Habits, Rituals, and the Evaluative Brain

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Key Words

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Abstract

Scientists in many different fields have been attracted to the study of habits because of the power habits have over behavior and because they invoke a dichotomy between the conscious, voluntary control over behavior, considered the essence of higher-order deliberative behavioral control, and lower-order behavioral control that is scarcely available to consciousness. A broad spectrum of behavioral routines and rituals can become habitual and stereotyped through learning. Others have a strong innate basis. Repetitive behaviors can also appear as cardinal symptoms in a broad range of neurological and neuropsychiatric illness and in addictive states. This review suggests that many of these behaviors could emerge as a result of experience-dependent plasticity in basal ganglia–based circuits that can influence not only overt behaviors but also cognitive activity. Culturally based rituals may reflect privileged interactions between the basal ganglia and cortically based circuits that influence social, emotional, and action functions of the brain.

Contents

Habit is the most effective teacher of all things.

—Pliny

We are what we repeatedly do. Excellence, then, is not an act, but a habit.

—Aristotle

Habit is second nature, or rather, ten times nature.

—William James

For in truth habit is a violent and treacherous schoolmistress. She establishes in us, little by little, stealthily, the foothold of her authority; but having by this mild and humble beginning settled and planted it with the help of time, she soon uncovers to us a furious and tyrannical face against which we no longer have the liberty of even raising our eyes.

—Montaigne

INTRODUCTION

Habit, to most of us, has multiple connotations. On the one hand, a habit is a behavior that we do often, almost without thinking. Some habits we strive for, and work hard to make part of our general behavior. And still other habits are burdensome behaviors that we want to abolish but often cannot, so powerfully do they control our behavior. Viewed from this broad and intuitive perspective, habits can be evaluated as relatively neutral, or as "good" (desirable) or as "bad" (undesirable). Yet during much of our waking lives, we act according to our habits, from the time we rise and go through our morning routines until we fall asleep after evening routines. Taken in this way, habits have long attracted the interest of philosophers and psychologists, and they have been alternatively praised and cursed.

Whether good, bad, or neutral, habits can have great power over our behavior. When deeply enstated, they can block some alternate behaviors and pull others into the habitual repertoire. In early accounts, habits were broadly defined. Mannerisms, customs, and rituals were all considered together with simple daily habits, and habituation or sensitization (the lessening or increase in impact of stimuli and events with repetition) were included. Much current work on habit learning in neuroscience has pulled away from this broad view in an effort to define habit in a way that makes it accessible to scientific study. Much insight can also be gained by extending such constructs of habit and habit learning to include the rich array of behaviors considered by ethologists, neuropharmacologists, neurologists, and psychiatrists, as well as by students of motor control. Below, I review some of the definitions of habit that have developed in cognitive neuroscience and psychology and how these views have been formalized in computational theories. I then point to work on extreme habits and compulsions, ritualistic behaviors and mannerisms, stereotypies, and social and cultural "habits" and suggest that these are critical behaviors to consider in a neuroscience of habit formation.

This proposal is based on mounting evidence that this broad array of behaviors can engage neural circuits interconnecting the neocortex with the striatum and related regions of the basal ganglia. Different basal

ganglia–based circuits appear to operate predominantly in relation to different types of cognitive and motor actions, for example, in intensely social behaviors such as mating and in the performance of practiced motor skills. Remarkably, however, evidence suggests that many of these basal ganglia-based subcircuits participate during the acquisition of habits, procedures, and repetitive behaviors, and these may be reactivated or misactivated in disorders producing repetitive thoughts and overt behaviors.

A starting point is to consider defining characteristics of habits. First, habits (mannerisms, customs, rituals) are largely learned; in current terminology, they are acquired via experience-dependent plasticity. Second, habitual behaviors occur repeatedly over the course of days or years, and they can become remarkably fixed. Third, fully acquired habits are performed almost automatically, virtually nonconsciously, allowing attention to be focused elsewhere. Fourth, habits tend to involve an ordered, structured action sequence that is prone to being elicited by a particular context or stimulus. And finally, habits can comprise cognitive expressions of routine (habits of thought) as well as motor expressions of routine. These characteristics suggest that habits are sequential, repetitive, motor, or cognitive behaviors elicited by external or internal triggers that, once released, can go to completion without constant conscious oversight.

This description is familiar to many who study animal behavior and observe complex repetitive behaviors [fixed action patterns (FAPs)]. Some of these appear to be largely innate, such as some mating behaviors, but others are learned, such as the songs of some orcene birds. Repetitive behaviors and thoughts are also major presenting features in human disorders such as Tourette syndrome and obsessive-compulsive disorder (OCD). Stereotypies and repetitive behaviors appear in a range of other clinical disorders including schizophrenia and Huntington's disease, as well as in addictive states. I suggest that there may well be a common theme across these behavioral domains. Many of these repetitive behaviors, whether motor or cognitive, are built up in part through the action of basal ganglia–based neural circuits that can iteratively evaluate contexts and select actions and can then form chunked representations of action sequences that can influence both cortical and subcortical brain structures (**Figure 1**). Both experimental evidence and computational analysis suggest that a shift from

FAPs: fixed action patterns

OCD: obsessive-compulsive disorder

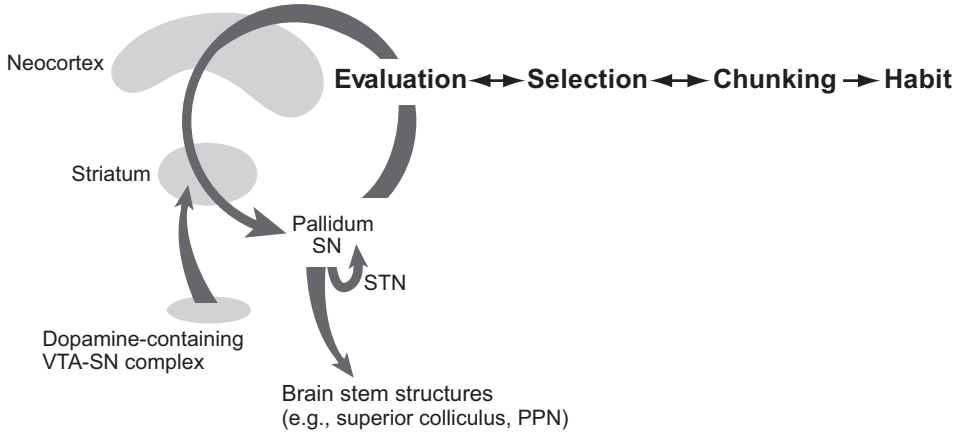

Figure 1

Schematic representation of the development of habits through iterative action of cortico–basal ganglia circuits. Circuits mediating evaluation of actions gradually lead to selection of particular behaviors that, through the chunking process, become habits. PPN, pedunculopontine nucleus; SN, substantia nigra; STN, subthalamic nucleus; VTA, ventral tegmental area.

S-R: stimulus-response

largely evaluation-driven circuits to those engaged in performance is a critical feature of habit learning. Chronic multielectrode recordings suggest that within the habit production system, as habits are acquired, neural activity patterns change dynamically and eventually settle into specific chunked patterns. This shift in neural activity from variable to repetitive matches the explore-exploit transition in behavioral output from a testing, exploratory mode to a focused, exploitive mode as habitual behaviors crystallize. This process may be critical to allow the emergence of habitual behaviors as entire structured entities once they are learned.

DEFINITIONS OF HABIT LEARNING IN COGNITIVE NEUROSCIENCE AND EXPERIMENTAL PSYCHOLOGY

Classic studies of habit learning distinguished this form of learning as a product of a procedural learning brain system that is differentiable from declarative learning brain systems for encoding facts and episodes. These definitions rest on findings suggesting that these two systems have different brain substrates (Knowlton et al. 1996, Packard & Knowlton 2002, Packard & McGaugh 1996). Deficits in learning facts contrast vividly with the preserved habits, daily routines, and procedural capabilities of patients with medial temporal lobe damage (Salat et al. 2006). By contrast, patients with basal ganglia disorders exhibit, in testing, procedural learning deficits and deficits in implicit (nonconsciously recognized) learning such as performance on mazes and probabilistic learning tasks in which the subject learns the probabilities of particular stimulus-response (S-R) associations without full awareness (Knowlton et al. 1996, Poldrack et al. 2001). The nonconscious acquisitions of S-R habits by amnesic patients has been documented most clearly by the performance of a patient who learned a probabilistic task with an apparent total lack of awareness of the acquired habit (Bayley et al. 2005).

Despite these distinctions, human imaging experiments suggest that both the basal ganglia (striatum) and the medial temporal lobe are active in such probabilistic learning tasks. When task conditions favor implicit learning, however, activity in the medial temporal lobe decreases as striatal activity increases, and when conditions favor explicit learning, the reverse is true (Foerde et al. 2006, Poldrack et al. 2001, Willingham et al. 2002). Moreover, in disease states involving dysfunction of the basal ganglia, medial temporal lobe activity can appear under conditions in which striatal activity normally would dominate (Moody et al. 2004, Rauch et al. 2006, Voermans et al. 2004). These findings demonstrate conjoint but differentiable contributions of both the declarative and the procedural memory systems to behaviors, as well as interactions between these two.

Comparable distinctions have been drawn for memory systems in experimental animals. The striatum is required for repetitive S-R or win-stay behaviors (for example, always turning right in a maze to obtain reward) as opposed to behaviors that can be flexibly adjusted when the context or rules change (for example, not just turning right, but turning toward the rewarded side even if it is now on the left). By contrast, the hippocampus is required for flexible (win-shift) behaviors (Packard & Knowlton 2002, Packard & McGaugh 1996). Nevertheless, the control systems for these behaviors cannot be simply divided into hippocampal and basal ganglia systems because both types of behavior can be supported by the striatum, depending on the hippocampal and sensorimotor connections of the striatal regions in question (Devan & White 1999, Yin & Knowlton 2004). Moreover, as conditional procedures are learned, neural activities in the striatum and hippocampus can become highly coordinated in the frequency domain (DeCoteau et al. 2007a).

In an effort to promote clearly interpretable experimentation on habit formation, Dickinson and his collaborators developed an operational definition of habits using characteristics of reward-based learning in rodents (Adams &

Dickinson 1981, Balleine & Dickinson 1998, Colwill & Rescorla 1985). In the initial stages of habit learning, behaviors are not automatic. They are goal directed, as in an animal working to obtain a food reward. But with extended training or training with interval schedules of reward, animals typically come to perform the behaviors repeatedly, on cue, even if the value of the reward to be received is reduced so that it is no longer rewarding (for example, if the animal is tested when it is sated or if its food reward has been repetitively paired with a noxious outcome). Dickinson defined the goal-oriented, purposeful, nonhabitual behaviors as action-outcome (A-O) behaviors and labeled the habitual behaviors occurring despite reward devaluation as S-R behaviors. Thus, in addition to habits being learned, repetitive, sequential, context-triggered behaviors, habits can be defined experimentally as being performed not in relation to a current or future goal but rather in relation to a previous goal and the antecedent behavior that most successfully led to achieving that goal.

A-O: action-outcome

The central finding from lesion work based on the reward-devaluation paradigm is that the transition from goal-oriented A-O to habitual S-R modes of behavior involves transitions in the neural circuits predominantly controlling the behaviors (**Figure 2**). Specifically, experiments suggest that different regions of the prefrontal cortex, the striatum, and the amygdala and other limbic sites critically influence these two different behavioral modes.

