

Received Wisdom Regarding the Roles of Craving and Dopamine in Addiction: A Response to Lewis's Critique of *Addiction: A Disorder of Choice*

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Abstract

Lewis's review of my book (2011, this issue) repeats widely shared understandings of the nature of addiction and the role that dopamine plays in the persistence of self-destructive drug use. These accounts depict addiction as a chronic relapsing disease and claim that drug-induced changes in dopamine function explain the transition from drug experimentation to compulsive drug use. In my book, I test the idea that addiction is a chronic, persistent state. Lewis fails to mention the results of the various tests, although they provide a handy test for his account of addiction and are surprising in light of the common verbal formula "addiction is a chronic, relapsing disease." Consequently, I review a few of the key findings in this response. Lewis faults me for not giving enough attention to dopamine. In my book, I conclude that there is more to the biology of addiction than dopamine, and in this response, I describe research that tests the idea that drug-induced increases in dopamine markedly reduce an individual's capacity to choose nondrug reinforcers. In one experiment, rats readily gave up cocaine for saccharin, even when they had been consuming massive amounts of the drug for weeks. Put more generally, well-established research results call for a revision of currently accepted understandings of addiction and the role that dopamine plays in drug use.

Keywords

addiction, remission rates, craving, dopamine, treatment seeking, drug dependence, recovery

In a scholarly and wide-ranging book on addiction, Edwin Brecher (1972) concluded that heroin was an "enslaving drug." On the basis of follow-up studies of clinic populations, he showed that heroin users became lifelong heroin victims. The reason, he explained, was that this was how heroin worked: It was enslaving. At virtually the same time Brecher was publishing his results, Robins, Helzer, and Davis (1975) were reporting that close to 90% of the young men who became addicted to opiates in Vietnam stopped using or became controlled opiate users upon their return to the United States. This was true for those who injected heroin in Vietnam, and it was true for those who used heroin recreationally from time to time when home. Which story better captures the nature of addiction? On the basis of Lewis's article (2011, this issue) one could not say. We learn about craving and dopamine, but not the basic facts regarding the natural history of drug use in addicts. What my book points out is that we now know whether Brecher's results or Robins' results are more representative. Data collected over the past 20 years and published in leading

journals provide a clear answer. Yet the findings (and the answer that they point to) have had little influence on the image of addiction found in clinical texts or the science pages of our leading newspapers and magazines. Addiction continues to be referred to as a chronic, relapsing disorder that requires lifetime care. Nevertheless, well-established research reveals that it is the psychiatric disorder with the highest remission rates.

One reason for the silence is that many of the key findings are buried in tables and appendices without pointers that might help the reader come to a sensible interpretation. For example, the numerators and denominators of the remission rates reside side by side in columns, not on top of one another. No one had bothered to calculate their quotients. Consequently, there was,

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I thought, an important story to tell. My book attempts to do this. It describes the typical trajectory of drug use in addicts who were scientifically selected for study, as well as the pattern of drug use in clinic addicts. The results are surprising; the factors that influence everyday decisions regarding nondrug commodities are also the factors that influence drug use in addicts. The data reveal a pattern of voluntary self-destructive behavior. This fits neither current medical understandings of addiction nor conventional economic understandings of consumer choice. Yet, it is what addicts do.

Lewis frames our disagreements as a "cold war" (p. 150), with the disease model and biology on one side and choice-based accounts of addiction and behavior on the other side, adding that I "reject biological explanations of addiction and replace them with principles drawn from behavioral economics and learning theory" (p. 150). I do not reject biological accounts. What I reject are accounts of addiction based on slogans ("it's a brain disease"), unrepresentative clinic populations, and simplistic understandings of behavior that assume that a biological influence implies an involuntary outcome. I sketch out a natural history of drug use informed by studies that selected subjects scientifically; that included subjects independent of their clinical history; and that experimentally tested whether drug use in addicts varies as a function of the factors that influence choice, such as economics, legal sanctions, and the opinions of others. I focused on this literature because descriptions of how addicts behave answer such critical questions as whether drug-induced neural adaptations turn voluntary drug use into involuntary drug seeking or whether a genetic predisposition for alcoholism renders excessive drinking compulsory. In contrast, many in the field of addiction have assumed that neural adaptation and genetic predispositions are *prima facie* evidence of involuntary drug use. Put another way, there are reasons to doubt received wisdom regarding the compulsive nature of drug use in addicts, and thus we need to test whether heroin and other drugs really are enslaving. The results of these tests provide criteria for evaluating theories of addiction, including Lewis's version that emphasizes craving and dopamine. Since Lewis omits virtually all of the key findings that I summarize, I will briefly outline some of what I discovered.

