

Question 3): What patterns of cannabis use would not be considered abusive? What basis would you use to distinguish abusive from therapeutic use? What properties of cannabinoids make them well suited for use as an analgesic? What are the most promising recently discovered properties of cannabinoids?

“Today, a discussion of the distinction between drug use and abuse seems anachronistic and unpatriotic.” So notes Eric Wish, PhD, colleague of the noted Norman Zinberg, MD (author of *Drug, Set, and Setting*) in a 1990 *JAMA* editorial. Nevertheless, at the risk of appearing unpatriotic and anachronistic, I will briefly try to make some general comments about human cannabis consumption patterns in order to tease out what might be appropriately considered using, abusing, and therapeutic consuming patterns.

A sensible place to begin is modern substance abuse disorder diagnostics as codified in the DSM-IV, the *Diagnostic and Statistical Manual for Mental Disorders*. As I noted in my general statement, major revisions in the history of the DSM brought about the modern substance abuse/dependence diagnostics and nosology. It turns out that the concept of ‘substance abuse’ was itself on the chopping block and just barely escaped deletion during the time between the DSM-III and DSM-III-R. Schuckit (1994) and Helzer (1994), writing in the *DSM-IV Sourcebook*, relay the following psychiatric lore:

The change between DSM-III and DSM-III-R represented an entire reorientation in the concept of abuse and dependence. As is described in more detail below, the term *dependence* was broadened considerably. As a consequence, the framers of DSM-III-R originally proposed to delete the concept of abuse, feeling that the entire spectrum of substance-related problems was now incorporated into the broad concept of dependence. At the last minute, however, pressure from the field required that the term *abuse* be reinserted into the manual. However, abuse was now viewed as a residual diagnosis that was to be applied only to individuals who still had some substance-related difficulties but who did not fit into even a broad approach to dependence” (p.7)

...

In a personal communication to the Substance Use Disorders Committee, Richard Frances recalled that there was an attempt to drop the term *abuse* in the DSM-III-R criteria, but that it was reinstated at the time of the field trials by the popular demand of those attempting to use the new DSM-III-R criteria. (p. 25)

Who knows who the most vocal opponents of the Committee’s plan of action may have been—the ‘squeakiest’ wheels? Nevertheless, seeing that we are now therefore stuck with this mental disorder of substance abuse, however tenuously it did survive near-deletion, and despite the protestations about the essentially pejorative nature of the diagnosis (Blackwell 1987; Peyhow and Gitlow 1988), how do we go about characterizing it? Let’s ask the experts. The following definition of substance abuse emerged by consensus when the question was posed to a panel of 99 substance abuse experts by Rindali and colleagues (1988). ‘Drug’ abuse is “any use of drugs that causes physical, psychological, economic, legal, or social harm to the individual user or to others affected by the drug user’s behavior” (quoted in Helzer, p. 24). The current DSM-IV definition of substance abuse, with its four free-standing criteria regarding the shirking of work/school obligations, engagement with physically hazardous behavior, the distress of legal problems, and social/familial disputes is essentially based on this consensus definition. I have noted in my previous statements (Preliminary and General) and in question 5 below the problems with this definition, especially with regards to the ‘legal’ harm criterion for the mental disorder, an issue of critical importance when it comes to distinguishing abusive patterns of cannabis use from non-abusive ones. Alexander (2003), writing in *The American Journal of Drug and Alcohol Abuse* (appropriate, given that in America 7 million cannabis-related arrests occurred in the last decade with three-quarters of a million per annum), notes some of the difficulties with this prohibition-endorsing criterion:

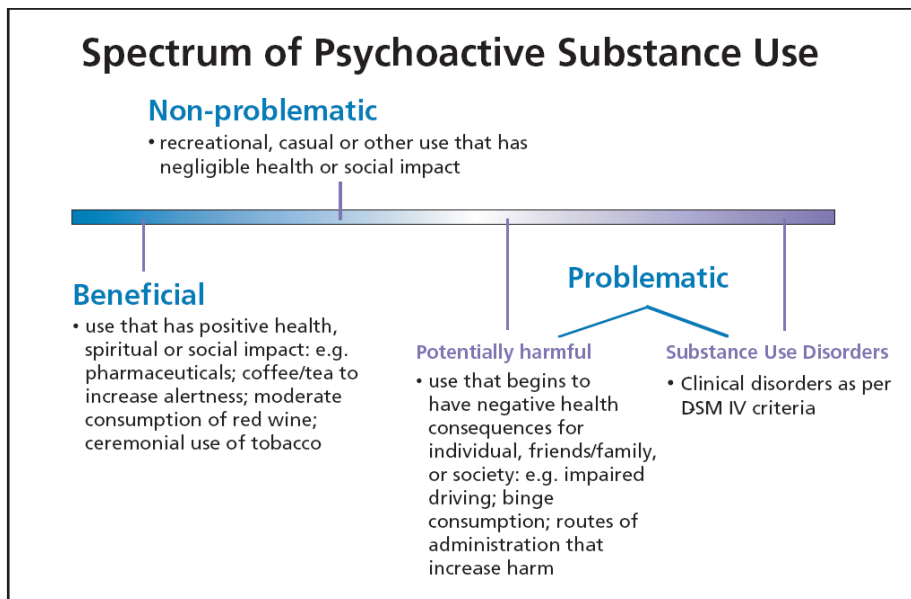
Subjective clinical judgment enters into Cannabis Abuse criterion distinctions regarding the meaning of ‘recurrent’ or ‘maladaptive pattern.’ For example, legal consequence risks are present with any marijuana use level, but may remain latent, or risk exposure only if a person drives or buys. Behavioral frequency cutoffs are

not sufficiently clear regarding ‘legal’ or ‘driving’ problems with marijuana to allow consistent clinical agreement that a ‘recurrent’ ‘maladaptive’ pattern exists. (p. 622)

My approach: forget about legal problems as a useful criterion to gauge cannabis abuse.

It is worthless and unreliable, not to mention unjust (Gettman, “The Cannabis Rescheduling Petition”). It is better to spend time focusing on particular problems associated with an individual’s cannabis consumption (Earlywine 2002; 2005) In fact, I would jettison the whole use/abuse dichotomy and transition to a spectrum view as has been adopted by the British Columbia Ministry of Health. In their framework for addressing problematic substance use, they include the figure below and note:

The Framework recognizes that instances or patterns of substance use occur along a spectrum from beneficial use to non-problematic use to problematic use (including potentially harmful use and substance use disorders). Substance use disorders represent the extreme and most damaging end of the spectrum. Some people choose to abstain from using psychoactive substances while some people choose to use only certain substances. It is important to emphasize that abstinence is a healthy lifestyle option. Nevertheless, many people choose to use substances and some do not develop serious problems because of this use. (p. 8)



Though they do not abandon the Substance Use disorders nosology (but would agree, I

am sure, as to the injustice of ‘drug’ prohibition), the public health officers in British Columbia take an enlightened approach to understanding psychoactive substance use. Applying this to cannabis, we can see that cannabis consumption can be beneficial, non-problematic, or problematic for the consumer. Distinguishing between problematic and non-problematic use is simple: probe for the existence of medical/psychosocial problems, leaving legal issues aside as a Dutch health care provider would be inclined to do, given the Netherlands’ system of de facto cannabis (re)legalization. If problems are identified, attention should be focused on reducing those particular harms associated with cannabis use for the patient-citizen. Distinguishing between non-problematic versus beneficial use of cannabis is more difficult, given the *relaxant* properties of cannabis use, and given consumers’ tendency to reduce or substitute for alcohol consumption, which has its own health benefits. Perhaps this determination, if it must be made at all, ought to be done on strictly subjective grounds, as per “the new subjective medicine” that seeks to take “the patient’s point of view” on matters related to health status and withdrawal of life-support (Sullivan 2003). Given that cannabis is not recognized as a medicine at the federal level and in 38 states, it is likely that consumers may not be ‘looking’ for medicinal or beneficial effects. A questionnaire that focuses on quality of life, stress reduction, spirituality, somaesthetics (Shusterman 1999), self-directed psychotherapeutics, self-care and related issues would likely help to elicit beneficial aspects of cannabis consumption that a consumer may only be dimly aware of on open-ended questioning.

