COMMONWEALTH OF MASSACHUSETTS SUPREME JUDICIAL COURT

MIDDLESEX COUNTY SJC-12279

COMMONWEALTH

v.

JULIE ELDRED

BRIEF OF *AMICI CURIAE* OF 11 ADDICTION EXPERTS IN SUPPORT OF APPELLEE

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QUESTION PRESENTED

May the probationer permissibly be required to "remain drug free" as a condition of her probation, and may she permissibly be punished for violating that condition, where the probationer suffers from substance use disorder [SUD], and where her continued use of substances despite negative consequences is a symptom of that disorder.

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INTEREST OF *AMICI CURIAE*

Amici curiae are addiction specialists in scholarship, practice and law. The social and legal implications of drug addiction have been a central focus of their work, which includes numerous important books, articles and public presentations on this issue.

Amici believe this case raises important questions about principles of behavior, criminal responsibility, and the sound and fair administration of criminal justice. Their teaching, research and clinical experience on the subject have given them a deep appreciation of whether the behavior of people who are addicted, including seeking and using prohibited substances, is responsive to incentives. This case squarely presents this question and hinges on the answer to it. We believe that we can provide the Court with conceptual, scientific and clinical considerations that demonstrate that the brain-disease model of addiction is highly contested and, indeed, contradicted by the data. These conceptual, scientific and clinical consideration also reveal that addicts have the capacity to respond to incentives and reasons.

A complete list of *amici* who reviewed and join in this brief is included in the attached Appendix. *Amici* file this brief solely as individuals and not on behalf of any institution with which they are affiliated.

Amici represent neither party in this action, and offer the following views on this matter.

SUMMARY OF THE ARGUMENT

This brief is a critique of the brain disease model and many supposed implications of that model. It begins with a brief history of the model and moves to a discussion of the motivations behind the characterization of addiction as a "chronic and relapsing brain disease." We follow with an enumeration of fallacious inferences based upon the brain disease model, including the very notion that addiction becomes a "brain disease" simply because it has neurobiological correlates. Regardless of whether addiction is labeled a brain disease, the real question, we contend, is whether the behavioral manifestations of addiction are unresponsive to contingencies. We then present an overview of data demonstrating that addiction is a set of behaviors whose course can be altered by foreseeable consequences. The same cannot be said of conventional brain diseases such as Alzheimer's or multiple sclerosis. The best scientific and clinical data we have do not support the view that addicts are unable to refrain from using substances by choice. By "choice" we mean the product of the capacity to respond to incentive and reasons, which obviously varies among addicts but which are virtually never entirely lost. Data amply show that addicts retain that capacity. Finally, we demonstrate how a decision in favor of the probationer could have significant implications for the future of treatment-based approaches to criminal justice, as well as for criminal responsibility more generally. We conclude that the probationer's claim should be denied because it rests on refuted scientific premises and will have negative consequences if it is accepted.

ARGUMENT

I. THE CHARACTERIZATION OF ADDICTION AS A BRAIN DISEASE IS SCIENTIFICALLY, CLINCALLY AND CONCEPTUALLY CONTESTED

This Part first addresses the origin of the brain disease model and is followed by a discussion of its rhetorical function. We show that despite claims that the model is "generally accepted," it is in fact highly contested and exceedingly controversial in the scientific community. Many eminent scholars reject it, and those who do accept it often do so based on reasons extraneous to its validity. It then turns to conceptual confusions inherent in the model, flawed analogies of addiction to other, recognized diseases and to the process of becoming addicted. The final section demonstrates that adopting a brain disease model of addiction does not reduce stigma. Although the QUESTION PRESENTED uses the term, substance use disorder (SUD), we use the far more common term for the phenomenon, "addiction," throughout this brief.

A. The Origin of the Brain Disease Model

Within the medical and research communities, the dominant narrative holds that addiction is a "brain disease." In a seminal article published 20 years ago in *Science*, "Drug Addiction is a Brain Disease, and It Matters," Alan Leshner PhD, then director of the National Institute on Drug Abuse, or NIDA, part of the National Institutes of Health, proclaimed that addiction was a brain disease on the ground that "addiction is tied to changes in brain structure and function." He had previewed the new formulation in 1995 to addiction experts, but the exposition two years later in *Science* is considered its official introduction to the broad scientific community. The brain-disease model has since become a staple of medical school education and drug counselor training and even appears in the antidrug lectures given to high-school students (Koop, 2007). Rehab patients routinely learn that they have a chronic brain disease. And the American Society of Addiction Medicine, the largest professional group of physicians specializing in drug problems, calls addiction "a primary, chronic disease of brain reward, motivation,

memory and related circuitry" (American Society of Addiction Medicine, 2011). Drug czars under Presidents Bill Clinton, George W. Bush and Barack Obama have all endorsed the brain-disease framework at one time or another (Smith, 2007). The Surgeon General under President Obama endorsed the formulation in a 2016 report on drug addiction, the first time the Office of Surgeon General ever addressed addiction outside of nicotine and smoking. The brain-disease model has been featured in a major documentary on HBO, discussed on talk shows and used in *Law and Order*, and has been on the covers of *Time* and *Newsweek*. The model has become dogma. Like all articles of faith, it is often believed without question, especially by addiction counselors and other clinicians. (Massing, 2000; Rose & Volkow, 2012; Lemonick, 2007; Interlandi, 2008).

Leshner's successor at the helm of NIDA, psychiatrist Nora Volkow, has been a strong proponent of the brain disease model. As she explained in an agency newsletter, the "brain [of an addicted person] is no longer able to produce something needed for our functioning and that healthy people take for granted, free will." According to Volkow, the inferior frontal gyrus, part of the brain's frontal lobe (a region that plays a key role in managing impulsive actions), serve as a set of "brakes" on drug consumption. Addiction disrupts the function of the brakes so that "even if I choose to stop," she told a radio audience, "I am not going to be able to;" the brakes can't perform their inhibitory function (Heyman, 2009).

Before Leshner and his NIDA colleagues designated addiction a disease of the brain — meaning that addiction is fundamentally a drug-induced disorder of disrupted brain function — doctors and much of the public regarded addiction as a vague sort of "disease" that manifested as an uncontrollable drive to use drugs or alcohol. Leshner coined a durable metaphor, writing that drugs "hijack" the brain's motivational and reward circuitry, thereby making the signs of addiction, the persistent seeking and using of substances, involuntary. "It may start with the voluntary act of taking drugs," he said, "but once you've got (addiction), you can't just tell the addict, 'Stop,' any more than you can tell the smoker 'Don't have emphysema'"

(Leshner, 1997). We explain in a later section why this is untrue and why addiction and conventional diseases are not analogous.

We also address the specific meaning of "compulsion" in the context of addiction and in relation to the *Diagnostic and Statistical Manual*, Fifth Edition, of the American Psychiatric Association, or DSM-5. In brief, the Manual does not even contain the word "compulsion" with respect to addiction, nor does it refer to drug taking as an "uncontrollable" act. Instead, it sets out criteria for "impaired control" and states that a patient might manifest impaired control by, among other things, "taking more drug than initially intended" and by relapsing. In other words, "compulsive" does not mean beyond one's control. Another relevant passage in DSM-5 that we subsequently discuss is its "Cautionary Statement for Forensic Use," which warns that the definitions of mental disorders it contains were developed to meet the needs of clinicians, public health professionals, and researchers but "not all of the technical needs of the courts and legal professionals" (at p.25). The statement also notes that a diagnosis, even one involving impairments of control, does not imply that the person so diagnosed cannot control his or her behavior" (*id.*)

Let us now consider the neuroscientific data that are marshaled in support of the brain-disease model. First, however, it is necessary to insert a caution concerning the methodology used in studies of the neurobiological underpinning of addiction because those findings apply to the work discussed in this and following sections. Virtually all neuroscience of addiction studies use as their subjects, addicts who are in treatment for addiction. This group of addicts is therefore <u>not</u> a random, representative sample of addicts. Compared with all addicts, the study population is disproportionately diagnosed with other mental disorders; in other words, the subjects are "co-morbid" ("dually diagnosed") for addiction and mental illness. This means that one cannot reliably know whether any brain findings associated with these subjects are accounted for by addiction, by the other disorder, or by some combination or interaction between the two. Even findings considered by some to be well-established must therefore be evaluated

and responded to cautiously. Although many neuroscientists who work on addiction write as if their findings disclose a strict causal connection between certain brain regions and addictive behavior, virtually all such studies indicate only association (correlation) and claims about causation are unwarranted. To complicate matters further, many brain imaging studies have not been replicated (reproduced) and probably could not be (Szucs & Ioannides, 2017). This "failure to replicate" is due to several factors including small sample sizes, statistical under-powering, and spatial uncertainty relating to measured regions of interest (ROI) in the brain. These and other problems have routinely led to the erroneous reporting of weak correlations as much stronger than they are (Eklund et al, 2016).