Since 1991, there have been four national surveys of psychiatric health in the United States. The subjects were selected so that their demographic characteristics approximated the demographic characteristics of the adult U.S. population (e.g., Anthony & Helzer, 1991; Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Robins & Regier, 1991; Stinson et al., 2005; Warner, Kessler, Hughes, Anthony, & Nelson, 1995). In each study, addiction was the psychiatric disorder with the highest remission rate. In the three studies that differentiated between addiction and abuse (which is considered less serious), the remission rates varied from 76% to 83%. Put more concretely, most of those who met lifetime criteria for addiction had not used drugs at clinically significant levels for at least a year. Further analysis indicates that these results are not an artifact of the epidemiological methodology. For example, the few prospective studies that are on hand led to virtually

identical estimates of the remission rates. On the basis of age data, most addicts appear to have quit using drugs at diagnostically significant levels by about age 30. It is important to note that a large majority of addicts did not seek treatment. This is relevant because addicts in clinics typically remit at much lower rates than do addicts who are not in treatment. The reasons for this difference are not well understood. The available data do not reveal systematic differences in pharmacological history. Indeed, there is some evidence that addicts who are not in treatment use drugs more heavily, yet quit more readily. There is, though, a difference in medical histories. Addicts in clinics are much more likely to suffer from non-drug-related psychiatric disorders than are addicts who are not in clinics. To be sure, not all addicts in treatment do poorly. As I note in my book, treatment approaches that provide incentives for quitting drugs often yield good results (e.g., Higgins et al., 1995). Thus, efforts to group addiction with chronic diseases, such as Alzheimer's and diabetes, are misleading. Most addicts quit drugs by about age 30 and do so independent of treatment.

The epidemiological studies do not provide any information as to what led so many addicts to stop using drugs. However, logic suggests some answers. Although in most cases there is no record of a clinical intervention, something must have changed. The most obvious hypothesis is that everyday pressures, particularly those demands that are correlated with adulthood, had some influence. Clinical, biographical, and experimental studies support this inference. In report after report, drug use in addicts decreased under the pressure of age-related changes in familial responsibilities, incentives, penalties, and cultural values that stress sobriety. However, external pressures cannot be the whole story. Although every addict gets older, not every addict quits. Clearly there are important individual differences that to varying degrees insulate drug users from the forces that typically lead to quitting. However, with the exception of additional medical disorders, the factors that differentiate addicts who quit drugs from those who do not are largely unknown.

These findings are central to the understanding of the nature of addiction and are surprising, given received wisdom. Yet Lewis gives them barely a nod. Instead, he focuses on the nature of my explanatory principles, the definition of addiction, craving, and the role of dopamine in drug use. These are important issues, and as they are what Lewis gives most attention to, I will devote the remainder of my response to these topics.

What Counts As an Explanation?

