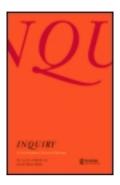
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Inquiry: An Interdisciplinary Journal of Philosophy

Publication details, including instructions for authors and subscription information: <u>http://www.tandfonline.com/loi/sinq20</u>

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To cite this article: Gene M. Heyman (2013): Addiction: An Emergent Consequence of Elementary Choice Principles, Inquiry: An Interdisciplinary Journal of Philosophy, DOI:10.1080/0020174X.2013.806126

To link to this article: <u>http://dx.doi.org/10.1080/0020174X.2013.806126</u>

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Addiction: An Emergent Consequence of Elementary Choice Principles

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(Received 6 April 2013)

ABSTRACT Clinicians, researchers and the informed public have come to view addiction as a brain disease. However, in nature even extreme events often reflect normal processes, for instance the principles of plate tectonics explain earthquakes as well as the gradual changes in the face of the earth. In the same way, excessive drug use is predicted by general principles of choice. One of the implications of this result is that drugs do not turn addicts into compulsive drug users; they retain the capacity to say 'no'. In support of the logical implications of the choice theory approach to addiction, research reveals that most addicts quit using drugs by about age 30, that most quit without professional help, that the correlates of quitting are the correlates of decision making, and, according to the most recent epidemiological evidence, the probability of quitting remains constant over time and independent of the onset of dependence. This last result implies that, after an initial period of heavy drug use, remission is independent of any further exposure to drugs. In short, there is much empirical support for the claim that addiction emerges as a function of the rules of everyday choice.

I. Introduction

Malaria is caused by parasitic microorganisms that infest and eventually rupture red blood cells. The resulting symptoms include fevers, chills, anemia, severe joint aches and, in some cases, death. According to Siddhartha Mukherjee some tumors and hematological cancers include a small number of cancer stem cells that generate new tumors.¹ For both diseases, an underlying, difficult-to-detect process produces overt harmful symptoms in the host,

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¹For example, Mukherjee, Emperor of All Maladies.

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and in both cases the underlying pathological process and the symptoms are readily distinguishable. That is, getting rid of the symptoms does not necessarily mean the disease has been cured. Most current models of addiction follow the same logic. In disease theories of addiction this is explicit. Their basic claim is that drug-induced changes in the brain promote excessive, uncontrollable drug use.² Or, consider the second sentence of the abstract for the conference (Agency and Addiction) at which an earlier version of this paper was presented:

Addictions to smoking, alcohol, gambling and illegal drugs cause much suffering. Large resources and strong interventions are used every year to discourage and punish these behaviors, as well as treat the underlying addiction.³

Although this was not a medical conference, the abstract presumes that excessive drug use and gambling are accompanied by an 'underlying' problem, and that clinicians should (and do) target the underlying processes. Thus, it is not unreasonable to say that among most researchers, clinicians and the informed public, addiction is a brain disease. Nevertheless, my goal in this paper is to convince the reader that this way of thinking about addiction is misguided. My argument is that, although addiction is a pathological form of behavior, there is no underlying pathology. The 'culprit' is the general principles that guide all choice.

My account proceeds in three parts. It begins with a discussion of the idea that certain phenomena emerge as a function of how their component parts combine rather than as a function of an underlying state. Second, I show that, under certain conditions, the principles that guide everyday choice produce highly excessive levels of consumption of whatever commodity was initially most favored. We shall see that a logical implication of this result is that drug-induced changes in the structure and function of the brain (which of course do exist) do not eliminate the capacity to voluntarily quit using drugs. Third, I introduce experimental, clinical and epidemiological evidence that support the first two claims. The data reveal that addicts remain susceptible to the persuasive powers of nondrug rewards and punishments. Indeed, the factors that influence everyday decision eventually persuade the vast majority of addicts to stop using drugs at clinically significant levels.

²For example, Kalivas and O'Brien, 'Drug Addiction'; Spanagel and Heilig, 'Addiction and its Brain Science'.

³Agency and Addiction Conference, University of Oslo, November 10-11, 2011.

II. Hurricanes and Addiction: Emergent Phenomena

Large and/or unusual events evoke explanations on the same scale, particularly when the events are not well understood. For example, until relatively recently, received wisdom explained catastrophic events, such as earthquakes and plagues, as the work of agents of equal magnitude, such as angry gods and other supernatural forces. However, it is now routine for scientists to explain natural disasters by the same principles that they use to explain everyday events. The principles that apply to continental drift, daily weather patterns and ocean waves apply to earthquakes, hurricanes and tsunamis. The extreme events do not reflect special principles, but the manner in which particular elements combine in accordance with general principles. For instance, hurricanes are infrequent but predictable disturbances, characterized by gale-force winds, mountainous waves and torrential downpours. When they reach land, they cause immense damage and by the standards of everyday weather are extreme events. Yet their etiology involves the elements of everyday weather and climate, such as latitude, the rotation of the earth, condensation and heat exchange. These elements are usually in balance, but as the oceans heat up in late summer, they can combine synergistically, resulting in a temporary, heatdriven feedback loop that produces powerful winds and heavy rains, which then re-combine to yield yet stronger winds and downpours. Notice that there is no distinct, dissociable, underlying 'hurricane' process. Rather, hurricanes and everyday weather differ in how their common elements interact. By contrast, recall that malaria requires the presence of a microorganism and that, for at least some chronic diseases, it is also possible to distinguish an underlying disease process—for instance the symptoms of diabetes (e.g. high blood sugar) are typically associated with insulin resistance and insufficient insulin production.

