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Computational models of motivated action selection in corticostriatal circuits

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Computational models of the basal ganglia have matured and received increasing attention over the last decade. This article reviews some of the theoretical advances offered by these models, focusing on motor and cognitive action selection, learning, and the interaction between multiple corticostriatal circuits in selection and learning.

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Introduction

The last decade has seen an explosion in the development of and attention to computational models of basal ganglia (BG) function. A Google Scholar search for articles mentioning both 'computational model' and 'basal ganglia' yields 2960 matches in the last twenty years, with 2550 of them appearing in the last decade alone, comprising more than a ten-fold increase. This surge seems not related to increased attention to either modeling or BG research by themselves: either term alone yielded a roughly even distribution across decades.

What accounts for this selective jump in focus on models of basal ganglia? A potential clue comes from another search: the number of articles mentioning the general term 'action selection' — perhaps the primary function attributed to the BG — has increased sharply from 2000 in the 1990s to 16,100 across decades. Action selection is a computational problem which, particularly when combined with the notorious complexity of BG circuitry, lends itself well to modeling. Indeed, the joint terms 'computational model', 'basal ganglia', and 'action selection' yield 694 results in the last decade, compared with just 29 in the 1990s — comprising a twenty-fold leap.

Several other factors are undoubtedly at play. But as James [1] noted, 'selection is the keel on which our mental ship is built'. The connection between the BG and action selection has been studied intensively [2–5], with computational accounts initially and/or most prominently offered by Houk [6,7] and Barto [8], followed by Doya [9], Gurney [10], and several others [11–13].

Building on these approaches, many BG models now exist at different levels of analysis — from biophysical to algorithmic — each attempting to account for varying degrees of physiological, behavioral and pharmacological data across species. Some of the main issues simulated in network models are: the fundamental disinhibitory mechanisms of BG processing which act to 'gate' motor programs [14]; pathological states associated with disease (e.g. Parkinson's); the transformations from motivation to action, from motor to cognitive to affective states, goals and their interactions. Below is a brief survey.

Basic action selection framework

In 1999, Redgrave and colleagues noted that the BG are well preserved across species and proposed that, by implementing a central switchboard-like selection mechanism, it constitutes a 'vertebrate solution to the selection problem' [5]. Detailed computational simulations of the proposal ensued [10]. In these models, the cortex represents the salience of multiple competing actions in separate 'channels'. The primary function of the BG is to inhibit all of these channels (via tonically active GABAergic neurons in BG output structures). The striatum can then disinhibit one of these channels by releasing tonic inhibition and selectively boosting activation of the most salient channel. Thus the BG do not select the actions themselves but rather facilitate their execution via the 'direct pathway' from striatum to BG output structures, consistent with the proposal of Mink [4]. In contrast, the classical 'indirect pathway', which traverses the pallidum and subthalamic nucleus before targeting BG output structures, was generally thought to antagonize the direct pathway by suppressing unwanted movements [15,4]. Gurney et al recast this pathway in terms of a control process to support 'capacity scaling' — that is to ensure that no matter how many channels are active in cortex, only one will be gated. These models have evolved substantially in the past decade, and there are now large scale and more physiologically realistic versions

[16••], as well as abstract algorithmic approaches linking the architecture of BG circuitry to optimal decision making theories [17].

Learning to select

The above models assume that to-be-selected action is signaled by the input channels (e.g., cortex) to be most salient, with this information passed unmodified by the BG. Other approaches focus on how the BG may learn which actions are most rewarding by experience. This literature is influenced by a wide range of evidence that the BG, and particularly the modulation of its activity and plasticity by dopamine, plays a key role in reinforcement learning [2] and is necessary for the acquisition, but not always expression, of simple stimulus–response associations [18,19]. Many computational models have linked the phasic bursts and dips observed in midbrain dopamine neuron activity [20] to reward prediction error signals that serve to drive learning in the BG [8,9,21••].

