

The Basal Ganglia and Chunking of Action Repertoires

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The basal ganglia have been shown to contribute to habit and stimulus–response (S–R) learning. These forms of learning have the property of slow acquisition and, in humans, can occur without conscious awareness. This paper proposes that one aspect of basal ganglia-based learning is the recoding of cortically derived information within the striatum. Modular corticostriatal projection patterns, demonstrated experimentally, are viewed as producing recoded templates suitable for the gradual selection of new input–output relations in cortico-basal ganglia loops. Recordings from striatal projection neurons and interneurons show that activity patterns in the striatum are modified gradually during the course of S–R learning. It is proposed that this recoding within the striatum can chunk the representations of motor and cognitive action sequences so that they can be implemented as performance units. This scheme generalizes Miller's notion of information chunking to action control. The formation and the efficient implementation of action chunks are viewed as being based on predictive signals. It is suggested that information chunking provides a mechanism for the acquisition and the expression of action repertoires that, without such information compression would be biologically unwieldy or difficult to implement. The learning and memory functions of the basal ganglia are thus seen as core features of the basal ganglia's influence on motor and cognitive pattern generators. © 1998 Academic Press

INTRODUCTION

James Parkinson might have been quite surprised if told that the basal ganglia, disabled in the disease that bears his name, have a prime function in learning and memory. He might also have questioned the view that the basal ganglia function mainly in the automatic execution of motor acts or in implementing habits. His idea was that the basal ganglia translate the dictates of the will into action, or, in more current phrasing, that the basal ganglia translate intention into action. In this paper, it is argued that these disparate views of basal ganglia function are neither contradictory nor mutually exclusive. This paper suggests that the basal ganglia may bring together these functions in part by promoting the building up of performance units made up of multiple acts that can be implemented in a particular temporal order—a sensorimotor form of chunking (Miller, 1956).

I take as a starting point the convincing experimental evidence that the

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basal ganglia function in some forms of implicit or "habit" learning (Graybiel, 1995; Hirsh, 1974; Knowlton, Mangels, & Squire, 1996; Mishkin, Malamut, & Bachevalier, 1984; Salmon & Butters, 1995; White, 1997). In humans, this learning is characterized by two key features: lack of awareness of the algorithm learned, and a slow rate of acquisition. In experimental animals, the apparently equivalent form of learning, or stimulus-response (S-R) learning, is also slowly acquired as a result of repeated pairing between stimulus and response. Both in humans and in other animals, habit learning can be dissociated from forms of explicit learning, mediated by hippocampal-medial temporal cortical systems, and from affect-related learning associated with limbic structures such as the amygdala (Kesner, Bolland, & Dakis, 1993; Knowlton et al., 1996; McDonald & White, 1993; Packard & McGaugh, 1996; Reading, Dunnett, & Robbins, 1991). It is becoming clear that tuning of neural responses is a very general phenomenon in the central nervous system, so that in many and perhaps even all cortical areas the response properties of neurons are modifiable, not fixed (Bichot, Schall, & Thompson, 1996; Chen & Wise, 1995, 1996; Iriki, Tanaka, & Iwamura, 1996; Mitz, Godschalk, & Wise, 1991; Wang, Merzenich, Sameshima, & Jenkins, 1995). The challenge to the neurobiologist in trying to understand the learning and memory functions of the basal ganglia is thus to determine how the particular behavioral characteristics of basal ganglia-based learning and memory are related to neural activity in the system, and to understand how the learning and memory functions of the basal ganglia relate to the core functions of the basal ganglia in the control of action.

CORTICOSTRIATAL MAPPING: COHERENT ACTIVATION, COMPARTMENTS, AND CONVERGENCE REMAPPING

The largest single input to the striatum is from the neocortex, and this input system is thought to be the main information source for basal ganglia circuits. Most of these inputs terminate on the spines of the main type of striatal neuron, the medium spiny neurons. As many as 10,000 cortical inputs may converge on a single projection neuron, and any single input fiber may contact many such cells (Wilson, 1995). Wilson and his coworkers have shown that the membrane potentials of these striatal projection neurons fluctuate between down-states, in which they are hyperpolarized to ca. -80 mV and in which they do not fire action potentials, and up-states, in which the neurons are partially depolarized (to ca. -50 mV) and in which afferent inputs can generate action potentials (Wilson, 1993). The cortical inputs are glutamatergic, and the projection neurons have both non-NMDA and NMDA receptors (Kita, 1996). When the projection neurons are in down-state, NMDA receptors are blocked, and glutamate acts through AMPA/kainate receptors. Under conditions of strong and temporally coherent activation, driving the neurons into up-state, NMDA receptors begin to become active with further inputs, promoting longer postsynaptic response times to favor input summation and also favoring NMDA-receptor mediated forms of plasticity (Kita, 1996).

