CHAPTER

8

A Case Study in Computational Psychiatry: Addiction as Failure Modes of the Decision-Making System

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Because addiction is so hard to define, the DSM-IV defined drug dependence and avoided the word *addiction* (DSM-IV-TR, 2000). However, more recent studies have suggested that addiction-like behaviors can underlie nondrug decision problems as well (Holden, 2001; Schüll, 2012;

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Robbins and Clark, 2015). But then we run into the problem of whether all continued behaviors are addictions. Do we really want to say that Brett Favre was "addicted" to football because he continued playing long after the game had damaged his body? Do we really want to say that Osip Mandelstam was "addicted" to poetry because he continued to write even after Stalin had sent him to the Gulag (where he eventually died)? To avoid this difficult definition, we will unask the question and instead concentrate on specific decision-making errors and relate that to problematic behaviors often categorized as addiction (Redish et al., 2008; Heyman, 2009, 2013; Redish, 2013).

Current models of psychiatry suggest that psychiatric disorders should be defined in terms of "harmful dysfunction" (Wakefield, 2007). This definition includes a scientific component (dysfunction) and a sociological component (harm). For example, illiteracy is harmful but is not usually considered a brain dysfunction. (On the other hand, dyslexia is both harmful and a brain dysfunction (Norton et al., 2015; Jaffe-Dax et al., 2015).) Synesthesia is due to a brain dysfunction but is not generally considered harmful (Cytowic, 1998). Treatment needs to be predicated on fixing those things that are harmful, but the appropriate treatment depends on the dysfunction. For example, most clinicians do not feel a need to treat synesthesia, but both dyslexia and illiteracy need treatment. Both of these problems require treatment, but because the causes are different, treatments for illiteracy and dyslexia will likely need to be different. In this chapter, we will make the case that addiction is a symptom, not a disease, and that because the underlying causes (the underlying dysfunctions) for addiction are varied the necessary treatments must be varied (Redish et al., 2008). We will make the case that rather than categorizing subjects in terms of their addiction (cocaine addiction, heroin addiction, gambling addiction), we should be defining them in terms of their decision-making dysfunctions (overvaluation, errors in expectation, reactions to anxiety). Lastly, we will argue that treatments should be guided by identifying the underlying dysfunction in an addict's decision-making circuitry to allow clinicians to individualize treatments.

A key concept that we will build this chapter on is that of a *vulnerability* or *failure mode*—a breakdown in a process due to a malfunctioning component. These terms come from the field of reliability engineering where one tries to identify the underlying breakdown that has caused a system-wide failure. A flat tire, for example, is a *failure mode* of automobiles (and bicycles) because a tire is a thin rubber tube filled with air. If that tube becomes punctured, then the air leaks out and the car is no longer riding on the normal air cushion. On the other hand, tank treads (which do not ride on air) are not susceptible to going flat (although they are vulnerable to being split). Just as cars and tanks have different failure modes that depend on their underlying mechanisms, so too do decision-making systems.

8.1 THE MACHINERY OF DECISION-MAKING

To understand how decision-making systems can go wrong, one needs to understand the fundamental mechanisms by which they work. Ultimately, decisions are about interactions with the world or changes in one's behavior that affect the world. For completeness, we will include both actions as visceral changes (heart rate, thermal regulation, hormone levels) as well as external, physical actions (pushing a button, lighting a cigarette, going to some location, signing on a dotted line). By this definition, ultimately, all decisions entail taking an action. At the point where we have defined decision as action selection, we are now in the domain of computational information processing—all decisions are, ultimately, a consequence of processing information about one's present circumstances (perception), information about one's past (memory), and information about one's needs/goals (motivation); however, as we will see below, these components do not have to be explicitly represented to be a part of the process—sometimes they can be hidden within the process itself.

