23
ONE CHEER FOR THE BRAIN-DISEASE INTERPRETATION OF ADDICTION

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Introduction

A minimum criterion for a useful scientific model of addiction is that it makes accurate predictions of drug use. According to this barebones standard, the brain-disease model of addiction fails. As documented in this chapter and elsewhere, its predictions are simply wrong (Klingemann et al., 2010; Robins, 1993). However, as discussed briefly in this chapter, it has prompted interesting research on drugs, brain, and behavior. Hence, in the spirit of E.M. Forster's "Two Cheers for Democracy," I think the right number of plaudits for the idea that addiction is a brain disease is "one." Also, and this is a matter of personal preference, I believe the idea that addiction is a brain disease should be referred to as an "interpretation." Models typically involve propositions that can be logically arranged to yield novel predictions or corollaries, whereas the idea that addiction is a brain disease contains no such propositions. Rather, it is linked to a set of empirical claims, three of which I test in this chapter: individuals who meet the criteria for addiction are involuntary drug users; addiction is a chronic state; however, abstinence, which is distinguished from recovery or cure, is possible with professional (lifelong) help. Each fails reasonable empirical tests.

What is at stake: a sampling

Human nature

Is it possible for voluntary behavior to be consistently self-destructive (akrasia)? Socrates famously said "no." Addiction provides the means to test Socrates's conclusion. It is a pattern of persistent, self-destructive behavior. Everyone agrees that addiction starts as voluntary drug use, so that the issue is: do addicts also voluntarily quit using? This is testable, which is to say, we can empirically evaluate whether Socrates's observation is true. (See Heather (2017) for a more complete discussion of addiction as akrasia.)

Expectations and outcomes

The Pygmalion myth and the research it has spawned attest to the importance of expectations on psychological phenomena. For example, negative expectations heighten the intensity of post-surgery pain and its disruptive effects, whereas positive expectations have just the opposite effect (e.g., Peerdeman et al., 2016; Sobol-Kwapinska et al., 2016). Thus, it is important to ensure that the brain-disease claim that addiction is a chronic, relapsing state, characterized by involuntary drug use, is true. No one wants drug users to fail to take positive actions because they falsely believe their drug use is not controllable.

Clinical practices and outcomes

Supporters of the brain-disease interpretation promote pharmacological interventions, whereas choice model interventions focus on expectations, rewarding alternatives to drugs, and social skills that preclude

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heavy drug use. These are not mutually exclusive approaches. The brain is the organ of choice so that, in principle, pharmacological treatments could help promote better decision making. Conversely, supporters of the brain-disease interpretation have no reason to reject efforts which expand the behavioral repertoire of heavy drug users. However, in recent years, National Institute of Drug Abuse (NIDA) officials have been among the most vigorous promoters of the brain-disease interpretation of addiction, and, accordingly, they put much more emphasis on pharmacological interventions than on choice-based approaches (e.g., Volkow & Koob, 2015). Thus, testing the brain-disease interpretation has implications for federal support for research on interventions and the sort of interventions that are available for those dependent on drugs.

What is not at stake

Punishment vs. treatment is not the issue

The claim that supporting the disease interpretation amounts to supporting treatment over punishment is a misleading, simplistic argument, based on a false distinction. There is a host of ways to influence the behavior of others, including education, persuasion, providing alternatives, promoting role models, etc. For instance, Alcoholics Anonymous (AA), which is neither medical nor punitive, helps alcoholics stay sober. Indeed, data presented in this chapter show that, of the available interventions, it was the one that had the highest correlation with remission. As described elsewhere, it works by offering positive role models, new ways of thinking, the opportunity to help others, and social activities that do not involve alcohol (e.g., Heyman, 2009; Kelly, 2017; Vaillant, 2005).

Whether drugs change the brain is not the issue

Addictive drugs are intoxicating; hence, they change the brain both in the short and long terms. No one can dispute this. However, the question is not whether drugs change the brain – but whether they change the brain such that drug use is no longer voluntary.

That drug use has antecedents is not the issue

As discussed in the section on the conceptual basis of the brain-disease interpretation, the claim that drug use has determinants does not make addiction a disease. Choices have antecedents and are rooted in the brain. Accordingly, they reflect genetic factors, early environment, and a host of influences that no one can possibly control. Thus, that addiction has antecedents is not in itself evidence of a disease. To be sure, there are differences between voluntary and involuntary behavior, but the supposed distinction between "free" and "determined" behaviors is not one of them.

The claim that no one chooses to be an addict is not the issue

One of the arguments in favor of the brain-disease model is that "no one chooses to be an addict." With a few exceptions, this is true. (Some individuals are on record for saying they wanted to be an addict, e.g., "Linda" in Zinberg et al., 1977.) Rather, addiction emerges as a function of a series of choices, not one of which is the decision to be an addict. For example, a novice smoker becomes an addicted smoker one cigarette at a time. Conversely, one day of not smoking does not turn a current smoker into an ex-smoker. Many outcomes in life are a function of a series of small choices, rather than of just one momentous decision (see Prelec and Herrnstein [1991] for a discussion of this important point).

Drugs and addiction: a schematic outline

A brief outline of some key features of addictive drugs and of addiction will help provide a context for evaluating the claims of the brain-disease interpretation.

Addictive drugs

Drugs act on neurons, binding to the same receptor sites as do the endogenous neurotransmitters that are integral to the physical basis of all thought, emotion, and action. However, drug doses can be arbitrarily large, whereas the circulating neurotransmitters are limited to the pico and nanogram level (e.g., Lechin et al., 2004). As a result, drugs can produce dramatic psychological effects. These include intoxication, hallucinations, unique sensory experiences, and intense waves of pleasure. The following comments from heroin users offer some insight into the subjective effects of addictive drugs (from Heyman, 2009, pp. 46–48).

Subjective effects

1 It was cool . . . the ultimate high.
2 And then came a surge of astonishing pleasure . . . it makes you feel everything is fine when it isn't.
3 warm, calm, dreamy, filling me up with a sensation . . . I'd never felt before.
4 I was invincible, without the energy of being invincible . . . . People try to put into words the feeling smack brings you . . . that's just the problem . . . it doesn't . . . . It was the most intense nothingness there ever was.

Addictive drugs produce these remarkable sensations without triggering the normal satiating processes which typically accompany appetitive behaviors. In contrast to food, heroin and cocaine do not fill you up. Rather, what controls consumption are their toxic effects (e.g., passing out) and, short of incapacitation, the user's judgment – but judgment is what intoxication undermines.

Along with their unique subjective effects, addictive drugs have a unique hedonic profile that is matched by few non-drug experiences. The positive effects are virtually instantaneous, whereas their direct negative effects, such as hangovers and withdrawal, are delayed by hours – or in the case of drug-related diseases such as lung cancer and cirrhosis of the liver, the delays are numbered in years and may not occur at all. The socially mediated negative effects, such as problems at home or work, can be delayed by weeks, months, and even years.

In sum, addictive drugs offer an immediate, reliable source of pleasure and escape. Their costs come later or not at all. And they have few or no substitutes – just as, say, medical care has few or no substitutes. They have cornered the market on intoxication.