How could this coherence-dependent mechanism affect the mapping of cortical inputs to the striatum? One answer may lie in the distributed yet clumpy anatomy of many corticostriatal projection systems. Inputs from a small site in the neocortex tend to be widely distributed in the striatum, but they also tend to form a labyrinthine series of partially interconnected clusters. The

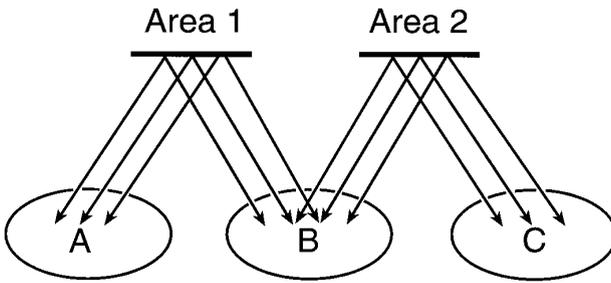


FIG. 1. Modular remapping in corticostriatal systems. Example shows two cortical areas (Area 1, Area 2) with distributed, clumpy projections to matrixomes in the striatum (A, B, C). It is proposed that such differential patterns of modular divergence and convergence set up templates in the striatum that can be used to recode cortical information.

multiple input clusters from functionally related areas can overlap selectively and quite precisely. For example, inputs from the cortex of the hand representations of primary somatosensory (SI) areas 3b and 1, and of the primary motor (MI) hand cortex representation overlap with one another, whereas they do not overlap with input from cortex of the contralateral MI hand representations (Flaherty & Graybiel, 1993). We have suggested that systems of input modules (called matrixomes) could serve as templates for forming new associations in the striatum and for selecting which inputs lead to output activation (Flaherty & Graybiel, 1994; Malach & Graybiel, 1986; Parthasarathy & Graybiel, 1992). The fact that the dendritic trees of many projection neurons in the striatum are also oriented and in some cases curved at boundaries of modules provides anatomical support for this idea (Walker, Arbuthnott, Baughman, & Graybiel, 1993; Walker & Graybiel, 1993).

This input organization not only may implement ongoing corticostriatal transmission promoting selection of output lines, but also could critically shape corticostriatal remapping, given the up-state/down-state fluctuations of striatal projection neurons. In the example shown in Fig. 1, inputs from cortical areas 1 and 2 overlap in striatal matrixome B but not in matrixomes A and C. This situation, given equal input efficacies, should promote B neurons achieving up-state more readily than A and C neurons. This would lead to more firing in B neurons, but also would lead to more activation of NMDA receptors in B neurons, which in turn could lead to long-term potentiation or depression (LTP or LTD), both demonstrated for corticostriatal synapses (Calabresi, Pisani, Mercuri, & Bernardi, 1996; Calabresi, Salardi, Pisani, Baik, Centonze, Mercuri, Bernardi, & Borrelli, 1997; Charpier & Deniau, 1997). On this basis, we suggest that coherent up-state transitions in modular groups of striatal projection neurons can promote long-term changes as well as transient activation evidenced by bursts of action potentials. A function of convergence mapping onto matrixomes thus would be to allow gradual changes in the input-output relationships of particular populations of striatal neurons. The incremental nature of the corticostriatal reordering, dependent on repetition of particular coherent input patterns, could underlie in part the gradual learning mediated by the basal ganglia. Not all cortical inputs are equally patchy (see, e.g., Eblen & Graybiel, 1995; Parthasarathy & Graybiel, 1992), and modularity is not necessary for convergence. Yet nearly all cortical areas show some modu-

larity. This could provide a fairly general mechanism for corticostriatal remapping during learning (and a critical one during postnatal development, given that dendrites and their synaptic inputs mature postnatally).

CORTICOSTRIATAL REMAPPING AND S-R LEARNING

Why should this form of remapping promote an S-R learning function for the striatum (basal ganglia) rather than an S-S or context learning function (thought to be the specialty of the hippocampal/medial temporal system) (Eichenbaum, 1995)? At the systems level, one answer is that the outputs of the striatum (via its pallidal and nigral targets) are oriented mainly toward motor and cognitive action systems of the frontal cortex and toward brainstem premotor regions such as the superior colliculus. By contrast, much of the outflow from the hippocampal formation and medial temporal cortex leads to other association cortex and to the septum and anterior thalamic/mammillary body circuits (Suzuki, 1996). The output pattern of the striatum suggests that sufficient coherent activation of striatal neurons leads to the generation of action potentials off up-states, and that these can set off changes in activity leading through the system of connections and loop circuits of the basal ganglia.

In the example of cortical areas 1 and 2 becoming active, B neurons would again be favored over A and C neurons by coherent activation of areas 1 and 2, and repetitions could lead to an increased or decreased probability of B responses, respectively, through LTP or LTD (Fig. 2). Based on fast GABA transmission from the striatum to the pallidum and the substantia nigra, LTP would lead to decreased outflow on activation of B neurons and LTD to increased outflow. But other long-term effects could also occur, in part through release of NMDA receptor-based effects in up-state. Coherent cortical activation imposed experimentally can lead to activation of immediate-early gene expression in striatal neurons (see Berretta, Parthasarathy, & Graybiel, 1997a; Liste, Rozas, Guerra, & Labandeira-Garcia, 1995; Sgambato, Abo, Rogard, Besson, & Deniau, 1997) and this activation is NMDA receptor dependent (Berretta et al., 1997a; Liste et al., 1995) and can be selective for particular matrisomes (Parthasarathy & Graybiel, 1997). Interestingly, among the documented genes known to be regulated in striatal projection neurons by cortical inputs are those coding for modulatory neuropeptides that are contained along with GABA in these neurons.