Importantly, how information is stored changes how easy it is to access and how it generalizes to new situations (Redish and Mizumori, 2015). For example, if you are looking for a specific book to cite on a topic, it will be easiest to find if your bookshelf is sorted by a library catalog system, such as the Dewey decimal or Library of Congress system, rather than if you have sorted your books by size. However, if you are looking for a book to level out the table with a short leg, then sorting by size is going to get you to your target faster. This point is one of the main discoveries of computer science in the last century—data structures matter (Cormen et al., 1990).

In the same way that data structures matter in a digital computer, so too do the information processes that are being used to select an action. Current taxonomies have suggested that there are four key action-selection information processing systems that store (and generalize) information differently (Rangel et al., 2008; van der Meer et al., 2012): **Reflexes**, **Pavlovian action-selection systems**, **Procedural decision-making systems**, and **Deliberative decision-making systems**. Each of these systems uses different computational information processing mechanisms through different brain structures and thus has different failure modes (Montague et al., 2012; Redish, 2013).

- **Reflexes** select actions based on immediately available perceptions and have stored an appropriate action in the spinal circuitry (Sherrington, 1906; Eaton, 1984), which was learned by the species through genetic variation and selection (a genetic learning algorithm). Both the memory and the motivational components are hardwired into the circuitry through genetically controlled wiring.

- **Pavlovian action-selection systems** entail species-specific actions that one learns when to release (Rangel et al., 2008; van der Meer et al., 2012). At first, these relationships are hardwired (salivate when presented with food), but with experience, one can learn to take these actions in response to predictive cues (salivate when you hear the dinner bell, Pavlov, 1927). Here, the motivational components remain in the circuitry through genetic learning, but the predictive stimuli can be learned (perception/memory) (Rescorla, 1988; Domjan, 1998).
- **Procedural action-selection systems** entail learning an arbitrary action sequence that one can release in an appropriate situation (hitting a fastball) (Squire, 1987; Mishkin and Appenzeller, 1987; Saint-Cyr et al., 1988). Here the motivational components are cached in the circuitry once learned, but they are learned through individual experience. Importantly, Procedural systems require learning in the perceptual system (situation recognition) that goes well beyond stimulus recognition (such as learning to differentiate a fastball from a curveball and being able to identify where and when the ball will cross the plate, McClelland and Rogers, 2003; Redish et al., 2007; Gershman and Niv, 2010).
- Finally, **Deliberative action-selection systems** entail an explicit imagination, evaluation, and planning process by which one creates a simulated (hypothetical) future (imagining the consequences of one's actions) and then evaluates that simulated future to select the best action (Gilbert and Wilson, 2007; Buckner and Carroll, 2007; Johnson et al., 2007). Interestingly, current models of imagination suggest that the same perceptual systems are reused for imagination (Kosslyn, 1994; O'Craven and Kanwisher, 2000), which implies that deliberation will depend on the same situation recognition processes as procedural learning (Pearson et al., 2015). Similarly, current models suggest that the deliberative process uses structures originally evolved to measure current rewards and punishments (this cake tastes good, that wool shirt is itchy) to evaluate the imagined worlds (Andrade and Ariely, 2009; Phelps et al., 2014). This explains why current emotional states can affect one's decisions about the future (like why you buy more food at the grocery store when you are hungry).

8.2 ADDICTION AS FAILURE MODES OF DECISION-MAKING SYSTEMS

A corollary of having multiple decision-making systems is that there are multiple ways for those systems to fail. Failure can occur at multiple targets in any given decision-making system, and each failure point can generate a subtly different behavioral phenotype (Table 8.1).

TABLE 8.1 Some Failure Modes of the Decision-Making System That Can Lead to Addiction. Obviously Incomplete