On the basis of studies with rats and primates, we have learned that natural rewards such as food, water, and social interactions with conspecifics trigger the release of dopamine in reward circuits (e.g., the striatum). Stimulants (e.g., cocaine and amphetamine) also trigger the release of dopamine in reward circuits. These and related findings encouraged the idea that neuroscientists have a successful understanding of addiction. For example, Nora Volkow in a talk titled "*The Unyielding Power of Dopamine"* (Volkow, 2017) argued that all drugs cause addiction by way of their effects on dopaminergic neurons. However, she ignored well-established research showing that marijuana, nicotine, and opioids typically have little impact on striatal dopamine, yet are addictive. For example, in a study with heroin addicts, a 50 mg dose of heroin produced a desirable euphoric high but no release in dopamine in the striatum (Daglish et al., 2008). Moreover, as is the case for brain studies of addiction in general, the evidence for a causal link is missing. For example, rats continue to prefer saccharin to cocaine even though stimulants have a much greater impact on dopamine than saccharin does (Lenoir et al., 2007).

In a scholarly review of the dopamine brain theory of addiction, published recently in *Nature Reviews Neuroscience,* Nutt and his colleagues (2015) cautioned their fellow neuroscientists that addiction is a complex phenomenon and that brain theories, like all theories,

require scrutiny. Put more generally, the article's point was that enthusiasm for brain theories of addiction, and in particular those based on dopamine, has greatly outpaced research addressing causal relations between the brain and addiction.

The second part of the brain-based account of addiction is that addicts are compulsive drug users because drugs change the brain (e.g., Leshner, 1999, 2001). However, as we show in Sections I and II, this assumption is wrong. As research progresses, we will learn much more about the role of the brain in addiction, but what we learn will not change the well-established facts that addicts retain the capacity to choose to stop using drugs.

B. Rhetorical Functions of the Brain Disease Model

Efforts to position addiction as a "brain disease" were intended to persuade politicians and society to take the problem seriously other than as a moral failure. The model's appeal is obvious: It is tidy. It signifies medical gravitas and neuroscientific sophistication. In practical terms, advocates of the brain-disease model hoped that this portrayal would inspire insurance companies to expand coverage for addiction and politicians to allocate more funding for research and treatment (Babor, 1990; Rosen & Savory, 2012). Prior to serving as NIDA director, Leshner served as acting director of the National Institute of Mental Health. There, he saw how brain-disease "branding" could prompt Congress to act. "Mental health advocates started referring to schizophrenia as a 'brain disease' and showing brain scans to members of Congress to get them to increase funding for research. It really worked," he said (Leshner, 2009).

Several scientists have expressed the opinion that re-classifying addiction would help them recruit more young scientists into the field. "I think one of the issues that has kept scientists from working on this is the same [moral] stigma," says a scientist quoted in a paper by psychologist Rachel Hammer of the Mayo Clinic and colleagues. "I think if we had a way of making this process be thought of as a disease you are going to have a lot more scientists willing to roll up their sleeves to work on the

problem," the scientist continues. Thus, Hammer and colleagues conclude that "addiction-as-disease was an important factor in scientists' efforts to obtain funding and build research teams." (Hammer et al, 2013).

Many proponents of the brain-disease concept were deeply committed to dispelling the stigma surrounding addiction. As Leshner wrote in *Science* in 1997,"The gulf in implications between the 'bad person' view and the 'chronic illness sufferer' view is tremendous." Medicalizing the condition, they hoped, would rehabilitate the public image of addicted individuals, transforming them from undisciplined deadbeats to people struggling with an ailment. This approach had its roots in the world of mental health advocacy. Until the early 1980s, many people blamed parents for their children's serious mental problems. Then advocates began to publicize neuroscientific discoveries, demonstrating, for example, that schizophrenia is associated with abnormalities of brain structure and function. (Goldstein & Rosselli, 2003; Phelan et al., 2002; Illes et al., 2008; Borgelt et al., 2011; Buchman et al., 2013). The science was not as secure as advocates for the mentally ill had hoped, but the idea of using the same de-stigmatizing strategy for addiction took hold.

Many experts credit the brain-disease narrative with enhancing the profile of their field. The late Bob Schuster, head of NIDA from 1986 to 1991, admitted that although he did not think of addiction as a disease, he was "happy for it to be conceptualized that way for pragmatic reasons… for selling it to Congress" (Schuster, 2007). For decades, addiction research had been a low-status field, disparaged by other researchers as a soft science that studied drunks and junkies. Now the field of neuroscience was taking greater notice. "People recognize that certain decision makers and others are very impressed with molecular biology," said Robert L. Balster, director of the Institute for Drug and Alcohol Studies at Virginia Commonwealth University (Balster, 2004).

Psychiatrist Jerome Jaffe, an eminent figure in the field and the first White House adviser on drugs (the precursor of the "drug czar"), saw the adoption of the brain-

disease model as a tactical triumph but a scientific setback. "It was a useful way for particular agencies to convince Congress to raise the budgets (and) it has been very successful," he said. Indeed, neuroimaging, neurobiological research, and medication development consume over half of the NIDA research budget. In light of the agency's reach – it funds almost all substance-abuse research in the United States – it sets the national agenda regarding which research gets funded and therefore the nature of the data produced and the kinds of topics that investigators propose. But Jaffe argues that the brain-disease paradigm presents "a Faustian bargain – the price that one pays is that you don't see all the other factors that interact (in addiction)" (Jaffe, 2007).

C. Conceptual Confusions Associated with the Brain Disease Model

This sub-section addresses a number of conceptual confusions. It starts by explaining that brain changes associated with persistent seeking and using substances do not mean that these behaviors are the signs of a brain disease and that such brain changes do not mean that persistent seeking and using of substances are involuntary actions in the legal sense of the term, "involuntary." This sub-section then shows that the analogy of addiction to conditions that are unquestionably diseases is false and concludes with a demonstration that although no one chooses to be an addict, addicts choose to persistently seek and use.

1. Brain Changes Do Not Necessarily Signify Brain Disease

On the one hand, every experience changes the brain – from learning a new language to navigating a new city. On the other hand, not all brain changes are equal; learning French is not the same as acquiring a crack habit. If brain changes signified a disease state per se, however, we would all be diseased all the time. In addiction, intense activation of certain systems in the brain makes it challenging, but by no means impossible, for users to quit, but this does not mean that characterizing addiction as a brain disease is necessarily the most useful model for explanation and treatment.

Endowing brain changes with too much clinical significance reflects the power of the increasingly neurocentric perspective that researchers and the public apply to behavioral conditions. Neurocentrism is a term coined to describe the view that human behavior can be best explained by looking solely or primarily at the brain. In an instance of a brain disease, psychotic disorders, and some others, the best initial treatments often manipulate the brain directly (though medication, mainly). Addiction is not such a condition. It is true that replacement opioids such as buprenorphine can be stabilizing because they prevent withdrawal symptoms and suppress craving, but instead of focusing narrowly on neurobiology, as a brain-disease model implies, there is greater value in viewing addiction as a multifaceted behavior that operates on several levels at once, including molecular function and structure, brain physiology, motivation, personality, the psychosocial environment, culture, and social relations. The lower levels of explanation, particularly those involving the brain, are simply one part of an enormously complex causal and clinical story. Indeed, they are not necessarily the most informative for most practical purposes, such as the prevention and treatment of addiction.

