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Contributions of the Basal Ganglia to Visual Perceptual Decisions

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Abstract

The basal ganglia (BG) make up a prominent nexus between visual and motor-related brain regions. In contrast to the BG's well-established roles in movement control and value-based decision making, their contributions to the transformation of visual input into an action remain unclear, especially in the context of perceptual decisions based on uncertain visual evidence. This article reviews recent progress in our understanding of the BG's contributions to the formation, evaluation, and adjustment of such decisions. From theoretical and experimental perspectives, the review focuses on four key stations in the BG network, namely, the striatum, pallidum, subthalamic nucleus, and midbrain dopamine neurons, which can have different roles and together support the decision process.

Keywords

striatum; pallidum; saccade; choice; reaction time; sequential analysis; signal detection theory

1. INTRODUCTION

Visual perceptual decision making is the process by which visual input is converted into a categorical choice based on perceived features of that input. To understand how the brain implements this process, numerous seminal studies focused on the roles of neural representations of the visual input in the visual pathways. However, these sensory representations alone do not govern decision-making behavior. For example, simple decisions about whether a dim light was detected can change depending on how cautious the subjects are instructed to be (Barlow 1956). Moreover, neural signals that encode the evolving perceptual decision process, not just the final choices, are commonly observed in motoric and higher-order brain regions (Gold & Shadlen 2007), reflecting the close link between perception and action in natural behaviors (Gibson 1966). These observations argue strongly that brain regions outside the visual pathway are needed to form visual perceptual

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decisions. In this review, I synthesize evidence showing that the basal ganglia (BG) are major contributors to the process of converting representations of visual input into decisions that guide behavior.

The BG receive projections from nearly every part of the cerebral cortex, leading early neuroscientists, as well as modern-day superhero movie makers, to ascribe to the BG seemingly all-encompassing roles, including “the seat of the sensorium commune” (Thomas Willis, 1667, as cited in Wilson 1914, p. 430), “the royal road of the sensations of the body to the soul” (Emanuel Swedenborg, 1740, as cited in Wilson 1914, p. 430), movement control (Ferrier 1873; Wilson 1914), and mind control (*Black Widow*, 2021). Of these roles, the BG’s contributions to movement control and reinforcement learning have been studied most extensively. Our growing understanding of how the BG are involved in these roles has also led to more effective therapeutic interventions for diseases with BG dysfunctions, such as Parkinson’s disease (PD) and addiction.

In contrast, although several early studies of BG function implied potential roles in perceptual decision making, the evidence was indirect and often not examined in depth. For example, lesions in the BG can lead to impairments in certain visual spatial and pattern discrimination tasks (Buerger et al. 1974, Divac et al. 1967). A BG output nucleus, the substantia nigra pars reticulata (SNr), is linked to the famous Sprague effects, whereby blindness from unilateral lesions of the visual cortex can be rescued by lesions of the contralateral superior colliculus (SC) (Sprague 1966; Wallace et al. 1989, 1990). The underlying mechanisms for these visual perception–related phenomena remain unclear.

Over the past 25 years, the BG’s roles in perceptual decision making, and higher cognitive functions more generally, have received increased attention. This review summarizes our recent progress in understanding, from both theoretical and experimental perspectives, how the BG pathway supports computations needed for visual perceptual decision making. Section 2 introduces the computational components commonly associated with perceptual decision making. Section 3 gives a broad overview of how the neural implementation of these computational components may benefit from several major anatomical and physiological features of the BG pathway. Section 4 describes in more detail the possible contributions of individual BG nuclei to specific computational components in visual decision making, based on available experimental data. Section 5 reviews our understanding of the BG’s roles in flexibly adjusting the decision process to meet task goals. Section 6 summarizes recent efforts to model how computations proposed to occur in the BG pathway can, in principle, support these decision-making functions. Section 7 highlights the major knowledge gaps in our understanding and possible future research directions.

2. COMPUTATIONAL COMPONENTS FOR VISUAL PERCEPTUAL DECISIONS

Two prominent theoretical frameworks in the field of perceptual decision making are signal detection theory (SDT) and sequential analysis (SA) (Green & Swets 1966, Stone 1960, Wald 1947). Studies of BG function in visual perceptual decision making have relied heavily on these frameworks, which is why I review them briefly in this section. The commonality

and differences in mathematical and neuroscientific terms between these two frameworks have been reviewed extensively (e.g., Griffith et al. 2021). Central to both frameworks are three general components that (*a*) transform sensory evidence into a decision variable, (*b*) apply a decision rule to the decision variable to reach a discrete choice, and (*c*) modulate these two components to incorporate additional decision-related factors such as the prior probabilities of the alternative stimuli and expected rewards and costs of each outcome. A key difference between the two frameworks is that SDT is typically used to account for choice behavior independent of time (e.g., when the decision is based on a single piece of evidence), whereas SA is used to account for time-dependent choice behavior (e.g., when multiple pieces of evidence are presented over time). For tasks in which subjects control the evidence viewing time, SA can also account for reaction time (RT) and its relationship with choice behavior.

For a typical two-alternative forced-choice task, an SDT-based model typically computes the decision variable as the likelihood ratio between the two alternatives, given the evidence observation(s) (Figure 1*a*). The decision rule is implemented by a decision criterion such that decision variable values greater or less than the criterion lead to choices for each of the two alternatives, respectively. Modulation of the decision process can be achieved by either changing the likelihood-ratio computation (e.g., improving the d' value of the evidence; Figure 1*b*) or changing the criterion (e.g., increasing the criterion would lead to more choices for one alternative and fewer for the other; Figure 1*c*).