In rats, lesions in either the sensorimotor striatum (dorsolateral caudoputamen) or the infralimbic prefrontal cortex reduce the insensitivity to reward devaluation that defines habitual behavior in this paradigm. With such lesions, the animals exhibit sensitivity to

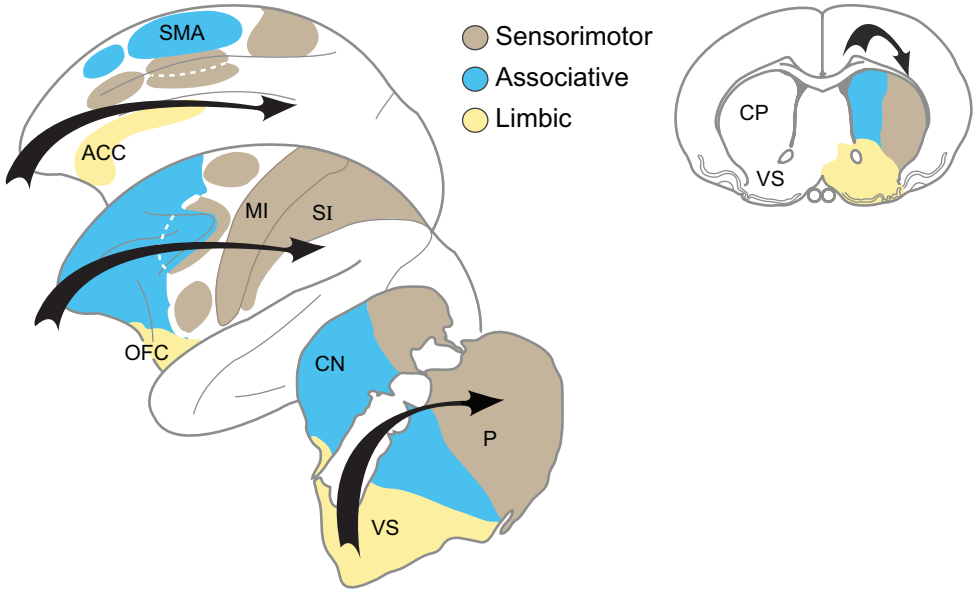

Figure 2

Dynamic shifts in activity in cortical and striatal regions as habits and procedures are learned. Sensorimotor, associative, and limbic regions of the frontal cortex (medial and lateral views) and striatum (single hemisphere) are shown for the monkey (*left*), and corresponding striatal regions are indicated for the rat (*right*). These functional designations are only approximate and are shown in highly schematic form. ACC, anterior cingulate cortex; CN, caudate nucleus; CP, caudoputamen; MI, primary motor cortex; OFC, orbitofrontal cortex; P, putamen; SI, primary somatosensory cortex; SMA, supplementary motor area; VS, ventral striatum.

reward value (A-O behavior) rather than habitual (S-R) behavior, even after overtraining (Killcross & Coutureau 2003, Yin & Knowlton 2004). By contrast, lesions of either the caudomedial (associative) striatum or the prelimbic prefrontal cortex reduce the sensitivity to reward devaluation that defines goal-oriented behavior in this paradigm; the animals are habit driven (Killcross & Coutureau 2003, Yin et al. 2005). The fact that lesions in either the striatum or the frontal cortex are disruptive suggests that the controlling systems represent neural circuits that have both cortical and subcortical components. In macaque monkeys, the basolateral amygdala and the orbitofrontal cortex are also required for sensitivity to reward devaluation (Izquierdo et al. 2004, Wellman et al. 2005). Thus multiple components of the goal-oriented system have been demonstrated across species, and these include regions strongly linked with the limbic system (Balleine et al. 2003, Corbit & Balleine 2005, Gottfried et al. 2003, Wellman et al. 2005).

Like the declarative vs. habit system distinction made in studies on humans, the distinction based on these experiments between action-outcome vs. stimulus-response systems is not absolute (Faure et al. 2005). Evidence suggests that these are not independent "systems." For example, after training that produces habitual behavior in rats, goal-oriented behavior can be reinstated if the infralimbic prefrontal cortex is inactivated (Coutureau & Killcross 2003). This finding suggests that the circuits controlling goal-directed behavior may be actively suppressed when behavior becomes habitual (Coutureau & Killcross 2003). The dichotomy between A-O and S-R behaviors also does not reflect the richness of behavior outside the narrow boundaries of their definitions in reward-devaluation paradigms (for example, when multiple choices are available or different reward schedules are used). The idea that there is a dynamic balance between control systems governing flexible cognitive control and more nearly automatic control of behavioral responses supports the long-standing view from clinical studies that frontal cortical inhibitory zones can suppress lower-order behaviors. This view has become important in models of such system-level interactions (Daw et al. 2005).

Most of these studies have been based on the effects of permanent lesions made in parts of either the dorsal striatum or the neocortex. The use of reversible inactivation procedures suggests that during early stages of instrumental learning, activity in the ventral striatum (nucleus accumbens) is necessary for acquisition of the behavior (Atallah et al. 2007, Hernandez et al. 2002, Hernandez et al. 2006, Smith-Roe & Kelley 2000). This requirement for the nucleus accumbens is apparently transitory: After learning, inactivation of the nucleus accumbens has less or no effect. Notably, inactivating the dorsolateral striatum during the very early stages of conditioning does not block learning and can even improve performance. This last result at first glance seems to conflict with the many reports concluding that the dorsolateral striatum is necessary for habit learning. However, these results fit well with the view, encouraged here, that the learning process is highly dynamic and engages in parallel, not simply in series, sets of neural circuits ranging from those most tightly connected with limbic and midbrain-ventral striatal reward systems to circuits engaging the dorsal striatum, neocortex, and motor structures such as the cerebellum.

Several groups have suggested that eventually the "engram" of the habit shifts to regions outside the basal ganglia, including the neocortex (Atallah et al. 2007, Djurfeldt et al. 2001, Graybiel 1998, Houk & Wise 1995, O'Reilly & Frank 2006). Evidence to settle this point is still lacking. There could be a competition between the early-learning ventral striatal system and the late-learning dorsal striatal system (Hernandez et al. 2002), an idea parallel to the proposal that, in maze training protocols that eventually produce habitual behavior, the hippocampus is required for learning early on, whereas later the dorsal striatum is required (Packard & McGaugh 1996). However, things are not likely to be so simple. The dorsal striatum can be engaged very early in the learning process (Barnes et al. 2005, Jog et al. 1999). And

"the striatum" and "the hippocampus" each actually comprise a composite of regions that are interconnected with different functional networks.

COMPUTATIONAL APPROACHES TO HABIT LEARNING: HABIT LEARNING AND VALUE FUNCTIONS

Work on habit learning has been powerfully invigorated by computational neuroscience. A critical impetus for this effort came from the pioneering work of Sutton & Barto (1998), which explicitly outlined the essential characteristics of reinforcement learning (RL) and summarized a series of alternative models to account for such learning (RL models). For experimental neuroscientists, this work is of remarkable interest because neural signals and activity patterns are being identified that fit well with the essential elements of RL models (Daw et al. 2005, Daw & Doya 2006). The key characteristics of these models are that an agent (animal, machine, algorithm) undergoing learning starts with a goal and senses and explores the environment by making choices (selecting behaviors) in order to reach that goal optimally. The agent's actions are made in the context of uncertainty about the environment. The agent must explore the environment to reduce the uncertainty, but it must also exploit (for example, by selecting or deselecting an action) to attain the goal. Sequences of behaviors are seen as guided by subgoals, and the learning involves determining the immediate value of the state or state-action set (a reward function), the estimated (predicted) future value of the state in terms of that reward (a value function). To make this value estimate, the agent needs some representation of future actions (a policy). Then the choice can be guided by the estimated value of taking a given action in a given state with that policy (the action value). These value estimates are principal drivers of behavior. Most behaviors do not immediately yield primary reward, and so ordinarily they involve the generation of a model of the action space (environment) to guide future actions (planning) in the sense of optimal control. Thus the control of behavior crucially depends on value estimates learned through experience.

A pivotal convergence of RL models and traditional learning experiments came with two sets of findings based on conditioning experiments in monkeys (**Figure 3**). First, dopamine-containing neurons of the midbrain substantia nigra pars compacta and the ventral tegmental area (VTA) can fire in patterns that correspond remarkably closely to the properties of a positive reward prediction error of RL models such as in the temporal difference model (Montague et al. 1996, Romo & Schultz 1990, Schultz et al. 1997). Second, during such conditioning tasks, striatal neurons gradually acquire a response to the conditioning stimulus, and this acquired response depends on dopamine signaling in the striatum (Aosaki et al. 1994a,b). These two sets of findings suggested a teacher (dopamine)–student (striatum) sequence in which dopamine-containing nigral neurons, by coding reward-prediction errors, teach learning-related circuits in the striatum (Graybiel et al. 1994). The actor-critic architecture and its variants, in which the critic supplies value predictions to guide action selection by the actor, have been used to model these relationships (Schultz et al. 1997). Many studies have now focused on identifying signals corresponding to the parameters in models of this learning process.

The firing characteristics of midbrain dopamine-containing neurons suggest that they can signal expected reward value (reward probability and magnitude including negative reward prediction error) and motivational state in a context-dependent manner (Bayer & Glimcher 2005, Morris et al. 2004, Nakahara et al. 2004, Satoh et al. 2003, Tobler et al. 2005, Waelti et al. 2001), that they are specialized to respond in relation to positive but not aversive reinforcements, and that they may code uncertainty (Fiorillo et al. 2003, Hsu et al. 2005, Niv et al. 2005, Ungless et al. 2004) or salience (Redgrave & Gurney 2006). These characteristics may, among others, account for the remarkable capacity for placebo treatments to

RL: reinforcement learning

VTA: ventral tegmental area

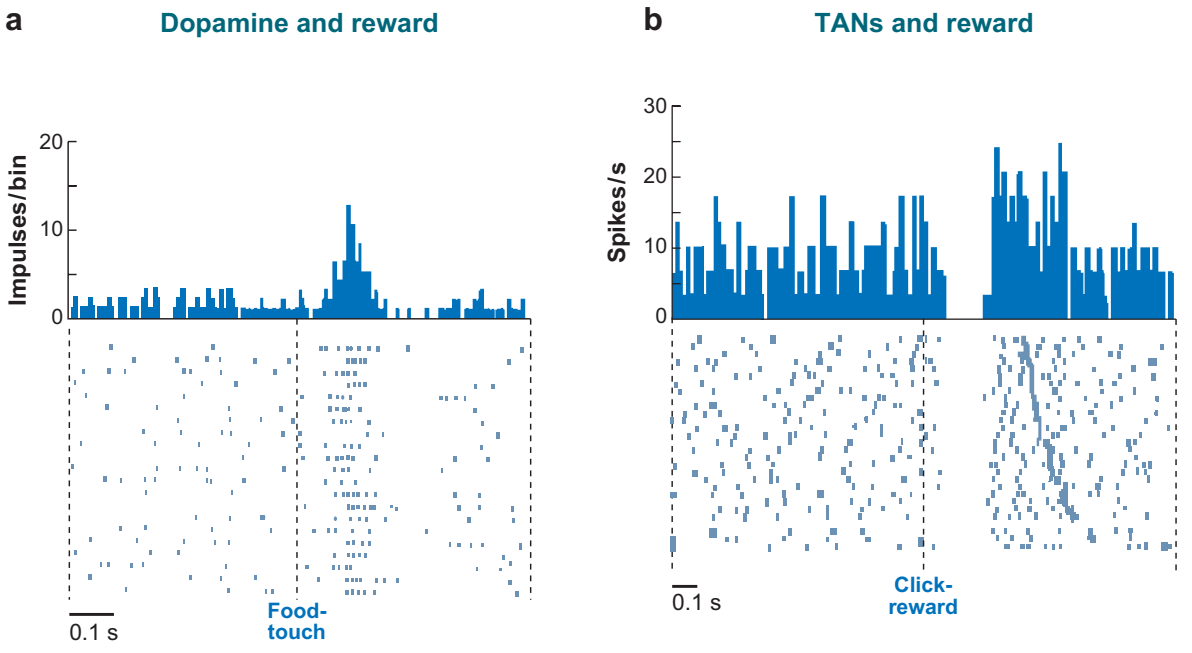

Figure 3

Reward-related activity of dopamine-containing neurons of nigral and striatal neurons. (*a*) Activity of nigral-VTA complex neurons (from Romo & Schultz 1990). (*b*) Activity of tonically active neurons in the striatum (from Aosaki et al. 1994b) Spike rasters (*below*) and histograms of those spikes (*above*) are aligned (*vertical lines*) at touch of the food reward (*a*) and at the conditional stimulus click sound indicating reward.

elicit dopamine release in the striatum (de la Fuente-Fernandez et al. 2001). Action value encoding was not detected by the original experimental paradigms used for recording from the dopaminergic neurons, which focused mostly on noninstrumental learning. Morris et al. (2006), using a decision task with a block design, have now shown that the action value of a future action can be coded in the firing of these neurons. This result is important in favoring computational models that take into account the value of a given action in a given state (the Q value). Remarkably, the dopaminergic neurons can signal which of two alternate actions will subsequently be taken in a given experimental task with a latency of less than 200 ms after the start of a given trial. This fast response suggests that another brain region has coded the decision and sent the information about the forthcoming action to the nigral neurons (Morris et al. 2006; compare Dommett et al. 2005). Models that incorporate the value of chosen actions in a particular state include those known as the state-action-reward-state-action or SARSA models and advantage learning models.

