

Question 1B): Because of my having taught Geog 512, and because of my interest in linguistic precision, I would appreciate a brief foray into 2 phrases used in your 1st paragraph. A "recognizing the human body as both a materially and discursively created natural object", uh, discursively, how, why? what's the matter with socially? B "holistic perspective.... sophisticated poststructuralist and feminist understanding of the human body that.... integrates... subjectivity and embodiment" etc.... Why and how is a feminist understanding of the body different from a masculinist understanding? I'm serious about this. Doesn't it set up an essentialist dichotomy that at best is a cliché?

A) "...recognizing the human body...as both a materially and discursively created natural object..."

Nothing at all is the matter with referring to the human body as a socially created or socially situated 'natural object.' Replacing 'discursively' with 'socially' in this phrase would create a statement I definitely would agree with. For indeed, we, as mammals who are of necessity nursed and reared by others of our own species, *are social and socially created creatures in every way*—just as our primate and many of our non-primate animal cousins are as well. With my diction of 'discursively created', I had in mind the medium by which the thick sociocultural dimensions of a living, embodied human being come to be expressed, communicated, and understood in intersubjective encounters such that the unique, subjective aspects of embodied human life come to be duly acknowledged and appreciated. Being that medical geography is one of the medical social sciences (which also include medical anthropology, medical sociology, medical psychology, medical economics, medical history, and medical ethics), with my chosen phraseology I especially have in mind a 'recognizing' of 'the human body' that seeks to deepen and strengthen the intersubjective encounter between a health/medical care provider and a patient-citizen (Sullivan 2003). While these 'clinical' encounters certainly

are social encounters between natural, biophysical, material objects—bodies—which are also socially created human subjects, it is through intersubjective and interpellating discourse, both verbal and nonverbal, that the humanistic aspects of the ‘clinical encounter’ take shape. The human body is the object of a vast amount of knowledge and information; this constellation of facts and theories, which is discursively passed on, prefigures the clinical encounter. But it by no means determines it. Thus, by saying that the body is ‘discursively *created*’, I am referring to the discursively *mediated* ongoing recognition and appreciation of a human being’s vast, underlying inner world of subjective complexity lying ‘beneath’ outward biological and material appearance.

Here, I must admit that I am putting myself in the role of a health-care-provider/medical geographer-in-training who is seeking to understand the lived experience of other subjects. In my training thus far, I have witnessed clinical encounters that serve as good examples to demonstrate why ‘discursively created’ is appropriate phraseology in describing the body. One example I would like to share was my observation of a clinical encounter in 2004 with a gentleman who was seeking medical asylum. This man from Romania claimed that he had been subjected to brutal forms of torture and punishment such as beatings and electrocution during a period of politically-motivated incarceration. This highly charged history, discursively communicated with the aid of an interpreter, was then cross-checked by my attending physician via examination and clinical judgment. Bodily markings, scars, and other signs were cross-referenced with the patient-citizen’s narrative of torture and abuse to check for consistency. His body thus was both materially and discursively created and understood. What is critical to recognize about this example is that the asylum seeker offered a

narrative discourse that is politically at odds with official pronouncements of Romanian government representatives and other members of the asylum-seeker's social milieu. If given the opportunity, they would question the patient-citizen's narrative and vehemently deny any abuse allegations. It is only with the patient-citizen's interdigitating discursive acts and bodily presentation in the clinic that the prevailing governmentally sanctioned social narratives about his life could be challenged. A similar discursive and material bodily co-construction occurs when a care provider grants a recommendation for the medicinal use of cannabis to a patient-citizen. Marijuana medical decision-making is often predicated on a patient-citizen's attestations of relief felt with the use of cannabis preparations, many of which may not be easily objectively verified not to mention legally sanctioned. Dr. Jerome Kassirer, former editor of the *New England Journal of Medicine*, put it this way: "The noxious sensations that patients experience are extremely difficult to quantify in controlled experiments. What really counts for a therapy with this kind of safety margin is whether a seriously ill patient-citizen feels relief as a result of the intervention, not whether a controlled trial 'proves' its efficacy" (1997). To this bit of editorializing, I would add that this is the case for all patient-citizens, whether "seriously ill" inpatients or generally well outpatients in no acute distress. Only through discursive acts on the part of the patient-citizen can relief felt from noxious sensations come to be known by another. Discourse about therapeutic benefits from cannabis use is certainly at odds with present official pronouncements about 'marihuana' from numerous government agencies. Even with social mores in some places 'against' marijuana, patient-citizen discourse can allow for the disruption of such static and prevailing notions and the expression of antithetical knowledge (Said 1981). Thus, from these examples, we

see that the notion of the body as a 'discursively created natural object' allows for both socially- and individually-derived categories of bodily inscription and description.

However, being attuned to discourse in itself does not automatically allow for the awareness that prior socially prevailing notions are challenged in the lived body of the patient-citizen. Critical thinking must prevail. As Foucault (1972) writes: "We must question those ready-made syntheses, those groupings that we normally accept before examination...we must...obscure forces by which we usually link the discourse of one man with that of another...they must be driven out from the darkness in which they reign" (p.22).

B) "holistic perspective.... sophisticated poststructuralist and feminist understanding of the human body that.... integrates... subjectivity and embodiment, in addition to strictly objective and material ones, in theorizing states of health and illness."

Can we grant that there is such a thing as a 'feminist approach' or a 'masculinist approach'? Do these categories even exist? Certainly, we can at least agree that there exists an essential biological category of 'sex' whose differentiation is driven by genes, mediated by hormones, and knowable upon anatomical inspection. Perhaps we would also agree that there is such a concept as 'gender' which, being more of a psychosocial phenomenon, connotes the embodied experience and constructed nature of sex identity. What of feminist and masculinist approaches? These concepts have no respective essentialized mappings onto women and men. Feminist approaches, while they arose historically from the writings and lived experiences of women, are not in any way relegated simply to women or only knowable through living as a woman. Likewise for masculinist approaches. All women do not employ feminist analysis, and all men do not

apply masculinist analysis. Feminist and masculinist approaches are, in my estimation, nothing at all like sex and gender roles but rather better understood as social theoretical, political, and moral movements that urge the adoption and appreciation of certain clusters of perception and ways of knowing (“Masculinism”). They are discursive strategies to generate appreciation of alternative, non-dominant viewpoints. Feminism does not critique maleness, but rather male-dominance, machismo, and patriarchy. Likewise, masculinism does not critique femaleness, but rather discursively seeks to shatter stereotypes about men’s ‘rough-and-tumble’ activities as necessarily equated with violence, domination, and competitiveness. Both discourses have emancipatory potential and are not opposites of each other.

However, in this present historical moment, moving towards a better understanding human health and illness, in all its shades of complexity, properly requires an infusion of feminist discourse to counter prevailing mechanistic, disembodied, and distanced notions of health and illness (Haraway 1991). The feminist perspective’s persistent focus on embodiment and partial/situated knowledge helps to locate states of health and illness as lived experiences that are only partially knowable. This helps to counter the one-size-fits-all approach to therapeutics and health status evaluation. A masculinist approach to understanding states of health and illness is also possible. But I do not think that it is lacking. Feminist understandings help to restore fluidity and balance to a sociomedical view of health and illness that can be rigid, strictly objective and disembodied. Thus, my view is not based in dichotomous essentialism, but rather a purposeful application of multiplicity-making discourse.

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Question 2): In your statement you make an argument for using a political ecology of health/disease framework in your dissertation and discuss core requirements for such an approach (pages 6-7 of your statement). Can you identify, discuss, and analyze examples in the literature which best exemplify prior uses of such an approach or at least examples which come closest to the ideal integrated approach as you have identified it?

Literature that explicitly identifies itself as contributing to the further understanding and elucidation of “the political ecology of health” or “the political ecology of disease” is actually quite limited, relatively speaking. A search of the National Library of Medicine via PubMed with the search phrase “political ecology” produces just 10 hits. Thus, it should not be surprising that prior approaches that exemplify in all dimensions the particular political ecology of health/disease approach that I have proposed are not found in the published peer-reviewed literature of medical geography, medical anthropology, and related disciplines, to my knowledge. However, the framework that I have proposed is drawn from a piecing together of various concepts and approaches from medical geography and medical anthropology and takes as its starting point the political ecology of disease formulation proposed by Mayer (1996, 2000). I will review here the literature that supports the ‘critical political ecology of disease’ framework I have proposed beginning first with literature that is explicitly in the realm of political ecology of health/disease, followed by examples from the general political ecology literature, and concluding with work from medical anthropology, medical geography, and related areas.

