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The Habitual Brain: An "Adapted Habit" Theory of Substance Use Disorders

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Behavioral habits are essential to human and animal life. We consider the many ways that habits—which are normally adaptive—can be expressed as drug use behavior and addiction. Although habit theories of substance use disorders have been proposed (e.g., Tiffany, 1990), the behavioral science and underlying neurobiology of habit development, maintenance, and change is only now being studied. We first define "adapted habit." We then propose that the etiology of an adapted habit represents the combination of: (a) initial "capture" of a habit, (b) development of behavioral action schemata, and (c) an overlay of cognitive expectancies concerning aspects of the habit. This combination conspires to make an intractable adapted habit such as substance abuse and addiction. Many intractable habits change, including substance use disorders such as cigarette smoking. As part of a science of habits, we need a real understanding of how to change habits to avoid or minimize harm.

Keywords habit; addiction; automaticity; adaptation; motivation; habit formation

> Drug addiction is [*an adapted, complex, routinized, entrained, stereotyped, over-learned, homeostatic*], chronic, relapsing disease . . . of the brain [bracketed text added]. (Leshner, 1997, p. 45)

A World without Habits

To appreciate some of the functions of habits, imagine having to completely relearn how to eat every time there is food available or one is hungry. A baby does this until it learns the *habit* of eating in a particular way; the child then rarely deviates from this habit when food is presented. This is true despite the fact that the food itself is highly variable and the circumstances in which eating takes place vary considerably. The entrainment of eating schedules and procedures are vitally important in the child's development, just as maintenance of eating habits is essential to adult functioning. One does not have to "reinvent the wheel" three times a day in order to maintain body weight and energy balance. The development of behavioral habits is equally important and functional for a broad array of life circumstances, some that are essential to survival and reproduction and others that are not.

There would probably be no substance dependence in a world without habits—if in fact there were any human or non-human animal survival at all! That is, we argue that habit formation is integral and necessary for the development of drug addiction, and perhaps,

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for life, itself. A common laboratory procedure to induce the drug withdrawal syndrome in rodents is to implant a pellet of morphine or an osmotic pump that releases the drug continuously and automatically over days. The animal then receives a specific antagonist (such as naloxone) to induce drug discontinuation effects or the pump is removed to achieve the same effect. This procedure certainly produces tolerance and the withdrawal syndrome, both cardinal features of drug dependence and addiction. It generally does not elicit other symptoms such as compulsive drug-seeking behavior or narrowing of attentional focus and the behavioral repertoire toward continued drug use that are equally important in most diagnostic systems. The development of a drug-seeking and drug-taking *habit* involves learning and behavioral actions that a morphine pellet does not provide. We argue that the inability to form and maintain a *behavioral habit* in an "ideal world" would preclude addiction to drugs (and probably life, itself).

Central Thesis

The central thesis of this discussion is that (a) the proclivity to develop behavioral "habits" is a fundamental characteristic emerging from brain architecture and (b) brain circuitry that supports habit formation is a major psychobiological substrate in the etiology of substance use disorders (SUDs). The formation of habits is characteristic of all mammalian species and is universal among human individuals—male and female—across widely divergent cultures. Although habits are far from unique to humans and are likely conserved across species that are much more diverse and phylogenetically old than are mammals, they are a vital component of what we colloquially refer to as "human nature." In fact, habits may best characterize "animal nature." We argue that habit formation is an "adaptation," meaning it is biologically based, though habits are highly plastic (at least initially) and idiosyncratically expressed in a myriad of ways within the same person and across individuals, societies, and cultures. More importantly, they are necessary for survival and reproduction, so the propensity to develop and sustain habits has been sharply honed by evolutionary selective pressures over eons of time.

We propose that habit formation and maintenance are two essential components of drug addiction (although certainly not the only components). This is implied in the formal diagnostic criteria for drug dependence in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, text revision (DSM-IV-TR, American Psychological Association [APA], 2000). These criteria emphasize that drug dependence is characterized by habitual behavior that narrowly focuses on obtaining and continuing to use the drug, often despite potentially serious consequences and neglect of other activities and responsibilities.

We argue, in a necessary oversimplification, that there is a "habit capture" system in the brain, dominated by autoshaping learning processes (primarily sign-tracking and feature-positive effects) and controlled by motivational processes characteristic of the corticomesolimbic dopamine system of the brain, such as the ventral tegmental area and the nucleus accumbens. Experimental drug use is captured by this habit-capture system such that, in some individuals (and in some non-human animals), drug abuse becomes an established and potentially intractable habit. The neurocircuitry of the "habit maintenance" system is different; it is controlled primarily by the basal ganglia in terms of the formation and execution of behavioral action schemata (such as drug-seeking and drug self-administration). Established habits are also characterized by positive outcome expectancies concerning what effects the habitual behavior will have; these cognitive expectancies (e.g., alcohol as a "Magic Elixir") are subserved primarily by prefrontal cortical areas of the brain. In general,

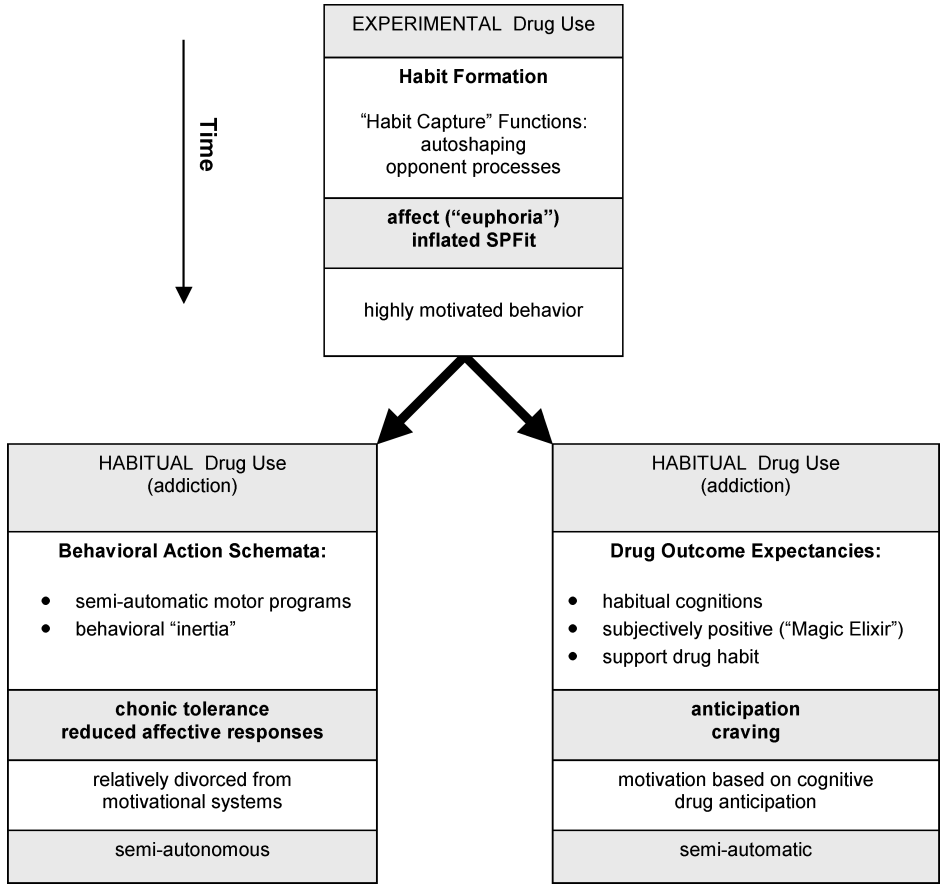

Figure 1. Schematic diagram of psychological processes involved in the transition from experimental drug use to established drug habit.