Ironically, a main candidate for a neural structure that could deliver the action value signal to the midbrain dopamine-containing neurons is the striatum, the region originally thought to be the student of the dopaminergic substantia nigra. Many projection neurons in the striatum encode action value when monkeys perform in block design paradigms in which action values are experimentally manipulated (Samejima et al. 2005). Other structures projecting to the nigral dopamine-containing neurons are also candidates, including the pedunculopontine nucleus (one of the brain stem regions noted in **Figures 1** and **5**), the raphe nuclei including the dorsal raphe nucleus, the lateral habenular nucleus and forebrain regions including the amygdala and limbic-related cortex,

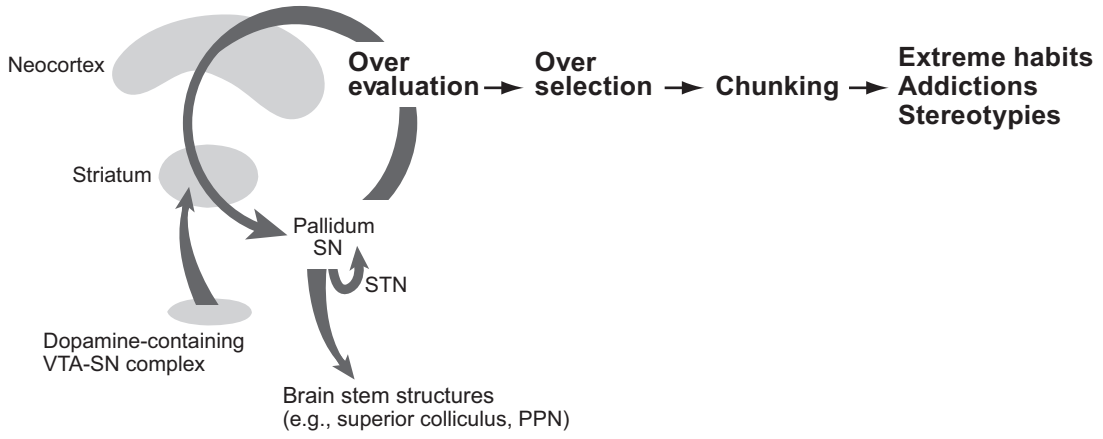

Figure 4

Schematic diagram suggesting the progression of functional activation in cortico-basal ganglia circuits as highly repetitive habits, addiction, and stereotypies emerge behaviorally. Note that in contrast to normal everyday habit learning (**Figure 1**), even the early stages of extreme habit formation involve steps that tend not to be readily reversible. PPN, pedunculopontine nucleus; SN, substantia nigra; STN, subthalamic nucleus; VTA, ventral tegmental area.

and also the striatum itself, including the striosomal system.

These findings highlight the difficulty of assigning an exclusive teaching function to any one node in interconnected circuits such as those linking the dopamine-containing midbrain neurons, the basal ganglia, and the cerebral cortex. Reinforcement-related signals of different sorts have been found in all of these brain regions (e.g., Glimcher 2003, Padoa-Schioppa & Assad 2006, Paton et al. 2006, Platt & Glimcher 1999, Sugrue et al. 2004), suggesting that signals related to reinforcement and motivation are widely distributed and can be used to modulate distributed neural representations guiding action. Reward-related activity has even been identified in the primary visual cortex (Shuler & Bear 2006) and the hippocampus (Suzuki 2007), neither of which is part of traditional reinforcement learning circuits. How these distributed mechanisms are coordinated is not yet clear.

Many of the ideas in reinforcement learning models and their close allies in neuroeconomics are now central to any consideration of habit learning. Experiments on goal-directed behavior in animals, including some with reward devaluation protocols, are increasingly being interpreted within the general framework of reinforcement learning (Daw & Doya 2006, Niv et al. 2006). For example, Daw et al. (2005) have proposed a model with two behavioral controllers. One (identified with the prefrontal cortex) uses a step-by-step, model-based reinforcement learning system to explore alternatives and make outcome predictions (their "tree-search" learning system for goal-oriented behaviors). The second, identified with the dorsolateral striatum, is a nonmodel-based cache system for determining a fixed value for an action or context that can be stored but that then is inflexible, corresponding to the habit system. The transition between behavioral control between the tree-search and cache systems is determined by the relative Bayesian uncertainty of the two systems. Allowing for interactions between these two systems would bring them into correspondence with the goal-oriented and habit systems of the reward devaluation literature. It seems unlikely, however, that there are only two learning systems or that these are dissociable as being exclusively cortical and subcortical.

The shift from ventral striatal to dorsal striatal activation during habit learning is also being incorporated explicitly into modified

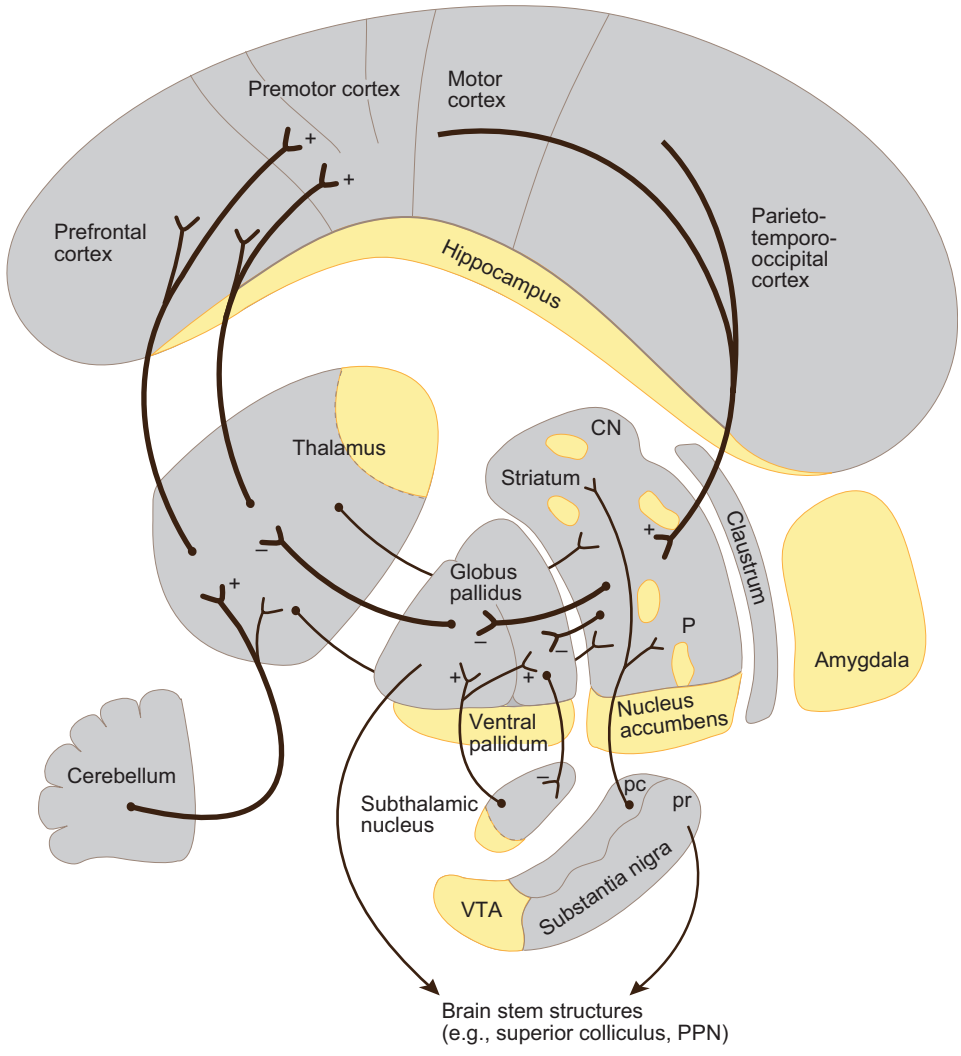

Figure 5

Basal ganglia circuit anatomy shown in simplified form with motor control pathways (*gray*) and regions related to limbic-system function (*yellow*). Descending pathways to brain stem structures (such as the superior colliculus and pedunculopontine nucleus) are not shown in detail. CN, caudate nucleus; P, putamen; pc, substantia nigra pars compacta; pr, substantia nigra pars reticulata; VTA, ventral tegmental area.

RL models. For example, evidence from human brain-scanning experiments has implicated the ventral striatum—active during the initial stages of learning—in reward-prediction error encoding whether or not responses by the subject are required. By contrast, reward-prediction error signaling that occurs during instrumental responding is differentially associated with activity in the dorsal striatum, especially dorsomedially (O'Doherty et al. 2004). These findings favor modified RL actor-critic models including advantage learning (Dayan 2002), in which the critic (ventral striatum) is influenced by motivational state (e.g., hunger) as well as by the ongoing evaluation of the particular state, whereas the dorsal striatum (actor) is engaged when actions are instrumental in bringing reward (Dayan & Balleine 2002).

This reformulation of the actor-critic model adds the critical feature of motivation to RL treatments. Haruna & Kawato (2006) have drawn a contrast between the caudate nucleus (and ventral striatum) as being associated with reward-prediction error and the putamen as being associated with a stimulus-action-dependent reward-prediction error. The early

requirement for ventral striatal activity in procedural learning has prompted the view that the ventral striatum is the director, guiding the dorsal striatal actor (Atallah et al. 2007). Imaging technologies cannot yet detect the neurochemically defined striosome and matrix compartments of the dorsal striatum, but the limbic-associated striosomes likely share some characteristics of the ventral striatum (Graybiel 1990, 1998). In line with this possibility, striosomes may represent state value in actor-critic architectures (Doya 2000, 2002) and thus may be critical components of striatum-based learning circuits.

The convergence of computational approaches with neuroimaging in humans has also led to new cognitive neuroscience models in which cognitive evaluation and choice depend on current reinforcement and expected future reinforcement and also on the value of behaviors not chosen so that choices themseves can be evaluated by comparing alternatives (Montague et al. 2006). The ventral-to-dorsal gradients found in these and other experiments (e.g., Tanaka et al. 2004, Zald et al. 2004) may in part reflect the predominance of immediate rewards (ventral striatum) and future rewards (dorsal striatum) in influencing behavior (Tanaka et al. 2004). This view emphasizes what I, below, call mental exploration.

EXTREME HABITS

Investigations of normal habit learning have immediate relevance for the field of addiction research. Habits induced by exposure to drugs of abuse can dominate behavior and produce states of craving and drug-seeking that persist for years. The dopamine-containing VTA and its striatal target region (the ventral striatum including the nucleus accumbens) have long been identified as forming a reward circuit essential for the initiation and expression of addictive behavior. Experiments on addiction have led to conclusions that are strikingly similar to those emerging from study of the normal transition from goal-directed to habit-driven behavior (**Figure 4**) (Everitt et al. 2001, Hyman et al. 2006, Ito et al. 2002, Kalivas & Volkow 2005, Porrino et al. 2004, Wise 2004). (*a*) The addictive process involves plasticity in neural circuits, not just in a single brain region. (*b*) The circuits critical for addiction include midbrain dopamine-containing cell groups and the striatum (in particular, the ventral striatum) and the neocortex (especially the anterior cingulate cortex and orbitofrontal cortex), as well as limbic parts of the pallidum (ventral pallidum), the thalamus (mediodorsal nucleus), and the amygdala and extended amygdala (Everitt & Robbins 2005, Kalivas et al. 2005, Kalivas & Volkow 2005). (*c*) The neocortex (especially the anterior cingulate and orbitofrontal cortex) powerfully influences this circuit at multiple levels. (*d*) Within this cortico-subcortical circuitry, different subcircuits appear to be essential at different stages of acquisition: the VTA and shell of the nucleus accumbens for the initial learning process in addiction, and the core of the nucleus accumbens and neocortical regions for the expression of the learned behaviors. Thus, the predominant activity in a given learning context shifts over time as the addiction becomes fixed (Sellings & Clarke 2003).

A striking example of this dynamic patterning comes from imaging studies in which radiolabeled dopamine-receptor agonist ligands are given to addicted subjects. Given exposures to a drug (IV methylphenidate), increased dopamine-related signaling (displacement of the ligand) in cocaine addicts occurs in the ventral striatum (and anterior cingulate and orbitofrontal cortices). But when cocaine addicts view video tapes showing cues associated with cocaine use, it is the dorsal striatum (including the putamen) that exhibits the differentially heightened dopamine signal (Volkow et al. 2006, Wong et al. 2006).

These findings are supported by work on experimental animals. In macaque monkeys, cocaine self-administration alters metabolic activity mainly in the ventral striatum after a brief period of self-administration, but with extended self-administration, such changes occur increasingly in the dorsal striatum (Porrino

et al. 2004). The gradients in these effects fit with anatomical evidence that the dopamine-containing inputs to the striatum, via indirectly recursive connections between the striatum and substantia nigra, follow such a ventral-to-dorsal gradient (Haber et al. 2000). Many other neurotransmitter-related compounds also follow such gradients, however, so that many aspects of striatal circuitry are modulated differently in ventral and dorsal striatum (and in medial and lateral striatal regions as well).