Over-emphasizing the neural level of analysis when conceptualizing addiction impedes our progress in treating and preventing it because it distracts us from paying needed attention to users' motives, to their unappreciated decision-making capacity, and to their abilities to respond to incentives. To be sure, addiction can be partially explained according to how it operates at the level of neurons and brain circuits. In this respect, arguably, addiction is a brain problem. But it is also a personality problem, a motivational problem, a social problem, a cultural problem, and so on. There is no scientific or clinical reason to privilege one level of analysis above all of the others unless doing so enhances our ability to respond effectively. At every one of those levels, we can find causal elements that contribute to excessive and repeated drug use and to potential strategies that can help bring the behavior under control.

In a 2016 article in the *New England Journal of Medicine*, Nora Volkow and others contend that the more we understand the neurobiological elements of addiction, the more we come to understand that this condition is a brain disease (Volkow et al. 2016). But this makes as much sense as concluding that because we now know more about the role of personality traits, anxiety and impulse control deficits in increasing addiction risk, we can, at last, recognize that addiction is a disease of personality. This contention that neurobiological understanding of a behavior entails that the behavior is a disease simply begs the question. As Volkow et al. write:

> In the past two decades, research has increasingly supported the view that addiction is a disease of the brain.… the underlying concept of substance abuse as a brain disease continues to be questioned, perhaps because the aberrant, impulsive, and compulsive behaviors that are characteristic of addiction have not been clearly tied to neurobiology… (at p. 363)

The model does not "continue to be questioned" because the linkages between addiction and biological processes are not obvious enough, however. It is clear and wholly expected, that a behavior as dramatic as addiction would have neural correlates (Volkow, 2006). Assuming we had sufficiently advanced science to identify such correlates, the absence of them would represent a profound upheaval of everything we know about biology in general and neuroscience in particular. But those linkages show only that the brain is involved with drug addiction, much as the brain is involved with all discrete behaviors. The linkages do not, per se, make the case that addiction is best defined as a brain-based phenomenon. Indeed, we fully expect more details about the biology of addiction to be uncovered in the near future. But that won't make it any more a brain disease than it is now.

2. Brain Changes Do Not Per Se Signify Involuntariness

Essentially any experience that changes behavior or consciousness must also change the brain. For instance, brain-imaging studies report persistent changes in the brain as a function of reading a novel (Berns et al., 2013). Reading and writing this brief changes the brains of the readers and writers. Thus, the question is not whether drugs change the brain, but whether they do so in a way that renders drug use no longer voluntary. The primary reason to reject this claim is conceptual. Section II. infra reviews the scientific literature**.**

Let us begin with meaning of "involuntariness," which can be either literal or metaphoric and normative. A human bodily movement is literally involuntary if it is a pure mechanism and not an action at all. Spasms and reflexes are examples. In criminal law, this is instantiated in the "voluntary act" doctrine that is an element of all crimes. Metaphorical compulsion exists when the bodily movement is an action, but it is done under a situation of constraint or hard choice. Duress is a classic example. The prohibited act a defendant performs in a do-it-or-else situation is surely an intentional human action, but if the choice is too hard to expect most people not to yield to the threat, then we may excuse the agent. Which choices are "too hard" is of course a normative social, moral and ultimately legal question.

Duress involves two parties, but in one party cases, such as giving in to a strong desire one knows one should not satisfy, it is much harder to assess the level of constraint because the subjective variables involved in deciding whether sufficient constraint exists are hard to assess. Such considerations led both the American Bar Association (1983, 1989) and the American Psychiatric Association (1983) to officially recommend the abolition of "control" tests for legal insanity. The test for metaphorical involuntariness in one party cases most always rests on a behavioral analysis because intentional action is being evaluated and there is no proxy measure, whether it is psychological or biological, that is available to reliably assess the level of constraint in these cases.

Despite laboratories all over the world working to find an adequate measure of whether a person could not control herself, the line between "could not" and simply "did not" is still elusive. There is no such consensually accepted measure (Morse, 2002, 2004, 2016, and in press). The conclusion that the agent could not control herself is typically based on a reverse inference. If the agent continues to engage in the behavior despite negative and sometimes disastrous consequences and the agent reports that she would like to stop, then we conclude that it must be the case that she could not control herself. But this is not a scientific judgment. It is at best a common sense inference, and the question is whether it is correct, a question addressed in Section II.

Addiction is a quintessential case of one-party metaphorical involuntariness. The necessary, foundational criteria of addiction—persistent seeking and using substances—are unquestionably human actions and not mechanisms like spasms or reflexes. If the agent stops persistently seeking and using, as so many addicts do, either occasionally or permanently, the agent is not then addicted. Moreover, in few cases is the agent forced to use drugs by an external threat. The motivation is entirely internal.

We explained above that <u>all</u> human action has biological causes at the level of the brain. That is simply a fact about human beings. If your brain is dead, then so are you and you are not acting. We also explained that the brain is constantly changing in response to various stimuli. If changes in the brain signified involuntariness, then all human action would qualify as involuntary. Some scientists and even some philosophers think that this is true, but it is simply an example of unrefined biological determinism that holds that if an action has biological roots, as all do, then it must be a mechanism or beyond the agent's control. This philosophically contestable view—and it is a minority view among philosophers of mind, action, and responsibility— is certainly inconsistent with the moral and legal structure of our society that makes the morally powerful distinction between voluntary and involuntary actions.

Once again, whether sufficient constraint exists to lead to the conclusion of an action being involuntary in a one party case must be assessed behaviorally. We must assess the psychological processes, such as desires, beliefs, memory, and judgment, for example, that are implicated in all human action, including yielding to untoward desires. Neuroscience may eventually help us understand those processes better, but simply because addiction seems to be associated with certain characteristic brain changes, does not per se mean that persistent seeking and using of substances is involuntary.

We typically do not know whether and to what degree the differing brain characteristics of addicts and non-addicts are the result of addiction itself or pre-existed the addiction. There are also not yet any population-based studies large enough to understand the statistical range of brain activity and brain anatomy. Consequently, we usually do not know if there are brain "changes" or simply different brains. It would not be surprising if both were true because everything, including the persistent use of substances, changes your brain. But ethical constraints prevent us from doing prospective, long-term controlled studies on human subjects to answer this question. Using animal subjects sheds some light on the problem, but with all due respect, most other animals (especially the rodents that are the focus of most neuroscience work on addiction) are not creatures like us that act for psychological reasons, such as deep-seated angst, profound boredom, or concerns about the future, although they may be profoundly intelligent in some respects. An addict acts for these and other psychological reasons; an addicted mouse or rat does not. In any case, the existence of brain changes does not per se mean the behavior is involuntary.

One might claim that, regardless of cause, the different characteristics of the "addicted brain" are "abnormal" and therefore associated behavior is involuntary, but this claim is confused. We typically conclude that the changes in brain are abnormal because they are associated with behavior we have decided on other grounds is abnormal. But that just regresses the issue to one involving the assessment of behavior. After all, scientifically rigorous neuroscientists do not go on "fishing

expeditions." They first identify what they believe is a well-validated or well-characterized behavioral condition that they wish to investigate because the behavior is of interest for some reason other than it will have neural correlates. Thus, scientists study addiction because they have already decided that the behavior is abnormal for reasons other than differing anatomy or function. However, they presume a priori that the brain is the source of the problem and go on to study the anatomy and function of the brains of those people who persistently seek and use drugs despite the negative consequences of these actions. And as explained earlier in this sub-section, there are no consensually validated behavioral measures of control capacity. Consequently, the virtually always correlational findings of neuroscientific studies of this capacity must be approached with genuine caution.