If natural disasters provide a model for the understanding of addiction, then a plausible version of this approach is that under certain conditions the principles that guide everyday choice lead to excessive levels of consumption. For instance, logic demands that all drug use is initially voluntary, so that we should consider whether under certain conditions, the rules of choice generate excessive amounts of voluntary behavior (just as laws of physics might generate hurricanes and earthquakes). This idea has consequences for the understanding of the relationship between the brain and addiction and also for how we talk about addiction.

First, the 'emergent' etiology implies that there is no need to assume a pathological, underlying enduring brain state that causes excessive levels of drug use. A corollary of this point is that, since drugs do change the brain, then the changes do not play a critical role in etiology and perhaps even in the persistence of drug use. (For instance, some heart murmurs are functionally significant whereas many are not.) This implication runs counter to the widely held assumption that drug-induced neural changes play an important

role in drug use. For instance, conventional wisdom is that the neural correlates of drug use cause addiction, by which is meant uncontrolled, compulsive drug use—the 'inability to say no'. However, the data are correlational. Often it is not possible to tell if the neural correlates preceded or followed drug use, and in no case has a causal tie been established between the neural correlates of drug use and compulsive drug use. Moreover, as discussed in subsequent sections of this essay, recent data indicate that such data will not be forthcoming.

Second, the criterion for saying someone is an addict should be the level and pattern of drug use, not an underlying state. By this standard, someone who once but no longer meets the criteria for drug dependence is not an addict. By contrast, the statements 'once an addict, always an addict' or 'addiction is a chronic, relapsing disease' go hand in hand with the claim that those who once met the criteria for addiction but no longer do so are, nevertheless, still addicts. This turns the diagnosis into a matter of faith rather than observation. For example, leading researchers and the authors of clinical texts routinely claim that addicts need lifelong care,⁴ even though every major epidemiological survey conducted in the United States in the last twenty years shows that addiction is the psychiatric disorder with the highest remission rate.⁵

III. Three Principles of Voluntary Behavior

There are a number of well-known choice theories of addiction.⁶ However, we can build a theory on the basis of three elementary observations that hold for all voluntary actions. The result is identical in form to Herrnstein and Prelec's theory of addiction; however, starting with elementary principles leads to a somewhat different emphasis.⁷

(1) Preferences are dynamic. Preferences for a substance or activity change as a function of previous choices and/or the passage of time. These patterns can be simple or complex, and they reflect both the nature of the commodity and the consumer. Most substances and activities quickly decrease in value because of such factors as satiation, fatigue, and boredom. However, some substances whet our appetite, which is to say they increase in value as a function of consumption, at least for a while (e.g. potato chips). Individual differences play a role in these relationships. Avid fans and enthusiasts become even more avid and enthusiastic as

⁴O'Brien and McLellan, 'Myths'; McLellan et al., 'Drug Dependence'.

⁵For example, Anthony and Helzer, 'Syndromes of Drug Abuse'; Stinson et al., 'Comorbidity'.

⁶For example, Ainslie and Monterosso, 'Hyperbolic Discounting'; Becker and Murphy, 'Theory of Rational Addiction'.

⁷Herrnstein and Prelec, 'Theory of Addiction'.

they learn more about their pet interests. By contrast, some people never become passionate about anything. However, regardless of the particulars, an item or activity's value invariably changes as a function of time, choice and consumption. Notice that consumption-dependent changes in preference must reflect changes in the brain, which is but another way of saying that changes in the brain are not in themselves evidence of either pathology or health.

- (2) Individuals always choose the better option. This is true by definition. However, what is best is ambiguous because there is more than one way to frame the options.
- In a series of choices between two or more items, it is possible to frame (3)the options in different ways. Imagine a host who keeps offering his guests after-dinner brandies, and a guest for whom the value of the brandy outweighs the value of not having a brandy at each offer. The perspective is 'one drink at a time', and under these conditions the obliging guest will keep accepting drinks—until too drunk to accept another. But now let us add the facts that this guest is someone for whom brandy quickly loses its value (e.g. tolerance) and has perverse effects on the future value of all his other activities. Moreover, these future costs are so great that, when the drink is evaluated in terms of the future as well as the present, it is disadvantageous to have even one drink (although at the moment it trumps not taking a drink). For example, his family will disown him if he comes home drunk one more time. Thus, if this guest frames his host's offer as a single brandy, he chooses it, and will do so at every opportunity to have a drink. But if he frames the offer in terms of his entire evening and ensuing plans (his ideal rate of drinking, given the overall consequences), he never chooses the brandy. The first frame of reference is local; the second is global and corresponds to what economists mean when they talk about consumers choosing among competing 'bundles' or 'market baskets' (i.e. the ideal combination of drinks and nondrinks).

IV. The Three Choice Principles Predict Binging on Drugs

Figure 1 presents a highly simplified but quantitatively faithful summary of these three basic choice principles. The left panel shows the local (one-drink-at-a-time) frame of reference. The *x*-axis represents the number of occasions in the last 30 that the guest accepted the offer for a drink. The *y*-axis represents the value of the drink and the value of the alternatives to drinking. As described above, on a choice-by-choice basis, the drink always has a higher value than skipping the drink, and, also as noted, each drink lowers the value of future drinks as well as the value of nondrinking activities. For instance, drinking may spoil relations at home or on the job. This, in turn, implies that skipping a drink increases the value of future drinks as well

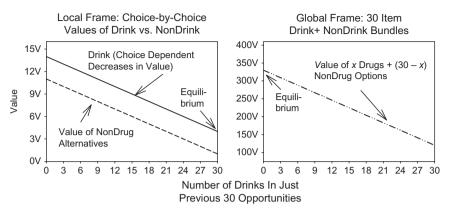


Figure 1. The relationship between value and choice for a commodity that mimics the properties of addictive drugs.

