COMMONWEALTH OF MASSACHUSETTS

SUFFOLK, ss **BOSTON MUNICIPAL COURT**
CENTRAL DIVISION
DOCKET # 0701CR7229

COMMONWEALTH)
)
v.)
)
RICHARD CUSICK)
Defendant)

and

DOCKET # 0701CR7230

COMMONWEALTH)
)
v.)
)
RUSSELL K. STROUP)
Defendant)

AFFIDAVIT OF LESTER GRINSPOON, MD IN SUPPORT OF DEFENDANTS' MOTION TO DISMISS, AND REQUEST FOR AN EVIDENTIARY HEARING ON THE RATIONALITY OF THE STATUTE CONSTRAINED BY THE ADMISSIBILITY TEST FOR EXPERT SCIENTIFIC TESTIMONY ARTICULATED IN COMMONWEALTH V. LANIGAN

I, Lester Grinspoon on oath depose and state:

1) I am a resident of Wellesley, Massachusetts.

2) My name is Lester Grinspoon. I am an associate professor of psychiatry, emeritus at Harvard Medical School. Shortly after graduating from Harvard Medical School in 1955, I joined the faculty and over the ensuing 45 years I combined research and teaching with clinical practice. My curriculum

vitae is attached.

3) I am the author of *Marihuana Reconsidered* (published by Harvard University Press in 1971, second edition in 1977, republished as a classic in 1997). Based on my research I concluded that marijuana was far less harmful to the individual than the use of either alcohol or tobacco. I further concluded that the greatest harm associated with marijuana arose not from any inherent psychopharmacological properties of the drug, but rather from the way we as a society were dealing with it.

4) In the time since my initial research, I have devoted much of my professional career to studying the physical and psychological effects of marijuana, and I have to this day remained current and up to date with the latest science in this area.

5) The Massachusetts Supreme Judicial Court stated in Marcoux:

> *On review, we took note of the scientific evidence and concluded, as has the trial judge, that the Legislature could believe with reason that the use of marijuana created dangers both to users and others justifying public control. Marijuana is a psychoactive drug whose immediate effects may include inducement of a state of euphoria or anxiety or panic, reduction in motor control of, and alterations of time perceptions and memory. Although the causal links could be disputed, there was ground to suspect that the use of the drug was*

> *a factor in psychotic incidents experienced by some smokers, in the descent of users to harder and more dangerous drugs, and in the occurrence of highway accidents. As to the last danger, the difficulty or impossibility of testing drivers* for *marijuana intoxication could be thought to justify a ban on the substance rather than a qualified regulation of it.* [Marcoux, v. Atty.General, 375 Mass.63, page 65]

6) In this affidavit I will first address the three above noted areas of concern expressed in the Marcoux decision. I will then address several additional areas of concern that have arisen since that 1978 decision.

Marijuana and Mental Health

7) Over a century of research reveals that marijuana does not cause mental illness. To date, the 3,281 page report of the Indian Hemp Drugs Commission remains the most comprehensive work on this topic. It documents no association between use of marijuana and mental illness when other contributors to mental illness are taken into account.[1]

8) More recent research also confirms that marijuana use does not cause psychological problems.[2] A longitudinal study of 800 New Zealanders revealed no

[1] Indian Hemp Drugs Commission (IHDC) (1894). Report of the Indian hemp drugs commission. Simla, India: Government Central Printing Office.

association between marijuana use and the onset of anxiety and mood disorders.[3] Compelling evidence also comes from epidemiological studies of different nations. Countries with relatively high rates of marijuana use within their populations have no statistically higher rates of mental illness than countries where marijuana use is less prevalent.[4]

9) Those few papers that do suggest modest links between marijuana and mental illness often fail to properly control for other contributing factors. These contributors include subjects' use of cigarettes,[5] alcohol,[6] hard drugs[7] and the onset of

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[2] Denson, T. & Earleywine, M. (2006). Decreased depression in marijuana users. Addictive Behaviors, 31, 738-742.

[3] McGee, R., Williams, S. A., Poulton, R. & Moffitt, T. (2000). A longitudinal study of cannabis use and mental health from adolescence to early adulthood. Addiction, 95, 491-503.

[4] Hall, W. & Solowij, N. (1998). Adverse effects of cannabis. Lancet, 352, 1611-1616.

[5] Cujpers, P. Smit, F., ten Have, M., de Graaf, R. (2007). Smoking is associated with first-ever incidence of mental disorders: a prospective population-based study. Addiction, 102, 1303-1309.

[6] Fidalgo, T., da Silveira, E. & da Silveira, D. (2008). Psychiatric comorbidity related to alcohol use among adolescents. The American Journal Of Drug And Alcohol Abuse, 34, 83-89.

[7] Bosack, C., Camus, D., Kaufmann, N., Aubert, A. C., Besson, J., Baumann, P., Borgeat, F. Gillet, M., & Eap, C. B. (2006). Prevalence of substance use in a Swiss psychiatric hospital: interview reports and urine screening.

symptoms occurring prior to marijuana use. In addition, many of these studies suffer from methodological problems that make it impossible to conclude whether or not marijuana caused a disorder. Inappropriate generalizations from small samples, statistical errors, biases in the assessment of symptoms, or failing to distinguish between momentary intoxication and chronic disorders are common in this literature. Once the potential confounders are taken into account, the link between marijuana and mental illness disappears.[8]

10) For example, one commonly cited article in the scientific literature claims to show increased anxiety in young women who smoke marijuana daily.[9] However, a close examination of this study reveals that while the authors extensively assessed subjects' marijuana use for the previous six months, they only assessed the subjects' use of alcohol, cigarettes, and hard drugs for the previous seven days. Obviously, participants in the study could have used these drugs extensively prior to the

[8] Earleywine, M. (2002). *Understanding Marijuana*. Oxford University Press. New York: New York.

previous week. In fact, subjects might have chosen to stop using these same drugs because of an anxiety reaction. Nevertheless, investigators' failure to assess other drug use in the same manner that they assessed marijuana use artificially inflates the link between marijuana and mental health problems. Other frequently-cited studies on this topic make comparable mistakes when assessing for subjects' use of cigarettes, alcohol, or hard drugs.[10] [11]

11) Additionally, the literature on marijuana and mental illness frequently confuses self-medication of symptoms with factors that might generate disorders. If marijuana actually caused mental illness, marijuana use must precede the disorder. By contrast, research suggests that symptoms precede

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[9] Patton, G.C., Coffey C, Carlin J.B., Degenhardt L., Lynskey M., Hall W. (2002) Cannabis use and mental health in young people: cohort study. BMJ 325, 1195-1198.

[10] Bovas, G. (2001). Cannabis abuse as a risk factor for depressive symptoms. American Journal of Psychiatry, 158,2033-2037.

[11] Caspi, A., Moffitt, T., Cannon, M., McClay, J., Murray, R., Harrington, H., Taylor, A., Arseneault, L., Williams, B., Braithwaite, A., Poulton, R., & Craig, I. (2005). 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. Biological Psychiatry, 57, 1117-1127.

rather than follow marijuana consumption. [12] [13] Once initial symptoms of the disorder are taken into account, the link between marijuana and mental illness invariably shrinks.

12) Another common methodological error in this literature involves authors making gross generalizations based on small samples of individuals with psychological problems. For example, a widely-cited study purports to link marijuana abuse with depression.[14] However, a close look at this study reveals that only 10 people of a sample of 1,920 were diagnosed with cannabis abuse and subsequently developed depression.

13) Another widely-cited article claims that people with a specific genetic risk who are heavy cannabis users early in life are more likely to develop schizophreniform disorder.[15] The results are

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[12] Thornicraft, G. (1990). Cannabis and psychosis: Is there epidemiological evidence for an association? British Journal of Psychiatry, 157, 25-33.