Also, even if an abnormal variable is causally involved in some action, it does not mean the action is metaphorically involuntary. Imagine an armed robber who intermittently has episodes of hypomania characterized by exceptionally high levels of energy, overconfidence and self-importance. He only robs when in a hypomanic episode because only then does he feel sufficiently energetic and confident enough to engage in such physical, high risk behavior. But for his hypomania, he does not rob. His robbing is surely action and it is surely voluntary. Depending on the nature of his psychological state at the time of the crime, we might excuse him because he suffered from significant rationality impairments, for example, but that does not mean his actions were involuntary. Moreover, the brain of an individual who has hypomania surely instantiates certain brain states that differ from the brains of those without this condition. Even abnormal brain changes do not per se mean that persistent seeking and using substances are involuntary.

Everything we say about whether brain changes associated with addiction per se prove the involuntariness of persistent seeking and using of drugs also applies to the genetic bases for addiction (Morse, 2011, 2014). Twin and adoption studies have repeatedly demonstrated a genetic predisposition for alcoholism (e.g., Cloninger, 1987), and the limited amount of research on the genetics of illicit

drug use suggests the same for drugs such as heroin, cocaine and marijuana (Tsuang et al., 2001). Excepting such variables as linguistic accent and choice of religious affiliation, all individual differences in behavior are partly attributable to differences in genes, however, including voluntary acts. The brain is the biological organ that is necessary for choice, and brain structure and development follow the blueprint set by DNA. Thus, there is no necessary connection between heritability and involuntariness. In support of this point, monozygotic twins are much more likely to share similar religious and political beliefs than are dizygotic twins, even when they grow up in different homes and were separated before the age of one year (e.g., McCourt et al., 1999; Waller et al., 1990).

These beliefs, like all mental states and actions, have genetic underpinnings but they are not necessarily involuntary. The relevance to addiction is that a genetic predisposition is not a deterministic cause of involuntary behavior, just as drug-induced brain changes are not. If the genetic basis for a behavior were a condition that negated responsibility, no one would be responsible for any behavior (Morse, 2011). We agree that some brain alterations are associated with psychological states that can make it more challenging for addicts to make certain choices, but Section II demonstrates that those changes do not come close to eradicating the capacity to refrain from persistent seeking and using substances.

Finally, we question the assumption that the symptoms of addiction were ever officially designated as "uncontrollable." This is an important matter given the charge that the probationer has been subject to "cruel and unusual punishment" because she was expected by the court to modify a behavior (drug taking) that is intrinsically beyond modification. An examination of DSM-5 is highly relevant here and it offers no support for the assumption that SUD symptoms are "uncontrollable."

According to the Manual, a person can meet criteria for severe SUD if she meets criteria that fall under four organizing categories. The category most relevant to the matter at issue is called "impaired control." Among the

criteria that count as impaired control, according to DSM-5, are "wanting to cut back on problematic use but being unable to do so" or "using much more of the substance than originally intended." These are ambiguous criteria. What does it mean to be "unable" to do something? How do we distinguish between an act that is truly irresistible and one in which an urge to act is simply not resisted although the agent could do so? Brain scans are of no use here. And the ample evidence on contingency management show that addicts are indeed capable of resistance – whether they exercise that capacity is another matter. Similarly, what does it mean to persist in an activity when we don't want to? We often do something longer than intended (e.g., a Boston-based professor taking a moment to see how the Red Sox are doing when preparing a lecture; staying at the office longer than promised; hanging out with friends rather than mowing the lawn, etc.) and relapse may equally well indicate ambivalence rather than "lack of control." These interpretations comport more faithfully with what we know about how and why addicts quit drugs than the idea that addicts continue to use because, thanks to brain changes, they cannot do otherwise. It is worth noting that after 1980, the DSM architects adopted an "atheoretical" approach to formulating diagnostic criteria. The idea was to keep definitions strictly descriptive and not comment upon whether symptoms are modifiable.

3. Comparisons with Conventional Brain Diseases Are Flawed

What's more, addiction is a condition whose symptoms (persistent drug use despite negative sequelae) can be coerced or incentivized to cease. True, one might coerce or incentivize a diabetic to observe his diet and take medication regularly, activities which will likely result in improved glucose control, but one is not coercing or incentivizing the underlying mechanism. After all, you cannot tame insulin dysregulation, the underlying pathology, with contingencies.

Granted, some patients with diabetes, hypertension, and asthma will experience exacerbation of their conditions despite having followed instructions faithfully (some autonomous physiological disruption clearly is at

work), but addiction is not a condition that worsens independent of the behavior of the addicted person. Conversely, if the user follows expert advice – "do not use drugs" – she no longer has the "disease" of addiction. This is because, again, addiction is defined by signs and symptoms alone. The addiction itself – that is, persistent drug use despite negative consequences – is not diagnosed by brain changes: the behavior *is* the disease. In other words, observable drug taking *constitutes* the disease state, it does not *manifest* it. Addiction is not a latent entity (alterations in brain function) that causes observable manifestations (drug use); rather it *is* the observable manifestation.

4. No One Chooses to Become an Addict, but They Choose Nonetheless To Use

The paradox at the heart of addiction is this: How can the capacity for choice coexist with self-destructiveness? "I've never come across a single person that was addicted that wanted to be addicted," says neuroscientist Nora Volkow (Gugliotta, 2003). One could say the same of an obese person: how many of us have ever come across a heavy person who exercised his or her freedom expressly toward the goal of becoming fat? Many undesirable outcomes in life arrive incrementally.

But if addiction is a choice, why would anyone "choose" to engage in such a self-destructive set of behaviors? People don't choose to use addictive drugs because they want to be addicted. People choose to take addictive substances because they want immediate relief, or in some cases to seek out novel psychological or physiological sensations.

Let's follow a typical trajectory. At the start of an episode of addiction, the drug increases in enjoyment value while once-rewarding activities such as relationships, job or family recede in value. Although the appeal of using starts to fade as negative consequences pile up – spending too much money, disappointing loved ones, attracting suspicion at work – the drug still retains value because it salves psychic pain, suppresses withdrawal symptoms and douses intense craving. The brain disease model cannot

accommodate the fact that people use drugs and continue to use them for *reasons*.

The idea that no one wants to become an addict leads to the much-promoted false dichotomy captured in a statement by the former Surgeon General, Vivek Murthy. In a 2016 report on addiction, the Surgeon General presents a choice: "It's time to change how we view addiction," he writes, suggesting that addiction is solely a brain disease (U.S. Department of Health and Human Services, Office of the Surgeon General, 2016). True, addiction per se is not a character flaw, but neither is it an involuntary process, which is precisely what "hijacked" neurobiology and "brain disease" imply. But we should be able to create a vibrant middle ground in which we both recognize the choice-making capacities in addiction and leverage them to therapeutic ends while advancing public investment in humane care (Heather, 2017).

D. There Is No Evidence That the Brain Disease Model Meaningfully Reduces the Stigma Associated With Addiction

A robust literature indicates that biological explanations of behavior do not produce some of the responses that brain disease advocates had hoped for. For example, they appear to foster pessimism about the likelihood of recovery and the effectiveness of treatment (Schomerus et al., 2012). This finding may seem counterintuitive. One might think that a biological explanation would be good news to a patient – and to be sure, some people with mental illness do indeed find it a relief. But when the patient's affliction is addiction and there are no medical cures to restore an addict's disrupted brain, emphasizing the biological dimension seems misguided. We offer just two examples of a more extensive literature on the effect of framing behavior as mediated solely by biological processes.

Rachel Hammer and colleagues conducted in-depth, semi-structured interviews with 63 patients in treatment for addiction in alcohol and/or nicotine treatment centers in the US Midwest and with 20 addiction scientists of various kinds (Hammer et al., 2013). Interviewees were

asked about their understanding of addiction, including whether they considered it to be a disease. The authors' conclusion from these data was that, despite popular arguments that framing addiction as a disease will improve treatment outcomes and decrease moral stigma, such a framing is not only unnecessary, but may be harmful. They observe: "Rather than a malady of the weak-willed, addiction reframed as a pathology of the weak-brained (or weak-gened) bears just as much potential for wielding stigma and creating marginalized populations." (p. 28)

Kvaale and colleagues (Kvaale et al., 2013) carried out the first meta-analytic review of studies looking at the effects on stigma of biogenetic explanations of mental disorders, including substance use disorders. Samples included in the review consisted of lay people, professionals, and individuals themselves affected by psychological problems. The main finding was that biogenetic explanations did appear to reduce blame but also induced pessimism over the future prospects of those suffering from these disorders. It was also found that biogenetic explanations increased endorsement of the stereotype that people with psychological problems are dangerous, an understandable reaction to the idea that addiction, for example, is the result of permanent changes to brain mechanisms over which the sufferer has no control.

