MARIJUANA AND DRIVING IN MICHIGAN:
IDENTIFYING THE “HIGH”

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SECTION I. - INTRODUCING MARIJUANA

Marijuana is the second most popular drug in North America, second only to alcohol, but with the legalization of marijuana becoming an actuality it could soon be number one. In the State of Michigan, marijuana is still illegal for recreational use; however, it is one of the numerous states that has enacted a medical marihuana statute. The Michigan Medical Marihuana Act (hereinafter known as “MMMA”) only applies to specific individuals following the parameters of the Act, yet recreational users fall into a completely different category.

An individual operating a motor vehicle with the presence of any amount of marijuana in his or her blood or system is guilty of drunk driving, which is known as “zero-tolerance.” There has been recent development in case law and its interpretation of marijuana and drunk driving cases, especially for MMMA patients. Still there remains issues with the current ability to actually and faithfully conclude that an individual is “high” or intoxicated while operating a motor vehicle.

The current testing parameters and legislative intent create issues for many individuals because of the severity attached with drunk driving charges, which last for the

1 MAHMOUD A. EL SOHLY, MARIJUANA AND THE CANNABINOIDS 277 (Humana Press, 2007).
2 In 2012, both Colorado and Washington made recreational use, possession, and delivery of marihuana legal within its borders and many states are also considering legalizing or have already passed legislation for medical use, such as Michigan.
3 See MCL § 333.7403 (2012); see also MCL § 333.7404 (2012); see also MCL § 333.26421, et al. (2008).
4 See MCL § 333.26424 (2008); see also MCL § 333.26428 (2008).
5 Compare MCL § 333.26424 and MCL § 333.26428 with MCL § 257.625.
6 MCL § 257.625(8).
8 See People v. Koon, 494 Mich. 1, 832 N.W.2d 724 (2013) (concluding that individual patients qualifying for MMMA protection will not automatically be considered “under the influence” of marijuana simply because it is found within their systems, yet the MMMA does not define “under the influence.” Therefore, as for now those types of drunk driving cases will be based upon the circumstances and facts of the case and whether they substantiate allegations of operating under the influence); see also id. at 8-9.
9 See infra Sections III.-VI., pp. 8-15.
individual’s lifetime. Therefore, drunk driving charges extending from marijuana use or marijuana intoxication must be examined carefully, and will be dependent on the individual facts and circumstances of the case and medicinal versus non-medicinal use.

This article begins with Section II., discussing the pharmacokinetics of the drug, and then explains how the drug actually enters the system in a Section III. Section IV. outlines how the drug is detected while Section V. explains the correlation between detection and intoxication, which leads to Section VI., the conclusion of intoxication or not for MMMA patients.

SECTION II. - MARIJUANA PHARMACOKINETICS

The best way to understand when an individual is or could be intoxicated by marijuana is to examine the internal functioning of the drug once it has entered the body, i.e. the pharmacokinetics. Approximately sixty-five “cannabinoids have been detected in the cannabis plant.” The active ingredient Delta-9-Tetrahydrocannabinol (hereinafter known as “THC”) was not identified until 1964, even though the general chemical structure of phytocannabinoids were characterized thirty years prior to 1964.

Besides THC, the following chemical compounds can also be found within marijuana: THC’S primary metabolite 11-Nor-9-Carboxy-Delta-9-THC (hereinafter known as “THC-COOH”), Cannabidiol (hereinafter known as “CBD”), Cannabinol (hereinafter known as “CBN”), Cannabigerol (hereinafter known as “CBG”), Cannabichromene

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10 See generally, MCL § 257.625
11 The most important factor, currently in Michigan, will be whether the individual is a medical marijuana cardholder or not. Recreational users will continue to be without cause when it is found within their system, unless there is a legislative rewrite; a judicial court overruling the statute itself; or a showing of inadequate testing procedures, methods, or findings within a court of law.
13 Id. (citing W.L. Dewey, Cannabinoid Pharmacology, 38 PHARMACOL. REV. 151 (1986); L.E. Hollister, Health Aspects Of Cannabis, 38 PHARMACOL. REV. 1 (1986)).
(hereinafter known as “CBC”), and a secondary THC metabolite 11-Hydroxy-Delta-9-THC (hereinafter known as “11-OH-THC”). All compounds are a form of THC, cannabis or marijuana.