[13] Schiffman, J., Nakamura, B., Earleywine, M., & LaBrie, J. (2005). Symptoms of schizotypy precede cannabis use. Psychiatry Research, 134, 37-42.

[14] Bovas, G. (2001). Cannabis abuse as a risk factor for depressive symptoms. American Journal of Psychiatry, 158,2033-2037.

[15] Caspi, A., Moffitt, T., Cannon, M., McClay, J., Murray, R., Harrington, H., Taylor, A., Arseneault, L., Williams, B., Braithwaite, A., Poulton, R., & Craig, I. (2005).

reported in percentages, but a simple conversion reveals that the number of people who have the genetic risk, early cannabis use, and schizophreniform disorder is 7 (out of 803). Generalizations from such small samples are patently absurd. (Both of these studies also failed to provide proper controls for cigarette, alcohol, and hard drug use.)

14) Studies like these often lose their statistical significance if a single cannabis user is removed from the sample. For example, recent media reports suggest that psychiatric disorders are more prevalent in multiple sclerosis (MS) patients who use marijuana medicinally, based on an in-press article.[16] The study actually found only 9 MS patients with psychological distress who also used medical marijuana. If the number had been 8 instead of 9, the effect would not have reached statistical significance. There would have been no headlines

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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. Biological Psychiatry, 57, 1117-1127.

[16] Ghaffar, O., and Feinstein, A. (In Press; 2008). Multiple sclerosis and cannabis: A cognitive and psychiatric study. Neurology.

proclaiming a lack of mental illness in marijuana users, either.

15) Biases in the assessment of symptoms also commonly appear in this literature. At least five studies purport to link marijuana use to symptoms of schizotypal personality disorder, an illness typically associated with eccentric beliefs and odd experiences. [17] [18] [19] [20] [21] Schizotypal personality disorder is often assessed with The Schizotypal Personality Questionnaire.[22] Recent work reveals that items on this questionnaire are biased against cannabis users, making them appear to have symptoms when they do not. For example, one item asks

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[17] Skosnik, P., Spatz-Glenn, L., Park, S., 2001. Cannabis use is associated with schizotypy and attentional disinhibition. Schizophren. Res. 48, 83-92.

[18] Mass, R., Bardong, C., Kindl, K., Dahme, B. (2001). Relationship between cannabis use, schizotypal traits, and cognitive function in healthy subjects. Psychopathology 34, 209-214.

[19] Dumas, P., Saoud, M., Bouafia, S., Gutknecht, C., Ecochard, R., Dalery, J., Rochet, T., d'Amato, T. 2002. Cannabis use correlates with schizotypal personality traits in health students. Psychiatry Research, 109, 27-35.

[20] Bailey, E. & Swallow, B. (2004). The relationship between cannabis use and schizotypal symptoms. European Psychiatry, 19, 113-114.

[21] Schiffman, J., Nakamura, B., Earleywine, M., & LaBrie, J. (2005). Symptoms of schizotypy precede cannabis use. Psychiatry Research, 134, 37-42.

[22] Raine, A. 1991. The SPQ: A scale for the assessment of schizotypal personality based on the DSM-III-R criteria. Schizophrenia Bulletin, 17, 555-564.

participants to respond True or False to the statement “I use words in strange and unusual ways.” Marijuana users are more likely than non-users to respond “True” to this statement. However, it would be expected that subjects who may come from a marijuana subculture with its somewhat unusual slang and argot might claim to use words in strange and unusual ways. It is not a sign of pathology. When this item is removed from the questionnaire, the scores of marijuana users and non-users are equivalent.[23]

16) A further issue related to diagnosis concerns the separation of momentary intoxication from symptoms of disorders. A great many studies of marijuana and psychological functioning have confounded genuine symptoms of pathology with transient effects of the plant. Marijuana intoxication creates experiences that many users deem pleasant, but these experiences can also appear odd. If people report these experiences but do not explain that they were induced by cannabis, researchers can misinterpret the experiences as a sign of psychosis. Large doses

[23] Earleywine, M. (2006). Schizotypy, marijuana, and differential item function. Human Psychopharmacology: Clinical and Experimental, 21, 455-461.

of ingested marijuana or of hashish can create odd experiences, but this situation is not the same as schizophrenia. It lacks the formal thought disorder and inappropriate emotions.[24] It also dissipates relatively quickly, while schizophrenia remains a chronic mental illness. Few of the studies performed to date have separated genuine symptoms of a disorder from fleeting moments that occurred subsequent to marijuana use. Thus, they make marijuana users look more deviant.

17) A recent meta-analysis by Moore, et al.[25] of seven longitudinal studies of the relationship of marijuana and psychoses has received a remarkable amount of attention, particularly in the lay literature. None of these studies randomly assigned people to use cannabis, of course, so individuals who might be prone to psychosis as well as cannabis use could have accounted for results. That is, these results are correlational, not causal. A predisposition to develop psychosis might vary with

[24] Basu, D., Malhotra, A., Bhagat, A. & Varma, V. K. (1999). Cannabis psychosis and acute schizophrenia: A case control study from India. European Addiction Research, 5, 71-73.

[25] Moore,T.H., Zammit,S., Lingford-Hughes,A.,Barnes, T.R, Jones, P.B. & Lewis,G. (2007). Cannabis use and risk of

a tendency to try cannabis, but neither would need to cause the other.

18) The general approach of the studies included in the Moore et al. analysis was to divide people into putatively psychotic and putatively normal groups. The researchers then compared the proportions of non-users to the proportions of marijuana users who end up in the group labeled psychotic. Three of the studies actually showed no difference in the rates of being labeled psychotic when comparing those who ever used marijuana to those who did not. Averaging across all seven studies, the people who used marijuana were 40% more likely to be placed in the psychotic group than those who did not. Nevertheless, a close look at the original studies reveals that there is markedly less here than meets the eye. Although a 40% increase sounds like a great deal, this effect is actually small for such studies. To put the results in perspective, recent data show that people who smoke cigarettes early in

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psychotic or affective mental health outcomes: a systematic review. Lancet, 28, 319-28.

life show a 128% increase in the rate of a schizophrenia diagnosis.[26]

19) Unfortunately, media accounts of the Moore paper suggested a 40% increase in the chance of developing schizophrenia, which is patently untrue. If the results implied a 40% increase in the chance of a marijuana user developing schizophrenia, the prevalence of schizophrenia should have increased significantly over the last four decades; no such increment has been identified. The problem apparently lies largely in the way in which psychosis was determined in the original studies.

20) The determination of who was and who was not in the psychotic group is very misleading in the majority of these studies. Schizophrenia is one of a number of psychotic disorders. Because schizophrenia is so rare, six of the seven studies examined psychosis very broadly defined; only one of these studies actually examined schizophrenia as an outcome. Of the remaining studies, one looked at

[26] Weiser, M., Reichenberg, A., Grotto, I., Yasvitzky, r., Rabinowitz, J., Lubin, G., Nahon, D., Knobler, H., & Davidson, M. (2004). Higher rates of cigarette smoking in male adolescents before the onset of schizophrenia: a historical-prospective cohort study. The American Journal of Psychiatry, 161, 1219-1223.

schizophreniform disorder. Schizophreniform disorder is a less serious condition with symptoms comparable to those of schizophrenia that last at least one month. Nevertheless, this study did not require any impairment in subjects' functioning. Thus, perfectly productive people who did not consider themselves ill in any way could be placed in the psychotic group. The remaining studies chose to look at any allegedly psychotic symptom. These studies labeled those people as psychotic, again, without requiring any evidence of subjects' impairment. Thus, the participants in these studies did not need to demonstrate hallucinations or poor functioning to be considered to be suffering from a psychotic disorder. Instead, the people labeled psychotic in these studies could be anyone who is not particularly emotional (flat affect, a decrease in emotional range, is one of the symptoms of schizophrenia).