II. ADDICTS RETAIN THE CAPACITY TO CHOOSE TO REFRAIN AND DO RESPOND TO INCENTIVES AND REASONS

In the hands of those who subscribe to and promote the brain-disease model, brain imaging is often intended as a visual refutation of the existence of the addict's capacity to refrain from using substances. In a typical imaging experiment conducted with positron emission tomography (PET) or functional magnetic resonance imagining (fMRI), addicts watch videos of people handling a crack pipe or needle, causing their prefrontal cortices, amygdala, and other brain structures to activate beyond the base rate of activity in the region of interest (the entire brain is active all the time) (Goldstein & Volkow, 2002). Videos of neutral

content, such as landscapes, induce no such heightened response while the brains of comparison subjects presented these stimuli are being scanned. The resultant technicolor images of affected brain regions, which are simply graphic representations of complex mathematical data and are not "pictures" of the brain, are undeniably arresting. These images are meant to convince us that the mere will to change or choice in the face of rewards or punishment cannot be expected to override these tissue or physiological changes. After all, it appears that one can "see" the damage inflicted on the now allegedly "broken" brain.

But seeking and using drugs can be affected by the will and does respond to incentives, as this Section will demonstrate. As psychologists and philosophers have underscored, and as we explained above, the common interpretation of pathological behavior as involuntary is often informed by a primitive form of biological determinism. If biological roots can be found, then we reflexively think "disease," and assume that its signs, like seeking and using substances, are not actions but pure mechanisms. Addiction may narrow addicts' focus and reduce their ability to take pleasure in non-drug experiences, but it does not turn them into automatons or slaves to their desires. They remain agents who can and do react to a variety of sanctions and incentives.

The data show that individuals who meet the American Psychiatric Association's criteria for "substance use disorder" (the technical term for "addiction") stop using drugs as a function of the factors that influence choices for all people, such as economic concerns, legal concerns, family issues, and moral values. To help put this conclusion into context, these same factors do not affect the symptoms of cancer, schizophrenia, or even diabetes, a disease with significant behavioral aspects. That is, drug use in addicts differs from the symptoms of widely recognized diseases. But first, consider a few of the basic features of addiction.

As we explained previously, addictive drugs act on the brain, producing virtually instantaneous changes in psychological state that often include intense feelings of pleasure, freedom from worry, and peace. However, with continued use, the strength of the immediate pleasurable drug effects decrease and negative effects begin to

accumulate. Direct negative drug effects include tolerance and withdrawal, and indirect negative effects include socially mediated problems, such as fear of arrest, possible job loss, problems with family members, and the host of issues that accompany illegal or frowned upon activities. Although this is a highly simplified, abbreviated account, it yields clear predictions regarding the course of addictive drug use given the assumption that addicts retain the capacity to choose not to use drugs.

(1) We should expect an initial, positive "honeymoon period" of escalating drug use.

(2) Then, there should be a period of ambivalent drug use, for example, addicts will quit using then start up again, then quit using, etc. (Indeed, many experts think that ambivalence is an almost invariant feature of addiction.)

(3) Finally, drug use ends.

The costs and benefits of drug use vary from individual-to-individual, and alternatives to drugs vary across individuals. Consequently, we should expect large individual differences in how long each stage of addiction lasts. Notice that this account differs markedly from the claim that addiction is usually, let alone invariably, a chronic, relapsing disease.

Recall that the idea that addiction is a disease is based in large part on studies of addicts in treatment (e.g., McLellan et al. 2000; O'Brien & McLellan, 1996). However, it is widely acknowledged that since most addicts do not seek help from clinics, clinic-based research may provide a biased account of addiction (e.g., Robins, 1993). To avoid these biases, researchers organized large, nation-wide epidemiological studies that recruited participants scientifically. For instance, the subject pools numbered in the thousands and matched the demographic characteristics of the nation as a whole (see Robins & Regier, 1991 and Heyman, 2009 for the historical background of these ground-breaking epidemiological studies). Some participants had been in treatment but this was not a necessary criterion. To date, four major surveys have been published (Anthony & Helzer, 1991; Conway et al., 2006; Kessler et al., 2005; Stinson et al., 2005; Warner et al., 1995).

The basic findings include the following: Most of those who were addicted to illegal drugs stopped using by about age 30. Addiction to legal, more readily available, drugs (e.g., alcohol and cigarettes) persisted considerably longer than dependence on illegal drugs (e.g., cocaine). Most addicts quit using without professional medical or psychological assistance For instance at about age 42, between 75% and 83% of those addicted to opiates, cocaine, and marijuana no longer met the criteria for addiction. In support of this result, a study that carefully tracked the time course of addiction revealed that the asymptotic remission rates were higher than 90% for illegal drugs (Lopez-Quintero et al., 2011). Importantly, the high rates at which addicts quit drugs was not an artifact of high mortality rates or other methodological issues (Heyman, 2013).

Most addicts quit and do so on their own. Addiction seems to be among the most spontaneously "remitting" of all the conditions termed major mental disorders, which is a very inconvenient fact for the position that addiction is a "chronic and relapsing brain disease." Consequently, it is reasonable to speculate that quitting was due to the gradual accumulation of the negative effects of drug use, particularly those related to the responsibilities that often accompany early and middle adulthood. A large body of research supports this line of thinking.

In interviews and memoirs, addicts identify both practical and moral reasons for quitting drugs. The following paraphrased quotes are typical: "I wasn't raised to be a bad parent," "I wanted my parents to be proud of me," "I was too old to go back to jail," "I could no longer afford drugs and groceries," "I knew I would die if I didn't stop," "I wasn't born in order to become a drug addict." In a study of heroin addicts, Waldorf (1983) quantified the explanations for quitting. In order of most frequent to least frequent, they were: "It was time to do other things," "Had no alternative," "Fears of loss of significant others," "Fears of returning to prison," "Concerns for health."

Another type of evidence is based on the changing history of the legal status of drugs and on changes in the widespread understanding of their health effects. During

the 20th century, there have been several nation-wide changes in the legal status of addictive drugs and in the understanding of their health risks. These events have many of the features of scientific experiments, but we do not have to wonder whether the results apply outside of the lab. In the late 19th and early 20th century, opiates and other addictive drugs were legal and could be purchased at pharmacies and mail-order companies (e.g., Sears, Roebuck and Company). The Harrison Act of 1914 outlawed the non-medical use of opiates and cocaine. The result, according to historians and early monographs from the 1920s (Courtwright, 2009; Kolb & DuMez, 1981), was an approximately 50% decrease in opiate addiction.

The Volstead Act, popularly known as Prohibition (1920), was followed by a marked increase in the price of alcohol and a concomitant decrease in alcoholism, as indexed by abrupt decreases in the rates of cirrhosis of the liver (Miron & Zweibel, 1991; Seeley, 1960). Since heavy drinking is a prerequisite for cirrhosis, the decrease in cirrhosis rates suggests that Prohibition must have brought about a decrease in alcoholism. These facts and their implications have been overshadowed by the unpopularity of Prohibition and the gradual return of widespread heavy drinking in the late 1920s.

For much of the early and mid-20th century, tobacco companies successfully undermined research that demonstrated a connection between smoking and cancer. However, in 1964 the Surgeon General published a well-documented, strongly worded rebuttal that convinced much of the public that smoking entailed severe health risks including an increase in the likelihood of cancer. The report was followed immediately by a striking, approximately linear decrease in the prevalence of smoking, despite the fact that most smokers who quit were pack-a-day addicted smokers (USDHHS, 1990, 1964; see Heyman, 2013 for graphs of these results).