In the SA framework, the most widely used model is the drift-diffusion model (DDM) (Gold & Shadlen 2007, Ratcliff 1978, Ratcliff et al. 2016). The DDM computes the decision variable as the temporal accumulation of an internal representation of evidence, which is assumed to be Gaussian distributed and related to the likelihood ratio between two alternatives (Figure 1*d*). The decision rule is implemented as two decision bounds. If the decision variable reaches one of the two bounds, then the decision process is terminated and the corresponding choice is committed. Otherwise, the decision process continues to gather further evidence until a choice can be made. The decision time is thus determined by the timing of the first bound crossing, which depends on the rate of accumulation (drift rate) and the bound heights. The total RT is the sum of decision time and nondecision times associated with sensory and motor processing delays. The DDM is also similar to the sequential probability ratio test (SPRT), with the latter assuming that evidence is presented in discrete times and that its internal representation is not necessarily Gaussian distributed (Bitzer et al. 2014, Bogacz et al. 2006).

Modulation of the decision process in both models can be achieved by changing the decision variable or decision rule (Figure 1*e*–*i*). For example, improving sensitivity may increase the likelihood ratio associated with each piece of evidence, which effectively increases the absolute drift rate in the DDM (Figure 1*e*). Lowering both bounds equally means that it takes less time on average for the decision variable to reach a bound (faster decisions), and the bound crossing is more susceptible to uncertainty in the internal representation of evidence (lower accuracy) (Figure 1*f*). The bounds can also be modulated dynamically to maximize reward rate (Drugowitsch et al. 2012, Thura et al. 2012). For example, gradually lowering (collapsing) bounds over time can shorten the deliberation time for difficult

choices with only minor reduction in the overall accuracy. The collapsing bounds can also be implemented with a gradually increasing offset in the decision variable (urgency signal). To create a decision bias, fictive evidence for one choice can change the drift rates asymmetrically toward the two bounds (Figure 1*g*), while shifting the starting point of the accumulation effectively changes the bound heights asymmetrically for the two alternatives, leading to more decisions and shorter decision time for the alternative associated with the lower effective bound height and fewer decisions and longer decision time for the other (Figure 1*h*,*i*).

These computational components in the SDT and SA frameworks beg the neuroscientific question: How are they implemented in the brain? In the next section, I review, in very broad strokes, several key features of the BG circuits that could be especially helpful for supporting computations needed for perceptual decision making.

3. KEY ANATOMICAL AND PHYSIOLOGICAL FEATURES OF THE BASAL GANGLIA PATHWAY

3.1. Interactions of Three Main Pathways Within the Basal Ganglia Can Support Complex Computations

The mammalian BG pathway consists of multiple anatomical and functional loops that share a common projection pattern (Albin et al. 1989, Alexander et al. 1986, DeLong 1990, Gerfen 1984, Haber 2003). In each loop, the striatum and the pallidum are the main input and output stations, respectively. The striatum consists of medium spiny projection neurons, which are estimated to be approximately 95% of the population, plus interneurons with distinct types defined by anatomical and physiological properties. The striatal projection neurons, which project primarily to deeper BG nuclei, are GABAergic inhibitory neurons with low baseline spike rates. The pallidal neurons are GABAergic inhibitory neurons that maintain high baseline spike rates. It is generally assumed that, under baseline conditions, the high-spiking pallidal outputs are necessary for inhibiting neurons in the thalamus and/or SC to suppress unwanted behaviors. When the pallidal neurons are inhibited themselves, such as via increased activation of directly projecting striatal neurons, thalamic and/or SC neurons are disinhibited and released from pallidal suppression to mediate appropriate behaviors.

The input-output functions of the BG depend on interactions among three main pathways connecting the striatal inputs and pallidal output (Figure 2*a*). In the direct pathway, a subset of striatal projection neurons directly inhibit pallidal output neurons, thus lessening their inhibitory output. In the indirect pathway, a different subset of striatal projection neurons inhibit pallidal neurons in the external segment of the globus pallidus (GPe). These GPe neurons do not themselves project outside the BG, but rather inhibit neurons in the subthalamic nucleus (STN) that excite pallidal output neurons in the internal segment of the globus pallidus (GPi) and SNr, thus increasing their inhibitory output. In the hyperdirect pathway, STN neurons receive direct cortical inputs, bypassing the striatum, and excite pallidal output neurons, also increasing their inhibitory output. In addition to these pathways within the BG, which have different combinations of excitatory and inhibitory projections

connecting the striatal inputs and pallidal output, a feedback projection from the pallidum, indirectly via the thalamus back to the cortex, adds further flexibility to implement complex computations in the BG circuit.

3.2. Convergent Projections into the Basal Ganglia Can Support Integration of Information from Diverse Sources

A striking feature of the striatum is that it has massive afferents from cortical regions. A single cortical region can project to multiple striatal regions, and multiple cortical regions can project to a single striatal region (Flaherty & Graybiel 1991, Selemon & Goldman-Rakic 1985) (Figure 1*b*). Such a mixture of divergence and convergence can provide to the BG heterogeneous representations of information combined from multiple cortical sources. Inside the BG, these representations are processed through the highly convergent feedforward projections from the striatum to the pallidum, with an estimated 100:1 ratio in the rat and approximately 300:1 in the human BG (Oorschot 2010, Wilson 2013). These anatomical organizations suggest that the BG are well positioned to integrate inputs from multiple sources, including external sensory inputs and internally generated cognitive signals, to guide decision making (Graybiel et al. 1994).