As I mentioned in my previous statement, Mayer’s 1996 paper was significant in that it provided the most robust elucidation to date of the outlines of a political ecology of disease framework using illustrative examples such as HIV/AIDS in sub-Saharan Africa, cryptosporidiosis in Wisconsin, and Lyme disease in Connecticut. Several papers in the literature have taken up Mayer’s formulation of the political ecology of health/disease, in

whole or in part, and applied it to a wide range of health and hazards issues. These include the refugee crisis in Africa (Kalipeni and Oppong 1998), the global tuberculosis pandemic (Gandy 2001), landmines in Africa (Oppong and Kalipeni 2005), self-reported health status of 'Namgis First Nation members affected by salmon aquaculture (Richmond et al. 2005), land degradation and disease ecology in Mozambique (Collins 2001; 2002), deforestation and environmental health in southern Malawi (Kalipeni and Fedder 1999), polio resurgence in Africa (Oppong 2006), flood hazard planning in Bangladesh (Paul 2005) and schistosomiasis and dam-building in Ghana (Hunter 2003). All of these authors explicitly refer to Mayer's 1996 political ecology of disease model with the exception of Hunter, who does not refer to political ecology, but whose pioneering study is a sine qua non of the political ecology of disease. They all emphasize local-global linkages and disease ecology or other ecologically-informed approaches. Other work has been done under the "political ecology of health/disease" rubric that does not satisfy the Mayer model, and this work will not be comprehensively reviewed here.

In a 2000 *Social Science and Medicine* article, Mayer briefly reviewed the political ecology of disease framework again, noting recent theoretical developments in the field of political ecology since 1996 (p. 948-950). The developments he points to mainly arise from the incorporation of critical perspectives into political ecology that strengthen its political economy-derived politics/power and profit/revenue calculus. However, Mayer once again underscores the fact that these recent developments in political ecology fail to mention health or disease, echoing similar sentiments from his 1996 article: "morbidity and mortality owing to disease are barely mentioned by those espousing political ecology, yet disease has been and remains a profound aspect of

human existence” (p. 448). In attempting to imagine what a political ecology of disease that incorporates the critical perspectives of Peet and Watts (1996) (and others) would look like, Mayer (2000) writes: “Using an interpretive framework developed in the context of advocating social change provides a challenge for the sociomedical interpretation of disease” (p.949). No examples are provided because, we can conclude, no political ecology studies that challenge the sociomedical interpretation of disease exist!

In my literature search, I did, however, find one study from 1996 published in *Medical Anthropology Quarterly* that does succeed in using a rudimentary political ecology of health framework to challenge sociomedical understandings of a behavior that is often understood as maladaptive and unhealthy: female circumcision. Gruenbaum (1996), an anthropologist at CSU Fresno, seeks to critique the prevailing medical anthropological interpretation of female circumcision as a ‘maladaptive cultural pattern’ by conducting a political-economic analysis of the practice in central Sudan. Gruenbaum argues that “whether a cultural practice is adaptive or maladaptive depends to a large extent on whose viewpoint is taken, where the political forces for and against it lie, and whose interests are served by change” (p. 470). In holding this view, Gruenbaum is echoing views cogently expressed by medical anthropologist Singer (1989) in a paper entitled: “The Limitations of Medical Ecology: The Concept of Adaptation in the Context of Social Stratification and Social Transformation.” Singer recognized the dangers inherent in irresponsible and apolitical application of the notion of ‘maladaptive patterns of behavior’ to describe individual and group behaviors that challenge prevailing unjust norms. In critiquing McElroy and Townsend’s (1978) view on health as a measure

of environmental adaptation (interestingly, the same authors are also a foil for Gruenbaum), Singer writes: “the adaptationist perspective appears to assign inequities in social relationships to the environment, thereby not only legitimizing those inequities as natural, but implying that the noxious consequences of exploitation are indicators of the maladaptation of politically and economically subordinate groups” (p.226). Singer adds that the oft stated assertion that health and disease are a measure of the effectiveness with which humans adapt to their environment “ignores not only the social origins of disease...but also the degree to which diagnosis is a social process, disease a medically constructed category, and illness a socially regulated role” (p.228). Singer’s paper essentially lays a foundation for both Gruenbaum’s work in Sudan and for the critical political ecology of health/disease framework that I have argued for which seeks to challenge sociomedical diagnostics of what constitutes unhealthy and maladaptive human-environment-related behaviors. Gruenbaum challenges the notion that female circumcision is maladaptive by showing that it does not reduce fertility rates, that it is accepted by many villagers, and that it does not foreclose the possibility of sexual pleasure and orgasm. She also points out that a quarter of a million Sudanese died from starvation in 1989-90 and raises the possibility that the female circumcision issue may not be one that require as urgent attention as it receives and is serving other agendas. Gruenbaum argues for a view “in which circumcision is understood as just one of many obstacles to healthy lives for women and girls” (p. 457). Although this paper does not at all end the international debate on the issue of female circumcision (or better called: female genital cutting), it is an example of a political ecology of health study (in this case, based on a fusion of medical ecology with political economy) that challenges

sociomedically accepted notions of maladaptive, unhealthy behaviors and therefore comes closest to the ideal political ecology of health/disease approach as I have identified it. It does not however employ the fully integrated political-economy/cultural ecology model proposed by Mayer.

The political ecology of health/disease model that I have proposed is really a political ecology of psychopathology and health focusing on Cannabis Abuse and other Cannabis-related mental disorders that seeks to challenge the sociomedical understanding that certain patterns of cannabis consumption are maladaptive and therefore unhealthy and mentally disordered behaviors. It seeks to add the anticipated critical dimension to the political ecology of disease framework proposed by Mayer. Similar moves have already been in the wider political ecology literature. Arguing for an anti-essentialist political ecology, Escobar (1999) eschews reified terms such as ‘nature’ and ‘environment’ and offers an expansive definition of an antiessentialist political ecology concerned with “the study of the manifold articulations of history and biology and the cultural mediations through which such articulations are necessarily established” (p. 3). This sort of political ecology has absorbed elements of the poststructuralist analyses of social movements and expert knowledge biases, and Escobar believes that applying political ecology cast in such a light can have significant implications for “social struggles, collective identities, and the production of expert knowledges” (p. 2). Escobar reminds readers that the “natural sciences are not ahistorical and nonideological” (3) and thus are open for political ecological scrutiny. Though he does not identify them as such, diagnostic definitions of disease and disorder are one example of expert knowledges that can be challenged with an antiessentialist political ecology. Escobar goes on to describe

three different “regimes of articulation” of nature and biology which are often in conflict: “capitalist nature: production and modernity”; “organic nature: culture and local knowledge”; and “technonature: artificiality and virtuality”. All three history-biology articulations are relevant to the political ecology of psychopathology and health framework I have outlined. The capitalist nature regime, which Escobar identifies as the best known, is associated with new ways of seeing such as the linear perspective, the objectification of landscape as vista, the privileging of the visual gaze with a concomitant rise in large scale surveillance and monitoring (governmentality), and the production of nature as commodity through the mediation of labor. Importantly, for Escobar, the distanced totalizing gaze associated with the capitalist nature regime also gives rise to modern sciences such as clinical medicine, in whose early history occurred what Philo (2000) reminds us Foucault called ‘primary spatializations’ in which tabulated diagnoses were mapped onto inanimate bodies of *Homo sapiens* (also see Sullivan 2006). Escobar recognizes that diagnostics and disease definition are a way of “integrating the individual (and the biological) into rational discourse” and “creating an alliance ‘between words and things’” (p.6). He shows how disease diagnostics *can* be understood through the lens of political ecology and thus exemplifies the first requirement—disease definition—of the framework I have proposed. This insight allows one to better understand how a political ecology of disease might approach and challenge sociomedical understandings of disease along lines of cultural ecology, political economy, and medical geography. Mounting challenges to pathodiagnostic ‘bodily inscriptions’ are well covered by Parr (1999; 2002; 2004). She writes: “...some individuals...sometimes resist a totalising medical ‘naming’ of their states of mind/body. Hence, when we discuss a ‘geography of mental illness and

mental health', we should be doing so critically, with an eye to the alternative definitions and understandings that individuals and groups have of their own mental states" (1999, p. 183). Escobar's organic nature regime, with its emphasis on local knowledge and 'experiments in living', exemplifies the approach of the second requirement in the political ecology of health/psychopathology model I have proposed regarding localized individual/group interpretations and health-related experiences related to particular human-environment interactions. The clash between localized understandings of particular human-environment interactions and medical/public health policy interpretations of those same interactions creates stressful conditions to which individuals and groups adapt. More on the notion of adaptation shortly. Escobar's third regime, technonature, is all-encompassing of new and emerging developments in technoscience such as biotechnology and human-computer interfacing. One important axis for this political ecology that Escobar identifies is organicity-artificiality. Regarding my proposed work, this provides an analytic for understanding how particular chemical isolates/extracts of the cannabis plant have been marketed, sold, subsidized, and heavily profited from while the organic version remains prohibited. It also helps to understand the transformation of human excrement and hair into biomaterials routinely screened and scrutinized at the molecular level for metabolic evidence of cannabis consumption.

