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Dopamine and the Neural "Now": Essay and Review of *Addiction: A Disorder of Choice*

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Abstract

Rather than view addiction as a disease, Heyman sees it as a choice—one that works like other choices, whereby immediate rewards outshine long-term gains. He rejects neuroscientific explanations of addictive behavior, because he believes they cast it as involuntary or disease-like. I argue that the disease-versus-choice debate creates a false dichotomy: Neuroscience does not have to frame addiction as a disease. Rather, it can help explain how addicts make impulsive choices in the moment and distort appraisal and decision-making habits in the long run. Specifically, the salience of drug-related cues is enhanced by dopamine activity in the ventral striatum, orbitofrontal cortex, and amygdala, due to the intense hedonic impact of repeated drug experiences. Moreover, dopamine-based craving peaks when drug (or alcohol or gambling) rewards become available, in the moment, and this rapid increase in attractiveness preempts rational judgment. Finally, repeated dopamine enhancement modifies brain structures to maximize the appeal of addictive activities, minimize the appeal of competing rewards, and undermine the cognitive capacities necessary to choose between them. I conclude that addiction is not a monolithic state but a recurrent series of choices that permit negotiation, and sometimes cooperation, between immediate and long-range goals.

Keywords

neuroscience of addiction, dopamine, craving, choice vs. disease model, hyperbolic discounting

Efforts to understand addiction have crystallized into a cold war between the disease model and an alternative model based on the exercise of choice. The disease model emphasizes the physiological changes that go with addiction, including changes in the function and structure of the nervous system (e.g., Volkow & Fowler, 2000; Washton, 1989). The choice model is based on epidemiological studies, psychological research, and self-report data (e.g., Peele, 2000; Schaler, 2000). In his clever and provocative book, Heyman (2009) asserts that the disease model is wrong. Drug addicts can and do recover, they sometimes stop indefinitely, and their decisions to take drugs or to quit are executed voluntarily. Diseases don't work that way. You can't get rid of cholera or cancer through volition. Thus, addiction appears to be a choice rather than a disease.

Heyman rejects biological explanations of addiction and replaces them with principles drawn from behavioral economics and learning theory. For him, addiction works like other choices, in which immediate rewards take precedence over long-term gains. But to my mind Heyman's model, and choice models in general, cannot be convincing as long as they dismiss or ignore the brain. Neuroscience doesn't have to cast addiction as a disease. Rather, it can offer a way out of the disease–choice dichotomy by identifying the biological processes that connect reward to decision making in the moment and goal-seeking habits over time. In this article, I critique Heyman's argument and propose an alternative view that is based on the neuroscience of addiction and the role of dopamine in motivated action.

Heyman's Message

Heyman recognizes that no one specifically chooses to be an addict. But he claims that the decision to take drugs has the same form as normal day-to-day decisions. For him, the problem of addiction is an extreme version of the more general

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problem of choosing what is most rewarding in the moment at the expense of long-term goals: choosing "locally" rather than "globally." Heyman is right that people usually choose what is most fun (or least painful) in the immediate future while devaluing rewards (or punishments) that come later. He cites studies of delay discounting that investigate these effects. For ordinary people in ordinary circumstances, this tendency leads to excess, which is, as Heyman states, "a fundamental feature of voluntary behavior" (p. 134). But for addicts, the consequences are more serious.

The problem, says Heyman, is that repeatedly choosing immediate rewards sets both immediate and long-term rewards to lower and lower values. To demonstrate the generality of this phenomenon, he compares the conventional reward of Chinese food to the drug reward of heroin. In either case, repeatedly choosing the immediate reward (Chinese food or heroin) lowers its value over time. Chinese food becomes boring. Heroin becomes expensive, physically addictive, marriage-destroying, and so forth. The decline in long-term value parallels day-to-day decrements in short-term value. Yet this decline is self-perpetuating: It makes it harder to ignore the attraction of immediate rewards on any single occasion because, according to Heyman's formula, the local choice continues to be valued above the global choice. In other words, an immediate reward—"one more time," as addicts often tell themselves—is always more attractive than waiting for the long-term picture to get brighter.

