
Addiction may or may not be a highly prevalent condition, but the concept of addiction is undeniably ubiquitous. From the people who cheerfully and publicly announce their addiction to coffee, or chocolate, or shopping, to those who ruefully and perhaps only in very special settings admit their addiction to alcohol or drugs, “addiction” is an oft-invoked explanatory frame for the presentation and characterization of individual behavior. Lately, it has even been applied to the behavior of super-personal entities, as in America’s “addiction” to oil.

Although the ubiquity of the concept is surely a sign of its usefulness, it also gives one pause; can a term of such broad application really have precise meaning (compare the word “thing”)? And if not—if there is nothing that all the “addicted” entities have in common—then why is the concept so apparently useful, and what is it useful for? Such questions may seem tailor made for Ivory Tower semantic analysis, but in fact the matter is much more urgent than that. For we live in a world where involuntary commitments and other coercive measures are sometimes considered justified in the course of dealing with addicted persons. Why is this so? What could be wrong with addicted persons that would justify such treatment? And why is the word extended to apply to persons for whom such treatment would presumably not be justified? These are some of the several questions asked by the authors of Midbrain Mutiny, and they have not just scientific, but also political and philosophical motivations for wanting to answer them.

So what is an addict? One possible definition—one that would seem to accord with the widespread use of the term—is an agent with abnormal preferences, in the sense that the addict is willing to pay far more, and to pay for far higher quantities of a good than is the average consumer. Here we should think not just of the novelist maniacally devoted to the twin pleasures of writing and alcohol, but also of the late Steve Irwin “addicted” to contact with dangerous animals, or of the infinitely more prosaic CEO who devotes all of her time to work. Although the typical person might reject such choices for themselves, and describe those who lead such lives in addiction-inspired language (e.g. the “workaholic”), few would argue that they are addicted in the sense required for paternalistic intervention—at least insofar as the persons described have freely chosen their respective lives, and continue to endorse them.

This last stipulation is of particular importance, for it points to something characteristic of “true” addicts, but not, say, to libertines: episodes of regret following the pursuit of the good in question, leading to efforts to end the relevant pursuit, but which eventually result in further consumption. In the more precise language of the authors, “true” addicts are characterized not just by relapse or repetition, but by the simultaneous investment in efforts to stop consumption and efforts to enable it. For Ross et al., it is this particular irrationality—this conflict in desires and behavior of the addict—that is both the necessary condition for any paternalism, and one of the primary hallmarks of the condition in need of scientific explanation. Noticing and accepting this characterization is thus important both as a step towards a better scientific understanding of the core nature of addiction, but also as way of protecting those with minority preferences from those wishing to promote and enforce more conservative—or just more mainstream—behaviors. In fact, the authors suggest, it is precisely the wish to blur the distinction between unusual preferences and irrational compulsion that promotes the broad use (they would argue mis-use) of the word “addiction”. Whether the word is used by individuals wishing to be excused from responsibility for their own tastes, or by those looking for justification to forcibly alter behavior they consider improper, these uses of the concept of addiction threaten both the foundation of liberal democracy, and the prospect of properly identifying and treating real addiction. As noted above, there is more at stake here than mere semantic correctness.

What, then, explains the characteristic irrationality of the addict? One prominent explanation—the one that according to the authors remains the favored economic model of addictive behavior—is that addicts occupy disordered reward circumstances. To understand this suggestion, one must reflect on the fact that not every good has the same price for every agent, nor for a given agent in every circumstance. It is more costly to pursue illegal or socially proscribed ends (marijuana is by
many measures more costly in the US than in Amsterdam); likewise the emphysema patient pays more for each cigarette than does a healthy smoker. People in such circumstances may both want, and not want, to pursue the end in question, and these preferences may shift with changes in perceived cost. One can imagine a person who would indulge in minority sexual preferences but for social circumstances making the cost of discovery high, or the drug user who would stop using but for the high cost of withdrawal. If this is the right story, then what looks like irrationality from the outside is in fact the rational pursuit of desired goods under circumstances that allow occasional pursuit of the end, but make consistent pursuit too costly. The agent in these circumstances might even recognize that her circumstances are disordered in just this way, but still be unable to remove herself from them for fear of paying the very costs that drive her globally incoherent behavior.

Although this is no doubt an accurate description of and explanation for the behavior of some agents, Ross et al. argue that it does not reflect the situation of the true addict, for a very simple reason: no rational agent who recognized the nature of their disordered circumstances, and was freed from them, would willingly put themselves back into that very situation; the gay man who pays the price of his outing does not typically go back into the closet. But the drug addict who has gone through rehab—who has thus been freed from his circumstances and who therefore has a very clear idea of the full costs of his behavior—nevertheless all too typically steps right back into them.