I agree with Anderson's (2000) critique of Escobar remaining "too 'essentialist' in his discussion of three types of construction of the natural realm" (p. 105). I also feel that his association of capitalist nature with objective clinical medicine is somewhat tenuous. While both rely on objectification and parsing, one does not necessarily imply the other. However, Escobar's insights do point the way forward towards a radically new

and radically simple way of understanding human-environment interactions. Robbins (2004), another accomplished and respected writer in political ecology, offers a similar view of the basic contestations in political ecology that provides a useful framework for addressing questions I wish to raise:

In recent history, powerful modern institutions and individuals ([e.g.], environmental ministries, multinational corporations, corrupt foresters) have gained undue and disproportionate power by explicitly attempting to divide and police the boundaries between human and non-human nature, even while allying themselves and building new connections to the non-human world, leading to unintended consequences and pernicious results. In the process, resistance emerges from traditional, alternative, and progressive human/non-human alliances marginalized by such efforts (usually along lines of gender, class, and race) (p. 213).

Robbins' widened view of the basic political struggles taking place at the sites of human-environment interaction is fundamental to the political ecology of biotic Substance consumption-related psychopathology and health I have argued for.

The notion of adaptation is central to the framework I have argued for. Phrasing the question in Robbins' terms, how do individuals and groups adapt to (and challenge) the division and policing of human/non-human nature boundaries by powerful institutions and individuals? The modern concept of human adaptation has its roots in the cultural ecological work of the mid-1950s spearheaded by Julian Steward, student of the renowned anthropologist Alfred Kroeber (Grossman 1977; Singer 1989). The geographer Bennett (1969) in his book on the inhabitants of the Great Plains of North America, helped to bring the human adaptation concept into geography. He offers valuable insight to the nature and type of adaptation patterns that individuals and groups practice when responding to problems and stressors. He sees adaptive behaviors as coping mechanisms that take a multitude of forms including "problem-solving, decision-making, consuming and not consuming, inventing, innovating, migrating, staying" (p. 11).

To define or measure adaptation, Bennett suggests looking in terms of goal-satisfaction and resource conservation (p. 13). He also insists on making the very useful distinction between *adaptive strategies* and *adaptive processes*. Adaptive strategies pertain to “the pattern formed by the many separate adjustments that people devise in order to obtain and use resources and solve immediate problems” and are generally conscious decisions. Adaptive processes pertain to “changes introduced over relatively long periods of time by the repeated use of such strategies or the making of many adjustments” and usually can be seen only by outside observers (p.14). The study of human adaptation patterns is a significant part of work in human geography and has been examined in diverse contexts, from natural hazards and threats to subaltern studies of peasant resistance strategies. Given the universal, embodied human experience of distress and threat, it should be no surprise that adaptation to various types of environmentally-induced distress may take similar forms. Mitchell (1974), in a review on the geographic study of natural hazards states as much: “the insights of natural hazard research may aid in developing general theories of man-environment relations. The possibility exists that models of human response to environmental threat may also function as analogs for research on man’s adjustment to more pervasive forms of social stress” (p. 312). The topical diversity of the studies using Mayer’s political ecology of disease framework also attests to this. Mayer (1996) also recognizes the relevance of socially stressful stimuli for a political ecology of disease framework: “it is important in the context of political ecology to ascertain the causes, both intentional and unintentional, of social isolation and marginalization” (p.451). He would agree, I am sure, that it is equally important to ascertain the responses and adaptations of individuals and groups to conditions that produce social stress.

Literature in ecological anthropology, such as work by Vayda and McCay (1975), has made significant headway in showing that how category of hazards can subsume “social and psychological insults” which produce demonstrable “psychological and behavior adaptations strategies.” In their excellent review of work in this area, they write broadly about the nature of various hazards that face organisms and groups and their responses. They are particularly concerned with those hazards that lead to “the risk of losing an ‘existential game’ in which success consists simply in staying in the game” (p.293). This aptly describes the hazards faced by those who produce and consume cannabis and other forbidden substances. Indeed, Vayda and McCay see the notion of ‘hazards’ to encompass not only “extreme geophysical events such as floods, frosts, droughts, hurricanes, and tornadoes” but also “predation by warfare, plundering or raiding...exactions of tribute and taxes...or acts of religious persecution” (294). Those affected by the ‘war on drugs’ prohibitions, aka ‘the pharmacratic inquisition’ or ‘psychopharmacological Calvinism’ (Ott ‘95;’96;’97; & Riley ’00), do certainly perceive their life hazards in the terms such as predations, raiding, and persecution. These hazards form the backbone of the adaptation pressures I have argued for in my framework.

The critical aspects of the political ecology of disease approach I have argued for which challenge sociomedical understandings of diseases may only be applicable to illnesses characterized by signs and symptoms perceived as maladaptive, such as those often found in psychopathology (Sarason 2002), e.g. DSM-IV substance-related disorders diagnostics, wherein the term *maladaptive* is left purposely undefined. However, specific modifications may allow for its application to other projects of human-environmental depathologization seeking to explain new patterns of health and health hazards.

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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:

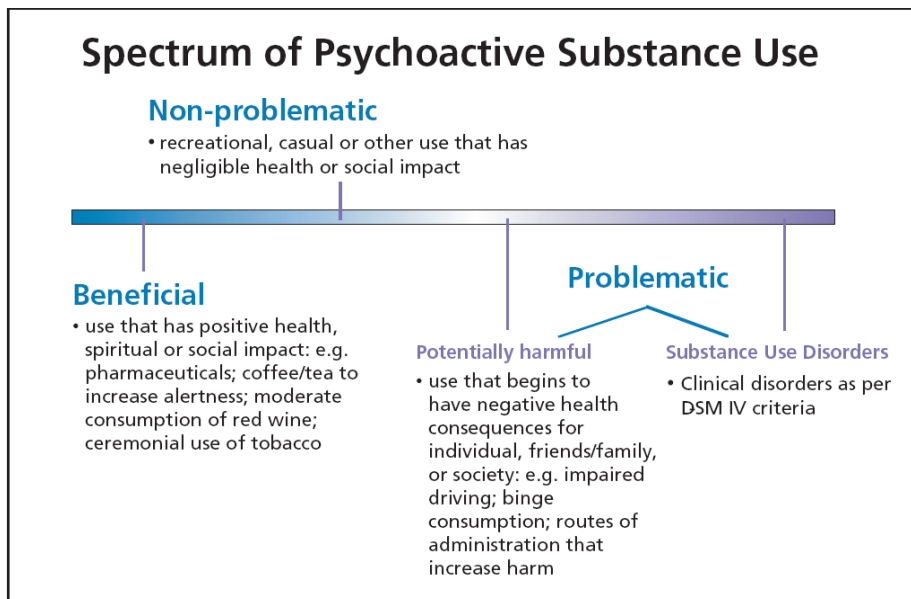
Subjective clinical judgment enters into Cannabis Abuse criterion distinctions regarding the meaning of ‘recurrent’ or ‘maladaptive pattern.’ For example, legal consequence risks are present with any marijuana use level, but may remain latent, or risk exposure only if a person drives or buys. Behavioral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

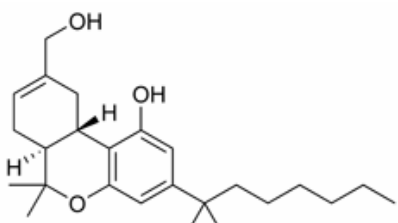


Figure 1: HU210: 3-(1,1-Dimethyl heptyl)-(-)-11-hydroxy- Δ^8 -tetrahydrocannabinol

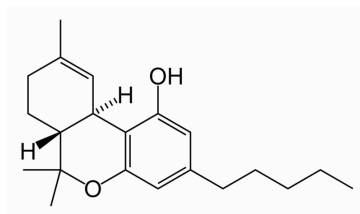


Figure 2: THC: (-)- Δ^9 -tetrahydrocannabinol (1/100th of the potency of HU210)

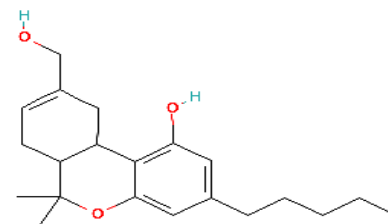


Figure 3: 11-OH- Δ^8 -THC (cannabis metabolite)

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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Question 4): One of your stated areas of expertise is clinical epidemiology. Thus, please consider one of the two articles attached to this exam (S Bent et al, Saw palmetto for benign prostatic hyperplasia. New England Journal of Medicine 2006;354:557-66 OR JA Blumenthal et al., Effects of exercise and stress management on markers of cardiovascular risk in patients with ischemic heart disease: a randomized controlled trial. JAMA 2005;293:1626-34. Do not read commentaries that accompanied or followed the publication of the study.