Differential release of dopamine in the nucleus accumbens in response to drug-associated cues has now been demonstrated directly in the rat by measuring extracellular dopamine by electrochemical detection with intrastriatal electrodes (Phillips et al. 2003). Cues produce dopamine release after the drug habit has been established (Ito et al. 2002). If the drug-taking behavior is extinguished and then is reinstated by exposing rodents to cues associated with the drug, as a model of relapse, dopamine release is also prominent in the neocortex and amygdala. These studies, in their entirety, suggest that as an individual moves from spaced, cognitively controlled experience with an addictive drug to increasingly repeated experience with the drug and then to the addicted state of compulsive drug use, major circuit-level changes occur at both cortical and subcortical levels. Within the striatum, there is a strong gradient from ventral to dorsal regions during the course of the addictive process.

Studies on normal habit learning and these studies on addictive habit learning both indicate a gradual change from ventral striatal control over the addictive behaviors to engagement of the dorsal striatum and neocortex (Everitt & Robbins 2005, Fuchs et al. 2006, Nelson & Killcross 2006). Such shared dynamic patterning (**Figure 2**) suggests that both in addiction and in the acquisition of nondrug-related habits, a similar set of coordinated changes in basic activity occurs at the circuit level. These dynamic patterns are a major clue for future experimental and computational work (Redish 2004).

A particular advantage of studies on addiction is that they can be used to uncover cellular and molecular mechanisms that promote transition to the addicted state (Hyman et al. 2006, Robinson & Kolb 2004, Self 2004). Many of these mechanisms are likely conserved and influence the learning processes that lead to repetitive, compulsive habits that are not triggered directly by drugs (Graybiel et al. 2000, Graybiel & Rauch 2000, Hyman et al. 2006). Links between the neurobiology of addictive and nonaddictive habits have already begun to be established by considering commonalities and interactions between drug effects and learning (e.g., Everitt & Robbins 2005, Fuchs et al. 2006, Nelson & Killcross 2006, Nordquist et al. 2007, Willuhn & Steiner 2006, Wise 2004).

LEARNING HABITS AND LEARNING PROCEDURES

How does habit learning relate to procedural or skill learning? Both involve learning sequential behaviors, but there are obvious differences: Learning how to ride a bicycle is quite different from having the habit of biking every evening after work. The skilled procedural performance of the baseball player is distinct from the habits and rituals for which baseball players are famous. And a regularly practiced habit may actually not be very skilled. Nevertheless, the learning of sequential actions to the point at which they can be performed with little effort of attention is common to both. So is the fact that, once consolidated, most acquired habits and procedures can be retained for long periods of time.

This transition from novice to expert is called proceduralization in the literature on human skill learning, most of which lies outside the reinforcement learning framework. These studies typically use reaction times (either simple or serial) as performance measures, along with a gradual reduction in interference if other tasks must be performed (Logan 1988, Nissen & Bullemer 1987). A large literature summarizes the varieties of motor sequence encoding in monkeys performing movement

sequences (Georgopoulos & Stefanis 2007, Matsuzaka et al. 2007, Tanji & Hoshi 2007). This work demonstrates that neurons can have responses related to individual or multiple elements of movement sequences and that these sequence-selective activities can occur in widely distributed brain regions, both cortical and subcortical (**Figure 5**). Notably, imaging and other studies demonstrate dynamic changes in the patterns of neural activity in the neocortex and striatum that closely parallel those observed in studies of habit-learning (**Figure 2**). With practice, the activity shifts from anterior and ventral cortical regions to more posterior zones of the neocortex, and from more ventral and anterior striatal regions to more caudal zones in the striatum (Doyon & Benali 2005, Graybiel 2005, Isoda & Hikosaka 2007, Poldrack et al. 2005). These shifts have been interpreted as representing a shift in the coordinate frames of the early and late representations (Hikosaka et al. 1999, Isoda & Hikosaka 2007). Doyon et al. (2003) propose a key function for corticostriatal circuits in this process, contrasting these with cerebellum-based learning processes that allow adjustments of motor behavior to imposed changes (Kawato & Gomi 1992).

A function in online correction has been ascribed to the striatum (Smith & Shadmehr 2005), which would be consistent with evidence for the basal ganglia–based song system in birds (Brainard & Doupe 2000). The experiments of Haruno & Kawato (2006) suggest that the differential engagements of the more anterior striatum (caudate nucleus) and more posterior striatum (putamen) at different points during learning reflect a fundamental difference in the representations of learning-related signals in the caudate nucleus and ventral striatum (representing reward-prediction error) and the putamen (representing stimulus-action-reward association). These and related studies (Samejima et al. 2005) indicate the value of extending reinforcement learning and neuroeconomics models of habit learning to the study of the proceduralization process itself and underscore the dynamic shifts in focus of neural activity as learning proceeds. However, these models will likely be revised as more is learned about the concurrent neural activities in the neocortex, striatum, and cerebellum. For example, cerebellar input can reach the putamen via a cerebello-thalamo-striatal pathway (Hoshi et al. 2005). Activity in the putamen may in part reflect input from a cerebellar circuit. Perhaps the striatum evaluates the forward models of the cerebellum.

These findings may help in understanding the progression of symptoms in Parkinson's disease patients. The advance of dopamine denervation in Parkinson's disease follows a posterior-to-anterior gradient, the reverse of the gradient found for procedural learning and habit learning. The predominant difficulty of Parkinson's patients to perform even well-known procedures, like rising from a chair or trying to do two things at once, may reflect learning gradients seen during proceduralization.

HABITS, RITUALS, AND FIXED-ACTION PATTERNS

Work on habits and habit learning has clear connectivity with the study of repetitive behaviors that include species-specific, apparently instinctual action sequences: the fixed action patterns described by ethologists. Like habits, FAPs are regularly contrasted with voluntary behaviors and relegated to low-level behavioral control schemes in which a particular stimulus/context elicits an entire behavioral sequence, for example, a chick pecking the orange spot on the beak of its parent (Tinbergen 1953). Here, value functions are estimated over evolutionary time.

Rituals are common across animal species and can be either remarkably complex, as in the nest building of the bowerbird (Diamond 1986), or relatively simple, as in grazing animals following set routes to a water source. Rituals related to territoriality, mating, and social interactions of many types seem to dominate the lives of animals in the wild. These behaviors share cardinal characteristics of habits. They are

repetitive, sequential action streams and can be triggered by particular cues.

Nearly all so-called fixed action patterns exhibit some flexibility (vulnerability to experience-based adjustment), and many rituals are acquired. A particularly instructive example of ritualistic behavior in animals is the use of song in mating behavior and other social behaviors in orcine birds. Their songs are composed of fixed sequences and, though in some species these appear to be genetically determined, in many species the songs are acquired by trial-and-error learning aimed at matching a tutor's template song. Bird song learning critically depends on a forebrain circuit that corresponds to a cortico–basal ganglia loop in mammals. Lesions within this circuit (called the anterior forebrain pathway) prevent the development of a mature song matching the tutor's song. The early song becomes prematurely stereotyped. At adulthood, such lesions impair the corrective adjustments of song required when abnormalities in singing behavior are induced experimentally (Brainard & Doupe 2000, Kao & Brainard 2006, Olveczky et al. 2005), and in some species the lesions can seriously degrade the mature song (Kobayashi et al. 2001). The song system is likely strongly influenced by dopamine, as the central core of the song circuit, area X (corresponding to the mammalian striatum and pallidum), receives a dopamine-containing fiber projection from the midbrain. Altogether, many aspects of avian song learning and song production bear a strong resemblance to the learning and performance of what we think of as habits and procedures in mammals: Both involve the acquisition of ordered sequences of behavior that are learned by balancing exploration and exploitation to attain a goal. And both require the activity of basal ganglia–based circuits as they are learned.

STEREOTYPIES

Very highly repetitive behavioral routines in animals are qualitatively distinguished as stereotypies on the basis of their apparent purposelessness and their great repetitiveness. In contrast to the habitual displays and rituals that are triggered in the course of normal behaviors, stereotypies (extremely rigid, repetitive sequences of behavior) are most prominent under aversive conditions, including stress, social isolation, and sensory deprivation (Ridley 1994). Exposure to psychomotor stimulants such as amphetamine also induces stereotypies, either of locomotion or of naturalistic behaviors such as sniffing, rearing, mouthing, and huddling. These categories can be likened to the route stereotypies and focused stereotypies of unmedicated animals. A distinction is often made between such highly repetitive, apparently purposeless behaviors and highly repetitive behaviors that appear goal directed, for example, repetitive grooming (barbering) of another animal or self-grooming.

Work on drug-induced stereotypies demonstrates that the basal ganglia are central to the development of these repetitive behaviors (Cooper & Dourish 1990). Low doses of drugs such as amphetamine (which releases dopamine and other biogenic amines) and cocaine (which blocks the reuptake of dopamine) induce prolonged and often repetitive bouts of locomotion. This behavior requires the functioning of the ventral striatum. The same drugs, given at higher doses, also induce bouts of highly stereotyped behavior, but these are more focused behaviors such as sniffing and grooming. These behaviors depend on the dorsal striatum (Joyce & Iversen 1984). Such dose-dependent drug effects ranging from locomotion to focused stereotypies follow the ventral striatal-to-dorsal striatal gradient that is thought to underlie both the acquisition of normal procedures and habits and the acquisition of addictive habits.

The strength of drug-induced stereotypic behaviors is correlated with differential activation of the striosomal compartment of the striatum, relative to activation of the matrix (**Figure 6**). This relationship has been demonstrated in rats, mice, and monkeys by the use of early-response gene assays (Canales 2005, Canales & Graybiel 2000, Graybiel et al. 2000, Saka et al. 2004). These findings tie the acquisition of at least one class of stereotypic

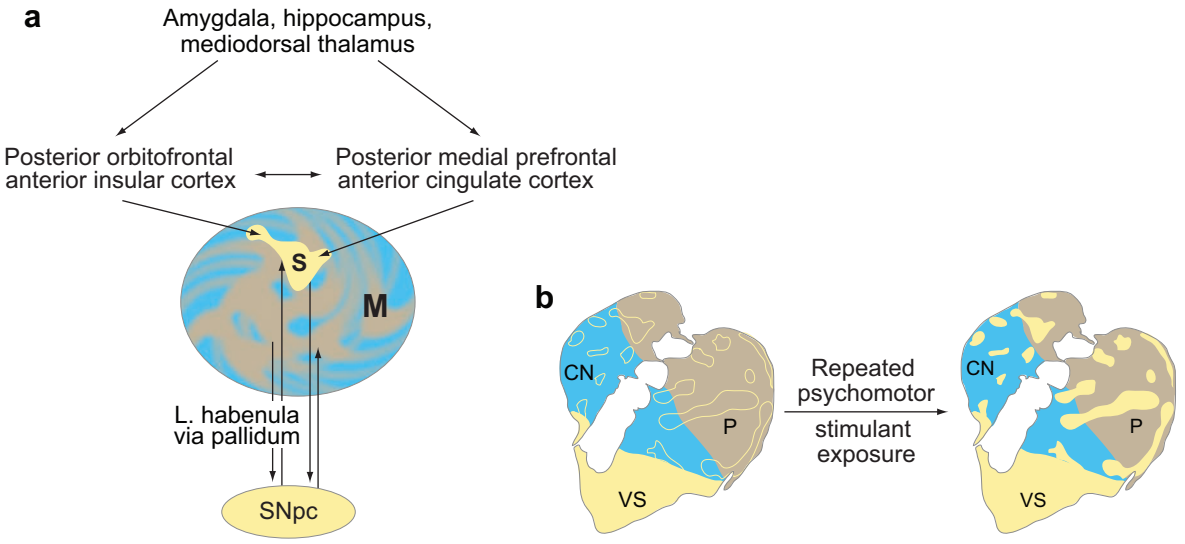

Figure 6

The striosomal system of the striatum. (*a*) Connections of striosomes with forebrain and brain stem regions including projections to nigral dopamine-containing neurons and/or nondopamine-containing nigral neurons near them. (*b*) Schematic illustration of the progressive accentuation of activity in striosomes that occurs as psychomotor stimulant-induced stereotypies develop. (Modified from Graybiel & Saka 2004). CN, caudate nucleus; M, matrix; P, putamen; S, striosome; SNpc, substantia nigra pars compacta; VS, ventral striatum.

behavior to a specialized set of striosome-predominant basal ganglia–based circuits. Striosomes in the anterior part of the striatum receive inputs from the anterior cingulate cortex and posterior orbitofrontal cortex and project to the substantia nigra both directly and indirectly via the pallidum and the limbic lateral habenular nucleus (Graybiel 1997). Through these connections, the striosomal system likely integrates and evaluates limbic information and affects the firing of dopamine-containing neurons in the nigral complex or immediately neighboring neurons. The striosomal system could thus alter the set point of subsets of the dopaminergic neurons and be a driving force behind some classes of stereotypic behaviors. We do not yet know what signal is transmitted by striosomes. One possibility is that they gate the magnitude of positive reward-prediction error or signal negative reward-prediction error through a connection with the pars compacta or nearby nigral neurons. They may also indirectly influence the lateral habenula, itself a source of negative reward-prediction error (Matsumoto & Hikosaka 2007).

