining the magnitude and duration of acute △9-tetrahydrocannabinol (△9-THC)-induced driving and cognitive impairment: A systematic and meta-analytic review

Highlights:

- Meta-analyses confirm that acute Δ^9-THC administration impairs aspects of driving performance.
- Meta-regression analyses suggest regular cannabis users experience less Δ^9-THC-induced impairment than occasional users.
- Other factors also influence the degree of impairment observed (e.g. dose, post-treatment time interval, type of skill).
- Most driving-related skills are predicted to recover within ~5-hs (and almost all within ~7-hs) of inhaling 20 mg Δ^9-THC.
- Oral Δ^9-THC-induced impairment may take longer to subside.

(Source: *https://www.sciencedirect.com/science/article/pii/S0149763421000178?via%3Dihub November 2021***)**

The analysis concluded as much of the other research has at least landed on that 'minimum times of waiting until doing sensitive tasks' applied, and the disturbing caveat that "regular cannabis users experience less THC induced impairment..." Before any further challenging of some of these findings, not least is the concern that self-reporting capacity to drive from the seasoned cannabis user can easily be related to the alcoholic who believes they too can manage to drive safely over the legal limit, regresses our world leading drink/drug driving regulations back to old 'sobriety tests' for every subjective situation.

That (as important as it is) aside, we submit the following review of this research.

- The proposal of permitting a medical exemption to the "presence offense" if there was no impairment and the cannabis was prescribed and taken as prescribed is a low-risk modification to Australian laws. But it's the camel's nose in the tent problem. Pretty soon you have the whole camel in your sleeping bag. (As per the alcohol issue above)
- Few users would be impacted by the proposal. It seems that only 1.8% of "medical" marijuana users get it by prescription and 89% of that is oils and sprays. Because of the admittedly large number of users who supplement their "medication" with illicit product, we see the proposal fairly innocuous except for the nose in the tent problem.

- The current cannabis warning label is insufficient. It only recognizes drowsiness as a consequence of taking THC-containing products. If the recommendation is adopted, there should be a much stronger warning about the dangers of both impairment and brain damage especially to adolescents.

- The crash risk studies chosen are ones on the low side, yielding relative risks (RR) or odds ratios (OR) in the 1.2-1.4 range, whereas the accepted average level is closer to two. If the average OR is 2.0, for example, that means that some subjects are below that number, and some are above that number. Because of the way in which the OR studies were done, measuring crash risk for drivers with a presence of THC, even though they may not have been impaired significantly, the number of study subjects is dominated by drivers with an OR of 1.1 or less. Those who are truly impaired have to have an OR well above 2.0 for the average to be 2.0. Drummer, for example, showed OR of 10.0 for those with very high THC levels and I have found RR to be in the 7-10 range for drivers convicted of DUI where THC was the only drug found (unpublished data currently in peer review). There is frequently a tendency to discount the danger of THC impairment, but we need to recognize that impairment is a dose-related phenomenon. The higher the dose, the greater the impairment. And that holds for both occasional users as well as for addicts who have developed some level of tolerance. So someone on a high dose of THC will be more dangerous than someone on a low dose of alcohol. The fact is that these people are impaired. They should not be permitted to put others at risk. A 9 mm bullet is half as deadly as a .45 caliber bullet and a .22 caliber bullet is half as deadly as a 9mm bullet. That doesn't mean we should shoot people with .22 caliber bullets because it's safe to do so.

- Page 5 cites studies by Cook and Santaella-Tenorio saying that there is no increase in traffic fatalities when medical marijuana is permitted. See the attached unpublished letter to the American Journal of Public Health criticizing the Santaella-Tenorio study. The journal has a habit of publishing pro-marijuana studies, unfortunately. And they declined to publish other critiques.

- Our last comment refers to their statement that medical users develop a tolerance to the impairing effects of THC. They are very careful to state this correctly, *"development of tolerance to impairing effects in patients could be expected to partially, but not fully, diminish potential effects on driving skills compared with an occasional recreational cannabis consumer taking a similar dose."* But his entirely misses the point that when tolerance takes effect, the user simply increases the dose. This is recognized in two places (P2 and P8) in the manuscript.

Research published in Accident Analysis and Prevention in 2021, investigated driving impairment due to cannabis use, by comparing occasional and regular users of the substance. The issue of 'tolerance' was a key focus in this work, as proponents of cannabis use have argued, subjectively, that driving impairment is lesser with those who are regular or chronic users, as they have developed a tolerance for it's affect and therefore are less likely to be involved in traffic accidents due to intoxication. (As we mention at different times, the same argument for the alcohol using driver does not give them a 'pass' from prosecution).

The Lambert Initiative – Cannabis Industry Article

In a recent release from the University of Sydney's Lambert Initiative for Cannabinoid Therapeutics decided that, according to the article … "that blood and oral fluid THC concentrations are relatively poor or inconsistent indicators of cannabis-induced impairment." Professor MacGregor went on to reiterate the *'perception of impairment' argument in the following statement in the article*

> *"A cannabis-inexperienced person can ingest a large oral dose of THC and be completely unfit to drive yet register extremely low blood and oral fluid THC concentrations. On the other hand, an experienced cannabis user, might smoke a joint, show very high THC concentrations, but show little if any impairment.*
>
> *"We clearly need more reliable ways of identifying cannabis-impairment on the roads and the workplace. This is a particularly pressing problem for the rapidly increasing number of patients in Australia who are using legal medicinal cannabis yet are prohibited from driving"*

This circles back to the to an retrograde argument by many 'seasoned drinkers' posited in opposition to 'breathalysers' that their ability to drive was barely influence by their blood alcohol limit. Many tragic examples exist of people, who could arguably be legally 'dead' with Blood Alcohol Limits of over 3.4, actually driving with only little 'impairment'.