21) Participants with a single odd belief could also get labeled psychotic. For example, the idea that one is watched by the police could be labeled psychotic, but it could in fact be real for people who use an illicit drug. Thus, although the media

misinterpreted these results to mean that marijuana use leads to a 40% increase in the chances of someone who has used marijuana in the past becoming schizophrenic, they actually mean that there might be a 40% increase in being unemotional or a little odd.

22) Nevertheless, even with these caveats, this 40% increase is actually an overestimation. As the Moore paper reveals, this 40% increase does not take into account the use of other drugs or the presence of other confounding factors like previous mental illness, personality traits, or low socioeconomic status. Of the studies that were reviewed, when other drug use was assessed, it was often not done so with the same degree of detail as the assessment of cannabis use. By including other drugs and other potential confounders Moore et al. found that the size of the link between cannabis and psychosis dropped an average of 45%. Thus, the 40% increase is actually a 22% increase on average once the use of other drugs is taken into account. Although this increase remained statistically significant, it is at odds with the fact that there has been no increase over the last four decades (a period over

which there has been a significant increment in the use of marijuana) in the prevalence of schizophrenia.

23) Media accounts have also misinterpreted research and implied that marijuana causes depression. This assertion is contrary to the experience of many clinicians who since the mid-19th century have found that cannabis is useful as an antidepressant in many patients.[27] Recent work suggests that elevated rates of depression in marijuana users disappear once people who use it for medical conditions are omitted from the sample. As one would imagine, people who use cannabis in an effort to combat aspects of cancer and AIDS are more depressed than others.[28] Thus, some studies report an artificial link between depression and marijuana use.

24) Other work that purports to link marijuana and depression suffers from methodological problems as well. For example, a widely-cited study purports to

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[27] Grinspoon,L. and Bakalar,J.B. *Marijuana, the Forbidden Medicine*, Yale University Press, Second Edition, 1997, p138.

[28] Denson, T. & Earleywine, M. (2006). Decreased depression in marijuana users. Addictive Behaviors, 31, 738-742.

link marijuana abuse with depression.[29] A close look reveals that only 10 people of a sample of 1,920 were diagnosed with cannabis abuse and subsequently developed depression. A recent study purporting to link marijuana to suicidal thought in high school students failed to determine if the suicidal thoughts occurred before or after students tried cannabis.[30] Another study on suicidal thoughts in Aborigines ran over 14 different statistical tests and failed to control for the fact that many participants were also inhaling gasoline fumes and using other drugs.[31] Errors like these are common in this literature, suggesting that there is little genuine evidence for the idea that marijuana catalyzes depression.

25) The sum of these errors makes it clear that any link between marijuana and mental illness is illusory and likely stems from methodological

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[29] Bovas, G. (2001). Cannabis abuse as a risk factor for depressive symptoms. American Journal of Psychiatry, 158,2033-2037.

[30] Chabrol, I., Chauchard, E., & Girabet, J. (2008). Cannabis use and suicidal behaviours in high-school students. Addictive Behaviors, 33, 152-155.

[31] Clough,A.R., Lee,K.S., Cairney, S., Maruff, P., O'Reilly,B. d'Abbs.P,& Conigrave, K.M. (2006). Changes in cannabis use and its consequences over 3 years in a remote

artifacts or confounding with previous psychological distress or hard drug use.

Marijuana and the Gateway Myth

26) Although adverse effects of marijuana are minimal, many worry that use of the plant might influence users toward the use of hard drugs. If it were the case that marijuana leads to problems with hard drugs, three statements must be true: (1) marijuana use must correlate with hard drug problems, (2) the associations must not arise because of a factor common to both, and (3) the use of marijuana must precede the development of hard drug problems.

27) Any causal relationship must show an association, the precedence of the cause, and the absence of any alternative, third-variable explanations. In fact, the correlation between marijuana use and hard drug problems is small, many who experience problems with hard drugs do not use marijuana first, and any association between marijuana and hard drugs arises because of factors common to both. Essentially, the link between marijuana and hard drug problems is

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indigenous population in northern Australia. Addiction, 101, 696-705.

spurious.

28) An extreme example can help illustrate the error in reasoning common among proponents of the gateway theory. Suppose data revealed that the crime rate in a city rises as the number of churches increases. This association might lead some cynic to hypothesize that churches cause crime. Data may suggest that the churches are built prior to the increases in crime, further supporting the theory. Both the association between the number of churches and crime, and the precedence of the churches appear. Nevertheless, these two facts alone do not establish that churches cause crime. An alternative explanation remains: as cities grow larger, both crime and the number of churches increase. The size of the population accounts for both of these increases. A third variable accounts for the association making it clear that churches do not, in fact, cause crime.

29) Similar evidence is actually available for marijuana and hard drugs. A small association exists between the two, but it arises because of personality traits in hard drug users, not an effect of marijuana. In addition, hard drug use does not

invariably follow marijuana use and many people who use hard drugs problematically did not start with marijuana first.

30) National survey data reveal that the correlation between marijuana and hard drugs is small. The vast majority of people who try marijuana never try hard drugs. Most regular marijuana users do not become regular users of hard drugs, and fewer people still develop hard drug problems. Statistically, the chance of becoming a regular user of crack cocaine after trying marijuana is less than 1 in 200. The chance of becoming a regular user of heroin after trying marijuana is less than 1 in 333.[1] To put these numbers in perspective, this is worse than the odds of flipping a coin and getting "Heads" 8 times in a row. Gateway proponents suggest that hard drug problems are more common among those who use marijuana regularly, and some studies find a small but statistically significant effect consistent with this idea, but a close look at the work reveals that this association arises because of factors common to both behaviors, not because one causes the other.

[1] Earleywine, M. (2002). Understanding Marijuana. Oxford University Press. New York: New York.

31) Marijuana does not cause the use of hard drugs. A subset of people who develop problems with hard drugs also happen to use marijuana, based on aspects of personality, neighborhood, and parenting. Miller[2] analyzed data from 4 national surveys including over 6,000 participants and found that characteristics of individuals, not the cannabis plant, account for links between marijuana and hard drugs. The small subset of individuals in this sample who used marijuana and hard drugs essentially accounted for the entire association between the two. Another study of over 58,000 people found that marijuana use is not causally linked to hard drug use (even non-problematic use) once characteristics of the individual are taken into account. When these individual characteristics are ignored, as they often are in most research on this topic, the spurious association between marijuana and hard drugs returns.[3] Essentially, marijuana does not cause hard drug problems, but a subset of people who used marijuana also happened to develop trouble with

[2] Miller, T. Q. (1994). A test of alternative explanations for the stage-like progression of adolescent substance use in four national samples. *Addictive Behaviors*, 19, 287-293.

hard drugs. It is the individual's propensity to use drugs, and not an effect of marijuana, that creates the illusion that marijuana co-varies with hard drug problems.

32) Finally, if marijuana caused hard drug problems, its use would have to precede these hard drug problems. In fact, studies of treatment samples of people who live in neighborhoods of low socioeconomic status consistently show 12 to 39% use hard drugs prior to use of marijuana.[4] [5] An illustrative study showed that 39% of a sample of active hard drug abusers and distributors used hard drugs before trying cannabis.[6] Clearly, the alleged path from marijuana to hard drug use is not invariable. Most marijuana users do not use hard

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[3] Morral, A., McCaffrey, D. F., Dock, S. M. Reassessing the marijuana gateway effect. *Addiction*, 97, 1493-1504.