A second line of evidence that the basal ganglia are required for some stereotypic behaviors comes from work on the highly repetitive movement sequences made by rodents during bouts of grooming called syntatic grooming (Berridge 1990). The grooming movements are strictly repetitive and temporally ordered, and lesion studies in the rat have demonstrated that a region in the mid-anterior dorsolateral caudoputamen must be intact for syntatic grooming to occur. Recording experiments suggest that some single striatal neurons in that region respond selectively to grooming movements that are part of a given syntatic grooming bout (Aldridge & Berridge 1998). The dopaminergic system can modulate the intensity of the grooming as well as the intensity of drug-induced stereotypies (Berridge et al. 2005).

How do stereotypies relate to habits? Evidently, they are different phenotypically. Stereotypies are more repetitive, are not as regularly elicited by triggers, and may not be governed in the same way by reinforcement learning. Yet many types of stereotypic behavior

depend on basal ganglia–based circuits for their development and production, as do habits. Stereotypies, like addictions, seem to reflect an extreme state of functioning of these brain circuits, in which flexibility is minimal and repetitiveness is maximal. The well-learned song of a zebra finch, the product of a basal ganglia circuit analogue in the avian brain, is as stereotyped as a typical drug-induced stereotypy. The fact that one is naturally prompted and the other is not should not obscure a potential common neural basis of these behaviors in the operation of cortico–basal ganglia circuits and related brain networks.

HABITS, STEREOTYPIES, AND RITUALISTIC BEHAVIORS IN HUMANS

Habits and routines are woven into the fabric of our personal and social lives as humans. One can scarcely call to mind the events of a day without running up against them. In fact, "getting in a rut" is so easy to do for many of us that we must fight the tendency in order to get a fresh look at life. Yet, as William James and many others have argued, those ruts, fashioned carefully, are invaluable aids to making one's way through life and are critical in social order: famously, the flywheel of society (James 1890).

Helpful as habits can be in daily life, they can become dominant and intrusive in neuropsychiatric disorders, including obsessive-compulsive disorder (OCD), Tourette syndrome, and other so-called OC-spectrum disorders (Albin & Mink 2006, Graybiel & Rauch 2000, Leckman & Riddle 2000). They are major features of some autism spectrum disorders and can dominate in some of these conditions (for example, in Rett syndrome). Stereotypies are pronounced in unmedicated schizophrenics and in some patients with Huntington's disease or dystonia, and they appear in exaggerated form in some medicated Parkinson's disease patients. In these disorders—as in normal behavior—habits, stereotypies, and rituals can be cognitive as well as motor, and often, cognitive and motor acts and rituals are interrelated. This commonality across cognitive and motor domains is a crucial attribute that suggests that there may be commonality also in the neural mechanisms underlying repetitive thoughts and actions.

In OCD, classified as an anxiety disorder, the repetitive behaviors that often occur do so in response to compelling and disturbing repetitive thoughts and felt needs (obsessions) that drive the repetitive behaviors (compulsions). The repetitive thoughts are dominant despite the fact that the person usually is aware that they are happening and does not want them to happen. Notably, the most common obsessions and compulsions are strongly cross-cultural. Checking ("Is it done? Did I do it just right?"), washing and cleaning due to obsessions about contamination, and repetitive ordering (lining up, straightening) are universal. OC-spectrum disorders include grooming and body-centered obsessions and compulsions, for example, extreme hair pulling or trichotillomania, for which barbering in animals is thought to be a model.

In Tourette syndrome, tics take the form of highly repetitive movements or vocalizations that range from abrupt single movements (twitches or sounds) to complex sequences of a behavior (including vocalizations) that appear as whole purposeful behaviors. They are exacerbated by stress, are sensitive to context, often occur in runs or bouts, and though suppressible for a time, often break through into overt expression. These actions are often prompted by an internal sensation or urge that builds up and produces stress; the urge can be relieved for a time by the expression of the tics. Nearly any piece of voluntary behavior can appear as a tic in Tourette syndrome. Sensory tricks, for example, touching a particular body part in a particular way, can also relieve the tics in some instances. It is as though a circuit goes into overdrive during a bout of tics but can be normalized by receiving new inputs.

At the behavioral level, obsessions and compulsions also can focus on a wide range of behaviors from grooming to eating to hoarding. Some specific genetic disorders involve compulsive

engagement in one or more of these fundamental behaviors (for example, ritualistic hyperphagia and food-related obsessions in Prader-Willi syndrome). These disorders seem to accentuate one or another of a library of species-specific and culturally molded behaviors. This remarkable characteristic has led many investigators, from ethologists and neuroscientists to neurologists and psychiatrists, to make extensive comparisons between these human behaviors and the rituals, stereotypies, and FAPs of nonhuman species (Berridge et al. 2005, Graybiel 1997). These behaviors also have high relevance for the study of habit. Across the spectrum of OC disorders, the behaviors are repetitive and usually sequential; they are often performed as a whole after being triggered by external or internal stimuli or contexts (including thoughts); and they can be largely involuntary.

It is not yet known whether the brain states generated by these disorders are akin to the brain states generated as a result of habit learning. However, dysfunction of basal ganglia–based circuits appear to be centrally implicated in these disorders, from OCD-Tourette and other OC-spectrum disorders to eating disorders to the punding of overmedicated Parkinson's disease patients (e.g., Graybiel & Rauch 2000, Palmiter 2007, Voon et al. 2007). A disordered OCD circuit has been identified in imaging studies, and this circuit includes the regions most often implicated in drug addiction: the anterior cingulate cortex and the posterior orbitofrontal cortex and their thalamic correspondents, and the anterior part of the caudate nucleus and the ventral striatum. In Tourette syndrome, differential degeneration of parvalbumin-containing neurons in the striatum and pallidum has been reported (Kalanithi et al. 2005), suggesting that the basal ganglia also contribute to the symptomatology in this disorder. The focal nature of tics may reflect local abnormal activation of subdivisions in the matrix compartment of the striatum (matrisomes) (Mink 2001) or defects in particular cortico–basal ganglia subcircuits (Grabli et al. 2004). The efficacy of sensory tricks in some Tourette patients may also reflect this architecture, whereby inputs and outputs are matched in focal striatal domains (Flaherty & Graybiel 1994). Because of the limited spatial resolution of current imaging methods, it is not known whether the striosomal system is differentially involved; this would not be surprising, however. Differential vulnerability of striosomes has been reported for a subset of Huntington's disease patients in whom mood disorders, including OC-like symptoms, were predominant at disease onset (Tippett et al. 2007). Predominant striosomal degeneration has also been reported in X-linked dystonia-parkinsonism (DYT-3, Lubag), in which many affected individuals exhibit OCD-like symptoms and suffer from severe depression (Goto et al. 2005). Sites being targeted for deep brain stimulation therapy in OCD and Tourette syndrome are also within these basal ganglia–based circuits; anterior sites near the nucleus accumbens are favored for OCD, and intralaminar and internal pallidal sites are favored for Tourette syndrome (Mink et al. 2006).

HABITS AND RITUALS: THE BASAL GANGLIA AS A COMMON THEME

Habits, whether they are reflected in motor or cognitive activity, typically entail a set of actions, and these action steps typically are released as an entire behavioral episode once the habit is well engrained. Here, I have reviewed evidence that this characteristic expression of an entire sequential behavior is also typical of well-practiced procedures and extends to stereotypies and rituals, including personal and cultural rituals in humans. Neural mechanisms that could account for such extended, encapsulated behaviors are not yet understood, but clues are coming from experiments in which chronic electrophysiological recordings were made from ensembles of neurons of rats and monkeys performing repetitive tasks to receive reward (**Figure 7**) (Barnes et al. 2005; Fujii & Graybiel 2003, 2005; Graybiel et al. 2005; Jog et al. 1999).

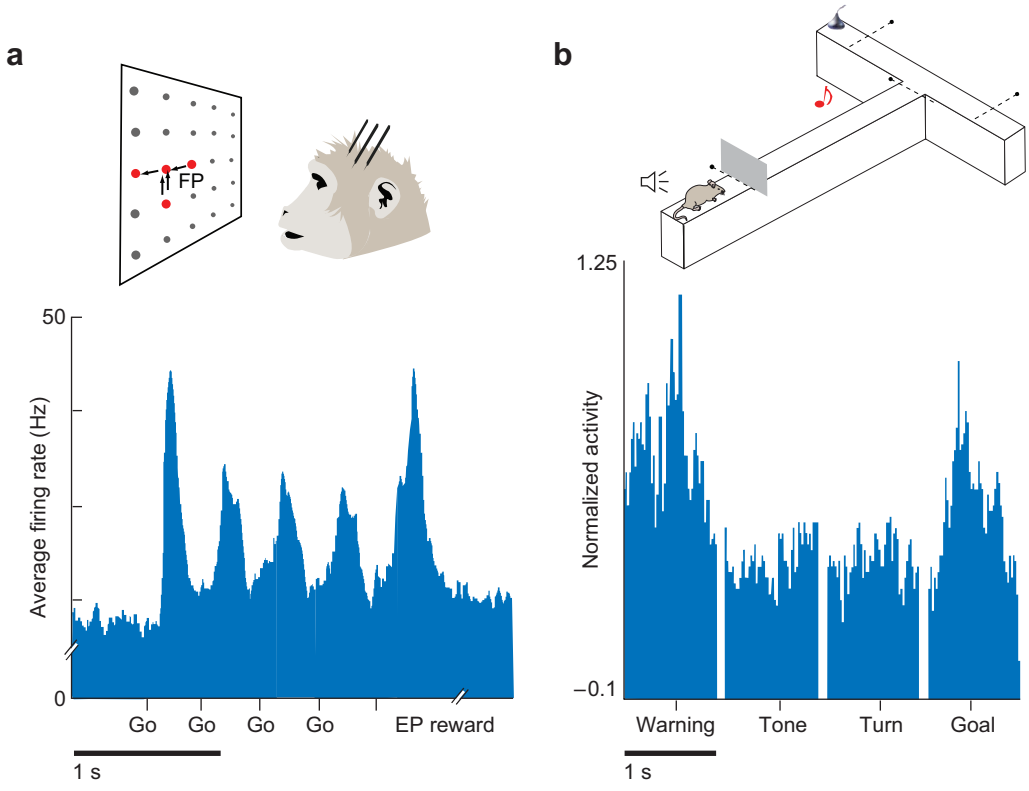

Figure 7

Heightened neural responses at action boundaries. Comparison between neural activity in prefrontal cortex accentuating the beginning and the end of saccade sequences in macaque monkeys performing sequential saccade tasks (*a*) and activity in sensorimotor striatum (dorsolateral caudoputamen) accentuating beginning and the end of maze runs in rats performing maze runs (*b*). Part *a* is modified from data in Fujii & Graybiel (2003), and part *b* is modified from data in Barnes et al. (2005).

In monkeys thoroughly trained to make sequences of saccadic eye movements, subsets of neurons in both the dorsolateral prefrontal cortex and the striatum have accentuated responses related to the first and last movements of the sequences, as though marking the boundaries of the action sequences (Fujii & Graybiel 2003, 2005). The accentuated beginning and end representations can be detected not only by looking at the task-related activity of the neurons, but also by looking at the temporal resolution of the neural representations (Graybiel et al. 2005). Time can be decoded with higher resolution at the beginning and the end of the movement sequences than during them. This last finding suggests that the neural representation of boundaries involves both action and time.

