

A submission to The Senate enquiry into an “Amendment (removing Commonwealth Restrictions on Cannabis) Bill 2018 otherwise known as ‘a Bill for an Act to Remove Commonwealth restrictions on cannabis and for related purposes’.

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Dear Committee members,

Thank you for agreeing to receive this late submission as agreed over the phone before I recently left for a two-week study trip to Bangladesh. I appreciate the latitude and, as agreed, forward this submission soon after my return.

As I understand from the ‘Explanatory Memorandum’ circulated under the authority of the proposer of the Bill, Senator Leyonhjelm, the Bill would allow any state or territory to legalise and regulate the distribution of cannabis on the grounds that adults should be free to make their own choices as long as they do not harm others, that criminalization increases pressure on the judicial system and induces organized crime, and that legalization would both save and earn money for the government.

Whereas I can empathise with the above the arguments, respectfully, I would like to draw attention to the collateral damage associated with the adult freedom of choice: ‘others’ inevitably will be harmed, especially the unborn and adolescents, and the marginalized in society. Also, whereas the ‘Explanatory Notes’ provide an overview of fiscal advantage in terms of reduction of policing costs and increase of taxation, they do not mention the direct and indirect costs of increased cannabis consumption on mental and physical health. Lastly, whereas the Bill is declared to be ‘compatible with human rights because it protects the right of self-determination’ if enacted it will surely result in increased exposure of the unborn, children and adolescents to the adverse effects of cannabis and thus challenge ‘The Declaration of the Rights of the Child’ which declares ‘The child shall enjoy special protection and shall be given opportunities and facilities, by law and by other means to enable him to develop physically, mentally, morally, spiritually and socially in a healthy and normal manner...In the enactment of laws for this purpose, the best interests of the child shall be the paramount consideration.’

Why would the passage of the Bill not be in the best interests of Australian children?

1) Because exposure to cannabis will influence the balance of the child’s innate endocannabinoid (eCB) system.

Cannabinoids are manufactured in the human brain where they exert a wide spread, complex, fundamental influence on brain development and function¹. Cannabinoids are also manufactured in certain plants from which they can be extracted and imbibed for euphoric and other effects. Extractions from plants are not ‘pure’ and predictable but represent a variable mixture of some 400 chemical compounds including cannabinoids, terpenoids and flavonoids that produce individual and interacting effects². 9-Tetrahydrocannabinol (THC) is the principle psychoactive component but its proportion of any extract varies with the breeding of the plant as well as with such local factors as the position of the bud in the plant, the season, humidity, temperature and fertilizer. As well as THC there are some 70 other

cannabinoids with their individual effect on the brain, and their modifying effects on the action of THC. Cannabidiol, for example, may counter the psychomimetic effects of THC³. The effects of the innate endocannabinoid system within the brain are closely controlled with effects regulated by release of minute amounts of particular components and their metabolites which, in sequence with other compounds, function for brief, controlled periods of time according to the needs of the body.

Exogenous Cannabis is lipid soluble and, therefore, crosses the blood-brain barrier to infuse itself in large, interacting, variable quantities and qualities into the intricacies of the innate system. Not only is the innate system subjected to large volumes of various types, the length of exposure to the foreign compounds will extend much longer than the natural because of the prolonged ½ life of their metabolism. Given the essential role of endocannabinoids in neurodevelopment, it is not surprising that laboratory studies confirm the susceptibility of the growing brain to adverse effects of intrusive exogenous cannabis.⁴⁵

2) Because the lipid solubility of cannabis components permits them to cross the placental from mother to unborn baby.

As well as influencing brain development, in ways not yet fully understood, the endocannabinoid (eCB) system also plays a fundamental role in reproduction itself. Exogenous cannabis has the potential to disrupt implantation, placentation and the development of the unborn in various ways⁶.

(a) **Length of gestation.** Studies with Australian authors have concluded ‘increasing use of marijuana among young women of reproductive age is a major public health concern’, being a ‘significant risk factor’ for premature birth if smoked before 20/52 of gestation, and for restricted foetal growth if smoked for longer⁷. Not surprisingly, the greater the consumption, the more premature the birth: taking cannabis 100 times in the three months before 20 weeks of gestation was likely to induce labour by 36-37 weeks of gestation, even if smoking had ceased by that 20 weeks. Considering the Australian component of this international research, the authors concluded ‘almost 12% of SPTB (spontaneous preterm birth) could be attributable to maternal marijuana use’.