[4] Golub, A. and Johnson, B. D. (1994). The shifting importance of alcohol and marijuana as gateway substances and among serious drug abusers. *Journal of Studies in Alcohol*, 55, 607-14.

[5] Blaze-Temple, D. & Lo, S. K. (1992). Stages of drug use: a community survey of Perth teenagers. *British Journal of Addiction*, *87*, 215-225.

[6] Mackesy-Amiti, M. E., Fendrich, M. & Goldstein, P. J. (1997). Sequence of drug use among serious drug users: typical vs. atypical progression. *Drug and Alcohol Dependence,* 45, 185-196.

drugs and many hard drug users do not start with marijuana.

33) Marijuana use clearly does not cause hard drug problems. The alleged gateway pattern can be explained by common characteristics of those who use marijuana and other drugs.

Marijuana and Driving

34) Marijuana is the most common illicit substance consumed by motorists who report driving after drug use.[1] Epidemiological research also indicates that marijuana is the most prevalent illicit drug detected in fatally injured drivers and motor vehicle crash victims.[2] There are two reasons for this: first, marijuana is by far the most widely used illicit drug in the United States with nearly one out of two Americans admitting to have tried it[3] and second, cannabis is the most readily detectable illicit drug in toxicological tests. The primary

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[1] US Department of Health and Human Services, Substance and Mental Health Services Association, Office of Applied Studies. *Driving After Drug or Alcohol Use*, 1998. http://www.oas.samhsa.gov/driverrprt/toc.htm

[2] US Department of Transportation, *National Highway Traffic Safety Administration. The State of Knowledge of Drugged Driving: Final Report.* September, 2003. http://www.nhtsa.dot.gov/people/injury/research/StateofKnwlegeDrugs/StateofKnwlegeDrugs/

psychoactive compound in cannabis, tetrahydrocannabinol (THC), may be detected in blood for days after last use, long after any psychoactive effects have disappeared.[4] Furthermore, non-psychoactive byproducts of marijuana, known as metabolites, may be detected in the urine of regular users for weeks after last use.[5] Other common drugs of abuse such as alcohol, cocaine and amphetamines are detectable for only short periods of time after their last use. Therefore, because marijuana's prevalence in toxicological evaluations of US drivers does not necessarily indicate psychoactivity at the time of the urine test, the results of those tests do not indicate causality in automobile accidents. Rather, its prevalence merely affirms that cannabis is far more popular and is far more easily detectable on drug screening tests than other psychoactive substances.

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[3] October 23-24, 2002 CNN/Time poll conducted by Harris Interactive.

[4] Karschner et al. 2008. Plasma cannabinoid concentrations in Davie cannabis users during seven days of monitored abstinence. In*: Proceedings of the American Academy of Forensic Sciences*, Annual Scientific Meeting: February 18-23, Washington, DC.

[5] Paul Cary. 2005. The marijuana detection window: determining the length of time cannabinoids will remain

35) While it is well established that alcohol consumption increases accident risk, evidence of marijuana's culpability in driving accidents and injury is far less clear. Although acute cannabis intoxication following marijuana smoking has been shown to mildly impair psychomotor skills, this impairment is seldom severe or long-lasting. In closed course and driving simulator studies, the acute effects on psychomotor performance include minor impairments in tracking (eye movement control) and reaction time, as well as variation in lateral positioning, headway (drivers under the influence of cannabis tend to follow less closely to the vehicle in front of them), and speed (drivers tend to decrease speed following marijuana inhalation).[6] In

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detectable in urine following smoking. *Drug Court Review* 5: 23-58.

[6] Franjo Grotenhermen. *Drugs and Driving: Review for the National Treatment Agency, UK.* Nova-Institut (Germany). November 2007. Other summaries include: Ramaekers et al 2006. Cognition and motor control as a function of delta nine THC concentration in serum and oral fluid: Limits of impairment. *Drug and Alcohol Dependence* 85: 114-122; David Hadorn, "A Review of Cannabis and Driving Skills," In: *The Medicinal Uses of Cannabis in Cannabinoids.* (eds:Guy et al). Pharmaceutical Press, 2004; Canadian Senate Special Committee on It Illegal Drugs, *Cannabis: Summary Report; Our Position for a Canadian Public Policy, 2002. (*See specifically Chapter 8*: "Driving under the Influence of Cannabis"*); Alison Smiley, "Marijuana: On-road and Driving Simulator Studies," In: *The Health Effects of Cannabis*

general, these variations in driving behavior are noticeably less consistent or pronounced than the impairments exhibited by subjects under the influence of alcohol.[7] Also, unlike subjects impaired by alcohol, individuals under the influence of cannabis tend to be aware of their impairment and try to compensate for it accordingly, either by driving more cautiously[8] or by expressing an unwillingness to drive altogether.[9]

36) Accordingly, cannabis-induced variations in performance do not appear to play a significant role in on-road traffic accidents when THC levels in a driver's blood are low and/or marijuana is not

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(eds. Kalant et al) Canadian Centre for Addiction and Mental Health, 1999.

[7] David Hadorn, 2004,op cit. and US Department of Transportation, 2003, op cit.

[8] According to the US Department Of Transportation, 2003, op.cit., "the extensive studies by Robbe and O'Hanlon (1993), revealed that under the influence of marijuana, drivers are aware of their impairment, and when the experimental task allows it, they tend to actually decreased speed, avoid passing of the cars, and reduce the risk-taking behaviors."

[9] Menetrey et al. 2005. Assessment of driving capability through the use of clinical and psychomotor tests in relation to blood cannabinoid levels following oral administration of 20 mg dronabinol or a cannabis decoction made with 20 and 60 delta-9-THC. *Journal of Analytical Toxicology* 29: 327-338. http://www.ingentaconnect.com/content/pres/jat/2005/00000029/00000005/art00008;jsessionid=34n6g1asv4qhe.alice?format=print&crawler=true

consumed in combination with alcohol.[10] [11] For example, a 1992 National Highway Traffic Safety Administration review of the role of drug use in fatal accidents reported, "There was no indication that cannabis itself was a cause of fatal crashes" among drivers who tested positive for the presence of marijuana. [12] A more recent assessment by Blows and colleagues noted that self-reported recent use of cannabis (within three hours of driving) was not significantly associated with car crash injury after investigators controlled for specific confounders (e.g., seat-belt use, sleepiness, etc.)[13] A 2004 observational case-control study published in the journal *Accident, Analysis and Prevention* reported that only drivers under the influence of alcohol or benzodiazepines experience an increased crash risk compared to drug-free controls. Investigators did

[10] United Kingdom Department of Environment, Transport and the Regions, Road Safety Division; *Cannabis and Driving: a Review of the Literature and Commentary*.

[11] Gregory Chesher and Marie Longo. Cannabis and alcohol in motor vehicle accidents," *In: Cannabis and Cannabinoids: Pharmacology, Toxicology, and Therapeutic Potential.* (eds. Grotenhermen et al.) Haworth Press, 2002

[12] US Department Of Transportation, National Highway Traffic Safety Administration. *The Incidence and Role of Drugs in Fatally Injured Drivers: Final Report.* October, 1992

observe increased risks (though they were not statistically significant) among drivers using amphetamines, cocaine and opiates. They reported, "No increased risk for road trauma was found for drivers exposed to cannabis."[14]

37) A handful of more recent studies have noted a positive association between very recent cannabis exposure and a gradually increasing dose-related risk of vehicle accident. Typically these studies reveal that drivers who possess THC concentrations above 5ng/ml of blood (implying very recent marijuana inhalation and acute intoxication) [15] [16] experience an elevated risk of accident compared to drug-free controls.[17] [18] Motorists who test positive

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[13] Blows et al. 2004. Marijuana use and car crash injury. *Addiction* 100: 605-611. http://www.blackwell-synergy.com/doi/abs/10.1111/j.1360-0443.2005.01100.x

[14] Movig et al. 2004. Psychoactive substance use and the risk of motor vehicle accidents. *Accident Analysis and Prevention* 36: 631-636.