Is the study convincing? Is the methodology appropriate? Are there problems in the study design? Are these problems significant enough to affect the conclusions? Are the statistical methods appropriate? What alternative statistical methods could have been used?

I will consider the Blumenthal et al. (2005) study on the effects of exercise and stress management on markers of cardiovascular risk in patient-citizens with IHD (ischemic heart disease). This recently published study is convincing to me for several reasons. The improvements in several intermediate cardiovascular risk markers and psychosocial distress/depression measures for the exercise and stress management groups over usual medical care group are convincing because they are sizable and statistically significant in several cases, internally consistent across multiple cardiovascular endpoints, and biologically plausible. A demonstration of “dose-response” would make the benefits of the interventions even more convincing; presumably more rigorous exercise and stress management/reduction regimens would lead to further improvements in cardiovascular function. The fact that the three groups did not have comparable percentages of individuals with annual incomes >\$50,000 or “at least some college” (p. 1630, Table 1) was worrisome given that such measures correlate well with increased psychosocial stress and adverse health outcomes. Greater attention should have been paid in making sure the randomization process made these measures more uniform across treatment groups.

The general methodology of this randomized controlled trial is appropriate, though there are a few small problems which mainly have to do with ambiguity. The study participants were recruited in an appropriate way with sensible inclusion and exclusion

criteria (p. 1627, col. 1). However, while the authors specify what they mean by the inclusion criterion of “documented IHD”, they are ambiguous about what constitutes “evidence of exercise-induced myocardial ischemia within the past year.” Is this evidence from clinical treadmill testing or from self-report of exercised induced angina, or both? Clarification would be helpful for a better understanding of the study participants’ baseline health status. The study uses appropriate methods to generate exercise-induced and mental stress-induced myocardial ischemia. It is good that the authors noted that such methods were found to be effective in previous studies. With regards to subject preparation, the authors write: “Unless medically contraindicated, patients discontinued anti-ischemic medications...at least 48 hours prior to testing” (p. 1627, col. 2). Given that all the patient-citizens being tested have stable IHD, one could make the case that discontinuing medication in all of their cases would be contraindicated. The authors should specify how such decisions of contraindication were made. One of the mental stress induction techniques—judged public speaking on a controversial topic—is innovative and interesting but not all people respond with apprehension in public speaking and not all topics are understood by all to be controversial. Were the topics randomly assigned? Or by some other method? The method of radionuclide ventriculography for determination of the presence of myocardial ischemia is appropriate and was appropriately performed at multiple time points during the testing. Measurement methodology of LVEF (left ventricle ejection fraction) was also appropriate. The method for measurement of segmental wall motion abnormalities (WMA) was ambiguous. “Wall motion for each of the 4 segments was rated by consensus of at least 2 experienced physicians.” Why “at least”? A more appropriate methodology to reduce bias would be

to specify an exact number of experienced WMA readers. The methodology for measuring the flow-mediated dilation index of vascular endothelial function, specifically in the brachial artery, was appropriate. However, glyceryl trinitrate response may be less reliable in those patient-citizens who are taking long-acting nitrates. The method used to measure heart rate variability during deep breathing (HRV-DP) was problematic in that only data from patient-citizens in whom R-R interval lengthening was observed was analyzed. This introduces the possibility of researcher bias in the selection of some patient-citizens with slightly greater or less R-R interval lengthening. A fixed R-R interval change should have been specified or no R-R interval lengthening-based discrimination should have been done in the measurement of HRV-DP. The methods for measuring baroreflex sensitivity, cardiorespiratory fitness, and psychosocial functioning were appropriate and meaningful intermediate clinical endpoints in that poor measurements in all three categories are associated with adverse cardiovascular outcomes. The methodology used in the intervention and “usual care” groups was appropriate but sometimes ambiguous. The method for exercise training was appropriate, although authors should have said something about how such high attendance rates were achieved across the consecutive 16 week period. What kinds of incentives/disincentives were offered? The methodology for stress management training, while appropriate, could have been improved significantly by including games/play activities, film/theatrical/art appreciation, therapeutic recreation, suspension therapy, yoga/massage/meditation/manipulation, and therapeutic landscape exposure. The authors were basically vague about the details of the stress management program; such details would be helpful in understanding their methodology, especially given the success of the

intervention. For example, when the authors write that “social support was considered to be a key aspect of the program” (p. 1629 col. 1), particular examples of how this was achieved would have been very helpful. Finally, the “usual care” group was ambiguously defined. Did any stress management or exercise activities occur in these populations? Even though they monitored to ensure that usual care patient-citizens “had not joined any exercise or stress management training program[s]” (p. 1629 col. 2), this does not exclude the possibility that such activities were occurring in the course of their usual medical care. The authors seem to indicate that only medication management occurred in this group. More clarification is needed.

I do not believe that there were any major problems in the study design. The small number of problems in study design did not significantly effect the study conclusions. Study recruitment methods and sample sizes were appropriate given the desire to achieve a study powered to detect ~15% greater improvements in the treatment groups with a 2-sided test and 5% type I error rate. Block and two-stage randomization was appropriate given the authors’ desires to accommodate availability of patient-citizens for study and to begin active patient-citizen participation in the study no more than 4 weeks following baseline evaluations. The patient-citizens were randomized into three groups—usual care, usual care + exercise, and usual care + stress management training—and received baseline and post-treatment assessments. It would have been very interesting if a fourth group that included both stress management training and exercise would have been created in order to see if the combination of both treatments resulted in synergistically improved outcomes. The study design had a small problem in that all patient-citizens did not undergo HRV-DB assessment because these measurements were

initiated only after the trial had begun. Only 47 patient-citizens were assessed. While appropriate statistical adjustments were made so as not to adversely effect study conclusions, the design could have been improved by including all patient-citizens in this assessment.

The study's statistical methods are appropriate. A general linear model was used in which post-treatment measures served as dependent variables, treatment group as the between-subject factor, and baseline measures/demographics as covariates. Other models were used for specific assessments. The models were checked to make sure they met certain assumptions such as additivity, linearity, and distribution of residuals. I assume they sought to ensure a normal distribution of residuals. This all seemed appropriate. The authors compared exercise and stress management versus usual care and exercise training versus stress management. The main data values on Table 3 are expressed as fitted means with standard errors in parentheses and are adjusted for age, sex, prior MI, pretreatment resting LVEF, and pretreatment level of the corresponding outcome under consideration. Significance is calculated presumably with ANOVA testing. This is all done appropriately. Alternatively, they could have also included an analysis of the stress management group versus usual care and the exercise group versus usual care. They did this occasionally but not consistently. The authors appropriately used an intention-to-treat analysis; for drop-outs, baseline measures were carried forward. The authors also describe a propensity score statistical approach. According to Rosenbaum and Rubin (1983) whom they cite, "The propensity score is the conditional probability of assignment to a particular treatment given a vector of observed covariates." They did not specify how they arrived at their propensity score or how pretreatment values were

treated as covariates. They simply reported that the propensity score analysis was “essentially the same as our primary models” (p. 1629 col. 3). Further clarification would have been helpful.

Overall, the study was well-designed, executed relatively well, and analyzed properly. The results are important for stress-reduction, cardiac, and mind-body medicine.