activation of the habit-capture system is characterized by intense affective responses (such as "euphoria" as well as fear) and strong motivation, while the habit-maintenance system is relatively divorced from the motivational processes that originally captured the system—unless the habitual behavior is blocked by intent or by circumstances. In this case, craving is prominent as the brain switches back to the habit-capture system to re-establish the drug habit. These relations are schematized in Figure 1. Everitt and Robins (2005) hypothesized that "the change from voluntary drug use to more habitual and compulsive drug use represents a transition at the neural level from prefrontal cortical to striatal control over drug seeking and drug taking behavior as well as a progression from ventral to more dorsal domains of the striatum" (p. 1481).

We propose that this "passing of the baton" from habit-capture to habit-maintenance, and from corticomesolimbic dopamine system to basal ganglia (action schemata) and prefrontal cortex (cognitive drug schemata), is the sine qua non of drug addiction. Leshner's (1997) famous metaphor of the development of addiction as "flipping a switch in the brain" might be better served by the "baton passing" analogy we offer for brain systems in the addictive process.

Routine Eating and Drug Addiction

To pursue our example of eating habits, we noted some of the positive aspects of routine eating above. Food, like drugs of abuse, is a primary reinforcer. In a remarkable paper, Woods (1991) discussed the negative aspects of eating and the implications of its "dark side." He views eating as an imminent and serious threat to the organism. Specifically, eating food causes a cascade of internal bodily (and brain) events that are highly stressful to the animal (or human) and that must be anticipated, tolerated, and controlled to prevent adverse events following meals. For example, eating food (or anticipating eating) increases cephalic insulin, apparently to prevent or escape the well-known adverse effects of high levels of fuels (e.g., glucose) in the brain.

Satiety mechanisms have been studied extensively, but little thought has been given to their biological significance. Woods (1991) argued that satiety mechanisms serve to limit meal size, which is necessary to prevent the adverse events that might otherwise occur in the brain with the large metabolic changes brought on by eating very large meals. He noted, too, that animals consume a great deal of water post-prandially, another mechanism to prevent adverse events from eating—in this case, from osmotic shift. Eating increases stress hormones such as epinephrine, norepinephrine, adrenocorticotropic hormone (ACTH), glucocorticoids, and beta-endorphin in what appears to be a classic stress response not unlike other stressors that may impact the animal and threaten its survival. These brain compensatory changes to prevent adverse events due to consuming food represent classic biological adaptations.

In addition to the internal—mostly brain—events that must be tolerated when eating, we add that an animal may be particularly vulnerable to prey when eating, when fully sated and lethargic, and especially when eliminating waste. The dangers of food poisoning, which was likely a common occurrence in our distant ancestral past, are further downsides of eating that could affect survival.

It would appear that routinely eating food is a dangerous habit! We argue, as did Woods (1991), that there are striking parallels between eating and taking drugs of abuse. These are listed in Table 1. The similarities between eating and drug-taking are many, and they span a wide range of domains of functioning, from behavioral and subjective to physiological and functional. The major difference between eating and drug use is that eating is necessary for survival but self-administering drugs of abuse is not. This is true even in the addicted individual.

How can these parallels be understood? Based on evolutionary psychology, we assume (for the moment) that the human brain is particularly optimized through natural selection for the following functions:

1. hunting/foraging/scavenging;
2. hierarchical social dominance relationships;
3. kinship selection and other kin relations;
4. mating and childcare.

This list is far from exhaustive and is not necessarily in order of importance, but considers the primary capacities/skills upon which evolutionary forces of natural selection might have operated eons ago (and today!). We view both eating and self-administering abused drugs in the context of general-purpose hunting/foraging/scavenging systems of the brain. Humans were called upon to hunt and scavenge for food and other resources (such as tools), and addictive behavior is best understood as a modern-day extension of this foraging behavior.

Table 1
Similarities between routine eating and substance use disorders (SUDs)

Characteristic	Similarity
Behavioral	Self-directed
	Goal-directed
	Ritualized
	Stereotyped
Subjective	"Like" (pleasurable)
	"Want" (crave)
	"Need"
	Cognitive expectancies
Physiological	Stressful
	Mimics endogenous processes
	Withdrawal syndrome
	Dangerous
	Vulnerable to prey
Brain	Dopamine N. acc. shell
	Basal ganglia
Learning	Primary reinforcer
	Conditionable
	Conditioned aversions
	Automaticity
	Over-learned
Temporal dynamics	Biphasic
	Anticipatory cascade
	Post-consumptive cascade
	Temporally entrained
	Liking declines, wanting doesn't
Functional	Adaptive
	Homeostatic
	Nonlinear dose-effect

In emphasizing the downside of routine eating, Woods (1991) placed eating in the context of the biological adaptations that were required to accomplish this (necessary) task while protecting the brain and body against the insult of taking a foreign substance into the body and incorporating it. The parallel with drug-taking may arise from similar considerations. We propose that self-administering abused drugs (though unnecessary) is captured by the same foraging system(s) of the brain as is eating food. We will discuss (below) some of the neural substrates that may be involved in these habits. Behavioral habits are particularly important for the foraging system(s) as successful strategies become codified as habits while unsuccessful strategies are less likely to be captured by this system.

History

Habit Strength

Habit theory became infamous in the second half of the twentieth century. Hull (1952) and Spence's (1956) elaborate theories of "habit-strength" simply collapsed of their own weight

as Skinnerian (1938, 1963) and Pavlovian (Pavlov, 1927) techniques proved to be vastly more parsimonious and powerful. The result was that the theory and explicit study of habits was laid to rest, at least in name. This article (and others, cf. Tiffany, 1990) seeks to build an unashamed habit theory of addiction.

Although the excessive fetish with schedules of reinforcement that succeeded Hull and Spence added little to our understanding of habit, psychopharmacology, or SUDs, it did lead (although certainly not inevitably) to three major advances that have contributed enormously to the addictions field: (a) drug self-administration (e.g., Pickens and Thompson, 1968), (b) respondent learning models of drug tolerance and addiction (Wikler and Pescor, 1967; Siegel, 1975), and (c) the wedding of neuropharmacology with behavioral experimental paradigms.