An explicit attempt has been made in rodents to track the neural activity patterns that occur in the striatum as habits are acquired (Barnes et al. 2005, Jog et al. 1999). Rats were trained to run a T-maze task in which they were cued to turn right or left to receive reward. The recordings were made in the dorsolateral caudoputamen, the region identified with S-R habits in reward devaluation studies and RL models. According to such studies, one would expect neural activity to be low during initial training but to increase during overtraining. The activity should be maintained during reward devaluation. This was not found. Instead, early in training, activity was strong throughout the maze runs. With extended overtraining, the activity did not increase. Instead, the activity changed in its

pattern of distribution over the course of the maze runs. Task-related neural firing became concentrated at the beginning and end of the maze runs, in a pattern resembling the action boundary pattern found in the overtrained monkeys performing saccade sequences (**Figure 7**). Simultaneously, apparently nontask-responsive neural firing in the striatum was markedly reduced. When extinction training was then given (akin to reward devaluation), these acquired neural activity patterns were not maintained, but instead were gradually reversed. They were not lost or forgotten, however, because once reacquisition training began, the acquired activity patterns reappeared.

These findings suggest that one result of habit learning is to build in the sensorimotor striatum chunked, boundary-marked representations of the entire set of action steps that make up the behavioral habit. The changes in neural activity found in the maze experiments have been interpreted as representing the neural analog of explore-exploit behavior (Barnes et al. 2005, Graybiel 2005). During initial training, the animals shifted from an exploratory (variable) mode to a repetitive mode of running the maze. In parallel, the neural activity in the sensorimotor striatum measured across task time was initially variable (neural exploration), in that activity occurred throughout the maze runs. As the behavior became repetitive, the neural activity took on the accentuated beginning and end patterns (neural exploitation). This shift could represent part of the process by which action sequences are chunked for representation as a result of habit learning: When they are packaged as a unit ready for expression, the boundaries of the unit are marked and the behavioral steps unfold from the first to the last boundary marker (Graybiel 1998).

These shifts in neural activity during learning could be carried to the striatum by its input connections—notably, inputs from the cerebral cortex and the thalamus. Alternatively, they may be produced by intrastriatal network activity or by some combination of such circuit processing. Evidence favors a role for striatal interneurons in such circuit-level plasticity. For example, as monkeys are taken through successive bouts of conditioning and extinction of conditioned eyeblink training, the tonically active neurons of the striatum (thought to be the cholinergic interneurons) successively gain and extinguish conditioned responses in parallel with the shifts in eyeblink behavior (Blazquez et al. 2002).

Further findings from the maze-learning experiments suggest that the formulation of the beginning and end pattern may depend, in part, on the rats learning the association between the instruction cues and the actions they instruct (T. Barnes, D. Hu, M. Howe, A. Dreyer, E. Brown, A. Graybiel, unpublished findings). The chunking process in sensorimotor tasks may thus occur as a result of S-R learning and be a hallmark of activity in the part of the striatum most directly engaged in affecting motor output. Work in progress also suggests that, at least in rats, the associative (dorsomedial) striatum exhibits different patterns during learning (Kubota et al. 2002a,b; C.A. Thorn, H. Atallah, Y. Kubota, A.M. Graybiel, unpublished findings). More work needs to be done to characterize these other learning-related striatal patterns along with those in other regions, including the neocortex; the locus coeruleus, identified as modulating exploration-exploitation balances (Aston-Jones & Cohen 2005); and serotonergic and cholinergic nuclei of the brainstem, which can modulate nigral neurons and reward functions (e.g., Doya 2002).

These results suggest that models epitomizing what physiological changes occur as habits and procedures are learned need to be revised to allow for repatterning of activity in different parts of the striatum and corresponding cortical and subcortical circuits. The gradients in activity traced from ventral to dorsal, anterior to posterior, and medial to lateral regions during habit learning do not necessarily mean that one region is transiently active and then becomes inactive as another region takes over, as if the habits are stored in just one site. The electrophysiological recordings suggest that we need dynamic models in which activity can occur simultaneously in multiple cortico-basal ganglia loops, not move in toto

from one site to another, and models in which, as the learning process occurs, activity patterns change at all these sites. We need to capture how such simultaneous, dynamically changing activity patterns can become coordinated over time through the actions of plasticity mechanisms that act on neurotransmitter signaling systems that themselves are expressed in differential gradients and compartmental patterns across the striatum. An interesting possibility is that oscillatory activity helps to coordinate these activities (DeCoteau et al. 2007b, Thorn & Graybiel 2007). For example, even though different patterns of neuronal activity are found with simultaneous recordings in dorsomedial and dorsolateral regions of the striatum during maze learning, these two regions exhibit coordination of oscillatory activity in particular frequency ranges (Thorn & Graybiel 2007).

If this dynamic repatterning is a general function of cortico–basal ganglia loops, then it should occur for cognitive activity patterns as well, with a shift from mental exploration to mental exploitation as habits of thought are developed. Some evidence suggests that the striatum and associated cortico–basal ganglia loops are involved in such processing and chunking in human language (e.g., Crinion et al. 2006, Lieberman et al. 2004, Liegeois et al. 2003, Tettamanti et al. 2005). A system of executive control using such start and end states has been proposed for activity in Broca's area in the human, supporting the idea of boundary markers in hierarchically organized domains (Fujii & Graybiel 2003, Koechlin & Jubault 2006).

Could the chunking process also relate to the low levels of attention that we typically need to pay to a familiar behavior when performing it as a habit? The deaccentuation of neural activity in between the accentuated beginning and end activities in cortico–basal ganglia circuits could reflect this attribute of habits. The eventual chunking of action repertoires during habit learning is an endpoint of successive shifting of neural activity from regions more closely related to the limbic system to regions more closely related to motor and cognitive output. These shifts represent stages of evaluation, and if the stages are successfully met—if the behavior is evaluated sufficiently positively—it is rerepresented in a chunked, readily releasable form. Thus, the relation between habits and the evaluative brain is that habits are an endpoint of the valuation process. Altogether, this process may engage a range of different cortico–basal ganglia loops and other neural circuits, potentially influencing different types of habit, from seemingly innocent mannerisms and rituals to dominating addictions. Studying this process should help investigators identify the neural systems underlying the shift from deliberative behavior controls to the nearly automatic, scarcely conscious control that we associate with acting through habit. Tracking this process may help us to understand the conscious state itself.

The power of social rituals may in part reflect an endpoint of this progressive evaluation process (**Figure 8**). The basal ganglia are strongly tied to the control and modulation of social behaviors. Human brain-imaging experiments have demonstrated strong activation of the dorsal striatum in experiments tracking activation for maternal love and romantic love (Aron et al. 2005, Bartels & Zeki 2004) and in social situations mimicked by interactive games and cost-benefit protocols (de Quervain et al. 2004, Elliott et al. 2004, Harbaugh et al. 2007, King-Casas et al. 2005, Montague & Berns 2002, O'Doherty et al. 2004, Tricomi et al. 2004, Zink et al. 2004). The nucleus accumbens and its dopamine receptors are necessary for monogamous pair bonding and for the maintenance of these bonds in the prairie vole (Aragona et al. 2006). Both language and song, with strong cortico–basal ganglia neural bases, serve social communication and are self-generated. They have the characteristic of agency, which heightens activation in the striatum in instrumental tasks relative to striatal activity in passive but otherwise corresponding tasks (Harbaugh et al. 2007, Zink et al. 2004). Finally, many of the rituals encountered in normal societies, and many of the ritualistic behaviors in neuropsychiatric disorders, have a strong social element, both in their content and

Figure 8

A ritual in humans (bull jumping in ancient Greece). Fresco from the East Wing of Knossos Palace, ~1500 B.C., Herakleion Museum, Crete.

in their likelihood for expression, and rituals and stereotypies in animals can also be strongly influenced by social context. Neural processing in circuits related to the basal ganglia, with their widespread interconnections with both limbic and sensorimotor systems, provides a common mechanistic theme across this large array of behaviors.

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The author is not aware of any biases that might be perceived as affecting the objectivity of this review.

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LITERATURE CITED

Adams CD, Dickinson A. 1981. Instrumental responding following reinforcer devaluation. *Q. J. Exp. Psychol.* 33:109–21

Albin RL, Mink JW. 2006. Recent advances in Tourette syndrome research. *Trends Neurosci.* 29:175–82

Aldridge JW, Berridge KC. 1998. Coding of serial order by neostriatal neurons: a "natural action" approach to movement sequence. *J. Neurosci.* 18:2777–87

Aosaki T, Graybiel AM, Kimura M. 1994a. Effects of the nigrostriatal dopamine system on acquired neural responses in the striatum of behaving monkeys. *Science* 265:412–15

Aosaki T, Tsubokawa H, Ishida A, Watanabe K, Graybiel AM, Kimura M. 1994b. Responses of tonically active neurons in the primate's striatum undergo systematic changes during behavioral sensorimotor conditioning. *J. Neurosci.* 14:3969–84

Aragona BJ, Liu Y, Yu YJ, Curtis JT, Detwiler JM, et al. 2006. Nucleus accumbens dopamine differentially mediates the formation and maintenance of monogamous pair bonds. *Nat. Neurosci.* 9:133–39

Aron A, Fisher H, Mashek DJ, Strong G, Li H, Brown LL. 2005. Reward, motivation, and emotion systems associated with early-stage intense romantic love. *J. Neurophysiol.* 94:327–37

Aston-Jones G, Cohen JD. 2005. An integrative theory of locus coeruleus-norepinephrine function: adaptive gain and optimal performance. *Annu. Rev. Neurosci.* 28:403–50

Atallah HE, Lopez-Paniagua D, Rudy JW, O'Reilly RC. 2007. Separate neural substrates for skill learning and performance in the ventral and dorsal striatum. *Nat. Neurosci.* 10:126–31

Balleine BW, Dickinson A. 1998. Goal-directed instrumental action: contingency and incentive learning and their cortical substrates. *Neuropharmacology* 37:407–19

Balleine BW, Killcross AS, Dickinson A. 2003. The effect of lesions of the basolateral amygdala on instrumental conditioning. *J. Neurosci.* 23:666–75

Barnes T, Kubota Y, Hu D, Jin DZ, Graybiel AM. 2005. Activity of striatal neurons reflects dynamic encoding and recoding of procedural memories. *Nature* 437:1158–61

Bartels A, Zeki S. 2004. The neural correlates of maternal and romantic love. *Neuroimage* 21:1155–66

Bayer HM, Glimcher PW. 2005. Midbrain dopamine neurons encode a quantitative reward prediction error signal. *Neuron* 47:129–41

Bayley PJ, Frascino JC, Squire LR. 2005. Robust habit learning in the absence of awareness and independent of the medial temporal lobe. *Nature* 436:550–53

Berridge KC. 1990. Comparative fine structure of action: rules of form and sequence in the grooming patterns of six rodent species. *Behaviour* 113:21–56

Berridge KC, Aldridge JW, Houchard KR, Zhuang X. 2005. Sequential superstereotypy of an instinctive fixed action pattern in hyperdopaminergic mutant mice: a model of obsessive compulsive disorder and Tourette's. *BMC Biol.* 3:4

Blazquez P, Fujii N, Kojima J, Graybiel AM. 2002. A network representation of response probability in the striatum. *Neuron* 33:973–82

Brainard MS, Doupe AJ. 2000. Interruption of a basal ganglia-forebrain circuit prevents plasticity of learned vocalizations. *Nature* 404:762–66

Canales JJ. 2005. Stimulant-induced adaptations in neostriatal matrix and striosome systems: transiting from instrumental responding to habitual behavior in drug addiction. *Neurobiol. Learn. Mem.* 83:93–103

Canales JJ, Graybiel AM. 2000. A measure of striatal function predicts motor stereotypy. *Nat. Neurosci.* 3:377–83

Colwill RM, Rescorla RA. 1985. Postconditioning devaluation of a reinforcer affects instrumental responding. *J. Exp. Psychol. Anim. Behav. Process.* 11:120–32

Cooper SJ, Dourish CT, eds. 1990. *Neurobiology of Stereotyped Behaviour*. Oxford, UK: Clarendon

Corbit LH, Balleine BW. 2005. Double dissociation of basolateral and central amygdala lesions on the general and outcome-specific forms of pavlovian-instrumental transfer. *J. Neurosci.* 25:962–70

Coutureau E, Killcross S. 2003. Inactivation of the infralimbic prefrontal cortex reinstates goal-directed responding in overtrained rats. *Behav. Brain Res.* 146:167–74

Crinion J, Turner R, Grogan A, Hanakawa T, Noppeney U, et al. 2006. Language control in the bilingual brain. *Science* 312:1537–40

Daw ND, Doya K. 2006. The computational neurobiology of learning and reward. *Curr. Opin. Neurobiol.* 16:199–204

Daw ND, Niv Y, Dayan P. 2005. Uncertainty-based competition between prefrontal and dorsolateral striatal systems for behavioral control. *Nat. Neurosci.* 8:1704–11