Lewis writes, "Heyman's model, and choice models in general, cannot be convincing" (p. 150) if they do not include information about the brain. This seems to me an unreasonable claim. If taken seriously, we would have to put all the well-established, powerfully predictive, and sometimes subtle and elegant, quantitative models of choice in economics and psychology that have emerged over the past hundred years on hold while waiting for neuroscience confirmation. Take, for example, a choice model that Lewis emphasizes. In scores, if not hundreds, of laboratory studies, steeply bowed hyperbolic discount curves

have proven better predictors of preference for delayed rewards than have the exponential curves that economists have assumed (e.g., Ainslie, 1992). The residuals for the hyperbolic curves are smaller, and thus they provide a more convincing account of the psychological effects of delay on preference. In other words, we do not have to consult brain research to decide which principle is more convincing—the behavioral results provide the answer. It is possible that a more successful, nonhyperbolic model will emerge, but it too will be judged on the basis of how well it fits the data. Brain scans and other biological measures will certainly enrich our understanding of how delay influences choice, but they will not tell us whether we were right (or wrong) to prefer hyperbolic discount curves to exponential ones. Thus, it is hard to make sense of Lewis's comment that explanations that ignore the brain are not convincing.

What Is Addiction and How Should We Go About Discovering Its Nature?

The *Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 1994)* provides research based criteria for classifying psychiatric cases. The diagnostic categories are a work in progress, based on research, reliability studies, and the collective wisdom and experience of expert committees. The criteria are behavioral and have proven useful as measured by research and clinical outcomes. Hence, I focus largely on studies that use the *DSM* criteria for distinguishing drug addicts from drug users.

Lewis ignores the *DSM* account of addiction and instead relies on Robinson and Berridge's theory of addiction. Following their lead, he writes that "cue-triggered wanting" is the "*sine qua non*" of addiction (p. 151). He does not provide evidence as to why this definition is useful. Rather, he supports this approach with the comment that Berridge and his colleagues are "recognized leaders in the neuroscience of addiction" (p. 151). However, we need not rely on authority; we can put craving to the test. Does craving or the *DSM* criteria provide a better index of addiction?

Craving was included in the third edition of the *DSM* account of addiction, but it was later dropped because of complaints that it was hard to measure and seemed to add little to the elementary observation that addicts often used drugs (e.g., Kozlowski & Wilkinson, 1987). However, committees are not the last word on scientific truth, and addicts frequently do report that they have a strong desire or craving to take drugs. Thus, it is not unreasonable to tentatively accept Lewis's claim that craving is the defining feature of addiction and to check if the data support this definition.

The basic findings are that addicts, as is widely assumed, often report a strong desire or craving to get high. However, further questioning reveals that they do not necessarily act on these feelings. Addicts who quit often say that they learned to ignore their cravings and that their cravings subsided in time (Waldorf, Reinerman, & Murphy, 1991). Similarly, we do not always consummate our cravings for food or sex. Other studies reveal weak or nonexistent correlations between craving and

relapse, including decreases in craving at the onset of abstinence (e.g., Shiffman et al., 1997)—which is just the opposite of what should happen according to the understanding of addiction as drug craving. In several studies, cravings waxed and waned as a function of drug availability (e.g., Carter & Tiffany, 2001; Meyer & Mirin, 1979). When the drug was not available, cravings decreased. That is, increases in the likelihood of drug use increased cravings, rather than vice versa. These results do not imply that cravings are unimportant. For instance, once engendered, they may increase the motivation to take the drug. However, the bidirectional and often weak connections between cravings and drug use do show that craving is not the essence of addiction. Under certain circumstances, cravings are one of the many factors that influence drug use. The larger lesson is that addiction is a field with a rich empirical literature and that we need not rely on opinion to determine whether craving is the core feature of drug use in addicts; we can check what the research says.

Do Drug-Induced Increases in Dopamine Preclude Reason?

Lewis puts drug-induced increases in dopamine at the heart of addiction. He contends that dopamine distorts the user's sense of time, exaggerating the importance of now at the cost of later; that dopamine levels predict preferences; and that the addict is someone who has become caught in a "dopaminergic time machine" that "captures . . . momentary brain process far older than the capacity to reflect" (p. 153). In other words, drugs increase dopamine levels, and dopamine decreases reason. This picture is vivid and persuasive, but do the data support it?