Drugs as Primary Reinforcers

Just as habits have a dark side, so too do some of these scientific advances. The naïve behavioral proclamation that drugs of abuse are primary reinforcers was revolutionary at the time, but it had a finality to it that many interpreted as an "explanation" for SUDs. This notion now seems almost tautological. In particular, the neglect of individual differences in human and nonhuman animal studies of drug self-administration persisted almost until the twenty-first century. Obviously some people use drugs for a short time but do not become addicted, whereas others seem virtually "pre-wired" to develop dependence. This is true of nonhuman animals as well. In juxtaposition with intracranial self-stimulation research, it also led to an equally naïve idea that these drugs activate simple "reward center(s)" in the brain that, in turn, resonate to increase the likelihood of further drug self-administration. The remnant of this simplistic view is that dopaminergic mesolimbic areas of the brain are now referred to as "reward pathways" (never as "reward centers") even though there is overwhelming evidence that these same reward pathways are also activated by stressful, noxious stimuli!

Respondent Conditioning

Siegel's Pavlovian (respondent learning) model of drug tolerance (Siegel, 1975) proved highly controversial (e.g., Baker and Tiffany, 1985). Although Siegel's research demonstrated beyond any reasonable doubt that learning factors contribute importantly to drug tolerance and addiction, the compensatory conditioned response that was critical to the model proved elusive, even in his own research. More recent studies (e.g., Krank, 2003; Kelsey and Arnold, 1994) have tended to dampen this criticism, but the addictions field has moved on to some extent to other, related research questions. Examples of these more recent experimental paradigms are drug craving (primarily in humans) and reinstatement of drug-seeking behavior following extinction (mostly non-human animals). Note that these latter paradigms build heavily on learning theories of addiction.

Homeostatic Models of Addiction

In relation to learning theories of SUDs, the confluence of respondent conditioning models of addiction (and their evanescent compensatory responses) with opponent process theory (Solomon, 1980) was consistent with and contributed substantially to homeostatic models of addiction. Homeostatic theories hearken back to Himmelsbach's (1943) classic studies

of the opioid withdrawal syndrome among individuals dependent on heroin. In contrast, instrumental drug self-administration is usually understood in simple hedonistic terms: the animal or human works for the drug because it produces "rewarding" subjective effects (presumably).

Tiffany's (1990) Cognitive Model of Drug Use Behavior

Although not couched in habit terminology, Tiffany's theory of drug use (but definitely *not* his model of drug urges or craving) is clearly a habit theory. The "drug use action plans or schemata" to which he refers (p. 154) are nearly synonymous with a definition of drug habit. His model is derived from cognitive theories of automatic versus controlled information processing. Therefore, Tiffany (1990) defined "drug use action schemata" as cognitive, behavioral, motor, and autonomic responses or skills that are stored in long-term memory as fully integrated semi-automatic processes. We certainly agree with this as a working definition and theory of drug habits—as far as it goes.

We believe the functional concepts of "biological adaptation" and "motivation" are missing from this definition and from Tiffany's (1990) theory. The cognitive theories that informed Tiffany's model of drug use assumed that higher cognitive processes are general-purpose rather than specialized. Though constrained by bottlenecks in performance (limited capacities in attention and memory), they were thought to have been virtually unlimited in flexibility and to have arisen from a tabula rasa cortex (Pinker, 2002). The cognitive model of drug use behavior that Tiffany developed makes similar assumptions. Tiffany (1990) was explicit that concepts of motivation (e.g., reinforcement and reward), or specific biological capacities and constraints on this behavior, were external or even incidental to his theory.

Toward a Psychology of Habits

Functions of Habits

Given our argument that habits are an essential part of addiction, we might reasonably ask whether habits are functional? Yes, absolutely, as we implied in our discussion of "a world without habits." Table 2 lists some of the functions that may make the capacity to form and maintain habits an evolutionarily optimal adaptation. We will discuss below the criteria that have been applied to the question of whether a psychobiological function can be reasonably considered a biological adaptation.

The Price of Habits

People (and nonhuman animals) pay a steep price for the habit adaptation. Drug addiction is one, but it takes little imagination to list the many "bad" habits that people develop—that is, habitual behavior that is counter-productive in the sense that it interferes with the biological, Darwinian (Darwin, 1859) imperatives to enhance survival and reproductive fitness. Table 3 lists some of the obvious problems with habitual behavior. We can only assume that the evolutionary advantages of this adaptation outweighed the disadvantages, at least in the ancient environment in which this capacity developed. Given that habits are fundamental to most non-human animals, we assume that this biological adaptation developed long before humans arrived on the scene.

Table 2
An apologia for adapted habits

Characteristic of habits	Explanation	Colloquialism
Efficiency	Habits are "fast and frugal" action heuristics	How not to reinvent the wheel every time
Minimality	Habits reduce bottlenecks in behavioral output	Why we can walk and chew gum at the same time
Utility	Habits have minimal response cost	Think, how can we dodge this work?
Quality	Habits are over-learned	How is drug addiction like shooting foul shots?
Optimality	Habits are behaviorally refined	If it ain't broke, don't fix it
Priority	Habits are high in the response hierarchy	Me first
Stability	Habits return the brain to homeostasis	If in doubt, do something
Reliability	Habits are predictable to others	Avoid unpredictable animals and people
Soothing	Habits are self-calming	Taming the savage beast within
Pre-emptive	Habits displace ongoing affect and behavior	The only game in town
Activating	Habits are an active default mode	I did it without thinking
Robustness	Habits are resistant to disruption by anxiety	Comes through in a clutch
Adaptive	Habits have some situational specificity	Is that behavior really appropriate?

Theoretical Context

The three developments noted above—drug self-administration, learning theories of SUDs, and behavioral neuropharmacology—have important implications for an adapted habit model of drug addiction. We propose that respondent conditioning is particularly important in the early stages of developing a drug habit, although our interpretation of the psychobiological functions—particularly "reward"—involved in this process (Newlin, 2002) differ

Table 3
Caveat emptor for adapted habits

Characteristic	Explanation	Colloquialism
Intractability	Highly resistant to change	Dyed in the wool
Unforgettability	Deeply engrained	Like riding a bicycle
Conditionability	Easily triggered by environmental stimuli	Spreads like a social disease
Automaticity	Only tenuously linked to brain mechanisms of self-control	Loose cannons on deck

Table 4
Major differences between the proposed "Habit Capture" and "Habit Maintenance" brain systems