There is an estimated convergence of between about 30:1 (rat) to 80:1 (monkey) in striatal projections onto its target neurons (Percheron, 1989; Oorschot, 1996). This means that, again, coherent network-level activity must be crucial in determining whether the pallidal and nigral targets of striatal projection neurons fire. We have found one anatomical pattern that may serve this purpose (Flaherty & Graybiel, 1994). At least some of the input matrisomes of the primate putamen coincide with output matrisomes identified as clusters of projection neurons whose outputs converge toward small sites in the pallidum. This sets up a divergence-reconvergence architecture that bears a notable resemblance to the adaptive mixture of experts architecture of some learning theories (Jacobs, Jordan, Nowlan, & Hinton, 1991; Graybiel, Aosaki, Flaherty, & Kimura, 1994; see Fig. 3). In effect, the striatum then becomes like a hidden layer in which cortico-basal ganglia throughput can be tuned on moment-to-moment and long-term bases.

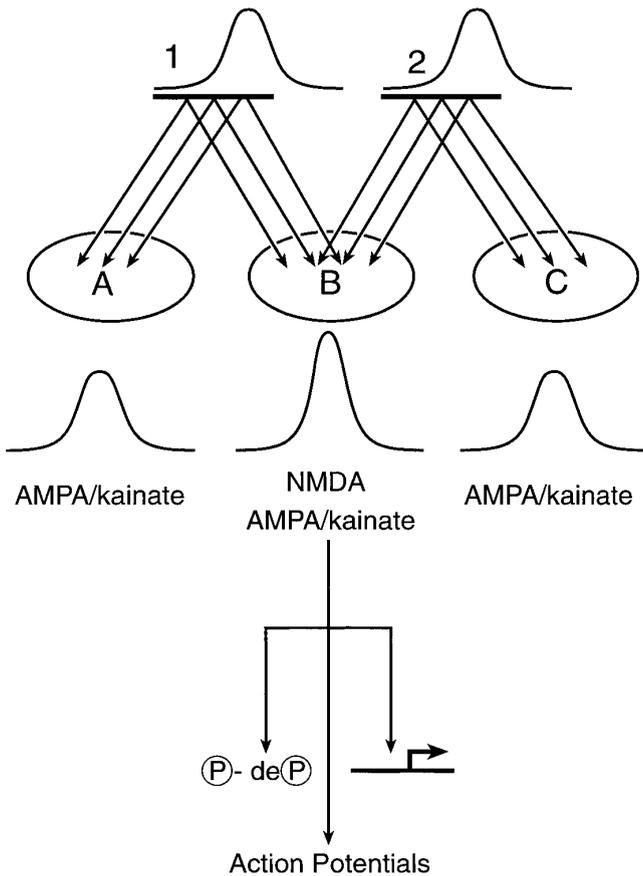


FIG. 2. Modifiable corticostriatal templates. The example shows two cortical areas (1 and 2, as in Fig. 1) with their similar activity levels shown by schematic X - Y plots. The areas project to striatal matrixosomes A, B, and C and their projections overlap in matrixosome B. It is proposed that such patterns of overlap and nonoverlap can have consequences for on-line corticostriatal throughput to striatal output targets (e.g., producing greater probability of up-state transitions and action potential firing of projection neurons in B), and also longer-term consequences, symbolized by changes in phosphorylation (P) states of intracellular messengers and receptors, and changes in gene expression ($\square \rightarrow$).

MODIFIABLE RESPONSES OF STRIATAL INTERNEURONS, GRADUAL S-R LEARNING, AND BINDING

“Context dependence” is a major response characteristic of striatal neurons, and the responses of many cells recorded in alert monkeys and other species are so specific (for example, to a particular sequence of movements or to a remembered stimulus) that the response must have been acquired through learning (Aosaki, Tsubokawa, Ishida, Watanabe, Graybiel, & Kimura, 1994b; Hikosaka, Sakamoto, & Usui, 1989a; Kermadi & Joseph, 1995; Romo, Scarnati, & Schultz, 1992; Schneider & Lidsky, 1981; Thorpe, Rolls, & Maddison, 1983).