[15] Huestis et al. 1992. Blood cannabinoids: absorption of THC and formation of 11-OH-THC and THCCOOH during and after smoking marihuana. *Journal of Analytical Toxicology* 16: 276-282. http://www.ncbi.nlm.nih.gov/pubmed/1338215

[16] Mushoff et al. 2006. Review of biologic matrices (urine, blood, hair) as indicators of recent or ongoing cannabis Use. *Therapeutic Drug Monitoring* 2: 155-163.

[17] Drummer et al. 2004. The involvement of drugs in drivers killed in Australian road traffic crashes. *Accident, Analysis and Prevention* 36: 239-248. http://linkinghub.elsevier.com/retrieve/pii/S0379073803001348

for the presence of THC in the blood at concentrations below this threshold typically do not have an increased risk compared to controls.[19] However, even this elevated risk is below that presented by drivers who have consumed even small quantities of alcohol.

38) Two recent case-controlled studies have assessed this risk in detail. A 2007 case-controlled study published in the *Canadian Journal of Public Health* reviewed 10 years of auto-fatality data. Investigators found that US drivers with blood alcohol levels of 0.05% (a level well below the legal limit of intoxication) were three times as likely to have engaged in unsafe driving activities prior to a fatal crash as compared to individuals who tested positive for marijuana.[20] A 2005 review of auto accident fatality from France revealed similar results, finding that drivers who tested

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[18] Grotenhermen et al. 2007. Developing per se limits for driving under cannabis. *Addiction* 102: 1910-1917. http://www.blackwell-synergy.com/doi/abs/10.1111/j.1360-0443.2007.02009.x

[19] Grotenherman et al. 2007. op cit.

[20] Bedard et al. 2007. The impact of cannabis on driving. *Canadian Journal of Public Health* 98: 6-11. http://www.cpha.ca/en/cjph/articles/ABS098N01.aspx

positive for any amount of alcohol had a four times greater risk of having a fatal accident then did drivers who tested positive for marijuana in their blood.[21] In the latter study, even drivers with low levels of blood alcohol (below 0.05%) experienced a greater elevated risk as compared to drivers who tested positive for high concentrations of cannabis (above 5 ng/ml). Both studies noted that, overall, fewer traffic accidents appeared to be attributed to drivers operating a vehicle while impaired by marijuana.

39) There has been growing interest in the development of a simple roadside test for the presence of marijuana-induced psychoactivity. Two different kinds of approaches to the problem are being explored, one which will reveal impaired driving capacity through a simple roadside psychomotor test and the other to develop a practicable roadside test which determines, directly or indirectly, blood THC level. In Australia, efforts have been made to adapt elements of the roadside Standardized Field Sobriety

[21] Lauman et al. 2005. Cannabis intoxication and fatal road crashes in France: a population base case-control study, *British Medical Journal* 331: 137 http://www.bmj.com/cgi/reprint/332/7553/1298.pdf

Test (SFST) to make it sensitive to drivers who may be under the influence of cannabis. Scientific evaluations of these tests have shown that subjects' performance on the modified SFSTs may be positively associated with dose-related levels of marijuana impairment.[22] Similarly, clinical testing for marijuana impairment among suspected drugged drivers in Norway has been positively associated with identifying drivers with THC-blood concentrations above 3 ng/ml.[23] While the technology to instantly identify at the roadside a blood level of THC which signifies driver impairment (as the breatholizer test does for alcohol) does not yet exist, much effort is being made to develop such a technology and there is little doubt that it will soon be available.

[22] Papafotiou et al. 2005. An evaluation of the sensitivity of the Standardised Field Sobriety Tests (SFSTs) to detect impairment due to marijuana intoxication. *Psychopharmacology* 180: 107-114.

[23] Khiabani et al. 2006. Relationship between THC concentration in blood and impairment in apprehended drivers. *Traffic Injury Prevention* 7: 111-116.

Marijuana and Pulmonary Risk

40) Both tobacco smoke and marijuana smoke contain non-specific quantities of a variety of respiratory irritants. Chronic exposure to these irritants has been associated with increased incidences of cough, sputum production, mild bronchitis, and wheezing compared to nonsmokers.[1] [2] [3] However, a recent meta-analysis of 30 years of data by investigators at Yale University reported that the inhalation of marijuana smoke, even long-term, is not associated with a decline in pulmonary function (e.g., emphysema).[4]

41) Like tobacco smoke, marijuana smoke also contains small quantities of some carcinogenic polycyclic aromatic hydrocarbons (PAHs), such as benzopyrene.

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[1] Tetrault et al. 2007. Effects of marijuana smoking on pulmonary function and respiratory complications: a systematic review. *Archives of Internal Medicine* 167: 221-228.

http://archinte.ama-assn.org/cgi/content/abstract/167/3/221

[2] Sherill et al. 1991. Respiratory effects of non-tobacco cigarettes: a longitudinal study in general population. *International Journal of Epidemiology* 20: 132-137. http://ije.oxfordjournals.org/cgi/content/abstract/20/1/132

[3] Donald Tashkin. 1990. Pulmonary complications of smoked substance abuse. *The Western Journal of Medicine* 152:525-530. http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1002405

[4] Tetrault et al. 2007. Op-cit.

According to the findings of a 2007 study published in the journal *Chemical Research in Toxicology*, mainstream cannabis smoke produces lower levels of these PAHs than tobacco smoke.[5]

42) Unlike tobacco smoke, marijuana smoke contains cannabinoids such as tetrahydrocannabinol (THC) and cannabidiol (CBD) which are non-carcinogenic and demonstrate anti-cancer properties *in vivo* and *in vitro*.[6] By contrast, nicotine promotes the development of cancer cells and their blood supply.[7] In addition, cannabinoids stimulate other biological activities that may mitigate the carcinogenic effects of smoke.[8]

43) Recently, clinical trials have demonstrated that

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[5] Moir et al. 2007. A comparison of mainstream and sidestream marijuana and tobacco cigarette smoke produced under two machine smoking conditions. *Chemical Research in Toxicology* (E-pub ahead of print). http://pubs.acs.org/cgi-bin/abstract.cgi/crtoec/2008/21/i02/abs/tx700275p.html

Field Code Changed

[6] Sarfaraz et al. 2008. Cannabinoids for cancer treatment: progress and promise. *Cancer Research* 68: 339-342. http://cancerres.aacrjournals.org/cgi/content/abstract/68/2/339

[7] John Minna. 2003. Nicotine exposure and bronchial epithelial cell nicotinic acetylcholine receptor expression in the pathogenesis of lung cancer. *Journal of Clinical Investigation* 111: 31-33. http://www.jci.org/111/1/31/pdf

[8] Robert Melamede. 2005. Cannabis and tobacco smoke are not equally carcinogenic. *Harm Reduction Journal* 2: 21. http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1277837

individuals greatly reduce their intake of cannabis-smoke byproducts by utilizing vaporization as an alternative to smoking. Marijuana vaporization is a process whereby the plant material is heated to within a temperature window within which cannabinoids vaporize. The lower and upper limits of the window are typically from about 360°F to 445°F which is below the ignition point of marijuana. Because the high end of the window is below the point of combustion, noxious smoke and PAHs are not produced.[9] Vaporizers are becoming ever more popular with both recreational and medicinal users.