Habit capture system	Habit maintenance system
Neuroanatomical	
Extended limbic network	Sensorimotor network
Corticomesolimbic	Basal ganglia
Medial prefrontal	Sensorimotor cortex
Medial dorsal striatum	Lateral dorsal striatum
Ventral striatum	Brainstem motor networks
Neurophysiological	
Broad activation	Narrowed activation
Primarily dopamine	
Functional control	
Goal-directed behavior	Antecedent stimuli
Action outcome sensitivity	Outcome insensitive
Outcome deflation/inflation	
Contingency degradation	
Outcome evaluation	Stimulus bound
Reward expectancy	Reward-independent
Flexible choice	Inflexible
Learning	
Autoshaping/sign-tracking	Overlearned/overtrained behavior
Early in instrumental learning	Late in instrumental learning
Rewarded choice behavior	
Cognition	
Explicit	Implicit
Controlled processing	Automatic processing
Memory systems	
Declarative memory	Procedural memory (nondeclarative)
Affect systems	
Emotionally laden	Relatively emotion free

Table 5
"Passing the Baton" from habit capture to habit maintenance

Habit capture system	Habit maintenance system
Experimental use	Habitual (addictive) use
Corticomesolimbic	Basal ganglia (action schemata)
Dopamine system	Prefrontal cortex (cognitive expectancies)
Autoshaping	Action schemata
Sign-tracking	Behavioral "inertia"
Feature-positive	Cognitive schemata
Controlled processing	Automatic processing
Working memory	Procedural memory
Drug sensitization	Drug tolerance

strongly from current dogma. We propose that respondent conditioning, particularly autoshaping, is a necessary component of the "hi-jacking" or co-option process that leads (sometimes, in some people) to drug habits and addiction. We view the corticomesolimbic dopamine system as most important in the drug experimentation phase, but as a drug habit develops, particularly among those at high genetic and/or environmental risk for SUDs, the brain areas that are most key to habitual use and addiction are the basal ganglia on the one hand and the prefrontal cortex on the other. This proposed change in the major sites of brain activity is parallel to the automatization of the drug habit (basal ganglia) and the blooming of subjectively positive drug outcome expectancies (cognitive habits) (Goldman, Brown, Christiansen, and Smith, 1991; Leigh, 1989) that support and promote the habitual use of these drugs (prefrontal cortex).

Autoshaping

Autoshaping is a laboratory procedure in which the animal is exposed to cued, noncontingent reinforcers (Hearst and Jenkins, 1974). In this situation, the animal readily approaches and contacts the cue, such as a lever or light that is turned on that is predictive of "reward" even though there is no contingency between the animal's behavior and presentation of the reinforcer. The food pellet or other reward (such as a receptive female to a male rat) will be presented no matter what the animal does, if anything. In most cases, the animal will come to reliably press the lever or contact the discrete cue of imminent reward before it appears, which is why it is usually referred to as "autoshaping." In some ways, the conditioned stimulus or "sign" of impending reinforcement becomes a substitute for the unconditioned stimulus (the reinforcer); the animal contacts the sign as if it were the food or other appetitive stimulus. This is a respondent (Pavlovian) conditioning procedure, but instead of an autonomic or affective response (such as salivation), the conditioned response is skeletal motor behavior. Autoshaping tends to produce behavior that is remarkably resistant to extinction; most animals will continue to approach and contact the conditioned stimulus (cue) even when such approach behavior actually prevents consumption of the reinforcer (such as food)! Based on this evidence, Hearst and Jenkins (1974) argued that autoshaping is in some ways more powerful than the "Law of Effect."

Technically, autoshaping is a procedure rather than an organismic process, although the term is often used to refer to the behavioral phenomenon as well as the procedure. The presumed psychological mechanism of autoshaping is sign-tracking (Hearst and Jenkins, 1974), or the tendency for animals to be attracted to and experience as salient cues that are predictive of positive events, such as the presentation of a reinforcer. In humans, this has sometimes (Newman, Wolff, and Hearst, 1980) been studied as the "feature-positive effect" in which people are inordinately attentive to features in a stimulus complex that are positively associated with reward rather than negative signs, or cues that signal the absence of reward. This has led to the assertion that people and non-human animals are particularly oriented toward, learn from, and even exaggerate positive correlational relationships in the world around them, to the point of ignoring or de-emphasizing zero-order correlations or negative correlations.

Autoshaping and Drug Use

We argue that experimental use of abused drugs is often captured by an autoshaping process because in human drug use, there are usually salient and reliable cues that the drug response is imminent. For example, initial drug use is usually deliberately done in a secretive environment to avoid detection, and the individual user is very aware that a drug response will

follow self-administration. These conditions are analogous to the rat or pigeon that receives a reliable cue that the drug reinforcer will arrive soon, even if it is presented intravenously via an implanted catheter. Experimental drug addiction is typically conceptualized in terms of instrumental conditioning rather than respondent conditioning or autoshaping.

Autoshaping and Drug Craving

Newlin (1992) proposed that drug craving is an example of autoshaping, the process in which appetitive approach behavior is conditioned through Pavlovian processes to drug stimuli. Therefore, this autoshaping model of drug craving incorporates aspects of both respondent and instrumental conditioning. It is a motivational model in that it emphasizes approach toward stimuli, such as drug paraphernalia, old drinking buddies, and situations in which the drug has been used many times in the past, that have become predictive of the drug effect for that individual.

Although originally proposed as a model of craving for drugs of abuse (Newlin, 1992), we now argue that autoshaping is also particularly characteristic of the experimental phase of drug use before the addictive process has developed fully (in some individuals). At its essence, respondent conditioning concerns anticipatory affective responses and the acquisition of motivation in relation to the conditioned and unconditioned stimuli. Therefore, notions of autoshaping are particularly apt in the experimental use of drugs because the emotional responses to the drug and to its anticipation are so strong. Robinson and Berridge (1993) emphasized that drug "liking" changes with addiction to drug "wanting" and to "needing." It is one of the great ironies of drug addiction that people with drug dependencies report little pleasure or reward from the drug to which they are addicted, a phenomenon that is difficult to reconcile with instrumental conditioning models. In these views, the "reward" from the drug effect reinforces or increases the likelihood of self-administration behavior. However, in a habit theory of addiction, the behavior becomes semi-autonomous and partially or wholly divorced from the motivational processes that originally promoted it. Importantly, the habitual behavior also becomes untethered from the internal self-control mechanisms that might otherwise alter or abolish it. That is, it takes on "a life of its own" that is neither pleasurable nor exciting (as it may have been initially). It becomes a mundane habit. Neurobiologically, it represents a "hand-over" from ventral striatal activation that is primarily dopaminergic (though modulated by a host of different neurotransmitters and second messenger systems) to the basal ganglia.

A particularly appropriate example of this is cigarette smoking. The euphoria or reward from smoking an individual cigarette by an established smoker is either absent or subtle at best, yet this is clearly an addiction in most chronic smokers. Instrumental conditioning accounts of smoking addiction find this problematic because if there is little or no reward, then there should be little or no smoking. However, if we assume that the habit of smoking has been effectively "handed off" to the basal ganglia, which has, in turn, established action schemata that maintain smoking behavior, then this continuation of the habit is understandable. It is then smoking *cessation* that engenders strong affective responses (i.e., nicotine withdrawal syndrome) rather than nicotine use itself. We suggest, as did Di Chiara (2000), that cessation leads to re-activation of the habit capture system until it is again handed back over to the habit maintenance system as the smoking habit is rapidly re-established.