A majority of users smoke (or inhale) marijuana rather than ingest the substance, which influences the drug’s effects on the individual. The reasoning for smoking is theoretically unknown, but it could be and is likely due to the fact that smoking increases “the efficiency and speed of delivery of the drug.” Essentially, marijuana enters the body immediately as THC; then begins its rapid absorption and distribution within seconds of entering the system. Less than thirty-minutes later, THC is metabolized into 11-OH-THC, and hours later 11-OH-THC is metabolized into THC-COOH, and elimination ensues.

II.A. - ABSORPTION

The “[r]oute of drug administration and drug formulation determine the rate of drug adsorption.” “Cannabinoids interact with a multitude of neurotransmitters and neuromodulators,” which help explain many of the drug’s pharmacological effects on individual users. In the last decade the knowledge obtained concerning THC has increased dramatically, allowing for a better understanding of its kinetics. “Significant

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14 Grotenhermen, supra note 12, at 15.
17 See id. at 21. (indicating that “30% of the THC in marijuana . . . is . . . destroyed by pyrolysis during smoking”) (citing S.L. Dalterio, et al., Acute Delta-9-Tetrahydrocannabinol Exposure Alters Ca2+ Atpase Activity In Neuroendrine And Gonadal Tissues In Mice, 137 EUR. J. PHARMACOL. 91 (1987)).
18 See infra Section II.A., pp. 3-5.
19 See infra Section II.B.-II.D., pp. 5-8
21 Grotenhermen, supra note 9, at 17 (citing Dewey, supra note 12, at 151; D. Baker, et al., The Therapeutic Potential Of Cannabis, 2 LANCET NUEROL. 291 (2003)).
22 Huetis, supra note 16, at 19.
binding . . . [occurs] in the striatum, cerebral cortex, and hippocampus [all] correlating with cannabinoid effects on perception, cognition, memory, learning, endocrine function, food intake, and regulation of body temperature.”\(^{23}\)

It has also been reported that “peak plasma concentrations of 100 to 200 ng/ml are routinely encountered” once the drug has fully absorbed and generally fall below 5 ng/ml within three hours of smoking.\(^{24}\) The exposure to the drug is influenced by “number, duration and spacing of puffs, hold time and inhalation volume,”\(^{25}\) and the drug’s potency.\(^{26}\) When marijuana is smoked it reaches the central nervous system and begins absorption almost immediately.\(^{27}\) It can be detected in plasma within seconds of inhalation, and the peak concentrations have been reported to occur within nine minutes of the last hit or puff sequence.\(^{28}\) The drug then travels from the lungs to the brain and through the rest of the body, yet there is an uncertainty in the amount of dose delivery due to the dynamics involved with smoking.\(^{29}\)

\(^{23}\) Id. at 18 (citing L.D. Chait & J.L. Perry, \textit{Acute And Residual Effects Of Alcohol And Marijuana, Alone And In Combination, On Mood And Performance}, 115 \textit{PSYCHOPHARMACOL.} 340 (1994); D.R. Compton, et al., \textit{Cannabinoid Structure Activity Relationship: Correlation Of Receptor Binding And In Vivo Activities}, 265 \textit{J. PHARMACOL. EXP. THER.} 218 (1993); R.E. Hampson & S.A. Deadwyler, \textit{Cannabinoids, Hippocampal Function And Memory}, 65 \textit{LIFE SCI.} 715 (1999)).


\(^{26}\) \textit{NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., supra note 24, at 9.}

\(^{27}\) See Huestis, \textit{supra} note 16, at 21.