Failure-Point	Clinical Consequence
Changing allostatic set points (Koob and Le Moal, 2006; Koob and Volkow, 2010)	Physiological needs, craving
Cue-outcome associations elicit prewired visceral actions (Damasio, 1994; Bechara and Damasio, 2002; Bechara, 2005)	Incorrect action selection, craving
Escape from negative emotions (Koob, 2009)	Incorrect action selection
Mimicking reward (Volkow et al., 2002; Wise, 2005; Dezfouli et al., 2009)	Incorrect action selection, craving
Errors in expected outcomes (Goldman et al., 1999; Jones et al., 2001; Redish and Johnson, 2007)	Incorrect action selection
Increased likelihood of retrieving a specific expected action-outcome path (Redish and Johnson, 2007)	Obsession
Overvaluation of expected outcomes (Robinson and Berridge, 2001, 2003)	Incorrect action selection
Overvaluation of learned actions (Di Chiara, 1999; Redish, 2004)	Automated, robotic drug use
Timing errors (Ross, 2008)	Preferences for unpredictable events
Overfast discounting processes (Bickel and Marsch, 2001; Bickel and Mueller, 2009)	Impulsivity
Changes in learning rates (Franken et al., 2005; Gutkin et al., 2006; Redish et al., 2008; Piray et al., 2010)	Excess drug-related cue associations
Selective inhibition of the deliberative system (Bernheim and Rangel, 2004; Bechara, 2005; Bickel et al., 2008, 2012; Baumeister and Tierney, 2011)	Fast development of habit learning
Selective excitation of the habit system (Everitt and Robbins, 2005; Bickel et al., 2008; Keramati and Gutkin, 2013)	Fast development of habit learning
Misclassification of situations: overcategorization (Redish et al., 2007)	Illusion of control, hindsight bias
Misclassification of situations: overgeneralization (Redish et al., 2007)	Perseveration in the face of losses

Modified from Redish, A.D., 2013. The Mind within the Brain: How We Make Decisions and How Those Decisions Go Wrong, Oxford Univ. Press, Oxford, UK.

Biological organisms, for instance, actively regulate crucial biological parameters homeostatically (Mayr, 1998). Although at any given moment there is a set value that the organism will attempt to maintain, this set point can and does vary as a function of context (allostasis, Koob and Le Moal, 2006). Drugs of abuse are capable of altering an individual's natural set point and thus changing the biological needs of an organism (Meyer and Mirin, 1979; Benowitz, 1996; Koob and Le Moal, 2006). Cessation of drug use can thus disrupt the new drug-induced set point and result in withdrawal. These reflex-driven withdrawal symptoms would lead to highly negative sensations that require relief, which can drive drug seeking from multiple decision systems, including both Pavlovian and Deliberative. Importantly, however, there are other failure modes that can also drive drug seeking long after withdrawal symptoms have been eliminated; withdrawal and craving are dissociable (Childress et al., 1988).

A second well-studied failure mode can arise from cues that have come to predict upcoming drug administration, which can activate compensatory mechanisms in an addict. Heroin addiction offers a striking illustration of this system at work (Meyer and Mirin, 1979): When an addict prepares to administer the drug in the same setting that the drug is typically taken, physiological mechanisms (enzyme changes, modulation of receptor kinetics) will prepare the user's body for an upcoming dose and thus temporarily provide the individual with heightened tolerance. However, if the drug is taken in a novel setting the user is liable to overdose due to a failure of these compensatory Pavlovian mechanisms to provide that conditioned tolerance. Similarly, alcoholics have reduced alcohol-related coordination deficits in bars and other places where they expect to drink than in nonalcohol-associated environments (such as offices) (Hunt, 1998).

Because deliberative systems depend on evaluation circuits that evolved to evaluate ongoing needs (Phelps et al., 2014; Redish, 2016), expectations of future outcomes can depend on immediate needs. Thus, the compensation processes that occur on cue delivery can drive positive evaluations of drug-related outcomes (providing relief from the allostatic shifts, Koob, 2009). Because the recall of memory from a search process depends on recall and framing components (Redish and Johnson, 2007; Winstanley et al., 2012), it can also be guided toward these reminded outcomes. For example, video poker machines at the entrance to a grocery store can cue a whole imagined scenario of potential game playing (Schüll, 2012). This is a process termed "Pavlovian-to-Instrumental" transfer (Kruse et al., 1983; Talmi et al., 2008).