One aspect of cannabis consumption that risks total neglect (and ‘abuse’, if you will) in substance use/abuse and related discourses is the relationship that human beings develop with biota that they discover, produce and consume, such as plants, and in

particular the cannabis plant. Appreciation, seed planting, nurturing, harvesting, and consumption of cannabis are all part of a human-environment relationship between two biotic species that both descended from a common evolutionary ancestor between 1 and 2 billion years ago (Dawkins 2004). Health/medical geographer Hester Parr, in her recent talk at the UW Geography Colloquium, spoke about the emotional benefits that mental patient-citizens glean through their experience with gardening and plant care. Her research showed that horticultural practices helped to “ground” patient-citizens. One respondent noted: “You slow your thoughts down to the speed of the plant and what’s happening to it.” Another said: “...you go into a sort of trance.” A third said: “You can go into this place that is not you and it’s not the world” (2006, personal notes from lecture). Clearly, human-plant relationships can have cultural, healing, and therapeutic aspects to them. This side of cannabis consumption and production is totally elided in modern ‘use/abuse/dependence’ discourse.

The properties that make cannabinoids well-suited for analgesia are their extremely high safety, remarkably low toxicity, and significant efficacy for relieving a wide range of pain states, from neuropathic pain to muscle ache / joint pain, to migraine pain. With whole cannabis, with its 68 cannabinoids, these three properties hold true. With other synthetic, single molecule cannabinoid therapeutic options, such as dronabinol, nabilone, levonantradol, and ajumelic acid, these properties of safety, low toxicity, and efficacy also hold, but to a lesser degree, and with intolerable side effects such as drowsiness, dysphoria, and increased toxicity reported in pre-clinical and clinical data. In its 4,000+ years of documented use, there is no report of death from overdose with cannabis. The theoretical LD₅₀ in humans is 1:20,000 or 1:40,000, using a single

cannabis cigarette as a unit of dose. In other words, one would have to consume ~628 kg of combusted cannabis by inhalation over 15 minutes in order to die (Carter et. al 2004, p. 465). In contrast, consuming 2 grams of dried opium poppy juice is lethal in humans as a result of severe respiratory depression. If a very large dose of cannabis is consumed, which typically occurs via oral ingestion of a concentrated preparation of cannabis flowers (e.g., in the form of an alcohol tincture or oil extract), agitation and confusion, progressing to sedation, is the generally the result (Mikuriya 2006). Some have even called this an ‘acute cannabis psychosis’, and this exacerbates fears that cannabis consumption, in the long term, might lead to schizotypy such as chronic, debilitating psychosis. Review of the current epidemiological data shows that such fears are unfounded (Erowid 2005; Armentano 2006; Gardner 2005; Mirken et al. 2005). However, in light of the minor associations that have been documented in a small number of longitudinal studies between heavy cannabis consumption and later schizotypy, those who are early- or pre-teens and those who have pre-existing symptoms of mental illness, should abstain from cannabis consumption, especially in large amounts. What are the schizotypy risks to the general population related to cannabis consumption? The United Kingdom’s Advisory Council on the Misuse of Drugs, a statutory and non-executive, non-departmental, independent public body of experts that advises the UK government on drug-related issues, offered the following words of wisdom after careful and extensive consideration:

In the last year, over three million people appear to have used cannabis but very few will ever develop this distressing and disabling condition. And many people who develop schizophrenia have never consumed cannabis. Based on the available data the use of cannabis makes (at worst) only a small contribution to an individual’s risk for developing schizophrenia....For individuals, the current

evidence suggests, at worst, that using cannabis increases the lifetime risk of developing schizophrenia by one per cent (2005).

So, in light of these considerations, it is clear that cannabis has an extremely high safety level and remarkably low and manageable toxicity level as an analgesic. Unlike opioids, they do not promote appetite loss, wasting, and constipation, but instead can be used to therapeutically treat these symptoms.

Cannabis is also highly efficacious as an analgesic. A synergistic and entourage effect in which endogenous cannabinoids are also involved likely results in the superior analgesia of whole plant cannabis. Carter et al. summarize this as follows:

“Cannabinoids produce analgesia by modulating rostral ventromedial medulla neuronal activity in a manner similar—but pharmacologically distinct from—that of morphine. This analgesic effect is also exerted by some endogenous cannabinoids...” (2004; p. 949).