Other studies have not confirmed this serious problem: for example, reports published in 1995⁸, 2005⁹ and 2010¹⁰ did not find an excess of premature labour, but most of such early studies were undertaken before legalization of marijuana in the United States and, therefore, maternal acknowledgement of consumption is likely to have been under-reported. Furthermore, in those earlier years, the strength of street cannabis was markedly lower.

Recent laboratory research on mice confirms ‘smoking marijuana during pregnancy even at low doses can be embryotoxic and fetotoxic’¹¹. The study revealed that pregnant mice exposed to ‘five minutes of daily (low dose) exposure’ produced pups with a mean reduction of 9.9% in body weight, associated with significant reduction in weight of brain, lungs and thymus. The placenta was hypertrophied and the ratio of its weight with that of the foetus was markedly increased, suggesting failure in its ability to transfer adequate nutrition to the foetus.

Reviews highlight the importance of eCB to the process of implantation and placentation, and thus the overall development of the baby¹²¹³ and warn that exogenous cannabis may interfere with the process. Indeed, the levels of endocannabinoids should decrease as

pregnancy progresses: lower levels are necessary for normal progression of pregnancy¹⁴. Smoking marijuana leads to sustained elevation in the level of placental and foetal systems and runs the risk of causing miscarriage and growth restriction.

(b) **Formation of the baby.** Components of the innate endocannabinoid system have been identified in the foetus from as early as 16-22 days from conception¹⁵. They are known to have a role in development of the **central nervous system** and as the fundamental structure of the brain is being laid down at this stage, it is not surprising a 'significantly increased risk for anencephaly' has been described after exposure at that time¹⁶. In this tragedy, the brain does not develop, and the cranium remains a vacuum space.

After involvement in the laying down of the basic template of the central nervous system, the eCB goes on to 'shape neuronal circuitry in the developing foetus as well as modulating development of various neurotransmitter systems...'^{17,18} Exogenous cannabis may be expected to 'target the cannabinoid receptor...disrupting migration, differentiation and synaptic communication in the developing neurotransmitter system'^{19,20} thus explaining 'the neurobehavioural deficiencies...observed in newborns exposed to marijuana'²¹, and neurodevelopmental problems in growing children and adolescents.

Whether exposure to cannabis induces defects in other foetal structures has been debated. A number of studies have not found any major consequences, but most were conducted before legalization of marijuana and, therefore, rely on maternal reporting of consumption which has been considered inaccurate. Use of Bayesian models to assess the effects of such under-reporting in the US 'National Birth Defects Prevention Study, 1997-2005', revealed a positive statistical association of maternal cannabis consumption with the major foetal abnormalities of oesophageal atresia, diaphragmatic hernia and gastroschisis, with odds ratios around 1.7 after 'correction for exposure misclassification'²²

Gastroschisis is a strange birth defect in which there is an opening in the anterior abdominal wall near the umbilicus through which intestine protrudes. It is not uncommon in northern Australia where the incidence in Indigenous mothers was revealed by a research team in which I was involved to be greater than published in any other ethnic grouping in the world²³. Fortunately, it usually responds to early surgery and prolonged intensive care.

(c) **Morbidity and mortality of the baby.** An association between maternal cannabis use (as confirmed by analysis of the concentration of the drug in umbilical cord tissue) and stillbirth has been found²⁴, as has an association with neonatal illness and death. Serious neonatal problems were found in 14.1% of users compared with 4.5% of non-users, with infection morbidity in 9.8% compared with 2.4%, and neurologic morbidity of 1.4% vs 0.3%. After adjustment for confounding consumption of tobacco, maternal use of cannabis was still associated with a three times higher rate of neonatal morbidity and death²⁵. Additionally, cannabis exposed babies were shown to have almost twice the risk of being admitted to a neonatal intensive care unit²⁶.