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Question 5): In the section on substance-related disorders, DSM-IV distinguishes 11 categories of substances. For most of these substances, it also distinguishes between substance abuse and substance dependence. Among these many disorders, is Cannabis Abuse disorder uniquely suspect? Are the abuse disorders more suspect than the dependence disorders? Are the Cannabis disorders more suspect than those of other related substances: alcohol (high prevalence), hallucinogens (similar pharmacology), opioids (endogenous analogs), nicotine (similar health hazards). Does the political ecology of cannabis use cast suspicion on the disorder diagnosis or on its legal status or both? Why?

The Substance-Related Disorders in the DSM-IV are divided into two groups: the Substance Use Disorders (Substance Dependence and Substance Abuse) and the Substance-Induced Disorders (Substance Intoxication, Substance Withdrawal, Substance-Induced Delirium, Substance-Induced Persisting Dementia, Substance-Induced Persisting Amnesic Disorder, Substance-Induced Psychotic Disorder, Substance-Induced Mood Disorder, Substance-Induced Anxiety Disorder, Substance-Induced Sexual Dysfunction, and Substance-Induced Sleep Disorder). Of these, the mental disorders that I call into suspicion are the Substance Use Disorders, especially Substance Abuse but also Substance Dependence to some extent, and the Substance Intoxication disorders. With regards to Substance Abuse mental disorders, Cannabis Abuse disorder is not uniquely suspect considering the full extent to which all transgressions of public health code dealing with Substance Abuse disorder prevention and control are understood as maladaptive within modern mental disorder diagnostics. For all 11 substance categories, the DSM-IV tells us that if persistent or recurrent substance-related legal problems arise in conjunction with substance use, then Substance Abuse mental disorder is the likely cause or underlying pathology that explains the patient-citizen's "clinically significant...distress." It should be noted that pathognomonic diagnosis of Substance Abuse disorder via criteria A3, "recurrent substance-related legal problems", represents only one of 15 possible diagnostic criteria combinations that satisfy the diagnosis using the algorithm of the DSM-IV. The diagnoses arrived at with this single criterion that are

uniquely suspect are those that take into consideration legal problems that arise from ‘victimless infractions’ which are violations of substance prohibition laws. The legal problems that fall into this category would be those related to the possession, manufacture/preparation, and pharmacological delivery of substances or metabolic evidence of their consumption. My basic contention is that if health professionals are going to prominently figure such kinds of legal problems into mental disorder diagnostics, the strength, justification, effectiveness, and fairness of the public health regulation laws which have been transgressed—their context—must be considered as well.

In my Preliminary Statement, I made a list of all the occurrences of the concept of legal problems in the DSM-IV, all of which appear in Substance-related disorders section of the manual with the exception of the reference to “legal difficulties” in the description of conduct disorder. I will reproduce that list here, with some additions, because it also helps to address several parts of this question. I have added underlining to highlight the legal problems that arise from nonviolent and victimless infractions. Reading simply the underlined words brings into relief how these legal problems translate into mental illness.

First, there is “arrests for substance-related disorderly conduct” which appears as a parenthetical example immediately following the diagnostic Criterion A3 of **Substance Abuse**. Under **Alcohol Abuse (305.00)** mental disorder, it is written: “Legal difficulties may arise because of alcohol use (e.g., arrests for intoxicated behavior or for driving under the influence).” Under **Cannabis Abuse (305.20)** mental disorder, it is written “...legal problems that may occur as a consequence of arrests for cannabis possession.” Under **Cocaine Abuse (305.60)** mental disorder, it is written: “Legal difficulties may result from possession or use of the drug.” Under **Hallucinogen Abuse (305.30)** mental disorder, it is written: “...legal difficulties may arise due to behaviors that result from intoxication or possession of hallucinogens.” Under **Amphetamine Abuse (305.70)** mental disorder, it is written: “Legal difficulties typically arise as a result of behavior while intoxicated with amphetamines (especially aggressive behavior), as a consequence of obtaining the drug on the illegal market, or as a result of drug possession or use. Occasionally, individuals with Amphetamine Abuse will engage in illegal acts (e.g., manufacturing amphetamines, theft) to obtain the

drug; however, this behavior is more common among those with Dependence.” Under **Inhalant Abuse (305.90)** mental disorder, we find: “Users can also become agitated and even violent during intoxication, with subsequent legal and interpersonal problems.” Under **Opioid Abuse (305.50)** mental disorder: “Legal difficulties may arise as a result of behavior while intoxicated with opioids or because an individual has resorted to illegal sources of supply.” Under **Phencyclidine Abuse (305.90)** mental disorder: “Legal difficulties may arise due to possession of phencyclidine or to behaviors resulting from Intoxication (e.g., fighting).” Other relevant passages of the DSM-IV on this topic are: “The category of Substance Abuse does not apply to caffeine and nicotine”; “The term abuse should be applied only to a pattern of substance use that meets the criteria for this disorder; the term should not be used as a synonym for “use,” “misuse,” or “hazardous use”; “The essential feature of Substance Abuse is a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of substances. In order for an Abuse criterion to be met, the substance-related problem must have occurred repeatedly during the same 12-month period or been persistent”; “There may be recurrent substance-related legal problems (e.g., arrests for disorderly conduct, assault and battery, driving under the influence) (Criterion A3)”; “Substance-Related Disorders are distinguished from nonpathological substance use (e.g., “social” drinking) and from the **use of medications for appropriate medical purposes** by the presence of a pattern of multiple symptoms occurring over an extended period of time (e.g., tolerance, withdrawal, compulsive use) or the presence of substance-related problems (e.g., medical complications, disruption in social and family relationships, vocational or financial difficulties, legal problems); “Although a diagnosis of Substance Abuse is more likely in individuals who have only recently started taking the substance, some individuals continue to have substance-related adverse social consequences over a long period of time without developing evidence of Substance Dependence.” [bold in original]

So what I am calling ‘suspect’ is the thorough-going insistence that legal problems arising from nonviolent substance prohibition infractions are psychopathognomonic for Substance Abuse disorders. While these sorts of legal problems can arise with any of the 292 controlled substances, they are specifically referred to in 6 of the 11 categories of substances in the DSM-IV. It should be noted that even if the substance in question is not part of one of the 11 categories of substances, the use of the substance can still be diagnosed as substance abuse with the label of **305.90 Other (or Unknown) Substance Abuse**. As noted above, the category of substance

abuse does not apply to caffeine or nicotine. Hughes (1994) notes in the *DSM-IV Sourcebook* that nicotine is the only substance in the DSM to have a dependence disorder but not an abuse disorder. He quotes from the DSM-III-R: “although Nicotine Abuse is logically possible...virtually no one who has not previously been dependent on nicotine uses nicotine-containing substances in a maladaptive way, e.g., episodic use of cigarettes that exacerbates a physical disorder” (pp. 169-170). Hughes goes on to note that “although social, occupational, and psychological problems may be caused by [tobacco] smoking (e.g., in restricted areas), there is no evidence that these are due to an intoxicated state or reach clinical significance” (pp. 112-113). The legal status of tobacco is clearly what keeps the mental disorder of Nicotine Abuse out of the DSM. The same thing could certainly be said about the reason that there is no Caffeine Abuse disorder. Nevertheless, there are clearly situations in which the self-directed consumption of caffeine and nicotine is significantly deleterious to one’s health, if not lethal. However, individuals who engage in this type of behavior do not qualify for substance abuse mental disorders. I should also note here that cannabis use does not have similar health hazards to nicotine use (as the question prompt states) aside from the potential for bronchial irritation and bronchitis when smoked. A recent large, population-based, retrospective, case-control study involving 1,651 Los Angeles area residents demonstrated strong, positive, and significant associations between tobacco smoking and the incidence of head, neck, and lung cancers but no significant positive associations with cannabis smoking and the incidence of those same cancers. In fact, a significant, albeit small, protective effect was demonstrated in one group of combusted cannabis consumers (Morgenstern et al. 2005). With regards to **Alcohol Abuse mental disorder**, as is noted above, the legal problems

that are potentially pathognomonic for this mental disorder are ones that arise from crimes with victims or with a very high potential for victimization. I am not calling this mental disorder into suspicion. Similarly, the legal problems alluded to that are consistent with **Inhalant Abuse mental disorder** also involve violent crimes (although in many places some victimless Inhalant use crimes exist). Surprisingly, for **305.40 Sedative, Hypnotic, or Anxiolytic Abuse mental disorder**, the DSM-IV does not mention any particular legal problems.