In addition, terpenoids, flavinoids, and essential oils present in phytocannabis preparations have been shown to have therapeutic effects on mood, inflammation, and pain (Russo 2002, p. 366; McPartland and Pruitt 1999). Additionally, cannabinoids are known to have antinociceptive effects in descending pain pathways, such as those mediated by the periaqueductal gray. Finally, cannabinoid-rich cannabis has anti-inflammatory properties (acting through prostaglandin synthesis inhibition and other cytokine-mediated mechanisms) and can presynaptically modulate the release of dopamine, serotonin, and glutamate—neurotransmitters involved in migraine, nausea, and many other noxious symptoms.

Cannabinoids are especially well-suited as analgesics because they work efficaciously. I will not review here the extensive clinical trials or historical literature showing this. Suffice it to say that under the State of Oregon’s Medical Marijuana

Program, as of July 1, 2006, 9748 of the 11143 state-wide registered medical cannabis patients, or 87%, report “severe pain” as a condition that is being eased with cannabis consumption (OMMP 2006). Beyond pain, there are numerous therapeutic applications for cannabis—Dr. Tod Mikuriya, a psychiatrist, Addiction medicine specialist, and Cannabinologist in Berkeley, CA, has documented 222 ICD-9 conditions that he has determined can benefit therapeutically from cannabis (Mikuriya 2004). More will undoubtedly be discovered in due time. Meanwhile, cannabis-related easement and pain relief will continue to grow as the knowledge of its remarkable benefits spreads.

There are many promising and emerging properties of cannabinoids that are being discovered in biomedical research. Two I will briefly mention are mounting findings of cannabinoids inhibiting tumor cell growth and the recently demonstrated neurogenic properties of cannabinoids. Regarding cannabinoids and cancer, several reviews have recently been published (Kogan 2005, Guzman 2003, Armentano 2006). Kogan writes: “Cannabinoids possess ... anticancer activity [and may] possibly represent a new class of anti-cancer drugs that retard cancer growth, inhibit angiogenesis (the formation of new blood vessels) and the metastatic spreading of cancer cells” (p. 952). The following news releases from NORML (National Organization for the Reform of Marijuana Laws) cite three studies that have been published only in the last two months (Ligresti et al. 2006; Caffarel et al. 2006; Carracedo et al. 2006):

Cannabidiol Dramatically Inhibits Breast Cancer Cell Growth, Study Says

June 1, 2006. Naples, Italy: Compounds in marijuana inhibit cancer cell growth in animals and in culture on a wide range of tumoral cell lines, including human breast carcinoma cells, human prostate carcinoma cells, and human colorectal carcinoma cells, according to preclinical trial data published in the May issue of the Journal of Pharmacology and Experimental Therapeutics.

Investigators at Italy’s Istituto di Chimica Biomolecolare assessed the anti-cancer activity of various non-psychoactive cannabinoids - including cannabidiol (CBD), cannabigerol (CBG), and cannabichromine (CBC) - in vivo and in vitro. Researchers reported that CBD acts as a more potent inhibitor of cancer cell growth than other cannabinoids, including THC, and noted that the

compound is particularly efficacious in halting the spread of breast cancer cells by triggering apoptosis (programmed cell death).

Cannabigerol and CBC also possess anti-tumor properties, but lack the potency of CBD, they found.

“These results suggest the use in cancer therapy for cannabidiol,” investigators concluded.

Previous studies have shown cannabinoids to reduce the size and halt the spread of glioma (brain tumor) cells in animals and humans in a dose dependent manner. Separate preclinical studies have also demonstrated cannabinoids to inhibit cancer cell growth and selectively trigger malignant cell death in skin cancer cells, leukemic cells, lung cancer cells, and prostate carcinoma cells, among other cancerous cell lines.....

Cannabinoids Halt Pancreatic Cancer, Breast Cancer Growth, Studies Say

July 6, 2006. Madrid, Spain: Compounds in cannabis inhibit cancer cell growth in human breast cancer cell lines and in pancreatic tumor cell lines, according to a pair of preclinical trials published in the July issue of the journal of the American Association for Cancer Research.

In one trial, investigators at Complutense University in Spain and the Institut National de la Sante et de la Recherche Medicale (INSERM) in France assessed the anti-cancer activity of cannabinoids in pancreatic cancer cell lines and in animals. Cannabinoid administration selectively increased apoptosis (programmed cell death) in pancreatic tumor cells while ignoring healthy cells, researchers found. In addition, “cannabinoid treatment inhibited the spreading of pancreatic tumor cells ... and reduced the growth of tumor cells” in animals.

