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If Addiction is not Best Conceptualized a Brain Disease, then What Kind of Disease is it?

Sally L. Satel · Scott O. Lilienfeld

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Abstract A modest opposition to the brain disease concept of addiction has been mounting for at least the last decade. Despite the good intentions behind the brain disease rhetoric – to secure more biomedical funding for addiction, to combat "stigma," and to soften criminal approaches – the very concept of addiction as a brain disease is deeply conceptually confused. We question whether Lewis goes far enough in his challenge, robust as it is, of the brain disease concept. For one thing, the notion that addiction is a disease (especially within the behavioral realm) is challenging to refute or confirm because the disease concept itself is poorly defined in medical and psychological nosology. More important, quibbling over what *kind* of disease addiction is unlikely to be productive. The rational response to adjudicating whether addiction is a brain disease is not to engage in potentially fruitless debates over the question of disease classification but rather to view addiction as an enormously complex set of behaviors that operate on several dimensions, ranging from molecular function and structure and brain physiology to psychology, the psychosocial environment, and social and cultural relations.

Keywords Addiction · Brain disease · Reductionism · Levels of analysis · National Institute on Drug Abuse · Neurocentrism · Philosophy of medicine · Over-medicalization

S. L. Satel
American Enterprise Institute, Washington DC, USA

S. L. Satel (✉)
Yale University School of Medicine, New Haven, CT, USA
e-mail: slsatel@gmail.com

S. O. Lilienfeld
Samuel Candler Dobbs Professor of Psychology, Emory University, Atlanta, GA, USA
e-mail: slilien@emory.edu

What is a Disease?

In his thought-provoking essay, Lewis makes the case that addiction is not a brain disease because it can be overcome as a result of willpower, changing perspectives, changing environments, mindfulness, or emotional growth. Although we are sympathetic to many of Lewis's arguments, we part ways with him in several respects.

Lewis, like most participants on both sides of this ongoing debate, appears to assume that the question of whether a given condition, in this case the condition of addiction, is or is not a "disease" is scientifically answerable. This assumption causes Lewis (and his champions and critics alike) to neglect a basic point: the very question of what constitutes a disease entity in psychiatry, and in other domains of medicine, is largely or entirely unresolved. Philosophers of medicine have long debated, and continue to debate, the criteria used to adjudicate whether a given condition is a disease. They have advanced various criteria, including statistical rarity, degree of social impairment, patient's or doctor's perceived need for treatment, biological dysfunction,

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and decreased evolutionary fitness. Yet all of these criteria have their serious shortcomings, and none appears to be sufficient to account for all conventional diseases [1, 2]. Others in the field, such as Donald Klein and Jerome Wakefield, have argued that disease is instead a hybrid concept involving a conjunction of evolutionary dysfunction (a breakdown in a naturally selected system) and harm (distress or impairment in everyday life), but this view, too, has its critics [3–5]. Still other authors have maintained that "disease," much like many other concepts in nature (e.g., mountain, continent, planet), is inherently a fuzzy, family-resemblance concept that does not lend itself to a strict definition [6, 7]. From this lattermost perspective, the question of whether a given condition (e.g., addiction) is truly a "disease" is as scientifically unresolvable as the question of whether a given large object orbiting the solar system (e.g., Pluto, Ceres) is truly a "planet" [8]. Although we do not intend to resolve this knotty conceptual controversy here, suffice it to say that before one can decide whether a given set of behaviors constitutes a brain disease, one must first decide whether it constitutes a disease. At present, it is not at all clear that this issue can be resolved strictly by means of scientific data.

If not a Disease, then What?

The rational response to adjudicating whether addiction is a brain disease, we believe, is not to engage in potentially fruitless debates over the question of disease classification but rather to view addiction as a complex set of behaviors that operates on several dimensions, ranging from molecular function and structure and brain physiology to psychology, the psychosocial environment, and social relations [6]. Given the myriad conceptual ambiguities associated with the disease concept, we are concerned here with the more pressing and arguably more important question of whether addiction is best construed as a *brain* disease.