A quick analysis of the Lambert Initiative article THC in blood and saliva are poor measures of cannabis impairment - The University of Sydney brings the following concerns to the fore.

Issue A:

"This study was funded by the Lambert Initiative for Cannabinoid Therapeutics." "Acknowledgements This research was not funded by a **specific** grant from any funding agency in the public, commercial, or not-for-profit sectors. However, D. M., R.C.K. and I.S.M. receive salary support from the Lambert Initiative for Cannabinoid Therapeutics, a philanthropically funded centre **for medicinal cannabis research** at the University of Sydney." The 'conflict of interest' is a best dubious.

- there is an EXTREME risk for bias due to financial interest in the product being investigated by those who are funded by organizations responsible for researching "the product"
- prolonging research leads to prolonged employment/salary for these "researchers"

Issue B:

They need to explain why the Hartman study Cannabis effects on driving lateral control with and without alcohol - PubMed (nih.gov) showed that 13.1 ng/ml THC created the same amount of weaving as 0.08 BAC. The hydrophobic THC molecule rapidly leaves hydrophilic blood since THC distributes readily into the brain - fatty tissue. The study shows the very low (2- 4 ng/ml THC levels within 1 - 2 hrs). Here was their ***admission* that this study did** perform appropriate assessments for impairment ***and*** the timely monitoring of THC levels/biomarkers: *"Very few studies have measured the effects of THC on SDLP in combination with a relevant (and appropriately timed) biomarker (Arkell et al., 2019a; Brands et al., 2019; Micallef et al., 2018; Hartman et al., 2015; Ronen et al., 2010; Fares et al., 2021). Further research using simulated and on-road driving methods (or other measures that have a known relationship with driving performance) would permit better characterisation of the relationships between THC-related biomarkers and driving impairment."*

- Yet the authors of the article simply call for ***more*** research rather than adopt their findings (a very common tactic used by the marijuana industry to allow more addiction and marijuana sales to occur - **not** to "adopt these results until additional research changes the conclusions")

Issue C:

The authors treat driving as a right - it is not. It is a privilege. There are many other options that ***do not*** involve having someone put the entire public at risk of losing their life (or being injured) from an impaired driver. Note: they ***do not*** make claims that marijuana is ***never*** impairing of one's ability to drive.

Issue D:

Zero tolerance or using the **US Depart of Transport (DOT)** standard of urine marijuana metabolites is ***a much safer*** alternative for the public **safety** (rather than allow **one** marijuana impaired driver to **kill** another human, or even themselves). The authors need to answer how many innocents can be injured or harmed to allow **one** marijuana-impaired driver to operate a vehicle. The US DOT uses urine levels for ***all drugs except*** alcohol - since they recognized a long time ago that blood level limits for these hydrophobic substances are NOT accurately measured in the blood. The same method (urine levels) should be used by all governments when looking at these impairing substances.

Issue E:

The authors use the delay is THC distribution phase (seen primarily with an orally administered intoxication) to make this claim *"Likewise, drivers who are impaired immediately following cannabis use may not register as such."* - oral peak THC blood levels may take hours (2-3 hrs) to attain - they acknowledge this distribution time when they state "A cannabis-inexperienced person can ingest a **large oral dose** of THC and be completely unfit to drive yet register extremely low blood and oral fluid THC concentrations"

- they go on to promote a claim *"A cannabis-inexperienced person can ingest a large oral dose of THC and be completely unfit to drive yet register extremely low blood and oral fluid THC concentrations"* but this is Assuming that testing will be made HOURS after a crash. A study that I was involved (attached) with shows that it takes usually 2 hours in a fatal crash to draw blood or those in which someone was injured (but not killed) - due to delays in processing the scene in these cases due to the mayhem involved.

Issue F:

We also note some concerns around the disingenuous use of words in this statement - *"blood and oral fluid THC concentrations are relatively poor or inconsistent indicators of cannabis-induced impairment."*

- it does ***not*** say that "blood and oral fluid THC concentrations cannot ever be used as indicators of cannabis-induced impairment."

- due to the justice system being warped into being more concerned about the defendant and ***not*** The victim - this claim is being warped even when blood levels are extremely high - 40 ng/ml THC 45 minutes after the crash - Judgement withheld on Brady Robertson's sobriety during deadly crash as constitutional challenge around driving laws & cannabis use continues | The Pointer

Issue G:

Re' "No significant relationship between blood THC concentration and driving performance was observed for 'regular' (weekly or more often) cannabis users."

- the reason why blood levels are inappropriate for chronic users - is that they may be chronically impaired and

they **do** have residual THC remaining in the fatty brain tissue which is coming out and being turned into THC metabolites (including the ***higher*** intoxicating THC-OH molecule than the parent THC molecule).