3) **Because legalization has resulted in higher rates of use.**

Laws and policies related to marijuana in the US have changed markedly in the last 20 years with 'medical marijuana' now legalized in 28 states and Washington DC and, since 2012, 'recreational marijuana' now also legalised in 9 states and Washington DC. A survey of consumption undertaken in 2015 suggested marijuana use had doubled since 2001-2, and that use was more common in residents of states which had legalized its medical use²⁷.

A later study examined data from adults who had participated in the former but, using different methodology, found a lower increase in consumption, but still one in which the 'overall number of marijuana users increased from 21.9 million in 2002 to 31.9 million in 2014'. It found daily users had increased from 3.9 million in 2002 to 8.4 million in 2014. On a population basis, it found use had increased from 10.4% of the population in 2002 to 13.3% in 2014. In that time, in accordance with increased use, it found the prevalence of 'perceiving great risk of harm from smoking marijuana once or twice a week' had decreased from 50.4 to 33.3%.

Consumption of marijuana had particularly increased after 2007 and was significantly higher in non-Hispanic black adults with reduced education and employment, higher use of tobacco, alcohol, drugs, and other psychotherapeutic agents.

Yet another study reported annual prevalence of any marijuana use by US high school graduates aged from 19-28 years had increased from 29.3% in 2002 to 31.6% in 2014. In the same group, daily use had increased from 4.5 to 6.9%²⁸.

Research on the effect of legalization of marijuana for recreational use in Colorado and Washington states in November 2012 has revealed, in Washington, a reduction in the perception of its harmfulness by 14.2 and 16.1% in 8th and 10th grade children respectively²⁹. In contrast, in states which had not legalized recreational marijuana, perceived harmfulness decreased by 4.9% and 7.2% in similar aged children.

After legalization, consumption of marijuana had increased by 2% and 4.1% in 8th and 10th graders in Washington but, in that period, consumption had actually decreased by 1.3% and 0.9% respectively in states which had not legalized the drug. No change in consumption or perception of risk was noted in Colorado after legalization for recreation, probably because of the publicity associated with the prior legalization of marijuana for 'medical purposes'. Thus, rates of harm perception were already lower, and consumption was already higher in Colorado compared with Washington and non-legalising states. Before legalization for recreation, consumption in 8th graders was reported to be 8.9% in Colorado, 6.2% in Washington and 7.6% in non-legalizing states. After legalizing, rates were 8.9, 8.2 and 6.3% respectively.

In 10th graders, before legalization for recreation in those states consumption was 17, 16.2 and 17.3% respectively, changing to 13.5, 20.3 and 16.4%. And, in 12th graders consumption before legalization was 27.4, 21.2 and 22.3% changing to 25.5, 21.8 and 22.1% respectively. Other studies in Colorado since legalization for recreation have been scarce but an upward trend in use has been confirmed³⁰. More significantly, another study measuring marijuana levels in neonatal meconium revealed the concentration had increased from 213 to 361ng/g, suggesting increased consumption or potency³¹. Indeed, in the last two decades the average THC content in cannabis has increased from 4-12%, but levels as high as 30% have been detected as the legal cannabis market has inspired selective growing methods to increase profits³².

These epidemiological studies reveal the extent of cannabis consumption in adolescents in association with the progressive legalization of marijuana in the United States in the last 20 years. This is of great concern because, as the American Academy of Pediatrics declares, 'marijuana alters brain development with detrimental effects on structure and function'³³.

4) Because of the association of cannabis consumption with psychosis and behavioural problems.

One alarming feature of the increased exposure to cannabis in children from the unborn to adolescence is that it is co-incidental to stages of great brain development. It should be emphasized that brain development is known to continue into the mid-twenties with vital changes in organization and pruning of neuronal connections in association with the widespread and complex effects of natural endocannabinoids.

Association of cannabis consumption with psychosis has long been observed but proof of causality has been difficult to establish because of the rightful impossibility of controlled trials. Nevertheless, a number of cohort studies strongly suggest cannabis is causative.³⁴ The Swedish Conscript Study revealed a dose-response relationship between cannabis use by age 18 and onset of schizophrenia by age 45, with a threefold increase in risk if cannabis had been consumed more than 50 times by 18³⁵.