The natural history of addiction

Given the unique, intoxicating, and often highly pleasurable effects of addictive drugs, it is understandable that some individuals end up using them excessively. Initially, the positive effects of the drug dominate. The costs have yet to emerge, except perhaps for hangovers. However, as drug use becomes more frequent, the direct positive effects weaken (e.g., tolerance), and the direct negative effects strengthen (e.g., withdrawal symptoms). Also, in time, the indirect, socially mediated penalties mount, leading to problems with family, friends, and work. Some statements from heavy drug users help give life to this abstract account (cited in Heyman, 2009, pp. 52–54).

1 Heroin is something I have to take every day. When I don't get it I feel horrible . . . . I was never born to be swollen like this. I wasn't born to limp. I wasn't born for my bowels to lock for me . . .
2 You're so damn sick . . . and you have the remedy for all your problems right in your hand.

According to this outline, the penalties of heavy drug use eventually outweigh the benefits, yet drug use continues. As described elsewhere, this balance is not necessarily contradictory. There are well-established, research confirmed, quantitative choice principles that predict the persistence of activities that produce a mix of immediate positive outcomes and delayed negative outcomes (e.g., Ainslie, 2013; Herrnstein & Prelec, 1992; Heyman, 2009). Consequently, under some conditions, say when talking to a spouse or counselor, a long-term drug user can

sincerely declare, "I am going to quit"; but then, under other circumstances, say when meeting a drug buddy, he or she – just as sincerely – resumes use. This is often described as loss of control, but "ambivalence" fits just as well.

Some features of addiction that are often overlooked

Addiction's idioms

Idioms emerge spontaneously out of shared experience. You have to, in effect, be there to understand what "giving someone the cold shoulder" means. The two best known addiction idioms are "kicking the habit" and "going cold turkey." Both refer to quitting drugs, all at once, without professional assistance.

Heavy drug use requires planning

Although popular opinion views addicts as impulsive, regular heavy drug use requires planning and, under most circumstances, subterfuge. Illegal drugs are not freely available, which means that addicts typically face a variety of hurdles to maintain a regular habit. Legal drugs are easier to obtain, but, as with illegal drugs, they often need to be used secretly in order to avoid sanctions from friends, family, and coworkers. Secrecy and getting around hurdles require purposeful, planful action.

The brain-disease interpretation claims: are they valid?

The following quotations outline the brain-disease interpretation of addiction.

1 1997, Leshner: [a] metaphorical switch in the brain seems to be thrown as result of prolonged drug use. Initially, drug use is a voluntary behavior, but when that switch is thrown, the individual moves into the state of addiction characterized by compulsive drug seeking and use . . . . That addiction is tied to changes in brain structure and function is what makes it, fundamentally, a disease (p. 46).

2 2004, Volkow & Li: The aberrant behavioural manifestations that occur during addiction have been viewed by many as "choices" of the addicted individual, but recent imaging studies have revealed an underlying disruption to brain regions that are important for the normal processes of motivation, reward and inhibitory control in addicted individuals. This provides the basis for a different view: that drug addiction is a disease of the brain, and the associated abnormal behaviour is the result of dysfunction of brain tissue, just as cardiac insufficiency is a disease of the heart and abnormal blood circulation is the result of dysfunction of myocardial tissue (p. 963).

3 2006, Martin: I find it useful to conceptualize addiction as the cancer of behavior. "How else could one fathom the mother who buys cocaine for herself instead of food for her children . . ." (p. 2301).

4 2007: National Institute of Drug Abuse, *NIDAInfoFacts*: Drug addiction is a *brain disease* because the abuse of drugs leads to changes in the structure and function of the brain (emphasis in original).

5 2014, Volkow: However much we may wish that a person's choices were free in all instances, it is simply a fact that an addicted person's failures in the realm of choice are the product of a brain that has become greatly compromised – it is readily apparent when we scan their brains.

6 2015, Volkow: To explain the devastating changes in behavior of a person who is addicted, such that even the most severe threat of punishment is insufficient to keep them from taking drugs – where they are willing to give up *everything they care for* in order to take a drug – it is not enough to say that addiction is a chronic brain disease. What we mean by that is something very specific and profound: that because of drug use, a person's brain is no longer able to produce something needed for our functioning and that healthy people take for granted, *free will* (emphases in original).

7 1609, John Downame (a minister, spelling updated, cited in Heyman, 2009, pp. 98–99): they who addict themselves to this vice, do find it so sweet and pleasing to the flesh, that they are loath to part with it, and by long custom they turn delight into necessity . . . and however the manifold mischiefs into which they plunge themselves . . . against all rules of reason, they hold fast their conclusion, the come what come may, they will not leave their drunkenness.

Taken together, these remarks tell the following story: drug use starts off as voluntary; however, drug-induced changes of the brain transform voluntary drug use into involuntary (compulsive) use. Brain imaging results provide the proof that drug use is involuntary and also the proof of which brain regions cause compulsive drug use. Other evidence for the claim that the brain is in a disease state is that drug use continues even though it is patently destructive – to the drug user and those he or she is close to.

These claims have some truth to them, but they also contain critical logical gaps and empirical claims that upon testing do not hold up. First, consider the conceptual issues.

1 It is, of course, true that drugs change the brain, but this is not sufficient grounds for saying addiction is a disease (see, Lewis, 2017, for an interesting discussion of this issue). All experience that leads to changes in behavior changes the brain. Reading a novel changes the brain (Berns et al., 2013); learning to play an arpeggio changes the brain (Ungerleider et al., 2002); and, if you are a rat pup, getting licked and groomed by your mom changes your brain (Kaffman & Meaney, 2007). That the brain changes is not in itself proof of involuntary behavior. Conversely, animal research indicates that there are drug-induced changes in the brain that appear to have no effect on the animal's preferences for the drug in question. For example, self-administered cocaine at doses that produced marked changes in cortical dendritic structure (e.g., Robinson et al., 2001) failed to increase preference for cocaine relative to saccharin (e.g., Lenoir et al., 2007). Under a wide range of conditions, the rats preferred saccharin to cocaine, brain changes notwithstanding. This is surprising, because saccharin is non-caloric and a weak reinforcer relative to even tepid (1.25%) sucrose solutions (Heyman, 1997). As noted in the "What is at stake" section, the question is not whether drugs change brains, but whether they do so in ways that render drug use no longer voluntary – which is a behavioral question.