- they have conveniently NOT included the Doroudgar study which showed chronic impairment in chronic users

Issue H:

The article **never** addresses the issue of multi-substance impaired driving - which is on also on the rise, with cannabis and alcohol use a common pairing. There is **no way** to determine the numerous amounts of combinations to determine accurate impairing blood (or oral levels) of each substance when combining. ***The Only safe measure is ZERO tolerance.***

The paper titled ***Simulated driving performance among daily and occasional cannabis users*** revealed the following:

Highlights:

- *Occasional users had a similar drug effect as daily users but lower blood THC.*
- *Smoked cannabis led to an increase in SDLP among daily and occasional users.*
- *Only daily cannabis users drove slower after smoking cannabis (15–30% THC).*

The **Objective** of the paper was *'Daily cannabis users develop tolerance to some drug effects, but the extent which this diminishes driving impairment is uncertain. This study compared the impact of acute cannabis use on driving performance in occasional and daily cannabis users using a driving simulator.'*

The conclusion of the research revealed that 'tolerance' did not attribute to safer or more competent driving *.. We observed a decrement in driving performance assessed by standard deviation of lateral placement (SDLP) after acute cannabis smoking that was statistically significant only in the occasional users in comparison to the non-users. Direct contrasts between the occasional users and daily users in SDLP were not statistically significant. Daily users drove slower after cannabis use as compared to the occasional use group and non-users. The study results do no conclusively establish that occasional users exhibit more driving impairment than daily users.*

(Source: *Science Direct Accident Analysis & Prevention Volume 160, September 2021, https://doi.org/10.1016/j.aap.2021.106326***)**

A 2018 paper, one of the earliest on the issue of THC impact on driving abilities was published in the USA National Institute of Health's National Library of Medicine concluded the following:

> ## The effects of cannabis intoxication on motor vehicle collision revisited and revised
>
> ***Conclusions:*** *Acute cannabis intoxication is associated with a statistically significant increase in motor vehicle crash risk. The increase is of low to medium magnitude. Remaining selection effects in the studies used may limit causal interpretation of the pooled estimates.*
>
> **(Source:** *PubMed.Gov https://pubmed.ncbi.nlm.nih.gov/26878835/***)**

A review of this research and conclusions raised some concerns, not least depth-integrity of research. Rogeberg and Elvik attempted to determine the Odds Ratio (OR) of being in a crash after using THC. It did so using a meta-analysis that is akin to a weighted average of research papers published by others. Two of those studies were ones done by Li and by Romano.

Even both Li and Romano used identical FARS data for subjects and identical National Survey data for controls for overlapping time periods, they reached opposite conclusions. Li reported the OR for a fatal crash associated with marijuana to be 1.83 (95% CI 1.39, 2.39). Romano reported the OR to be 0.92 (95% CI 0.6, 1.40), essentially saying that use of marijuana exerted a crash protective effect on the user. Since both researchers used the same data to arrive at different conclusions, Romano published another study, examining why that happened. He concluded that there were biases in the selection of FARS data to be included, more so in his paper than in Li's paper. When he removed those biases from both papers, he ended up with results similar to Li's. You can see his analysis at Romano E, Torres-Saavedra P, Voas RB, Lacey JH. ***Marijuana and the Risk of Fatal Car Crashes: What Can We Learn from FARS and NRS Data?*** *J Primary Prevent (2017) 38:315-328*.

But perhaps Romano's most important conclusion was"...the FARS database should neither be used to examine trends in drug use nor to obtain precise risk estimates."

The 21 studies used in Rogeberg's meta-analysis reported Odds Ratios ranging from 0.22 to 13.40. He weighted some as low as 0.46% of the total and others as high as 10.68% of the total. The flawed NHTSA report and three different FARS-based reports received a combined weight of 35.41% in Rogeberg's meta-analysis. Rogeberg included Romano's 0.92 OR in his meta-analysis, rather than the higher one that Romano admitted to later. For the NHTSA report, Rogeberg chose to use the later OR 1.0 result, rather than the first-released OR 1.05. He should have consistently chosen either the first-published result by those authors, or the corrected result, preferably the latter.

Rogeberg and Elvik's study used a biased selection of previously published work that included discredited NHTSA and FARS reports, and weighted those discredited reports more highly than they deserved.

The Colorado Department of Health and Environment has empanelled a group of "experts" to review the literature to answer that question and many others pertaining to THC. They concluded that there is substantial evidence that *"waiting at least 6 hours after smoking less than 18 mg allows driving impairment to resolve or nearly resolve."* However, a typical joint has 400 mg of flower. If the THC concentration is a very modest 15%, that provides 60 mg of THC, and if the bioavailability is 30% (due to pyrolysis, side-stream loss, etc) you'll get an 18 mg dose administered. So, they 'resolve' time is at the very least out by some factor for ever the low use cannabis smoker.

Conclusion

Both the limited research and the clear unpredictability of Cannabis intoxication, along with the idiosyncratic nature of THC impact on individual biological units, should be enough to move forward, only with extreme caution.

As this product has very limited evidence-based impact on health issues, but a considerable placebo effect, it's therapeutic outcomes in no way come close to the accompanying risks of driving whilst medicating.

Our Nation has worked long and hard to arrest and 'wind in' drink driving and the incredible toll it has taken on our communities. To add any mechanism to legislation that allows or even permits any other version of intoxicated use over a vehicle is at best incredibly unwise – at worst culpable.

The campaign in play at the moment to have Cannabis is 'medicinal' form excised from the legislation to enable the users of such formulations to consume this psychotropic substance and drive with impunity is ill-advised at best. It is our conclusion that enabling people who use cannabis to drive – even as 'medicine' – is not on the best interest of public safety.

Research Team @ Dalgarno Institute

Appendix

Cannabis & Driving - THC, How Much is Too Much?