Note: The resulting functions correspond, in a schematic way, to the DSM definition of substance dependence. However, the underling process is voluntary choice, not compulsion.

as future nondrink options. Nevertheless, since no current option outpaces the value of the drink, at every opportunity the drink is chosen. This leads to the equilibrium in the lower, right corner, which is a 30-plus drink binge. Indeed, if nothing changes, the preferences and point of view displayed in the left-panel imply a never-ending binge.

Figure 1 also reflects the 'official' understanding of addiction. Clinicians, researchers and the courts use the American Psychiatric Association's (APA) DSM criteria for substance dependence to distinguish between addiction and ordinary drug use.⁸ According to the DSM:

the essential feature of substance dependence [addiction] is a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues to use the substance despite significant substance related problems, such as being caught driving under the influence, repeated black outs, extreme weight increase, etc.

The downward sloping lines represent the 'significant substance related problems'. These include direct drug effects, such as tolerance and withdrawal, and indirect effects, such as drug-related job loss, legal problems and disapproval of family and friends. The absolute difference between the value of the drug and the nondrug alternative represents the fact that drug use persists (its decreasing value notwithstanding).

⁸American Psychiatric Association, *Diagnostic and Statistical Manual*.

The right panel shows the same options but from the perspective of a series of choices that include both drinks and nondrinks, or what economists call 'bundles'. The x-axis is—again—the number of drinks in the previous 30 opportunities to have a drink. The y-axis, though, is different. It is the value of different combinations of drink and nondrink choices (or, equivalently, the value of different rates of drinking) over a longer period of time. For example, the midpoint of the y-axis is the value of 15 drinks plus 15 nondrink activities, and the endpoints are zero drinks out of 30 drink opportunities and 30 drinks out of 30 drink opportunities, respectively. Importantly, the values of drinks and of nondrink activities are exactly the same as in the left panel. For example, given the same history of drinking, the local consumer and the global consumer would estimate the value of the next drink and next nondrink (each taken by themselves) exactly the same. However, the global consumer does not consider choices on a drink-by-drink basis (there is no 'next drink'); he or she evaluates bundles composed of different numbers of drinks and nondrinks over an extended period. The right panel shows the value of drinking and nondrinking when framed this way. The value of bundles containing one or more drinks is always lower than the value of bundles without any drinks. Thus, in this example, the two ways of framing choices produce diametrically opposed behavioral patterns: never drink or never refuse a drink.

Two qualifications are in order. First, for real commodities, consumptiondependent changes in value must be much more complex than those displayed in Figure 1. Second, for most commodities, the local and global equilibria are not very different.⁹ However, for commodities that have the potential to undermine the value of competing activities, such as drugs, the local and global equilibria can be quite different. This is, I believe, why drugs are so problematic, and why they are invariably among the commodities most likely to be prohibited by governments.

V. Addiction, Context and Individual Differences

Land masses rob hurricanes of their warm water heat source thereby bringing them to a halt. Figures 2 and 3 show that a highly valued, beneficial nondrug option (or set of options) brings addiction to a halt, whereas a lower-valued, ordinary option paves the way for high levels of drug use. The solid lines in the left panels represent the value of the drug as consumption increases. In both cases, costs of drug use grow faster than the benefits in identical ways.¹⁰ The two dashed curves in Figure 2 represent the value of

⁹For examples, see Heyman, Addiction.

¹⁰In both graphs, the curves representing the value of the drug reflect the idea that the costs of drug use increase according to the frequency of use raised to a power of 2.0, whereas the benefits increase linearly as a function of use: $V = -AX^2 + BX + C$, where X is the number of drug choice in the last 10 opportunities. The constants were then adjusted so that the function would approximate 0.0 when 10 of the last 10 decisions were to use the drug.

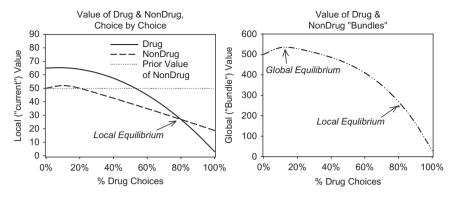


Figure 2. The relationship between value and choice in a setting in which the value of the nondrug alternative is undermined by drug use.

Note: In these conditions, the left panel shows that a consumer who re-evaluates his or her options at each choice ends up choosing the drug about 80% of the time. By contrast, the right panel shows that a consumer that chooses on the basis of the best combination of drug and nondrug experiences, chooses the drug about 18% of the time.

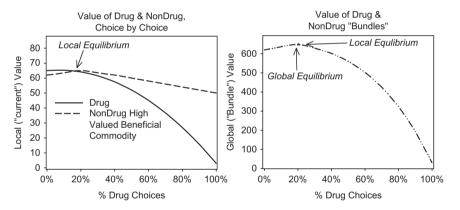


Figure 3. The relationship between value and choice in a setting in which the nondrug alternative is beneficial.