“These findings may contribute to ... a new therapeutic approach for the treatment of pancreatic cancer,” authors concluded.

In the second trial, investigators at Spain's Complutense University reported that THC administration "reduces human breast cancer cell proliferation [in vitro] by blocking the progression of the cell cycle and by inducing apoptosis." Authors concluded that their findings “may set the bases for a cannabinoid therapy for the management of breast cancer.”...

A first-ever human clinical trial published on July 13, 2006 demonstrating tumor volume shrinkage with intratumor THC injections in several patient-citizens with recurrent glioblastoma multiforme is a landmark (Guzman et al. 2006). The sad fact about cannabinoid anti-cancer therapy is that it has been hidden and suppressed for so long. As Armentano (2006 “Cannabinoids and Cancer Hope”) writes, citing (Munson et al. 1975):

For over 30 years, US politicians and bureaucrats have systematically turned a blind eye to scientific research indicating that marijuana may play a role in cancer prevention -- a finding that was first documented in 1974. That year, a research team at the Medical College of Virginia (acting at the behest of the federal government) discovered that cannabis inhibited malignant tumor cell growth in culture and in mice. According to the study's results, reported nationally in an Aug. 18, 1974, *Washington Post* newspaper feature, administration of marijuana's primary cannabinoid THC, “slowed the growth of lung cancers, breast cancers

and a virus-induced leukemia in laboratory mice, and prolonged their lives by as much as 36 percent.

The second extremely promising recently discovered property of cannabinoids is that they promote neurogenesis. A lab in the Neuropsychiatry Research Unit, Dept of Psychiatry, University of Saskatchewan, administered the compound in Figure 1 to adult rats by injection twice daily for 10 days and found a ~40% increase in the number of new and integrated neurons in the hippocampal dentate gyrus as compared to controls (Jiang et al. 2006) after the treatment. The effect was blocked with a CB1 antagonist.

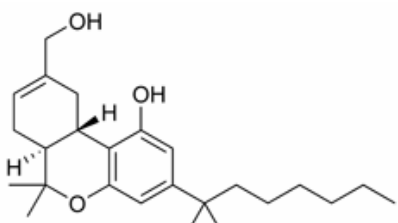


Figure 1: HU210: 3-(1,1-Dimethyl heptyl)-(-)-11-hydroxy- Δ^8 -tetrahydrocannabinol

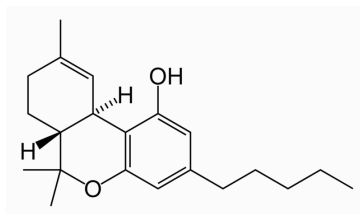


Figure 2: THC: (-)- Δ^9 -tetrahydrocannabinol (1/100th of the potency of HU210)

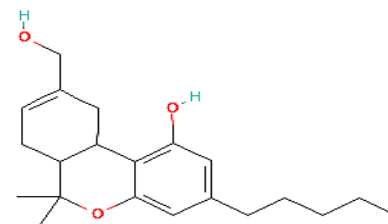


Figure 3: 11-OH- Δ^8 -THC (cannabis metabolite)

Though global media coverage of this finding downplayed it, these results are highly relevant to cannabis consumption. Note that Δ^8 -tetrahydrocannabinol also exists in phytocannabis preparations, biosynthesized at roughly one-tenth the level of Δ^9 -tetrahydrocannabinol (Latta and Eaton 1975). Liver metabolism of Δ^9 -THC (Figure 2) and Δ^8 -THC yield the hydroxylated products 11-OH- Δ^9 -THC and 11-OH- Δ^8 -THC (Figure 3), respectively. The latter compound differs from neurogenic HU210 (Figure 1) by only a single 4-carbon unit in the alkyl chain, inserted between the chain and its connection to the aromatic ring at the 3-position (meta- to the -OH substituent). This shows that HU210 (Fig. 1) and 11-OH- Δ^8 -THC (Fig. 3) are virtually identical structurally, and thus raises the distinct possibility that cannabis consumption yields biometabolites that, over a period of exposure, can lead to an increased number of neurons in the brain.

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