High Truths on Drugs and Addiction. Edward Wood, Founder and President of DUID Victim Voices. Marijuana drugged driving.

Alcohol-Marijuana-and-Driving-21-3.pdf (drugfreekidscanada.org)

AJGP report (The Royal Australian College of General Practitioners 2021):
The AJGP report relies on a badly flawed and previously referenced above pair of studies from Rogeberg and Elvik that the risk of crash from cannabis-positive drivers is a mere 1.1-1.4. We have some concerns as to why the NHTSA report should be ignored. The Brubacher report had an average time of 101 minutes from the crash before taking a blood sample for testing. Since it has been shown that the peak THC blood concentration can decline an average of 76% within the first 25 minutes after starting to smoke a joint, the Brubacher report is pretty meaningless.

NIDA report

NIDA report referenced the following:

- Two large European studies that found drivers with THC in their blood were roughly twice as likely to be culpable for a fatal crash than sober drivers,
- Several meta-analysis showed a significant crash risk - double or more, and
- A NHTSA study failed to find a significant crash risk due to cannabis.

You need to understand the following:

Impairment, whether it be from alcohol, THC, or some other drug, is a function of four things:

- ✓ The dose consumed,
- ✓ The mode of consumption,
- ✓ The time since consumption,
- ✓ Biological variables

When determining the effect of alcohol on crash risk, virtually all studies do so by measuring crash risk as a function of the blood level of alcohol in the driver (or breath level, converted to BAC equivalents). That can be done because there is a very high correlation between BAC and crash risk.

When determining the effect of THC on crash risk, researchers typically study crash risk as a function of a dichotomous independent variable (presence or absence of THC). They do this because there is absolutely no correlation between THC blood levels and the level of impairment.

But in measuring crash risk as a function of the presence or absence of THC, the pool of drivers with THC being present is not homogeneous. Some are very highly impaired (crash risk of 10 times or more) as well as those who are functionally unimpaired (THC remains in the blood, even though their acute impairment has subsided or the dose was too small to create impairment or...).

Consequently, the results of the European studies and the meta-analyses are of limited value. They aren't to be discarded, but their value is limited. They do NOT conclude that someone impaired by THC is only twice as likely to be culpable.

The pool, for example, could consist of 20 drivers, all positive for THC. 10 were unimpaired, 8 were modestly impaired with an Odds Ratio of 2.0, similar to someone with a BAC of .08 gm/dL, 2 were more seriously impaired with an Odds Ratio of 10.0. On average, the Odds Ratio would be 2.3. But that doesn't represent the crash risk of any of the 20 drivers in the pool.

Data published by Colorado's Office of Research and Statistics, for example, allows us to assess the crash risk of drivers who were convicted of impaired driving when THC was the only intoxicant found in blood. Since they were convicted of DUI, one should expect that they were likely more impaired than someone who simply had THC on board. That pool of drivers had a 7.1% incidence of crash, compared with 24.8% incidence of crash for drivers convicted of impairment by alcohol only. The alcohol-only pool of drivers had a mean and median BAC of .166 and .160 respectively. Drivers with that much alcohol on board typically have a crash risk of 25-30, depending on which research report you wish to rely upon. Clearly, the THC-impaired drivers who were convicted of impaired driving had a far higher risk than 2.0. These data are still being reviewed for publication.

I've appended that ORS report as well.

The last study by NHTSA is problematic. It is commonly referenced by the pot lobby to claim the study found there was no correlation between THC use and crash risk. That's incorrect. In fact, the study failed to find a statistically significant relationship between crash risk and the use of any drug (including methamphetamine, heroin, etc.) except for alcohol. But an absence of evidence is not evidence of absence. It's like when you can't find your car keys, it's not because the keys no longer exist. You just didn't look where they do exist.

In the NHTSA case, the results are because the study was never designed to detect any such correlation in the first place. There were four major flaws in the study, including reliance on volunteers only. It's not clear why someone who knew they were impaired would volunteer for the study, but we know that some did, since they did find a correlation with crash risk and alcohol.

So the NHTSA study should simply be ignored. It was a waste of $6 million in taxpayers' money. Even worse, it muddies the waters about drug impairment.

University of Sydney Arkell study

The U of Sydney press release of Arkell's study was a bit misleading. The study consisted of 14 subjects with a history of light cannabis use. The intent of the study was to determine if a 50:50 mix of THC:CBD had a less impairing effect than THC alone. Some have speculated that CBD would reduce the impairing effects of THC since it does lower some of the effects of THC. It didn't reduce impairment. The study used a very low vaporized dose of THC – 125 mg of 11% THC concentration. Typical doses are 300-500 mg with a minimum of 15% THC concentration flower. So, any conclusions about impairment lasting 4 hours should be limited to the conditions studied.