2 According to Volkow, the criterion for knowing that someone is a compulsive drug user is brain scans. She pushes back against the idea that addicts voluntarily choose to use drugs with reference to brain scans. This is not sensible. Compulsion and choice refer to behavioral phenomena. We can only tell whether an activity is voluntary by: (1) identifying the behavioral criteria for distinguishing voluntary from involuntary, and (2) then carrying out the designated behavioral tests. Related to this point, leading brain scan researchers have cautioned against the simplistic interpretation of brain scans that informs Volkow's comments (e.g., Logothetis, 2008; Poldrack, 2018). In a discussion of reward, titled "Brain Activity Doesn't Tell Us What Somebody Is Experiencing," Poldrack (Chen, 2018) points out that there is no one-to-one relationship between brain regions and psychological function:

> The question is, if we take somebody and we don't know what they're doing, but we see activity in that part of the brain, how strongly should we decide that the person must be experiencing reward? If reward was the *only* thing that caused that sort of activity, we could be pretty sure. But there's not really any part of the brain that has that kind of one-to-one relationship with a particular psychological state. So you can't infer from activity in a particular area what someone is actually experiencing . . . the remarkable complexity of the brain rules

> out untested claims about behavior based strictly on brain imaging.
>
> (emphasis in original)

3 Martin wants to call addiction a disease because addicts behave destructively, even when they hurt their own children. Volkow, in a very moving blog (2015), makes the same case. Both repeat the argument made by Downame in the early 17th century that habitual drunkenness is a disease because it is irrational (see earlier in this chapter). However, self-destructive – yet voluntary – behavior is a long-standing theme in poems, novels, movies, biographies, and history. Agamemnon exchanged the life of his daughter for the promise that his ships would safely leave port and more speedily carry out his plan to sack Troy. But once underway, he undermined his plans by demanding that Achilles, his greatest warrior, turn over his loveliest female slave. Achilles, furious at Agamemnon, pulls his troops from the war. In both instances, Agamemnon acted selfishly and, in doing so, caused and/or risked great harm to himself and others. Move forward, several millennia: Bill Clinton repeatedly risked his reputation and even his presidency for a string of fleeting sexual dalliances. Both were uniquely able leaders; their missteps, in contrast, were quite ordinary.

4 Related to these points, it is sometimes argued that a genetic predisposition for addiction implies that addiction is a disease (e.g., Miller & Chappel, 1991). This isn't simply a truism (given that we are biological creatures). Research reveals that addiction has a substantial heritability, particularly alcoholism (Palmer et al., 2019). However, heritability does not imply involuntary behavior. Political orientation and voting patterns also have substantial heritabilities (e.g., Dawes & Weinschenk, 2020; Hatemi et al., 2014), yet individuals acquire and often change their political beliefs, and in many countries, voting is the epitome of a "free choice." Put more generally, both voluntary and involuntary behavior have a genetic basis.

Voluntary and involuntary behavior: what's the difference?

Choices are subject to the same cause and effect relations as are other natural phenomena; they have antecedents. Not only do genes influence choice, as noted previously in this chapter in regards to voting, but so does home environment, childhood neighborhood, and a host of factors no individual could possibly control. Rather, the distinction between voluntary and involuntary behavior has to do with the type of causal relations.

Voluntary behaviors are guided by feedback; involuntary behaviors are elicited by stimuli

The behaviors that we call "voluntary" are those that can be influenced by such factors as values, the opinions of others, and costs and benefits. In contrast, the activities that we call "involuntary" are little influenced by their costs and benefits. They are ballistic, triggered by stimuli. For example, socially mediated reinforcers (e.g., disapproval, approval) can influence the frequency of winking and putting on rouge, but they do not affect the frequency of blinking and blushing. Given that all but the simplest activities are an amalgam of learned and innate factors, the distinction is a matter of degree. For example, a careful study of feeding in herring gulls, aptly titled "How an Instinct Is Learned" (Hailman, 1969), revealed that food begging in baby herring gulls emerged from the rich interplay of primitive behavioral tropisms and experience. Learning to forage food (from their parents) did not start from scratch. The author referred to the end product as a "behavioral mosaic." Thus, the issue is not the presence or absence of causal factors, but the kinds of causal relations. In general, voluntary acts are governed largely by expectations and feedback; involuntary acts are governed largely by eliciting stimuli.

There is much more to say about the distinction between voluntary and involuntary behavior, and the unique properties of human voluntary behavior, such as the ability to self-regulate desires, including drug cravings (Heyman, 2017). However, this is a

chapter about the brain-disease interpretation, and the distinction between elicited control and feedback control is sufficient for determining whether drug use in addicts is involuntary or voluntary.

Is addiction a chronic disorder, and how likely is unassisted recovery?

Next, I test two ideas central to the disease interpretation of addiction: (1) since there is no cure, addiction is a chronic, lifelong malady, but (2) addicts can remain abstinent – albeit not cured – with professional help. In particular, they cannot remit on their own. Promoters of the disease interpretation make these points as follows.

In an article in the *Lancet,* titled "Myths About the Treatment of Addiction," O'Brien and McLellan (1996) write that for most addicts, the expectation of a "cure" is unrealistic and that "[a]ddictive disorders should be considered in the category with other disorders that require life-long treatment" (p. 237). Leshner (1999) says much the same in an article outlining the brain-disease interpretation, published in *JAMA*. He writes: "Once addicted, it is almost impossible for most people to stop the spiraling cycle of addiction on their own without treatment" (p. 1315). In a 2018 article on the brain-disease interpretation, a psychiatrist who specializes in addiction combines the earlier views in two pithy sentences: "Like most chronic medical illnesses, there is no 'cure' for addiction. It needs to be managed and mitigated by continuous treatment" (Morse, 2018, p. 163).

A simple test of the claim that addiction is a chronic disorder is to determine if it lasts at least as long as other psychiatric disorders. The data are from two of the four major epidemiological surveys of psychiatric disorders in the U.S. population: the Epidemiological Catchment Area Study (ECA: Anthony & Helzer, 1991; Robins & Regier, 1991) and the National Comorbidity Study (NCS: Kessler et al., 1994; Warner et al., 1995). The researchers' goals were to determine the prevalence, persistence, treatment history, and correlates of psychiatric disorders in representative populations. Lee Robins, who helped usher in the scientific study of behavioral disorders, led the ECA.

Figure 23.1 compares the remission (recovery) rates for a number of the more common and/or well-researched psychiatric disorders. On the x-axis is type of disorder; on the y-axis is the percentage of those who once met the criteria for the identified disorder but no longer did so. (The surveys were retrospective.) "Substance use" refers to the abuse of or dependence on illegal drugs.

Although the two projects were conducted ten years apart by different researchers, the remission rates were quite similar – except for substance use disorders. The reasons are methodological. The ECA included abuse and dependence, whereas the NCS ignored those who were not dependent. In support of this methodological explanation, when the NCS researchers recalculated their results according to criteria that were more similar to those used by the ECA, the substance use remission rates decreased from 74% to 63%, much closer to the ECA value of 59%. Thus, the "discrepancies" between the ECA and NCS substance disorders confirm the reliability of their methods.

However, the important point for this chapter is that the remission rates for drug disorders were

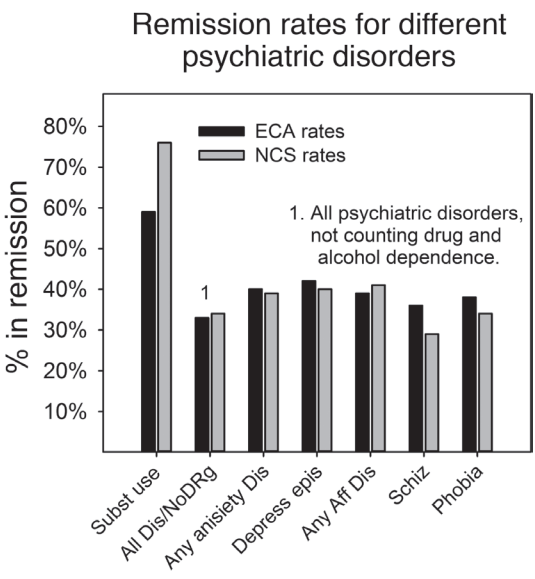

Figure 23.1 On the y-axis is the percentage of survey participants who no longer met the diagnostic criteria for the disorder listed on the x-axis. Drug disorders had the highest remission rates. The text explains why the NCS and ECA drug disorder remission rates differed. Other details are in Heyman (2009, p. 72).

about twice as high as those for other psychiatric disorders. Addiction was the "least chronic" psychiatric disorder. In support of this point, remission rates for dependence on illegal addictive drugs at age 30 and older exceed 50% (Heyman, 2013).