A Dutch study found cumulative cannabis use was associated with psychotic outcomes three years later³⁶. A California study reported a large association with cannabis use disorder and later hospitalization for schizophrenia³⁷. A New Zealand study found association between cannabis dependence and psychosis after adjustment for confounding influences³⁸. A meta-analysis published in *The Lancet* in 2007, reported 'the risk of psychosis increased by roughly 40% in people who have used cannabis, and there is a dose-response effect, leading to an increased risk of 50-200% in the most frequent users'³⁹. An accompanying Comment on the review, written by psychiatrists from Copenhagen University Hospital, declared that a causal relationship would 'would mean that 14% of psychotic outcomes in the UK might not occur if cannabis was not used'. They warned the findings of the review had 'tremendous implications for young people, their families, and society'. Extrapolating, they concluded 'around 800 yearly cases of schizophrenia in the UK could be prevented through cessation of cannabis consumption'⁴⁰. The accompanying Editorial declared that although in 1995 *The Lancet* had uttered the 'much quoted words' the smoking of marijuana, even long term, is not harmful to health...research published since then...leads us now to conclude that cannabis use could increase the risk of psychiatric illness'.

Apart from the worry that cannabis consumption may at least precipitate if not actually cause psychosis, other neurological adverse associations have been observed: chronic use before 18 years of age has been reported to result in greater decline in intelligence by 38 which persisted despite reduction of consumption in the preceding year.⁴¹ Interference with attention, memory and inhibitory control have also been reported regularly⁴² and would be expected to interfere with education. Overall, a cannabis addiction rate of 10% is likely to compound all the above problems, by rendering desistance much more difficult.

While these above studies relate to adolescents, some studies, but not others, have suggested significant decline in cognition after exposure before birth. One review of research concluded there was 'growing evidence that psychological health may be particularly vulnerable to the adverse effects of in-utero exposure'⁴³. It concluded that increased aggressive behavior and attentional problems in early childhood, followed by hyperactivity, impulsivity and delinquent behavior, depression and anxiety in later childhood, were associated with maternal consumption of one or more joints per day. Heavy maternal use was associated with delinquency, and with the early onset smoking of marijuana.

5) Because Indigenous Australians are very exposed to cannabis.

Research into why Indigenous women continue to experience rates of stillbirth, preterm birth and low birth weight two to three times higher than other women in high income countries, revealed 1 in 5 women used cannabis in pregnancy and 52% smoked cigarettes, with an almost 4 times higher risk of negative birth outcomes⁴⁴. (Adjusted OR low birth 3.9). The weights of the affected babies were on average 565 gm lighter than expected (OR 6.5) and to be disproportionately small for gestational age (adjusted OR 3.9), suggesting interference with in-utero growth. Overall, '51% of mothers using cannabis experienced adverse perinatal outcomes compared with 30% of mothers smoking cigarettes alone, and with 24% of mothers not using either substance during pregnancy'. Overall, younger mothers were at risk: 26.8% of mothers aged 14-19 at the birth of their first child reported marijuana consumption, as did 31.5% of those aged 15-19 at the time of the study. Also, cannabis use was increased in those with lower levels of education, lack of employment and with those suffering adverse stressful events in their lives. Another study has concluded that 15% of indigenous women reported a 'mean of seven cones or joints per day during pregnancy'⁴⁵.

The relatively high rates of gastroschisis in North Queensland may be associated with this high intake of marijuana, especially because, internationally, the incidence of this birth defect is known to be higher in the younger mothers.

6) Because low birth weight from maternal consumption is associated with both acute and chronic complications. The importance of low birth weight is that whether associated with prematurity or reduced growth for gestational age it is associated with increased morbidity and mortality. The acute complications of premature birth include immaturity of lungs and, therefore, dangers of respiratory failure; cardiac instability and reduced oxygenation of the brain; decreased feeding and therefore failure to thrive, and susceptibility to anaemia, low blood glucose, high blood bilirubin, and infection. Acute complication of reduced intra-uterine growth is related to the cause eg placental malfunction leads to foetal undernutrition which may impact upon brain growth. Long term complications include neurological impairment including cerebral palsy, decreased IQ and educational achievement, and increased psychopathology.