To try to understand this modifiability, we are taking the approach of recording from the striatum during learning. We first studied, with Aosaki, Kimura, and colleagues, the tonically active neurons (TANs) of the primate striatum, because they were known to exhibit conditioned sensory responses

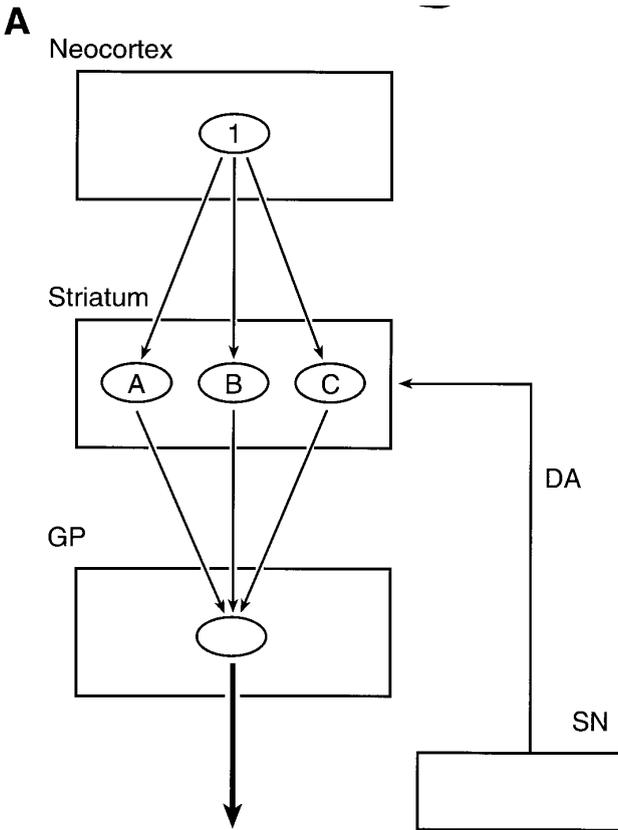


FIG. 3. Remapping in cortico-basal ganglia circuits as a mechanism for learning. (A) Example of divergent mapping from neocortex (1) to striatal matrixes (A, B, C) with reconvergence at the level of the globus pallidus (GP). Adapted from Flaherty and Graybiel (1994) and Graybiel *et al.* (1994). The network is shown to be modulated by dopamine (DA)-containing inputs from the substantia nigra (SN). Compare with the learning network in (B). (B) Diagram modified from Jacobs *et al.* (1991) showing adaptive mixture of networks model. Note similarity of architecture to pattern of connectivity in (A).

in monkeys highly trained on a sensorimotor conditioning task (Aosaki *et al.*, 1994b). The TANs are thought to be the cholinergic interneurons of the striatum (Aosaki, Kimura, & Graybiel, 1995). Recording during acquisition, we found that the TANs acquired these conditioned responses very gradually during the course of training. The gradual recruitment of TANs as the behavioral response was acquired fits well with the slow acquisition of implicit (S-R) learning. We reason that the TANs could serve an important function in the gradual stamping in of new S-R patterns in the striatal projection neurons influenced by TANs; they do not lead the behavioral response itself (Aosaki *et al.*, 1994b, 1995; Graybiel *et al.*, 1994). A particular characteristic of the TAN responses suggests that they could have an important influence on the time-sensitive aspects of this learning as well. The occurrence of their acquired responses (pauses in tonic firing) coincides within a similar ca. 270-ms time frame, which starts about 70 ms after the conditioned stimulus across a wide sector of the striatum (Aosaki *et al.*, 1995). This characteristic suggests that

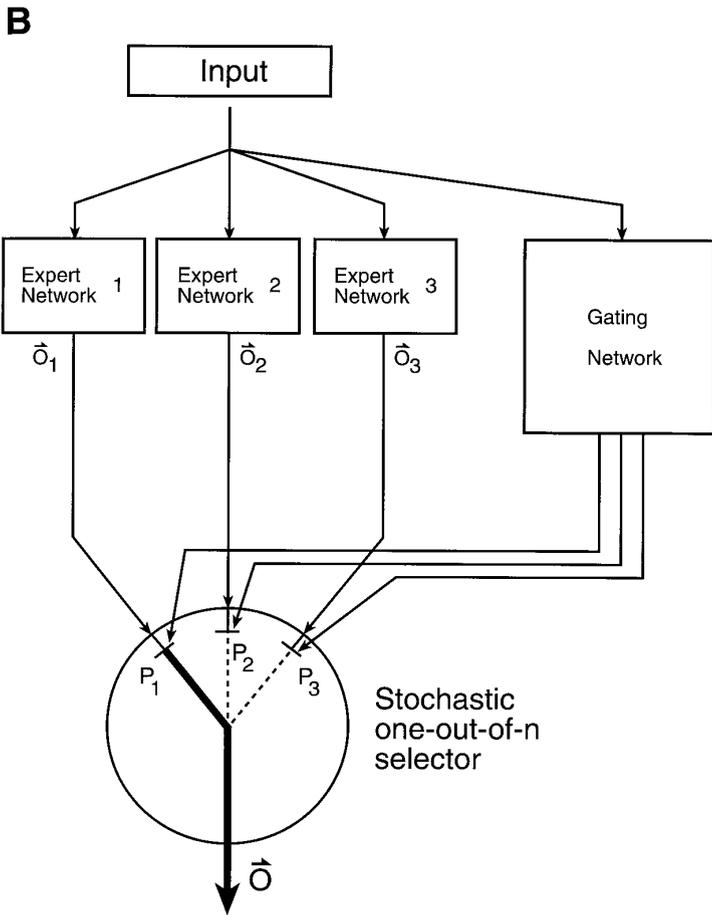


FIG. 3—Continued

these sparsely but widely distributed striatal interneurons could play a role in motor binding (Graybiel et al., 1994). For anterior parts of the striatum preferentially influencing prefrontal cortex, the TANs could serve a comparable binding function in the striatum (Graybiel, 1997). The acquired TAN responses could, as argued below, also produce a time-stamp signal useful in neuronal coding of serial-order tasks.