There are other methodological issues, such as the validity of self-reports and the possibility that those who remit may be more likely to participate in research projects than those who don't remit. These are important issues but, as discussed elsewhere (Heyman, 2009, 2013), they do not, I believe, have much influence on the results presented in this chapter.

What accounts for the high remission rates?

According to the brain-disease interpretation, remission depends importantly on treatment. Recall Leshner's comment (1999): "Once addicted, it is almost impossible for most people to stop the spiraling cycle of addiction on their own without treatment (p. 1315)." The next three graphs test this claim.

Differences in treatment and non-treatment groups

I compared remission rates for those who were ever in treatment with those who were never in treatment for alcohol, marijuana, and cocaine dependence. The National Epidemiological Survey of Alcohol and Related Conditions (NESARC: Grant & Dawson, 2006) survey provided the data. This is the largest psychiatric survey to date. The researchers interviewed more than 40,000 participants, with the goal of establishing a nationally representative sample of the non-institutionalized adult population of the United States. Accordingly, the subset of individuals who met the criteria for addiction is likely to be representative of American addicts in general. Treatment was broadly defined. It included self-help (such as AA), counseling, in-patient detoxification, psychiatric care, specialized addiction treatment, and so on. The comparisons took two forms. Survival curves and a multiple regression analysis which included controls for the "stage of treatment" and covariates. All the participants met the criteria for dependence. A previous research group plotted the cumulative remission rates from the NESARC survey, but without distinguishing between those in treatment and those not in treatment (Lopez-Quintero et al., 2011).

Covariates

The covariates included demographic, psychiatric, familial, and drug history measures that are correlated with drug use (e.g., Anthony & Helzer, 1991; Warner et al., 1995). Differences between the treatment and non-treatment groups were greatest for alcohol dependence. Of the 28 measures, 14 were statistically significant (*t*-tests). Those in treatment were more likely to be older, to be divorced, to have a family history of drug use, to have a personality disorder diagnosis, to have a family history of drug use, to report physical and emotional problems, to drink more heavily, and to have been dependent longer. Those in treatment were also more likely to have less education and a lower family income. For those dependent on marijuana and cocaine, the differences between the treatment and non-treatment groups were similar, but smaller and not statistically significant in 21 of the 28 comparisons.

The dependent measure for the statistical analyses was rate of remission; for example, one analysis tested if males or females remitted at a higher rate, holding all other variables constant at their mean. Treatment was "time differentiated" in that the rates of remission were calculated for years 0–3, 4–9, and 10 or more of treatment. For the analyses of treatment effects, the standard of comparison was the average rate of remission for those not in treatment.

The results were complex. In the first three years of treatment, those dependent on alcohol and marijuana remitted at significantly higher rates than the average rates for those not in treatment. In contrast, those who remained in treatment for cocaine dependence for four years or more remitted at a lower rate than those not in treatment, and the same was true for those who remained in treatment for ten years or more for marijuana dependence. Overall the remission rate was only significantly higher

for those in treatment for alcohol dependence. However, as shown next, the effect as measured in years of dependence was negligible. Although the treatment groups differed somewhat in terms of the covariates, the only covariate that had a significant correlation with remission rates for each of the three drugs was gender: females remitted at higher rates than males.

Table 23.1 lists how long dependence persisted for those in treatment and not in treatment. Figure 23.2, Figure 23.3, and Figure 23.4 show the probability of remission as a function of time since the onset of dependence and the onset of treatment. These analyses do not include the role of the covariates. On the x-axis is time since the onset of dependence. On the y-axis is the probability of still being dependent (i.e., of not remitting). The step functions for those not in treatment and the entire sample start at the onset of dependence. The step function for the treatment group starts at the average gap from the onset of dependence to the start of treatment. This gap was highly variable. For instance, its standard deviation was 21% greater than the mean interval. The data points were calculated according to the Kaplan-Meier procedure (e.g., Clark et al., 2003). This approach was motivated by how to best measure the effects of an intervention on survival, as in cancer research. The problem is that the study ends before the fate of all of the subjects is known (they are still surviving) or some have simply stopped participating in the research. For example, in the NESARC study, the duration of dependence is unknown for those participants who did not remit. The missing data are referred to as "right censored." The key assumption of the Kaplan-Meier approach is that the same survival function describes the subjects with known and unknown outcomes. For retrospective data, this is a reasonable assumption.

For those who were ever in treatment and those who were never in treatment, the step functions decrease to well below 0.50. This means that remission was the typical outcome. The filled circles show that once treatment started, the remission rates were roughly similar to those not in treatment, the details regarding different "stages" of treatment notwithstanding. For instance, once treatment starts, the step functions are roughly parallel. In keeping with the graphs, Table 23.1 shows that the median durations of dependence for those not in treatment were 15, five, and three years for alcohol, marijuana, and cocaine dependence, respectively, whereas the median durations of dependence from the start of treatment to remission were quite similar: 15, six, and four years, respectively. Of course, given that treatment did not start instantaneously with the onset of dependence, overall, those in treatment were dependent longer.

Table 23.1 Median duration of dependence for those ever in treatment and those never in treatment

	Alcohol (1,000/3,800)[a]	**Marijuana (150/450)**	**Cocaine (150/200)**
Age at interview	38.2 (0.23)[c]	32.3 (0.51)	37.5 (0.65)
Average gap between onset of dependence and start of treatment (years)	6.54 (7.43)	4.0 (5.15)	3.74 (4.53)
Overall median duration of dependence [b]	16 (15–17)[d]	5 (5–6)	4 (4–5)
Median duration of dependence for those not in treatment[b]	15 (14–17)	5 (4–6)	3 (3–4)
Median duration of dependence for those in treatment, counting pre-treatment period[b]	18 (17–20)	9 (6–11)	7 (5–9)
Median duration from start of treatment to remission[b]	15 (13–16)	6 (4–8)	4 (2–6)

[a] Treatment/no treatment participants. These are rounded rather than exact numbers, according to the Census Bureau restrictions See Acknowledgments regarding U.S. Census Bureau.
[b] Median duration according to Kaplan-Meier analysis (see text)
[c] Standard deviation (years)
[d] 95% confidence interval