Dayan P. 2002. Motivated reinforcement learning. In *Advances in Neural Information Processing Systems*, ed. TG Dietterich, S Becker, Z Ghahramans, pp. 11–18. San Mateo, CA: Morgan Kaufmann

Dayan P, Balleine BW. 2002. Reward, motivation, and reinforcement learning. *Neuron* 36:285–98

DeCoteau WE, Thorn CA, Gibson DJ, Courtemanche R, Mitra P, et al. 2007a. Learning-related coordination of striatal and hippocampal theta rhythms during acquisition of a procedural maze task. *Proc. Natl. Acad. Sci. USA* 104:5644–49

DeCoteau WE, Thorn C, Gibson DJ, Courtemanche R, Mitra P, et al. 2007b. Oscillations of local field potentials in the rat dorsal striatum during spontaneous and instructed behaviors. *J. Neurophysiol.* 97:3800–5

de la Fuente-Fernandez R, Ruth TJ, Sossi V, Schulzer M, Calne DB, Stoessl AJ. 2001. Expectation and dopamine release: mechanism of the placebo effect in Parkinson's disease. *Science* 293:1164–66

de Quervain DJ, Fischbacher U, Treyer V, Schellhammer M, Schnyder U, et al. 2004. The neural basis of altruistic punishment. *Science* 305:1254–58

Devan BD, White NM. 1999. Parallel information processing in the dorsal striatum: relation to hippocampal function. *J. Neurosci.* 19:2789–98

Diamond J. 1986. Animal art: variation in bower decorating style among male bowerbirds *Amblyornis inornatus*. *Proc. Natl. Acad. Sci. USA* 83:3042–46

Djurfeldt M, Ekeberg Ö, Graybiel AM. 2001. Cortex-basal ganglia interaction and attractor states. *Neurocomputing* 38–40:573–79

Dommett E, Coizet V, Blaha CD, Martindale J, Lefebvre V, et al. 2005. How visual stimuli activate dopaminergic neurons at short latency. *Science* 307:1476–79

Doya K. 2000. Complementary roles of basal ganglia and cerebellum in learning and motor control. *Curr. Opin. Neurobiol.* 10:732–39

Doya K. 2002. Metalearning and neuromodulation. *Neural. Netw.* 15:495–506

Doyon J, Benali H. 2005. Reorganization and plasticity in the adult brain during learning of motor skills. *Curr. Opin. Neurobiol.* 15:161–67

Doyon J, Penhune V, Ungerleider LG. 2003. Distinct contribution of the cortico-striatal and cortico-cerebellar systems to motor skill learning. *Neuropsychologia* 41:252–62

Elliott R, Newman JL, Longe OA, Deakin JFW. 2004. Instrumental responding for rewards is associated with enhanced neuronal response in subcortical reward systems. *Neuroimage* 21:984–90

Everitt BJ, Dickinson A, Robbins TW. 2001. The neuropsychological basis of addictive behaviour. *Brain Res. Brain Res. Rev.* 36:129–38

Everitt BJ, Robbins TW. 2005. Neural systems of reinforcement for drug addiction: from actions to habits to compulsion. *Nat. Neurosci.* 8:1481–89

Faure A, Haberland U, Conde F, El Massioui N. 2005. Lesion to the nigrostriatal dopamine system disrupts stimulus-response habit formation. *J. Neurosci.* 25:2771–80

Fiorillo CD, Tobler PN, Schultz W. 2003. Discrete coding of reward probability and uncertainty by dopamine neurons. *Science* 299:1898–902

Flaherty AW, Graybiel AM. 1994. Input-output organization of the sensorimotor striatum in the squirrel monkey. *J. Neurosci.* 14:599–610

Foerde K, Knowlton BJ, Poldrack RA. 2006. Modulation of competing memory systems by distraction. *Proc. Natl. Acad. Sci. USA* 103:11778–83

Fuchs RA, Branham RK, See RE. 2006. Different neural substrates mediate cocaine seeking after abstinence versus extinction training: a critical role for the dorsolateral caudate-putamen. *J. Neurosci.* 26:3584–88

Fujii N, Graybiel A. 2003. Representation of action sequence boundaries by macaque prefrontal cortical neurons. *Science* 301:1246–49

Fujii N, Graybiel A. 2005. Time-varying covariance of neural activities recorded in striatum and frontal cortex as monkeys perform sequential-saccade tasks. *Proc. Natl. Acad. Sci. USA* 102:9032–37

Georgopoulos AP, Stefanis CN. 2007. Local shaping of function in the motor cortex: motor contrast, directional tuning. *Brain Res. Rev.* 55:383–89

Glimcher PW. 2003. *Decisions, Uncertainty, and the Brain: The Science of Neuroeconomics*. Cambridge, MA: MIT Press

Goto S, Lee LV, Munoz EL, Tooyama I, Tamiya G, et al. 2005. Functional anatomy of the basal ganglia in X-linked recessive dystonia-parkinsonism. *Ann. Neurol.* 58:7–17

Gottfried JA, O'Doherty J, Dolan RJ. 2003. Encoding predictive reward value in human amygdala and orbitofrontal cortex. *Science* 301:1104–7

Grabli D, McCairn K, Hirsch EC, Agid Y, Feger J, et al. 2004. Behavioural disorders induced by external globus pallidus dysfunction in primates: I. Behavioural study. *Brain* 127:2039–54

Graybiel AM. 1990. Neurotransmitters and neuromodulators in the basal ganglia. *Trends Neurosci.* 13:244–54

Graybiel AM. 1997. The basal ganglia and cognitive pattern generators. *Schizophr. Bull.* 23:459–69

Graybiel AM. 1998. The basal ganglia and chunking of action repertoires. *Neurobiol. Learn. Mem.* 70:119–36

Graybiel AM. 2005. The basal ganglia: learning new tricks and loving it. *Curr. Opin. Neurobiol.* 15:638–44

Graybiel AM, Aosaki T, Flaherty AW, Kimura M. 1994. The basal ganglia and adaptive motor control. *Science* 265:1826–31

Graybiel AM, Canales JJ, Capper-Loup C. 2000. Levodopa-induced dyskinesias and dopamine-dependent stereotypies: a new hypothesis. *Trends Neurosci.* 23:S71–77

Graybiel AM, Fujii N, Jin DZ. 2005. Representation of time and states in the macaque prefrontal cortex and striatum during sequential saccade tasks. *Soc. Neurosci. Abstr. Viewer/Itiner.* 400.7

Graybiel AM, Rauch SL. 2000. Toward a neurobiology of obsessive-compulsive disorder. *Neuron* 28:343–47

Graybiel AM, Saka E. 2004. The basal ganglia and the control of action. In *The New Cognitive Neurosciences*, ed. MS Gazzaniga, pp. 495–510. Cambridge, MA: MIT Press. 3rd ed.

Haber SN, Fudge JL, McFarland NR. 2000. Striatonigrostriatal pathways in primates form an ascending spiral from the shell to the dorsolateral striatum. *J. Neurosci.* 20:2369–82

Harbaugh WT, Mayr U, Burghart DR. 2007. Neural responses to taxation and voluntary giving reveal motives for charitable donations. *Science* 316:1622–25

Haruno M, Kawato M. 2006. Different neural correlates of reward expectation and reward expectation error in the putamen and caudate nucleus during stimulus-action-reward association learning. *J. Neurophysiol.* 95:948–59

Hernandez PJ, Sadeghian K, Kelley AE. 2002. Early consolidation of instrumental learning requires protein synthesis in the nucleus accumbens. *Nat. Neurosci.* 5:1327–31

Hernandez PJ, Schiltz CA, Kelley AE. 2006. Dynamic shifts in corticostriatal expression patterns of the immediate early genes Homer 1a and Zif268 during early and late phases of instrumental training. *Learn. Mem.* 13:599–608

Hikosaka O, Nakahara H, Rand MK, Sakai K, Lu X, et al. 1999. Parallel neural networks for learning sequential procedures. *Trends Neurosci.* 22:464–71

Hoshi E, Tremblay L, Feger J, Carras PL, Strick PL. 2005. The cerebellum communicates with the basal ganglia. *Nat. Neurosci.* 8:1491–93

Houk JC, Wise SP. 1995. Distributed modular architectures linking basal ganglia, cerebellum, and cerebral cortex: their role in planning and controlling action. *Cereb. Cortex* 5:95–110

Hsu M, Bhatt M, Adolphs R, Tranel D, Camerer CF. 2005. Neural systems responding to degrees of uncertainty in human decision-making. *Science* 310:1680–83

Hyman SE, Malenka RC, Nestler EJ. 2006. Neural mechanisms of addiction: the role of reward-related learning and memory. *Annu. Rev. Neurosci.* 29:565–98

Isoda M, Hikosaka O. 2007. Switching from automatic to controlled action by monkey medial frontal cortex. *Nat. Neurosci.* 10:240–48

Ito R, Dalley JW, Robbins TW, Everitt BJ. 2002. Dopamine release in the dorsal striatum during cocaine-seeking behavior under the control of a drug-associated cue. *J. Neurosci.* 22:6247–53

Izquierdo A, Suda RK, Murray EA. 2004. Bilateral orbital prefrontal cortex lesions in rhesus monkeys disrupt choices guided by both reward value and reward contingency. *J. Neurosci.* 24:7540–48

James W. 1950 [1890]. *The Principles of Psychology*. New York: Dover

Jog M, Kubota Y, Connolly CI, Hillegaart V, Graybiel AM. 1999. Building neural representations of habits. *Science* 286:1745–49

Joyce EM, Iversen SD. 1984. Dissociable effects of 6-OHDA-induced lesions of neostriatum on anorexia, locomotor activity and stereotypy: the role of behavioural competition. *Psychopharmacology (Berl.)* 83:363–66

Kalanithi PS, Zheng W, Kataoka Y, DiFiglia M, Grantz H, et al. 2005. Altered parvalbumin-positive neuron distribution in basal ganglia of individuals with Tourette syndrome. *Proc. Natl. Acad. Sci. USA* 102:13307–12

Kalivas PW, Volkow N, Seamans J. 2005. Unmanageable motivation in addiction: a pathology in prefrontal-accumbens glutamate transmission. *Neuron* 45:647–50

Kalivas PW, Volkow ND. 2005. The neural basis of addiction: a pathology of motivation and choice. *Am. J. Psychiatry* 162:1403–13

Kao MH, Brainard MS. 2006. Lesions of an avian basal ganglia circuit prevent context-dependent changes to song variability. *J. Neurophysiol.* 96:1441–55

Kawato M, Gomi H. 1992. A computational model of four regions of the cerebellum based on feedback-error learning. *Biol. Cybern.* 68:95–103

Killcross S, Coutureau E. 2003. Coordination of actions and habits in the medial prefrontal cortex of rats. *Cereb. Cortex* 13:400–8

King-Casas B, Tomlin D, Anen C, Camerer CF, Quartz SR, Montague PR. 2005. Getting to know you: reputation and trust in a two-person economic exchange. *Science* 308:78–83

Knowlton BJ, Mangels JA, Squire LR. 1996. A neostriatal habit learning system in humans. *Science* 273:1399–402

Kobayashi K, Uno H, Okanoya K. 2001. Partial lesions in the anterior forebrain pathway affect song production in adult Bengalese finches. *NeuroReport* 12:353–58

Koechlin E, Jubault T. 2006. Broca's area and the hierarchical organization of human behavior. *Neuron* 50:963–74

Kubota Y, DeCoteau WE, Liu J, Graybiel AM. 2002a. Task-related activity in the medial striatum during performance of a conditional T-maze task. *Soc. Neurosci. Abstr. Viewer/Itiner.* 765.7

Kubota Y, DeCoteau WE, Liu J, Graybiel AM. 2002b. Task-related activity in the medial striatum during performance of a conditional T-maze task. *Soc. Neurosci. Abstr. Viewer/Itiner.* 765.7

Leckman JF, Riddle MA. 2000. Tourette's syndrome: when habit-forming systems form habits of their own. *Neuron* 28:349–54

Lieberman MD, Chang GY, Chiao J, Bookheimer SY, Knowlton BJ. 2004. An event-related fMRI study of artificial grammar learning in a balanced chunk strength design. *J. Cogn. Neurosci.* 16:427–38

Liegeois F, Baldeweg T, Connelly A, Gadian DG, Mishkin M, Vargha-Khadem F. 2003. Language fMRI abnormalities associated with *FOXP2* gene mutation. *Nat. Neurosci.* 6:1230–37

Logan GD. 1988. Toward an instance theory of automatization. *Psychol. Rev.* 95:492–527