\(^{29}\) See \textit{id.} at 21 (citing S. Agurell, et al., \textit{Pharmacokinetics And Metabolism Of Delta-Tetrahydrocannabinol And Other Cannabinoids With Emphasis On Man}, 38 \textit{PHARMACOL. REV.} 21 (1986); A. Ohlsson, et al., \textit{Single Dose Kinetics Of Deuterium Labeled Delta-1-Tetrahydrocannabinol In Heavy And Light Cannabis Users}, 9 \textit{BIOMED. ENVIRON. MAS. SPECTROM.} 6 (1982)).
Ingested marijuana, on the other hand, will absorb at a slower rate than smoked marijuana, which brings about a delayed peak concentration ranging from four to six hours or one to five hours.\textsuperscript{30} Ingested marijuana also maintains lower peak concentrations as compared to smoked marijuana.\textsuperscript{31} Furthermore, the bioavailability of the drug is reduced during ingestion because of first pass metabolism,\textsuperscript{32} along with “variable absorption [and] degradation of the drug in the stomach.”\textsuperscript{33} It has also been reported that metabolite concentrations of THC are found to be higher than active THC concentrations when the substance is ingested as compared to when it is smoked.\textsuperscript{34}

\textbf{II.B. - DISTRIBUTION}

THC distributes itself throughout the entire body and is bound to lipoprotein, “including the brain . . . while less highly perfused tissues accumulate [the drug] more slowly as THC redistributes from the vascular compartment to tissue.”\textsuperscript{35} The “[s]low release of [the] drug from fat and significant enterohepatic recirculation contribute to THC’s long terminal half-life in plasma,” which has been determined to be 4.1 days or greater for chronic users.\textsuperscript{36} It has also been reported that chronic users have a positive test

\textsuperscript{30} See id. at 21-22 (citing M.E. Wall & H.L. Taylor, \textit{Conjugation Of Acidic Metabolites Of Delta-8 And Delta-9-THC In Man in MARIHUANA ’84—PROCEEDINGS OF THE OXFORD SYMPOSIUM ON CANNABIS} (IRL Press Ltd., 1984); A. Ohlsson, et al., \textit{Plasma Delta-9-Tetrahydrocannabinol Concentrations And Clinical Effects After Oral And Intravenous Administration And Smoking}, 28 CLIN. PHARMACOL. THER. 409 (1980)).


\textsuperscript{32} See NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., supra note 24, at 8.

\textsuperscript{33} Huestis, \textit{supra} note 31, at 1175.

\textsuperscript{34} See id. at 1776 (citing T. Nadulski, et al., 29 ANAL. TOXICOL. 782 (2005)).

\textsuperscript{35} Huestis, \textit{supra} note 16, at 21-22 (citing D.J. Harvey, \textit{Absorption, Distribution, And Biotransformation Of The Cannabinoids in MARIJUANA AND MEDICINE} 91 (Humana Press, 2001)).

\textsuperscript{36} Id. (citing E. Johansson, et al., \textit{Prolonged Apparent Half-Life Of Delta-1-Tetrahydrocannabinol In Plasma Of Chronic Marijuana Users}, 713 J. PHARMA. 111 (1998)).
for THC of 2 ng/ml after 12 hours of abstinence.\(^{37}\) It was further indicated and reported that 1 ng/ml of THC had been found in plasma in chronic users for up-to seven days.\(^{38}\)

Recirculation occurs when, for example, THC enters the brain tissue and is then released to the blood.\(^{39}\) It has been suggested that this slow release from the tissue(s) is correlated to a long half-life for THC in blood and plasma, especially in chronic users who continue to increase the storage of THC each time the drug is used.\(^{40}\) This initial distribution occurs quickly during smoking, yet the release of THC from brain tissue is slower than the rate of release of THC in blood.\(^{41}\)

THC in plasma, however, decreases rapidly after the individual has ceased smoking, which is due likely to the rapid distribution and metabolism of the drug.\(^{42}\) In occasional users it has been found that THC concentrations will fall below the levels of quantitation within eight to twelve hours.\(^{43}\) The levels of quantification found after ceasing use for several hours is said to be 1 to 2 ng/ml, and these levels were indicated to have potential impairment effects on some psychomotor functions.\(^{44}\) However, there was no explanation on the actual impairments or reductions in functionality, but rather simple assumptions or hypothesized opinions.\(^{45}\)

\(^{37}\) See MAHMOUD A. EL SOHLY, MARIJUANA AND THE CANNABINOIDs 284 (Humana Press, 2007).
\(^{39}\) See Huestis, supra note 31, at 1779.
\(^{40}\) Id.
\(^{41}\) Id.
\(^{42}\) See id. at 1778.
\(^{43}\) See NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., supra note 24, at 8.
\(^{45}\) See generally, id. 284.
II.C. - METABOLIZATION