3.3. Combination of Focused and Diffuse Projections Within the Basal Ganglia Can Support Selection

The output neurons of the BG in the pallidum receive inhibitory input from the striatum and excitatory input from the STN (Figure 1*c*). The striatopallidal projections follow segregated patterns at multiple levels (Alexander et al. 1986, Haber 2003, Parent & Hazrati 1993). The three striatal nuclei in primates, namely the caudate, putamen, and nucleus accumbens, project primarily to the SNr, GPi, and ventral pallidum, respectively, forming three parallel BG pathways. Within each pathway, a single striatopallidal axon focuses its distal branches to entwine the dendrites of its primary pallidal target (Parent & Hazrati 1993). In contrast, a single STN neuron innervates a large number of pallidal neurons with similar anatomical intensity. The combination of these focused and diffuse projections, reminiscent of the center-surround visual receptive field, has led to the hypothesis that the BG pathway plays roles in implementing a filtering or selection process for movement control and decision making more generally (Berns & Sejnowski 1995, Mink 1996, Redgrave et al. 1999).

3.4. Neuromodulatory Projections Can Support Decision Evaluation, Contextual Modulation, and Learning

The BG receive inputs from major neuromodulatory systems that can influence the perceptual decision making process (Figure 1*d*). The striatum receives dense dopaminergic innervations from the substantia nigra pars compacta (SNc) and ventral tegmental area (VTA) in the midbrain. Activation of dopamine neurons carries reward-related information, including reward prediction errors (RPEs), reward uncertainty, stimulus salience, and sensory features associated with reward (Bromberg-Martin et al. 2010; Fiorillo et al. 2013; Schultz 1998, 2019; Takahashi et al. 2017). Dopaminergic input to the striatum can differentially modulate the direct and indirect pathways in the BG by activating two families of receptors, D1-like and D2-like, expressed by striatal projection neurons in each of the two pathways, respectively (Gerfen et al. 1990). The BG receive cholinergic innervations

from the pedunculopontine nucleus-laterodorsal tegmental complex, which is hypothesized to play roles in reinforcement learning and action evaluation (Albin et al. 2022), and internally from the cholinergic interneurons inside the striatum, which respond to reward-predicting stimuli with a pause followed by rebound activation and are sensitive to spatial and temporal contextual influence (Apicella 2017). The striatum also receives noradrenergic and serotonergic innervation from the locus coeruleus and dorsal raphe, respectively (Parent et al. 1983). These neuromodulators have only begun to be directly examined in the context of perceptual decision tasks. Nonetheless, their presence in the BG can provide additional neural mechanisms to support flexible decisions and learning in different behavioral contexts and/or performance evaluation related to reward outcomes.

4. PERCEPTUAL DECISION–RELATED ROLES OF EACH KEY BASAL GANGLIA NUCLEUS

The anatomical features of the BG are suitable for implementing computations related to decision formation and evaluation. In this section, I review available experimental evidence for each key nucleus' activity patterns and possible computational roles.

4.1. Striatum

The striatum is the main input station of the BG and receives sensory-related inputs from almost all parts of the sensory cortex and thalamus (Cowan & Powell 1956, Yeterian & Pandya 1995). The striatum consists of three regions in the primate brain: the putamen, caudate, and ventral striatum. These three regions are parts of the motor, prefrontal-oculomotor, and limbic BG loops, respectively (Alexander & Crutcher 1990), and may make different contributions to perceptual decision making. It is generally assumed that the caudate–putamen complex is more involved in decision formation, and the ventral striatum is more involved in decision evaluation.

To understand the striatal contributions to decision formation, neural signals in the caudate and putamen have been examined in the context of two types of perceptual decision tasks: (*a*) categorization tasks with briefly presented stimuli (evidence) and/or (*b*) discrimination tasks that benefit from evidence accumulation over time. Functional magnetic resonance imaging (fMRI) studies in humans have consistently shown that the caudate nucleus is activated for both types of tasks (Filimon et al. 2013, Forstmann et al. 2008, Green et al. 2012, Mansfield et al. 2011, Nagano-Saito et al. 2012, Nomura & Reber 2008, Seger & Cincotta 2005).

In monkeys performing visual categorization tasks, caudate activity reflects the rules that map the categorization of a visual feature to the appropriate response. As the animal learns the visuomotor mapping, caudate responses to the visual cues become selective for cue category or the action associated with the category (Pasupathy & Miller 2005, Seo et al. 2012). When the experimenter changes the rules such that the visual feature is associated with a different category, the predecision activity of many caudate neurons follows the category change instead of the visual feature (Muhammad et al. 2006). Category-selective predecision activity has also been described in putamen neurons in monkeys performing

a tactile categorization task (Merchant et al. 1997). Such putamen activity reflects only the categorical choice without additional graded modulation by stimulus property. Similar striatal activity has been observed in the direct pathway striatal projection neurons in mice (Wang et al. 2018). Comparisons between caudate activity and the activity of cortical areas that project to the caudate nucleus have revealed interesting differences. For example, when the categorization demand is low (few categories need to be learned), the category selectivity of cue-related activity develops sooner and is more strongly represented in caudate neurons than in prefrontal cortical neurons (Pasupathy & Miller 2005, Seo et al. 2012). However, as the categorization demand increases, e.g., with novel categories or more categories, the category-selective activity emerges earlier in the prefrontal cortex than the caudate (Antzoulatos & Miller 2011). These results suggest that the neural implementation of categorization-related computations may shift from striatum to cortex, depending on task complexity.