Autoshaping with Drugs of Abuse

Autoshaping in which an abused drug is the unconditioned stimulus has been amply demonstrated. Carroll and Lac (1993, 1997, 1998) and Uslander, Acerbo, Jones, and Robinson

(2006) found robust autoshaping in rats with intravenous cocaine as the unconditioned stimulus, although Kearns and Weiss (2005) failed to autoshape rats with cocaine in an experimental procedure that did produce autoshaping with food. Krank (2003) found consistent evidence of autoshaping with alcohol as the unconditioned stimulus. Tomie et al. (Tomie, Aguado, Pohorecky, and Benjamin, 1998; Tomie, Di Poce, Derenzo, and Pohorecky, 2002; Tomie, Wong, Apor, Patterson-Bukendahl, and Pohorecky, 2003; Tomie, Tirado, Yu, and Pohorecky, 2004) performed a series of studies to investigate autoshaping primarily with alcohol, but also with chlordiazepoxide (Tomie, Wong, and Pohorecky, 2005).

Neurobiology of Autoshaping

Everitt and Robbins (2005) and their colleagues, in particular, have studied the brain substrates of autoshaping behavior. Lesion studies implicate the nucleus accumbens, a mesolimbic brain structure that is extremely important for drug-seeking behavior, in autoshaping performance. For example, lesioning the core (as opposed to the outer shell) of the nucleus accumbens severely impaired approach behavior to the conditioned stimulus while lesions of the anterior cingulate cortex had more limited deleterious effects and lesions of parts of the amygdala had no effect on autoshaping with food (Cardinal et al., 2002). Parkinson, Willoughby, Robbins, and Everitt (2000) investigated these connections, finding that lesions of the nucleus accumbens core and lesions that disconnected the anterior cingulate cortex from the nucleus accumbens core were effective in blocking appetitive instrumental approach (autoshaping). Further evidence (Cardinal et al., 2003) that the anterior cingulate cortex was involved in autoshaping came from evidence that lesions of this structure impaired discrimination in autoshaping between the predictive sign and the nonpredictive sign. Lesions of the subthalamic nucleus (Wistanley, Baunez, Theobald, and Robbins, 2005) and the pedunculopontine tegmental nucleus (Inglis, Olmstead, and Robbins, 2000) also profoundly impaired acquisition of autoshaping, suggesting that it is controlled at least in part by distributed subcortical structures.

Dopamine, a major "reward"-related neurotransmitter that is prevalent in the nucleus accumbens, is important in autoshaping (Dalley et al., 2002). Dopamine-depleting lesions of the nucleus accumbens impaired autoshaped learning, and particularly when approach behavior prevented food delivery (an important procedure for demonstrating that the process is respondent rather than instrumental conditioning). However, apomorphine, a potent dopaminergic agonist, strongly impaired learning, leading Dalley et al. (2002) to conclude that dopamine energizes or drives rather than guides autoshaping behavior. The dopamine antagonist flupenthixol infused into the core of the nucleus accumbens impaired both acquisition and performance of autoshaping, although a glutamatergic antagonist only reduced acquisition of autoshaped learning (Di Ciano, Cardinal, Cowell, Little, and Everitt, 2001). Dalley et al. (2005) found that dopamine D1 and NMDA receptors in the nucleus accumbens were critical in the acquisition of appetitive instrumental behavior, although dopamine D2 receptors were not. In addition, there is evidence from another research team that drug sensitization with amphetamine, a dopaminergic drug, leads to perseverative responding in a reward devaluation paradigm that is used to discriminate between goal-directed behavior and behavior that is controlled by action schemata of the basal ganglia. Therefore, dopaminergic stimulation speeded the "hand-off" from habit capture system to habit maintenance system, despite an intact ability of the animals to perform habitual behavior. Since virtually all drugs of abuse stimulate dopamine in the nucleus accumbens (Di Chiara and Imperato, 1988), it suggests a mechanism by which drug habits are learned unusually rapidly compared to non-drug habits.

Evidence from human studies with functional magnetic resonance imaging (fMRI), a noninvasive brain imaging technique, indicates that the dorsal and ventral striatum are particularly responsive to the salience of stimuli during learning. This has been manipulated by the amount of monetary reward (Zink, Pagnoni, Martin-Skurski, Chappelow, and Berns, 2004) and the degree to which a salient stimulus interrupts cognitve processes, even without reward (Zink, Pagnoni, Chappelow, Martin-Skurski, and Berns, 2006). This is consistent with the idea that the habit capture system is attuned to stimuli that are biologically relevant; that is, they are salient because they impinge on survival or reproductive motivation. Further fMRI evidence (Somerville, Heatherton, and Kelly, 2006) indicates the ventral part of the anterior cingulate cortex is more sensitive to social criticism, while the dorsal anterior cingulate responds more to expectancy violation.

Therefore, there is good evidence that the nucleus accumbens, particularly the core, is essential in autoshaping behavior and that dopamine is an important neurotransmitter in this function. Robbins and his colleagues (Dalley, Cardinal, and Robbins, 2004) have also studied prefrontal cortical contributions to autoshaping. Orbitofrontal cortex, but not infralimbic cortex lesions, impaired acquisition of autoshaped learning, producing perseverative responding (Chudasama and Robbins, 2003). Dalley et al. (2004) implicated the orbitofrontal cortex in lower-order discriminations, such as reversal learning (when the contingencies change) and learning with delayed reinforcement. These functions are important in autoshaping as well, particularly when approach toward the predictive sign is changed to prevent consumption of the unconditioned stimulus (such as food). In terms of neurotransmitter systems, Tomie et al. (2004) found that autoshaping for food increased plasma corticosterone as well as increases in norepinephrine and serotonin in the prefrontal cortex. This pattern indicated that autoshaping has both arousal-inducing and stressful aspects.

Neurobiology of Habits

Yin and Knowlton (2006) provided an excellent integrative overview of habit systems in the brain. The neuroanatomical structures implicated strongly in autoshaping play important roles in the systems that Yin and Knowlton's (2006) identified. They specified three hierarchically related striatal systems in the brain, all involving the striatum. These consisted of a limbic network (including the ventral striatum or nucleus accumbens), an associative network (including the caudate in primates or dorsomedial striatum in rats), and the sensorimotor network (including the putamen in primates or dorsolateral striatum in rats). Specifically, the limbic (or mesolimbic) network has supervisory control from the orbitofrontal and ventral prefrontal cortex. It is this network that we have termed the "habit-capture" system. It is characterized by goal-directed behavior that is sensitive to action-outcome contingencies and affective valence, and it has strong dopaminergic control. In cognitive terms, it exhibits controlled processing that is explicit, declarative, and in humans amenable to verbal self-report.