Addiction is typically associated with brain changes, to be sure, but in contrast to conventional brain pathologies, such as Alzheimer's disease, those alterations rarely if ever preclude individuals' capacity to alter their behavior based on foreseeable consequences. The term "brain disease," which in most cases implies a lack of control over behavior, obscures that crucial distinction. Moreover, although severe addictions are partly rooted in genetic predispositions that are themselves manifested in brain functioning, these conditions can be profitably understood at multiple levels of analysis (e.g., psychological, social, cultural) in addition to the neural level.

These dimensions are best organized not as a simplistic reductionist scheme arrayed hierarchically from least complex to most, but rather as a side-by-side arrangement in which each level is complex in its own way. We imply no sense of a pinnacle, or ultimate frame of analysis; the point is that each dimension plays a distinctive explanatory role in addiction, with some being more clinically or scientifically relevant than others depending upon the question posed and the context in play. We start with the neurobiological level, which comprises the central nervous system, and most prominently the brain and its constituent cells. Genes help to direct neuronal development; neurons assemble themselves into brain circuits. Information processing, or computation, and neural network dynamics are next. The subsequent dimension consists of conscious mental states, such as thoughts, feelings, motives, perceptions, knowledge, and intentions. Social and cultural spheres, which play powerful roles in shaping our thoughts, feelings, and behavior, occupy the next phases.

Problems arise when we ascribe too much importance to only one dimension, as in the case of the brain disease model and brain-based explanations, and not enough to psychological or social ones. Psychiatrist Kenneth Kendler warned of the hazards of "fervent monism," that is, the tendency to value one approach to human nature while underemphasizing or neglecting others [9]. The heart of what we term "neurocentrism" is a particular brand of fervent monism, one that prioritizes brain-based explanations for human behavior above all others [10–12]. Just as one obtains differing perspectives on the layout of a sprawling city while ascending in a skyscraper's glass elevator, we can acquire different insights into human behavior at different dimensions of analysis [13].

Consider a da Vinci painting It is an assemblage of subatomic particles, but it is not merely an assemblage of such particles. Conceptualizing it exclusively at only the subatomic level misses critical aspects of analysis, especially the level of the meaning afforded by the nuanced higher-order interactions among the features of the painting [14]. Philosopher Daniel Dennett referred to this brand of eliminative reductionism as "greedy reductionism," because it advocates replacing one level of analysis with others rather

than attempting to integrate them by constructing bridging laws across them [15, 16]. Psychologist Steven Pinker has aptly argued for an alternative brand of reductionism: "Good reductionism (also called hierarchical reductionism) consists not of replacing one field of knowledge with another but of connecting or unifying them" [17]. Such an ecumenical approach to addiction would, in our view, would be far more fruitful than the effort to conceptualize this enormously complex behavioral condition by means of a single and inevitably myopic lens.

The key to this approach is recognizing that some explanations are more informative for certain purposes than others. This principle is profoundly important in therapeutic intervention. A scientist trying to develop a medication for Parkinson's disease will work within the biological realm of the explanatory spectrum, perhaps developing compounds aimed at boosting the activity of dopamine in affected brain regions. In contrast, a psychotherapist helping a distraught spouse who has recently undergone a divorce must focus on the psychological realm of analysis. Efforts by this therapist to understand the patient's psychological pain by subjecting his or her brain to an fMRI could be worse than useless because doing so would draw attention away from his or her thoughts, feelings, and assumptions about the world — the domains in which intervention would be most helpful.

Overemphasizing the neural dimension of analysis when conceptualizing addiction impedes our progress in treating and preventing it. This is because it distracts us from considering users' motives, their unappreciated, if constrained, decision-making capacities, and their well-documented abilities to respond to incentives, central psychological factors in the theatre of addiction. Ironically, Lewis' emphasis on the neurobiological dimensions of addiction in his essay and in his 2011 book, *Memoirs of an Addicted Brain* inadvertently reinforces the notion that addiction is a brain disease. Although he focuses on the "learning" aspects of addictive behavior, an undeniably useful perspective on mechanism, the story he tells is rendered almost exclusively in the language of biology – the lingua franca of physical disease [18]. As a consequence, we are doubtful that Lewis' otherwise careful analysis will sway champions of the brain disease model of addiction because it does not explicitly address the value of levels of explanation beyond the biological. To be fair, Lewis does present textured patient vignettes in depth in his 2015 book, *The Biology of Desire*, and he mines his own experience in his 2013 book, *Memoirs of an Addicted Brain*. Indeed, he attributes his own recovery to psychological transformation.