Substance Abuse mental disorders are understood to be a residual category for individuals who do not meet the diagnostic criteria for Substance Dependence mental disorder. The DSM-IV notes: “diagnosis of Substance Abuse is preempted by the diagnosis of Substance Dependence if the individual’s pattern of substance use has ever met the criteria for Dependence for that class of substances (Criterion B).” For Substance Dependence, one must demonstrate a “maladaptive pattern of substance use, leading to clinically significant impairment or distress” as manifested by satisfying at least 3 simultaneous diagnostic criteria (none are pathognomonic). Two of the criteria have to do with tolerance and withdrawal, the hallmarks of physiological dependence. I find nothing suspect about these, aside from the potential of confusing the negative effects of ceasing consumption of a substance that provides therapeutic benefits with a syndrome of withdrawal from that substance. Other substance dependence diagnostic criteria could be demonstrated to be present in a particular substance consumer simply due to the fact that the substance is prohibited. For example, a portion of Criterion A5—“a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances)” —could be satisfied solely due to the fact the substance is

prohibited and therefore is unavailable for local or home production and/or distribution (especially true for biotic substances). Furthermore, if a substance is being used medicinally or therapeutically, it could certainly be the case that, as Criterion A3 states, “the substance is often taken in larger amounts or over a longer period than was intended.” Often, individuals ‘discover’ the therapeutic benefits of a substance that was initially intended to be consumed sparingly under an environment of prohibition. Once this therapeutic discovery is made, more of the substance will be needed than was previously intended. Moreover, one may go to greater lengths to obtain it (Criterion A5), similar to the lengths that people may go to in order to obtain any good medicine, even if the medicinal benefit is palliative rather than curative or complementary rather than central. Given the environment of prohibition and the importance of the consumption of the substance to the maintenance of one’s health, the time and effort involved in procurement may cut into time that could be used for doing other activities, such as those enumerated in Criterion A6: “important social, occupational, or recreational activities are given up or reduced because of substance use.” These scenarios could certainly be present in opioid, cannabis, and hallucinogen consumers, and thus their corresponding dependence mental disorder diagnostics for **304.30 Cannabis Dependence** mental disorder, **304.50 Hallucinogen Dependence** mental disorder, and **304.00 Opioid Dependence** mental disorder are suspect.

The substance intoxication mental disorders are also suspect. I believe that in the descriptions of these mental disorders, biases exist that run deeper than those related to judgments about behaviors involving substance prohibition code infractions found in substance abuse and substance dependence mental disorder diagnostics. These biases are

more so of the ethnopharmacologically-centric type. Substance intoxication disorders are understood to be applicable to those who demonstrate “clinically significant maladaptive behavior or psychological changes” following the consumption of a particular substance. Again, nicotine is exempt. In the introduction to the substance intoxication mental disorders section, the DSM-IV makes the following general observations:

Many substances may produce physiological or psychological changes that are not necessarily maladaptive. For example, an individual with tachycardia from excessive caffeine use has a physiological intoxication, but if this is the only symptom in the absence of maladaptive behavior, the diagnosis of **[305.90] Caffeine Intoxication** would not apply. The maladaptive nature of a substance-induced change in behavior depends on the social and environmental context. The maladaptive behavior generally places the individual at significant risk for adverse effects (e.g., accidents, general medical complications, disruption in social and family relationships, vocational or financial difficulties, legal problems).

Let us take a look at the particular criteria involving **292.89 Cannabis Intoxication** mental disorder, **292.89 Opioid Intoxication** mental disorder, and **292.89 Hallucinogen Intoxication** mental disorder. In order to diagnose a patient-citizen with Cannabis Intoxication mental disorder, the following three criteria must be satisfied:

Criterion A: “Recent use of cannabis.”; Criterion B: “Clinically significant maladaptive behavioral or psychological changes (e.g., impaired motor coordination, euphoria, anxiety, sensation of slowed time, impaired judgment, social withdrawal) that developed during, or shortly after, cannabis use.”; Criterion C: “Two (or more) of the following signs, developing within 2 hours of cannabis use: (1) conjunctival injection, (2) increased appetite, (3) dry mouth, (4) tachycardia”; and Criterion D: “The symptoms are not due to a general medical condition and are not better accounted for by another mental disorder.”

What is suspect here is the judgment/bias that the “symptoms” of ‘euphoria’ and ‘the sensation of slowed time’ are somehow clinically significant maladaptive psychological changes. How is euphoria maladaptive? Euphoria comes from the Greek word *euphoros* which means a feeling of well-being, health, happiness or elation. How can a feeling of

health and well-being be maladaptive? Euphoria can be understood as a maladaptive psychological change only if one believes that the social conditions that one *should* be adapting to are not conducive to feelings of health, well-being, and happiness—a deeply suspect position to hold and endorse. Similarly, the ‘sensation of slowed time’ is not necessarily one that is a maladaptive psychological change, especially if this allows one to appreciate moment-to-moment experiences more fully. These are psychocultural states that are de facto pathologized in the DSM-IV. Any ‘social withdrawal’ and/or ‘anxiety’ must be contextualized with the experience of ‘marijuana stigma.’ One should ask: how does the criminalization of cannabis use figure into the lived experience of the cannabinated state and the life experience of a cannabis user in general? With regards to other items, might some not find the benefits of substance consumption to be so positive that they would accept the temporary and reversible risks and impairments that accompany them? For example, in the case of opioid intoxication mental disorder, which is defined similarly to cannabis intoxication mental disorder, a maladaptive psychological change identified is “initial euphoria followed by apathy.” The apathy that follows may be rationally acceptable given the benefits of the experience of euphoria. Returning to cannabis intoxication mental disorder, items listed in Criterion D are simply objective signs of cannabis consumption to help the diagnostician confirm recent cannabis consumption by the patient-citizen. There is nothing pathological about any of these signs, with the exception of tachycardia in some cases. Some of these signs may in fact be indicia of therapeutic effects in some patient-citizens. Similar difficulties and suspicions arise when considering Hallucinogen Intoxication mental disorder. Diagnostics are structured similarly to Cannabis Intoxication mental disorder. One of the

maladaptive psychological changes associated with Hallucinogen Intoxication according to the DSM-IV are “ideas of reference” that developed during, or shortly after, hallucinogen use (Criterion B). The DSM-IV glossary of technical terms defines ‘ideas of reference’ as:

“The feeling that casual incidents and external events have a particular and unusual meaning that is specific to the person. This is to be distinguished from a *delusion of reference*, in which there is a belief that is held with delusional conviction.” A “delusion is of reference” is: “A delusion whose theme is that events, objects, or other persons in one’s immediate environment have a particular and unusual significance. These delusions are usually of a negative or pejorative nature, but also may be grandiose in content. This differs from an *idea of reference*, in which the false belief is not as firmly held nor as fully organized into a true belief.”

A criterion of psychopathology then is the fact that recent consumption of a hallucinogen leads one to *falsely* believe that incidents and events in one’s immediate environment have a particular and unusual meaning that is specific to oneself. Could this not actually be a *true* experience of meaning and reverence? Such experiences are common with several of the biotic substances that I listed in my general statement, such as cannabis, peyote, magic mushrooms, iboga, and ayahuasca (*Cannabis sativa* L., *Lophophora williamsii* J.M.C., 186 *Psilocybin-containing fungi* spp., *Catha edulis* Vahl, *Tabernanthe iboga* L., *Banisteriopsis caapi* C.V.M. & *Psychotria viridis* Ruiz&Pav.) What the DSM-IV may call “Hallucinogen-Induced Toxicity” consumers may come to view as a powerful spiritual experience that may proffer a kind of knowledge (*gnosis*) about oneself and the world. One type of knowledge that people report as an outgrowth of their use of certain psychoactive substances is a sense of unity and at-onement in a non-dualistic sense about themselves vis-à-vis the world. This ‘intuitive knowledge’ can in fact be grounded in modern day systems theory, Gaianism, and deep ecology—all these offering

a modern ‘scientific’ language to describe *gnosis* gleaned from the unitive experiences catalyzed by such substances, which are appropriately called entheogens (Capra 1982, 1988, 1996; Lovelock 1979; Bache 2000; Baetson 1980; Ruck et al. 1979).