CHANGES IN ACTIVITY PATTERNS OF STRIATAL PROJECTION NEURONS DURING LEARNING

In ongoing experiments to be reported elsewhere (Jog, Connolly, Hillegaart, Wilson, & Graybiel, 1996; Jog, Kubota, Connolly, Iyengar, & Graybiel, 1997; Jog, et al., in preparation), we are recording with multiple tetrodes from populations of neurons in the striatum of rats undergoing behavioral training in a T-maze task. With this method, we record activity from projection neurons and can record from one small region for days without having to move the electrodes. This gives us samples of activity in populations of neurons across days of training on this task, chosen as an example of a striatum-based “win-

stay" task (McDonald & White, 1993). According to the behavioral characteristics of striatum-based learning, we would predict that the striatal projection neurons would only gradually change their firing patterns during behavioral training. And given the circuit-level considerations posed above, we would predict finding groups of nearby neurons with similar properties that would exhibit increased coherent activity during learning. These properties appear to hold. A particularly interesting finding is that at some recording sites the responses shift temporally, toward earlier phases of the behavioral trial (Jog et al., in preparation). This key feature may account for the repeated observation that in highly trained monkeys, "overtrained" on a variety of behavioral tasks, a large fraction of striatal neurons fire in an anticipatory mode early in the task (Alexander, 1987; Alexander & Crutcher, 1990; Hikosaka, Sakamoto, & Usui, 1989b; Jaeger, Gilman, & Aldridge, 1993). We hypothesize elsewhere that this predictive firing can be built up on the basis of repeated task exposure with the kinetics of implicit learning (Jog et al., in preparation).

MODIFIABLE CORTICOSTRIATAL TEMPLATES AND S-R LEARNING

These new ensemble recording data demonstrate that gradual changes occur in the firing of striatal projection neurons as animals acquire behavioral learning. They do not show whether these changes are initiated in the striatum or elsewhere (for example, in the neocortex). There is ample reason to think, however, that some of the changes occurring during learning involve altering the parameters of corticostriatal transmission.

The most extensively studied modifier of corticostriatal transmission is the nigrostriatal system (see, e.g., Calabresi et al., 1996; Kötter, 1994, for review). Dopamine receptor activation (or inactivation) affects both on-line activity of striatal neurons and plasticity in these neurons. For example, the production of LTP and LTD by tetanic stimulation *in vitro* critically depends on dopamine receptor activation (Calabresi et al., 1996, 1997; Wickens, Begg, & Arbuthnott, 1996). Dopamine receptor function also affects the capacity of cortical stimulation to induce gene expression in striatal neurons via an NMDA-dependent mechanism (Berretta, Sachs, & Graybiel, 1996; Berretta, Sachs, & Graybiel, in preparation; Liste et al., 1995). These results suggest that dopamine can affect the efficacy of corticostriatal transmission with long-term consequences that could affect striatum-based learning and memory.

This conclusion is strongly supported by behavioral and physiological evidence. The early experiments of Routtenberg and his colleagues demonstrated that dopaminergic inputs to the rat's striatum are necessary for memory consolidation in a passive avoidance test (Routtenberg & Kim, 1978). This role for dopamine has been confirmed (White, 1997). In the monkey, we found that dopaminergic inputs are required for the expression of acquired responses of TANs to conditioned stimuli (Aosaki, Graybiel, & Kimura, 1994a). It is now known that dopamine-containing neurons in the midbrain nigral/ventral tegmental area complex can fire preferentially in response to novel salient stimuli and primary rewards and, in conditioned monkeys, to conditioned stimuli but no longer to the rewards themselves (Schultz, 1997). These experiments suggest that the dopamine-containing neurons of the midbrain serve a reinforcement function during learning and a predictive function after learning and that they affect the expression of learned responses.

Other striatal afferent systems are also likely to produce major effects on striatal neurons during learning. These include inputs from the thalamus and the amygdala, recursive inputs from the pallidum, and the large serotonergic inputs to the striatum and to the substantia nigra pars compacta (see Parent & Hazrati, 1995). Few direct studies of such potential directional effects have yet been made. But our collaborative work with Kimura and associates shows that inputs to the striatum from the intralaminar nuclei are necessary for expression of acquired TAN responses (Matsumoto, Hanakawa, Maki, Graybiel, & Kimura, 1997), and Packard and colleagues have shown that inputs from the amygdala carried by the stria terminalis are necessary for striatal cholinergic enhancement of learning in a conditioned avoidance paradigm (Packard, Introini-Collison, & McGaugh, 1996). These experiments suggest that remapping of corticostriatal throughput is multiply determined. Finally, striatal interneurons other than the TANs are likely to have important effects in shaping corticostriatal transmission (Kita, 1996; Parthasarathy & Graybiel, 1997).