The historical trends are exactly as expected if addicts retain the capacity to quit drugs. In contrast, new laws, increases in prices, and newly published scientific information do not slow down the growth of cancer cells,

restore the capacity to remember in Alzheimer's patients, or restore a receptor's affinity for insulin.

Data from interventions that focus explicitly on the determinants of choosing to continue or desist from drug use are also supportive of the view that addicts can be affected by incentives and reasons. Steve Higgins, a psychologist at the University of Vermont, developed an addiction treatment program based on the ideas that addicts would choose to stop using drugs if there was a concrete and relatively immediate reason to do so and that they would remain abstinent if they became involved in rewarding, new nondrug activities. The initial trials were with cocaine addicts. If a drug test was negative, the client earned a voucher for goods and services, such as educational programs, and recreational activities. Conversely, a positive urine test reduced the value of the voucher. One control group received counseling but no contingency, and a second control group got vouchers independent of whether they had been abstinent. All subjects met the then DSM criteria for cocaine addiction.

At every test date, the contingency group had higher abstinence scores (Higgins et al., 1994, 1995). A surprising feature of this success is that the vouchers were never worth more than $12.50. This amount is likely less than the client had been spending on cocaine. Yet, they chose the voucher instead. This finding persisted at follow-up and has been replicated. If cocaine addicts are stubbornly compulsive, then once the immediate reward for abstinence is gone, they should start using cocaine again. However, at every follow-up date, voucher subjects were more likely to have drug-free urines. Most interestingly, the percentage of drug-free samples increased from about 60% to almost 80% for the voucher group.

The subjects in the Vermont study were treatment seekers, who presumably wanted to change. Would contingencies work on drug addicts who did not volunteer to seek help? Physicians and airplane pilots who are on probation for drug use provide a handy test of this question. The physicians and pilots were compelled to enter treatment and forced to make themselves available for random testing. If they tested positive, they risked

permanently losing their license to practice and the income and status associated with such prestigious professions. Given how much was at stake for the lives of these subjects, they had very strong reasons to cease using and they did. The abstinence rates were typically above 80% and averaged close to 90% (Coombs, 1997, graphs in Heyman, 2009).

It is reasonable to suppose that such high abstinence rates reflect the individual characteristics correlated with the responsibilities and skills involved in medicine and piloting an airplane. However, a similar program with men and women on probation in the criminal justice system obtained similar results. Hawaii's Opportunity Probation with Enforcement (HOPE) program is an innovative approach to the problem of high rates of drug use among men and women in prison (Hawken & Kleiman, 2009). The program focuses on the post-release probation period, with the aim of breaking the vicious correlation between drug use and recidivism. The terms of parole include a contract to abstain from illegal drugs. To ensure that the probationers maintained their end of the bargain, they were subject to random drug tests. Positive tests resulted in a few days back in jail. The key finding was that drug use decreased by more than 80% within the first three months of the contingency plan and by more than 90% at six months (Hawken & Kleiman, 2009). A control group composed of probationers who were not under threat of immediate consequences for drug use continued to use drugs at the same rate as at the start of their probation.

The intervention results tell the same story as the epidemiological research, self-reports by addicts, and the historical record: addicts can choose to stop using drugs; they retain the capacity to quit. In contrast, the interventions that help addicts to quit drugs would not alleviate the symptoms of diseases that defenders of the disease model say addiction is similar to (e.g., McLellan et al., 2000; O'Brien & McLellan, 1996). No one could reasonably suppose that rewards persuade tumors to shrink. But rewards persuade addicts to reduce drug use.

Many other studies also confirm that addicts respond to incentives. Here are a few excellent further examples. A

classic demonstration of the power of incentives was the military's Operation Golden Flow (Gupta, 2015). In Vietnam, between ten and twenty-five percent of GI's were addicted to high-grade, freely-available heroin. In 1971, President Nixon commanded the military to begin drug testing. No soldier could board a plane home until he had passed a urine test. As word of the new directive spread, most GIs stopped using narcotics, and almost all soldiers who were detained passed the test on their second try. Once they were home, heroin apparently lost its appeal (Robins et al., 1974). Opiates may have helped them endure the war's alternating bouts of boredom and terror, but stateside, civilian life took precedence. Only five percent of the men who became addicted in Vietnam relapsed within ten months after return, and just twelve relapsed briefly within three years.

Consider the following fMRI experiment by researchers at Yale and Columbia. They found that the brains of smokers reporting a strong desire to smoke displayed enhanced activation of reward circuitry, as would be expected (Westbrook et al., 2011). But they also suggested that subjects could reduce craving by considering the long-term consequences of smoking, such as cancer or emphysema, while observing videos depicting people smoking. When subjects did so, their brains displayed enhanced activity in areas of the prefrontal cortex associated with focusing, shifting attention, and controlling emotions. Simultaneously, activity in regions associated with reward, such as the ventral striatum, decreased (Kober et al., 2010).

Investigators at NIDA observed the same pattern when they asked cocaine users to inhibit their craving in response to cues. Subjects underwent positron emission tomography (PET) scanning as they watched a video of people preparing drug paraphernalia and smoking crack cocaine. When researchers instructed the addicts to control their responses to the video, they observed inhibition of activity in brain regions normally associated with drug craving. When not deliberately suppressing their cravings, the addicts reported feeling their typical desire to use, and the PET scans revealed enhanced activation in brain regions that appear to be implicated in craving (Volkow et

al. 2010). We use the word, "appear," advisedly. Recall that for methodological reasons, most such studies indicate only association (correlation), and conclusive inferences about causation are unwarranted.

These powerful findings illuminate the capacity for self-control in addicts. They also underscore the idea that addicts persist not because of an inability to control the desire to use, but from a failure of motivation. Granted, summoning sustained motivation can be a great challenge. It takes a lot of energy and vigilance to resist persistent craving. Studies on the regulation of craving also help to distinguish behavior that people simply do not control from behavior that they cannot control. We are again referring to the elusive line between "did not" and "cannot." Imagine, by way of contrast with the behavior of addicts, promising a reward to people with Alzheimer's if they could keep their dementia from worsening. That would be both pointless and cruel because the kinds of brain changes intrinsic to dementia leave the sufferer largely resistant to rewards or penalties. In short, contingencies cannot produce recovery or full remission of these conditions as it can in individuals addicted to drugs.

Finally, we close this Part with reference to *Powell v. Texas*, 292 U.S. 514 (1968). It is emblematic of our core argument. Powell, was a chronic alcoholic who spent all his money on wine and who had been frequently arrested and convicted for public drunkenness. In the present case, Mr. Powell argued that he was afflicted with "the disease of chronic alcoholism,...his appearance in public [while drunk] was not of his own volition," (p. 517) it was "part of the pattern of his disease and is occasioned by a compulsion symptomatic of the disease," and thus to punish him for this behavior would be a violation of the Eighth Amendment prohibition of cruel and unusual punishment. This is an extremely sympathetic case for a compulsion excuse. The crime was not serious and the criminal behavior, public intoxication, was a typical manifestation of Powell's alcoholism (he had been arrested for public drunkenness over one hundred times).

The Supreme Court rejected Mr. Powell's claim for many reasons. Among them, Justice Marshall's plurality opinion was skeptical of the compulsion claim and

concluded that it went too far on the basis of too little knowledge. (As Part III. discusses further, we think the same is evidently true for the probationer's claim.) It pointed to the uncertainty about the meaning of the concept of "compulsion." (Likewise.) Finally, the opinion also suggested that it was unclear that providing a defense in such cases would improve the condition of alcoholics. (Likewise.)

Powell himself testified about his undisputed chronic alcoholism. He also testified that he could not stop drinking. Powell's cross-examination concerning the events of the day of his trial is worth quoting in full, as Justice Marshall did.