To be sure, addiction can be explained in part according to how it operates neurobiologically. Indeed, scientists studying the role of dopamine transmission in reinforcement will, naturally, address themselves to to neurophysiology. In this respect, arguably, addiction is *in part* a pathological condition of the brain. At the same time, however, it is every bit as much a personality disease, a motivational disease, a social disease, a cultural disease, and so on. Why privilege one aspect of analysis above all of the others? On every one of those dimensions we can find elements that contribute to excessive and repeated drug use. Understanding those elements can lead to strategies that help individuals bring their behavior under control.

The NIDA Misconception

Some background about the nature of the debate surrounding the "brain disease" formulation is in order. By the mid-1990s, the longstanding truism "once an addict, always an addict" was reinvigorated and repackaged with a neurocentric twist: "Addiction is a chronic and relapsing brain disease" [19]. This view was promoted tirelessly by psychologist Alan I. Leshner, then the director of the National Institute on Drug Abuse (NIDA), the nation's premier addiction research body and part of the National Institutes of Health. It is now the dominant view of addiction in the scientific field [20]. The brain disease model is a staple of medical school education and drug counselor training and even appears in antidrug lectures given to high-school students [21, 22]. Rehab patients similarly learn that they have a chronic brain disease. 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" [23]. Drug czars under Presidents Bill Clinton, George W. Bush, and Barack Obama have all endorsed the brain disease framework at one time or another [24–26]. From being featured in a major documentary on HBO, on talk shows and *Law and Order*, and on the covers of *Time* and *Newsweek*, the brain disease model has become dogma in most

quarters. Like all articles of faith, it is typically accepted without question, as Lewis observes [24, 27–29].

A recent article in the *New England Journal of Medicine* confirms that the brain disease formulation is the government-approved narrative for addiction. Three esteemed researchers, including NIDA head Dr. Nora Volkow and National Institute on Alcohol Abuse and Alcoholism chief Dr. George Koob, both of whom work under the auspices of the National Institutes of Health, wrote the article, entitled "Neurobiologic advances from the brain disease model of addiction." They laid out the neural pathways involved in various stages of drug abuse, such as anticipation and craving, intoxication, and withdrawal. It is a fine summary of the latest brain-based findings.

They begin: *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* [30].

As two vocal critics of the brain disease model, we can attest to the fact that their interpretation is incorrect. The brain disease model indeed "continues to be questioned" by us and others, but not because the linkages between addiction and biological processes have not always been clearly tied to neurobiology. Those linkages, regardless of how detailed they are, demonstrate only that the brain is somehow involved with drug addiction. This assertion is merely a scientific truism given that virtually all contemporary neuroscientists agree that the mind is ultimately what the brain and rest of the central nervous system do. The linkages between brain and behavior do not, per se, make the case that addiction is best conceptualized as a brain-based phenomenon. Nor does the trivial fact that a da Vinci painting is composed of subatomic particles implies that it is best conceptualized as a subatomic phenomenon that is most profitably analyzed by chemists and physicists. Indeed, we fully expect more details about the biology of addiction to be uncovered in the near future; as important as they may be, such discoveries will not make addiction any more a brain disease than it is – or is not – at present.

Similarly, to contend that knowing about the brain in addiction makes addiction a brain disease makes no more sense than concluding that because we now know more about the role of personality traits, such as neuroticism, in increasing risk for anxiety disorders, we can, at last, recognize that anxiety disorders are purely disorders of personality. The brain disease model captures only one important part of a remarkably multilayered causal story.