More recent mechanistic network models have integrated the selection and learning mechanisms into a single model including the direct, indirect and hyperdirect pathways of the BG, with different model neurons to simulate physiological properties of the different nuclei [13,12,22]. These models adopt the notion that the cortex generates candidate actions for a given sensory context, and that the BG selectively gate one (or a subset) of these actions by disinhibiting thalamocortical activity for the winning action(s). However, instead of always gating the most salient actions, these models assume that the striatum transforms the cortical representations into reinforcement values, such that actions with the highest value are most likely to be gated. Dopamine modifies both activity and plasticity in the striatum. Phasic bursts of dopamine enhance corticostriatal synaptic plasticity via D1 receptor stimulation in the direct pathway [23,13,9,12], such that high value actions become more strongly represented. Some models also include a separate function for the indirect pathway, generally consistent with earlier ideas that this pathway acts to suppress movement [15,24,4], but does so in proportion to the learned negative value of an action [13]. Specifically, midbrain dopamine neurons reliably pause as a function of negative reward prediction errors (i.e. when outcomes are worse than expected; [21••]). In the model striatum, the resulting drop in dopamine concentration transiently increases excitability and strengthens corticostriatal synaptic plasticity in indirect pathway striatal cells, by removing the tonic inhibitory effect of dopamine onto high affinity D2 receptors predominantly expressed in these cells [13,25]. Conversely, when positive outcomes occur, dopamine bursts further inhibit D2 cells and act to weaken these synapses. As a result, indirect pathway cells differentially respond to actions that have negative value. Because direct and indirect pathway cells compete at BG output, the action most likely to be gated is a function of the difference in activity in these pathways for each action in parallel.

Evidence for these posited model mechanisms has mounted over the last decade. Electrophysiological studies have identified separate populations of striatal cells associated with action facilitation vs. suppression [26,27] and that code for positive and negative action values [28,29]. These studies could not identify whether these populations correspond to the direct and indirect pathways. However, experiments using sophisticated genetic manipulations have confirmed selective roles of direct and indirect pathways in the facilitation and suppression of behavioral output [30••], with opposing modulations of synaptic plasticity in these pathways as a function of D1 and D2 receptor stimulation [31••], that support reward and punishment learning, respectively [32••]. When dopamine levels are elevated pharmacologically, optogenetic stimulation of the direct or indirect pathway enhances or diminishes reward learning [33]. These findings all converge with the above model mechanisms suggesting that dopamine promotes reward learning by modulating activity-dependent plasticity in striatal direct and indirect pathway cells in opposite directions. Analogous findings have been found in humans: striatal dopamine manipulation influences the degree to which individuals learn more from positive or negative outcomes, with DA elevations enhancing reward learning but impairing punishment learning, and vice-versa for DA depletion [34–36], and these learning modulations are accompanied by altered striatal responses to reward prediction errors [37••]. Further, genetic variants affecting striatal D1 and D2 receptor function are predictive of individual differences in learning from positive and negative prediction errors [38, for review].

What might be the advantage of having two opposing pathways instead of just a single pathway that learns a single probabilistic reward value for each actions? First, it is possible that the anatomical intermingling of direct and indirect pathway cells allows the system to act as a differential amplifier by subtracting away correlated noisy activity from both projection pathways, so that what is left at BG output is only the difference in learned value for each action. Second, simulations showed that the dual pathway mechanism, together with sufficient dynamic range in dopamine signals, allows networks to resolve subtle differences in probabilistic reward values of actions depending on the combination of stimuli [13]. Here the indirect pathway can act as a 'veto' to prevent actions that would normally be considered adaptive from being executed in a particular stimulus context [13,12,39]. Recent physiological data provide a novel mechanism by which this veto function could occur: indirect pathway cells were found to inhibit their direct pathway neighbors (via inhibitory recurrent collaterals), but not vice versa [40]. Third, having separate representations of positive and

negative value across pathways allows these representations to be differentially emphasized during action selection. Specifically, tonic dopamine levels can also be modulated separately from phasic signals, and optimized as a function of motivational state [41]. Because dopamine modulates striatal activity in opposite directions in the two pathways, tonic dopamine can act as a knob to primarily emphasize learned positive or negative prospective outcomes when making decisions (e.g., higher levels would suppress the representation of negative value by inhibiting the indirect pathway). Supporting this depiction, tonic dopamine manipulations influence the degree to which action selection is sensitive to previously learned benefits vs. costs [42,43], the latter of which are coded in the indirect pathway [44]. Finally, the two pathway model can explain aspects of appetitive and aversive/avoidance learning due to dopaminergic manipulations that are quickly renewed after extinction [45••,46].