Thus, for Ross et al., it is not just the appearance of irrationality that is characteristic of the addict, but a real and deep-seated disconnect between cost/benefit calculation and resulting action. Nor is it an issue of impulsivity (although there are such disorders), for the addict's behavior is typically careful and deliberate. This is a problem for economic models of rational agency, because addicts would appear to be agents with perfect knowledge of price and a normal degree of control over their behavior, who nevertheless deliberately act against their own self-interest. Thus, the authors argue it is necessary to supplement economic theory with data from cognitive science, and generate a hybrid neuroeconomic model of addiction to account for the behavior of (at least) this class of agents.

As the reader will not be surprised to learn, that model turns out to be somewhat complex, but the core idea is relatively straightforward. Animals who need to predict the outcomes of their actions (will eating this result in nourishment or sickness?), and what in general to expect from the world in various circumstances (does that noise presage the appearance of a predator?) also typically have mechanisms for adjusting those predictions when they prove inaccurate. These work in part, and roughly, as follows: prediction errors cause the creation of (internal) circumstances that reward the repetition of a given action so as to reduce uncertainty about its outcome. As prediction errors decrease, so does the reward associated with the action, and thus also the motivation to repeat it. In humans, we typically associate the stages of the learning cycle with surprise, curiosity, and boredom, respectively, but the conscious experience of these (and similar) states is not a necessary part of the learning-motivation mechanism. Likewise, although we talk of “satisfying” our curiosity, the reward for uncertainty reduction is only loosely associated with hedonic pleasure. What is important is only that our brains contain a sub-personal mechanism designed to internally reinforce certain actions to the degree that it expects to learn something from the outcome. These reward processing mechanisms are implemented largely—although not exclusively—in midbrain structures like the substantia nigra, ventral tegmental area, and amygdala, and reward appears to be mediated by—or, perhaps one might say consists in—the release of dopamine in these regions.

The central hypothesis of Midbrain Mutiny is that in addicted agents this apparatus malfunctions in such a way that participation in the addictive behavior continually produces prediction errors, thus setting the conditions for internal reward upon repetition of the action. In addicts, the feedback loop that decreases reward as expectations line up with outcomes never kicks in; it is as if the addict (or, really, the addict's brain) always expects to learn something new the next time he indulges. The precise cause of this malfunction differs for different addictive targets—some drugs like cocaine apparently just directly flip the “surprise” switch—but the authors argue that the common cause underlying all real addiction is this particular dysfunction of a reward system designed in part to keep us in tune with our environments.

This brings us to gambling. The reader may well be wondering how we could have gotten to this part of the review without once mentioning what is ostensibly the focus of the volume. The answer is simple: Midbrain Mutiny is about gambling only insofar as it is about addiction in general. One of the more interesting claims in the book is that, precisely because gambling does not involve the ingestion of substances with numerous and varied psychotropic effects, gambling addiction showcases the disorder in its purest form. For what gambling does—in at least some people, some of the time—is manipulate the midbrain reward system by continually producing “surprising”, locally unpredictable outcomes. Whether that outcome is good (a win) or bad (a loss) is immaterial from the standpoint of a system whose primary function is the accurate prediction of outcomes; thus, every outcome reinforces the next bet.

Of course, if this is the right story—and the evidence the authors marshal is largely convincing—one might well wonder why not everyone is addicted to gambling. For in a strict sense the reward system is operating exactly as designed; the agent’s behavior may be disordered as a result, but the system is not malfunctioning on its own terms. Thus, it is not gambling addiction that needs to be explained, but casual gambling. It is one of the strengths of the book that the authors do not shy away from this question, and what emerges from their discussion is a rich picture of human agency, in which multiple kinds of rewards, systems for monitoring and pursuing them, and cognitive strategies for focusing some pursuits over others, all compete and cooperate in particular environments to give shape to the behavioral trajectory of the person. This discussion falls short of an explanation for why some people are more susceptible to addiction than others—although it offers an interesting characterization of the relevant differences—but what it certainly does do is make clear that the economic
model of rational agency, like the ideal gas law, is a perfectly workable abstraction under many circumstances that breaks down in significant ways when dealing with real agents in particular conditions.1

There is much to like about Midbrain Mutiny. It is interesting, deliberate, and very reasonable, treating delicate issues like the proper relations between various sciences—e.g., why and how much economics should care about the detailed, neuroscientific model of agency emerging from the cognitive sciences—with the care and precision they deserve. Refreshingly, it avoids the knee-jerk reductionism that mars so much work attempting to bring knowledge from the “basic” sciences to bear on issues in the higher-order behavioral and social ones. In one typical passage of this sort, the authors affirm that “what counts as behavioral pattern p in one ecological context might not count as another instance of p in a different ecological context.” (11) That is, what would count as the same action when described in low-level terms like joint angles and velocities, would not count as the same action when viewed in its full behavioral context, and it is the latter that matters to the task of individuating and interpreting actions. Indeed, this work doubles as an extended meditation on the issue of what it is to explain anything at all, what level and sort of reduction is warranted, and how to negotiate the competing demands of various cognitive and scientific desiderata.