44) In one study of vaporizers, published in the *Journal of Pharmaceutical Sciences* in 2006, investigators reported that vaporization delivered set doses of marijuana's primary active ingredient, THC, to subjects in a reproducible manner while suppressing the intake of respiratory toxins. The authors concluded: "[These] results show that with the Volcano [one of many commercially available types of marijuana vaporizers], a safe and effective

[9] Gieringer et al. 2004. Cannabis vaporizer combines efficient delivery of THC with effective suppression of pyrolytic compounds. *Journal of Cannabis Therapeutics* 4: 7-26.

cannabinoid delivery system seems to be available to [the public.] . . . . The final pulmonary uptake of THC is comparable to the smoking of cannabis, while avoiding the respiratory disadvantages of smoking."[10]

45) A second trial, published in the journal *Clinical Pharmacology & Therapeutics* in 2007, also reported that subjects who inhaled marijuana via the Volcano vaporizer significantly reduced their intake of gaseous combustion toxins, including carbon monoxide. Investigators concluded: "Vaporization of marijuana does not result in exposure to combustion gases, . . . and [was] preferred by most subjects compared to marijuana cigarettes. . . . The Volcano [vaporizer] device is an effective and apparently safe vehicle for THC delivery."[11]

46) However, because most cannabis-using populations do not utilize vaporization technology, concerns persist regarding the potential health risks associated with the inhalation of marijuana smoke.

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[10] Hazekampt et al. 2006. Evaluation of a vaporizing device (Volcano) for the pulmonary administration of tetrahydrocannabinol. *Journal of Pharmaceutical Sciences* 95: 1308-1317. http://www.ncbi.nlm.nih.gov/pubmed/16637053.

[11] Abrams et al. 2007. Vaporization as a Smokeless Cannabis Delivery System: A Pilot Study. *Clinical Pharmacology & Therapeutics* 82: 572-578. http://www.nature.com/clpt/journal/v82/n5/abs/6100200a.html

Nevertheless, presumptions regarding cannabis use as a risk factor for the development of significant pulmonary complications, particularly lung cancer, are not affirmed by the scientific literature.

47) While a handful of anecdotal reports and two small case-control studies[12] [13] associate heavy marijuana use with increased risk of head, neck and lung cancers in a minority of chronic users, no large scale population studies have replicated these results. In 2001, investigators at Johns Hopkins University reported that neither "lifetime use" nor "ever use" of cannabis was associated with head, neck or lung cancer in adults in a large, hospital-based case-control study of 164 oral cancer patients and 526 controls. Researchers concluded, "The balance of evidence from this, the largest case-control study addressing marijuana use and cancer to date, does not favor the idea that marijuana as commonly used in the community is a major causal

[12] Aldington et al. 2008. Cannabis use and risk of lung cancer: a case-control study. *European Respiratory Journal* 31: 280-286. http://erj.ersjournals.com/cgi/content/abstract/31/2/280

[13] Zhang et al. 1999. Marijuana use and increased risk of squamous cell carcinoma of the head and neck.*Cancer Epidemiology Biomarkers & Prevention* 8: 1071-1078. http://www.ncbi.nlm.nih.gov/pubmed/10613339

factor for head, neck or lung cancer in young adults."[14]

48) More recently, the results of a 2004 case-control study of 407 individuals diagnosed with oral squamous cell carcinoma and 615 healthy controls found "no association" between marijuana use and incidences of oral cancer, regardless of how long, how much, or how often individuals had used it.[15] A second 2004 case-control study of 116 oral cancer patients and 207 matched controls also failed to identify any association between self-reported cannabis use and oral cancers in adults age 45-years-old or younger, although only 10 percent of patients in the study identified themselves as heavy users of marijuana.[16]

49) A 1997 retrospective cohort study examining the relationship of marijuana use to cancer incidence in

[14] Ford et al. 2001. Marijuana Use is not Associated With Head, Neck or Lung Cancer in Adults Younger Than 55 Years: Results of a Case Cohort Study. In: *National Institute on Drug Abuse (Eds) Workshop on Clinical Consequences of Marijuana: Program Book.* Rockville: Maryland.

[15] Rosenblatt et al. 2004. Marijuana use and risk of oral squamous cell carcinoma. *Cancer Research* 64: 4049-4054. http://cancerres.aacrjournals.org/cgi/content/abstract/64/11/4049

[16] Llewellynn et al. 2004. An analysis of risk factors for oral cancer in young people: a case-control study. *Oral Oncology* 5: 78-79.

65,171 men and women 15 to 49 years of age in California found that cannabis use was not associated with increased risks of developing tobacco-use related cancers – including lung and upper aerodigestive tract cancers, breast cancer, prostate cancer, colorectal cancer, or melanoma. Investigators reported no cases of lung cancer among men and women who used marijuana but did not smoke tobacco.[17]

50) Most recently, a small case-control study from New Zealand reported that subjects who reported ever having used cannabis had no statistically significant elevated risk of lung cancer compared to non-using controls.[18]

51) Government reviews have also failed to affirm a positive association between marijuana smoking and cancers of the lung, head, or upper aerodigestive tract. For example, a comprehensive 1998 report by the British House of Lords, Science and Technology Committee concluded, “There is as yet no

[17] Sidney et al. 1997. Marijuana use and cancer incidence (California, United States). *Cancer Causes & Controls* 8: 722-728. http://www.ncbi.nlm.nih.gov/pubmed/9328194

[18] Aldington et al. 2008. Cannabis use and risk of lung cancer: a case-control study. *European Respiratory Journal* 31: 280-286.

epidemiological evidence for an increased risk of lung cancer" in cannabis smokers.[19] An 18-month study by the US National Academy of Science, Institute of Medicine also affirmed, "There is no conclusive evidence that marijuana causes cancer in humans, including cancers generally related to tobacco use."[20]

52) More recent epidemiologic reviews published in the journals *Alcohol* and *Lancet Oncology* provide similar conclusions. A review of two cohort studies and 14 case-control studies assessing the association of marijuana and cancer risk by Hashibe and colleagues concluded, "[R]esults of cohort studies have not revealed an increased risk of tobacco related cancers among marijuana smokers."[21] A 2005 review by Hall and colleagues concluded, "There is a conspicuous lack of evidence on the association between cannabis smoking and lung

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[19] British House of Lords, Science and Technology Committee. 1998. *Ninth Report*. London. http://www.parliament.the-stationery-office.co.uk/pa/ld199798/ldselect/ldsctech/151/15101.htm.

[20] National Academy of Sciences, Institute of Medicine. 1999. *Marijuana and Medicine: Assessing the Science Base*. National Academy Press: Washington, DC. (page 119).

[21] Hashibe et al. 2005. Epidemiologic review of marijuana use and cancer risk. *Alcohol* 35: 265-275. http://www.ncbi.nlm.nih.gov/pubmed/16054989.

cancers."[22]

53) Finally, in 2006, the results of the largest population-based case-control study ever to assess the risk of marijuana use and lung cancer (1,212 cases and 1,040 demographically matched controls) failed to find any association between marijuana smoking and cancer, even among subjects who reported smoking more than 22,000 marijuana cigarettes (joints) over their lifetime. Investigators concluded, "[C]ontrary to our expectations, we found no positive associations between marijuana use and lung or upper aerodigestive tract cancers. . . . It is possible that marijuana use does not increase cancer risk … [and may provide] a protective effect" against the development of certain types of cancers in humans.[23]

Marijuana and Dependence

54) Cannabis is widely accepted by the National Academy of Sciences, Institute of Medicine (IOM) and

[22] Hall et al. 2005. Cannabinoids and cancer: causation, remediation, and palliation. *Lancet Oncology* 6: 35-42. http://www.ncbi.nlm.nih.gov/pubmed/15629274.