Moving beyond cue associations, the ability to encode refined cached action chains (i.e., habits) that are released in the appropriate situation is crucial to many forms of expertise (Graybiel, 1995; Klein, 1999). Current

theories suggest that this system entails the recognition of situations and the release of cached actions (Daw et al., 2005; Dezfouli and Balleine, 2012; van der Meer et al., 2012). However, these learned situation-action sequences are inflexible (because they evolved to respond quickly) and can turn maladaptive but well-practiced behaviors into tenacious habits. Whereas Pavlovian-to-Instrumental transfer can allow cues to increase the likelihood of deliberative systems to drive behavior toward drug seeking, a failure in the cached action system will make it such that, on recognizing and categorizing a situation, an inflexible and automated (potentially drug-related) action sequence will be released. For example, a smoker who has made it a matter of mindless routine to light up a cigarette first thing every morning (Tiffany, 1990) or the video poker player who gets lost in the flow of the game (Schüll, 2012) are two examples of these learned situation-action procedural mechanisms gone awry.

Higher level cognitive dysfunctions are also major contributors to drug abuse and relapse. To plan for the future, for example, an agent must evaluate available actions and their expected outcomes (Redish, 2016). Drugs of abuse often disrupt this planning and evaluation process, which leads to distorted outcome expectations (Goldman et al., 1987, 1999; Jones et al., 2001; Oscar-Berman and Marinkovic, 2003). The orbitofrontal cortex and nucleus accumbens are both key structures implicated in outcome evaluation (O'Doherty, 2004; McDannald et al., 2011; van der Meer et al., 2012) and a failure to receive, process, or generate the appropriate signal in these structures would negatively impact behavior because of a misvaluation of expected outcomes. Both orbitofrontal cortex and nucleus accumbens are often disrupted in drug users (Carelli and Wondolowski, 2003; Schoenbaum et al., 2006; Kourrich and Thomas, 2009; Koob and Volkow, 2010).

Many theories suggest that the evaluation of rewards in some systems, particularly the Pavlovian and Procedural systems, is due to changes in dopamine release (Montague et al., 1996; Schultz et al., 1997). Dopamine signaling increases to unexpected rewards, and dopamine neurons shift their firing to earlier cues that reliably predict those rewards (Schultz and Dickinson, 2000). Correctly predicted rewards produce flat rates of dopamine spiking, whereas expected but undelivered rewards produce a decrease in dopamine signaling. These three components mean that dopamine could signal "reward prediction errors," which can be used to train reactive decision systems such as Pavlovian or Procedural systems by driving estimates of value in the direction of the predicted error (Rescorla and Wagner, 1972; Montague et al., 1996; Sutton and Barto, 1998; Schultz and Dickinson, 2000). Redish (2004) noted that if a pharmacological agent (such as a drug) provided dopamine in a way that bypassed the normal neural calculations, it would lead to addictive behaviors

because the value would be driven to infinity. (Of course, neuronal representations would have to renormalize value, which could explain why nondrug rewards can lose their value in the face of extensive drug experiences, Goldstein, 2000; Heyman, 2009.) Importantly, this would only be one of many potential failure modes that could lead to addictive behaviors (Redish et al., 2008), and dopamine neurons signal other information as well as prediction errors (Bromberg-Martin et al., 2010). Nevertheless, the excess reward prediction error theory predicted that drugs of abuse should not show Kamin blocking, a phenomenon whereby predictive cues are not associated with already predicted stimuli (Redish, 2004). Interestingly, several subsequent experiments found that animals do show Kamin blocking using cocaine delivery as a reward (Panlilio et al., 2007; Marks et al., 2010). However, a further study found that while most animals showed Kamin blocking in a nicotine access experiment, the subset of animals that showed uncontrollable nicotine seeking did not (Jaffe et al., 2014). This elucidates one of the main points of this chapter: drug seeking can occur due to many potential failure modes; different individuals may have different reasons for their drug seeking. Treatment will need to identify the active failure mode to successfully treat addicts.