Lewis's account echoes those found in countless journal articles, clinical texts, and National Institute on Drug Abuse public information bulletins. However, Lewis does not check if the claims are valid; rather, as before, he relies on expert opinion and again cites Berridge and his colleagues. For instance, to support his views, he writes, "Berridge and Aldrige (2008) use phrases like 'frenzied pursuit' . . . and . . . 'eager hovering' to describe addictive activities . . ." (p. 152). Berridge's interpretation of dopamine's psychological effects is based largely on his rat research, and, indeed, most of what is known about dopamine and behavior is based on animal studies. However, the results of these studies are often hard to interpret because dopamine also mediates motor function and because the experiments rarely include potent, competing, nondrug reinforcers that could provide a meaningful test of whether the rats were indeed too "frenzied" to make nondrug choices. For example, when a rat is faced with a choice between drug and water but is not water deprived, there is really no reason for the rat to ever drink the water. Under these conditions, the drug could have virtually no reinforcing value but still attract close to 100% of all responses. This might appear to some observers as compulsive drug seeking.

Serge Ahmed and his colleagues (Lenoir et al., 2007) conducted a study that offers a rare and valuable exception to the

general trend of rat drug studies. They offered rats a choice between cocaine and saccharin. This is a telling comparison. Cocaine markedly increases the availability of dopamine in reward circuits and is said to be highly reinforcing, whereas saccharin is only moderately reinforcing (relative to sucrose; e.g., Heyman, 1997) and has relatively little influence on dopamine levels (e.g., Lenoir et al., 2007). To help insure the relevance to addiction, the researchers developed a unique behavioral procedure that promoted escalating amounts of cocaine consumption. It is likely that no rats have ever consumed more cocaine at higher rates than did the rats in the Lenoir experiment. However, as soon as saccharin became available, every rat switched to it. After a few sessions, the rats preferred saccharin to cocaine. As measured by shifts in preference, the rats were as behaviorally flexible after weeks of cocaine administration as they were at the start of the experiment, and despite long-term exposure to huge amounts of cocaine, they preferred the substance that was correlated with substantially lower dopamine levels. Other researchers have found similar results in primates (e.g., Nader & Woolverton, 1991).

Lewis's characterization of dopamine as the biological basis of addiction is consistent with current received wisdom. However, current conventional wisdom regarding dopamine and addiction ignores critical control conditions for interpreting drug effects and ignores the results of studies in which the researchers allowed access to a viable nondrug reinforcer.

Related to the role of dopamine in drug preference is the more general matter of whether drug-induced changes in the brain play a role in addiction. Lewis says that I "strenuously reject this idea" (p. 153). My position is that drug induced neuroadaptations do not necessarily imply compulsive drug use. Rather, as noted earlier, the question is whether drug-induced neural changes decrease the user's susceptibility to the determinants of choice. In light of current research, I think the most cautious answer to this question is that if there are drug-induced changes in the capacity to make choices, they are not of sufficient magnitude to prevent most addicts from quitting.

The first five chapters of my book outline the natural history of drug use, emphasizing studies that recruit addicts scientifically rather than relying on self-selected clinic populations. These results are, I believe, essential to the understanding of addiction. For instance, to ignore the first five chapters is to ignore drug use in the majority of those who meet the criteria for addiction. The last two chapters provide a framework for making sense of the research findings. This framework is based on three self-evident principles of choice. The principles can be combined so as to generate simple equations and graphs. In this form, they predict key features of addiction, such as alternating periods of heavy use and abstinence, the properties that make substances more likely to become the focus of an addiction, and even the excuses that accompany relapse, such as "This is the last time." The model helps explain addiction, and since it is not specific to drugs, it also leads to the prediction that any favored good, regardless of its nature, will be overconsumed. This may help explain why excessive appetites and cultural institutions

dedicated to curbing excessive appetites have for so long been prominent in human affairs. The Greeks preached moderation—we complain about supersized Big Macs. My argument is that both phenomena reflect the same underlying principles of choice, despite their great historical and cultural differences.

Lewis ends with an on-target outline of the dynamics of addiction. He observes that addiction is not a "monolithic state" (p. 150), but a recurrent series of varying patterns of drug use, accompanied by varying brain states. I agree that this is what addiction is like, and my book was written in the spirit of this characterization. It shows, I believe, that the varying drug consumption levels reflect varying choices. What this response to Lewis's essay adds is that we are now ready for a more convincing account of the varying brain states that accompany the varying choices.