Part of the dorsal striatal network to which Yin and Knowlton (2006) refer builds stimulus-response associations that are necessary in habit capture. In humans, it includes the caudate and associative pallidum, with inputs from prefrontal and parietal cortical areas. In contrast, and a different part of the dorsal striatum (putamen), is the sensorimotor network, which is associated with action schemata and well-established habits. In cognitive terms, this system involves automatic processing that is implicit and not associated with affect, and may not be readily accessible to consciousness. In Yin and Knowlton's (2006) system of habit networks, it is strongly stimuli bound in the sense that relevant stimulus semi-automatically elicit behavioral habits with little supervisory control. This network is characteristic of

overlearned behavior or very late in the learning curve of instrumental conditioning. It is this network to which Tiffany's (1990) habit theory of drug abuse might refer, and which we refer to as a "habit maintenance" system. As Yin and Knowlton (2006) emphasized, these networks are hierarchically interrelated and different networks are sequentially predominant over the course of learning. As we saw, they can be teased apart to some extent with lesion and neuropharmacological interventions and with electrophysiological and neuroimaging measures, although they normally function relatively seamlessly to promote habit formation and continuation.

One of the primary applications of this research is drug abuse and addiction. We argue that the habit formation and maintenance systems evolved to promote survival and reproductive functions with so-called natural rewards. However, they can become hi-jacked or co-opted by the artificial introduction of abused drugs that mimic endogenous neurotransmitter and other receptor systems of the brain. Experimental use of these drugs is often "captured" by the ventral striatal habit system as if they promoted survival and reproductive functions (even though they generally do not), and then maintained by dorsal striatal mechanisms in the form of chronic, habitual drug habits (addiction).

This conceptualization parallels the SPFit theory of Newlin (2002) but is much more specific in terms of neurobiological mechanism. SPFit theory was couched in terms of the habit capture system as well as the prefrontal cognitive expectancies that surround chronic drug use. We now specify some of the neural substrates that may underlie the development and maintenance of drug habits. We emphasize that these are universal and highly adaptive (and adapted) systems of the brain that normally support behavioral habits which are essential to life. These brain mechanisms happen to be amenable to capturing and maintaining drug habits that are usually destructive (i.e., they impair biological fitness).

The Seven Deadly Sins of Adaptationism

We have adopted an adaptationist approach to drug habits. Panksepp and Panksepp (2000) critiqued evolutionary psychology's penchant for seeing adaptations in all phenomena. They structured their criticisms in terms of Seven "Sins" that are often committed in this area of research (and Seven Solutions!). We find this a useful background for considering the limitations of our approach. We conclude by evaluating whether habits qualify as "adaptations" according to the criteria enumerated by Schmitt and Pilcher (2004) that are often used to justify adaptationism.

Sin 1

> We can attempt to estimate the emergence of various general principles only to the extent that we have established credible brain structure/function relationships in many related species. (p. 113)

This is a tall order, although the experimental tools to investigate the neurobiology of habits in non-human animals are powerful, indeed. Neuroscience approaches to understanding SUDs have achieved ascendancy in the addictions field precisely because neurogenetic, pharmacological, and neurophysiological methods have developed greater precision and finesse than have the behavioral, cognitive, and cultural research tools that the clinical sciences have used to date. With the advent of brain imaging technologies (such as functional

magnetic resonance imaging, positron emission tomography, neuromagnetic imaging, and diffuse optical imaging), this methodological rigor is spreading to the clinical sciences. Neuroimaging techniques are far from fully developed. Their power will improve as the technologies become more precise and as researchers learn to more fully exploit them.

A serious problem in studying animal models of habitual behavior is the degree to which the animal model replicates the human phenomenon. The power of strong control over experimental variables in an animal is undercut if the model itself poorly represents the human situation. For example, a large empirical literature has been concerned with reinstatement of drug-seeking behavior. In this laboratory paradigm, non-human animals are first trained to self-administer a drug, then that behavior is extinguished by nonreinforcement (e.g., lever pressing for cocaine injections that no longer elicit an injection), followed by resumption of reinforcement (reinstatement). An alternative procedure is to make the instrumental response (and the drug reinforcer) completely unavailable for a period of time, then place the animal back in the environment with the same drug-operant contingencies (reinstatement). In many ways, the latter procedure, which is rare in animal studies, is more akin to the human situation and makes a more ecologically valid animal model. Fuchs, Branham, and See (2006) compared these two procedures, finding that virtual lesions of the dorsolateral caudate-putamen, structures that Yin and Knowlton (2006) identified as part of the sensorimotor habit network (habit maintenance system in our terminology), reduced cocaine seeking following abstinence but not after extinction training. This implies that reinstatement following extinction led to re-activation of the habit-capture system, while simple abstinence left the habit maintenance system operative.

Sin 2

> Human proclivities are commonly discussed independently of what we share with other creatures. (p. 114)

Our primary thesis is that habit formation is a fundamental characteristic of the architecture of human nature. Having written that, we readily accept the notion that most of human nature is not uniquely human at all but shared with most, if not all, mammalian species—that is, most of that which makes us "human" is strongly conserved across animal species. This makes non-human animal studies of habit formation and habit change particularly appropriate and useful. For example, initial evidence (Pickens and Thompson, 1968) that animals readily learn to self-administer drugs of abuse in a Skinnerian paradigm and these drug "habits" are highly resistant to extinction irreversibly changed the addiction field's view of the etiology of SUDs.

Drug abuse and dependence are far from uniquely human phenomena, nor have they ever been (Dudley, 2002). But does this imply that SUDs and habit formation are not characteristic of human nature? Certainly not. Instead, it implies that SUDs emerge in uniquely human ways from biologically fundamental brain processes that normally serve to promote survival and reproductive functions in both human and nonhuman animals. In Panksepp and Panksepp's (2000) terminology, human (and non-human) drug use recruits higher cognitive functions (particularly drug outcome expectancies) in the maintenance of habitual behavior that is tethered to primitive (reptilian?) subcortical functions of affect and motivation. In Newlin's (2002) model of SUDs, drug use develops as it hi-jacks corticomesolimbic motivational systems and the basal ganglia that evolved as primary brain structures for promoting survival and reproductive behavior.

Sin 3

> There surely should be little doubt that [basic emotional and motivational] systems reflect profound cross-species adaptations. (p. 114)

Panksepp and Panksepp (2000) identified seven adapted emotions/motivations they consider fundamental—rage, fear, lust, care, panic, play, and most relevant to drug abuse, seeking. Although we do not quibble with these emotions as basic adaptations (among other possible affects), we take an alternate view of motivation. Newlin's (2002) SPFit model of SUDs proposed that survival motivation (related to feelings of power, control, and social rank) and reproductive motivation (related to feelings of sexual and personal attractiveness, sexiness, and nurturance) are the most fundamental adaptations. It follows that these authors' primary affects (above) derive from promotion (i.e., lust, care, play) or frustration of (i.e., rage, fear, panic) these survival/reproductive motivations. We argue that basic affects are difficult to understand and even more difficult to study in humans with noninvasive techniques (although they can be studied well among non-human animals with invasive procedures) when considered in isolation from the motivational systems they evolved to support. It also implies that the propensity for habit formation and persistence evolved to subserve survival and reproductive functions.