I will conclude with a straightforward and succinct answer to the final question: *Does the political ecology of cannabis use cast suspicion on the disorder diagnosis or on its legal status or both? Why?* The political ecology of cannabis use casts suspicion on both the mental disorder diagnoses of Cannabis Abuse, Cannabis Dependence, and Cannabis Intoxication and on the legal status of cannabis consumption—prohibition. The political ecology of Cannabis-related psychopathology reveals the emergency demands for collective resistance and reform and the imperative of heading pleas for the repeal of prohibition and the adoption of a policy of restoration comprised of relegalisation, amnesty, and restitution (d’Oudney et al. 2006). Suspicion is cast on the diagnosis of Cannabis Abuse given the fact that clinically significant distress that manifests as recurrent or persistent cannabis-related legal problems does not remotely imply *de facto* psychopathology (Earlywine 2004), but, in my opinion, flawed and insincere public health regulation of Cannabis Abuse prevention and control, which a critical political ecology of Cannabis Abuse disorder can expose. Suspicion is cast on diagnoses of Cannabis Dependence because some of the described features of this disorder can be better explained with cannabis consumption political ecologies of health forced to operate under an environment of prohibition. The political ecology of cannabis use also casts suspicion on the diagnosis of Cannabis Intoxication, given that it shows how cultural ecological aspects of the psychological states induced by cannabis consumption are inappropriately pathologized in current diagnostics.

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Question 6): Discuss the differential expression of pain in various cultures and cultural groups. Why do these variations exist? What are the therapeutic implications? Implications for the use of cannabinoids? Discuss both “purely” cultural factors as well as genetic factors, citing appropriate empirical and theoretical studies. Is there a geography of pain?

To begin a discussion of the differential expression of pain in various cultures and cultural groups, some ground-laying definitions are in order. What is ‘pain’ and ‘pain expression’? What do we mean by ‘cultures’ and ‘cultural groups’? Just making sense of these terms alone is enough material for several separate dissertations. But perhaps we can find some pithy agreeable common ground. Morris (2001) puts the challenge presented by this question in the following terms: “The challenge of ethnicity is to understand how far the human experience of pain embodies not only measurable differences that distinguish specific groups but also deep similarities that bind us together—despite our diverse, historically changing ethnic and cultural backgrounds.”

Pain is a subjective biopsychosocial phenomenon. In 1979, the International Association for the Study of Pain’s Subcommittee on Taxonomy, composed of 14 distinguished world experts in pain medicine, published a definition of pain they felt would be suitable for scientific discourse. Pain, they said, is “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (Morris 1991, p. 16). Already from this definition, we see non-materialist dimensions of pain that raise important questions. The definition states that pain is an “emotional experience” as well as a sensory one. Given what we know about the somatization of psychological and emotional states of distress (MacLachlan 2004, p.34), and given the falsity of the prevailing “Myth of Two Pains” regarding the separability and distinctiveness of ‘physical pain’ and ‘mental pain’ (Morris 1991, p.9), can we not simply see “psychological and emotional pain” as part and parcel with pain,

even if there is no predictable “sensory” component described “in terms of” “actual or potential tissue damage” accompanying the pain? Furthermore, what all is encompassed by “potential tissue damage”? Certainly, this includes the threat of pain being inflicted, but what level of threat potential needs to be present for the experience of that threat to register as pain in our definition? How imminent must the potential be? And combining this with the earlier query, what if it is infliction of emotional and psychological pain that one is threatened with rather than “actual tissue damage”, at least at the outset? If we see this unpleasant experience too as pain, then would this definition encompass the pain inflicted by imprisonment or the threat of imprisonment? My hope is that this definition of pain would be this expansive, given the particular historical moment we are currently living in with regards to the rise of the penal state and other forms of state violence. The noted Norwegian criminologist Nils Christie, in his book *Limits to Pain* (1981) has written that penal law is really pain law and that carceral criminal justice is really the “calm, efficient, and hygienic operation” of “pain delivery” (Chapter 2). Interestingly, Christie relates that as he was writing his book in 1980, he received in the mail a copy of the March issue of the journal *Nordisk medicin* which reported on the work and mandate of the IASP. Following a quote from the journal, Christie writes:

The research is interdisciplinary. One wonders what would happen if penal experts were included. Would they then compare notes, and try to construct all the other parties’ negations? Penologists might thereby learn more efficient ways of creating pain, doctors more efficient ways to prevent it.

But of course, penologists in our cultures would not opt for membership in the Interdisciplinary [sic] Association for the Study of Pain. They would become provoked and angry even by the mere suggestion. Attendance would make clear what is now blurred. There might be only small problems in delivering pain in societies where pain is the explicit destiny for most people: pain on Earth, pain in Hell. (Even though the ambiguous status of the hangman indicated that the problems back in time were not insignificant.) But that society is not ours. We

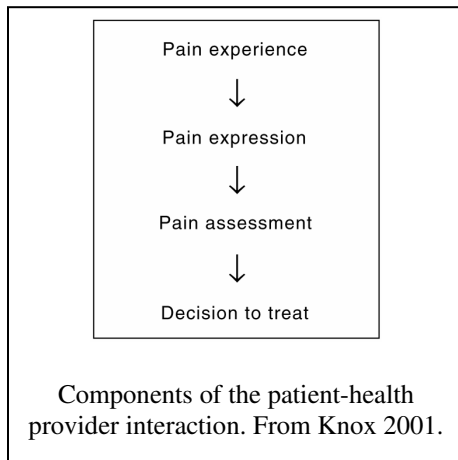
have abolished Hell, and have pain-reduction on earth as one of the major goals. In such a society it is difficult to let people suffer intentionally. (Chapter 2)

I could not agree more with Christie's observations. Understanding the workings of the penal state can indeed help doctors learn "more efficient ways" to prevent pain. But doctors must first come to see the essential operations of the penal state as "pain delivery." And this is as much a cultural issue as it is a medical one. Craig Haney, a psychology professor and legal scholar who attempts to use psychological theory to craft humane penal policies (1997), describes the well-known psychological antecedents to violent behavior and the injustice of meting out unlimited 'penal pain' based on a flawed model of psychological individualism. He begins his article entitled "Psychology and the Limits to Prison Pain" with a quote from Nils Christie: "A suitable amount of pain is not a question of utility, of crime control, of what works. It is a question of standards based on values. It is a cultural question." I shall return at the end to the implications that this view of pain has for 'the geography of pain.'

Returning to the matter of culture and pain expression, a discussion that takes place at the intersection of the 'inner subjective world' and outer 'social world', I again draw insight from the writings of Morris. He writes that "pain...places us within a social world where what we feel cannot be easily disentangled from what we learn from our culture and from how other people respond to us...Pain is also a subjective experience, perhaps an archetype of subjectivity, felt only within the solitude of our individual minds. It is, in addition, always saturated with the visible or invisible imprint of specific human cultures" (Morris 1991, p. 14). For a definition of "human culture", it might be helpful to consider two rather broad definitions such as "the universal human capacity to classify, codify, and communicate experiences symbolically" (Wikipedia) and from UNESCO:

the set of distinctive spiritual, material, intellectual and emotional features of society or a social group...encompass[ing], in addition to art and literature, lifestyles, ways of living together, value systems, traditions and beliefs. (2002)

What we are considering here with regards to members of various cultures and cultural groups is not so much how they differ with regards to pain experience, but more precisely how they differ with regards to ‘pain expression.’ We can understand by ‘pain expression’ as the manner in which an individual’s pain experience is communicated to others, whether intentional or not, including verbal and nonverbal modalities. Certainly, health care services are not the only venues in which pain expression occurs, and treatment of pain does not always occur in the context of a hospital or clinic, but often occurs in the home without medical supervision. But for the purposes of further



narrowing this question, I will only consider pain expression as it occurs in the context of help-seeking for pain alleviation and easement. Pain expression is important in health care provision because it is on the pathway to making the decision to treat or not to treat pain (see Figure, left). All points along this interaction schema can be influenced by cultural

factors such as the cultural background of the patient-citizen, the cultural background of the care provider, and the context of the hospital or clinic where the interaction takes place, steeped as it is in ‘biomedical’ culture.

Tremendous human-to-human variation occurs in pain experience, especially along the dimension of pain sensitivity or pain threshold. It would be sensible to theorize that some of this variation is accounted for by factors such as cultural background,

genetic susceptibility, and environmental exposures. To get an idea of this variation, it is worth remembering that here are those who, from birth, or as a result of head trauma, have conditions which make them totally impervious to pain. One famous man who had such a condition as a result of a head trauma at age seven became a circus sideshow phenomenon in the 1920s known as “The Human Pincushion” (Morris 1991, p. 12). The psychosocial variation in pain experience is also significant. For example, there are members of cultural groups or subcultures that take pleasure in the inflicting pain on themselves. There are individuals who have I heard about from personal acquaintance-eye-witnesses who ‘enjoy the endorphin rush’ of being suspended from the ceiling by twine that is attached to 6 stainless steel hooks piercing the skin flaps of their backs bilaterally in rows of two. The long cross-cultural traditions of flagellations and other acts of pain self-infliction also attest to a spiritual dimension of the experience of pain that some enjoy, similar in some ways to those who enjoy pain-laden masochistic sex acts.