Note: This means that its value increases as a function of consumption, and the drug fails to undermine the value of the beneficial good. Under these conditions, drug use does not escalate, and local bookkeeping and global bookkeeping produce nearly identical (and nearly optimal) outcomes.

nondrug alternatives, with the higher one indicating the value of these activities prior to the onset of drug use. Notice that, as drug use proceeds, the value of the nondrug alternative decreases. The decrease represents what the APA refers to as 'significant substance related problems'. As a result, the local equilibrium shifts to the right so that drug use increases. This increase comes about because of changes in the values of the nondrug alternatives. Thus, drug use can increase even though the value of the drug has decreased and even though we have made no assumptions about the capacity to say 'no'. Indeed, in Figure 2, there are no assumed changes in the susceptibility to nondrug reinforcers.

The nondrug option in Figure 3 is beneficial. It increases in value as a function of consumption. Time spent in this activity pays off in the present and promises even greater rewards in the future. Consequently it does not decline in the face of drug use. As a result, drug use remains moderate, and the local and global equilibria are nearly identical. This means that drug use does not lead to an overall decline in the quality of life as in Figures 1 and 2. Put in more general terms, whether or not drug use escalates to problematic levels may depend largely on the nature and magnitude of the available nondrug reinforcers. However, it should be added that the magnitudes of the nondrug reinforcers reflect characteristics of the consumer as well as characteristics of the settings. Environments vary in the degree to which they offer beneficial activities, and individuals differ in their capacities to take advantage of beneficial activities.

VI. Evidence Regarding the Local/Global Analysis of Choice

Figures 1–3 outline several possible relationships between choice and the values of the commodities and activities that guide choice. However, they do not specify the processes or mechanisms that govern choice. For instance, individuals could evaluate their options just as shown in the graphs, so that consciousness mirrored the actual contingencies. Alternatively, individuals could be influenced by factors that indirectly reflect the contingencies depicted in the graphs. For instance, those under the influence of Apollian cultural traditions would make decisions that correspond to global decision making, and those under the influence of Dionysian cultural traditions would make decisions that correspond to local decision making. Or, the contingencies depicted in Figures 1-3 may operate in a piecemeal manner according to different commodities and activities and individual differences. For example, it is not unreasonable to suppose that most people are local bookkeepers most of the time, but when it comes to commodities such as drugs or activities such as child rearing, they switch to a more global approach. However, we need not establish how individuals make choices to test whether the local/global analysis of choice is relevant. If it correctly describes the correlations between action and outcome, particularly the ambiguous nature of these relations it will predict key features of everyday life, including addiction.

The local choice equilibrium is consistent with the matching law result.¹¹ The matching law is an empirical generalization, which in its ratio form says that choice ratios approximate reward ratios: e.g. B1/B2 = R1/R2. By rearrangement this is also an equity principle, which says that equal work gets equal pay: R1/B1 = R2/B2, and in this form it is identical to the local equilibrium. The matching result is also one of the most robust results in the choice literature. In hundreds of studies on how individuals allocate behavior among two or more competing reinforcement sources, the typical result is matching.¹² Matching holds for different species, for different settings (e.g. in the lab or in the 'wild'), for different reinforcers (e.g. brain stimulation, money, food, social approval), and for different response requirements (e.g. pushing a button, looking at a person or target, running in a wheel). In other words, over a remarkably wide range of conditions, individuals make choices as predicted by the local equilibrium of Figures 1–3.

There is also empirical support for the global equilibrium. Although choices typically gravitate to the local equilibrium, researchers have arranged conditions such that the choices approximate the global equilibrium.¹³ As suggested by the fact that this result is much less common, special efforts are usually required to insure the global equilibrium, such as additional stimuli that reveal the trial-to-trial dependencies. The global equilibrium is also the result that economic theory predicts. Textbooks and articles depict choice as a matter of competing bundles, each composed of different combinations of goods. Thus, when the consumer chooses the best bundle, he or she automatically becomes a global maximizer. Economics' growth and influence suggest that individuals and firms behave in just this way under some conditions. Thus, there is no shortage of empirical support for local and for global choice. The local equilibrium approximates the results of hundreds of research studies, and the global equilibrium approximates how economists say choices should be made—and on occasion are made.

VII. Why Are Drugs the Most Likely Focus of Addictions?

Figures 1–3 show that elementary rules of choice (principles 1–3) can combine so as to yield excessive levels of consumption that approximate the APA criteria for addiction. However, of the many substances and activities that humans find rewarding only a handful become 'addictive'. As outlined elsewhere in more detail, these substances and activities have distinct (and

¹¹Herrnstein et al., 'Utility Maximization and Melioration'; Vaughan, 'Melioration, Matching, and Maximization'.

¹²For example, Davison and McCarthy, *Matching Law*; Herrnstein, 'On the Law of Effect', *Matching Law*; Williams, 'Reinforcement, Choice'.

¹³For example, Heyman and Tanz, 'How to Teach a Pigeon'; Kudadjie-Gyamfi and Rachlin, 'Temporal Patterning'.

unusual) properties.¹⁴ They include the capacity to undermine the value of competing substances and activities (as in Figure 1), low rates of satiation, few if any substitutes, the capacity to intoxicate, immediate benefits and delayed costs. Not all addictive substances have all of these properties, but those that do not prove to be special cases that end up confirming this list. For example, cigarettes are not intoxicating but fill a niche that until recently had no other competitors.¹⁵ Thus, as is the case with many natural phenomena, addiction emerges as a function of how its elements combine with general principles. We need not assume a distinct, dissociable, underlying pathological process.