Apart from the actual effect of the cannabinoids in cannabis, other components of the smoke may adversely affect the unborn eg increased levels of carbon monoxide will compete with oxygen for transport in the red cells. Other substances known to have contaminated cannabis include cyanides and pesticides.

7) Because of increased presentations of children to hospital emergency departments⁴⁶, and because of an associated increase in road accidents⁴⁷. World-wide, regular use of cannabis has been associated with an increase in vehicle accidents, drug induced psychotic symptoms and psychiatric disorders, HIV, hepatitis B and C, infective endocarditis and tuberculosis⁴⁸. Some of these, of course, are not relevant to Australia but confirm the contribution of cannabis to the health burden.

Conclusion.

I have emphasized the effects of exposure to cannabis on neonates, children and adolescents because of Senator Leyonhjelm's caveat that adults should be free to choose their behavior as long as it does not harm other people. I have emphasised the harm that can be inadvertently inflicted on

children by an adult's freedom to smoke marijuana: harm from actual exposure to cannabis as in the unborn, or harm by reducing the perception of danger and thus indirectly increasing use. Such reduced perception is induced by observing adult consumption and by the advertising associated with marketing. Even if promotion of cannabis is restricted to 'adult hours' in the media, merely walking down main streets in 'legalised states' is sufficient to observe attractive outlets. Thus, legislation for medical and then for recreational use has been associated with increased consumption by mothers and adolescents as it has reduced the perception of harm, rendered the drug more available and even lowered its price by competition. In accordance with demand for more euphoric effect, cannabis with greater THC content has entered the market, rendering the effect increasingly potent.

One sadness is that consumption has been shown to be highest in those less able to handle it: the youngest, the poorest, marginalized, uneducated, psychologically unstable and those already burdened by significant stress in life. Indigenous Australians are a case in point. A question is...will legalization of marijuana reduce or increase 'the gap' between Indigenous and non-Indigenous health?

Freedom of 'normal adults' to consume marijuana is likely to increase the health burden and the social and educational difficulties of the marginalized in society. Rights are thus challenged by responsibilities. *Noblesse oblige* pertains. We are 'our brother's keeper'.

Senator Leyonhjelm is rightly concerned with fiscal responsibility but his considerations do not include the costs of health care imposed by cannabis consumption. If it is true that 12% of low birth weight deliveries could be prevented by reducing cannabis consumption, in that small area alone, savings will be considerable in costs of actual care and preserved potential. Incalculable savings would accrue from preservation of intellectual and motivational potential. If cannabis use is associated with 800 new cases of schizophrenia each year in the UK, the financial and social costs are enormous.

Lastly, who will benefit from legalization of marijuana for recreational use? Relatively few consumers will be stable adults who can afford and tolerate the distraction. Most users will be the marginalized poor in society.

Who will receive their money? Attendance at an investor's conference in Denver revealed to this author that the real beneficiaries will be those poised to invest in 'big agriculture' with industrialized growth of plants under automatic control of temperature, fertilizer and humidity, in glass house conservatoria to maintain the best growing environment and protect from the vicissitudes of weather and insects. Reaping will be automatized and travelling laboratories will measure and ensure potency.

The concept of a few plants being available for private consumption is fanciful: it is a delusion distracting from the reality of agricultural mechanization for large scale production. At that conference, millions of dollars were said to be waiting upon legalization to be made in Australia. If that happens, agricultural profit will accrue from the addiction of the marginalized. Is that profit to which the government should aspire? Is that to do with human rights, when we are supposed to be protecting 'the best interests' of children?

Lastly, will legalization decrease criminalization? Even that is arguable: the government will be seeking to tax production and sale and will, therefore, add to consumer price. Law enforcement will be needed to prevent and punish private unregulated production and distribution.

If passed, Senator Leyonhjelm's Bill is likely to be followed by unconsidered consequences. Costs of preventive and actual health care and reduced potential can be expected to rise. Profit is likely to be disappointing and tainted by its origin. Human rights will be challenged by responsibilities: no construction of the argument will persuade that legalization is in the best interests of children. And, unlimited freedom to smoke cannabis will not be matched by unlimited freedom to produce it. Criminals will still exist.

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