Source	Key points / Key words	Dalgarno Institute Summary
Signed letter dated 22 October 2021 addressed to one of our network affiliates, Drug Free Australia **From:** **MINISTER FOR POLICE; ROAD SAFETY; DEFE CE INDUSTRY; VETERANS ISSUES** - WA Minister for Road Safety the Hon. Paul Papalia **Attached - 1. WA - Letter Office of the Hon. Paul Papalia CSC MLA_20211022**	..."The McGowan Government have been a **consistent supporter** of **more progressive laws** for **access** to **medicinal cannabis products. Since winning government in 2017**, the government has cut red tape and **made it easier** for patients to **access medicinal cannabis, invested** in **developing local medicinal cannabis cultivation** and **processing** opportunities, and **continues** to **advocate** for the **inclusion** of **medicinal cannabis products** on the **Commonwealth Pharmaceutical Benefits Scheme**. The **scientific evidence is clear** that **consumption** of **cannabis** or **cannabis derived products containing** the **psycho-active compound THC**, whether **consumed lawfully or unlawfully**, can **affect** a person s **ability** to **safely drive** a vehicle. It is also **clear** that a **high proportion** of **recreational cannabis users** are **young people**, who are **already over-represented in road crash and road trauma** statistics. I can **assure you** that any **reforms to WA's drug driving laws** will be **very carefully considered, based** on **solid scientific evidence**, and done **in a manner** that **does not compromise road safety outcomes.** The Road Safety Commission is **monitoring developments in both cannabis testing technologies and related drug driving laws across Australia and overseas. Until there are substantial developments in testing** for **cannabis related impairment** at the **roadside**, we will **continue** to take a cautious approach and maintain the **current legislative framework** that does **not allow a person to drive within detectable traces of the drug in their system.**	Since coming into office, McGowan government has consistently supported more laws by cutting ...*"red tape.."* to enable easier access to medicinal cannabis (MC) products for patients. The government has invested in local development and cultivation of MC and is an advocate for the inclusion of MC products on the Commonwealth Pharma Benefits Scheme. The government is aware based on scientific evidence that consumption of cannabis or cannabis derived products consumed lawfully of unlawfully can affect one's ability to safely drive a vehicle and that a high proportion of recreational drug use is by young people *.."who are already over-represented in road crash and road trauma statistics"..,* however; the government provides an assurance that any changes to drug driving laws will be carefully considered, done in a manner that will not compromise road safety based on scientific evidence. **Important take away** The government is providing an assurance that any reforms to drug driving laws will be based on solid scientific evidence and in a manner that does not compromise road safety and the Road Safety Commission is monitoring cannabis testing technologies and related drug laws across Australia and overseas. Until there is substantial testing for drug related-impaired driving the government will continue to take a cautious approach maintaining the current legislative framework in relation to drug driving.

Govt of WA Road Safety Commission-annual-crash-statistics-2014 https://www.wa.gov.au/system/files/2021-07/annual-crash-statistics-2014.pdf	..."**Keywords** Road crash statistics, **Fatal crashes, Blood alcohol concentration, Drink driving, Drugs, Fatality,** Helmet use, Injury, Restraint use, Road environment, Metropolitan area, Regional area, Seat belt, Speeding, Vehicle type, Western Australia. **..."Minister's foreword** ..."Unfortunately, **speeding, drug and alcohol use were contributing factors** in many of those crashes. ..."While the rate of fatalities on Western Australian roads has dropped 25 per cent since 2008... ...we lost 182 people on our roads this year ...each a loved one to somebody. As the Minister for Road Safety, I acknowledge that **the positive trend in the statistics will be of no comfort to the families of those taken** so suddenly. **...Sadly, alcohol, drugs** and speeding **remain strong contributing factors** with speeding a factor in around a third and **alcohol around a fifth of all fatal crashes. Illegal drugs** were also a factor in a **fifth of this year's fatalities.** We know that **25 per cent** of the **motorcyclists killed** this year **had illegal drugs in their system.** This figure was 19 per cent for drivers."... **..."Key road crash facts for 2014** **... Alcohol** • Almost one fifth (17%) of police attended fatal crashes involved at least one driver/rider with a **blood alcohol content of 0.05g/100ml or above. Illegal drugs** • Around a fifth of police attended fatalities had an illegal drug detected in their system. • Drivers and motorcyclists	**The W.A. Government Thus Far** Historically the WA government has published road crash data yearly, which included road crashes (including fatalities) involving drivers with alcohol or illegal mind-altering substances (drugs) detected in their system. As per the WA governments 2014 report the information on road crash fatalities with drugs detected in their system was provided by the Chemistry Centre of Western Australia - Francois Oosthuizen https://www.wa.gov.au/system/files/2021-07/annual-crash-statistics-2014.pdf As per the WA governments 2015 report the information on road crash fatalities with drugs detected in their system was provided by the Chemistry Centre of Western Australia https://www.wa.gov.au/system/files/2021-07/annual-crash-statistics-2015.pdf 2016 summary Preliminary fatal and critical injuries on Western Australian roads report **no mention of drugs** https://www.wa.gov.au/system/files/2021-07/annual-prelim-crash-statistics-2016.pdf 2017 report (PDF) **the word "drug" appears in the report only once** Foreword *.." We have **restored the primacy of the work** of the Road Safety Council, **put in place new laws to protect** cyclists and roadside emergency workers, **and are strengthening penalties for** drink and **drug drivers**, including strict bans for recidivist offenders.."* https://www.wa.gov.au/system/files/2021-07/annual-prelim-crash-statistics-2017.pdf