VIII. Do Drug Induced Neuroadaptations Lead to Compulsive Drug Use (the Inability to Say 'No')?

The thesis that drug-induced changes in the brain turn voluntary drug use into compulsive, involuntary drug use implies that addiction is a chronic disease (since there is no cure and disease symptoms are by definition involuntary), but that treatment can keep addicts off of drugs, just as treatment can help keep the symptoms of other chronic diseases at bay. The logical implications of this account are that addicts should be in lifelong treatment,¹⁶ that remission depends on treatment,¹⁷ that the correlates of remission are the correlates of chronic diseases and, conversely, the correlates of remission are not the correlates of choice (e.g. values, legislation, the threat of arrest, public opinion, and so on). These ideas are central to the disease interpretation of addiction and are endorsed and promoted by representatives of the federal health agencies (e.g. National Institute on Drug Abuse), neuroscientists,¹⁸ clinicians,¹⁹ the informed media,²⁰ and the informed public. But what do the data say?

The findings vary somewhat as a function of drug, with the distinction between legal and illegal drugs yielding the greatest variation.²¹ For illegal drugs, the following generalizations have now been replicated several times. Most individuals who meet the APA criteria for addiction are in 'remission' by age 30, and about 80% are in remission by about age 42.²² Remission is

²⁰For example, Quenqua, 'Rethinking Addiction's Roots'.

¹⁴Heyman, Addiction.

¹⁵Ibid.

¹⁶For example, McLellan et al., 'Drug Dependence'; O'Brien and McLellan, 'Myths'.

¹⁷For example, Goldstein et al., 'Neurocircuitry of Impaired Insight'; Leshner, 'Addiction is a Brain Disease'.

¹⁸For example, Kalivas, 'Glutamate Homeostasis Hypothesis'.

¹⁹For example, McLellan et al., 'Drug Dependence'; O'Brien and McLellan, 'Myths'.

²¹For example, Heyman, 'Quitting Drugs'.

²²For example, Anthony and Helzer, 'Syndromes of Drug Abuse'; Conway et al., 'Lifetime Comorbidity'; Kessler et al., 'Lifetime Prevalence', 'Prevalence, Severity, and Comorbidity'; Stinson et al., 'Comorbidity'; Warner et al., 'Prevalence and Correlates'.

typically stable so the proper word is probably 'resolved'.²³ Most of those who remit have not been in treatment.²⁴ And the correlates of quitting include economic concerns, worries about going to jail, pressure from friends and family, and the desire to behave more responsibly, as in becoming a better parent or a more worthy son or daughter.²⁵ In short, the correlates of quitting drugs are the correlates of choice. By contrast, economic considerations, financial worries and pride have little or no influence on recovery from those diseases to which addiction is said to be similar, such as diabetes, Alzheimer's, heart disease and cancer.

The findings could not be less supportive of the idea that addiction is a brain disease. Of course, we could call addiction a disease because it seems humane or convenient to do so, but to do so means failing to make key distinctions. Changes in legislation, threat of arrests, and new familial responsibilities are on record for bringing drug use to a halt in addicts; there is no analogous record for Tourette's syndrome, Alzheimer's disease and schizophrenia. Put another way, calling drug use in addicts 'compulsive' and calling addiction a 'disease' makes the world less sensible.

IX. Changes in Preference or Changes in Capacity?

It is difficult to distinguish between choices that reflect differences in the value of the alternatives and choices that reflect differences in the capacity to make decisions. For instance, what is the correct disposition when an addict continues to keep taking drugs, although at times he says he want to quit and in fact stops using for at least a while? Is drug use beyond his control or is he changing his mind? Indeed, distinguishing between these two possibilities may be a fool's errand. However, there are relevant observations that support the distinction between changes in preference and changes in the capacity to choose.

IX.i. Do cocaine-induced changes in the brain's 'pleasure' and 'planning' centers affect preference for cocaine?

Animal drug self-administration studies provide unambiguous information on drug-induced neural adaptations. By contrast, in human research the causal links between drug consumption and neural adaptation are often ambiguous. For example, since human brain research drug studies are rarely longitudinal, the observed differences between the brains of drug users and nondrug users could have been in place prior to drug use or could be due to

²³For example, Heyman, Addiction, 'Quitting Drugs'.

²⁴For example, Anthony and Helzer, 'Syndromes of Drug Abuse'; Stinson et al., 'Comorbidity'.

²⁵ For example, Biernacki, *Pathways from Heroin Addiction*; Waldorf, 'Natural Recovery'; Waldorf, Reinarman, and Murphy, *Cocaine Changes*.

the many correlates of heavy drug use, such as mental disorders, a sedentary lifestyle, unemployment and low educational achievement. Of course, researchers try to control for these variables, but it is not practical to do so, and it may prove impossible to control for the potentially highly perverse effects of the lifestyle that accompanies heavy drug use. Thus, animal studies are particularly valuable sources of information about the relationship between brain, drug use and behavior.