Matsumoto M, Hikosaka O. 2007. Lateral habenula as a source of negative reward signals in dopamine neurons. *Nature* 447:1111–15

Matsuzaka Y, Picard N, Strick PL. 2007. Skill representation in the primary motor cortex after long-term practice. *J. Neurophysiol.* 97:1819–32

Mink JW. 2001. Basal ganglia dysfunction in Tourette's syndrome: a new hypothesis. *Pediatr. Neurol.* 25:190–98

Mink JW, Walkup J, Frey KA, Como P, Cath D, et al. 2006. Patient selection and assessment recommendations for deep brain stimulation in Tourette syndrome. *Mov. Disord.* 21:1831–38

Montague PR, Berns GS. 2002. Neural economics and the biological substrates of valuation. *Neuron* 36:265–84

Montague PR, Dayan P, Sejnowski TJ. 1996. A framework for mesencephalic dopamine systems based on predictive Hebbian learning. *J. Neurosci.* 16:1936–47

Montague PR, King-Casas B, Cohen JD. 2006. Imaging valuation models in human choice. *Annu. Rev. Neurosci.* 29:417–48

Moody TD, Bookheimer SY, Vanek Z, Knowlton BJ. 2004. An implicit learning task activates medial temporal lobe in patients with Parkinson's disease. *Behav. Neurosci.* 118:438–42

Morris G, Arkadir D, Nevet A, Vaadia E, Bergman H. 2004. Coincident but distinct messages of midbrain dopamine and striatal tonically active neurons. *Neuron* 43:133–43

Morris G, Nevet A, Arkadir D, Vaadia E, Bergman H. 2006. Midbrain dopamine neurons encode decisions for future action. *Nat. Neurosci.* 9:1057–63

Nakahara H, Itoh H, Kawagoe R, Takikawa Y, Hikosaka O. 2004. Dopamine neurons can represent context-dependent prediction error. *Neuron* 41:269–80

Nelson A, Killcross S. 2006. Amphetamine exposure enhances habit formation. *J. Neurosci.* 26:3805–12

Nissen MJ, Bullemer P. 1987. Attentional requirements of learning: evidence from performance measures. *Cognit. Psychol.* 19:1–32

Niv Y, Duff MO, Dayan P. 2005. Dopamine, uncertainty and TD learning. *Behav. Brain Funct.* 1:6

Niv Y, Joel D, Dayan P. 2006. A normative perspective on motivation. *Trends Cogn. Sci.* 10:375–81

Nordquist RE, Voorn P, de Mooij-van Malsen JG, Joosten RN, Pennartz CM, Vanderschuren LJ. 2007. Augmented reinforcer value and accelerated habit formation after repeated amphetamine treatment. *Eur. Neuropsychopharmacol.* 17:532–40

O'Doherty J, Dayan P, Schultz J, Deichmann R, Friston K, Dolan RJ. 2004. Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science* 304:452–54

Olveczky BP, Andalman AS, Fee MS. 2005. Vocal experimentation in the juvenile songbird requires a basal ganglia circuit. *PLoS Biol.* 3:e153

O'Reilly RC, Frank MJ. 2006. Making working memory work: a computational model of learning in the prefrontal cortex and basal ganglia. *Neural Comput.* 18:283–328

Packard MG, Knowlton BJ. 2002. Learning and memory functions of the basal ganglia. *Annu. Rev. Neurosci.* 25:563–93

Packard MG, McGaugh JL. 1996. Inactivation of hippocampus or caudate nucleus with lidocaine differentially affects expression of place and response learning. *Neurobiol. Learn. Mem.* 65:65–72

Padoa-Schioppa C, Assad JA. 2006. Neurons in the orbitofrontal cortex encode economic value. *Nature* 441:223–26

Palmiter RD. 2007. Is dopamine a physiologically relevant mediator of feeding behavior? *Trends Neurosci.* 30:375–81

Paton JJ, Belova MA, Morrison SE, Salzman CD. 2006. The primate amygdala represents the positive and negative value of visual stimuli during learning. *Nature* 439:865–70

Phillips PE, Stuber GD, Heien ML, Wightman RM, Carelli RM. 2003. Subsecond dopamine release promotes cocaine seeking. *Nature* 422:614–18

Platt ML, Glimcher PW. 1999. Neural correlates of decision variables in parietal cortex. *Nature* 400:233–38

Poldrack RA, Clark J, Pare-Blagoev EJ, Shohamy D, Creso Moyano J, et al. 2001. Interactive memory systems in the human brain. *Nature* 414:546–50

Poldrack RA, Sabb FW, Foerde K, Tom SM, Asarnow RF, et al. 2005. The neural correlates of motor skill automaticity. *J. Neurosci.* 25:5356–64

Porrino LJ, Lyons D, Smith HR, Daunais JB, Nader MA. 2004. Cocaine self-administration produces a progressive involvement of limbic, association, and sensorimotor striatal domains. *J. Neurosci.* 24:3554–62

Rauch SL, Wedig MM, Wright CI, Martis B, McMullin KG, et al. 2006. Functional magnetic resonance imaging study of regional brain activation during implicit sequence learning in obsessive-compulsive disorder. *Biol. Psychiatry* 61:330–36

Redgrave P, Gurney K. 2006. The short-latency dopamine signal: a role in discovering novel actions? *Nat. Rev. Neurosci.* 7:967–75

Redish AD. 2004. Addiction as a computational process gone awry. *Science* 306:1944–47

Ridley RM. 1994. The psychology of perseverative and stereotyped behaviour. *Prog. Neurobiol.* 44:221–31

Robinson TE, Kolb B. 2004. Structural plasticity associated with exposure to drugs of abuse. *Neuropharmacology* 47(Suppl. 1):33–46

Romo R, Schultz W. 1990. Dopamine neurons of the monkey midbrain: contingencies of response to active touch during self-initiated arm movements. *J. Neurophysiol.* 63:592–606

Saka E, Goodrich C, Harlan P, Madras BK, Graybiel AM. 2004. Repetitive behaviors in monkeys are linked to specific striatal activation patterns. *J. Neurosci.* 24:7557–65

Salat DH, van der Kouwe AJ, Tuch DS, Quinn BT, Fischl B, et al. 2006. Neuroimaging H.M.: a 10-year follow-up examination. *Hippocampus* 16:936–45

Samejima K, Ueda Y, Doya K, Kimura M. 2005. Representation of action-specific reward values in the striatum. *Science* 310:1337–40

Satoh T, Nakai S, Sato T, Kimura M. 2003. Correlated coding of motivation and outcome of decision by dopamine neurons. *J. Neurosci.* 23:9913–23

Schultz W, Dayan P, Montague PR. 1997. A neural substrate of prediction and reward. *Science* 275:1593–99

Self DW. 2004. Regulation of drug-taking and -seeking behaviors by neuroadaptations in the mesolimbic dopamine system. *Neuropharmacology* 47(Suppl. 1):242–55

Sellings LH, Clarke PB. 2003. Segregation of amphetamine reward and locomotor stimulation between nucleus accumbens medial shell and core. *J. Neurosci.* 23:6295–303

Shuler MG, Bear MF. 2006. Reward timing in the primary visual cortex. *Science* 311:1606–9

Smith MA, Shadmehr R. 2005. Intact ability to learn internal models of arm dynamics in Huntington's disease but not cerebellar degeneration. *J. Neurophysiol.* 93:2809–21

Smith-Roe SL, Kelley AE. 2000. Coincident activation of NMDA and dopamine D1 receptors within the nucleus accumbens core is required for appetitive instrumental learning. *J. Neurosci.* 20:7737–42

Sugrue LP, Corrado GS, Newsome WT. 2004. Matching behavior and the representation of value in the parietal cortex. *Science* 304:1782–87

Sutton RS, Barto AG. 1998. *Reinforcement Learning: An Introduction*. Cambridge, MA: MIT Press

Suzuki WA. 2007. Associative learning signals in the monkey medial temporal lobe. Presented at *Cosyne 2007*, Salt Lake City, Utah

Tanaka SC, Doya K, Okada G, Ueda K, Okamoto Y, Yamawaki S. 2004. Prediction of immediate and future rewards differentially recruits cortico-basal ganglia loops. *Nat. Neurosci.* 7:887–93

Tanji J, Hoshi E. 2007. Role of the lateral prefrontal cortex in executive behavioral control. *Physiol. Rev.* 88:37–57

Tettamanti M, Moro A, Messa C, Moresco RM, Rizzo G, et al. 2005. Basal ganglia and language: phonology modulates dopaminergic release. *NeuroReport* 16:397–401

Thorn CA, Graybiel AM. 2007. Medial and lateral striatal LFPs exhibit task-dependent patterns of coherence in multiple frequency bands. *Soc. Neurosci. Abstr. Viewer/Itiner.* 622.14

Tinbergen N. 1953. *The Herring Gull's World: A Study of the Social Behaviour of Birds*. London: Collins

Tippett LJ, Waldvogel HJ, Thomas SJ, Hogg VM, van Roon-Mom W, et al. 2007. Striosomes and mood dysfunction in Huntington's disease. *Brain* 130:206–21

Tobler PN, Fiorillo CD, Schultz W. 2005. Adaptive coding of reward value by dopamine neurons. *Science* 307:1642–45

Tricomi EM, Delgado MR, Fiez JA. 2004. Modulation of caudate activity by action contingency. *Neuron* 41:281–92

Ungless MA, Magill PJ, Bolam JP. 2004. Uniform inhibition of dopamine neurons in the ventral tegmental area by aversive stimuli. *Science* 303:2040–42

Voermans NC, Petersson KM, Daudey L, Weber B, Van Spaendonck KP, et al. 2004. Interaction between the human hippocampus and the caudate nucleus during route recognition. *Neuron* 43:427–35

Volkow ND, Wang GJ, Telang F, Fowler JS, Logan J, et al. 2006. Cocaine cues and dopamine in dorsal striatum: mechanism of craving in cocaine addiction. *J. Neurosci.* 26:6583–88

Voon V, Potenza MN, Thomsen T. 2007. Medication-related impulse control and repetitive behaviors in Parkinson's disease. *Curr. Opin. Neurol.* 20:484–92

Waelti P, Dickinson A, Schultz W. 2001. Dopamine responses comply with basic assumptions of formal learning theory. *Nature* 412:43–48

Wellman LL, Gale K, Malkova L. 2005. $GABA_A$-mediated inhibition of basolateral amygdala blocks reward devaluation in macaques. *J. Neurosci.* 25:4577–86

Willingham DB, Salidis J, Gabrieli JD. 2002. Direct comparison of neural systems mediating conscious and unconscious skill learning. *J. Neurophysiol.* 88:1451–60

Willuhn I, Steiner H. 2006. Motor-skill learning-associated gene regulation in the striatum: effects of cocaine. *Neuropsychopharmacology* 31:2669–82

Wise RA. 2004. Dopamine, learning and motivation. *Nat. Rev. Neurosci.* 5:483–94

Wong DF, Kuwabara H, Schretlen DJ, Bonson KR, Zhou Y, et al. 2006. Increased occupancy of dopamine receptors in human striatum during cue-elicited cocaine craving. *Neuropsychopharmacology* 31:2716–27

Yin HH, Knowlton BJ. 2004. Contributions of striatal subregions to place and response learning. *Learn. Mem.* 11:459–63

Yin HH, Knowlton BJ, Balleine BW. 2005. Blockade of NMDA receptors in the dorsomedial striatum prevents action-outcome learning in instrumental conditioning. *Eur. J. Neurosci.* 22:505–12

Zald DH, Boileau I, El-Dearedy W, Gunn R, McGlone F, et al. 2004. Dopamine transmission in the human striatum during monetary reward tasks. *J. Neurosci.* 24:4105–12

Zink CF, Pagnoni G, Martin-Skurski ME, Chappelow JC, Berns GS. 2004. Human striatal responses to monetary reward depend on saliency. *Neuron* 42:509–17

Crittenden J, Sauvage M, Cepeda C, Andre V, Costa C, et al. 2007. *CalDAG-GEFI modulates behavioral sensitization to psycho motor stimulants and is required for cortico-striatal long-term potentiation*. Presented at Annu. Meet. Am. Coll. Neuropsychopharmacol., 46th, Boca Raton, FL

Welch J, Lu J, Rodriguez RM, Trotta NC, Peca J, et al. 2007. Cortico-striatal synaptic defects and OCD-like behaviours in Sapap3-mutant mice. *Nature* 448:894–900

NOTE ADDED IN PROOF

Valuable new mouse models of disorders involving action since this review was submitted include emerging Sapap-3 mutant mice and CalDAG-GEF I mutant mice (Crittenden et al. 2007, Welch et al. 2007).

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