	comprised the **majority** of **road crash fatalities** with **illegal drugs in their system (38% and 32%).** **..."Information on road crash fatalities with drugs detected in their system was provided by the Chemistry Centre of Western Australia.** Acknowledgments The Road Safety Commission would like to thank the following people and organisations for their assistance in providing data: **Chemistry Centre of Western Australia - Francois Oosthuizen** Western Australia Department of Health - Vikki Mirosevich, Michael Anagno Main Roads Western Australia - Thandar Lim Western Australian Police - Stephen Temby	WA Road Fatalities and Serious Injuries 2020 WA Government Road Safety Commission **No mention of drugs** https://www.wa.gov.au/system/files/2021-10/WA%20Road%20Fatalities%20and%20Serious%20Injuries%202020.PDF **Note, below are the data resources on WA govt website:** Hard to make sense of reporting to be honest **- all over the place!**
Govt of WA Road Safety Comminssion_annual-crash-statistics-2015 https://www.wa.gov.au/system/files/2021-07/annual-crash-statistics-2015.pdf	**Keywords** Road crash statistics, Fatal crashes, **Alcohol, Drink driving, Drugs,** Fatality, Helmet use, Injury, Restraint use, Road environment, Metropolitan area, Regional area, Seat belt, Speeding, Vehicle type, Western Australia **Minister's foreword:** ..."Western Australia's **preliminary road toll in 2015** was the **equal lowest since records began in 1961.** ...It stands at **161 deaths in Western Australia**, which is a decrease of 21 fatalities on the 2014 figure. **While a downward trend is statistically pleasing**, **there's no doubt that these deaths are tragic for families, friends and communities** across our state. **It is a concern that the fatality rate in WA** (6.2 per 100,000 people) **is still behind the national average** (5.1 per	

100,000 people). **Despite mass media campaigns seeking to change drivers' behaviour,** poor driving continued to feature in this year's road crash fatalities. **37% of those killed were found to have an illegal drug in their system and the number of fatalities in alcohol related crashes was 25%,** about the same time as last year (26%).

Key road crash facts for 2015

Alcohol - 25% of fatalities were in an 'alcohol related crashes'. This was similar to the percentage for the previous year (26%).

Illegal drugs - 37% of fatalities had an **illegal drug in their system.**

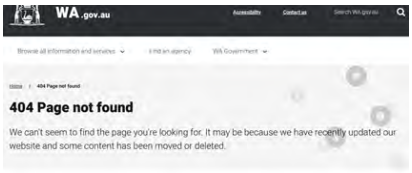
1.1. Data sources and acknowledgements

Data used in this report was sourced from a variety of government and research organisations.

Main Roads Western Australia (MRWA) Most of the statistics presented here were extracted from data on police-reported road crashes. These data were obtained from the IRIS maintained by MRWA.

The dataset used was extracted from IRIS on 22 June 2016 and changes made to the dataset after this date are not reflected in this report.

Australian Bureau of Statistics Fatality rates were calculated from the police-reported data using estimated resident population, vehicle registrations and estimated kilometres travelled data obtained from the Australian Bureau of Statistics (ABS). Bureau of Infrastructure, Transport Regional Economics Comparisons across Australian states and territories were derived from data provided by the Bureau of Infrastructure, Transport and Regional Economics (BITRE). **Western Australia Police Information** on whether speed, **alcohol,**

	in attention or fatigue were contributors to a crash was obtained from the Road Crash Casualty Database maintained by Western Australia Police. This database records information on crashes that were attended by police. **Chemistry Centre of Western Australia Information on road crash fatalities with drugs detected in their system was provided by the Chemistry Centre of Western Australia.** Reported road crashes in Western Australia 2015 | page 9 Western Australian Department of Transport The number of Western Australian motor driver licences on record for each year from 1974 onwards is provided in the Appendix A(ii). This information was obtained from the Western Australian Department of Transport.	
WA_annual-prelim-crash-statistics-2017 https://www.wa.gov.au/system/files/2021-07/annual-prelim-crash-statistics-2017.pdf	**...”Preliminary summary of fatalities in 2017** ... **35 deaths** were due to **alcohol-related crashes.** **NO MENTION of illegal drug related crashes.**	
WA Road Fatalities and Serious Injuries 2020 https://www.wa.gov.au/system/files/2021-10/WA%20Road%20Fatalities%20and%20Serious%20Injuries%202020.PDF Google search ***Why does the western Australian government no longer report on drug related road crashes and fatalities?***  **Click on what comes up *....Illicit drug and driving: and investigation of fatalities and traffic...”*** **And you get the adjacent response.**	...” Alcohol involvement is not available due to changes in data sharing arrangements **NO MENTION of illegal drug related crashes** **Words, DRUG or DRUGS <u>do not</u> appear at all!**  	2020 *..”Alcohol involvement is not available due to changes in data sharing arrangements”..*

Conclusion:

Informing the Public or the Public informing?

In April 2019 headlines trumpeted that, Medicinal cannabis supply to reach new high with first-ever pot factory in NSW, so goes all such cannabis focused by lines these days, unhelpfully painting cannabis in a 'health' light. Of course, click bait phrases are engaged for further reading. However, it was the announcement of a research facility to develop our own Cannabis Medicines – even though GW Pharma in England was just putting their decade long clinically trialled epilepsy medicine into the market.

As of this August the facility is producing.

One tabloid outlet, Channel 7's news program The Latest, made a story of this on the evening of 20th of August. The segment went, as most tabloid news goes, with shallow data and then lathers of personal, and clearly uniformed, commentary.

The host Michael Usher was quick to recount his experience of the 'miraculous' transformation he saw in a fitting child when plied with one of Colorado's 'home brew' cannabis tinctures (most likely the now infamous Charlottes Web). Then after further endorsing comments about people illegally importing the substance in desperation, was critical of authority's perceived lack of action.

Then he crossed to two 'social commentators' – not cannabis clinicians, or medical experts – just 'influencers' who then waxed lyrical about the need to not let 'big pharma' hijack this new wonder drug, and to top off this – Big Marijuana Playbook' pitch – the statement came; "We seem to forget that parents know more often that not what is best for their children."