A causal link between the striatum and categorical decisions has been further supported by perturbation studies in rodents and clinical studies of human patients. For example, in rats performing an auditory categorization task, optogenetic activation and inactivation of the corticostriatal projections induced choice biases in opposite directions, and these effects were more consistent and specific than the effects of activating cortical neurons more broadly (Znamenskiy & Zador 2013). In mice performing a visual orientation-change detection task, optogenetic activation of striatal projection neurons induced biases that reflected changes in the decision criterion in the SDT framework (Wang et al. 2018). This effect also had different spatial specificity for the direct and indirect pathways, implying pathway-specific contributions to the implementation of the decision criterion. In human patients, focal putamen lesions and PD (which affects dopaminergic innervations in the striatum) cause deficits in rule-based categorization performance (Ell et al. 2006, Maddox et al. 2005). These results together support a conserved striatal contribution to categorical decisions across species.

Caudate activity can also reflect a process of evidence accumulation akin to that found in the DDM. In particular, in monkeys performing visual discrimination tasks that involve accumulation of visual evidence over time, the activity of some caudate neurons encodes the gradual emergence of selectivity for evidence strength and the eventual choice (Ding & Gold 2010) (Figure 3*a*). For trials that end with a saccadic choice toward the neuron's response field, caudate activity ramps up in an evidence strength–dependent manner. During the ramp, spike count variability and temporal correlation also increase with time, consistent with the caudate activity being directly involved in evidence accumulation, instead of passively relaying ramp-like inputs (Ding 2015). Unilateral electrical microstimulation in the caudate nucleus induced changes in choice and RT that were consistent with biasing of the relative bound heights for the two alternatives (Ding & Gold 2012b). The dorsal striatum in rats showed similar causal involvement in evidence accumulation when the rats were tested with an auditory decision task (Yartsev et al. 2018). In humans, patients with focal putamen lesions used lower decision bounds than healthy controls (Winkel et al. 2016). These results suggest that, just like for perceptual categorization tasks, when evidence accumulation is required, the striatum plays causal roles in the evidence accumulation–dependent decision process.

The evidence accumulation–related activity in the striatum shares common features with reported cortical activity from the prefrontal, premotor, and parietal areas but differs in functionally significant aspects (Ding & Gold 2010, 2012a; Roitman & Shadlen 2002; Thura & Cisek 2014). First, in the cortex, evidence accumulation–related activity follows an accumulation-to-bound trajectory: For the choice linked to the neuron's response field, activity ramps up to reach a higher-than-before common bound level just before saccade onset. In contrast, in the caudate nucleus, similar activity either does not converge to a common level or converges to a level that has been reached well before saccade onset. In other words, in contrast to cortical activity that appears to reach a bound, caudate activity follows only the early accumulation phase without reflecting bound crossing. Second, caudate microstimulation–induced biases often favor choices associated with saccades toward ipsilateral targets, in contrast to the consistent contralateral biases from microstimulation of a parietal cortical area (Hanks et al. 2006), suggesting that the two areas have different anatomical organizations for choice-related computations. Third, in rats, precisely timed optogenetic experiments showed that striatal inactivation can induce robust choice biases throughout the decision formation period, whereas frontal cortical inactivation was effective only if applied near the end of decision formation (Hanks et al. 2015), suggesting that the striatum and frontal cortex may contribute primarily to evidence accumulation and decision termination, respectively. Fourth, within the SA framework, striatum-related microstimulation effects in decision formation (reflected in choice and the decision time portion of the RT) were often accompanied by effects in the motor-generation process (reflected in the nondecision time portion of the RT) (Ding & Gold 2012b, Winkel et al. 2016). Such observations reaffirm the well-known motoric roles of the BG pathway and raise the question of whether (*a*) the decision-formation and motor-generation processes are implemented by different striatal subpopulations or, (*b*) because the decisions are often linked to predictable actions in the lab setting, the decision formation process is embodied in the neural substrates responsible for motor generation.

In addition to contributing to decision formation, a substantial subset of caudate neurons also maintains selectivity for evidence strength during and after saccades are generated, suggesting that these neurons may serve additional functions beyond decision formation, such as postdecision evaluation. To maintain a satisfactory level of performance, three commonly studied signals can help to evaluate past decisions to guide appropriate adjustments: (*a*) choice confidence, which estimates the likelihood that a decision is correct; (*b*) reward expectation, which estimates how much reward can be expected from that decision before reward feedback; and (*c*) reward prediction error, which is the difference between the actual reward received and the reward expectation. These types of signals have been observed in the striatum, particularly the caudate and ventral striatum. In the caudate nucleus, many neurons show postdecision responses that remain modulated by stimulus strength (Ding & Gold 2010, Yanike & Ferrera 2014). The modulation patterns conform to a representation of choice confidence or reward expectation for activity before feedback and, in some cases, a representation of reward prediction error afterward. Similar stimulus strength–dependent fMRI signals in the human ventral striatum have been reported to reflect reward prediction error (Chen et al. 2015), categorical uncertainty (Grinband et al. 2006), choice confidence (Buzzell et al. 2016, Guggenmos et al. 2016, Hebart et

al. 2016), or sensory reliability (Bang & Fleming 2018). These evaluative signals in the striatum are likely linked to modulation by dopaminergic inputs that themselves reflect reward expectation and reward prediction error. For example, on a motion discrimination task with asymmetric rewards, DA neuron activity in the SNc reflected reward expectation before choice action (higher with stronger motion or if a larger reward is expected) and reward prediction error after reward feedback (higher when a difficult choice was rewarded and suppressed on error trials) (Lak et al. 2017, Nomoto et al. 2010). Similar patterns were observed for a vibrotactile detection task (de Lafuente & Romo 2011), in mouse DA neurons, and in the DA axonal activity in the mouse striatum (Lak et al. 2020, Moss et al. 2021, Tsutsui-Kimura et al. 2020).

