



## **AN OVERVIEW OF: MARIJUANA & THE SAFETY-SENSITIVE WORKER**

**By: Dr. Brendan Adams M.Sc. MD CCFP, FASAM, ABAM**

The full 21-page paper by Dr. Adams reviews the studies relevant to workplace impairment in safety-sensitive environments to provide clarity for the development of corporate and government policies. Despite a long history of marijuana use - originally illicit, however soon to be legalized – scientific studies are relatively sparse and often interpreted in apparently contradictory ways.

Key topics from the full paper are highlighted below. To read more about a specific topic, click on a green box below or you can view the full paper [right here](#).

IT IS IMPORTANT TO BETTER UNDERSTAND MARIJUANA IMPAIRMENT  
AND THE IMPLICATIONS FOR SAFETY-SENSITIVE WORKSITES



The effects of marijuana on an individual are complex. The dose (i.e. the amounts and types of psychoactive compounds) can be highly variable from one use to the next, and the mode of consuming the drug (smoking or eating) changes the intensity and duration of the effects. The experience of the individual and/or the possible development of a drug tolerance can alter the degree to which the effects are expressed as impairment. Mixing with other drugs, particularly alcohol, even in relatively small amounts, can greatly magnify the effects of marijuana.



MARIJUANA EFFECTS ARE COMPLEX AND HIGHLY VARIABLE  
BETWEEN INDIVIDUALS: EVERYONE REACTS DIFFERENTLY

Drawing parallels between impairment due to alcohol consumption and impairment due to marijuana consumption is a mistake for two reasons:

1. Alcohol affects both gross motor control and fine motor control (apparent in the extreme as staggering and slurring of speech). Marijuana affects higher order cognitive functions such as tracking, reaction time, visual function, short-term memory, divided attention and the ability to handle unexpected events (i.e. subtle effects, not always apparent to the casual observer).

ALCOHOL AND MARIJUANA IMPAIRMENT ARE NOT THE SAME:  
THEY AFFECT DIFFERENT AREAS OF THE BRAIN



2. Alcohol metabolites are eliminated from the body within a few hours and task performance returns to normal levels. In contrast, some individuals (particularly chronic users of marijuana) demonstrate residual effects many hours or even days later (e.g. reduced executive functioning such as decision-making, concept formation and planning).

UNLIKE BREATH ALCOHOL MEASUREMENTS, CURRENT SCIENCE DOES NOT PERMIT US TO SPECIFY SAFE (LOW-RISK) LEVELS OF MARIJUANA IN THE BODY



After surveying the findings of the relevant studies, this paper concludes that “there can be no question to an independent reviewer of the data comprising our current state of knowledge, that marijuana impairs the ability of humans to perform safety-sensitive duties on a balance of probabilities and in the vast majority of tasks.”



TO ASSURE A SAFE WORKSITE, WORKERS UNDER THE INFLUENCE OF MARIJUANA AND WORKERS RECENTLY CONSUMING MARIJUANA MUST BE EXCLUDED FROM SAFETY-SENSITIVE TASKS

Companies and governments formulating policy and regulations must recognize the high variability between individuals, the potential longevity of impairment and the absence of a definitive test for marijuana impairment. On safety-sensitive worksites, worker safety can be assured only by excluding both workers under the influence of marijuana and workers who have recently consumed marijuana.

Beyond the present-day safety risks to workers, employers should also be cognizant of the long-term health risks, including mental health outcomes, for marijuana-using employees. Young employees may be at particular risk of long-lasting cognitive impairments because physical growth and brain maturation that continues to develop into their early 20's.

WHETHER THE SOURCE OF IMPAIRMENT IS ILLICIT OR LEGALIZED MARIJUANA, IT IS IRRELEVANT TO MANAGING THE ONSITE SAFETY RISKS



# **Marijuana and the Safety Sensitive Worker**

## **A review for CLRA**

**Dr. Brendan Adams M.Sc. MD CCFP, FASAM, ABAM**

### Topic Overview

The issue of marijuana, or more specifically delta-9-tetrahydrocannabinol (THC) is topical and controversial. It becomes apparent, at this writing, that marijuana will soon be legalized in Canada, in some form, and on some level, for recreational use. The possession of marijuana for medical purposes has already been enshrined in law, and reinforced by Supreme Court rulings. As such, it behooves employers to understand this drug as much as possible, the effect it may have in the workplace, particularly with respect to safety sensitive duties, and what type of guidelines should exist in terms of procedure and policy for those individuals using the drug.

Good policy must be informed by good data. It is here that we face our first challenge. The words of one of the foremost researchers in marijuana and human performance are useful to quote here:

*“If there is one thing I have learned from studying marijuana for more than a decade, it’s that proponents and opponents of the drug will put opposite spins on these findings,” says Harrison Pope, a Harvard professor of psychiatry and leader of (the) research. “One day I will get a letter that will say, ‘we are shocked that you are so irresponsible as to publish a report that claims marijuana is almost harmless. That is a terrible disservice to our children’. The next day I will get a letter complaining that ‘I am irresponsible for implying there something wrong with smoking marijuana. You have set back the legalization of marijuana movement by twenty years’. As a scientist, I am struck by how passionately people hold opinions in both directions no matter what the evidence says. The other striking thing is how little we actually know about the effects of a drug that is been smoked for thousands of years and been studied for decades<sup>1</sup>.”*

It is in studying the data, and attempting to make sense of thousands of studies, many of them contradictory, that one first runs into a challenge. Answering a deceptively simple question such as ‘does marijuana affects one’s ability to drive safely?’ is fraught with difficulty, in that there are studies that purport to show completely opposite answers to this query. In order to make sense of this body of research, several overarching facts must be kept in mind. Firstly, marijuana is inevitably compared with alcohol and this is a mistake. The two are radically different drugs, operate on different brain subsystems, in different mechanisms, and are excreted from the body in a different fashion. The impairment caused by alcohol is quite separate and distinct to the impairment caused by marijuana, and even more complex is the impairment seen when these two drugs are combined. Secondly, the actions of marijuana are extremely complex and nuanced. This is not a “sledgehammer” drug, and consequently its effects, both acute, subacute and chronic, are often subtle and may be missed if the correct experimental design is not used.

It will take a combination of data, constantly evolved and re-examined, to make intelligent statements about this drug. Laboratory human performance data are only part of the puzzle, and equally intriguing are analysis of accident data in the real world. Lastly, the principles of pharmacology must be acknowledged, in that the mode of drug administration, individual user variability, the effect of the drug on naïve versus experienced users, and the concentration of the drug are all variables which appear to be extremely relevant when measuring human performance. The overall caveat, when studying human performance and marijuana, can best be summed up the phrase “it is complicated”. For this reason, overly simplistic stances on either side of the question should be viewed with a high degree of suspicion.

In crafting an opinion on marijuana, at the outset, it is critical to state the purpose of such an opinion, and this paper. This is not, and cannot be, an exhaustive review of marijuana, or its constituent chemicals. Such reviews have been performed already, and three such reviews to which the reader is referred are the “Update of Cannabis and its medical use” by Dr. B. K. Madras for the World Health Organization (335 references)<sup>2</sup>, the “Information for Health Care Professionals - Cannabis (marihuana, marijuana) and the cannabinoids” authored by Health Canada in 2013 (one thousand references)<sup>3</sup> – and soon to be released as an updated 2016 version, and the foundational text, “Principles of Addiction Medicine (marijuana pharmacology- 80 references)<sup>4</sup>. Rather, the purpose of the opinion is to help guide the thinking and subsequent interventions around the use of this drug and safety sensitive personnel.