On the other hand, the book also has some potentially significant weaknesses. The authors are quite right to note that a given bodily movement only gets meaning as an action in its full behavioral context, but they miss the fact that the same is true for the activity of the brain. Although it has been the prevailing practice in cognitive neuroscience to interpret regional brain activations in isolation from one another, work coming out of my lab and elsewhere (Anderson, 2007, 2008; Dagher, Owen, Boecker, & Brooks, 1999; Hagoort, 2005; Thompson & Varela, 2001; Uttal, 2001) suggests this is a profound mistake: the same low-level brain computation can be put to many different uses, depending on the overall neural and environmental context, and thus what a given regional activation “means”—what the region is “doing” for the system—can be different in different circumstances. They likewise make a simpler mistake in asserting that fMRI activations predict neural firing rates (255: fn. 5); in fact the BOLD signal2 that fMRI detects is much more highly correlated with the local field potential, which seems to be a measure of local dendritic processing (and not axonal spiking activity), and is in many cases entirely uncorrelated with spike rates (see, e.g., Logothetis, 2007). These two mistakes compound when the authors adopt the underlying assumption that more BOLD signal indicates that a region is doing more of whatever it is that the region has been associated with in other studies. This wouldn’t be true even if the BOLD signal were associated with firing rates; it is often the case that the firing rate is itself a carrier of information—for instance the angle of a certain joint, or the slant of a certain line—so that more activity simply indicates that the system is in a different informational state. The point is not that this is what “more” means everywhere in the brain, but that what more activity means is probably different for different regions, in different circumstances.

To see why these mistakes matter, consider the following passage addressing the observation that adolescents are at higher risk for gambling addition than adults. Their model implies that adolescents must get a greater reward for gambling, and that would seem to predict that there should be more BOLD response in the relevant midbrain structures. The authors write:

“Bjork et al. (2004) compared the BOLD responses of 12- to 17-year-olds with those of 21- to 28-year-olds on a delayed monetary reward task. Their results do not support the hypothesis that adolescents have increased reward cue-elicited anticipatory VS activation. They indicate an adolescent VS activation deficit rather than a VS activation surplus as a factor for risky behavior. According to this account, adolescents may seek more extreme incentives as a way of compensating for low VS activity levels (Spear, 2000). Either way, the risky behavior observed in adolescents appears to be due to faulty reward processing of some sort.” (175).

Naturally, any scientist should be suspicious about the predictive power and specificity of a model if both possible experimental outcomes support it. But more to the point, it is the narrowness of the authors’ understanding of the meaning of the BOLD signal that in the current case forces them into the unsatisfying post hoc explanation of the experimental result. In fact, since the BOLD response is an indirect, metabolically-driven measure of brain activity, variations can just as easily indicate differences in metabolic efficiency. Might it be the case that adolescents have a higher metabolic efficiency than adults?

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It would certainly be worth the authors’ time to find out. And indeed, since the BOLD response is an indirect, metabolically-driven measure of brain activity, variations can just as easily indicate differences in metabolic efficiency. Might it be the case that adolescents have a higher metabolic efficiency than adults?

1 The ideal gas law, \( PV = nRT \), describes the relation between the pressure (\( P \)), volume (\( V \)), temperature (\( T \)) and amount (\( n \)) of a hypothetical gas consisting of point particles that interact only via the exchange of kinetic energy during collisions, like tiny billiard balls in space (\( R \) is a constant that balances the equation). A triumph of 19th century scientific abstraction, the law provides a good approximation of the behavior of simple gasses at high temperature and low pressure; but as any engineer knows, the predictions can be seriously mistaken under certain conditions and for more complex gasses such as hydrocarbons. What conditions matter, and when, depends on the particular properties of the gas in question.

2 BOLD signal = Blood Oxygenation Level Dependent signal. Changes in regional neural activity are typically associated with changes in the amount of oxygenated blood flowing to the region in question. These changes can be detected using a Magnetic Resonance Imaging (MRI) device specially tuned for this purpose; the procedure, meant to reveal ongoing activity in the brain, is thus called functional Magnetic Resonance Imaging (fMRI).
References


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