4.2. Pallidum

In contrast to the large number of studies of the striatum in decision formation and evaluation, very few studies have experimentally measured or perturbed pallidal responses in the context of perceptual decision making. One of the few studies recorded from GPe and GPi neurons while monkeys predicted whether more tokens that started in a center jar would move to the left or right side (Thura & Cisek 2017). This task can be solved by updating the evolving likelihood for the two alternatives with each token movement. This solution could account for the monkeys' performance only if a choice-independent, gradually rising urgency signal was added to the likelihood values. Approximately one-third of GPe neurons and half of GPi neurons showed time-dependent activity changes that were consistent with this urgency signal. GPe and GPi activity was generally not modulated by likelihood changes and became choice selective only right before saccade onset (Figure 3*a*). These results suggest that, compared to the striatum, the GPe and GPi are less involved in evidence accumulation. Rather, they may mediate a time-dependent urgency signal to control how or when a decision is committed.

The GPi and SNr are commonly considered to be the BG output nuclei for skeletomuscular and oculomotor movements, respectively, and there are additional differences between their functions in perceptual decision making. For example, on the same saccade task and compared in the same monkeys, GPi and SNr neurons showed different response profiles in terms of the prevalence of visual or reward-related responses, whether the neuron pauses or increases firing around saccade time, and the extent of laterality in their responses (Shin & Sommer 2010). Although the SNr has yet to be examined systematically using perceptual decision tasks per se, previous studies using other visual-saccade tasks showed that (*a*) SNr neurons can encode the likelihood of a target location (Basso & Wurtz 2002) and (*b*) electrical microstimulation of the SNr broadens the RT distribution of memory-guided saccades (Basso & Liu 2007), which could be indicative of a change in the variability of the bound for initiating saccades. These results suggest that the SNr may contribute to evidence accumulation and decision commitment during perceptual decision making, beyond providing the urgency signal.

4.3. Subthalamic Nucleus

Two potential contributions of the STN have been proposed, loosely related to its position in the hyperdirect and indirect pathways, respectively: (*a*) The STN may control the

effective decision bound dynamically over time, and (*b*) it may also contribute to evidence accumulation by helping to normalize likelihood-related quantities, which can be equivalent to setting the effective bound. Both roles are supported by experimental data from human subjects performing perceptual decision tasks.

The bound-controlling role of the STN is supported by the observed effects of deep brain stimulation (DBS) of the STN in PD patients. In PD patients, DBS disrupts deliberation for difficult choices, leading to shorter RTs, which may reflect a lowering of the decision bound (Cavanagh et al. 2011, Coulthard et al. 2012, Frank et al. 2007, Herz et al. 2016, Zavala et al. 2014). Because the therapeutic effects of DBS resemble those of STN lesions, and DBS tends to disrupt movement-related STN activity patterns (Bergman et al. 1990, Schor et al. 2022), it may be assumed that DBS suppresses normal STN activation. The DBS effects are thus consistent with the observation from nonperceptual decision tasks, where STN neurons are activated when monkeys or rodents need to suppress the more automatic or default actions to generate the appropriate, rewarded responses (Isoda & Hikosaka 2008, Schmidt et al. 2013). These results together suggest that STN activation could increase the decision bound to reduce the possibility of inappropriate behaviors; STN suppression (e.g., DBS) may decrease the decision bound to terminate deliberation prematurely.

The evidence-normalizing role of the STN is supported by other aspects of DBS effects. Computationally, evidence normalization in models such as the SPRT and DDM requires interactions of signals associated with different alternatives. Without such interactions, these models are reduced to a race model, in which independent evidence accumulators for different alternatives compete, and the first accumulator to reach the decision bound determines the final choice. These models have different predictions of how choice and RT are related. If DBS disrupts STN activity and thereby the interactions, then subjects' performance is expected to show changes in the choice–RT relationship as the internal computations change from an SPRT- or DDM-like to a race model–like process. Indeed, in PD patients, DBS not only speeds up choices, as described above, but also induces changes in the choice–RT relationship, consistent with a disruption of the interactions (Green et al. 2013). These results together suggest that the STN may serve multiple roles in the decision process.

5. EXPERIMENTAL EVIDENCE FOR BASAL GANGLIA CONTRIBUTION TO FLEXIBLE PERCEPTUAL DECISIONS

The anatomical and physiological evidence described above has suggested specific contributions of the BG to implementing computations to support decision formation and evaluation in a stationary environment. The well-known context-dependent BG activity patterns and the multiple neuromodulatory projections to the BG have also led to the idea that the BG may be especially instrumental in modulating decisions to flexibly adapt to changes in the task environment. In this section, I review our current understanding of the BG's contribution to flexible control of perceptual decisions, based on experimental results using several common task manipulations.

5.1. Prior Bias

Prior bias in perceptual decisions can arise when the alternatives have different prior probabilities of being the correct one. With equal priors, the best strategy is typically to base the decision on evidence alone. With unequal priors, the best strategy is typically to favor the alternative associated with the higher prior probability, especially when evidence is weak (Bogacz et al. 2006, Green & Swets 1966). In the SDT framework, the favoring can be implemented as a shift in the decision criterion (Figure 1*c*). In the DDM framework, the favoring can be implemented as a bias in drift, starting value, and/or bounds, depending on the particular task parameters (Bogacz et al. 2006, Simen et al. 2009) (Figure 1*g–i*). Neural correlates of prior probability–related decision bias have been observed in sensory or sensory-adjacent cortical areas (Hanks et al. 2011, Mulder et al. 2012, Preuschhof et al. 2010, Rao et al. 2012, Summerfield & Koechlin 2008). There is some evidence that the BG can also contribute to prior probability–related biases. For example, in a random-dot motion discrimination task, fMRI signals in the human putamen are sensitive to premotion cues that indicate different prior probability values (Forstmann et al. 2010b). PD patients show impairments in incorporating prior probability information into their perceptual decisions, even when such information is explicitly presented and acknowledged by the patients themselves (Perugini et al. 2016). Other evidence, however, suggests that the BG involvement may be less instrumental or depend on specific task features. For example, fMRI signals in the human putamen did not reflect prior probability for a numeric comparison task and did not reflect computations related to the biases induced by unequal priors (Mulder et al. 2012, Scheibe et al. 2010). These apparently contradictory results call for further systematic investigation to elucidate the roles of the BG in mediating prior probability–related biases.