As such, we are specifically not concerned with whether the drug, or constituents/derivatives may eventually prove to be medically useful. We are also not concerned with issues around prohibition, legality, social order, and governmental drug policy. These are all extremely complex and nuanced areas, and subject to vigorous ongoing debate. They are well examined in the references cited above. Our sole concern has always been, and remains, the safety of workers at the job site. As such, any substance which may be shown to impair, in the broadest sense of the word, the performance of workers in safety sensitive occupations is of central concern to CLRA and is the major thrust of this paper. In simplest terms, does using marijuana impair an individual, and their ability to perform safety sensitive duties? If so, over what time course? Is it a safe drug to use for individuals involved in safety sensitive work?

Note: To aid the reader in accessing references, the following convention has been employed. Because this paper relies so heavily on large review papers, each citing hundreds of papers, the review paper itself will be referenced below, however original research papers as cited by the review paper, will have such references preserved in the quotation and those references may be accessed by obtaining the original review paper.

Before we begin it is also important to clarify what is meant by “marijuana” or even “cannabinoids”. For purposes of our discussion a cannabinoid will be deemed to be any chemical substance that interacts with cannabinoid receptors in the body, referred to as CB1 or CB2. (For completeness sake it must be noted that cannabinoids also interact with an increasingly diverse number of receptor sites in the body which are currently being actively

researched, but for purposes of our discussion, CB1 and CB2 are sufficiently precise). In terms of plant marijuana, there are more than four hundred discrete chemical structures synthesized by the hemp plant, approximately sixty of which are cannabinoids. The two cannabinoids which primarily concern us for purposes of this discussion are the psychoactive ingredient Delta nine tetrahydrocannabinol (THC) as well as the less active cannabidiol (CBD). That is not to say that some of the other cannabinoids may not have subtle psychoactive effects which are difficult to characterize. To further complicate matters there is the issue of synthetic cannabinoids, such as Nabilone (Cesamet), and a wide variety of illicit drugs generically referred to as “spice” or the like - which deserve separate consideration as their behavioural and experiential effects are quite separate and distinct from plant marijuana, and will not be specifically discussed here.

Completing the complexity of this definition is the fact that mode of administration and mode of preparation of the drug is of pivotal importance in terms of its resultant psychoactive effect. Firstly, drug concentration is relevant, as the effects of a very low concentration mixture such as one or two percent dried marijuana leaf is quite different than 80 or 90 percent pure resin extraction, colloquially referred to as “shatter” or “budder”. As an aid to understanding, this would be equivalent to comparing an alcoholic beverage such as light beer at 4% alcohol by volume, with over-proof beverage alcohol such as Everclear 75% abv. The active ingredient, in both cases is ethyl alcohol, but the concentration is so much higher in the second substance that its behavioural effects are vastly different. Mode of administration is also extremely relevant. In general, pharmacology principles state that the more rapidly a psychoactive substance reaches the brain, and the higher concentration of the substance, the more dramatic are the neurologic effects. In general, drugs of abuse are preferentially injected or smoked as this results in crossing the blood brain barrier in approximately 4 to 7 seconds. Oral administration or transdermal administration is much slower, resulting in a broader, shallower curve of drug concentration in the brain. Marijuana is currently administered most typically by smoking or vaporization which is pharmodynamically equivalent, and increasingly commonly, orally through foodstuffs or oil drops.

The first area to consider is that of impairment. At the outset, the author wishes to vehemently stress that the concept of impairment is usually poorly understood, and poorly discussed both in lay debate, and in scientific literature. Quite simply, for our purposes, impairment is any decrement in task performance (in this case, attributable to a drug effect), which contributes to the inadequate performance of that task which could lead, directly or indirectly, to an incident or accident. Typically “impairment” is confused with “intoxication”, and the two are related but distinctly different. Post-intoxication impairment can refer to such entities as “drug hangover”, withdrawal effects, post-intoxication metabolic disturbances, and chronic impairment not related to acute intoxication. It is scant comfort to have a worker injured or killed as result of a late drug effect, and rationalize the event as acceptable because it did not involve acute intoxication. Additionally, little attention is paid to such things as mood impairment, irritability, impulsivity, risk taking, apathy or low motivation, and how these altered states of neurologic activity may contribute to unsafe work practices. It is the intention of this review to take the broadest possible view of impairment, with respect to decrements in safe performance.

## Driving and marijuana

One of the topics that has received the most lay press coverage, and a substantial amount of research, is the question surrounding the use of marijuana and driving impairment. One recent (June 3, 2016, Booth) article in a driving magazine even went so far as to suggest that individuals may actually be safer when they are “baked” on marijuana, than sober<sup>5</sup>. The basis for the controversy, and conflict around marijuana related impairment while driving is in part due to the large, case-control study from the National Highway Traffic Safety Administration in the USA which found no significant increased crash risk attributable to cannabis after controlling for drivers’ age, gender, race, and presence of alcohol<sup>6</sup>.

Because driving is a complex motor task which also involves significant neurocognitive processes, it is viewed, rightly or wrongly, as a model for other forms of complex activities which are safety sensitive. Plainly, if it can be shown that the use of marijuana acutely or chronically impairs driving ability, then by extrapolation, marijuana may be inferred to adversely affect other forms of complex neurocognitively based safety sensitive activities. For a good review of the current state of knowledge, the reader is referred to the article “Clearing the Smoke on Cannabis – Cannabis Use and Driving- an Update” published in 2015 by the Canadian Centre on Substance Abuse<sup>7</sup>. The publication cites the following data. In terms of prevalence, a random sample of night time drivers in British Columbia tested for drugs and alcohol, indicates that 5.5 percent of the drivers tested positive for cannabis (Beasley, Beirness and Boase 2013). In a study of seriously injured drivers admitted to a regional trauma unit in Toronto, 13.9 percent tested positive for cannabis (Stoduto et al 1993). Unfortunately these prevalence studies do nothing to indicate whether cannabis was relevant to causation or was impairing.

To answer this question, the paper goes on to cite four papers (Ashton, 2001; Berghaus and Guol, 1995; Hartman and Heustis, 2013; Ramaekers, Robbe, and O’Hanlon, 2000)<sup>7</sup>. They note *“performance deficits have been found in tracking, reaction time, visual function, concentration, short-term memory, and divided attention. Studies of driving performance (both simulated and on road) show increased variability in lateral position in the lane, following distance, and speed as a function of cannabis use. Cannabis also impairs performance on divided attention tasks- those tasks that require the ability to monitor and respond to more than one source of information a time. Cannabis also compromised the ability to handle unexpected events, such as a pedestrian darting out on the roadway. Combining cannabis with even small amounts of alcohol greatly increased the negative effects on driving skills. (Downey et al., 2013, Raemakers et al., 2000).”*

These research findings are not particularly new or remarkable, so this gives rise to the immediate question as to why there is any controversy with respect to driving under the influence of cannabinoids. This becomes apparent upon reading the original papers, because significant variability is noted between subjects, with wide variation in terms of impairment, and impaired functioning, between users. It has been suggested that more experienced users have less decrement in performance, and it has also been suggested that, particularly with experienced users, they are aware of their impairment and compensate by slowing down or simplifying the task. Risk-taking also appears to decrease, at least acutely, under the influence

of marijuana as opposed to, for instance, alcohol. While acknowledging the controversy, it does not obviate the fact that there are clearly measurable decrements of performance, in a variable and relatively unpredictable fashion, in individuals who have consumed the drug. The counterargument to the idea that experienced users develop coping tactics and strategies, thereby mitigating risk, is well summed up by the authors of the review paper stating *“these tactics, however, may not be sufficient to compensate for all the impairing effects of cannabis—especially unexpected events and higher order cognitive functions such as divided attention tasks and decision-making. Attempts to compensate may be at the expense of vehicle control, for instance speed control, lane position variability, reaction time-reflecting deficits in the ability to allocate attention. In summary the research evidence leaves little doubt that cannabis has detrimental effects on driving performance, particularly when used in combination with other substances, most notably alcohol<sup>7</sup>.”*

The second obvious way to examine the question is to look at the risk of collision after using cannabis. Again, it is possible to find some studies that report no significant increase in collision risk. Gradually, however, more recent research, and larger studies, are indicating increased crash risk beginning at low levels of cannabis and that the risk escalates with dose. The review paper quotes four such studies (Drummer et al., 2004; Laumon, Gadegbeku, Martin, Biecheler, and SAM group, 2005; Mura et al. 2003, and Asbridge, Hayden and Cartwright, 2012). The meta-analysis by Asbridge is particularly interesting as it involves a large number of data points and concluded that cannabis doubled the risk of crash involvement.