> Q: You took that one [drink] at eight o'clock [a.m.] because you wanted to drink?
> A: Yes, sir.
> Q: And you knew that if you drank it, you could keep on drinking and get drunk?
> A: Well, I was supposed to be here on trial, and I didn't take but that one drink.
> Q: You knew you had to be here this afternoon, but this morning you took one drink and then you knew that you couldn't afford to drink anymore and come to court; is that right?
> A: Yes, sir, that's right.
> Q: Because you knew what you would do if you kept drinking, that you would finally pass out or be picked up?
> A: Yes, sir.
> Q: And you didn't want that to happen to you today?
> A: No, sir.
> Q: Not today?
> A: No, sir.
> Q: So you only had one drink today?
> A: Yes, sir (pp. 519-520).

On redirect examination, Powell's attorney elicited further explanation.

> Q: Leroy, isn't the real reason why you just had one drink today because you just had enough money to buy one drink?
> A: Well, that was just give to me.
> Q: In other words, you didn't have any money with which you could buy drinks yourself?

A: No, sir, that was give to me.
Q: And that's really what controlled the amount you drank this morning, isn't it?
A: Yes, sir.
Q: Leroy, when you start drinking, do you have any control over how many drinks you can take?
A: No, sir (p. 520).

Powell wanted to drink and had that first drink, but despite that last answer, his compulsion did not cause him to engage in the myriad lawful and unlawful means he might easily have used to obtain more alcohol if his craving was desperately compulsive. Although Powell was a core case of an addict, he could refrain from using if he had a good enough reason to do so.

In sum, although drugs change the brain and addiction has a biological basis, research shows that drug use in addicts remains voluntary; like other choices it is subject to economic, social, and legal sanctions, such as those imposed by the courts.

III. THIS CASE HAS PROFOUND CRIMINAL JUSTICE IMPLICATIONS AND SHOULD NOT BE DECIDED ON THE BASIS OF CONTESTED CONCEPTS AND SCIENCE.

This Section addresses, first, some of the potentially profound legal and social policy implications of granting the probationer's claim. It then turns to why caution mandates that this Court should not accept the claim

A. The Criminal Justice Implications of Granting the Claim.

The basis of the probationer's claim is that she cannot fairly be expected to refrain from using drugs as a condition of probation because she cannot control her drug use and therefore is not responsible for it. If the basis for this non-responsibility claim were firmly established, it would state a strong moral and legal claim. It is far from firmly established, however, as we believe we have shown in the previous sections. Nonetheless, we consider the

implications of the view that the addict is not responsible for her use of drugs.

Let us begin with the effect on probation and parole. Staying drug-free is a universal condition of criminal justice supervision. Not only is possession of controlled substances a crime in itself in all jurisdictions (*see, e.g.,* M.G.L. Part I, Title 15, Chapter 94c, §34), it is well-known that for many reasons, including feeding their habit, addicts often commit other crimes related to the addiction. If addicts cannot be sanctioned for violating this condition of probation and parole, the state will lose this powerful contingency management technique for assisting addicts to remain free of drugs and for protecting society. The threat of being incarcerated or re-incarcerated or sanctioned in some way gives the addict an extremely powerful incentive to stay clean. It will not always be successful, but as the Hawaii program described in Part II indicates, it decreases the rate of violations markedly, an outcome the elasticity of demand for addictive substances predicts. NIDA's funding of contingency management indicates that there is consensual understanding that such tools are profoundly positive intervention to reduce persistent seeking and using of substances. The existence of a specialty Adult Drug Court in Massachusetts also testifies to belief that imposing the condition of staying clean is efficacious.

What would be the effect of losing this tool on sentencing judges and parole authorities? The inability to impose sanctions will almost certainly increase recidivism substantially. Many judges and parole authorities who are conscious of their duty to protect society would hesitate before granting probation or parole that might otherwise give people a chance to live a productive life in freedom. In an age in which our society is criticized for too much incarceration, this would be an unfortunate outcome. Paradoxically, if judges no longer granted probation or parole and incarceration took its place, this might serve as a deterrent to possession because the "cost" of this crime would increase, but we doubt it. In any case, arguing against probation or parole without the condition of staying drug-free (and, indirectly for incarceration as a deterrent) would be an odd position for supporters of the probationer

to take because their argument in her favor currently rests on the claim, in effect, that addicts cannot be deterred.

Diversion programs would be imperiled, if not crippled. Various types of diversion programs for non-violent crimes, including specialty drug or mental health courts, depend for their success on the contingency management tool of making staying clean a condition of successfully completing the program with all the benefits that accrue. If the probationer's petition is granted, it entails that virtually no diverted addict could succeed. The rationale for these worthy programs would evaporate.

Can the effect of holding that probationers are not responsible for violating the condition of drug abstinence be limited to the context of probation and parole? There is no principled argument for so cabining the holding. If a jurisdiction deems addicts not responsible for possession—which is a proxy for use—then the state will lose its power to use the criminal sanction as one powerful regulatory tool in its armamentarium. If an addict cannot control herself and is "compelled" to possess in order to use, how can it be fair to blame and punish her? Many jurisdictions legislatively preclude using addiction as the basis of an insanity defense. As *Powell* and *Clark v. Arizona,* 548 U.S. 735 (2006) respectively teach, no jurisdiction is constitutionally compelled to include addiction as a basis for legal insanity or to adopt a "control" test for legal insanity. Massachusetts has such a test. Granting this petition, which entails adopting the contested disease model of addiction and the corollary that addicts are incapable of conforming to the law, will ensure that addiction will now be used to support the claim that an addicted defendant charged with illegal possession should be acquitted by reason of insanity. Such claims and the adjudication of whether the defendant is truly an addict will multiply.

The result will not be positive for addicts. As this Court wrote in *Commonwealth v. Bruneau*, 472. Mass. 510 (2015),

> "… a defendant found not guilty by reason of mental illness faces harsh consequences because the defendant is eligible for civil commitment under

> strict security, where he would be confined for an indefinite period of time. See G. L. c. 123, § 16."(at pp. 517-18)

Under such a threat, addicts will probably not be willing to raise the defense of legal insanity because the consequences of conviction will be much less harsh than a successful insanity acquittal. After all, potentially life-long commitment, a practice the Supreme Court approved in *Jones v. United States,* 463 US 354 (1963), a case involving a defendant acquitted by reason of insanity for shoplifting, will deter raising legal insanity. In the case of acquittals, the state will inevitably exercise its power for the purpose of preventive detention. This will simply confirm Justice Marshall's prediction in *Powell* that the effect of imposing a "compulsion" defense would result in incarceration. The only thing that would change is the name of the institution from jail to hospital. If the probationer's petition is granted, the insanity defense might be well-justified by the same reasoning, but defendants will be unwilling to use it.

It is by no means clear that a complete defense to crime for addicts could be limited to the offense of illegal possession. The most extensive discussion of this issue in case law is *United States v. Moore*, 486 F. 2d 1139 (D.C. Cir. 1973). Moore was an addict convicted of possession who claimed on the basis of uncontested expert evidence at trial that addiction was a disease and that he could not control his compulsion to possess and to use. The trial judge refused to grant Moore an instruction providing a defense to possession on that basis. The D.C. Circuit affirmed the conviction. There were numerous detailed concurrences in this result, but it was so held essentially for the following reasons: 1) there was controversy over whether addiction is a disease and whether we are able to know an addict's genuine capacity to refrain from using; 2) the defense would apply to any defendant with impaired behavioral controls, even in the absence of an allegedly objective cause such as a disease; 3) it would apply not only to possession, but also to any other crimes committed to support the addiction; and, 4) adopting such a defense would undermine the strong public policy supporting the prohibition of sale and possession of controlled substances.

There were two very strong dissents. The first, by Judge Skelly Wright, argued that the common law should embrace a new principle according to which a drug addict who lacks substantial capacity to conform his conduct to the requirements of the law as a result of drug use should not be held criminally responsible for mere possession for his own use. The opinion rejected the claim that deterrence would be undermined as too speculative. This dissent recognized that the compulsion claim might be difficult to limit to mere possession, but evaded the problem by arguing that Congress clearly intended that such a defense should not go this far. (The majority thought this argument was scant consolation.) In a second, partial dissent, the chief judge of the circuit, David Bazelon, argued that the principle behind adopting the defense applied to crimes other than mere possession and that juries should also hear evidence about compulsion arising from addiction when other crimes were charged, including armed robbery or trafficking.