Although the opposing pathway model has been questioned [47], the converging evidence for it implies that when faced with challenging data, parsimonious theories that explain a range of data need not be replaced altogether. Instead, they can be refined by new developments of anatomical and physiological constraints, and more nuanced dynamics [25]. For example, the subthalamic nucleus (STN), originally conceptualized as part of the indirect pathway [15,24], now forms the major node of a third hyperdirect pathway (from cortex to STN to BG output) which provides global inhibition of all actions. Simulated STN activity (and hence global inhibition) unfolds dynamically during response selection, and is particularly influential in situations of high conflict (i.e., when multiple actions are strongly activated simultaneously) to prevent premature responding [22] or to inhibit actions altogether if need be [48]. Thus, the cortico-STN pathway can modulate the dynamics of action selection by regulating the amount of striatal activity needed to gate a response (i.e., the threshold to disinhibit BG output structures), without interfering with the striatal valuation process itself. Simulations suggest that this mechanism is adaptive and can account for various physiological and behavioral data not considered in the original direct/indirect pathway model [22,49,48,50], while not replacing that model altogether.

Cognitive action selection and learning

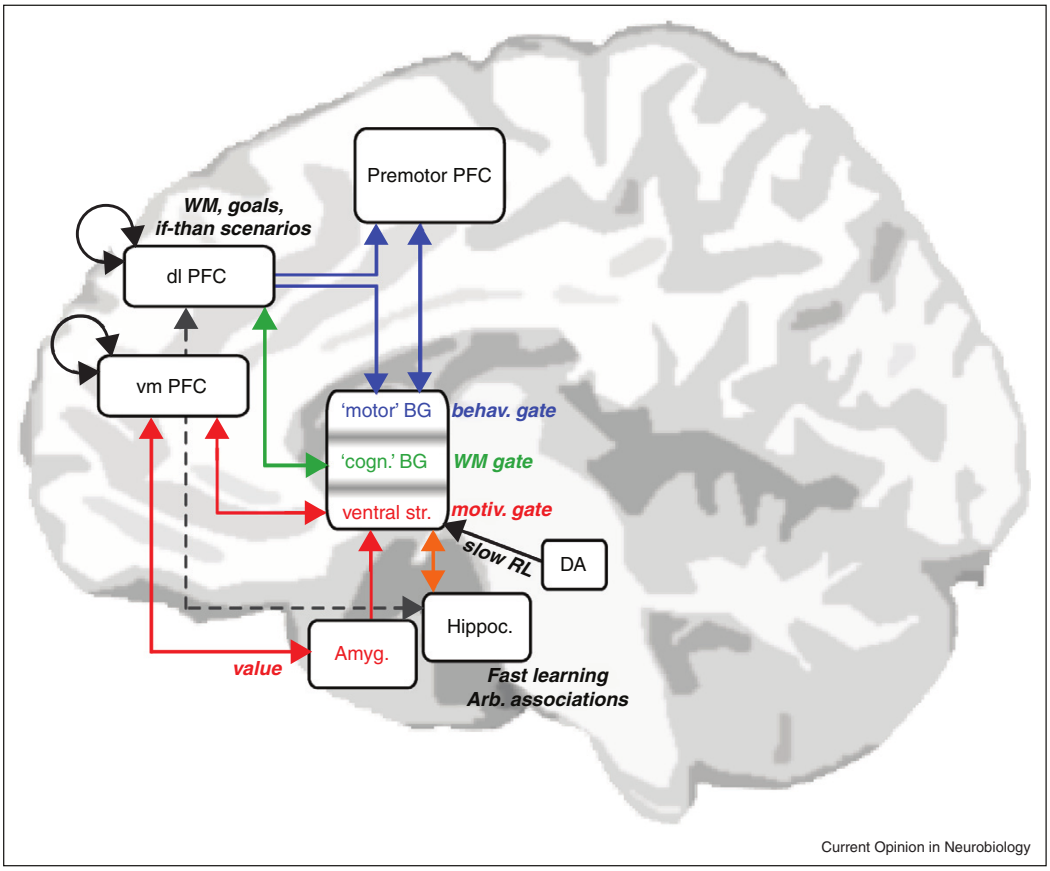
In many contexts, action selection encompasses much more than simply facilitating or suppressing motor actions as a function of learned value in particular sensory contexts. For example, action selection may be contextualized by prior states (sensory, motor, or cognitive), which can be maintained in working memory. In this scenario, action selection can proceed as usual, but with the stimulus context expanded to include internal states. Moreover, the decision of which states to update and subsequently maintain in memory, and which to ignore, is itself an action selection process that benefits from analogous gating and learning mechanisms in circuits linking striatum with prefrontal cortex [51,52]. Recent empirical data support this scheme [53••–55].