One of the drawbacks with these studies is that they all have varying definitions of “cannabis use” in terms of recency, dose, and blood level. Another drawback is the necessity for controlling for confounding variables such as sex, age, and other drug and alcohol use and ethnicity. Studies often examine crashes of different severity, and it is difficult to compare causation between fatal crashes, and more minor property damage crashes. Also of interest is the concept of chronic impairment, and whether cannabis in the withdrawal phase is also correlated in crash involvement. This would be extremely difficult to measure as blood levels may be negligible, and would be yet another interfering variable in the “control” group.

### Resolving the controversy

So what are we to make of studies such as the NHTSA (2007 and 2015) which show no effect, and other studies which do show an effect? In conflicts of this sort it is important to look at overall numbers, as well as study design and how accurately the use of marijuana was correlated with crashes. It is important to also read the comments of the NHTSA researchers themselves. It is instructive to consider the following: *“while the findings of this case-control study were equivocal with regard to the crash risk associated with drug use by drivers, these results do not indicate that drug use by drivers is risk free. The study limitations cited above, together with the findings of numerous other studies using different and complementary methods, need to be carefully considered before more definitive conclusions about drug use and crash risk can be reached. The findings of this study notwithstanding, the established body of scientific evidence on the subject of drug impairment indicates that in some situations, drugs other than alcohol can seriously impair driving ability. This study provides further confirmation*

*that driver impairment is a very serious safety concern and that it involves a very certain element of alcohol impairment and a less certain element of drug impairment.” “From a public health perspective, relatively little is known about the contribution of drugs other than alcohol to traffic crashes. Understanding the effects of other drugs on driving is considerably more complicated than is the case for alcohol impairment. This stems from the fact that there are many potentially impairing drugs in the relationship between dosage levels and driving impairment is complex and uncertain in many cases. Additional challenges include the large differences among individuals with regard to response to the same dose of many drugs, and differences in impairment resulting from acute versus chronic use of some drugs<sup>6</sup>.”*

In short, the studies that show no apparent effect once confounding variables are controlled for, are often misrepresented and misinterpreted as “proving that marijuana has no effect on motor vehicle crashes”. That is not what these studies are showing or saying, as commented upon by the authors above.

### Cognitive Impairment

This is perhaps the single most important area of discussion with respect to safety sensitive work. Again, the reader is cautioned that impairment for our purposes is defined as any alteration of central nervous system functioning which either causes or contributes to an accident or incident (near miss) in the safety sensitive work site. As has been previously stated, impairment is often mistakenly confused with intoxication in lay discussions of this issue. Impairment can refer to impaired memory, mood, ability to concentrate, ability to divide attention between two tasks, ability to engage in complex reasoning, ability to judge distance, speed and time or manipulate objects in a three-dimensional environment, or the more commonly understood effects on gross and fine motor coordination and control. In addition, a broader definition of impairment captures such issues as the onset of formal mental illness such as major mood disorder or psychosis (example schizophrenia).

A recent review article in New England Journal of Medicine<sup>8</sup> is useful in its review of seventy-seven papers and has this to say about impairment: “*marijuana use impairs critical cognitive functions, both during acute intoxication and for days after use*”. A follow-up review article from this year by the same authors examines the issue of cognitive impairment in greater detail<sup>10</sup>. The review series produced by the Canadian Centre on Substance Abuse<sup>7</sup> is worth quoting extensively here: “*Evidence suggests that chronic cannabis use does not produce severe or grossly debilitating impairment of memory, attention and other cognitive functioning; the effects on these cognitive abilities are generally more subtle. After about a month of discontinued use, chronic cannabis users have demonstrated performance deficits in psychomotor speed, attention, memory, and executive functioning as compared to non-using controls (Grant, Gonzalez, Carey, Natarajan & Wolfson, 2003; Medina et al., 2007). Dose-related alterations in brain activity have been noted in the frontal areas, hippocampus and cerebellum, which are regions of the brain responsible for decision-making, executive functioning, and memory (Bolla, Eldreth, Matochik & Cadet, 2005; Schweinsburg et al., 2008). Recent evidence also indicates that chronic cannabis use is associated with reductions in the hippocampal and amygdala brain structures (Yucel et al., 2008). In contrast, studies that have examined the effects of chronic*

*cannabis use following more than a month of abstinence have failed to find evidence indicating any significant cognitive impairment (Fried et al., 2005; Lyketsos, Garrett, Liang & Anthony, 1999; Pope et al., 2002), suggesting that cognitive deficits may be reversible after about a month of discontinued use and that impairment is related to recent but not cumulative use. The linking of neurocognitive impairment in memory, attention, and executive functioning with chronic cannabis use is biologically plausible. The regions of the brain primarily involved with these forms of cognitive functioning include the hippocampus, prefrontal cortex, and cerebellum. Delta-9-tetrahydrocannabinol (THC), the main psychoactive ingredient in cannabis, has been shown to cause deleterious effects on these areas of the brain, which are dense with cannabinoid receptors (Herkenham et al., 1990).”<sup>7</sup>*

One of the best systematic review papers published on cognitive impairment is that of Crean et al.<sup>9</sup> (64 references). The authors reviewed the literature in terms of acute, subacute and long term impairment. It is useful to quote extensively from this review:

*“Some cannabis-related executive function deficits improve after cessation of cannabis use (Pope et al., 2002), but growing evidence suggests that other deficits persist after cannabis is discontinued (Bolla et al., 2005)”*

*Adding to the complexity of this issue is the fact that many factors can impact cannabis-related impairment and recovery of executive functions, including age of onset of smoking cannabis, years of use, and amount of regular use (Grant et al., 2003). This clinical conundrum is compounded by the fact that treatment professionals may not be able to easily identify patients with cannabis-related impairment in executive functions without the benefit of neuropsychological assessment (Fals-Stewart, 1997).*

*Although there is convincing evidence that acute cannabis use generally affects cognitive and motor functions, it is less clear as to whether those deficits are short term and transient or if they are more enduring. Previously published reports (Pope et al., 2001; 2002) using traditional neuropsychological assessment methods typically show a resolution of deficits by 28 days of abstinence. However, as neuroimaging technology has improved, more recent reports show subtle, long-term effects of cannabis on cognition and brain functioning (Bolla et al., 2005). In addition, newly published reports suggest that the deficits change as a function of the quantity of cannabis consumed and duration of use (Solowij et al., 1995; 2002; Grant et al., 2003).”*

*“Summary of Acute Effects of Cannabis on Executive Functions*

*Research assessing the effects of acutely administered doses of cannabis on executive functioning has yielded mixed results (see Table 2). Evidence of the impairing effects of cannabis intoxication on attention and concentration is stronger in less experienced cannabis users than those with established drug tolerance; attention and concentration in the latter group is disrupted more by acute abstinence than acute cannabis administration, probably as a function of neuroadaptation to chronic, heavy cannabis use. Comparable effects were observed on tasks involving information processing, a function that is a basic building block for attention and concentration. Acute cannabis use has generally been found to impair aspects of planning and decision-making, e.g. in terms of response speed, accuracy and latency. Some studies also*