THC is mainly metabolized by the liver, and it has been reported that the clearance rates range from 36L per hour for naïve users to 60L per hour for chronic users.46 “The inactive THC-COOH metabolite and its glucuronide conjugate” are the major end products of biotransformation.47

Essentially, the metabolization of THC can generally be said to occur in two phases.48 Phase-I begins with oxidation reactions of THC, which in turn brings about hydroxylation of THC and leads to the production of the metabolite 11-OH-THC.49 In fact, “[o]xidation of active 11-OH-THC produces the inactive metabolite,” THC-COOH.50 Phase-II involves the metabolism of THC-COOH and the “addition of glucuronic acid,” which in turn “improves water solubility, facilitating excretion, but the renal clearance of these . . . metabolites [are] low due to extensive protein binding.”51 “Other tissues, including brain, intestine, and lung, may [also] contribute to the metabolism of THC.”52

Even though THC concentrations within the blood decrease rapidly after smoking, the concentrations of the THC metabolite begin to increase during this time.53 For chronic users, THC and its metabolite are retained for long periods of time, and it has been

47 EL. SOHLY, supra note 37, at 210.
48 See Huestis, supra note 31, at 1180.
49 Id. (indicating that early research believed that 11-OH-THC was the active substances, but inevitably it was discovered that this was not the case).
52 Id.
53 See Huestis, supra note 50, at 22 (citing Huestis, supra note 44, at 276).
suggested that fatty acid conjugates of THC and 11-OH-THC may be formed, which in-
turn also increases the stability of these compounds in fat.\textsuperscript{54}

**II.D. - ELIMINATION**

Once the elimination process begins, sixty-five percent “of the drug is excreted in
the feces, with approximately” twenty percent in urine.\textsuperscript{55} “After the initial distribution
phase, the rate limiting step in the elimination of THC is its redistribution from lipid depots
to blood,” meaning there is a very slow return of THC from where its originally stored in
the body to the blood.\textsuperscript{56} When THC is stored inside depots it becomes poorly perfused
within tissues, such as fat, and continued use of the substance increases the concentrations
within those areas, bringing about a formation of less accessible depots.\textsuperscript{57} In most cases,
nearly the entire drug is excreted within 5-days; however, this rapid elimination is slowed
dramatically once metabolite concentrations reach a value of 20 to 50 ng/ml in urine.\textsuperscript{58}

**SECTION III. - MARIJUANA EFFECTS**

“People variously use marijuana for its exhilarating, relaxing, hallucinogenic, anti-
nausea, and soporific effect,” and it “is most frequently smoked and less frequently”
ingested.\textsuperscript{59} Once an individual has been exposed to marijuana use alterations in cognitive
and psychomotor impairment occur, bringing about a “high;” however, a tolerance to the

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\textsuperscript{54} See Huestis, \textit{supra} note 31, at 1778 (citing F. Grotenhermen, 42 \textsc{Clin. Pharmacokinet.} 327 (2003)).
\textsuperscript{55} Huestis, \textit{supra} note 50, at 22 (citing M.E. Wall, \textit{et al.}, \textit{Metabolism, Disposition, And Kinetics Of Delta-9-
Tetrahydrocannabinol In Men And Women}, 34 \textsc{Clin. Pharmacol. Ther.} 352 (1983)).
\textsuperscript{56} \textsc{El. Sohly}, \textit{supra} note 37, at 219 (citing E.R. Garrett & C.A. Hunt, \textit{Pharmacokinetics Of Delta-9-
Tetrahydrocannabinol In Dogs}, 66 \textsc{J. Pharm. Sci.} 395-407 (1977)).
\textsuperscript{57} \textit{Id.} at 219.
\textsuperscript{58} See Huestis, \textit{supra} note 31, at 1782-83 (citing D.J. Harvey, \textit{Absorption, Distribution, And
Biotransformation Of The Cannabinoids in Marijuana And Medicine} 91 (Humana Press, 2001); M.M.
Halldin, \textit{et al.}, \textit{10 Drug Metab. Dispos.} 297 (1982)).
\textsuperscript{59} See \textsc{El. Sohly}, \textit{supra} note 37, at 277 (indicating that that the “profile from oral ingestion is much longer,
taking longer for the drug to be absorbed and for the active . . . THC to be distributed”).
effects of the drugs is created over time. The actual effects of the drug begin to appear within five to ten minutes, yet “[t]he degree of effect will differ from individual to individual” because of numerous and varying human and pharmacokinetic factors.