If one could overlook the immediate attractions of the drug, or the Chinese food, on any particular night, then one could concentrate on the long term instead. If one could do that night after night, the habit (call it an addiction or not) would be beaten! Heyman considers this a choice, and he cites epidemiological, survey, and self-report evidence that it is a choice addicts frequently make. By focusing on long-term rather than short-term outcomes, one can score the maximum amount of reward over time. For example, one can choose to go for a month or a year eating at a variety of different restaurants (saving Chinese food for weekends) and avoiding (or at least reducing) shots of heroin. The same applies to drinking (no thanks, not tonight), gambling, smoking, and so forth. The trick, Heyman concludes, is to acquire strategies for evaluating the overall summation of rewards over time and ignoring the attractions of immediate gain.

Nobody would argue that going to a Chinese restaurant is a disease, and few would deny that it is a choice. Heyman sees drug addiction in the same light. You can choose to do it or you can choose to stop. He doesn't say it's easy, but it is possible. These are voluntary acts, not products of a diseased brain. Heyman avoids the issue of will, free will, or willpower (highly overlapping and slippery concepts), and he minimizes the role of rationality. Instead, he emphasizes perspective, environmental context, societal and personal rules or values, the relative reward of competing alternatives, and learning itself. Some combination of these factors helps addicts to quit, he says. Yet it occurs to me that the alcoholic can choose his route home to avoid every bar, force himself to think about the consequences of failure, recite personal rules or biblical injunctions at every step, and still end his evening staring at the bottom of a glass.

The question, then, is why is it so *hard* to quit (and remain abstinent)? For me, this is by far the most important question for understanding addiction. Heyman asks the same question (p. 129), but his answer relies on abstract principles of bookkeeping and formulas for comparing perspectives. Can motivation really be left out of the analysis of choice? I agree with Heyman that time is of the essence, and immediacy (the local perspective) is the danger zone for addicts. But what is it about the moment, the immediate, that gives it such power? In the remainder of this article, I will show that questions of motivation and immediacy have to be connected in order to understand addiction, and these questions can only be formulated and resolved clearly and precisely when we consider the brain.

Cues, Conditioning, and Dopamine

One of Heyman's central claims is that voluntary behaviors vary according to their consequences, whereas "involuntary activities are elicited by preceding stimuli (e.g., urges) and are influenced little or not at all by their consequences" (p. 104). This means that involuntary behavior is a result of cue-driven or Pavlovian conditioning and that voluntary behavior is strengthened by operant reinforcements. Thus, in a number of case histories reviewed by Heyman, addicts voluntarily stop using drugs when the consequences get bad enough, and he extols the benefits of contingencies and incentives in programs for helping addicts abstain. It is notable that such a view echoes Skinner's own views on self-destructive behavior. In *Walden Two*, Skinner (1948) argued that strategic modification of contingencies is the best cure for self-indulgence. I'm not so sure that arguments based on conditioning resonate well with the issue of choice. But my claim in this section is that, by Heyman's own definition, addictive behavior is involuntary because it is primarily driven by cues and not by consequences.

Berridge and his colleagues, recognized leaders in the neuroscience of addiction, propose *incentive salience* or "cue-triggered wanting" as the *sine qua non* of addiction. Cues associated with drugs, such as paraphernalia, vodka ads, drug-taking buddies, or auditory tones for animals in cages, become more salient with recurring consumption and pairing of substance and stimulus. This is because drug taking triggers the release of dopamine, sucked up from the ventral tegmental area (VTA) in the midbrain. In turn, dopamine increases the impact of stimuli on the nucleus accumbens and ventral pallidum, both parts of the ventral striatum involved in reward seeking; the amygdala, involved in emotional conditioning; and the orbitofrontal cortex, where expectancies are encoded and value is assigned (Robinson & Berridge, 1993, 2003; Rosenkranz & Grace, 2002; Schultz, 2007a; Volkow & Fowler, 2000; Wyvell & Berridge, 2000). The pairing of cues with proximal drug experiences leads to their increasing power to induce psychophysiological responses (e.g., "wanting" or craving). The cues come to signify reward through Pavlovian conditioning and drive behavior through their powerful motivational effects.