*found risk-taking increased with higher doses of cannabis. Acute, impairing effects of cannabis on tasks assessing inhibition and impulsivity have also been documented. Verbal fluency appears intact following acute cannabis administration, but cannabis-related impairments in aspects of working memory are well-established.”*

The authors then review sub-acute (7 hours to 20 days) impairment:

*“Summary of the Residual Effects of Cannabis on Executive Functions*

*Investigations on the residual effects of cannabis on executive functioning show that recently abstinent cannabis users (7 hours to 20 days) may experience impairment in certain aspects of executive functioning. Attention, concentration, inhibition and impulsivity may or may not continue to be impaired during the interval associated with the elimination of THC and its metabolites from the brain. Decision-making and risk-taking capabilities have not been thoroughly studied during this period, but a single study by Whitlow et al. (2004) suggests that these abilities are impaired. In contrast to the acute effects of cannabis in working memory, deficits as a function of residual cannabis effects have not been found. Findings for verbal fluency are somewhat mixed, but may be due in part to sample differences in degree of cannabis exposure. Studies showing the greatest deficits in executive functioning used subjects who had been smoking heavy amounts of cannabis for long periods of time. It is likely that residual impairments are linked to the duration and quantity of cannabis use.”*

Lastly, chronic impairment (3 weeks or longer) is reviewed:

*“The long-term effects of cannabis use have received the greatest research attention in recent years. Nevertheless, this area of the literature has been fraught with inconsistencies in findings and is complicated by discrepant definitions of what constitutes “long-term effects.” For the purpose of this review, long-term effects refers to 21+ days since last using cannabis, which ensures that both the acute and residual effects of cannabis in the brain have been eliminated. Only a handful of researchers have examined these long-term effects of cannabis use on executive functions.”*

*“Summary of the Long-Term Effects of Cannabis on Executive Functions*

*Cannabis appears to continue to exert impairing effects in executive functions even after 3 weeks of abstinence and beyond. While basic attentional and working memory abilities are largely restored, the most enduring and detectable deficits are seen in decision-making, concept formation and planning. Verbal fluency impairments are somewhat mixed at this stage. Similar to the residual effects of cannabis use, those studies with subjects having chronic, heavy cannabis use show the most enduring deficits.”*

Another review paper examining the issue of impairment is helpful:

The essential role of working memory in the safe performance of complex tasks has been well described elsewhere. The comments of the World Health Organization document “Update of Cannabis and its medical use” by Madras<sup>2</sup> is instructive: “*THC and cannabis decrease working memory, apparently by actions in the hippocampus, a brain region critical for learning*

*and memory. The memory decrements induced by THC or cannabis resemble hippocampal lesions. These impairments may result from suppression of glutamate release in the hippocampus, which is responsible for the establishment of synaptic plasticity.<sup>77,78,79</sup>*

*“4.4.2 **Cannabis, employment, the workplace.** The effects of cannabis use on cognition in the context of work and everyday life, or whether off-site cannabis use endangers a worker or his colleagues while at work, has not been systematically investigated. One study that examined association between cannabis use and cognitive performance, mood and human error at work found that cannabis use was associated with impairment in both cognitive function and mood, though cannabis users self-reported no more workplace errors than controls. Users also displayed lower alertness, slower response organization, working memory problems at the start, and psychomotor slowing and poorer episodic recall at the end of the working week. Subtle effects on cognitive function may be exacerbated with fatigue or work - related demands<sup>177</sup>. During an economic downturn, cannabis use was recently shown to increase unemployment among users<sup>178</sup>. Combined with alcohol, vaporized cannabis yields higher maximum concentrations of blood THC (than without alcohol) detected 8.3 hours later, possibly explaining why performance is more impaired if cannabis is combined with alcohol in this manner<sup>179,180,181</sup>.”*

Also useful as a source of information is the document “Information for Health Care Professionals Cannabis (marihuana, marijuana) and the cannabinoids<sup>12</sup>”. (Current version 2013 – soon to be updated.) This document contains 1,000 references. It is worthwhile quoting, unabridged, the section on psychomotor performance:

*7.7.2 Psychomotor performance Although no studies have been carried out to date examining the effects of cannabis or psychoactive cannabinoid exposure on psychomotor performance in individuals using these substances solely for medical purposes, it is well known that exposure to such substances impairs psychomotor performance (118) and patients must be warned not to drive or operate complex machinery after smoking or eating cannabis or consuming psychoactive cannabinoid medications (e.g. dronabinol, nabilone, nabiximols). A double-blind, placebo-controlled, crossover study comparing the effects of a medium dose of dronabinol (20 mg) and of two hemp milk decoctions, containing medium (16.5 mg) or high doses (45.7 mg) of THC, reported severe impairment on several performance skills required for safe driving (953). A moderate dose (21 mg of THC) was associated with impairments in motor and perceptual skills necessary for safe driving (954). In one study, performance impairment appeared to be less significant among heavy cannabis users compared to occasional users, potentially because of the development of tolerance or compensatory behaviour (169). It has been suggested that, unlike alcohol, cannabis users are aware of their level of intoxication and compensate by becoming hyper-cautious; in tasks such as driving, this kind of behaviour results in decreased speed, decreased frequency of overtaking, and an increase in following distance (955,956). Others disagree with this assertion ((957) and also see (176)). A recent double-blind, placebo-controlled, randomized, three-way, crossover design study suggested that administration of dronabinol dose-dependently impaired driving performance in both occasional (defined as using*

a cannabinoid between 5 and 36 times per year) and heavy cannabis users (defined as using 1 - 3 joints per day, > 160 times per year) (958). However, the magnitude of the impairment appeared to be less in heavy users, possibly due to tolerance (958). The authors indicate that driving impairments after dronabinol were of clinical relevance and comparable to drivers operating their vehicles at a blood-alcohol concentration of greater than 0.8 mg/mL (0.08 g%) (958). Approximately 25% of the heavy users demonstrated impairment equivalent to, or worse than, that reported for drivers with a blood-alcohol concentration of 0.5 mg/mL (0.05 g%). Driving impairments after dronabinol use were evident even though THC plasma concentrations were relatively low (varying between 2 and 10 ng/mL) (175,958). A recent case-control study estimating accident risk for a variety of substances including alcohol, medicines, and illegal drugs found that the odds ratio for accident risk for all the THC concentrations measured (1 to > 5 ng/mL) was statistically significant (959). At whole-blood concentrations of  $\geq 2$  ng/mL THC, the risk of having an accident was significantly increased (959). One study found that the risk of responsibility for fatal traffic crashes, while driving under the influence of cannabis, increased with increasing blood concentrations of THC such that there was a significant dose-effect relationship between risk of responsibility for fatal traffic crashes and blood concentrations of THC. The study showed that the odds ratio of having a fatal crash increased from 2.18 if blood concentrations ranged between 0 and 1 ng/mL of THC, to 4.72 if blood THC concentrations were  $\geq 5$  ng/mL (960). The findings from this study further support the notion of a causal relationship between cannabis use and crashes (960). Another study suggested that drivers who were judged (by a police physician) as being impaired had higher blood THC concentrations than drivers judged not to be impaired (median: 2.5 ng/mL vs. 1.9 ng/mL) (961). Using a binary logistic regression model, the odds ratio for being judged impaired appeared to increase with increasing drug concentrations from 2.9 ng/mL onwards (961). Serum THC concentrations between 2 and 5 ng/mL have been identified as a threshold above which THC-induced impairment of skills related to driving become apparent (133,959). Performance impairment after cannabis intake was reported to be highest during the first hour after smoking, and between 1 - 2 h after oral intake, and declining after 3 - 4 h (or longer in the case of oral ingestion) (862,961). A recent meta-analysis of observational studies examining acute cannabis consumption and motor vehicle collision risk reported that driving under the influence of cannabis was associated with a significantly increased risk of motor vehicle collisions compared with unimpaired driving, with an odds ratio of 1.92 (95% Confidence Interval = 1.35 - 2.73;  $p = 0.0003$ ) (175). Collision risk estimates were higher in case-control studies and studies of fatal collisions, than in culpability studies and studies of non-fatal collisions (175). It has been reported that individuals who drive within 1 h of using cannabis are nearly twice as likely to be involved in motor vehicle accidents as those who do not consume cannabis (954). For this meta-analysis, only observational studies with a control or comparison group, including cohort (historical prospective), case-control, and culpability designs were included, and experimental laboratory or simulator studies were excluded (175). Furthermore, only studies that assessed acute or recent cannabis use were examined. This meta-analysis supports the findings of other studies which suggest that cannabis use impairs the performance of the cognitive and motor tasks that are required for safe driving, thereby increasing the risk of collision (175). Although driving simulator studies have reported a dose-response effect, in which elevated concentrations of THC were associated with increased crash risk, dose-response effects could