Declaration of Conflicting Interests

The author declared that he had no conflicts of interest with respect to his authorship or the publication of this article.

References

- Ainslie, G. (1992). *Picoeconomics: The strategic interaction of successive motivational states within the person*. Cambridge, England: Cambridge University Press.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders*. (4th ed.). Washington, DC: Author.
- Anthony, J.C., & Helzer, J.E. (1991). Syndromes of drug abuse and dependence. In L.N. Robins & D.A. Regier (Eds.), *Psychiatric disorders in America: The epidemiologic catchment area study* (pp. 116–154). New York: Free Press.
- Berridge, K.C., & Aldrige, J.W. (2008). Decision utility, the brain, and pursuit of hedonic goals. *Social Cognition, 26*, 621–646.
- Brecher, E.M. (1972). *Licit and illicit drugs*. Boston: Little, Brown.
- Carter, B.I., & Tiffany, S.T. (2001). The cue-availability paradigm: The effects of cue availability on cue reactivity in smokers. *Experimental and Clinical Psychopharmacology, 9*, 183–190.
- Heyman, G.M. (1997). Preference for saccharin sweetened alcohol relative to isocaloric sucrose. *Psychopharmacology, 129*, 72–78.
- Higgins, S.T., Budney, A.J., Bickel, W.K., Badger, G.J., Foerg, F.E., & Ogden, D. (1995). Outpatient behavioral treatment for cocaine dependence: One-year outcome. *Experimental and Clinical Psychopharmacology, 3*, 205–212.
- Kessler, R.C., Chiu, W.T., Demler, O., Merikangas, K.R., & Walters, E.E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*, 617–627.
- Kozlowski, L.T., & Wilkinson, D.A. (1987). Use and misuse of the concept of craving by alcohol, tobacco, and drug researchers. *British Journal of Addiction, 82*, 31–36.
- Lenoir, M., Serre, F., Cantin, L., & Ahmed, S.H. (2007). Intense sweetness surpasses cocaine reward. *PLoS ONE, 2*, e698.
- Lewis, M.D. (2011). Dopamine and the neural "now": Essay and review of Addiction: A disorder of choice. *Perspectives on Psychological Science, 6*, 150–155.
- Meyer, R.E., & Mirin, S.M. (1979). *The heroin stimulus: Implications for a theory of addiction*. New York: Plenum.

- Nader, M.A., & Woolverton, W.L. (1991). Effects of increasing the magnitude of an alternative reinforcer on drug choice in a discrete-trials choice procedure. *Psychopharmacology*, *105*, 169–174.
- Robins, L.N., Helzer, J.E., & Davis, D.H. (1975). Narcotic use in southeast Asia and afterward. An interview study of 898 Vietnam returnees. *Archives of General Psychiatry*, *32*, 955–961.
- Robins, L.N., & Regier, D. (1991). *Psychiatric disorders in America*. New York: Free Press.
- Shiffman, S., Engberg, J.B., Paty, J.A., Perz, W.G., Gnys, M., Kassel, J.D., & Hickcox, M. (1997). A day at a time: Predicting smoking lapse from daily urge. *Journal of Abnormal Psychology*, *106*, 104–116.
- Stinson, F.S., Grant, B.F., Dawson, D., Ruan, W.J., Huang, B., & Saha, T. (2005). Comorbidity between DSM-IV alcohol and specific drug use disorders in the United States: Results from the National Epidemiological Survey on Alcohol and Related Conditions. *Drug and Alcohol Dependence*, *80*, 105–116.
- Waldorf, D., Reinarman, C., & Murphy, S. (1991). *Cocaine changes: The experience of using and quitting*. Philadelphia: Temple University Press.
- Warner, L.A., Kessler, R.C., Hughes, M., Anthony, J.C., & Nelson, C.B. (1995). Prevalence and correlates of drug use and dependence in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *52*, 219–229.