In a frequently cited and representative study, Robinson and Kolb and their colleagues at the University of Michigan and University of Lethbridge trained rats to self-administer cocaine intravenously.²⁶ After about three weeks of daily sessions, during which cocaine self-administration increased about three-fold, the rats were sacrificed and brain regions that are analogous to those likely to play a role in human drug use were evaluated histologically. The stained brain slices showed increases in dendritic branching in the nucleus accumbens, which is a key component in reward pathways, and they showed changes in the dendrites of neurons in the prefrontal cortex, which the authors described as 'misshapen' and 'neuropathological'. As the prefrontal cortex in humans is involved in judgment and decision, and the nucleus accumbens is often described as a 'pleasure center', Robinson and his colleagues concluded that the drug-induced anatomical changes were a 'recipe for addiction'. What they assumed was that the increased dendritic branching in the accumbens magnified the reward value of cocaine, whereas the 'pathological' changes in the prefrontal cortex compromised decision making so the 'ability to make judgments about the future consequences of continued drug-taking becomes more and more impaired'.²⁷

Although Robinson and his colleagues drew motivational and cognitive conclusions from the brain slices, there were no behavioral tests of these ideas. For instance, if cocaine-induced neuroadaptations were in fact an anatomical 'recipe for addiction', then preference for cocaine should have increased, but this was not tested.

In a subsequent study, Serge Ahmed and his colleagues also trained rats to self-administer cocaine intravenously.²⁸ However, they included a procedure for testing changes in preference for cocaine. The study had two major findings. First, they were interested in developing a procedure that insured 'addicted rats'. To this end, they manipulated the contingency so that in a few weeks the rats were self-administering about three to four times as much cocaine a day as the rates in the Robinson et al. study (65–75 mg/kg/2 h and 17.5 mg/kg/h, respectively). This resulted in dose-dependent increases in locomotion, which are correlated with long-lasting changes in midbrain

²⁶Robinson et al., 'Cocaine Self-Administration'.

²⁷Ibid., 264.

²⁸Lenoir et al., 'Intense Sweetness'.

dopamine function and which have been interpreted as key neuroadaptations in the transition from cocaine use to cocaine addiction.²⁹ Second, the Ahmed group tested preference for cocaine relative to saccharin. The rats preferred saccharin, their history of heavy cocaine use and motoric signs of addiction notwithstanding. Both before and after weeks of cocaine binging, preference for saccharin relative to cocaine was typically above 75%. The researchers then arranged conditions that would decrease preference for saccharin, such as greater delays and work requirements for the nondrug reinforcer. As expected, these measures increased preference for cocaine (by devaluing saccharin), but even under these conditions, the rats never chose cocaine more than 50% of the time.³⁰

These last two manipulations are relevant to the claims that addictive drugs interfere with decision-making capacity. Extra delays and response requirements weakened preference for saccharin. This implies that the rats remained sensitive to quantitative changes in the consequences of their behavior—despite the huge amounts of cocaine they had ingested. In sum, no matter how much cocaine the rats consumed, their preference for cocaine never exceeded their preference for saccharin, and, similarly, despite weeks of heavy cocaine ingestion, the rats remained sensitive to the consequences of their behavior. To help put these results in context, it is useful to point out that saccharin is not a particularly strong reinforcer for rats. For instance, in the control conditions for a study on alcohol self-administration, rats strongly preferred a weak sucrose solution (1.25%) to the most strongly preferred saccharin solution.³¹ Thus, cocaine changes the brain, but in a study that actually tested whether these changes affected preference for cocaine, the results were negative.

IX.ii. Is there a dose–response relationship between drug use and the persistence of addiction?

Although much of the support for the disease interpretation of addiction is based on animal studies,³² we could dismiss Lenoir et al.'s study on the grounds that it is about rats not humans. However, a recent report on the likelihood of remission from illegal and legal drug dependence provides results that test whether the rat cocaine/saccharin preference tests are relevant to the understanding of addiction in humans.³³

²⁹For example, Robinson and Berridge, 'Addiction'; Hyman, Malenka, and Nestler, 'Neural Mechanisms of Addiction'.

³⁰Lenoir et al., 'Intense Sweetness'.

³¹Heyman, 'Preference for Saccharin'.

³²For example, Robinson and Berridge, 'Addiction'.

³³Lopez-Quintero et al., 'Probability and Predictors'.

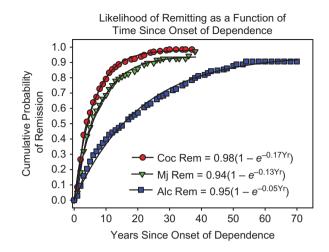


Figure 4. The cumulative probability of remitting from dependence as a function of time since the onset of dependence.

Note: I fit the equations on the basis of the assumption that each year a constant proportion of those still dependent would remit. The rate constants reveal that individuals who were dependent on cocaine were more than 3 times as likely to remit as individuals dependent on alcohol. A surprising feature of these results is that the likelihood of remitting was independent of onset time, which implies that remitting was also independent of how much drug has been consumed. *Source:* National Epidemiological Survey on Alcohol and Related Conditions (Lopez-Quintero et al., 2011).

Dose–response curves are a hallmark of pharmacological effects. Indeed, dose–response curves are the standard way for establishing that a chemical agent produced a physiological effect. Thus, if drugs change the brain in ways that undermine the capacity to say no to drugs, there should be a dose–response relationship between years of drug use and the likelihood of remitting from dependence. Figure 4 tests this prediction.³⁴ The graphs show remission rates in the most recent and largest nationwide survey of addictive disorders in the United States (National Epidemiological Survey on Alcohol and Related Disorders, NESARC).³⁵ The smooth curve is based on the idea that each year a constant proportion (of those who were still addicted) remitted, independent of how long they had been using drugs.³⁶

The NESARC researchers used a semi-structured interview schedule that included questions about the timing of psychiatric symptoms. For example, for those participants who reported that they had used an addictive drug,

³⁴Heyman, 'Quitting Drugs'; Lopez-Quintero et al., 'Probability and Predictors'.