*not be established in this study (175). A double-blind, counter-balanced, placebo-controlled driving simulator study reported that driving performance was more impaired in subjects who co-consumed alcohol and low or high doses of THC by smoking cannabis cigarettes (176). The level of THC detected in the blood was higher when cannabis was consumed along with alcohol than when consumed alone (176). It also appeared that regular cannabis users displayed more driving errors than non-regular cannabis users (176). A recent systematic review and meta-analysis concluded that, after adjusting for study quality, cannabis use was associated with a seven-fold estimated risk of being involved in a fatal accident, benzodiazepine use was associated with a two-fold estimated risk of a fatal accident, and opiate use with a three-fold estimated risk of a fatal accident (177). In contrast, cannabis use was associated with a 1.5-fold estimated risk of having an accident that only caused injury, benzodiazepine use was associated with a 0.71-fold estimated risk, whereas opiates were associated with a 21-fold estimated risk of having an accident that only caused injury (177)*

Thus, this extensive review of the literature highlights some of the difficulties with respect to marijuana and impairment. It is variable, depending on multiple factors, and generally complex and challenging to measure<sup>11</sup>.

Is there a drug level at which impairment may be inferred – as with alcohol?

This is a vexing question currently before authorities attempting to establish equivalent “drug driving levels” that correspond to the concept of blood alcohol. As is discussed throughout this paper, it is exceedingly unwise to extrapolate between psychoactive drugs as if one were equivalent or even comparable to another. Marijuana is unique in many of its properties including its pharmacology. It is instructive to quote from the American Society of Addiction Medicine “Principles of Addiction Medicine<sup>4</sup>”: *“the relationship between blood levels of THC and pharmacologic effects is not initially linear. A slight delay between the rapid appearance in plasma of THC and the onset of behavioural effects makes the impairment produced by THC difficult to predict based solely on plasma concentrations. Once THC is distributed completely to all body compartments, behavioural effects of THC are proportional to its plasma concentrations<sup>70</sup>. Lack of correlation of blood concentrations and pharmacologic effects is a confounder to the interpretation of impairment following THC use. Complex mathematical models allow for the estimation of time elapsed since marijuana usage based upon THC/metabolite ratios, a topic of importance in criminal and workplace cases in which liability is assessed based on drug use. Human controlled drug administration studies led to the development and validation of two equations for predicting time since last use: model one based upon THC concentration (for infrequent users) and model two based upon the THCCOOH/THC ratio (for all users and oral administration). Both models were found valid for forensic use with ninety-five percent confidence intervals of detection<sup>70</sup>.”*

Thus, two points flow from the above discussion, both of which must be underscored. Firstly, because marijuana metabolites are detectable for many days to weeks after cessation of use, a positive urinary drug screen does not imply impairment. Secondly, there is a well recognized lack of correlation between serum levels of marijuana and its metabolites, and impairment or

psychoactive effects. While some pharmacodynamic models have been developed to allow one to interpolate when a dose was taken, and what the serum levels were, in no way do these models directly measure impairment.

So while work proceeds urgently on this important social issue, it would be the position of this paper that attempting to find a particular blood level of THC or cannabinoids at which an individual might be viewed as impaired, or not impaired, is not a practical or useful goal to pursue in ensuring worker safety and fitness to work.

### Sativex

In examining the issue of drug side effects, and particularly impairment, it is also helpful to look at a licenced form of cannabinoids, in that the producing drug company (Bayer) is required to publish research based warnings in its product monograph.

From the fact sheet produced by Health Canada (2005): *“SATIVEX® is a cannabis based medicine containing Tetranabinex® and Nabidiolex® extracts of chemically and genetically characterised Cannabis sativa L. plants. The principal active components are delta-9-tetrahydrocannabinol (THC) and cannabidiol (CBD)<sup>13</sup>”.*

*“THC and CBD are the principal active components in SATIVEX®. THC can produce physical and psychological dependence and has the potential for being abused. THC has complex effects on the central nervous system. These can result in changes of mood, decrease in cognitive performance and memory, decrease in ability to control drives and impulses, and alteration of the perception of reality, particularly altered time sense. Fainting episodes have been observed with the use of SATIVEX®. Central nervous system effects, with dizziness being the most frequent (see table 2), appear to be dose-related, increasing in frequency with higher dosages, and subject to great interpatient variability. They usually resolve on reduction of doses, increasing the interval between doses or interruption of SATIVEX®. Because of the potential of THC to alter the mental state, SATIVEX® should be used only as indicated and prescription should be limited to the amount necessary for the period between clinic visits. Drug administration should be discontinued in patients experiencing a psychotic reaction or suicidal ideation and the patient should be closely observed in an appropriate setting until his/her mental state returns to normal. Patients should stop taking SATIVEX® if they become confused and disorientated. Patients should be warned not to drive or engage in activities requiring unimpaired judgment and coordination. Side effects may impair the mental and/or physical abilities required for certain potentially hazardous activities such as driving a car or operating machinery. Patients should also be cautioned about the additive/synergistic effects of side effects with other central nervous system depressants, including opiates, GABA inhibitors, sedative/hypnotics and alcohol.<sup>14</sup>”*

### Psychiatric Disorders as they affect Safety

It is a documented phenomenon, observed across several studies<sup>15, 15a,</sup> as far back as the mid-1980s in the landmark Swedish study by Andreasson<sup>15b</sup>, that marijuana contributes, in some as yet to be delineated fashion, in the emergence of schizophrenia in vulnerable individuals. This

has been speculated to be due to a polymorphism in one or more genes<sup>16, 16a</sup>. This is one of the more controversial areas of research and debate rages between those claiming a factitious or statistically insignificant association, and the school of thought pointing to a permissive if not causative factor. The exact details of this debate are not germane to this paper, however, it appears at this stage that there is some association, particularly between adolescent marijuana use, and the emergence of schizophrenia in vulnerable individuals. What is relevant to our considerations is the fact that the emergence of a psychotic thought process is diametrically opposed to safe work practices. There have been several well-publicized recent cases wherein professional airline pilots experienced a psychotic break while on duty, with the expected serious impact on safe completion of their flight.<sup>17</sup> As such, even though the exact mechanisms or associations have not yet been definitively elucidated, the possible role of marijuana in the emergence of psychosis is yet another concern in the safety sensitive work site.