LEARNING HABITS AND EXPRESSING HABITS

A paradox in the learning literature is that the basal ganglia are at once thought to be important for the production of habitual or "automatic" responses and yet are thought to be important for new S-R learning. Does the same mechanism subserve both learning and expression of habits? The modifiable template mechanism proposed above could do so; but another possibility is that different parts of the system are specialized for acquisition or expression phases, in part depending on the nature of their cortical inputs. This is the suggestion made by Jueptner et al. (Jueptner, Stephan, Frith, Brooks, Frackowiak, & Passingham, 1997a; Jueptner, Frith, Brooks, Frackowiak, & Passingham, 1997b). They have studied cerebral metabolic activity by PET imaging as subjects learn new sequences or execute learned sequences in a button-press task. They find that during new learning, the anterior striatum (caudate nucleus) is differentially active, along with the anterior prefrontal cortex and anterior cingulate gyrus. During the performance of already learned sequences, neither the anterior striatum nor the anterior cortical areas were differentially active; activity had shifted posteriorly, to the putamen and to the premotor and motor cortex. There is by no means unanimity about such anterior-posterior differences (see Brown, 1997, for review). However, when subjects in the Jueptner et al. study were instructed to pay attention to the task during the learned sequence production, the anterior regions again became more active. This result suggests a special function of the anterior system in "attention to action" (Jueptner et al., 1997a, 1997b) which may be important for some forms of procedural learning but not for others (Brown, 1997).

One notable feature of the anterior striatum, other than its massive inputs from prefrontal cortex, is that the neurochemically specialized striosomes are especially prominent there (Graybiel & Ragsdale, 1978). Striosomes are thought to provide a major source of input to the dopamine-containing neurons of the substantia nigra, which, as noted above, respond to salient (attention-attracting) stimuli and, in conditioned animals, respond to conditioned stimuli predictive of future reward. The major cortical inputs to the anterior striosomal system so far identified come from the anterior cingulate/medial prefrontal and caudal orbitofrontal cortex, regions interconnected with the amygdala and

hippocampus (Eblen & Graybiel, 1995). Physiological recordings suggest that neurons in these cortical areas fire in relation to reward or signal lack of predicted rewards as though providing an error signal (Thorpe et al., 1983). The anterior striosomal system may thus play a special role in influencing striatal processing in relation to knowledge of results. More generally, the striatal substrates for procedural learning and for execution of procedures may not be identical everywhere in the striatum, but specialized according to behavioral modality. The relative functions of the striatum in the acquisition and expression should thus not be considered to be exclusive of one another.

CORTICOSTRIATAL REMAPPING AND SEQUENCE LEARNING

Tanji and coworkers have demonstrated exquisitely selective coding for temporally ordered sequences of movements by neurons in the supplementary motor area (SMA) and pre-supplementary motor area (pre-SMA) of monkeys trained to perform three discrete movements in varying orders (Shima, Mushikake, Saito, & Tanji, 1996; Tanji & Shima, 1994). Populations of cortical neurons in these cortical areas respond selectively in relation to temporally correct sequences of two or all three of the movements, performed by memory. These firing patterns stand in sharp contrast to the activity patterns of most neurons recorded in primary motor cortex in the same task, which respond before single movements regardless of the sequence in which they are performed. In the pre-SMA, there is an added population of neurons that fire selectively when a new sequence is to be performed and the previously performed sequence must be abandoned. The pre-SMA has been singled out in an fMRI study as differentially active during the learning (but not during the later performance) of movement sequences (Hikosaka, Sakai, Miyauchi, Takino, Sasaki, & Putz, 1996). Sequence-specific responses have also been found in arcuate, premotor, and prefrontal cortex (e.g., Barone & Joseph, 1989). These findings suggest that in the prefrontal, supplementary motor, and premotor cortex, there is a step-by-step building-up of a code for movement sequences—the neural representation of elements of a motor plan.

There is also strong representation of sequential patterns of movement in the basal ganglia. A large number of observations in the human, ranging from PET and fMRI experiments to studies of patient populations, have implicated the basal ganglia as being active in the learning or execution of sequential behavior (Benecke, Rothwell, Dick, Day, & Marsden, 1987; Brown, 1997; Gerloff, Corwell, Chen, Hallett, & Cohen, 1997; Harrington & Haaland, 1991; Rauch, Savage, Brown, Curran, Alpert, Kendrick, Fischman, & Kosslyn, 1995; Rauch, Whalen, Savage, Curran, Kendrick, Brown, Bush, Breitner, & Rosen, 1997; Seitz, Roland, Bohm, Greitz, & Stone-Elander, 1990; Seitz & Roland, 1992; Vriezen & Moscovitch, 1990). In the most extensive demonstration of this in trained monkeys, Kermadi and Joseph (1995) have shown that many neurons in the caudate nucleus of highly trained monkeys code for learned sequences of eye and eye–hand movements. Both the responses of these neurons to the sequentially presented sensory cues in the task and their movement-related responses were sequence-dependent. Moreover, many striatal units fired in anticipation of such cued sequences. Kermadi and Joseph suggest that the caudate nucleus codes for the temporospatial movement plan, helps to construct the plan based on representing the sequence of targets to be