Heyman assumes that there is no place for choice in such a model, but this assumption is based on the incorrect premise

that addiction is either a choice or a biological problem (e.g., a disease). This is a false dichotomy. Biological explanations are not incompatible with choice. Human motivation cannot be reduced to fixed action patterns, and cues do not trigger human behavior in the way that flies trigger the flick of a frog's tongue. Rather, motivation and emotion influence behavior by narrowing appraisals and urging actions that address them (this is the party line in emotion theory; e.g., Frijda, 1986; Izard, 1991). Acknowledging the motivational force of drug-related cues doesn't dehumanize addiction—it helps capture the biological vulnerabilities that are part of what it means to be human.

The central place of dopamine in addiction is strongly supported by the dopamine-enhancing effects of an entire class of addictive drugs (methamphetamine and cocaine) that target the ventral striatum, the impact of dopamine antagonists, dopamine knockout and other manipulations on drug-taking and orbitostriatal activation (e.g., Nestler, 2005; Volkow & Fowler, 2000), and by numerous other findings, including the potentiation of gambling problems in Parkinson's patients treated with dopamine agonists (Dodd et al., 2005). Moreover, Pavlovian conditioning in the amygdala is directly enhanced by dopamine (Rosenkranz & Grace, 2002), where it has been found to correlate with cocaine self-administration (Hurd, McGregor, & Pontén, 1997). Yet, neuroscientists no longer believe that dopamine equals reward in the brain. Rather, striatal changes involving dopamine correspond with the feeling of craving, not the feeling of being high (Risinger et al., 2005) or "wanting" rather than "liking" in Berridge's model.

Heyman dismisses the importance of dopamine-powered motivation in addicts. He points out that all rewards, including "natural" rewards such as eating and watching cartoons, release dopamine, yet they are rarely seen as addictive. I would counter that eating is addictive to people with eating disorders and cartoons are addictive to young children. But the main point is that dopamine is a key neuromodulator for motivating and directing goal-related behavior. Its functions are certainly not restricted to drug taking, yet much of the natural reward-seeking subserved by dopamine can have addiction-like characteristics. So what's special about drug addiction? Heyman acknowledges that drugs are unique: They are "the most likely substances to become the focus of an addiction" (p. 150). But he doesn't say why. In my view, the motivational thrust or urge associated with drugs results from the excessive and repeated dopaminergic highlighting of an experience that is enormously exciting or pleasurable. Drugs are *designed* for maximum hedonic impact. The greater the hedonic impact of a stimulus, and the more it is repeated, the more rapidly and intensely dopamine will amplify the salience of its cues. Heyman tries another argument—he reminds us that "even aversive events increase dopamine levels" (p. 143). This fact is well established, but it is irrelevant. The fact that dopamine can motivate escape or avoidance in relation to aversive events does not negate its capacity to motivate approach in relation to positive events. Heyman concludes that "there is not a specific relationship between dopamine and addictive drugs" (p. 143). I agree that there is no specific relationship in terms of a built-in biological exclusiveness. Yet the extreme hedonic impact of the drug experience accounts for the extreme potency of the dopamine-induced urges that lie at the heart of drug addiction.

Craving and the Power of "Now"

Addiction is highly sensitive to time. It is a problem that pits the present tense against the future tense. Heyman recognizes this. His main thesis relies on a distinction between momentary gain (the local approach) and long-term gain (the global approach). Yet dichotomizing time in this way misses a fundamental fact of addiction: Under the sway of dopamine, "now" gets rapidly, even suddenly, more attractive as one approaches it. Berridge and Aldridge (2008) use phrases like "frenzied pursuit" (p. 642), "immediate reward" (p. 648), and "eager hovering" (p. 638) to describe addictive activities in psychological terms, and phrases like "magnified firing bursts . . . at the moment of the cue" (p. 642) to describe brain states that mediate those activities. Schultz (2007b) reviews a large body of findings indicating that the dopamine activation underlying stimulus-based reward prediction is restricted to a very short time range—a few hundred milliseconds—translating to behavioral activation at the scale of 1–2 s. One of the most revealing studies of drug-taking *in time* found that addicts' self-reports of craving and their striatal and orbitofrontal activity (measured with fMRI) both peaked just before the moment of drug administration and declined immediately after (Risinger et al., 2005). Thus, at both behavioral and neural levels of description, immediacy is fundamental, and craving is its motivational correlate.