The other mental health disorder widely reported with marijuana is that of dependency. The DSM 5 of the American Psychiatric Association sets out the criteria for cannabis use disorder, the more severe forms of which are colloquially referred to as “addiction”. The addiction liability of marijuana is generally viewed as being approximately ten percent of all users, and fifteen percent of those users who commenced regular use in their mid teens<sup>18</sup>. The relevance of any individual suffering from active addiction and safety sensitive work is the attendant chaos, distraction, and inability to focus on task which accompanies this disorder, regardless of the drug and its neurobiological effects. This is insightfully summed up in the preamble from Marijuana Anonymous recovery group: *“We who are marijuana addicts know the answer to this question. Marijuana controls our lives! We lose interest in all else; our dreams go up in smoke. Ours is a progressive illness often leading us to addictions to other drugs, including alcohol. Our lives, our thinking, and our desires center around marijuana—scoring it, dealing it, and finding ways to stay high<sup>19</sup>.”* Again, a detailed discussion of addiction and its phenomenology is outside the focus of this paper, however an individual who is experiencing active addiction typically has issues in multiple spheres such as relationships, finances, sleeping, self-care, and drug seeking behaviour all of which serve to distract and defocus the individual from the task at hand. In a worker performing safety sensitive duties this can contribute to accidents.

Beirness<sup>7</sup> summarizes well: *“A growing body of evidence also indicates that chronic cannabis use may increase the risk of mental health outcomes, including psychosis, depression and anxiety. Adolescent cannabis users may be at greatest risk, perhaps because their use becomes longstanding or due to the effects such exposure has on their developing brains. A biological mechanism may underlie the cannabis-psychosis relationship, but further research is needed to develop a clearer understanding of this possibility.”*

#### Marijuana use in younger adults

It is also important to note that marijuana’s effect in the developing brain is quite different than that in the brain of older users. The average age of initiation of marijuana in North America tends to be in the mid teens. Brain maturation and physical growth is generally acknowledged to have reached the end of its adolescent period in the early 20s, generally sometime around age 21 to 24. There is now evidence that suggests that measurable and long-lasting cognitive

impairments accumulate after long-term or heavy use in adolescent users<sup>20</sup>. Beirness et al<sup>7</sup> reviews the research: *“Evidence indicates that individuals who initiate cannabis use at an early age—when the brain is still developing—may be more vulnerable to lasting neuropsychological deficits than those who begin use later in life. Visual scanning is a cognitive function that undergoes a major maturational process around 12–15 years of age, and Ehrenreich et al. (1999) found that early-onset regular cannabis users (onset before age 16), but not late-onset regular users (onset at 16 years or later), exhibited significantly longer reaction times than controls on a visual scanning task<sup>1</sup>. Another study reported that long term cannabis users who had initiated use before age 17 had smaller brains, with a lower percentage of gray matter and a higher percentage of white matter, as compared to long-term users who had initiated use at age 17 or later (Wilson et al., 2000). Cannabis use before age 17 has also been associated with poor neurocognitive performance on tasks involving verbal ability (Pope et al., 2003); however, it is not clear from Pope and colleagues’ study whether these associated verbal decrements are directly related to cannabis use or if they may be accounted for by lower overall premorbid cognitive ability.”*

Since many industries that are safety sensitive tend to hire younger workers in their late teens or early 20s, this factor comes into play as a factor influencing the well-recognized predisposition of younger more inexperienced workers to suffer a higher accident or injury rate on the job. The review papers by Volkow<sup>8,10</sup> go on to note some of the controversy around whether such deficits are reversible, or not. *“Some studies suggest that long-term deficits may be reversible and remain subtle rather than disabling once a person abstains from use. Other studies show that long-term, heavy use of marijuana results in impairments in memory and attention that persist and worsen with increasing years of regular use and with the initiation of use during adolescence.”*

### Points of Confusion

This section is provided to address some of the more prevalent areas of debate concerning marijuana. It is not intended to be exhaustive.

While it has been said that “the first casualty of politics is truth”, it is actually a more profound observation on humans and their belief systems. People debating belief systems rarely do so based on scientific data or demonstrable fact. Rather, they tend to engage in highly emotional debate, and cherry pick the scientific and pseudoscientific literature for data that confirm what they already believe (confirmation bias). Likewise, as with any deeply held belief system, defence mechanisms come into play to prevent the individual having to face significant dissonance between what they believe, and what the science is telling them. As such, in the ongoing debate concerning marijuana, there exists common logical pitfalls and defence mechanisms, and a brief examination of these is useful to anyone seeking to understand this politically charged arena. Such an examination should be done with the caveat that engaging in debate with an individual who has a belief system around marijuana is unlikely to alter that individual’s beliefs. This section may, however, help those individuals who are confronted with some of the more common debate points around the use of marijuana.

### *False Dichotomy*

This has been sometimes described as the “would you rather be shot or stabbed?” conundrum. In forcing the individual into an artificial choice, neither one of which is acceptable, the debater seeks to rationalize their choice of drug as being superior. Various statements from online forums illustrate this concept:

*“Marijuana is much safer than alcohol”.*

*“Marijuana is illegal and yet people get drunk every weekend and no one says anything”.*

*“I would much rather have someone drive stoned than drunk”.*

*“No one ever died from marijuana”.*

What these statements fail to acknowledge are several realities. Firstly, it is extremely common for individuals to mix their drugs as has been observed<sup>21</sup>, and a significant percent of post-accident drug testing revealed both alcohol and marijuana present in the system. The idea that a clean dichotomy exists between one drug or the other is actually not borne out by observation of human behaviour. Secondly, it is vastly preferable to have an individual operating a motor vehicle, or working in safety sensitive areas, not psychoactively affected by *any* intoxicant. The idea that one intoxicant is marginally advantageous over another is a classic false dichotomy. The absence of any intoxicant is superior to either. The other issue that it is missed in these types of statements is the fact that just because something is legal, does not mean it is safe or healthy. Cigarettes are legal, but no one (outside the tobacco industry) is suggesting they are without harm. The statement that “no one ever died from marijuana” is in fact demonstrably false, and that there are well-documented accidents, involving fatalities, in which marijuana was imputed to be the significant contributing factor<sup>22</sup>. What the debaters actually mean to say is that the LD50 (lethal dose 50) for marijuana is much higher than that of alcohol when compared to a recreational dose.

#### *Incomplete data set/inadequate experimental design*

It is a truism in science that, if you are not looking for something, you are highly unlikely to find it. Thus to state that “no one has ever...” , or “no study has ever shown...” is valid only if one can demonstrate that well-constructed studies have looked for an effect, or a phenomenon, and failed to find it. This is a particularly prevalent problem in studies involving motor vehicle accidents and marijuana use. To date, no consensus exists on the exact way to quantitate marijuana intoxication/impairment and therefore to compare it to accident data is highly problematic. Correlation studies have been done, (and extensively quoted within this document), but as far as a dose-response curve between marijuana use and motor vehicle accident relative risk, such does not exist in a reliable, reproducible form. In short, the absence of data showing an effect, does not mean that there is no effect.

#### *Confusing Intoxication with Impairment*

This is one of the core concepts in this document. Typically, when discussing marijuana, individuals look at acute intoxication (which is highly variable between individuals but is generally accepted to be less than six hours). It is reasoned that once acute intoxication passes,

there are no relevant safety-related effects from consuming this drug. Again scientific data would dispute this point of view, particularly if impairment is viewed in its broader context, which is “any decrease in neurologically governed functions, such as mood, memory, motivation, problem solving and higher cognitive functions.” Once one stops looking for intoxication, and starts considering intermediate or long-term impairment as a safety relevant issue, some of the data with respect to marijuana are of concern. These data also cast in a negative light the commonly held justification that “what I do on my own time is of no concern of the employer” or that someone is fit to work in a safety sensitive environment because “they only use it after a shift, never at work”. The extensive discussions reviewed above invalidate this argument.