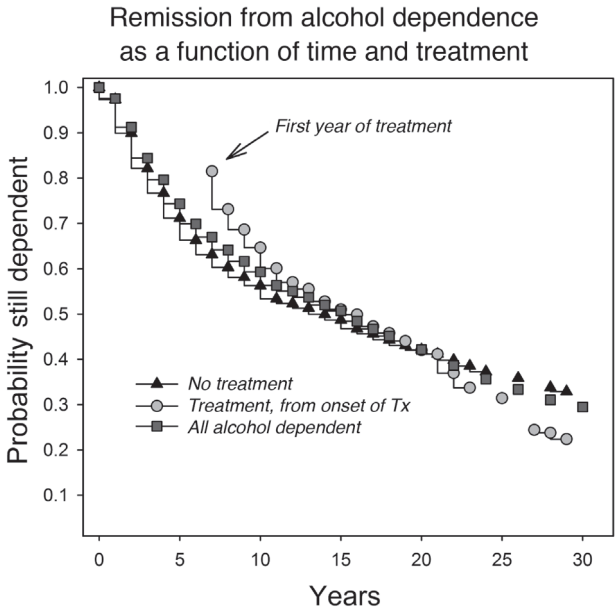

Figure 23.2 Kaplan-Meier survival curves for alcohol dependence. The x-axis is years since the onset of dependence. The y-axis shows the probability of still being dependent (not remitting). The step function for those who entered treatment starts at the average number of years separating the onset of dependence from the onset of treatment. The National Epidemiological Survey on Alcohol and Related Conditions (NESARC) collected the data (e.g., Grant & Dawson, 2006), and the U.S. Census Bureau reviewed the data to insure that no confidential information is disclosed. As noted in the Acknowledgments, "any opinions and conclusions regarding these data are those of the author and do reflect the views of the U.S Census Bureau." See the text for how the Kaplan-Meier curves are calculated and basic features of the NESARC survey. Sample sizes were roughly 1,000 for those ever in treatment and 3,800 for those never in treatment.

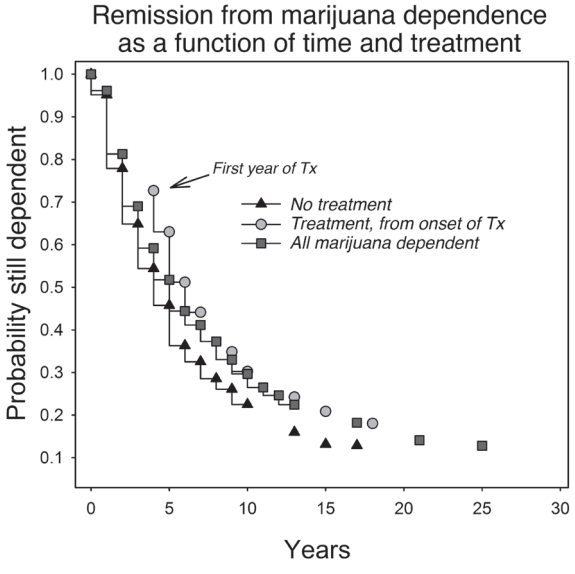

Figure 23.3 Kaplan-Meier survival curves for marijuana dependence. As in Figure 23.2, the x-axis is years since the onset of dependence; the y-axis shows the persistence of dependence; and the step function for those who entered treatment starts at the average number of years separating the onset of dependence from the onset of treatment. For other details see Figure 23.2 caption and the text. The treatment group numbered approximately 150, and the non-treatment group numbered approximately 450.

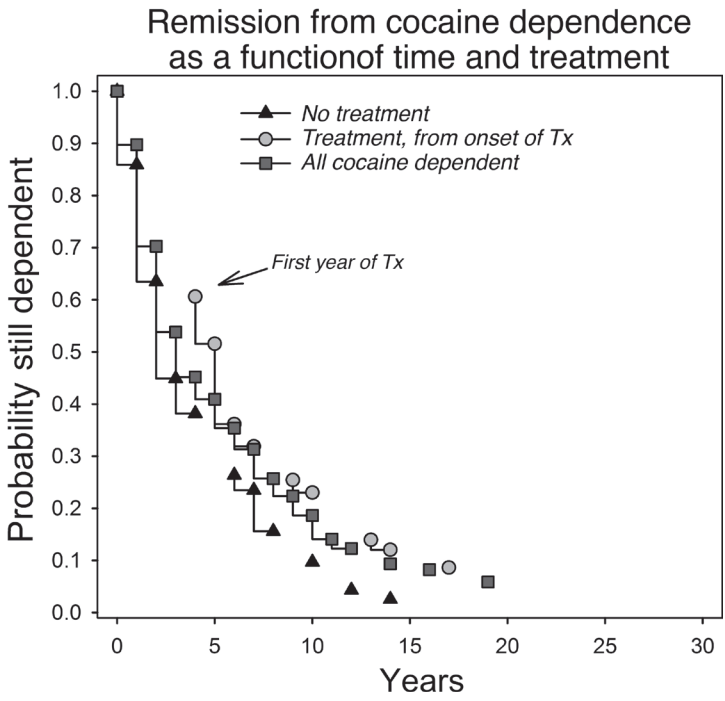

Figure 23.4 Kaplan-Meier survival curves for cocaine dependence. The axes and data source are the same as in Figures 23.2 and 23.3. See Figure 23.2 and text for description of the graph, the Kaplan-Meir procedure, and data source. The treatment group numbered approximately 150, and the non-treatment group numbered approximately 200.

There are two ways of looking at these results. As measured by overall years of dependence, the step functions and Table 23.1 reveal that treatment had little effect. Even with starting the clock at the start of treatment, those in treatment remained dependent about as long as those not in treatment. However, for those who entered treatment, remission rate was zero prior to the start of treatment. From this perspective, treatment made a big difference. Remission rates jumped from zero to a level that was initially (0–3 years) higher than the average remission rate for those not in treatment. Perhaps, then, there are two kinds of addicts: those who recover on their own and those who recover as a function of treatment. However, the covariates provide few clues as to the source of this difference, assuming that it exists. Recall that the only covariate that predicted lower remission rates in the time differentiated regressions for all three drugs was being male. But, regardless of the factors that contributed to cutting back on drug use, most of those who were dependent on alcohol, marijuana, or cocaine remitted.

Treatment was a heterogeneous category including both self-help and various forms of professional care. Thus, it was of interest to see if the type of treatment made a difference. There was a sufficient number of participants to carry out this comparison for alcohol dependence. Those in treatment were divided into three groups: those who participated only in AA, those who participated in AA plus some other form of treatment, and those who participated in any treatment other than AA. Figure 23.5 shows the survival step functions for each group. Those in AA had the highest remission rate, those in anything but AA had the lowest remission rate. Wald chi-square tests that controlled for all the covariates revealed that these differences were statistically significant.