This does not mean that all drug-related *activities* are executed within a second or two of the cue that initiates them. More often it is the *decision* to indulge that takes hold in the moment. But that is the point, and it refutes Heyman's argument all the more directly, because the decision to take drugs—the choice itself—is the thing that interests him (and me) the most.

Heyman acknowledges that "cravings . . . are one of the factors that influence drug use" (p. 111). But "an urge is not an obligation," (p. 111) he concludes. This seems a rather strained argument. To say that "[c]ravings . . . do not make drug use obligatory" (p. 111) is simply to recall that human motivation is not controlled by fixed-action patterns—the stimulus–response programs that contributed far more to the evolution of reptiles than mammals. Heyman is right: Addicts can *choose* to get away from the moment—a claim he bolsters with many examples of spontaneous remission or recovery. However, my point, and the point Heyman minimizes, is that it is extremely difficult to do so, and especially difficult time after time. That is the central issue in addiction and one that is rarely missed by the gathering of alcoholics in the church basement or by anyone who has gone back to smoking following the whiff of a nearby cigarette. What is it about *the moment* that makes it so difficult to escape? And why are cravings, though not obligatory, still so very important?

Utility theories, derived from economics and imported into psychology, provide an objective metric for defining parameters of choice making (Schultz, 2006). A common approach in utility theory is delay discounting, which shows an exponential curve describing the value of an outcome rising from the present moment to the moment of its (expected) occurrence. The curve starts out low, representing a devaluation of rewards far ahead of us in time, and then it rises more and more rapidly, showing how rewards increase in their rated value as their moment of payout approaches. Heyman is familiar with delay discounting (Heyman & Gibb, 2006), and he agrees that most people choose immediate rewards even though they are less valuable objectively. However, the exponential curves typical of delay discounting trace a gradual rise in value as rewards get closer in time. According to George Ainslie (2001), this is why conventional utility theories do such a poor job of explaining addiction. In his elegant and powerful book, *Breakdown of Will*, Ainslie shows that discounting curves do not change smoothly. Rather, the curve representing value remains low until just before the reward becomes available, when it rises suddenly, tracing a hyperbola. This universal tendency has one chief implication. Not only are nearby rewards overvalued, but their overvaluation accelerates rapidly just as they appear on the subjective horizon—that is, when they become imminent. For the addict, drinker, or gambler, the value of the immediate indulgence suddenly looms when drugs, booze, or big stakes become available, dwarfing the value of clean living that one aspires to in the hazy future. This is why addicts continually "choose" what Heyman calls the local choice. The problem is that calling it a choice overlooks the urgency addicts feel when their rewards become available—not next week, not tomorrow, but now!

Although Ainslie has no interest in the brain or its chemicals, he describes a fact about motivated choice that fits perfectly with Risinger et al.'s (2005) findings: Craving peaks just before the expected experience and it does so in synch with orbitostriatal activation. There is nothing gradual about it. Berridge's model makes the same prediction: Just before the reward is likely to appear, when the brain is flooded with dopamine, attraction suddenly skyrockets (Tindell, Berridge, Zhang, Peciña, & Aldridge, 2005). The anatomy of this mechanism is thought to be centered in the ventral striatum, where attention and action are focused by rewards, and/or the orbitofrontal cortex, where events are evaluated as rewarding (or not). Both systems are directly fueled by dopamine from the VTA. In sum, just before a reward becomes available, there is a rapid rise in craving and attraction subserved by brain systems devoted to appraising the rewardingness of the environment and directing appropriate actions. It seems likely that there have been distinct evolutionary advantages to the motivational highlighting of immediate gains. Thus, craving, and its foundation in "now," may be the byproduct of a brain designed to be maximally responsive to immediate rewards.