[23] Hashibe et al. 2006. Marijuana use and the risk of lung and upper aerodigestive tract cancers: results of a population-based case-control study. *Cancer Epidemiology, Biomarkers & Prevention* 15: 1829-1834. http://cebp.aacrjournals.org/cgi/content/abstract/15/10/1829.

other investigative commissions and committees as lacking the severe physical and psychological dependence liability associated with most other intoxicants such as alcohol, tobacco, cocaine, and heroin.

55) According to the IOM, “Millions of Americans have tried marijuana, but most are not regular users … [and] few marijuana users become dependent on it.”[1] Investigators further contend, “[A]though [some] marijuana users develop dependence, . . . they appear to be less likely to do so than users of other drugs (including alcohol and nicotine), and marijuana dependence appears to be less severe than dependence on other drugs.”[2]

56) Accordingly, authors of the IOM report estimate that fewer than 10 percent of those who try cannabis ever meet the clinical criteria for a diagnosis of “drug dependence” (based on DSM-III-R criteria).[3] Other surveys have placed this figure below five

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[1] National Academy of Sciences, Institute of Medicine. 1999. Marijuana and Medicine: Assessing the Science Base. National Academy Press: Washington, DC. (pages 92-96)

[2] Ibid. (page 98)

[3] Ibid. (page 95: Table 3.4 ‘Prevalence of Drug Use and Dependence in the General Population)

percent.[4] [5] In contrast, the IOM reports that 32 percent of tobacco users, 23 percent of heroin users, 17 percent of cocaine users, and 15 percent of alcohol users meet the criteria for "drug dependence."[6]

57) Those who allege that marijuana possesses addictive qualities similar to harder drugs like cocaine or heroin often cite figures regarding the rising number of individuals enrolled in drug treatment for having used cannabis.[7] In reality, few of these individuals are in "treatment" because they or their families believed that their marijuana use was adversely impacting their lives. Rather, most individuals in drug-treatment programs for cannabis were arrested for possessing minor amounts of marijuana and were referred to treatment by the

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[4] Anthony et al. 1994. Comparative epidemiology of dependence on tobacco, alcohol, controlled substances, and inhalants: Basic findings from the National Comorbidity Survey. Experimental and Clinical Psychopharmacology 2: 244-268.

[5] National Academy of Sciences, Institute of Medicine. 1999. (page 97)

[6] Ibid. (page 95)

[7] Karl Hille. "Pot withdrawal symptoms mirror nicotine's, study finds." Baltimore Examiner, January 25, 2008. ("Treatment admissions related mainly to marijuana use have more than doubled since the early 1990s, now ranking on par with cocaine and heroin nationwide, experts say.")

courts as a requirement of their probation.[8] In fact, data published by the US Substance Abuse and Mental Health Services Association (SAMHSA) indicates that voluntary drug treatment admissions for cannabis have fallen over the past decade, while criminal justice referrals for cannabis-related drug treatment have risen dramatically.[9] (During this same time period, arrests for minor marijuana offenses have risen from fewer than 300,000 per year in the early 1990s to more than 800,000 per year.) As a result, according to current state and national statistics, an estimated 60 to 70 percent of all individuals in treatment with a primary diagnosis of "marijuana dependence" are legally coerced into treatment.[10] [11]

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[8] US Office of Applied Studies, Substance Abuse and Mental Health Services Administration. June 24, 2005. The DASIS (Drug and Alcohol Services Information System) Report, Differences in Marijuana Admissions Based on Source of Referral: 2002. http://oas.samhsa.gov/2k5/MJreferrals/MJreferrals.htm.

[9] US Office of Applied Studies, Substance Abuse and Mental Health Services Administration. March 29, 2002. The DASIS Report, Treatment Referral Sources for Adolescent Marijuana Users. http://www.oas.samhsa.gov/2k2/YouthMJtx/YouthMJtx.htm.

[10] Copeland and Maxwell. 2007. Cannabis treatment outcomes among legally coerced and non-coerced adults. BMC Public Health 7: 111.

[11] US Office of Applied Studies, Substance Abuse and Mental Health Services Administration. June 24, 2005.

58) Finally, while a handful of recent clinical trials claim to have identified a distinctive “marijuana withdrawal syndrome”, it remains unclear whether such a syndrome is clinically significant. To date, reports of cannabis-associated withdrawal symptoms remain limited to subjective case reports of polydrug users[12], subjects enrolled in substance abuse drug treatment[13] [14] [15] [16] [17], or clinical trials involving very few participants[18] [19]. By contrast, discontinuing chronic heavy dosing of THC in

[12] Vandrey et al. 2008. A within-subject comparison of withdrawal symptoms during abstinence from cannabis, tobacco, and both substances. Drug and Alcohol Dependence 92: 48-54.

[13] Levin et al. 2005. Severity of dependence and motivation for treatment: comparison of marijuana- and cocaine-dependent treatment seekers. Journal of Addictive Diseases 25: 33-41.

[14] Budney et al. 2006. The time course and significance of cannabis withdrawal. Journal of Abnormal Psychology 112: 393-402.

[15] Vandrey et al. 2005. Cannabis withdrawal in adolescent treatment seekers. Drug and Alcohol Dependence 78: 205-210.

[16] Budney et al. 1999. Marijuana withdrawal among adults seeking treatment for marijuana dependence. Addiction 94: 1311-1322.

[17] Crowley et al. 1998. Cannabis dependence, withdrawal, and reinforcing effects among adolescents with conduct symptoms and substance use disorders. Drug and Alcohol Dependence 50: 27-37.

[18] Budney et al. 2001. Marijuana abstinence effects in marijuana smokers maintained in their home environment. Archives of General Psychiatry. 58: 917-924.

preclinical trials does not reveal withdrawal symptoms.[20]

59) According to the Institute of Medicine, marijuana withdrawal symptoms are "mild and subtle"[21] compared with the profound physical syndromes associated with ceasing chronic alcohol or barbiturate use – which can be fatal – or those abstinence symptoms associated with daily tobacco use, which are typically severe enough to persuade individuals to reinitiate their drug-taking behavior.

60) Marijuana's absence of significant withdrawal effects is likely because "under normal cannabis use, the long half-life and slow elimination from the body of THC … can prevent substantial abstinence symptoms" from occurring.[22] As a result, said symptoms, when identified, are typically limited to feelings of mild anxiety, irritability and insomnia.[23] [24] [25] These abstinence symptoms do not

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[19] Vandrey et al. 2005. A cross-study comparison of cannabis and tobacco withdrawal. American Journal on Addictions 14: 54-63.

[20] National Academy of Sciences, Institute of Medicine. 1999. (page 91)

[21] Ibid. (page 90).

[22] Ibid. (page 58).

[23] Ibid. (page 90)

[24] Vandrey et al. 2008. Op-cit.

appear to be severe or long-lasting enough to reinforce marijuana use in individuals who have ceased using the drug.[26]

61) This lack of reinforcing abstinence symptoms arguably explains why those who report using marijuana in early adulthood typically report voluntarily ceasing their cannabis use by age 30 with little physical or psychological difficulty.[27] [28] [29] By comparison, most cigarette smokers who pick up the habit early in life continue to smoke for the rest of their lives, despite making numerous efforts to quit.

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[25] Haney et al. 1999. Marijuana abstinence effects in marijuana smokers maintained in their home environment. Psychopharmacology 141: 395-404.

[26] Arendt et al. 2007. Withdrawal symptoms do not predict relapse among subjects treated for cannabis dependence. American Journal on Addictions 16: 461-470.