The ability to mentally construct imagined futures and play out competing scenarios to predict the anticipated value of a potential action confers a great advantage when making important "one-time-only" decisions (e.g., mentally simulating and comparing which of two job offers to accept, Gilbert and Wilson, 2007; Redish, 2016). However, these computational processes also have failure modes endemic to them. For example, either a miscalculation of the anticipated outcome or a misevaluation of a correctly anticipated outcome would lead to dysfunctional decision-making. The former can drive obsession and craving (Redish and Johnson, 2007), whereas the latter can drive decisions that will lead to negative outcomes. These can be seen in the heroin user looking for the orgasmic high of the first hit (Meyer and Mirin, 1979), the gambler trying to recreate the one time they won big at the machine (Lesieur, 1977; Custer, 1984) or the smoker underestimating the likelihood of getting cancer (Weinstein et al., 2005).

The etiology of addiction is not always driven by deficits in reward networks. Indeed, substance abuse has a high comorbidity rate with neuropsychiatric states. A well-studied example is that of alcoholism and its relation to anxiety disorders and major depression. In alcohol rehabilitation clinics alone, 50% of patients are diagnosed with either an anxiety disorder or major depression and these patients are twice as likely as their noncomorbid counterparts to relapse after leaving the clinic (Hobbs et al., 2011; Schadé et al., 2005). Although the comorbidity of

psychiatric disease and substance abuse is established, the causal relation between the two is a matter of debate. Intriguingly, a metaanalysis of epidemiological surveys and field studies found that comorbidity of alcoholism with anxiety was dependent on the type of anxiety disorder that the patient had—while agoraphobia and social anxiety were found to be risk factors for developing alcoholism, panic disorder and generalized anxiety were found to result from alcoholism (Hall, 1990). It would appear then that the causal relationship between anxiety and pathological alcohol consumption is bidirectional: pathological anxiety is a risk factor for alcohol abuse, but long-term alcohol use has the potential to induce pathological anxiety (Kushner et al., 2000).

A common argument for anxiety driving alcohol abuse is that individuals suffering from pathological anxiety might resort to alcohol as a means of self-medication (Quitkin et al., 1972). It has been suggested that the pharmacological profile of alcohol is such that it alleviates anxiety in a similar fashion as commonly prescribed anxiolytic compounds such as benzodiazepines and barbiturates (Liljequist and Engel, 1984). In support of this view, it has been shown that cross-tolerance occurs with alcohol and anxiolytics, thus highlighting a potential shared mechanism.

In contrast to the anxiolytic effects seen with acute alcohol administration, chronic alcohol use produces long-term changes in $GABA_A$ inhibitory receptors and in NMDA-sensitive glutamatergic receptors (Valenzuela and Harris, 1997; Littleton, 1998; Hunt, 1998) and is anxiogenic (Coffman and Petty, 1985; Tran et al., 1981). One explanation for why alcohol abuse could result in the development of an anxiety disorder hinges on the effect alcohol has on Deliberative and Pavlovian systems. Alcohol, for example, specifically impairs hippocampal and prefrontal function (Hunt, 1998; White, 2003; Oscar-Berman and Marinkovic, 2003), which could shift the balance from Deliberative to more Pavlovian and Procedural systems. Importantly, early alcohol use could depend on cognitive and social expectations (Goldman et al., 1987, 1999; Bobo and Husten, 2000), whereas later use may depend on dysfunctions in Pavlovian and Procedural systems (Dickinson et al., 2002; Oei and Baldwin, 2002).

It is important to note that the various decision-making mechanisms identified above interact to generate behavior. Failure in one system can affect other systems (such as evaluation errors affecting deliberative systems), but also a working system can be used to drive behavior when another system is dysfunctional. For example, social factors certainly play a part in the potential for alcoholism to induce anxiety, with social ramifications of alcohol abuse such as divorce and unemployment undoubtedly acting as potent anxiogenic stressors. We will see additional examples below.