Thus, in contrast, to the claims of the brain-disease interpretation of addiction, remission did not

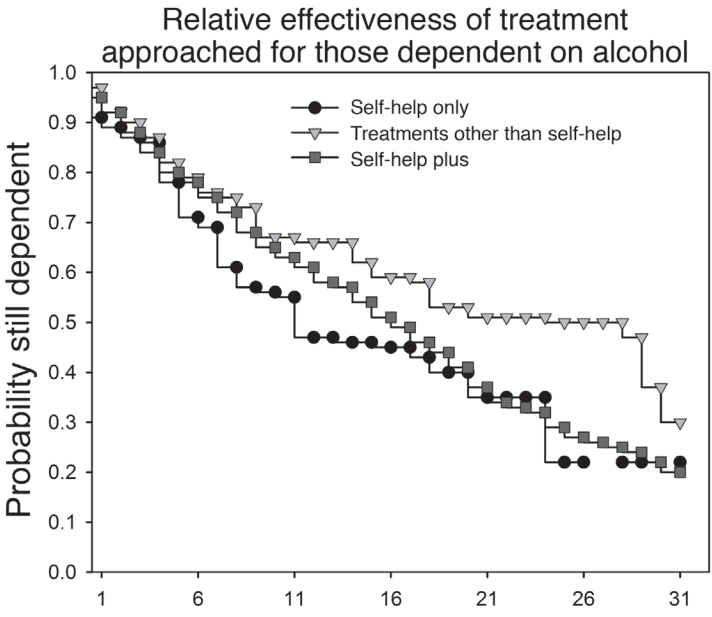

Figure 23.5 Kaplan-Meier survival curves for three alcohol dependence treatments: self-help (e.g., AA), any treatment other than self-help, and self-help along with additional treatments. Wald chi-square tests that included controls for the covariates revealed that remission rates for treatments that included and did not include self-help differed statistically ($p <= 0.05$ for self-help only ($n = 100$) vs. no self-help ($n = 200$) and $p < = 0.001$ for self-help plus ($n = 700$) vs. no self-help). See Figure 23.2 and the text for other information regarding Kaplan-Meier curves and the data.

depend on treatment, and – given that someone was in treatment – the most effective mode for the most prevalent form of dependence was organized self-help (AA), which is not a medical treatment.

What are the correlates of remitting?

If drug use is voluntary, then the correlates of quitting should include the costs and benefits of drug use, along with the various other factors that influence choices. Reviews confirm this prediction (e.g., Davis et al., 2016; Hawken & Kleiman, 2009; Heyman, 2013). For instance, the correlates of remission include marital status, changes in the legal standing of addictive drugs (e.g., the Harrison Narcotics Tax Act), increases in drug price, decreases in the acceptability of drug use, and moral and health concerns (e.g., Biernacki, 1986; Robins, 1993; Waldorf et al., 1992). Contingency management (Davis et al., 2016) and drug court follow-up studies (Hawken & Kleiman, 2009) show that the promise of parole, explicit rewards, and the chance of winning prizes encourage remission in drug users who have not responded positively to other types of interventions (see Higgins et al., 1994). Similarly, AA's techniques fit the choice model perfectly. It works by providing positive role models, positive social interactions that do not involve alcohol, the opportunity to help others as a sponsor, and encouraging its members to take stock of the costs of their drinking, as measured by the harm it has caused others (AA, 1939). In sum, the correlates of remission for those dependent on drugs are the correlates of choice.

This does not mean that all addicts will remit or that it is easy to quit. Quitting drugs involves giving up a visceral, immediate and reliable source of pleasure or escape in exchange for a future that is presumably better overall but at the moment of choice is abstract and uncertain. Moreover, the physical and social damage associated with years of drug use are likely to have undermined the value of activities that do not involve drugs, plus made them less

accessible. That is, heavy drug use perpetuates itself by weakening competing options.

The graphs tell a remarkable story. They show that the majority of those who were dependent on addictive drugs overcame the grave challenges posed by addiction, and did so regardless of how long they had been dependent.

Cocaine use, abstinence, and the brain

Several studies have shown that the density of prefrontal cortex gray matter is greater in former cocaine users than it is in current cocaine users (e.g., Connolly et al., 2013; Hanlon et al., 2011). Within-subject longitudinal studies show that these differences appear in well less than a year of abstinence (e.g., Moeller et al., 2012; Parvaz et al., 2017). These results are interesting and important. First, notice that the participants in these studies quit or greatly reduced their cocaine use prior to the abstinence-correlated gray matter increases. That is, the changes which were presumably wrought by cocaine did not prevent quitting cocaine. Similarly, the epidemiological studies revealed (indirectly) that the neural changes associated with cocaine use did not prevent remission. Second, the abstinence findings showcase the remarkable plasticity of the brain. Either abstinence reversed cocaine induced neural changes, and/or it triggered changes that were not directly related to cocaine but instead were driven by abstinence, such as the activities that comprise a healthier lifestyle. Which of these two factors plays the larger role has yet to be established, but however it turns out, the studies will provide new information on experience-dependent brain plasticity.

An alternative approach

One of the reasons that the brain-disease interpretation of addiction has won so many adherents – particularly among clinicians, the media, and public health bureaucrats – is that it was the only well-known, research-based account. This is changing. A number of researchers with backgrounds in the experimental study of choice have turned their attention to drug addiction, gambling, overeating, and related behavioral phenomena (e.g., Ainslie, 2013; Herrnstein & Prelec, 1992; Heyman, 2018; Rachlin et al., 2015). Their approach includes quantitative models that predict the persistence of choices that on balance yield negative returns but from moment to moment yield positive returns (so called "specious" rewards). This is precisely the temporal hedonic profile of long-term drug use. Moreover, the models predict that this pattern of behavior is most likely to occur when one of the options is a substance or activity that undermines the value of competing activities and does not satiate, two properties that apply best to addictive drugs. The choice models also predict that conditions can be arranged such that dependent drug users will choose non-drug alternatives. Drug courts (Hawken & Kleiman, 2009), contingency management interventions (Davis et al., 2016; Higgins et al., 1994), and Figures 23.2–23.5 of this chapter support this prediction. In sum, experimentally established, quantitative choice principles explain the emergence, persistence, and remission of drug dependence.

What's at stake, revisited

The "What's at stake" section at the beginning of this chapter listed three issues, one regarding human nature and the other two regarding practical applications. The first claim was that addiction provided an opportunity to test the assumption that individuals will not voluntarily engage in self-destructive behavior. This is a long-standing, widely held view, dating from the writings of early Greek philosophers. It is also one of the pillars of the disease interpretation of addiction. Early 17th century clergy concluded that habitual "drunkenness" was a symptom of disease because it was self-destructive, and 21st century clinicians and researchers make the same argument (e.g., Martin, 2006; Volkow, 2015). The data presented here and elsewhere (e.g., Toneatto et al., 1999) demonstrate that individuals who meet the criteria for dependence not only start using drugs voluntarily, but quit voluntarily. Thus, according to the addiction test, individuals do, in fact, voluntarily engage in self-destructive behavior. As described by research-based

choice theories (e.g., Ainslie, 2013; Herrnstein & Prelec, 1992; Rachlin et al., 2015), this happens one choice at a time, along the primrose path to addiction.

The practical implications of the observations and data reviewed in this chapter are that addiction is not a chronic, relapsing disease, Alcoholics Anonymous is an effective method for achieving recovery, and that many addicts remit according to methods that they have worked out for themselves – that is, without the ostensible help of experts. This is useful information. For those struggling with addiction, as well as their family, friends, and neighbors, it has to be encouraging to know that most addicts remit and that the paths to remission are accessible to all.

Recovery is a popular theme in fiction (e.g., Jean Valjean and Abel Magwitch). The remission graphs in this chapter tell the same story for many of those who became dependent on drugs. One can hope that as these results become better known, the survival analysis step functions that appear in future research reports will decline yet more sharply than those which appeared in these pages.