Ironically, most studies of emotion in humans attempt to study these affects entirely out of context from motivational processes. For example, a common procedure to elicit emotion in human subjects is by showing positive or negatively valenced slides, often in mixed positive and negative order. We would expect this to produce sharply attenuated affective responses compared to carefully contrived social interactions because it does not activate the motivational systems that place these emotions in context.

An important example of this in relation to drug addiction is convincing evidence that self-administration of drugs of abuse has different neurobiological effects than does passive administration. Most psychopharmacological studies in nonhuman animals use passive administration (i.e., injections by the experimenter independent of the animal's behavior) because it allows precise metering of the dose and total amount of drug delivered, while self-administration by the animal normally does not. Of course, humans almost exclusively self-administer drugs of abuse; passive administration would be a rare exception. Therefore, self-administration is in some ways a more appropriate or realistic laboratory analog of the human situation.

This evidence was reviewed by Jacobs, Smit, De Vries, and Schoffelmeer (2003). They noted that self-administration of cocaine and opioids produces greater neuropharmacological responses (such as increased dopamine in nucleus accumbens) than does passive administration. It is revealing that the brain structures showing the largest differences between self- and passive-administration were the ventral striatum (primarily ventral tegmental area and nucleus accumbens) and basal ganglia (e.g., caudate-putamen and pallidum). These are precisely those areas most important in the passing of the baton from habit capture system (primarily corticomesolimbic dopamine system) to habit maintenance system (primarily basal ganglia). We reason that passive drug administration does not activate these two habit systems to the extent that self-administration does. This may account, in part, for why passive administration of medicinal opioids for the control of pain (such as post-surgical pain) is much less habit-forming than is active self-administration in opioid abuse.

We noted above that Panksepp et al.'s (2004) seeking brain-behavioral system is most relevant of their seven affects/motivations to drug abuse and addiction. They view the seeking

system, which is primarily dopaminergic and glutamatergic, as providing a "go" signal for exploratory and reward-seeking in the context of goal-directed behavior. This system is particularly activated and sensitized in novel environments or those that hold the possibility for new rewards (e.g., food, sex, drugs, etc.). Our "habit capture system" is most similar to Panksepp et al.'s (2004) seeking function, although we emphasize the role of this brain-behavioral system in developing new behavioral habits that lead to reward and re-activating old habits. In other words, we view this system as providing far more than a relatively undifferentiated "go" signal or drive. Their seeking affect/motivational system does not correspond well with our notion of a habit maintenance system. This is true even though we view the habit maintenance system as "driven," albeit by stimulus-bound responses rather than exploration and capitalization of reward (functions characteristic instead of the habit capture system).

Sin 4

> Most higher cortico-cognitive functions are epigenetically created by the experiences of organisms. (p. 115)

Central to Panksepp and Panksepp's (2000) argument is that cortical processes subserve nonspecific, general-purpose computational functions, whereas subcortical processes are usually highly specific, adapted functions. While it is undoubtedly true that cortical functions are more plastic than their subcortical counterparts, language functions likely contradict their central tenet. Moreover, these authors did not (in this particular paper) consider the implications of cortical hemispheric specialization of function, which is particularly prominent for receptive and expressive speech. The division of the cerebral hemispheres is the grossest of gross neuroanatomy. There is more than ample evidence (Davidson, 1992a, 1992b; Davidson and Irwin, 1999) that the right and left hemi-globes are differentially specialized, to a greater or lesser degree, for the seven affects noted by Panksepp and Panksepp (2000). For example, electrophysiological and neuroimaging studies implicate fear and panic as primarily right prefrontal cortical functions, while rage and lust (and probably care and play) are more associated with left prefrontal processes. This substantiates a degree of specialized cortical adaptation that belies Panksepp and Panksepp's (2000) basic thesis.

In relation to habit formation and maintenance, Davidson (2003) has argued that hemispheric specialization of approach and avoidance motivation are superordinate over affect. That is, the left prefrontal areas are specialized for approach motivation and positive emotions as well as anger (Harmon-Jones and Allen, 1998) are characteristic affects associated with this psychological orientation. In contrast, the right prefrontal cortex is associated with psychological withdrawal motivation, and negative emotions (except anger) follow from this orientation. We hypothesize that drug-seeking is associated with greater left prefrontal activation (approach) and drug cessation with right prefrontal activity. These hemispheric dynamics have rarely (Zinser, Fiore, Davidson, and Baker, 1999) been studied in relation to drug abuse and dependence. Moreover, despite decades of neuropharmacological research using brain microdialysis to measure dopamine (Di Chiari and Imperato, 1988) and other neurotransmitters, particularly in the ventral striatum (nucleus accumbens and ventral tegmental area) of rodents, there are virtually no studies of bilateral asymmetry in response to drugs of abuse in these areas. This is a particularly promising area of research.

Inasmuch as the habit-capture system (and Panksepp's seeking system) concerns approach behavior (toward drugs and drug-related stimuli), we propose that it involves preferentially left prefrontal brain activation. We suggest further that subcortical areas of

this corticomesolimbic dopamine system, particularly the ventral striatum, are also left lateralized.

If these hypotheses were verified by human and non-human animal research, then it would again indicate a degree of specialization and localization of function that contradicts Panksepp and Panksepp's (2000) argument that higher cortical functions are relatively non-specific and non-adapted. It would also have important implications for the habit theory that we propose. Specifically, we would expect that autoshaping of drugs of abuse, whether in humans or non-human animals, would be associated with left prefrontal and left ventral striatal function as the drug habit is "captured." We would also predict that in habit maintenance, the basal ganglia would show greater activation on the right as drug action schemata were interrupted by cessation of drug-taking behavior. Similarly, the activation of drug outcome expectancies that develop and solidify with prolonged drug use would be associated with greater left frontal brain activity. We emphasize that these are eminently testable hypotheses with current neurotechnologies.

Sin 5

> The classic distinction between emotional and cognitive processes is sustained by abundant data indicating that the two can be dissociated (p. 116)

Although there is little doubt that emotional and cognitive processes are dissociable, our model of the habit capture system implies strong integration of these processes as prefrontal and ventral striatal systems work together to form new habits. The dissociation of affect and cognition to which Panksepp and Panksepp (2000) refer applies to a much greater extent in the maintenance of habits as cognitive expectancies and action schemata "go their separate ways." This dissociation may be particularly apparent in relation to drug addiction. In cessation, we would expect notable temporal delays between changes in drug outcome expectancies and the action schemata that they normally (loosely) support.