acquired, and helps to update the plan and to select the next movement, consonant with evidence reviewed by Marsden (1987) indicating that the basal ganglia anticipate the next movement. Mushiake and Strick (1995) have reported selective coding for sequences in the globus pallidus of monkeys trained on a button-press task, showing that sequence coding is present in basal ganglia output neurons. Finally, Berridge and coworkers suggest that there is a specialized representation of "instinctive" (i.e., unlearned) sequential grooming movements in part of the rodent striatum (Cromwell & Berridge, 1996).

Because each of the cortical areas implicated in coding movement sequences projects to the striatum, and the striatum, via the pallidonigral complex and thalamus, projects to and influences these cortical regions, it is not clear whether such representations of sequences are formed in the cortex or in the basal ganglia or in both. However, an ongoing collaborative study with the Kimura laboratory (Matsumoto et al., submitted) suggests that activity in the striatum may be critical to building up these representations. Two monkeys were trained on a three-button-press task, and a unilateral lesion of the nigrostriatal system was made prior to the training in one animal and after the training in the other. Behavioral measures of serial reaction times and EMG recordings from different sets of muscles engaged by the task showed the gradual acquisition of smooth, efficient performance only if the dopaminergic innervation of the striatum was present on the side contralateral to the arm being tested. The effects of dopamine depletion were strikingly evident when, after the monkeys were thoroughly trained on the button-press task (in which reward came after the last press), they were given reward early, one button earlier than expected. Both monkeys showed perseverative errors with the arm contralateral to the intact nigrostriatal system, continuing, as though by "habit," to press the last button. There were also perseverative button presses with the arm contralateral to the posttraining nigrostriatal lesion. But in the monkey with the pretraining lesion of the dopaminergic input to the striatum, perseverative pushes were absent with the contralateral arm. It was as though, when trained without an intact nigrostriatal system, the monkey failed to bind the sequence of pushes together into a "macro" or performance unit. The dependence of this behavior on the presence of dopaminergic innervation of the striatum suggests that the striatum itself (as modulated by dopamine) was necessary for the acquisition of the performance unit.

INFORMATION RECORDING IN CORTICO-BASAL GANGLIA LOOPS

It is clear that learned sequences of movements can be coded both in the neocortex and in the basal ganglia, and it would be surprising not to find such information about learned sequences broadly represented in the motor system. What is proposed here is that the basal ganglia may be critically involved in constructing performance units of such sequence representations. I adopt the term *chunk* from Miller (1956), who used it to convey the idea that efficiency in information content can be gained by recoding bits of information to form packages (chunks of information) that, once learned, can be treated as entities in memory. For the sensorimotor system, this process of recoding could yield the "automated" behaviors attributed to the basal ganglia, and could help to account for some of the difficulties in performing sequential actions suffered by patients with basal ganglia disorders or by animals with basal ganglia lesions.

What could do the recoding? The modular corticostriatal remapping discussed above might serve just such a purpose. By reordering cortically derived information within the striatum, notably within striatal matrixosomes (and striosomes), a more efficient action-oriented representation could be produced. For example, the new representation might reduce the degrees of freedom in the representation by reducing the number of distinctively represented units. Our data both on TAN activity during S–R learning in monkeys and on projection neuron activity during T-maze learning in rats suggest that the changes in neural responsiveness that occur with learning are slow, but that they incorporate two key features: a latch-on mechanism for temporal synchronization (exhibited by TANs) and a phase-advance tendency for predictive coding (exhibited by some projection neurons). The gradual tuning of cortically innervated modular populations of neurons (expert networks) in the striatum, the spatiotemporal binding by striatal interneurons, and the reconvergence of the information onto striatal output targets are candidate mechanisms for bringing about chunking.

THE BASAL GANGLIA AND INFORMATION RECORDING

The functions the basal ganglia perform in influencing motor pattern generators of the brainstem and spinal cord may have equivalents in the cognitive realm by virtue of influencing cognitive pattern generators in the forebrain (Graybiel, 1997). It seems reasonable, given the circuitry of the basal ganglia and their apparent involvement in some neuropsychiatric disorders, that any chunking functions of the basal ganglia may also extend beyond motor chunks to a range of action repertoires.