8.3 BEYOND SIMPLE FAILURE MODES

Neuropsychiatric symptoms often result in unhealthy and unsafe behaviors that themselves drive the expression of new symptoms (Borsboom and Cramer, 2013). Symptoms that tend to cooccur can then causally influence one another (sleep loss → fatigue → loss of interest, etc.). As such, an initial external event (e.g., a debilitating physical injury) is capable of triggering a symptom network (e.g., injury → stress → depressed mood → insomnia → impaired attention → etc.). Once activated, a symptom network might itself be diagnosed as a mental disorder. The degree to which neuropsychiatric states are the result of internally driven defects in the neural circuitry or the externally imposed ramifications of initial symptoms is a topic of debate. As has often been found with such scientific debates (e.g., nature vs. nurture), it is likely a combination of the two.

8.4 RELIABILITY ENGINEERING

Broadly, reliability engineering refers to a collection of methods designed to minimize the likelihood of a system failing. To address this issue, one identifies the potential failures of the components and asks how those potential failure modes would affect the function of the system as a whole. This deductive, top-down approach is known as fault tree analysis (MacDonald et al., 2016). In fault tree analysis, the relationship between elements in the system and the ramifications of a failure in any one element on the system as a whole are evaluated using probabilistic causal networks (Pearl, 1988, 2009).

Applying this systems engineering outlook to the nervous system has recently emerged as a valuable tool capable of providing insight into the etiology of mental illnesses (MacDonald et al., 2016; Redish and Gordon, 2016). Specifically, by identifying the relationship between neural circuits and the points at which they are susceptible to failure, reliability engineering offers a toolbox of techniques for predicting the underlying cause of a neuropsychiatric disease (Flagel et al., 2016). As a result, more effective and individualized treatments can then be designed to address an individual's specific constellation of network failures that underpin their specific neuropsychiatric phenotype.

Just as with any other clinical condition, neuropsychiatric disorders are often identified on the basis of outwardly observable symptoms that are thought to reflect an underlying physiological deficit. The unobservable biological dysfunctions that generate the observable symptoms are known as latent variables because, despite being the direct cause of the pathological symptoms, they are hidden from view.

The aim of generating a fault tree for a given neuropsychiatric disease is then to identify all the distinct latent variables (and relations between them) contributing to the disease state. With this tool, a clinician can understand the pattern of potential dysfunctional components in the system that could result in a given patient's symptoms. Seeing as there are multiple combinations of latent variable defects that can result in the same symptomatology, an inductive principle that could be used to make increasingly accurate predictions about the most likely cause of a given disorder would be useful. Such a principle for making claims about uncertain variables does in fact exist and can be found in the mathematical rules governing Bayesian inference (Pearl, 1988, 2009).

8.5 IMPLICATIONS FOR TREATMENT

The goal of treatment is to reduce the harm underlying the "harmful dysfunction" discussed in the opening of this chapter. For example, a number of treatments have been aimed at attempting to reduce identifiable dysfunctions occurring in addicts, such as treatments to mitigate the effect of heroin on the mu-opioid receptor in some heroin users (Meyer and Mirin, 1979), treatments to slowly ramp down the changed set point in some nicotine users (Hanson et al., 2003), as well as treatments that make imbibing alcohol unpleasant (Wright and Moore, 1990), and treatments to extinguish the cigarette-nicotine association with denicotinized cigarettes (Johnson et al., 2004; Buchhalter et al., 2005). By identifying the underlying failure modes, we can move toward personalized treatments that attempt to address the individual dysfunctional mechanisms that are active in any given user.

However, it is also possible to provide compensatory mechanisms that can alleviate the harm, even without treating the dysfunction itself. For example, eyeglasses reliably treat the harmful dysfunction of nearsightedness without actually repairing the dysfunctional lenses. Presumably, the reason that organisms evolved multiple decision-making systems is that they make more optimal decisions under different conditions. By switching to the most effective system in a given situation, an organism could outcompete other organisms trying to use a single information processing algorithm for all situations. Just as we saw in the shelving example above, where it was easier to find the next book in a series if it was organized by author, if we had multiple indices that provided pointers to where the book was, perhaps one index of subject classification and another of size, then we could use the subject classification index when we wanted to locate a book on a topic and the size index when we needed a book to stabilize the table. Which decision-making algorithm

will control behavior depends on a number of incompletely understood factors, but one factor is the situation that one is in. This means that it could be possible to change the situation and, as a result of this change in situation, change one's addictive behavior.