#### *Equating alcohol impairment with marijuana impairment*

Because our society is most familiar with alcohol, and its acute and subacute effects, we tend to generalize our understanding of the concepts of “impairment”. Alcohol is known to adversely affect fine motor and gross motor control. Hence an individual who is acutely intoxicated by alcohol will be slurring his speech because of the loss of fine motor control governing the lips/mouth/tongue, and weaving, because of loss of gross motor control. Neither of these are significantly impacted by marijuana, hence the common observation “he was not impaired because he was not slurring his speech or weaving”. As the discussions above reveal, the impairment caused by marijuana is typically more subtle and of higher function than that caused from alcohol. Discussions in which alcohol is juxtaposed with marijuana as purportedly a “better” or “safer” drug are relatively meaningless when taken in this context. It also indicates that significant impairment from marijuana may be before our eyes, but because we are not looking for it, we do not see it.

#### *Causation cannot be proved*

It is not widely understood amongst general readership that scientific studies cannot prove causation. The most illustrative example of this is the ongoing challenge that science faced in answering the question “do cigarettes cause lung cancer/chronic obstructive pulmonary disease/cardiovascular disease?” Firstly, it takes time for these entities to arise, so someone studying cigarette smoking in a population of twenty-year-olds who had been smoking for five years, is unlikely to find a significant incidence of lung cancer or chronic obstructive pulmonary disease. Secondly, studies can only prove correlation, and it is through increasingly tight correlation between cigarette smoking and these disease entities that the consensus is that “smoking cigarettes causes cancer”. Thus the argument that “scientific studies prove that marijuana does not cause accidents” is as invalid as “scientific studies prove that marijuana does cause accidents”. All that scientific studies can show is correlation.

#### *Reducing complex cognitive tasks to measurable sub-tasks*

It is impractical to study complex human behaviours in a laboratory setting. There are too many variables, accurately modelling the task inevitably oversimplifies it, and constraining outcomes to be measurable can be a challenge. As such, the vast majority of research on human performance under the influence of marijuana involves substitutes or analogues for complex safety sensitive tasks. This leads to the invalid extrapolation that, because no impairment in one

measure of complex performance can be found, that there is no effect on the safe performance of complex tasks. This is illustrated in research done on aviation simulator performance and marijuana use<sup>23</sup>. The more complex, and high-level the task, the more likely it is to show an effect of marijuana use on performance of that task.

### Medical Marijuana and the Safety-sensitive Jobsite

It is tempting, although in the opinion of the author a mistake, to “put medical marijuana on trial”. A common human trait is to wish to avoid appearing gullible. Marijuana is unique in that, in recent history, hundreds of “dispensaries” have opened, particularly on the West Coast, in a situation without parallel in the world of medical therapeutics. It strains credulity to believe that this plethora of retail outlets is necessary to dispense a drug for therapeutic reasons when no other medication has merited this treatment. The strongest parallel to the hundreds of retail outlets selling marijuana is that of liquor stores, leading to the arguable conclusion that the “dispensaries” are in fact selling recreational marijuana to recreational users, and that the “medical” necessity of this drug is simply a ruse to obtain what is still an illegal intoxicant by other means. Equally unparalleled is the proliferation of companies and individual physicians providing marijuana “prescriptions” based on Skype interviews or Internet contact, absent of any of the usual history taking or physical examination essential to the provision of quality medical services. In the face of these cultural realities, it is easy to understand the scepticism of safety sensitive industry as they confront the topic of medical marijuana in the workplace.

Another objection is that marijuana has not been subjected to rigorous double blinded, cross over controlled studies, has not been licensed for any particular therapeutic purpose, and in the words of the Health Canada website: *“Cannabis is not an approved therapeutic product and the provision of this information should not be interpreted as an endorsement of the use of this product, or cannabis generally, by Health Canada. Despite the similarity of format, it is not a Drug Product Monograph, which is a document which would be required if the product were to receive a Notice of Compliance authorizing its sale in Canada. (This document) is a summary of peer-reviewed literature and international reviews concerning potential therapeutic uses and harmful effects of cannabis (marihuana) and cannabinoids. It is not meant to be comprehensive and should be used as a complement to other reliable sources of information. (This document) should not be construed as expressing conclusions from Health Canada about the appropriate use of cannabis (marihuana) or cannabinoids for medical purposes<sup>12</sup>.”* As such, it is easy to dismiss all users of cannabis as recreational, in the same manner as “prescriptions” of alcohol were provided by complicit physicians during prohibition to enable recreational drinkers to access their beverage of choice in the face of government proscription.

Notwithstanding the foregoing, it is entirely possible that marijuana, or derivatives thereof, will prove to have some form of medical benefit. It would be wrong to dismiss wholesale the idea that cannabinoids are of no medical use<sup>3,4,12</sup>. As such, employers will be increasingly confronted with individuals of varying degrees of sincerity, insisting that THC is a medical necessity for them, and supported by a physician’s medical opinion, albeit of varying degrees of validity. Such an individual may fall under the protections of the Human Rights Act, as suffering from a disability, and deserving of accommodation to the point of undue hardship. Nothing in this paper

would prevent such accommodation in non safety-sensitive duties. It is not, however, acceptable to permit individuals, unfit to perform safety sensitive duties by virtue of psychoactive impairment, to perform such duties, merely because they insist that their medical condition requires cannabinoids in order to treat. It is not fair to them and it is not fair to their coworkers. As such, whether the source of marijuana happens to be by medical authorization, based on medical opinion, or obtained for recreational purposes through various sources, the net effect remains the same. Any individual seeking to use marijuana on an ongoing basis is unfit for safety sensitive duties. Employers would be well advised to seek legal counsel in including medical marijuana in their alcohol and drug policies, and guidance with respect to handling the accommodation claims of employees seeking dispensation to use medical marijuana.

### Summary

As has been demonstrated by the discussion above, marijuana is highly psychoactive in humans, and impairing in a wide variety of spheres. There is a great degree of variability between subjects, based on experience with the drug, age of the subject, and specifics of the task at hand. Notwithstanding this, there can be no question to an independent reviewer of the data comprising our current state of knowledge, that marijuana impairs the ability of humans to perform safety sensitive duties on a balance of probabilities, and in the vast majority of tasks. The time course of this impairment is highly variable but is clearly longer than the time course of intoxication. The fact that one particular subject, performing a particular task, may demonstrate little or no measurable impairment is of no comfort in the overall picture.

The following quote from the WHO paper<sup>2</sup> is a cogent summary: *"There is ample evidence indicating that neurocognitive impairment from cannabis persists from hours to weeks. A return to a non-intoxicated state does not ensure a full return of neurocognitive function in the workplace<sup>182</sup>. In a summary of the dilemmas that cannabis for medical use has created for the workplace, it was pointed out that ensuring safety of workers who are under the influence or who recently consumed cannabis is not possible<sup>183</sup>".*

It also becomes apparent, due to the variability cited in the preceding references, that a discrete level of blood concentration of THC cannot be accurately specified, below which safety impairment does not become a concern. Blood levels also fail to take into account such compounding variables as the emergence of psychosis and mood disorder, both of which have been linked to the use of marijuana. The only rational manner in which to proceed is to prohibit the use of the drug in safety sensitive tasks. Given the current state of technology, the only rational way in which to do this is to insist on negative drug screening for marijuana and its metabolites in safety sensitive personnel. The emergence of the phenomenon of "medical marijuana" in no way changes this reality. The source of THC is not relevant to the impairment of individual users with respect to safety sensitive duties. It only becomes relevant in a discussion of duty to accommodate which is beyond the scope of this paper. In summary, the use of THC in the safety sensitive work place, based on a preponderance of evidence demonstrating significant psychomotor impairment from various sources, is unacceptable.