Moreover, it has been reported that “[p]eak blood or plasma THC concentrations occur within a few minutes of the end of smoking and begin a rapid decline as the drug distributes from the central compartment into tissues,” and “the peak effects of the drug occur after the blood concentration has peaked and begun to decline.” In fact, the peak effect will occur once “THC and THC-COOH concentrations have reached equivalency”—generally 30-45 minutes after smoking.

The National Highway Traffic Safety Administration reports that the peak of the “high” lasts 10-30 minutes after smoking while the “high” itself typically remains for approximately 2 hours. Traces of the drug remain in the system for a much longer period, and thus long after the high has ceased. Moreover, “most behavioral and physiological effects return to baseline levels within [three to five] hours after drug use, although some investigators have demonstrated residual effects in specific behavior up to 24 hours,” including paranoia, depression, panic and irritability.

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61 *EL SOHLY*, supra note 37, at 277-78.
62 *Id.* at 283.
65 See supra Section II.B., p. 6.
66 NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., supra note 64, at 10.
67 See *EL SOHLY*, supra note 37, at 277.
It’s interesting to note that “[e]xperimental studies [for operating a motor vehicle] have shown modest functional impairment, but debate exists over how well these . . . studies translate in real-life . . . situations.” However, there are useful indicators revealing impairment, including reddening of the eyes, loss of convergence, or an affect on nystagmus (i.e. the smooth tracking of the eye), which becomes more prominent during high dose conditions. Drug Recognition Experts explain that marijuana symptoms include “a lack of pupillary convergence[,] . . . [p]ulse is usually evaluated; . . . [s]peech may be slow or slurred[,] . . . [possible] stale breath[,]. . . taste buds may be elevated as a result of irritation from the hot smoke[, and] . . . eyes will typically be bloodshot because of the vasodilatory effects of THC on the capillaries of the sclera.”

SECTION IV. - DETECTING MARIJUANA

“Scientific advances have improved [the] ability to identify and quantitate cannabinoids in body fluids; however, the interpretation of the results remains a difficult task.” Typically, in Michigan when there is a probable cause by a law enforcement officer that an individual is under the influence of a controlled substance, i.e. marijuana, he or she will seek to obtain blood from the suspected individual. It has been determined that THC-COOH “has no pharmacological effect on the body and its level in the blood correlates poorly, if at all, to the individual’s level of THC-related impairment.”

68 Mu-Chen Li, et al., Marijuana Use And Motor Vehicle Crashes, 34 EPIDEMIOLOGIC REV. 65 (2012) (citing R.A. Sewell, et al., The Effect Of Cannabis Compared With Alcohol On Driving, 18 AM. J. ADDICT. 185–93 (2009)) (indicating that marijuana use is second to alcohol in the most commonly detected substance in American drivers; however, “it is unclear whether [it] plays a significant role in crash causation”); id. at 66.
69 See EL. SOHLY, supra note 37, at 278.
70 Id. at 280; see also STEPHEN T. TALPINS, THE DRUG EVALUATION AND CLASSIFICATION (DEC) PROGRAM 1-7 (2004).
71 Huestis, supra note 49, at 31.
becomes critical that “test reports are specific as to both the level of . . . THC and its metabolite, THC-COOH, or the results may be misinterpreted.” Blood or plasma levels most closely reflect drug exposure. But even this type of analysis may not provide for an accurate depiction of intoxication or impairment when dealing with marijuana.

Chronic users have had THC concentrations of 2 ng/ml detected after 12 hours of abstinence and up-to 48 hours of abstinence in some cases. It has further been found that THC can be detected for 4 to 6 days after use for frequent users “due to extensive storage.” One study indicated that three of its subjects would have been considered impaired if an impairment cut-off rate of 2 to 5 ng/ml would have been used.

SECTION V. - END RESULTS: INTOXICATION?