Addiction epitomizes bad judgment and impulsivity, if not stark irrationality. But we can forgive the addict his addiction and the brain its impulsivity if we recognize that the dopaminergic machinery of the forebrain wasn't designed for rational decision making. Dopamine's job is to activate the power of cues. It evolved in vertebrates hundreds of millions of years ago because it highlights the value of immediate rewards and directs learning (Schultz, 2007a). Unfortunately, many acquisitions that are functional and adaptive (like upright spines) have their liabilities (like back aches). In the same vein, addiction may be a common ailment in brains such as ours. Other (more dorsal and more lateral) regions of the prefrontal cortex have to correct dopamine's impatience through a more reflective consideration of options and their consequences, and these regions are currently of great interest in the study of addiction. However, a sudden shift in the psyche triggered by the most rewarding of substances (i.e., stuff designed to make you feel good) cannot be reduced to poor judgment. Rather, a dopaminergic time machine captures the addict in a momentary brain process far older than the capacity to reflect.

Brain Changes Over Time

For Heyman as well as Ainslie, quitting depends on rules or strategies that highlight long-term gains, and it specifically requires the summation or "bundling" of future rewards into an aggregate goal that collapses future time points into something bigger, something more valued. Such a goal might be "sobriety," "self-love," or "having a happy marriage." Inhibiting or redirecting impulses is a crucial step toward this goal. The problem is that this kind of cognitive control requires a well-functioning brain. Many researchers conclude that brain changes brought about by addiction make it harder to quit, because they undermine inhibitory control, judgment, and/or reappraisal. Yet Heyman strenuously rejects the idea that brain changes underlie drug addiction. Who is right?

There is ample evidence that the brain is modified by addiction structurally as well as functionally, and some of these modifications hinder cognitive and self-regulatory capabilities. Nestler (2005) reviews data from many laboratories indicating that drugs of abuse induce lasting changes in regions associated with goal seeking. These changes include alterations in the responsiveness of the VTA, such that dopamine release is enhanced in relation to the drug and its cues but is decreased in relation to other stimuli—consistent with Robinson and Berridge's (1993) incentive-sensitization hypothesis. There are also shifts in other neuromodulators that interact with dopamine: for example, a major reduction in acetylcholine, leaving dopamine to dominate effortful behavior (Bechara & van der Kooy, 1992; Koob, 2009; Nader & van der Kooy, 1997). Because acetylcholine is the neuromodulator responsible for normal alertness and attention, the character of behavior now shifts from exploration, alertness, and volition to single-minded, desperate pursuit. Brain changes also include a primary target of dopamine, the nucleus accumbens, which begins to respond differently to inputs due to dendritic restructuring as well as altered receptivity to dopamine. Other regions associated with the VTA–accumbens loop may also be permanently altered, and these include the amygdala (emotional memory), hippocampus (declarative or explicit memory), hypothalamus

(involuntary behavior and the autonomic nervous system), and cortical regions. Volkow and colleagues have engaged in two decades of research into cortical changes underlying addiction. They conclude that the orbitofrontal cortex, critical for assigning value, and the anterior cingulate cortex, responsible for judging options and selecting among them, become dysfunctional both during addiction and during withdrawal (e.g., Goldstein & Volkow, 2002; Volkow & Fowler, 2000). They dub the resulting cognitive dysfunction "impaired response inhibition and salience attribution"—not a hopeful starting place for building new strategies.