## Major Points:

- Marijuana is a drug which has significant effects on psychomotor performance and cognition. These effects can be long-lasting, and persist far after the initial intoxication phase. In some cases they can be measured months later. Some data supports the view that some impairments may be permanent.
- Marijuana's effects are often subtle, and relate to higher cognitive processes such as impulsivity, working memory, decision-making and attention. As such, they may be missed by casual observation.
- With great respect, most primary care physicians are not trained in detecting subtle impairment in their patients, nor are they trained in occupational medicine, and their assurances that their patient taking marijuana (either recreational or medical) is not impaired is of no validity to determining fitness for safety sensitive work.
- Likewise, given the lengthy time course of impairment, as discussed in the foregoing paper, the assurances that a worker will only take marijuana outside of regular work hours, is of no relevance in determining their fitness to perform safety sensitive work.
- There is a high degree of inter-subject variability and this drug's effects on human performance is complex and nuanced.
- Because of the foregoing there is a degree of variation in experimental data which can lead to controversy and confusion.
- Because of the high degree of variability it is not reassuring that any particular individual, in a particular circumstance, may demonstrate no obvious impairment. A broader more population-based approach is the only fair and equitable solution to this complex issue.
- There is no particular test which measures impairment, nor is there any agreed-upon blood level of marijuana which would indicate impairment in a fashion that society is most familiar with in its dealings with beverage alcohol. Marijuana and alcohol are radically different drugs and must not be approached with the same mindset.
- The source of supply of marijuana, whether by medical authorization or by recreational street sources is irrelevant in terms of impairment and safety. Source of supply has large implications in the arena of Human Rights, which is outside the considerations of this paper.
- For all of the reasons quoted in the foregoing the only safe and equitable solution is to exclude marijuana, both medical and recreational, from the safety sensitive work site

## References

1. Cromie, W. J. (2001, October 15). Study: Intelligence, Cognition Unaffected by Heavy Marijuana Use. Retrieved November 18, 2012, from the Harvard University Gazette Web site: <http://news.harvard.edu/gazette/2001/10.11/marijuana.html>

2. Madras, B K, 37<sup>th</sup> Expert Committee on Drug Dependence (November 16-20, 2015) Agenda item 6.2, World Health Organization
3. [http://www.hc-sc.gc.ca/dhp-mps/alt\\_formats/pdf/marihuana/med/infoprof-eng.pdf](http://www.hc-sc.gc.ca/dhp-mps/alt_formats/pdf/marihuana/med/infoprof-eng.pdf)
4. "Principles of Addiction Medicine, 5<sup>th</sup> edition, published 2014, "The Pharmacology of Marijuana" p 217-232 (80 references).
5. <http://driving.ca/auto-news/news/motor-mouth-hysteria-over-high-driving-is-half-baked>
6. Compton RP, Berning A "Drug and Alcohol Crash Risk" Washington, DC: National Highway Traffic Safety Administration; 2015, DOT HA 812 117 - as quoted by NIH Research Report Series <https://www.drugabuse.gov/publications/research-reports/marijuana/does-marijuana-use-affect-driving>
7. <http://www.ccsa.ca/Eng/topics/Marijuana/Marijuana-Research/Pages/default.aspx>  
This is a series of review articles by the Canadian Centre on Substance Abuse. Specifically referenced were "Cannabis use and Driving" and "Chronic Use and Cognitive Functioning and Mental Health"
8. Volkow et al, "Adverse Health Effects of Marijuana Use", N Engl J Med 2014 Jun 5;370(231) 2219-2227
9. Rebecca D. Crean et al.; An Evidence Based Review of Acute and Long-Term Effects of Cannabis Use on Executive Cognitive Functions, J Addict Med. 2011 Mar 1; 5(1): 1–  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3037578/>
10. Volkow ND, Swanson JM, Evins AE, et al. Effects of Cannabis Use on Human Behavior, Including Cognition, Motivation, and Psychosis: A Review. JAMA Psychiatry. February 2016. doi:10.1001/jamapsychiatry.2015.3278.
11. <http://archpsyc.jamanetwork.com/article.aspx?articleid=2537046#tab10>
12. [http://www.hc-sc.gc.ca/dhp-mps/alt\\_formats/pdf/marihuana/med/infoprof-eng.pdf](http://www.hc-sc.gc.ca/dhp-mps/alt_formats/pdf/marihuana/med/infoprof-eng.pdf)
13. [http://www.hc-sc.gc.ca/dhp-mps/prodpharma/notices-avis/conditions/sativex\\_fs\\_fd\\_091289-eng.php](http://www.hc-sc.gc.ca/dhp-mps/prodpharma/notices-avis/conditions/sativex_fs_fd_091289-eng.php)
14. <http://www.bayer.ca/static/documents/news/en/SATIVEX%20PM-PT3-EN-31MAR-2015-141001.pdf> and  
[http://www.bayer.ca/static/documents/news/en/sativex\\_dhcpl\\_lapds\\_109461\\_e%20GW-2.pdf](http://www.bayer.ca/static/documents/news/en/sativex_dhcpl_lapds_109461_e%20GW-2.pdf)

15. 15. S. Zammit et al; Self reported cannabis use as a risk factor for schizophrenia in Swedish conscripts of 1969: historical cohort study, *BMJ* 2002;325:1199  
 15a: Radhakrishnan R, Wilkinson ST, D'Souza DC. Gone to Pot - A Review of the Association between Cannabis and Psychosis. *Front Psychiatry*. 2014;5:54. doi:10.3389/fpsy.2014.00054.  
 15b. Andreasson et al, Cannabis and schizophrenia. A longitudinal study of Swedish conscripts. *Lancet*. 1987 Dec 26;2(8574):1483-6  
 See also discussion in 7 above.
16. 16. Moderation of the effect of adolescent/onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: longitudinal evidence of a gene X environment interaction. *Biol Psychiatry*. 2005;57:1117-27  
 16a: Di Forti M, Iyegbe C, Sallis H, et al. Confirmation that the AKT1 (rs2494732) genotype influences the risk of psychosis in cannabis users. *Biol Psychiatry*. 2012;72(10):811-816. doi:10.1016/j.biopsych.2012.06.020.
17. <http://www.dailymail.co.uk/news/article-2185854/Clayton-Osbon-JetBlue-pilot-went-bersek-flight-psychotic-episode-prison.html>  
[http://www.huffingtonpost.ca/2013/06/06/screaming-air-canada-pilot\\_n\\_3398171.html](http://www.huffingtonpost.ca/2013/06/06/screaming-air-canada-pilot_n_3398171.html)
18. Lopez-Quintero C et al; probability and predictors of transition from first use to dependence on nicotine, alcohol, cannabis, and cocaine: results of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) Drug alcohol Depend. 2011; 115:120-30
19. <https://www.marijuana-anonymous.org/>
20. Meier et al, Persistent cannabis users show neuropsychological decline from childhood to midlife. *Proc Natl Acad Sci USA* 2012, 109(40): E2657-2564).
21. Stodudo, G (1993), Alcohol and drug use among motor vehicle collision victims admitted to a regional trauma unit: Demographic, injury and crash characteristic. *Accident analysis and prevention*, 25,411-420.
22. <http://www.tsb.gc.ca/eng/rapports-reports/aviation/2011/a11w0151/a11w0151.asp>
23. Leirer, VO et al; *Aviat Space Environ Med*. 1991 Mar;62(3):221-7  
 This study finds evidence for 24-h carry-over effects of a moderate social dose of marijuana on a piloting task. In separate sessions, nine currently active pilots smoked one cigarette containing 20 mg of delta 9 THC and one Placebo cigarette. Using an aircraft simulator, pilots flew just before smoking, and 0.25, 4, 8, 24, and 48 h after smoking. Marijuana impaired performance at 0.25, 4, 8, and 24 h after smoking. While seven of the nine pilots showed some degree of impairment at 24 h after smoking, only one reported any awareness of the drug's effects. The results support our preliminary study and suggest that very complex human/machine performance can be impaired as long as 24 h after smoking a moderate social dose of marijuana, and that the user may be unaware of the drug's influence.