It has been stated that impairment is possible when plasma THC concentrations are in-excess of 2 to 3 ng/ml or 1.6 ng/ml in whole blood. However, it has been indicated that it becomes complicated when looking at concentrations of frequent users because of

73 John R. Bradley, Driving Or Being In Actual Physical Control While Under The Influence Of Drugs Or The Combined Influence Of Alcohol And Any Drug in WASH. DUI PRACTICE MANUAL § 3.5 (Westlaw, 2013).
75 See generally, id. at 475.
76 See EL SOHLY, supra note 37, at 284.
78 Huestis, supra note 50, at 25.
79 See Karschner, supra note 74, at 475 (noting that for a 7-day period blood tests revealed individuals with THC concentrations greater than 1 ng/ml).
the possible storage of the drug for hours (and possibly days) after initial or last use.\textsuperscript{81} Therefore, it should be safe to assume that “a practical presumptive concentration of blood THC cannot be related to a measurable level of impairment” or intoxication.\textsuperscript{82} Furthermore, the National Highway Traffic Safety Administration has reported “that no clear relationship has been demonstrated between marijuana smoking and either seriously impaired driving or the risk of accident.”\textsuperscript{83}

It has also been reported that no study has been produced with definitive results “indicat[ing] that drivers who have used marijuana alone are any more likely to a cause serious accident than drug free drivers.”\textsuperscript{84} However, eight studies reveal a significant association between marijuana use and crash risk.\textsuperscript{85} The actual significance between marijuana use and crash rate needs additional research in order to accurately obtain results on crash risk as compared to dose, recency, and administration.\textsuperscript{86} There is “evidence . . . [of] a significant dose-response relationship between marijuana use and the degree of impairing effect[s].”\textsuperscript{87} Dose-response relationship measures the changes of drug effects, which is dependent upon the amount of drug, intensity, and duration of exposure.\textsuperscript{88}

\textsuperscript{81} See id. (citing M.R. Peace, et al., \textit{Performance Evaluation Of Four On-Site Drug-Testing Devices For Detection Of Drugs Of Abuse In Urine}, 24 J. ANAL. TOXICOL. 589 (2000)); see also supra Section II.D., pp. 7-8.  
\textsuperscript{82} Karschner, supra note 74, at 475 (explaining that THC is different than Alcohol, and thus it cannot be used as a practical comparison) (citing R.V. Blanke, et al., \textit{Drug Concentrations And Driving Impairment}, 254 J AM. MED. ASSOC. 2618 (1985); R.L. Hawks, \textit{The Constituents Of Cannabis And The Disposition And Metabolism Of Cannabinoids} in \textit{The Analysis Of Cannabinoids In Biological Fluids}, 125 (R. Hawks ed., 1982)).  
\textsuperscript{83} NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., supra note 77, at 9.  
\textsuperscript{84} Id.  
\textsuperscript{85} See Mu-Chen Li et al., \textit{Marijuana Use And Motor Vehicle Crashes}, 35 EPIDERMOL. REV. 65, 69 (2012).  
\textsuperscript{86} See id. at 70.  
\textsuperscript{87} MAHMOUD A. EL SOHLY, MARIJUANA AND THE CANNABINOIDS 291 (Humana Press, 2007).  
Furthermore, there are two mathematical models used to predict the time of cannabis use, which have been tested and revealed a ninety-five percent “confidence interval for more than [ninety percent] of the specimens evaluated.”  These models, due to a high rate of confidence, have been evaluated and are being used in criminal matters by forensic examiners to assist with explaining and interpreting impairment. The National Highway Traffic Safety Administration, on the other hand, indicated that it is inadvisable to predict effects based on blood THC concentrations alone, yet it has upheld the use of the mathematical models to possibly predict impairment.

The real difficulty in analyzing impairing effects of marijuana is due to the combined use of other drugs or alcohol within an individual’s system during studies evaluating the effects on the system. However, “[s]tudies that examine the casual effect through responsibility analysis . . . [again] indicated that THC alone did not increase accident risk.” Furthermore, the variation of effects associated with marijuana as compared to alcohol is far greater, which likely adds to the difficulty in determining marijuana intoxication.