Heyman does not deny that drugs change the brain. It is "a logical necessity as well as experimental fact," he says (p. 95). Yet he dismisses the importance of such brain changes by asserting that many if not all experiences change the brain. He concludes that neural plasticity underlies positive behavioral change, including voluntary actions, therapeutic improvement, and abstinence itself. Hence, brain changes can't be the problem in addiction. But this makes little sense. A very high proportion of the outputs from the striatum are inhibitory—that is, they limit activation in the areas that receive them. This can be considered the opposite of plasticity. Modifications to the orbitofrontal cortex, making it less responsive to nondrug reinforcers, also appear restrictive rather than enhancing. Clearly, not all brain changes are created equal. The problem is that Heyman devalues brain-change arguments to chip away at the disease model. "Drugs change the brain," he says, "but this does not make addiction a disease" (p. 97). It is this false dichotomy that muddies the waters once again. We can agree that addiction is not a disease. Now, rather than discard the lessons of several decades of research, we should look to neuroscience to help us resolve the more interesting question of what addiction is.

Heyman's view of rationality is hard to pin down. In some passages he argues that rationality is not essential for choice. This allows him to ignore the striking cognitive impairments associated with addiction-related brain changes. But elsewhere, he proposes that quitting (the best global choice) is facilitated by "the capacity to reflect upon the options" (p. 138), by "imagination and forethought" (p. 139), and by reappraising options "in more abstract terms . . . to exercise self-control" (p. 141). Thus, even for Heyman, higher order cognition, rationality, or judgment must be important for choosing the long-term benefits of quitting. In fact, "choice" without rationality risks becoming an empty label, based entirely on the precepts of learning theory. It follows that the capacity for meaningful choice, especially across multiple occasions, requires a well-functioning brain, capable of rationality, forethought, and self-control. Brain changes brought about by addiction work against this capacity.

The fact that addiction changes the brain does not make it a disease. It makes it a biological as well as psychological condition, based on an unusually strong (but not "unnatural") connection between hedonic experience and neural restructuring, facilitated by dopamine's pull for the immediate. Instead of ignoring addiction-related brain changes, I suggest that we look at them more closely. And since choice itself remains a mystery to philosophers as well as psychologists, let's hope that neuroscience can teach us something important about choice through the insights it has gained about addiction.

Conclusion

Based on the temporal properties of dopamine-induced craving and the neural changes that result from repeated dopamine enhancement, addiction and abstention are not choices made freely and flexibly. Yet, rather than lead to the notion of disease, these neural lessons suggest an approach to addiction compatible with Ainslie's model of *intertemporal bargaining*. Ainslie (2001) shows how temporal unevenness in assessing reward gives rise to a bargaining process between different "selves" or "positions." Each position takes the perspective of a different point in time—the immediate present, the anticipated future, or something in between—and "choice" arises from bargaining among these positions. Ainslie believes that addiction is a consequence of poor bargaining, and I think Heyman would agree. Heyman also highlights competing temporal perspectives leading to "conflicting motives that are experienced as ambivalence" (p. 172), and he recommends "conscious self-reflection" to achieve the best outcome. The best outcome is, of course, quitting (or abstaining). So the choice to quit (or remain abstinent) is captured by the phrase "intertemporal cooperation" (Ainslie, 2001, p. 104): lasting goodwill between the capricious urges of the current self and the realistic concerns of the future self.

Where I differ from both authors is in my emphasis on the knowledge we require to understand intertemporal bargaining. We need to put the brain into the picture. Neuroscience can help us make sense of addiction by bringing strong emotion and cognitive variability together in a single formula that predicts poor choices based on dopamine's distortion of time. From this perspective, intertemporal cooperation depends on escaping from dopaminergic desperation long enough to consider moments other than now. Momentary states of dopamine enhancement, triggered by a narrow range of cues, shut down intertemporal flexibility. Brain changes that accumulate with the recurrence of this cycle send it further out of reach. Yet addiction is not one monolithic brain state. It is a sequence of transitory states underlying impulse, reflection, and emotions that include shame and remorse. The recursive nature of this sequence provides windows of opportunity for the present self to influence future decisions—like the decision to quit—based on fluctuations in brain state, emotion, and cognitive functioning.

Viewed in this way, addiction is not a disease. Nor is it a single, global choice. It is neither entirely voluntary nor entirely involuntary. Rather, addiction comprises a recurrent series of brain states underlying a recurrent set of choices, whereby habit is interspersed with unexpected opportunities for change.

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

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