[27] Kandel et al. 1984,. Patterns of drug use from adolescence to young adulthood: Periods of risk for initiation, continued use, and discontinuation. American Journal of Public Health 74: 660-666.

[28] University of Michigan, Institute for Social Research. 1992. Monitoring the Future Occasional Paper No 35: Changes in Drug Use During the Post-High School Years (Ann Arbor).

[29] National Academy of Sciences, Institute of Medicine. 1999. (page 92) Marijuana and Dependence.

The Potency of Marijuana

62) Prior to the late 1990s, the potency of marijuana had averaged around 3 to 4% THC for two decades. According to the federally funded University of Mississippi at Oxford, which has been randomly testing seized samples of cannabis for THC content since the mid-1970's, average THC levels have increased since 1998 from approximately 4% to approximately 8% today.[1] However, the putative increment in potency does not mean increased risk. Because the effects of smoking cannabis are so much more rapidly perceived than are the effects of any psychoactive drug (including marijuana) taken orally, both recreational and medicinal users can quickly learn how to titrate the dose to achieve the desired effect. A user who smokes (or vaporizes) marijuana has to inhale less of a more potent sample and, conversely, more of one that is less potent. It follows that to the extent that inhalation of cannabis smoke is considered a risk factor for pulmonary disease, the more potent sample provides a healthier choice.

[1] US National Drug Intelligence Center. 2007. National Drug Threat Assessment, Washington, Dc. http//:www.usdoj.gov/ndic/pubs21/21137/marijuana.htm#Start

63) Claims that today's marijuana is far stronger and thus potentially more harmful to the health of the user than the cannabis available in previous decades are highly misleading for two other reasons. First, while a handful of cannabis strains significantly more potent than about 8% THC may be available in limited quantities today, these varieties comprise only a small percentage of the overall marijuana marketplace and are available only at prices that are cost-prohibitive to most users.[2] Of the thousands of pounds of marijuana seized by law enforcement annually, few samples ever test positive for especially high percentages of the drug's primary psychoactive ingredient THC. In fact, according to the US Drug Enforcement Administration (DEA), of the more than 4,600 domestic samples analyzed by the government between 1998 and 2002, fewer than two percent were found to contain THC levels above 20 percent.[3] As a result, the federal government concedes that most of the marijuana

[2] Forbes,D. "The Myth of Potent Pot," Slate.com, November 19, 2002, http://www.slate.com/?id=2074151

[3] US Department of Justice, Drug Enforcement Administration. 2005. Drugs of Abuse. Washington, DC.
http://www.dea.gov/pubs/abuse/7-pot.htm.

available in the domestic drug markets is lower potency commercial-grade marijuana.[4] Consequently, for the majority of today's cannabis consumers, marijuana remains essentially the same plant it has always been, with any relatively mild rise in average potency akin to the difference between beer and wine.

64) Second, unlike whiskey, or even aspirin, marijuana poses no risk of fatal overdose, regardless of THC content.[5] In fact, since 1985 when dronabinol (Marinol), which is 100% THC, was approved as a prescription pharmaceutical, physicians have been prescribing it to patients for a variety of illnesses in a wide range of doses with relatively few side effects. This more than two decades of legitimate medical use constitutes further evidence

[4] US National Drug Intelligence Center. 2007. National Drug Threat Assessment. Washington, DC. http://www.usdoj.gov/ndic/pubs21/21137/marijuana.htm#Start

[5] Australian National Drug and Alcohol Research Centre. 1994. The Health and Psychological Consequences of Cannabis Use. Canberra. (See specifically: Chapter 9, Section 9.3.1 Acute Effects: There are no recorded cases of fatalities attributable to cannabis, and the extrapolated lethal dose from animal studies cannot be achieved by recreational users, http://www.health.gov.au/internet/wcms/publishing.nsf/Content/health-pubs-drug-cannab2-home.htm.

that THC is remarkably non-toxic to the brain.[6] Furthermore there is growing evidence that the administration of cannabinoids may be neuroprotective against various types of brain injury, including damage associated with stroke[7] and acute alcohol poisoning.[8]

65) Finally, it should be noted that marijuana smokers moderate their cannabis consumption to avoid dysphoria and other unpleasant effects.[9] As a result, consumers of higher-grade cannabis typically take longer intervals between puffs and, overall, consume less marijuana by volume than they would if they were using lower quality marijuana.[10] [11]

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[6] Earleywine,M. 2005. Understanding Marijuana: A New Look at the Scientific Evidence. London: Oxford University Press. (p. 130)

[7] Hampson et al. 1998. Cannabidiol and Delta-9-tetrahydrocannabinol are Neuroprotective Antioxidants. Proceedings of the National Academy of Sciences 95: 8268-8273. http://www.pnas.org/cgi/content/full/95/14/8268

[8] Hamelink et al. 2005. Comparison of Cannabidiol, Antioxidants, and Diuretics in Reversing Binge Ethanol-Induced Neurotoxicity. Journal of Pharmacology And Experimental Therapeutics Fast Forward 314: 780-788. http://jpet.aspetjournals.org/cgi/content/abstract/314/2/780.

[9] Mikuriya and Aldrich. 1988. Cannabis 1988: Old Drug, New Dangers, The Potency Question. Journal of Psychoactive Drugs 20: 47-55.

[10] Herning et al. 1986. Tetrahydrocannabinol Content and Differences in Marijuana Smoking Behavior. Psychopharmacology 90: 160-162.

In conclusion:

66) I have been studying psychoactive drugs throughout my research and teaching career at Harvard Medical School. Cannabis has been of particular interest to me since 1967. During that time I have published two books and a number of papers on various aspects of marijuana and I have participated in innumerable national and international conferences. Over these years my growing understanding of marijuana both as a recreational drug and a medicine strengthened my belief that its physiological, mental and social toxicity is arguably the least of any drug with psychoactive properties; over the same period of time I observed that the number of annual arrests for possession rose from fewer than 100,000 in 1967 to about 800,000 in 2006. This proliferation of arrests is emblematic of the harm imposed by the law on these mostly young people as it damages their career prospects, undermines their respect for our legal institutions and places what for many of them is a severe financial burden of legal fees on themselves or their families. Large as it has

[11] Abrams et al. 2007. Vaporization as a smokeless cannabis delivery system: A pilot study. Clinical Pharmacology & Therapeutics 82: 572-578.

become, the number of citizens arrested annually on marijuana charges comprises only a fraction of the millions of citizens who use this drug regularly and the many more millions who have used it at one time or another. The marijuana prohibition criminalizes users and former users who understand firsthand that the "authorities" have and continue to greatly exaggerate its toxicity; they become resentful that they are seen as criminals and they tend to become cynical about what the government and organizations such as the Partnership for a Drug-Free America say about the legitimate dangers of other drugs. Where marijuana is concerned, many of these authorities' assertions are based on myths which arose in the 1930s "Reefer Madness" days.

67) Today, there is no excuse for the kinds of misunderstandings and misinterpretations which continue to fuel the belief that marijuana is so dangerous that it is necessary to criminally prohibit its responsible use by adults. During the past four decades many scientific studies addressing the question of cannabis toxicity have been completed; in fact, there are few other drugs in or outside of the *U.S.Pharmacopoeia* which have been so

carefully scrutinized. And just as with studies of the toxicity of pharmaceutical products, they have to be read critically because some are, as we as have seen above, methodologically flawed.

68) In addition to carefully following this developing scientific literature on marijuana over four decades, I have also accumulated a great deal of clinical experience with patients who have used cannabis as a medicine. I can confidently say based on my education, training, investigations and clinical experience that it is beyond reasonable scientific doubt that responsibly used marijuana is safe.

Signed under the pains and penalties of perjury this ___ day of March, 2008

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Lester Grinspoon M.D.