Another example of cognitive influences on action selection concerns 'goal-directed behavior'. Both animals and humans can flexibly decide to select a usually rewarding action depending on whether the anticipated outcome is currently desired. With repeated selection, actions can become 'habitual' and thereby insensitive to changes in valuation of the outcome [56]. Computational models have described these processes in terms of a competition between prefrontal and striatal systems for behavioral control, with the prefrontal cortex representing the anticipated reward outcome associated with current or future states, and the striatum implicitly (but less flexibly) learning probabilistic values of stimulus-response pairings as a function of reward prediction errors [57••,58]. As behaviors are well learned, the ingrained striatal associations dominate and habits emerge. Finally, several repeated pairings of sensory and cortical motor states gives way to a third stage, in which corticocortical associations are sufficiently strong to elicit automatized responding even before striatal gating signals occur [7,13,58,59].

Although these models have heuristic value, some aspects will need to be refined. Lesion and pharmacological studies have shown that the habit and goal-directed computations are supported by distinct corticostriatal circuits (with each system having both striatal and frontal components) rather than by PFC and striatum as two categorical competitive entities [56]. Recent physiological data suggest that learning in these two circuits occurs in parallel, and that the cognitive circuit simply prevents the habitual circuit from controlling behavior during initial task acquisition [60••].

Moreover, cognitive and motor corticostriatal circuits are not completely segregated. Indeed, anatomical data now suggest a substantial degree of convergence and crosstalk, such that prefrontal cortex can influence motor striatum [61] (Figure 1). Neural models have simulated this sort of interaction, whereby prefrontal cortical representations of instructed cognitive rules can directly guide striatal action selection before procedural learning occurs [62]. Further, this prefrontal modulation modulates not only striatal activity, but also activity-dependent plasticity, thereby sculpting striatal action policies to ingrain rule-like behaviors — even when the rule turns out to conflict with experienced reinforcement contingencies [62]. When modeled algorithmically, this prefrontal-BG 'Bias' model provided a better quantitative fit to human participant choices, and the neurogenetic predictors thereof, than did an alternative model in which the striatum simply competes with (or is overridden by) PFC rules at the level of

Figure 1



Interacting corticostriatal circuits contributing to action selection at various levels of analysis. Colored projections reflect subsystems associated with value/motivation (red), working memory and cognitive control (green), procedural and habit learning (blue), and contextual influences of episodic memory (orange). Subregions within the basal ganglia (BG) act as gates to facilitate or suppress actions represented in frontal cortex. These include parallel circuits linking the BG with motivational, cognitive, and motor regions within the prefrontal cortex (PFC). Recurrent connections within the PFC support active maintenance of working memory (WM). Cognitive states in dorsolateral PFC (dlPFC) can influence action selection via projections to the circuit linking BG with the motor cortex. Dopamine (DA) drives incremental reinforcement learning in all BG regions, supporting adaptive behaviors as a function of experience. Reprinted from Frank *et al*. (2009). Current Directions in Psychological Science 18:73–77.

motor output [62,63]. Moreover, this type of inter-circuit interaction has been extended to include multiple hierarchically nested corticostriatal circuits, whereby anterior 'higher level' circuits select more abstract actions that constrain attention, action selection and learning in the more posterior circuits, facilitating the discovery of higher order abstract rules [64]. Others have proposed a cascading interactive corticostriatal circuit model that supports sequencing of linguistic speech productions [65].

Conclusion

Much remains to be explored with models of basal ganglia, and this review constitutes only a very brief and selective survey of existing models. For example, models have begun to investigate the learned responses of cholinergic interneurons and their modulation of striatal plasticity [39]. Further work will need to consider the implications of back-projections from pallidum and thalamus to striatum, the role of striosomes, serotonin, adenosine, and a host of other factors. At the larger scale, models will have to consider communications not just between multiple corticostriatal circuits, but from hippocampus, cerebellum, and other structures to the BG. These interactions are likely involved in more sophisticated versions of action selection that will also benefit from formal analysis. As these complexities are taken into account, computational models — constrained and refined by emerging data — are expected to play an increasingly important role in shaping theorizing.

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