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References

Ainslie, G. (2013). Intertemporal bargaining in addiction. *Frontiers in Psychiatry*, *4*(5). http://doi.org/10.3389/fpsyt.2013.00063

Alcoholics Anonymous (1939). *Alcoholics Anonymous: The story of how many more than one hundred men have recovered from alcoholism* (1st ed.). Works Publishing Company.

Anthony, J. C., & Helzer, J. E. (1991). Syndromes of drug abuse and dependence. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorders in America: The epidemiologic catchment area study*. Free Press.

Berns, G. S., Blaine, K., Prietula, M. J., & Pye, B. E. (2013). Short- and long-term effects of a novel on connectivity in the brain. *Brain Connectivity*, *3*(6), 590–600. http://doi.org/10.1089/brain.2013.0166

Biernacki, P. (1986). *Pathways from heroin addiction: Recovery without treatment*. Temple University Press.

Chen, A. (2018, October 17). A neuroscientist explains the limits and possibilities of using technology to read our thoughts. *The Verge*. Retrieved May 26, 2020, from www.theverge.com/2018/10/17/17990876/russell-poldrack-new-mind-readers-neuroimagining-fmri-science-technology

Clark, T. G., Bradburn, M. J., Love, S. B., & Altman, D. G. (2003). Survival analysis part I: Basic concepts and first analyses. *British Journal of Cancer*, *89*(2), 232–238. https://doi.org/10.1038/sj.bjc.6601118

Connolly, C. G., Bell, R. P., Foxe, J. J., & Garavan, H. (2013). Dissociated grey matter changes with prolonged addiction and extended abstinence in cocaine users. *PLoS ONE*, *8*(3), e59645. https://doi.org/10.1371/journal.pone.0059645

Davis, D. R., Kurti, A. N., Skelly, J. M., Redner, R., White, T. J., & Higgins, S. T. (2016). A review of the literature on contingency management in the treatment of substance use disorders, 2009–2014. *Preventive Medicine*, *92*, 36–46. http://doi.org/10.1016/j.ypmed.2016.08.008

Dawes, C. T., & Weinschenk, A. C. (2020). On the genetic basis of political orientation. *Current Opinion in Behavioral Sciences*, *34*, 173–178. https://doi.org/10.1016/j.cobeha.2020.03.012

Grant, B. F., & Dawson, D. A. (2006). Introduction to the National Epidemiologic Survey on Alcohol and Related Conditions. *Alcohol Research & Health*, *29*(2), 74–78.

Hailman, J. P. (1969). How an instinct is learned. *Scientific American*, *221*(6), 98–106. http://doi.org/10.1038/scientificamerican1269-98

Hanlon, C. A., Dufault, D. L., Wesley, M. J., & Porrino, L. J. (2011). Elevated gray and white matter densities in cocaine abstainers compared to current users. *Psychopharmacology*, *218*(4), 681–692. http://doi.org.proxy.bc.edu/10.1007/s00213-011-2360-y

Hatemi, P. K., Medland, S. E., Klemmensen, R., Oskarsson, S., Littvay, L., Dawes, C. T., Verhulst, B., McDermott, R., Nørgaard, A. S., Klofstad, C. A., Christensen, K., Johannesson, M., Magnusson, P. K. E., Eaves, L. J., & Martin, N. G. (2014). Genetic influences on political ideologies: Twin analyses of 19 measures of political ideologies from five democracies and genome-wide findings from three populations. *Behavior Genetics*, *44*(3), 282–294. http://doi.org/10.1007/s10519-014-9648-8

Hawken, A., & Kleiman, M. (2009). Managing drug involved probationers with swift and certain sanctions: Evaluating Hawaii's HOPE: Executive summary. *NJC 2230444*. National Institute of Justice.

Heather, N. (2017). Addiction as a form of akrasia. In N. Heather & G. Segal (Eds.), *Addiction and choice: Rethinking the relationship* (pp. 133–150). Oxford University Press.

Herrnstein, R. J., & Prelec, D. (1992). A theory of addiction. In G. Loewenstein & J. Elster (Eds.), *Choice over time* (pp. 331–360). Russell Sage Foundation.

Heyman, G. M. (1997). Preference for saccharin-sweetened alcohol relative to isocaloric sucrose. *Psychopharmacology*, *129*(1), 72–78. http://doi.org/10.1007/s002130050164

Heyman, G. M. (2009). *Addiction: A disorder of choice*. Harvard University Press.

Heyman, G. M. (2013). Quitting drugs: Quantitative and qualitative features. *Annual Review of Clinical Psychology*, *9*, 29–59. http://doi.org/10.1146/annurev-clinpsy-032511-143041

Heyman, G. M. (2017). Do addicts have free will? An empirical approach to a vexing question. *Addictive Behaviors Reports*, *5*, 85–93. http://doi.org/10.1016/j.abrep.2017.02.001

Heyman, G. M. (2018). Deriving addiction: An analysis based on three elementary features of making choices. In H. Pickard & S. Ahmed (Eds.), *The Routledge handbook of the philosophy and science of addiction* (pp. 23–33). Routledge.

Higgins, S. T., Budney, A. J., Bickel, W. K., Foerg, F. E., Donham, R., & Badger, G. J. (1994). Incentives improve outcome in outpatient behavioral treatment of cocaine dependence. *Archives of General Psychiatry*, *51*(7), 568–576. http://doi.org/10.1001/archpsyc.1994.03950070060011

Kaffman, A., & Meaney, M. J. (2007). Neurodevelopmental sequelae of postnatal maternal care in rodents: Clinical and research implications of molecular insights. *Journal of Child Psychology and Psychiatry*, *48*(3–4), 224–244. http://doi.org/10.1111/j.1469-7610.2007.01730.x

Kelly, J. F. (2017). Is Alcoholics Anonymous religious, spiritual, neither? Findings from 25 years of mechanisms of behavior change research. *Addiction*, *112*(6), 929–936. http://doi.org/10.1111/add.13590

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H., & Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *51*(1), 8–19. http://doi.org/10.1001/archpsyc.1994.03950010008002

Klingemann, H. K., Sobell, M. B., & Sobell, L. C. (2010). Continuities and changes in self-change research. *Addiction*, *105*(9), 1510–1518. http://doi.org/10.1111/j.1360-0443.2009.02770.x

Lechin, F., Pardey-Maldonado, B., van der Dijs, B., Benaim, M., Baez, S., Orozco, B., & Lechin, A. E. (2004). Circulating neurotransmitters during the different wake-sleep stages in normal subjects. *Psychoneuroendocrinology*, *29*(5), 669–685. http://doi.org/10.1016/S0306-4530(03)00095-7

Lenoir, M., Serre, F., Cantin, L., & Ahmed, S. H. (2007). Intense sweetness surpasses cocaine reward. *PLoS ONE*, *2*(8), e698. https://doi.org/10.1371/journal.pone.0000698

Leshner, A. I. (1997). Addiction is a brain disease, and it matters. *Science*, *278*(5335), 45–47. http://doi.org/10.1126/science.278.5335.45