It is known that an increase in driving risk is found when marijuana and alcohol have been used together as compared to using either substance on its own. Even though it

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90 See id. at 31-32 (citing J.E. Man, *Temporal Indication Of Marijuana Use Can Be Estimated From Plasma And Urine Concentrations Of Delta-9-Tetrahydrocannabinol, 11-Hydroxy-Delta-9-Tetrahydrocannabinol, And 11-Nor-Delta-9-Tetrahydrocannabinol-9-Carboxylic Acid*, 25 J. ANAL. TOXICOL. 538 (2001)).


92 See id., supra note 48, at 44.

93 See id.

94 Mu-Chen Li, supra note 85, at 70 (citing R.A. Sewell, et al., *The Effect Of Cannabis Compared With Alcohol On Driving*, 18.3 AM. J. ADDICT. 185–93 (2009)).

95 NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., *supra* note 77, at 11.
has been reported that THC affects the cognitive and psychomotor tasks of an individual, which are both associated with driving, “any situation in which safety both for self and others depends upon alertness and capability of control of man-machine interaction precludes the use of marijuana.” 96 Furthermore, it has been stated that “subject[s] under marijuana treatment appear to perceive that they are indeed impaired, . . . [and w]here they can compensate, they do, for example by not overtaking, by slowing down, and by focusing their attention when they know a response will be required.” 97

Researchers have concluded that the effects of marijuana use on the individual are based upon numerous facts, which bring about inconsistent findings on cognitive functioning. 98 Those factors include “age, education, experience with the drug, gender and moods, . . . user expectations, . . . [and] manner in which marijuana is smoked.” 99 It has also been reported that the frequency and quantity smoked will change the effects on individuals, i.e. infrequent users maintain higher manual dexterity than chronic users. 100

SECTION VI. - UP IN-SMOKE?

The Michigan Supreme Court indicated that MMMA patients are not subjected to the per-say rule found in MCL § 257.652, and thus the State must show that the individual was actually under the influence of marijuana rather than simply having trace amounts of

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96 Huestis, supra note 89, at 44-45 (2002) (quoting H. Moskowitz, Marijuana And Driving, 17 ACID. ANAL. PREX. 323 (1985)).
97 NAT’L HIGHWAY TRAFFIC SAFETY ADMIN., supra note 77, 9 (quoting A.M. Smiley, et al., The Effects Of Marijuana Alone And In Combination With Alcohol On Driving Performance in ALCOHOL, DRUGS & TRAFFIC SAFETY, PROCEEDINGS OF THE 10th INTERNATIONAL CONFERENCE ON ALCOHOL, DRUGS & TRAFFIC SAFETY 133 (P.C. Noordzij & R. Roszbach eds., 1986)).
99 Id. at 286 (citing MARIJUANA AND THE WORKPLACE 6-7 (Charles R. Schwenk & Susan L. Rhodes eds., 1999)).
100 Id. at 287.
the drug in his or her system. Generally, unless the individual smoked a short time prior to driving, his or her levels will maintain a relatively low concentration of THC. Individuals protected by the MMMA can question these levels provided when charged with drunk driving. The scientific community understands some aspects concerning marijuana use and intoxication; however, it is limited and varying.

Therefore, attempting to use a per-say limit or quantification for MMMA patients in order to distinguish between sober driving and intoxicated driving is difficult to validate within the criminal justice system. Law enforcement, and almost all of the scientific community, are unable to specify a level of concentration for THC that correlates with intoxication (or being high) and the level of THC found in an individual’s blood, except when the concentrations are extremely high or peaking.

When handling a drunk driving matter involving an MMMA patient, it is essential to identify the correlation between a concentration level and actual intoxication or impairment. Unless the THC level associated with intoxication is extremely exasperated by an examiner in court, a typical level of THC will likely be low, and thus the possibility of intoxication is minimized. However, rebutting the State’s claim of intoxication, i.e. impairment, for the MMMA patient may require a defense expert or harsh examination of all the State’s experts and intoxication claims and facts.

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102 See supra Section IV., pp. 10-11.
103 This is irrelevant for recreational users within Michigan because of the zero-tolerance standard placed upon them, even though they may not be high or intoxicated; therefore, the issue regarding operating under the influence or while intoxicated is one limited to MMMA patients, unless there is a finding of error during the testing procedure bringing about a false positive otherwise contaminated specimen and test.
104 See supra Section V., pp. 11-14
105 See supra p. 11