The arguments in *Moore* are strikingly similar to those in this case. And recall that the claim in *Powell* involved public drunkenness, a claim distinct from possession, because that criminal behavior was allegedly part of the pattern of and a compulsion symptomatic of the disease of alcoholism (we recognize that possessing alcohol is not a crime, but the claim is analogous).

It might be argued that this case should adopt the disease and non-responsibility claims because these have now become firmly established. Sections I. and II. of this brief deny this, but granting the petition will cast the doctrines of criminal responsibility of addicts for many crimes into dangerous, uncharted waters. Moreover, the thinking behind these claims, which often rests on what we have termed "unrefined biological determinism," will support claims that the very concept of responsibility that is foundational for criminal law and our society is unjustified. We are mindful of the dangers of catastrophic thinking, but we fear that granting the probationer's petition may have the effect of starting to pry open the lid of Pandora's Box.

Another implication of granting the probationer's petition concerns civil commitment specifically aimed at addicts who are dangerous to themselves or others. Massachusetts has such a law (M.G.L., Chapter 132, Section 35). Although not criminal, such laws serve at least one goal in common with criminal law: incapacitation of potentially dangerous people. The primary purpose of such laws is to provide treatment, not to punish the people committed. If convicted defendants cannot be required to remain clean, these laws might be used in place of probation. Nevertheless, these laws do not provide the same level of due process protection as criminal law; it is not clear that the benefits intended occur; and they can have quite disquieting unintended consequences, such as housing in jails, rather than in treatment facilities, the people who have been committed (Depew et al, 2014). Granting the petition in this case and thus announcing that addicts who are potentially dangerous cannot be deterred by sanctions may well increase the use of this unfortunate approach to addiction, especially because the definition of dangerousness is so vague. In *Jones*, for example, the Supreme Court suggested that shoplifting a jacket was a sufficiently dangerous crime to justify potentially life-long involuntary civil commitment after acquittal by reason of insanity (at p. 365, n. 14). Justice Marshall's prediction of turning hospitals into jails would once more be confirmed. Granting the petition will not be of benefit to addicts in the long run.

B. The Need for Caution

We urge this Court to be cautious. As Justice Marshall wrote about the similar claim being made in *Powell*, "The difficulty with that position [that Powell should be excused because his crime was allegedly part of the pattern of and a compulsion symptomatic of the disease of alcoholism], is that it goes much too far on the basis of too little knowledge." (at p. 521). As Parts I. and II. have demonstrated, this is still true despite claims to the contrary.

It may be true that the majority of addiction specialists – but not necessarily premier scholars in psychiatry, psychology, or allied fields - adhere to the monolithic brain

disease model and believe that addicts cannot control the action of using drugs. Nonetheless, psychiatry and psychology are imperfect, constantly evolving fields. They do not control what the law may properly do. Their classifications and concepts have purposes that differ from the law's. The United States Supreme Court said this in *Powell* (at p. 526) and has repeatedly re-affirmed since, *e.g., Jones* at 365, n.13; *Kansas v. Crane,* 534 U.S. 407, 414 (2002); *Clark* at 752-53, 774-75. The American Psychiatric Association concurs. The "Cautionary Statement for Forensic Use" in DSM-5 states the following.

> Although the DSM-5 diagnostic criteria and text are primarily designed to assist clinicians in conducting clinical assessment, case formulation, and treatment planning, DSM-5 is also used as a reference for the courts and attorneys in assessing the forensic consequences of mental disorders. As a result, it is important to note that the definition of mental disorder included in DSM-5 was developed to meet the needs of clinicians, public health professionals, and research investigators rather than all of the technical needs of the courts and legal professionals (at p.25).

In *Powell,* Justice Marshall recognized that there was serious difficulty understanding the meaning of compulsion (at p. 526) As the contrasting majority and dissenting opinions in *Crane* disclose, there is continuing debate about the meaning of "serious difficulty" controlling one's behavior and similar terms, such as compulsion or loss of control, and about whether control capacity can be reliably assessed. Justice Breyer's majority opinion wrote that,

> …we recognize that in cases where lack of control is at issue, "inability to control behavior" will not be demonstrable with mathematical precision. It is enough to say that there must be proof of serious difficulty in controlling behavior (at p. 413).

In a rather colorful passage, Justice Scalia's dissent had the following to say on this question.

> This formulation of the new requirement [serious difficulty controlling behavior] certainly displays an elegant subtlety of mind….How *is* one to frame for a jury the degree of "inability to control…? Will it be a percentage ("Ladies and gentlemen of the jury, you may commit Mr. Crane under the SVPA [referring to Kansas' law governing the commitment of so-called mentally abnormal sexually violent predators] only if you find, beyond a reasonable doubt, that he is 42% unable to control his penchant for sexual violence")? Or a frequency ratio ("Ladies and gentlemen of the jury, you may commit …only if you find, beyond a reasonable doubt, that he is unable to control his penchant for sexual violence 3 times out of 10")? Or merely an adverb ("Ladies and gentlemen of the jury, you may commit Mr. Crane under the SVPA only if you find, beyond a reasonable doubt, that he is appreciably—or moderately, or substantially, or almost totally—unable to control his penchant for sexual violence")? None of these seems to me satisfactory (at pp.423-24).

DSM-5's "Cautionary Statement" is also instructive about control problems.

> Nonclinical decision makers should also be cautioned that a diagnosis does not carry any necessary implications regarding the etiology or causes of the individual's mental disorder or the individual's degree of control over behaviors that may be associated with the disorder. Even when diminished control over one's behavior is a feature of the disorder, having the diagnosis in itself does not demonstrate that a particular individual is (or was) unable to control his or her behavior at a particular time (at p. 25)

The preceding sections of this brief indicate that debates about the conceptualization and measurement of "loss of control" continue.

> As the majority concluded in *Clark*,

> Though we certainly do not "condemn mental-disease evidence wholesale" [citation omitted], the consequence of this professional ferment [concerning psychiatric classification and its implications] is a general caution in treating psychological classifications as predicates for excusing otherwise criminal conduct (at p. 775).

The history of psychiatry and psychology is littered with discredited paradigms and beliefs that were once considered orthodoxy or were "generally accepted." We urge this Court to be cautious and not to grant this petition because doing so will implicitly accept highly contested and evolving concepts and science as a basis for a legal policy that could have the profoundly negative consequences for addicts, the criminal justice system and for society discussed just above.

IV. CONCLUSION

This case raises important questions about principles of behavior, criminal responsibility, and the sound and fair administration of criminal justice. The probationer claims that she should not be held accountable for her failure to "remain drug free" as a condition of her probation because she suffers from addiction, or substance use disorder [SUD], wherein her continued use of substances despite negative consequences is a sign of that disorder. Her claim is flawed in a number of ways. As a straightforward matter of definition, we note that nowhere in the Diagnostic and Statistical Manual of the American Psychiatric Association, the most widely used taxonomy of psychiatric disorders, is drug use in the context of SUD regarded as a behavior completely beyond the control of the addicted user. More substantively, the core of her argument, which depends largely on the implications of the brain-disease model of addiction -- namely, that the brain changes associated with addiction render the addict incapable of behavioral control – is demonstrably untrue. The mere association of drug taking with expected neurobiological changes in the brain is not evidence that drug use is beyond control. This is abundantly evident from the large volume of data demonstrating that addiction is a set of behaviors whose course can be altered by foreseeable consequences.

The same cannot be said of conventional brain diseases such as Alzheimer's or multiple sclerosis.

In sum, the best scientific and clinical data are strongly at odds with the view that addicts are unable to choose not to use substances. We believe that a decision in favor of the probationer could have significant, even devastating, implications for the future of treatment-based approaches to criminal justice as well as for criminal responsibility more generally. We conclude that the probationer's claim should be denied because it is based on erroneous, refuted scientific premises and will have negative consequences if it is accepted.

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