5.2. Reward Bias

Reward bias in perceptual decisions arises when the alternatives are paired with different reward outcomes. Such a bias typically reflects the optimal strategy of favoring the alternative associated with the larger reward, especially when evidence is weak (Bogacz et al. 2006, Green & Swets 1966). Similar to the prior bias, the favoring can be implemented as a shift in decision criterion in the SDT framework and as a combination of biases in drift and relative bound heights in the DDM framework (Bogacz et al. 2006, Green & Swets 1966, Simen et al. 2009) (Figure 1*c*,*g–i*). Extensive studies of the BG's roles in reward processing, reinforcement learning, and value-based decisions have naturally led to the expectation that the BG are a key player also in mediating reward biases in perceptual decisions (Bogacz & Gurney 2007, Bogacz & Larsen 2011, Ito & Doya 2011, Kable & Glimcher 2009, Rao 2010, Ratcliff & Frank 2012, Summerfield & Tsetsos 2012).

Contrary to this expectation, results from several earlier studies in humans and rodents did not provide strong evidence for substantial involvement of the BG in mediating reward biases on perceptual decision tasks. For example, fMRI signals in the ventral striatum covaried with the reward obtained, but no signal in any BG nuclei correlated with the reward bias or reward-modulated decision variables estimated in the SA framework (Summerfield & Koechlin 2010). For a task that manipulated cost instead of reward, cost-sensitive fMRI signals were found in a region covering the STN and caudate, but such signals were not

sensitive to perceptual uncertainty (Fleming et al. 2010), suggesting that they most likely only reflected the conditions of asymmetric cost and might not directly modulate or mediate the reward bias. As subjects developed larger biases, the connectivity between the frontal cortex and ventral striatum also increased, but the activation of the ventral striatum itself did not seem to correlate with the reward bias (Chen et al. 2015). Striatal lesions also did not change the reward bias in rats performing an olfactory task with asymmetric reward conditions (Wang et al. 2013).

In contrast to these earlier negative results, recent results from my lab provide strong support for involvement of the caudate nucleus in mediating reward biases in perceptual decisions. We trained monkeys to perform a random-dot motion discrimination task and induced reward biases by assigning unequal rewards to the two choices (Fan et al. 2018). While the monkeys viewed the motion evidence, the activity of caudate neurons reflected various combinations of the quantities needed for making a decision, including reward context, motion direction, motion strength, and expected reward size. Many caudate neurons encoded, at the single-neuron level, both reward-related and evidence-related quantities (Doi et al. 2020, Fan et al. 2020), making them well-suited for the incorporation of reward and visual information. Electrical microstimulation in the caudate changed how the monkeys incorporated reward biases (Doi et al. 2020). More specifically, when examined in a DDM framework, both the monkeys' decision strategy and the effects of caudate microstimulation reflected a combination of reward context–dependent changes in drift and relative bound heights. Moreover, caudate microstimulation–induced changes mimicked the monkeys' reward bias strategy and were related to the tuning properties of neurons near the stimulation sites, establishing a crucial link between the monkeys' biased behavior and caudate activity.

Certain features of our results may also explain why previous fMRI and animal lesion studies did not find strong support for striatal involvement. First, striatal neurons are renowned for their heterogeneous patterns of activity (Nakamura & Ding 2017). In our caudate samples, each decision-related quantity was present at various times during the task and could correlate positively or negatively with neural activity (Doi et al. 2020, Fan et al. 2020), posing a challenge for its detection in aggregate neural measurements such as the fMRI signal. Second, in our task, monkeys adopted strategies that coordinated adjustments in the drift and relative bound heights, and the microstimulation effects followed the same coordination pattern (Doi et al. 2020). The microstimulation effect on each adjustment alone, however, varied greatly across sessions and stimulation sites and may give the impression of inconsistent caudate involvement. The perturbation effect's computational specificity, its dependence on the animals' voluntary decision strategy, and its relationship to the neural tuning properties can all pose challenges for understanding the involvement of a neural substrate in a complex decision process.

5.3. Speed–Accuracy Tradeoff

When the decision is based on evidence accumulation over time, faster decisions mean fewer evidence samples and therefore tend to be less accurate. In the SA framework, the speed–accuracy tradeoff (SAT) can be achieved with symmetric adjustments of decision bounds, which can simultaneously influence choice accuracy and decision time in patterns consistent

with behavioral performance in humans and animals (Heitz 2014, Wald 1947). Under certain conditions, decision bound adjustment can also provide the optimal solution for maximizing reward rates (Gold & Shadlen 2002).