To be sure, addiction is associated with and contributes to brain changes. Individuals with such brain alterations are sometimes less likely to make certain decisions and find it more difficult to act on certain choices, but those changes do not come close to eradicating the capacity to choose. Volumes of research show that most people who are addicted respond to incentives and consequences, such as small cash payments, the opportunity to participate in work programs, or threats of an overnight jail stay [31]. The implications for contingencies, choice, and motivation in recovery are well established [32, 33]. The data on the power of rewards and sanctions in reducing drug use are potent evidence that addiction is a set of behaviors that many, if not most, users can control when consequences are foreseeable. In contrast to many neurological illnesses, addiction thus has an important voluntary dimension. In his writing, Lewis makes the point that users indeed make choices, and that some of these are self-destructive whereas others are life-saving. He emphasizes that these positive changes, like addiction itself, also represent a form of learning. Imagine, by way of contrast, promising a reward to people with amytrophic lateral sclerosis (Lou Gehrig's disease) if they could prevent their neurological symptoms from worsening. That would be both pointless and cruel because the kinds of brain changes intrinsic to this illness leave sufferers resistant to rewards or penalties. The same would hold for dementias, such as Alzheimer's. Hence, the oft-cited analogy between addiction and most other neurological diseases does not hold.

One of the major shortcomings of the neurocentric view of addiction is that it ignores the fact that people use drugs and sustain their addiction because substances temporarily quell their pain: persistent self-loathing, anxiety, alienation, deep-seated intolerance of stress or boredom, and pervasive loneliness. The brain disease model is of little use here because it does not accommodate the emotional logic that triggers and sustains addiction.

The brain disease model can also lead us to overvalue medication. As commonly conceptualized, this model implies that addicts cannot stop using drugs until their brain chemistry returns to normal, and it

overemphasizes the value of brain-level solutions, such as pharmaceutical interventions. In 1997, Leshner ranked the search for a medication to treat methamphetamine addiction as a "top priority" [33]. A decade later, Volkow forecasted that "We will be treating addiction as a disease [by 2018], and that means with medicine" [21, 29]. The search for a magic bullet is folly— and even NIDA concedes that it has finally given up hope of finding a wonder drug— but the brain-disease narrative continues to inspire unrealistic goals and guide funding priorities.

Methadone and buprenorphine, both opiate substitutes, suppress withdrawal and craving and are thus stabilizing for many patients. But rarely are medications sufficient to change longstanding habits of behavior – responding to psychic distress with other self-defeating behavior, yielding to cue-induced craving – without professional counseling or a self-imposed schedule of self-binding. Self-binding refers to devising ways to shield oneself from predictable provocation, to cope with stress, and to maintain the motivation to do so.

Closing and Policy Implications

Lewis rightly views addiction as a learned behavior that can also be unlearned. This vision clashes with the standard NIDA-approved brain disease model that focuses on "tissue damage," as Lewis puts it, and that denies the capacity for choice making. We find Lewis' insights to be valuable. At the same time, we believe that a more explicit consideration of the alternative dimensions of analysis would enrich his conceptualization of addiction.

In his policy writings, Lewis recommends laissez-faire policies for addicts (e.g., sanctioned drug distribution; decriminalization of minor drug-related crime). His prescriptions reflect one way of seeing the addict: namely, as a person who can make better choices in a different environment and who does not require external control or benign paternalism. We draw somewhat different policy conclusions, however. We urge accountability and applaud strategies that divert non-violent substance abusers into contingency management programs with the goal of achieving abstinence or responsible use of opiate replacement medications. This course of recovery will involve shifts in personal identity, social networks, and values – dimensions that Lewis, too, rightly regards as important.

The recent opiate epidemic in America has afforded an opportunity to replay longstanding debates surrounding conceptualizations of drug addiction and the addict. The major emphasis among public health officials and addiction experts has been on opiate replacement because, as the standard narrative goes, addiction is a "brain disease." We agree that medications can be useful and in some cases even necessary – one of us works in a methadone clinic – but they must not be emphasized at the expense of the other dimensions of recovery that take place on the psychological, social, and environmental levels. A neurocentric view of addiction, which Lewis' analysis paradoxically reinforces despite his overt rejection of the simplistic brain disease model, can lead us into such a trap. Once the nature of choice in addiction is better appreciated, the value of employing contingency management and of modifying expectations of user behavior becomes apparent.

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