There you have it! What more is needed?

Tabloid news endorsement – personal anecdote, and a parent grieving heart, is all we need for a product to be declared a 'medicine' and then self-prescribed for whatever ailment you believe it will help with. However, any 'science' that disagrees with this new manufactured consensus, is at best ignored, as 'my gut feeling' is far better than evidence-based clinical research.

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In recent announcements too, Victoria and Tasmania are also to have/have new Cannabis Facilities of some kind – As nationally, other 'industry' permission is being granted for this apparent 'science skipping' policy progress.

The headlines pealed forth that Australia's biggest medicinal cannabis plant is to be built in Melbourne! (unintentional and amusing double entendre aside) It always remains a little tragic when our long-submitted warnings about 'thin end of wedge' policy creep emerges.

Of course, the relentless propaganda (disguised as 'health marketing') on the purported panacea properties of pot, combines with a doubling down on all efforts in the 'we have to make this weed thing work for us somehow', mentality lend themselves to an ever blinding (at the very least, myopic) bias that largely ignores not only minor 'side effects' of this unpredictable product, but the real and often irreparable long term harms.

Sure, at the moment, other than the pro-pot pushers, and their 'addiction for profit' financiers, no credible voice in the scientific and policy space is suggesting cannabis should attain the protected status of alcohol (not yet, anyway) but the every noisy clamor

to validate this product as 'medicine' is enabling creative, if not fallacious means, to give it even a spurious legitimacy in those important echelons.

State governments are trying to be the first to get big farms and processing plants up, believing a 'boon' in the faux pharmaceutical space will emerge – it hasn't anywhere else – but that isn't stopping the equity rescue pursuit of bad investment.

The most disturbing thing in this process, is that legitimate, thorough, and best practice clinical trials have been, if not negated, then massaged to fit a new approval mechanism.

Not a clear authoritative pre and proscription model – one that only thorough and exhaustive trials can produce – rather a 'recommendation', based on analysis of some evidence-based data, and a plethora of anecdotal testimonials.

It appears on the weight of the (often celebrity endorsed and pop-culture lauded) stories, long term impacts on consumers is of little regard if the 'felt need' is being met, even if only subjectively, and if only for the interim.

The message being sent to the most vulnerable of those consumers – Children is, 'as long as any symptom is managed, it matters little what may come next.'

The Potential Thalidomide Parallel and Big Tobacco Playbook

What is of grave concern is that the Thalidomide disaster that Australia could have avoided if it followed the USA lead back in the 1960's, is consistently being ignored in the reviewing and research space.

The United States FDA (Food & Drug Administration) never authorized this experimental drug for 'morning sickness' and consequently saw less than 50 babies born with deformities, whilst in Australia, thousands of casualties were had, whom all still live with disabilities today. Even the long fought for settlement for damages changed little to the permanent and ignored (or played down harms) of this purported panacea for morning sickness.

The caution exercised by the US Federal government is being worn thin by the predatorial nature of Pot promotors, consequently concerns about cannabis being a potential new Thalidomide threat are being largely and, it would appear, willfully ignored.

What vexes even more, is the same willful ignorance is being exercise around Big Marijuana's unabashed plagiarizing of the Big Tobacco marketing playbook. They are, arguably, following it to the letter, and somehow the authorities that took four decades to wake up to the con of the tobacco industry, are swallowing the pitch, hook, line, and sinker. It is as if they have never seen this before and in their newfound naivete are blithely embracing the strategy as fiscally wise and harm limited.

The Australian Push

In very recent weeks another license was granted. Medicinal cannabis producer and developer MGC Pharma (ASX: MXC) will see its logistics processes streamlined following the granting of a new import licence.

According to news reports... "Issued by the Australian Office of Drug Control (ODC) the import licence will enable MGC to directly import Schedule 4 (prescription only medicine) and Schedule 8 (controlled drugs) medicinal cannabis products into Australia from its European production facility."

This Canadian company, along with others have long looked to target Australia as a possible source of renewed revenue, as that nations burgeoning legalization experiment is failing to produce even a modicum of the cornucopian realities it promised and in current reality is just plain failing.

Perhaps the most concerning issue we see

emerging is, as we opened here, in the policy, practice and regulation space around this unpredictable and highly engineered product.

The Therapeutic Goods Administration (TGA) have, laudably, been reining in the misuse of another plant-based drug – opiates – as the harms from this misappropriated and misused pain manager exploded.

You would think this would act as a profound cautionary tale when it comes to releasing further 'medicines from nature', particularly the ones that have been so engineered that there is little resemblance to their natural state.

The fervency to unleash 'cannabis' as a medicine has been fueled by the relentless propaganda machine and the anecdotes, they engage to promote this product. Yet, the scientific bodies charged with ensuring not only efficacy, but safety – the AMA and TGA – seem (for the most part) to be bowing to public pressure and now we appear to be 'voting' on medicine, and that not via democratic political process, but 'social media consensus'.

The TGA 'Pass'?

Under the 'Accessing Unapproved Products' the TGA have listed the following 'caveat'.

Medical Cannabis Guidance Documents.

This new 'category' of allowance by the TGA, is not a recommendation, as no exhaustive clinical trial evidence exists to do so. Anecdotal and placebo effect data cannot pass for best practice science, that is why this new 'category' has been included and is it simply another step toward 'rescheduling' this drug?

Even the wording on the TGA site under 'Why are these Guidance Documents developed?' is indicting.