In one of the first experiments demonstrating the BG's roles in perceptual decision making, subjects were instructed to make visual motion discrimination decisions that prioritized speed, accuracy, or the subjects' choice between the two (Forstmann et al. 2008). Before the motion stimulus appeared, the anterior striatum was activated more when speed was prioritized. Across subjects, decision bound adjustments covaried with striatal activation and the connectivity strength between the frontal cortex and striatum (Forstmann et al. 2010a, Green et al. 2012), suggesting that the striatum is a key node for mediating bound adjustments for the SAT. Consistent with these findings, patients with focal striatum lesions use unusually low decision bounds compared to control subjects (Winkel et al. 2016). However, these patients can still modulate their decision bound according to accuracy or speed instructions, suggesting that the striatum is not the only route for SAT control.

The STN is another prime candidate for controlling the SAT and may have roles complementary to those of the striatum. In PD patients, low-frequency oscillations in the local field potential signals recorded in the STN covaried with decision bound adjustment, especially when accuracy was prioritized (Herz et al. 2017). In another study, healthy subjects were asked to adjust decision bounds based on cues (Mansfield et al. 2011). Depending on whether the cue was random, predictable but different from the previous trial, or repeated from the previous trial, different combinations of the striatum and STN were activated. In contrast, the presupplementary motor area (pre-SMA) showed similar activation regardless of cue uncertainty. As the pre-SMA projects to both the striatum and the STN, the cortical signals related to the decision bound are likely regulated to activate the two BG nuclei differentially to prioritize speed and accuracy, respectively, and to accommodate different levels of task uncertainty.

5.4. Attention

Studies of attention control of perceptual decisions have traditionally focused on cortical and thalamic-collicular circuits for top-down and bottom-up mechanisms, respectively. A recent proposal has suggested a different view of attention control as a product of value-based state estimation, a process that could be implemented through the BG pathway (Krauzlis et al. 2014, Samejima & Doya 2007). This idea is supported by observations of attention deficits in PD patients (Cepeda et al. 2008, Downes et al. 1989, Sharpe 1990) and more recent studies in animals. In monkeys performing visual attention tasks, striatal activity is modulated by the spatial location of attentional cues (Arcizet & Krauzlis 2018, Bogadhi et al. 2018, Caspari et al. 2015). Such modulation was present even in the absence of any spatial relationship between the cue location and the decision-indicating action, suggesting that the striatal involvement operates on the decision process itself. In mice trained to detect an orientation change in the visual stimulus, optogenetic activation of direct pathway striatal neurons increased both hit and false-alarm rates, consistent with a decrease in the decision criterion in the SDT framework (Wang & Krauzlis 2020). Collectively, these findings suggest that the striatum is causally involved in attention control of visual perception.

6. THEORETICAL STUDIES OF THE BASAL GANGLIA IN PERCEPTUAL DECISION MAKING

The experimental results reviewed above have implicated BG nuclei in multiple decision-related computations, including accumulation of evidence, application of decision rules, and evaluation of past decisions. In some cases, different BG nuclei appear to be involved in overlapping computations (e.g., both the striatum and STN may control decision rules; both striatal and DA neurons may support decision evaluation). How does the BG pathway as a whole support perceptual decision making, and what decision-related computations can be substantiated with the BG circuits? Several theoretical models explored these questions using available neurophysiological data and made new predictions that can be tested experimentally. Below, I highlight four examples that focus on different forms of decision-related computations.

The first two examples explored the link between BG circuits and a DDM-like process for two-alternative decision tasks. Wang and colleagues implemented a large-scale biologically realistic network model, including pools of neurons representing the cortex, the direct and indirect pathways of the BG, and the SC (Lo & Wang 2006, Wei et al. 2015). The cortical pool implements evidence accumulation, and the SC pool implements decision commitment based on the combined inputs from the cortex and BG. The BG pathway can thus modulate the effective decision bound applied onto the accumulated evidence. The model assumes that the cortical output to the SC takes the form of a gradual ramp from a low baseline level, and the BG output from the SNr to the SC takes the form of an abrupt pause in the high baseline level for one alternative only (Figure 3*b*). With these assumptions, a change in the synaptic weight of the cortico-striatal connection induces larger effects on the effective decision bound than the same weight change at the cortico-SC connection. In other words, adding the BG circuit can expand the range of decision bound adjustments beyond that of the cortico-SC network alone. The model also makes predictions about the roles of the STN and GPe. Because striatal neurons show evidence-dependent ramping activity and inhibit SNr neurons, for SNr neurons to exhibit the abrupt-pause activity pattern, the striatal inhibition needs to be balanced out with a similarly ramping excitatory input until close to the time of the pause. The model predicts that the indirect pathway, via the GPe and STN, provides such counterbalancing, ramping excitation (Figure 3*b*).

Ratcliff & Frank (2012) explored how the BG pathway, especially the hyperdirect pathway, may modulate decision commitment. Similar to the first example, their model also assumes that the cortex provides evidence accumulation signals to the BG. The model further assumes that the STN receives direct early-onset excitation from the cortex via the hyperdirect pathway. The STN activation at the beginning of the decision process prevents inhibition of GPi neurons and suppresses premature decisions. STN activity then gradually dissipates with the buildup of inhibition from the GPe and neural accommodation, allowing the regular DDM process to form decisions (Figure 3*c*). By comparing the simulated RT and choice data to DDM predictions, Ratcliff & Frank observed that the synaptic weight of the STN–GPi projection was parametrically related to the decision bound and nondecision times in the DDM: Larger weight raised the bound height and increased nondecision times.

Because of the early activation pattern of STN activity, if the BG simply relay accumulated evidence from the cortex, then the increase in nondecision times implies a delay in when the accumulated evidence is used. Alternatively, if the BG are directly involved in evidence accumulation, then such an increase in nondecision times implies that the BG may modulate when evidence accumulation itself begins.