Leshner, A. I. (1999). Science-based views of drug addiction and its treatment. *JAMA*, *282*(14), 1314–1316. https://doi.org/10.1001/jama.282.14.1314

Lewis, M. (2017). Addiction and the brain: Development, not disease. *Neuroethics*, *10*(1), 7–18. https://doi.org/10.1007/s12152-016-9293-4

Logothetis, N. K. (2008). What we can do and what we cannot do with fMRI. *Nature*, *453*(7197), 869–878. http://doi.org/10.1038/nature06976

Lopez-Quintero, C., Hasin, D. S., de los Cobos, J. P., Pines, A., Wang, S., Grant, B. F., & Blanco, C. (2011). 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*(3), 657–669. http://doi.org/10.1111/j.1360-0443.2010.03194.x

Martin, P. (2006). [Review of the book *Cell biology of addiction*]. *New England Journal of Medicine*, *354*, 2301–2302. http://doi.org/10.1056/NEJMbkrev39780

Miller, N. S., & Chappel, J. N. (1991). History of the disease concept. *Psychiatric Annals*, *21*(4), 196–205. http://doi.org/10.3928/0048-5713-19910401-06

Moeller, S. J., Tomasi, D., Woicik, P. A., Maloney, T., Alia-Klein, N., Honorio, J., Telang, F., Wang, G., Wang, R., Sinha, R., Carise, D., Astone-Twerell, J., Bolger, J., Volkow, N. D., & Goldstein, R. Z. (2012). Enhanced midbrain response at 6-month follow-up in cocaine addiction, association with reduced drug-related choice. *Addiction Biology*, *17*(6), 1013–1025. http://doi.org/10.1111/j.1369-1600.2012.00440.x

Morse, E. D. (2018). Addiction is a chronic medical illness. *North Carolina Medical Journal*, *79*(3), 163–165. https://doi.org/10.18043/ncm.79.3.163

National Institute of Drug Abuse. (2007). *Understanding drug abuse and addiction*. Retrieved November 13, 2008, from www.nida.nih.gov/Infofacts/understand.html

O'Brien, C. P., & McLellan, A. T. (1996). Myths about the treatment of addiction. *Lancet*, *347*(8996), 237–240. https://doi.org/10.1016/s0140-6736(96)90409-2

Palmer, R. H. C., Brick, L. A., Chou, Y., Agrawal, A., McGeary, J. E., Heath, A. C., Bierut, L., Keller, M. C., Johnson, E., Hartz, S. M., Schuckit, M. A., & Knopik, V. S. (2019). The etiology of DSM-5 alcohol use disorder: Evidence of shared and non-shared additive genetic effects. *Drug & Alcohol Dependence*, *201*, 147–154. http://doi.org/10.1016/j.drugalcdep.2018.12.034

Parvaz, M. A., Moeller, S. J., d'Oleire Uquillas, F., Pflumm, A., Maloney, T., Alia-Klein, N., & Goldstein, R. Z. (2017). Prefrontal gray matter volume recovery in treatment-seeking cocaine-addicted individuals: A longitudinal study. *Addiction Biology*, *22*(5), 1391–1401. http://doi.org/10.1111/adb.12403

Peerdeman, K. J., van Laarhoven, A. I. M., Keij, S. M., Vase, L., Rovers, M. M., Peters, M. L., & Evers, A. W. M. (2016).

Relieving patients' pain with expectation interventions: A meta-analysis. *Pain*, *157*(6), 1179–1191. http://doi.org/10.1097/j.pain.0000000000000540

Poldrack, R. A. (2018). *The new mind readers: What neuroimaging can and cannot reveal about our thoughts*. Princeton University Press.

Prelec, D., & Herrnstein, R. J. (1991). *Preferences or principles: Alternative guidelines for choice*. Harvard Business School.

Rachlin, H., Safin, V., Arfer, K. B., & Yen, M. (2015). The attraction of gambling. *Journal of the Experimental Analysis of Behavior*, *103*(1), 260–266. https://doi.org/10.1002/jeab.113

Robins, L. N. (1993). Vietnam veterans' rapid recovery from heroin addiction: A fluke or normal expectation? *Addiction*, *88*(8), 1041–1054. http://doi.org/10.1111/j.1360-0443.1993.tb02123.x

Robins, L. N., & Regier, D. A. (1991). *Psychiatric disorders in America: The epidemiologic catchment area study*. Free Press.

Robinson, T. E., Gorny, G., Mitton, E., & Kolb, B. (2001). Cocaine self-administration alters the morphology of dendrites and dendritic spines in the nucleus accumbens and neocortex. *Synapse*, *39*(3), 257–266. https://doi.org/10.1002/1098-2396(20010301)39:3<257::AID-SYN1007>3.0.CO;2-1

Sobol-Kwapinska, M., Bąbel, P., Plotek, W., & Stelcer, B. (2016). Psychological correlates of acute postsurgical pain: A systematic review and meta-analysis. *European Journal of Pain*, *20*(10), 1573–1586. http://doi.org/10.1002/ejp.886

Toneatto, T., Sobell, L. C., Sobell, M., & Rubel, E. (1999). Natural recovery from cocaine dependence. *Psychology of Addictive Behaviors*, *13*(4), 259–268. http://doi.org/10.1037/0893-164X.13.4.259

Ungerleider, L. G., Doyon, J., & Karni, A. (2002). Imaging brain plasticity during motor skill learning. *Neurobiology of Learning and Memory*, *78*(3), 553–564. http://doi.org/10.1006/nlme.2002.4091

Vaillant, G. E. (2005). Alcoholics Anonymous: Cult or cure? *Australian & New Zealand Journal of Psychiatry*, *39*(6), 431–436. http://doi.org/10.1111/j.1440-1614.2005.01600.x

Volkow, N. D. (2014). *Addiction and free choice*. Retrieved January 5, 2015, from www.drugabuse.gov/about-nida/noras-blog/2014/02/addiction-free-choice

Volkow, N. D. (2015). *Addiction is a disease of free will*. Retrieved January 23, 2016, from www.drugabuse.gov/about-nida/noras-blog/2015/06/addiction-disease-free-will

Volkow, N. D., & Koob, G. (2015). Brain disease model of addiction: Why is it so controversial? *The Lancet Psychiatry*, *2*(8), 677–679. http://doi.org/10.1016/S2215-0366(15)00236-9

Volkow, N. D., & Li, T. K. (2004). Drug addiction: The neurobiology of behaviour gone awry. *Nature Reviews Neuroscience*, *5*(12), 963–970. http://doi.org/10.1038/nrn1539

Waldorf, D., Reinarman, C., & Murphy, S. (1992). *Cocaine changes: The experience of using and quitting*. Temple University Press.

Warner, L. A., Kessler, R. C., Hughes, M., Anthony, J. C., & Nelson, C. B. (1995). Prevalence and correlates of drug use and dependence in the United States: Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *52*(3), 219–229. http://doi.org/10.1001/archpsyc.1995.03950150051010

Zinberg, N. E., Harding, W. M., & Winkeller, M. (1977). A study of social regulatory mechanisms in controlled illicit drug users. *Journal of Drug Issues*, *7*(2), 117–133. http://doi.org/10.1177/002204267700700203