Sin 6

> Computational-informational metaphors may not really be instructive for understanding the essential organic underpinnings of the human mind. (p. 117)

We certainly agree that the ability of neural networks or other computational systems to mimic and model behavioral phenomena may not reveal biological substrata (using current methods). However, these computational metaphors will likely prove very useful for studying habits in general and drug habits in particular. We note that drug addiction can be conceptualized as a local minimum in neural network terms and it is an "attractor" in linear dynamic systems theory or a "strange attractor" in nonlinear dynamic (chaos theory) terms.

At present, these computation approaches are primarily metaphorical. We argue that what is needed is a wedding of dynamical (linear and/or nonlinear) systems computational engines with the powerful neurotechnologies that have developed recently to study habitual behavior in general and drug habits in particular. Either approach in isolation has inherent limitations, as Panksepp and Panksepp (2000) emphasize, but the combination would be a technological tour de force that could reveal important characteristics of brain structure/function relationships.

Sin 7

> Much of mental life is fundamentally organic (p. 118).

There was a relative vacuum of neuroscience studies of habits until very recently. This dearth was particularly acute given our thesis that "much of mental life is fundamentally habit." Our partial review of neuroscience studies on the brain substrates of habit formation and maintenance indicates that much work has now been done. We anticipate that this work will continue unabated inasmuch as it sheds considerable light on habits, "good" and "bad," and on drug abuse and addiction in particular.

Conclusion

Is the human capacity and propensity to develop habits (and drug habits) an adaptation? We argue that it is. Schmitt and Pilcher (2004) considered criteria for determining whether a function is an adaptation, and these are summarized in Table 6. We have elaborated some of the fitness-enhancing aspects of habits, as well as the capacity to develop "bad" habits—such as drug abuse and addiction. In most cases, habits are highly functional because, among other reasons, they are so efficient. It is possible to carry out most daily activities, many of which are necessary for survival and reproduction, using precious few cognitive or affective resources. This automaticity has its down side and this may be particularly apparent in modern society as opposed to the ancient environments in which this adaptation evolved (probably in "lower" animals).

We propose that habits represent activity of a "horizontal module" in Fodor's (1983) nosology, meaning that it is accessible to a broad range of domains of functioning. It is not limited, for example, to phonological or grammatical processing, but represents a more general aspect of functioning. It can be co-opted by many different types of stimuli and environmental demands. Habits can be motor, affective, or cognitive, although they tend to morph into action schemata, or semi-automatic programs carried out primarily by the basal ganglia. We argue that habits are absolutely fundamental to human functioning.

Table 6
Schmitt and Pilcher's (2004) criteria for psychobiological adaptation

Criterion	Habit
Fitness enhancement	Highly effective foraging and scavenging system
○ functionality	○ functional; potentially dysfunctional in modern society
Design specificity	A horizontal adaptation/module?
○ domain-specific	○ (horizontal modules are not domain-specific)
○ complexity	○ disparate causative factors, integrated together
Universal yet interactive	Cross-culturally universal; idiosyncratically expressed
Efficient and economical	Habits require few cognitive or affective resources to maintain
Conserved across species	Habits are clearly present in some or all mammals

In addition to Schmitt and Pilcher's (2004) criteria for adaptations, we add cross-species universality in this particular case. We suggest that habits grow out of classic Hebbian characteristics of neural circuitry. As such, they may be as phylogenetically old as nervous systems, themselves.

RESUMEN

El Cerebro Habitual: "adaptó un modelo del hábito" de los desórdenes del uso de la sustancia

Los hábitos del comportamiento son esenciales para la vida humana y animal. Consideramos las muchas maneras eso ha'bito-que son adaptante-se pueden expresar normalmente como comportamiento y apego relacionados uso de la droga. Aunque las teorías del habito de los desórdenes del uso de la sustancia se han propuesto (e.g., Tiffany, 1990), la ciencia del comportamiento y la neurobiología subyacente del desarrollo, del mantenimiento, y del cambio del hábito ahora se está estudiando solamente. Primero definimos "habito adaptado." Entonces proponemos que la etiología de un hábito adaptado representa la combinación de: (1) inicial "captura" de un habito, (2) desarrollo de los esquemas del comportamiento de la accion, y (3) un recubrimiento de expectativas cognoscitivas referentes a los aspectos del habito. Esta combinación conspira hacer un habito adaptado insuperable tal como "abuso de la sustancia" y apego. Muchos habitos insuperables cambian, incluyendo desórdenes del uso de la sustancia tales como tabaquismo. Como parte de una ciencia de hábitos, necesitamos una comprensión verdadera de cómo cambiar hábitos para evitar o reducir al mínimo daño.

RÉSUMÉ

Le Cerveau Habitué: Un modèle de désordres d'utilisation de substances en tant qu "habitude adaptée"

Les habitudes comportementales sont essentielles à la vie humaine et animale. Nous considérons les nombreuses manières dont les habitudes-qui sont normalement adaptatives-peuvent être exprimées comme comportement et penchant reliés a utilisation de drogue. Bien qu'on ait proposé des théories d'habitude pour expliquer l'utilisation de drogue (par exemple, Tiffany, 1990), la science comportementale et la neurobiologie fondamentale du développement, de l'entretien, et du changement d'habitude commencent seulement a etre étudiées. Nous définissons d'abord "l'habitude adaptée." Nous proposons alors que l'étiologie d'une habitude adaptée représente la combinaison de: (1) la "capture" initiale d'une habitude, (2) développement des schémas d'action comportementaux, et (3) un recouvrement des expectatives cognitives au sujet des aspects de l'habitude. Cette combinaison conspire à creer une habitude insurmontable tel l'"abus de substance" et le penchant. Beaucoup d'habitudes insurmontables changent, y compris des désordres d'utilisation de substance tels que le tabagisme. Dans le contexte de la science des habitudes, nous avons besoin d'une reelle comprehension de la façon de changer les habitudes afin d'éviter ou de minimiser leurs consequences nefastes.

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Glossary

Autoshaping: a laboratory procedure in which the animal receives, in a classic respondent (Pavlovian) paradigm, repeated presentations of an arbitrary cue of impending reinforcement, such as food. The animal begins to contact the cue as if it were the reinforcement, and this learning is surprisingly resistant to extinction when the reinforcer (food, in this case) is omitted.

Drug-Seeking Behavior: the sometimes relentless preoccupation with and pursuit of drugs in the person's (or non-human animal's) environment. It is usually understood in terms of instrumental conditioning.

Habit-Capture: capturing a habit is when behavior that might only be performed a limited number of times is turned into a chronic habit. There may be systems in the brain that capture habits under certain circumstances that the behavior is performed (such as taking a drug).

Opponent Process Theory: this theory proposes that intense affective responses act as "a" processes that are balanced by "b" processes that are opposite in direction to the "a" process and which grow over repeated elicitations. Since the "a" and "b" processes are opponent and thought to combine additively, the "b" process tends to ameliorate the intense response of the "a" process.

Satiety Mechanisms: satiety, or satiation, is the quenching of a desire, such as hunger or thirst, when the object is consumed, such as food or water. If there were no mechanisms of satiety, then the organism would consume without end.

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