³⁵Grant and Dawson, 'National Epidemiological Survey on Alcohol'.

³⁶Heyman, 'Quitting Drugs'.

there was a series of follow-up questions regarding if and when the symptoms of dependence became evident and their time course. Figure 4 summarizes the result for the relationship between time since the onset of dependence and remission for cocaine, marijuana and alcohol.³⁷ On the *x*-axis is the amount of time in years since the onset of dependence. On the *y*-axis is the cumulative probability of remission. The fitted curves are negative exponentials, based on the assumption that each year the likelihood of remitting remained constant, independent of the onset of dependence. Since exposure to addictive drugs is necessarily correlated with years of dependence, the curve is in effect the null hypothesis for a pharmacological role in remission. The results fit the null hypothesis virtually perfectly. For every drug the probability of remitting was constant and independent of time since onset, that is to say independent of exposure to the addictive drug.

However, the rates of remission differed. The likelihoods of quitting cocaine and marijuana were much higher than the likelihood of quitting alcohol. The pattern indicates that drug availability played a large role in remission.

X. Discussion

Figures 1-3 show that principles that apply to all choices can produce the highly excessive consumption patterns that are characteristic of addiction The empirical data reveal that cocaine induced neural changes in the brain did not influence preference and that epidemiological data fail to reveal a doseresponse relationship between drug consumption and the persistence of drug use. Moreover, most addicts quit using drugs at clinically significant levels, and do so without professional help. That is, they voluntarily quit, even after years of drug use. Thus, logic and observation support the thesis that addiction, although a pathological form of behavior, does not reflect an underlying disease state. Rather the simplest interpretation of the data is that the etiology of problem drug use involves the manner in which the principles of choice interact with substances and activities that have the properties listed earlier: the capacity to undermine the value of competing commodities and activities, few if any substitutes, delayed costs, and so on. There are of course other factors, including individual differences and social conditions that favor local as opposed to global bookkeeping. However, the key point is that these factors combine with the basic rules of choice, Principles 1-3, to produce addiction. As is the case with other natural disasters, addiction emerges as a function of interacting parts according to general rules that apply to all behavior.

Understanding a problem does not necessarily lead to a solution. We cannot prevent hurricanes. However, we can make natural disasters yet worse by misdiagnosing them, as when witches and sinners are blamed for famines and

³⁷Carlos Blanco kindly made the data available, and versions of this graph without the fitted equations are in Lopez-Quintero et al., 'Probability and Predictors'.

floods. Similarly, we may be making addiction and the problems caused by addiction worse by blaming poor choices on a disease that does not exist. For instance, the natural consequence of a series of poor choices is a series of poor outcomes, but the disease interpretation shields drug users from the consequences of their poor choices. Thus, it seems reasonable to ask whether the disease interpretation of addiction is actually doing more harm than good, whether both addicts and society would do better if addiction were identified as an issue of choices and consequences.

References

- Ainslie, G., and J. Monterosso. 'Hyperbolic Discounting as a Factor in Addiction: A Critical Analysis'. In *Choice, Behavioural Economics and Addiction*, ed. R.E. Vuchinich and N. Heather, 35–61. Amsterdam: Pergamon/Elsevier Science Inc., 2003.
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders:* DSM-IV. 4th ed. Washington, DC: American Psychiatric Association, 1994.
- Anthony, J. C., and J. E. Helzer. 'Syndromes of Drug Abuse and Dependence'. In *Psychiatric Disorders in America: The Epidemiologic Catchment Area Study*, ed. L. N. Robins and D. A. Regier, 116–54. New York: Free Press, 1991.
- Becker, G. S., and K. M. Murphy. 'A Theory of Rational Addiction'. Journal of Political Economy 96 (1988): 675–700.
- Biernacki, P. Pathways from Heroin Addiction: Recovery without Treatment. Philadelphia, PA: Temple University Press, 1986.
- Conway, K. P., W. Compton, F. S. Stinson, and B. F. Grant. 'Lifetime Comorbidity of DSM-IV Mood and Anxiety Disorders and Specific Drug Use Disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions'. *Journal of Clinical Psychiatry* 67 (2006): 247–57.
- Davison, M., and D. McCarthy. *The Matching Law: A Research Review*. Hillsdale, NJ: Lawrence Erlbaum, 1988.
- Goldstein, R. Z., A. D. Craig, A. Bechara, H. Garavan, A. R. Childress, M. P. Paulus, and N. D. Volkow. 'The Neurocircuitry of Impaired Insight in Drug Addiction'. *Trends in Cognitive Sciences* 13 (2009): 372–80.
- Grant, B. F., and D. A. Dawson. 'Introduction to the National Epidemiologic Survey on Alcohol and Related Conditions'. Alcohol Research & Health 29 (2006): 74–8.
- Herrnstein, R. J. The Matching Law: Papers in Psychology and Economics. Ed. H. Rachlin and D. Laibson. Cambridge, MA: Harvard University Press, 1997.
- Herrnstein, R. J. 'On the Law of Effect'. Journal of the Experimental Analysis of Behavior 13 (1970): 243–66.
- Herrnstein, R. J., G. F. Loewenstein, D. Prelec, and W. Vaughan. 'Utility Maximization and Melioration: Internalities in Individual Choice'. *Journal of Behavioral Decision Making* 6 (1993): 149–85.
- Herrnstein, R. J., and D. Prelec. 'A Theory of Addiction'. In *Choice over Time*, ed. G. Loewenstein and J. Elster, 331–60. New York: Russell Sage, 1992.
- Heyman, G. M. Addiction: A Disorder of Choice. Cambridge, MA: Harvard University Press, 2009.
- Heyman, G. M. 'Preference for Saccharin Sweetened Alcohol Relative to Isocaloric Sucrose. *Psychopharmacology* 129 (1997): 72–8.
- Heyman, G. M. 'Quitting Drugs: Quantitative and Qualitative Features'. Annual Review of Clinical Psychology 9 (2013): 29–59.