In Miller's original proposal, the value of chunking was mnemonic: it lets us go beyond the 7 ± 2 limits of short-term memory (Miller, 1956). One of his points was that the recoding from bits to chunks of information needs to be automatic, or nearly so, to be useful. Another was that linguistic encoding is a critical example of chunking. What is proposed here is that a similar recoding to achieve information compression can occur in the formation of action repertoires, whether they be motoric or cognitive. One of the most compelling and well-documented examples of chunking of action repertoires comes from studies of patients with obsessive–compulsive disorder (OCD). The caudate nucleus, together with the anterior cingulate cortex and orbital frontal cortex, shows consistent patterns of metabolic activity in symptomatic OCD patients, and abnormalities in metabolic activity in these cortical regions abate with treatment that decreases symptomatology (Baxter, Schwartz, Bergman, Szuba, Guze, Mazziotta, Alazraki, Selin, Ferng, & Phelps, 1992; Rauch, Jenike, Alpert, Baer, Breiter, Savage, & Fischman, 1994; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996; Swedo, Pietrini, Leonard, Schapiro, Rettew, Goldberger, Rapoport, Rapoport, & Grady, 1992). The symptoms themselves include repetitive, intrusive thoughts and compulsions that lead to ritualistic behaviors such as washing, counting, and checking. These behaviors involve sequential acts. But they are performed as chunks, unitized and driven by the extraordinary imperative of urges and compulsions that the patient recognizes as abnormal and outside the controlled range of the person's intent. These compulsions are now viewed in the context of a lack of normal error feedback (that the action has been done, that the hands have been washed). Disruption of the anterior

learning-performance circuits involving the caudate nucleus, anterior cingulate cortex, and orbitofrontal cortex, which may carry such signals, could thus be critically involved in the genesis of the symptoms.

A BROADER VIEW

There is a much more extensive circuitry of the basal ganglia than discussed here, and other potential functions, such as response selection, that have not been touched on. No integrated treatment of the learning and memory functions of the basal ganglia could be complete without considering these other mechanisms. In the example of response selection, multiple levels within the basal ganglia may be important, and the interplay between direct and indirect pathways may be critical (Berns & Sejnowski, 1996; Mink, 1996). Similar arguments hold for other proposed basal ganglia functions, ranging from response stabilization and scaling (Inase, Buford, & Anderson, 1996; Turner and Anderson, 1997) to the decision of whether and when to move (Graybiel and Kimura, 1995). I have, however, emphasized cortico-basal ganglia circuits, the templates they form in the striatum, and the modifiability of striatal coding during learning to make a particular point: that one function of the striatum may be to chunk performance sequences. It may be these chunked representations that are selected and scaled by the output circuits of the basal ganglia.

How does this notion relate to the two key characteristics of habit or S-R learning emphasized at the outset of this paper, that it occurs slowly and without conscious awareness? The slow kinetics of S-R learning offer a great advantage for chunking in view of the fact that we do not want to collapse all encoded sequences (temporally ordered acts) into chunks. We need to select which sequences to recode in this form, because the behavioral macro or chunk, once formed, may be difficult to break apart. Neural coding of sequences, by contrast, appears to occur quickly and to be highly labile. For example, Tanji and coworkers can train monkeys on a series of three-movement sequences in a training session, and find corresponding switches in the encoding of the sequential movements in the SMA and pre-SMA cortex (Shima et al., 1996). Part of what goes on in S-R learning may be a selection of which sequential representations to chunk, based on reinforcement-related modulation.

The lack of conscious awareness in S-R learning may also be an advantageous property for a chunking mechanism in that the action chunks are treated as units (not, for example, as response chains). We do not want "supervisory attention" (Shallice, 1988) or "attention to action" (Jueptner et al., 1997a, 1997b) or conscious manipulation to intervene inside the macro unit itself. Chunks take their advantage from being manipulable as entities, and the intervention of consciousness or attention might actually disrupt their smooth implementation.

Prediction should be a crucial guide to the implementation of chunks and to their formation. The central place of prediction in corticobasal ganglia loop functions was experimentally demonstrated by Flowers (1978) in a series of experiments on patients with Parkinson's disease, and it was Flowers who suggested that a fundamental sensorimotor defect in Parkinson's patients is a lack of predictive capacity. He suggested that this defect forced the patients toward modes of movement performance in which each movement is guided by sensory input rather than by an internal model allowing prediction and forward planning. The function of action chunking proposed here for the basal ganglia fits

well within this framework. Prediction would allow the initiation of chunks and their formation. The recession in time that we are observing in the responses of some striatal projection neurons during learning is intriguing when viewed in this context.

The “dictates of the will” alluded to by Parkinson could in part be implemented by prediction under the control of intent leading with repetition to action chunking and eventually to the implementation of action chunks no longer under conscious supervision. We suggest, in other words, that there probably are different kinds of linkage between intent and action. Intent may elicit or suppress single acts or series of acts. And, as shown by the example of OCD, some acts and sequences are not under the control of intent even though consciously performed. They can even coexist with different states of conscious intent, at least in disorders affecting cortico-basal ganglia loops. It is for all of these reasons that we emphasize the compatibility of otherwise seemingly contradictory views of the basal ganglia as being involved in generating either automatic acts or willed (intended) acts, or as being involved in either motor or mnemonic processes. One attractive feature of the chunking function proposed here is that these different aspects of basal ganglia operations seem natural allies.

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