Many prescribers and dispensers know very little about medicinal cannabis because there has been little research on medicinal cannabis for many years and there is not much information on medicinal cannabis provided in most medicine and pharmacy courses.

Dr Kevin Sabet, former Drug Czar for three US Presidents, and Co-Founder and President of Smart Approaches to Marijuana (SAM) writes insightfully and compassionately in warning against rescheduling of Cannabis in any context, no less for the 'potential of medical use'.

So, back to the grand new industry announcements; they are investing millions to produce what exactly in Victoria (or elsewhere for that matter)?

- National inquiry into CBD producers is currently in play with no findings handed down,
- The Victorian Enquiry into Cannabis seeking submissions but will not commence review until September.
- TGA have only approved Cannabis based pharmaceuticals, Sativex ® and very recently Epidiolex®

So, what information does Cannvalate who are promoting pot product as if it was a clinically trialed medicine have that gives them confidence to invest so heavily?

- Is it because they already know they will be the key wholesaling enterprise so a 'Big Pharma brand' may start developing medicines, and such manufacturers have better access to primary product, without 'middle-men' and less potential for contamination/degradation in shipping processes? That certainly has a legitimate tone, but there is still no legislative pass to guarantee this, or this there?
- Are they aware that this new TGA 'rework' is just an echo of the U.S. Food and Drug Administration's lack of resources to act in accordance with best practice as the following reveals...

The FDA occasionally sends Warning Letters to online CBD dealers telling them to stop making unsupported medical claims for their products. For the most part, the dealers are quick to comply as their customer base is well-schooled in knowing precisely what it wants. Even here, however, there are legislative initiatives presently being considered that would redefine these products to exempt them from these FDA regulations.

If motivated to do so, the FDA could stop sales of CBD and THC. Pursuant to the FD&CA, when a substance is identified as a drug and approved as such by the FDA, all subsequent producers of the drug must obtain prior FDA approval before the drug can be marketed in food or medicine. Thus far, the FDA has shown little interest in enforcing this provision. Instead, the agency seems satisfied that warned CBD sellers have complied with the agency's letters and deleted unsupported medical claims, as ordered. One of the warned CBD dealers actually posted the FDA Warning Letter on its website and added a link to the agency's lab report to show customers just how high the CBD content was in its products.

Nominally, the DEA and Federal Bureau of Investigation (FBI) retain authority to enforce the marijuana and THC provisions of the CSA, but the actions of the 115th Congress in passing the Farm Bill weakened this authority and virtually eliminated it with respect to finished products containing hemp-based ingredients. When it comes to marijuana law enforcement, federal efforts are now focused mostly on cartel-related cross-border and maritime bulk smuggling operations. Oddly, success in reducing imported marijuana drives up consumer demand and increases the value of domestic supplies.

More so, we must ask,

- Who is 'prescribing' cannabis 'medicines' and in what formulation? If Sativex and Epidiolex, then fine, we don't need 'cannabis clinics' for this?
- Where are these patients coming from?
- How does one define the term 'patient' and as actual cannabis based medicines have no curative properties, and very limited symptom management value, what is this being 'prescribed for'?
- To repeat, the science is clear – cannabis as properly trialed pharmaceutical grade medicine is limited to two (perhaps three) current clinically trialed formulations – Sativex, Epidiolex and the rest is not medicine, at best is a poorly vetted 'supplement'.
- What are the unwitting public being led to believe and who is driving the decision making on this? Science, due process, and government? Or, are Anecdotes, celebrities and sentiment driving policy on medicine now?

Victorian Legislative Council called earlier this year for submissions from the public on how cannabis should be managed in Victoria, with a focus on what assisting Young People stay away from Cannabis may look like.

If one where skeptical – and we most certainly need to be with the momentum of the marijuana marketing machine driving perception, not fact or science – one could deduce this looks a lot like another preemptive ploy by the state government to back their unleashing of 'weed', as if it were a fait accompli? For instance, they maybe intimating that they have a campaign of 'protective elements' for young, whilst they enable the 'grown ups' to enjoy the weed, with impunity?

Of course, my cynicism could be entirely misplaced and no under handed blanket permission for cannabis use be given to the ever more 'cannibidazzled' public.

One thing will remain, and only time will bring our collective awareness to the inevitable and sad conclusion; increased use of this unvetted drug will only increase harms to individuals, families, communities, and the governments that foster this. That, as with Thalidomide and Tobacco, is a guarantee.

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For a genuine investigative journalistic examination of the Cannabis issue, both 'medicinal' and 'recreational', the exemplary report was **SBS Insight – Marijuana**. It is a must view for anyone serious about the health and well-being of citizens.

This public address by Health Worker **Scott Cagnon at Vermont 'Town Hall'** Meeting summarizes succinctly and accurately the chaos in policy that commercializing a psychotropic toxin will produce.

For those interested in understanding the 'underbelly' of the broken marijuana market and the egregious misuse of medical marketing then you must view the important documentary **Smokescreen**.

The Research and Communication Team,
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For more evidence-based research go to…

Cannabis, Policy & Your Community – What is Best Practice?

The International Academy on the Science & Impact of Cannabis – Library

Cannabis Resource Library

Smart Approaches to Marijuana

Monitoring Health Concerns Related to Marijuana in Colorado:
2016 Changes in Marijuana Use Patterns, Systematic Literature Review, and Possible Marijuana-Related Health Effects

Cannabis: The Health & Social Effects of Non-Medical Cannabis Use (W.H.O)

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