For example, one strategy for coping with addiction is to eliminate exposure to cues that are known to trigger maladaptive behavior by precommitment (Ainslie, 1992; Kurth-Nelson and Redish, 2012a). Crucial to this method is the notion of shifting between decision-making systems (Kurth-Nelson and Redish, 2010; Kurzban, 2010; Redish, 2013). By preventing oneself from having the option to engage in addictive behavior ahead of time, an addict can precommit to a choice (say via the Deliberative system) that allows them to avoid placing themselves in temptation's path (that might trigger a Pavlovian and Procedural action). For example, an alcoholic might decide ahead of time to avoid walking down the street that has a liquor store on the way home from work or a gambler might avoid driving by a casino. (This can be difficult if there are video poker machines in every store, even the grocery store, Schüll, 2012.) This strategy of precommitment is a commonly employed and often effective method for minimizing exposure to cues that trigger impulsive and addictive behaviors. Fundamentally, it depends on the existence of multiple value functions, such as would occur with multiple, competing decision-making systems (Kurth-Nelson and Redish, 2010).

A related strategy for overcoming compulsive behavior is known as bundling (Ainslie, 1992; Kurth-Nelson and Redish, 2012b). Bundling entails changing the space of potential outcomes, usually by looking beyond a single choice. Effectively, bundling is a way of saying "doing this will lead to that." For example, an alcoholic acknowledging that there is no such thing as "just one drink" realizes that if they choose to drink, they will end up drinking to excess. This knowledge changes the value of the two options (drink or do not) relative to having a third (now unavailable) option of drinking "just one drink." This simple reestimation of the space of potential outcomes can help individuals step out of the vicious cycle of distorted expectations and destructive behavior.

Another commonly employed method for breaking addictive behavior is called contingency management (Higgins et al., 2002; Petry, 2011). Contingency management introduces a reward system that serves as an alternative to the reward that an addict would obtain from engaging in their maladaptive behavior. Addicts, by remaining abstinent, earn prizes or credits that can be used to purchase goods, and it has been shown that this promise of future reward will often incentivize them to remain abstinent.

Although current hypotheses describe the reasons for contingency management's success in terms of alternate reinforcers and lost opportunity costs (Higgins et al., 2004; Bickel et al., 2007; Packer et al., 2012),

Regier and Redish (2015) did a comparison of expected decreases in drug use relative to the actual alternative compensations provided in contingency management. We found that contingency management worked much better than expected.

One hypothesis to explain this surprising effectiveness of contingency management is that the promise of earning a delayed reinforcer if the addict remains abstinent nudges the individual into using deliberative processes rather than more reactive processes. This hypothesis predicts that contingency management success rates will be positively correlated with the integrity of the abilities (such as executive function processes) that underlie deliberation. This implies that cognitive tests that measure the viability of an individual's Deliberative system could be used to determine whether a patient is a good candidate for contingency management treatment. Furthermore, if the patient's Deliberative system was also compromised, perhaps executive function training could be used beforehand to prepare a patient for contingency management.

8.6 CONCLUSIONS

Multiple decision-making systems coexist and interact with one another to generate complex behavior. These decision-making systems and their interactions are vulnerable to distinct failure modes that can provide multiple paths to addiction. A computational understanding of decision-making circuitry offers the promise of a powerful tool that can be of tremendous value to both researchers and clinicians. Clinically, a more thorough appreciation of addiction mechanisms, from the underlying computations that neural circuits perform to how deficits in those neural circuits relate to clinical phenotypes, can inform the design of more effective treatments. A deeper understanding of which decision-making system vulnerabilities give rise to which clinical phenotypes will lead to more accurate methods for identifying, categorizing, and treating addictive dysfunction in an increasingly meaningful and patient-specific fashion.

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