Indeed, human-to-human variation in pain experience is tremendous. How much of this variation is explainable through cultural factors? It turns out that the answer is: not very much. Zatzick et al.’s review of 13 cross-cultural studies that look at individual differences in the ability to discriminate experimentally induced noxious, painful stimuli found no significant ethnic differences (1990). The studies covered data in toto from 42933 subjects. Admittedly, there was wide variation in how the studies defined ethnic groupings and how they administered the experimental pain. But overall, Zatzick et al. conclude that “There is no evidence suggesting that the neurophysiologic detection of pain (i.e., pain threshold) varies across cultural boundaries. On the other hand, pain tolerance reflects the behavioral aspects of pain that are profoundly influenced by

culture” (p.554). This latter conclusion was also arrived at by Greenwald (1991) who found in his survey of 536 patients recently treated for various forms of cancers “no statistically significant relationships...between ethnic identity and measures of pain sensation. However, pain described in affective terms according to the McGill Pain Questionnaire did vary among ethnicities” (p. 157). The affective dimension of pain refers to the feelings of unpleasantness and emotions related to future implications that arise in people who suffer pain. Given the way in which information about the affective dimension of pain is elicited (via questioning from a researcher), it would make sense to consider Greenwald’s finding as a confirmation of the cross-cultural variability in pain expression. After all, subjects in his study ‘expressed’ to Greenwald the nature and content of their pain-associated feelings and emotions. It is reasonable to believe that those similar sensations and feelings were conveyed to those who were helping to alleviate their pain. Other studies have examined cross-cultural variation in pain experience by looking at the amount of analgesia that members of different cultural groups require for post-operative pain. For example, Morris (2001) reports studies showing that “10% of adult dental patients in China routinely receive anesthetic for tooth drilling, as compared to 99% of adult dental patients in America.” Other studies show no significant cultural variation in use of PCA in post-operative pain. The action and pain beliefs of both providers and patients are culturally implicated in these results. Before turning to the question of cultural variation in pain expression, it may be helpful to note how much human-to-human variation in pain experience/sensation is mediated by genetic factors. MacGregor et al.’s (1997) twin study on pain threshold studied pressure pain threshold in 609 female-female twin pairs, of which 269 were monozygotic and 340 were

dizygotic. They calculated a slight excess in the monozygotic intraclass correlation which corresponded to a non-significant heritability of only 10%. This was not altered when adjustments were made for age, current tobacco and alcohol use, current analgesic use, psychological status assessed by the general health questionnaire, and social class. The authors conclude that learned patterns of behavior within families, rather than genetics, are an important determinant of pain sensitivity. However, genotyping studies have had more success. Diatchenko et al. (2004) were able to account for ~11% of the variation in experimentally-determined pain sensitivity in a cohort of 206 healthy female volunteers with haplotype variation at a single gene locus. They showed that individuals who had inherited certain haplotype polymorphisms of the COMT gene (catecholamine-O-methyltransferase) located on the long arm of chromosome 22 (22q11.21) displayed increased or decreased pain sensitivity depending on genotype. Longitudinal follow-up with the cohort showed that those who were in the least-pain sensitive group based on COMT haplotype had a 2.3 times diminished risk of developing temporomandibular joint disorder, a common musculoskeletal pain condition. The question to answer now is whether COMT haplotype distribution varies culturally and geographically. Clearly, genes play a small, but significant role in human-to-human pain sensitivity and propensity for developing chronic pain conditions (Mogil et. al 2005).

Cultural variation in pain expression, as alluded to in the studies reviewed earlier is an empirically determined fact. Wolff et al's review (1985) of the research in this area, which dates back to the seminal work by Zborowski (1952), concludes that there is substantial evidence that variation exists in "pain behavior and response" amongst various "ethnocultural groups" notably along the lines of pain tolerance, verbal

complaints of pain, and attitudes towards pain. Morris (2001) notes in his review that this cultural patterning of pain expression likely occurs well before the acquisition of language by children, given the differences in acute pain response between infants of different ethnocultural groups (as a study of Chinese and non-Chinese Canadian infants demonstrated). Despite these documented differences, Knox (2001) cautions against making generalizations about pain expression in members of given ethnic groups given that the research in this area also “suggests that *intra*-ethnic variations are marked and often more prominent than inter-ethnic differences.”

So, from looking at pain experience, we moved on to look at pain expression, and now, we must consider the latter two points on Knox’s model for patient-provider interaction: ‘pain assessment’ and ‘decision to treat’. What implications does the cultural variation in pain expression have on treatment? Lasch (2002), in concluding her review of research on culture and pain, writes: “The cultural surround of the pain experience—narratives or stories that privilege us to grasp the meanings of pain, disease, life, and death—tells us much about what individuals in pain are experiencing and how we can help them.” Indeed, in cross-cultural interaction between patient-citizen and provider, the subjective experience of pain may take on potentially unfamiliar cultural forms of pain expression. Lasch suggests that attentiveness to narrative and story will help to bridge the gaps in understanding. Several studies report systemic undertreatment of pain in individuals who are members of different ethnic groups. If this is not a function of outright discrimination (let’s hope not), then it must be one of gaps in understanding. Harrison et al. (1996) showed in a 50-patient hospital study conducted in the United Arab Emirates that only nurses who shared the same mother tongue as patients were able to

provide researchers with pain ratings that matched those given by patients. Clearly, the cultural background of care providers plays a role in their baseline ability to understand patients' pain expressions and to effectively respond with treatment.

When it comes to pain treatment with cannabinoids, these same cultural considerations apply. The implications of differential pain expression by various groups and differential interpretation of pain expressions by care providers of multiple cultural backgrounds, including 'biomedical culture', are what must be considered when cannabinoid and cannabis therapeutics are at stake. Let us consider cannabinoid-rich cannabis, which contains 68 cannabinoids that act synergistically and by the entourage effect (Russo 2002, p.366). A physician's willingness to recommend cannabis for analgesia and his/her willingness to approve and grant asylum to a patient who is using cannabis to ease their pain will be mediated by the cultural background of the physician. If the physician has first-hand, direct experience with the unique, analgesic effects of cannabis (see question 3), he/she may be much more inclined to recommend or approve its therapeutic use. This knowledge would certainly be the product of "illegal" or "socially disapproved" behavior and thus would automatically be knowledge that is culturally variant. Physicians may also have second-hand knowledge of the analgesic effects of cannabis and thus may recommend or approve its use notwithstanding the authoritarian messages of zero-tolerance cannabis prohibition. To not be fearful of these latter messages in itself represents a culturally unique American doctor. On the other hand, if a physician has been steeped in cultural messages of the 'dope fiend' discourse, 'hippie-hating', or 'stupid pothead/marijuana stigma', they will be far less inclined to allow such pain control to go on. Indeed, even if they are not 'hippie-hating' etc., the

prevailing 'medical culture' with its hypersensitivity to litigiousness and accepted practice of 'defensive medicine', may influence the physician to steer clear of "Schedule I" substances. Likewise, a patient's willingness to approach a physician for asylum for cannabis therapeutics will be influenced by their perceptions of safety and trust they feel from the medical establishment. Their willingness to even initiate cannabis therapy will be influenced by their membership within certain cultural groups and their past exposures to cannabis consumption, both first-hand and second-hand, and past exposure to cannabis prohibition policing.

Finally, is there a 'geography of pain'? Yes, there is. Crombie, Crombie et al., and Von Korff (1999) sketch out the 'epidemiology of pain', but as with all other diseases in medical geography, the 'geography of pain' goes beyond this. Performing a 'cybergeographic sweep' via google revealed a chapter title by historical geographer Daniel Doepfers called "The Philippines in the Great Depression: a geography of pain"; several poetic, artistic, and literary references in which the term was variously understood as the pain humans inflict upon each other and on the environment and normally obscured by the darkness, the various emotional responses related to pain and coping with pain, the way that pain is physically etched on the body, the experience of going room-to-room in a Timorese building used for torture, and the varied shades of inner pain and suffering. To this I would add the intergenerational 'passing of pain' (Barash 1994), the various forms, meanings, and constructions that arise out of the experience of pain, the pain of imprisonment and its threat, interpersonal and political violence, and the culturally-influenced ways that people cope with this pain in spaces and places in the world. All this, plus the epidemiology of pain, characterize the geography of pain.

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