- Heyman, G. M., and L. Tanz. 'How to Teach a Pigeon to Maximize Overall Reinforcement Rate'. Journal of the Experimental Analysis of Behavior 64 (1995): 277–97.
- Hyman, S. E., R. C. Malenka, and E. J. Nestler. 'Neural Mechanisms of Addiction: The Role of Reward-related Learning and Memory'. Annual Review of Neuroscience, 20 (2006): 565–98.
- Kalivas, P. 'The Glutamate Homeostasis Hypothesis of Addiction'. Nature Reviews Neuroscience, 10 (2009): 561–72.
- Kalivas, P., and C. O'Brien. 'Drug Addiction as a Pathology of Staged Neuroplasticity'. *Neuropsychopharmacology* 33 (2008): 166–80.
- Kessler, R. C., P. Berglund, O. Demler, R. Jin, K. R. Merikangas, and E. E. Walters. 'Lifetime Prevalence and Age-of-onset Distributions of DSM-IV Disorders in the National Comorbidity Survey Replication'. *Archives of General Psychiatry* 62 (2005): 593–602.
- Kessler, R. C., W. T. Chiu, O. Demler, K. R. Merikangas, and E. E. Walters. 'Prevalence, Severity, and Comorbidity of 12-month DSM-IV Disorders in the National Comorbidity Survey Replication'. Archives of General Psychiatry 62 (2005): 617–27.
- Kudadjie-Gyamfi, E., and H. Rachlin. 'Temporal Patterning in Choice among Delayed Outcomes'. Organizational Behavior and Human Decision Processes 65 (1996): 61–7.
- Lenoir, M., F. Serre, L. Cantin, and S. H. Ahmed. 'Intense Sweetness Surpasses Cocaine Reward'. PLoS One 2, no. 8 (2007): e698.
- Leshner, A. I. 'Addiction is a Brain Disease, and it Matters'. Science 278 (1997): 45.
- Lopez-Quintero, C., D. S. Hasin, J. P. de los Cobos, A. Pines, S. Wang, B. F. Grant, and C. Blanco. 'Probability and Predictors of Remission from Life-time Nicotine, Alcohol, Cannabis or Cocaine Dependence: Results from the National Epidemiologic Survey on Alcohol and Related Conditions'. *Addiction* 106 (2011): 657–69.
- McLellan, A. T., D. C. Lewis, C. P. O'Brien, and H. D. Kleber. 'Drug Dependence, a Chronic Medical Illness: Implications for Treatment, Insurance, and Outcomes Evaluation'. *Journal* of the American Medical Association 284 (2000): 1689–95.
- Mukherjee, S. The Emperor of All Maladies. New York: Scribner, 2010.
- O'Brien, C. P., and A. T. McLellan. 'Myths about the Treatment of Addiction'. *Lancet* 347 (1996): 237–40.
- Quenqua, D. 'Rethinking Addiction's Roots, and its Treatment'. New York Times, July 10, 2011. http://www.nytimes.com/2011/07/11/health/11addictions.html (accessed October 29, 2012)
- Robinson, T. E., and K. E. Berridge. 'Addiction'. Annual Review of Psychology 54 (2003): 25-53.
- Robinson, T. E., G. Gorny, E. Mitton, and B. Kolb. 'Cocaine Self-Administration Alters the Morphology of Dendrites and Dendritic Spines in the Nucleus Accumbens and Neocortex'. *Synapse* 39 (2001): 257–66.
- Spanagel, R., and M. Heilig. 'Addiction and its Brain Science'. Addiction 100 (2005): 1813-22.
- Stinson, F.S., B. F. Grant, D. Dawson, W. J. Ruan, B. Huang, and T. Saha. '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 (2005): 105–16.
- Vaughan, W. 'Melioration, Matching, and Maximization'. Journal of the Experimental Analysis of Behavior 36 (1981): 141–9.
- Waldorf, D. 'Natural Recovery from Opiate Addiction: Some Social-Psychological Processes of Untreated Recovery'. *Journal of Drug Issues* 13 (1983): 237–79.
- Waldorf, D., C. Reinarman, and S. Murphy. Cocaine Changes: The Experience of Using and Quitting. Philadelphia: Temple University Press, 1991.
- Warner, L. A., R. C. Kessler, M. Hughes, J. C. Anthony, and C. B. Nelson. 'Prevalence and Correlates of Drug Use and Dependence in the United States. Results from the National Comorbidity Survey'. Archives of General Psychiatry 52 (1995): 219–29.
- Williams, B. A. 'Reinforcement, Choice, and Response Strength'. In Stevens' Handbook of Experimental Psychology, Learning and Cognition, 2nd ed., ed. R. C. Atkinson, R. J. Herrnstein, G. Lindzey, and R. D. Luce, 167–244. New York: John Wiley, 1988.