In the third example, Bogacz & Gurney (2007) used the BG circuit to implement a multihypothesis SPRT (MSPRT). Crudely speaking, with two alternatives, an SPRT accumulates evidence in the form of the difference in the likelihoods for the two alternatives given an observation. With multiple alternatives, the MSPRT accumulates evidence in the form of the likelihood of an observation given one alternative, normalized by pooled likelihoods for all alternatives. In the model, each striatopallidal channel (direct pathway) relays from the cortex the likelihood values given one alternative (Figure 3*d*). Likelihood values for all alternatives are pooled from the cortex to the STN. The normalization was implemented with a combination of exponent and logarithmic functions, substantiated by the STN and GPe, respectively. Under these assumptions, the pallidal output activity can reflect temporally accumulated, normalized likelihood values as required by the MSPRT. A pause in activity in one pallidal output channel triggers a choice, akin to crossing a decision bound.

These three example models used physiologically plausible parameters to link the BG circuits to prescribed decision-related computations in the SA framework. With these parameters, the models can generate choice and RT data consistent with human and animal performance. Additional DA-dependent plasticity at the corticostriatal synapses can support reward feedback–dependent tuning of these parameters to improve perceptual decisions under different task contexts (Bogacz & Larsen 2011, Hsiao & Lo 2013, Ratcliff & Frank 2012).

In the fourth example, Rao (2010) incorporated decision formation and reinforcement learning in a single decision process to learn the appropriate decision-related computations. The model maps a four-layer network (cortex, striatum, pallidum, and DA neurons) onto a partially observable Markov decision process. The cortical layer computes the posterior probability distributions over possible states of the environment (belief states), which are updated after each time step by incorporating all evidence received thus far. The striatal layer represents a set of Gaussian basis functions, and each neuron is activated in proportion to the similarity between the cortical input and its preferred basis function. Interactions between the striatal layer and DA neurons implement a temporal difference learning algorithm that tunes the synaptic weights at the corticostriatal and striatopallidal connections. After learning, the striatal layer efficiently represents the cortical belief-state input in a compressed form, and the pallidal layer implements the optimal policy for converting striatal output to a discrete action. The learned solution can approximate an accumulation-to-bound process with a collapsing bound for decisions under deadline and capture the performance of monkey and human subjects on a perceptual decision task (Huang & Rao 2013, Khalvati et al. 2021, Rao 2010).

These four example models thus used the BG circuits in different ways to implement accumulation-to-bound decision processes. The presumed roles for individual BG nuclei also differ among the models and await experimental tests (Figure 3*b–d*).

7. CONCLUDING REMARKS AND OPEN QUESTIONS

In this review, I highlight theoretical and experimental results that have clearly established that the BG circuits are key players in perceptual decision making. Several major knowledge gaps remain.

First, there is a lack of direct measurement of single-neuron activity in key BG nuclei such as the STN and SNr in the context of perceptual decision tasks. This gap in our knowledge leaves open several obvious questions. For example, do SNr neurons behave the same way as GPi neurons (i.e., providing an urgency signal only), or do they behave more like the striatal neurons (i.e., sensitive to evidence strength and/or choice)? Are the bound-controlling and evidence-normalizing roles of the STN mediated by different STN subpopulations? Answers to these questions are critical for constraining and refining BG models of perceptual decision making.

Second, it remains unclear how evaluative signals in the striatum are computed and used for effective evaluation of decisions. Does striatal activity reflect the most effective evaluation signal for a specific task, or does it maintain multiple forms of evaluative signals for enhanced flexibility? In both cases, how is the best evaluative signal identified and controlled in the brain?

Third, in simpler visuo-saccade tasks, the activity of BG neurons and the effects of their perturbations often depend on task contexts (e.g., Basso & Liu 2007, Hikosaka et al. 1989, Jantz et al. 2017, Kawagoe et al. 1998). To what extent does such task dependence extend to perceptual decision making? How do single neurons in the BG interact to support flexible perceptual decision making in different contexts?

Fourth, we know very little about how the three different BG pathways interact and even less about the computational contributions of different cell types (e.g., striatal interneurons) or noncanonical projections (e.g., from the GPe to striatum).

Finally, the BG are a part of the larger decision network that includes the cortex, the thalamus, and the SC. Our understanding of the BG's roles also requires a better picture of how the whole network interacts to make decisions and the extent of redundancy and interdependence. Such information is especially important for clinical diagnosis and treatments of patients with long-term diseases involving BG dysfunction.

To fill these knowledge gaps, systematic examinations are needed that combine rigorous behavioral control, large-scale recording techniques that track neural signals at the single-neuron and population levels across brain regions, and perturbation techniques that can probe the link between these neural signals and behavioral output. The advent of advanced genetic tools offers additional opportunities to target cell type– and/or pathway-specific neural populations. It is an exciting time in the field, and I am hopeful that our improved

knowledge of the BG's roles will help us to understand better the cognitive deficits in patients afflicted with BG-related diseases.

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SUMMARY POINTS

1. The basal ganglia causally contribute to perceptual decision making to guide actions.
2. The striatum is involved in evidence accumulation to support decision formation and evaluation.
3. The basal ganglia can provide flexibility in decisions that need to incorporate sensory evidence and nonsensory factors, such as prior bias, reward expectation, and the speed–accuracy tradeoff.

FUTURE ISSUES

1. How do single neurons in different basal ganglia nuclei interact to implement decision-related computations?
2. What are the organizational principles for the basal ganglia pathways, based on their roles in motor control, perceptual decision making, value-based decision making, etc.?
3. How do the basal ganglia interact with the cortex and other subcortical regions to ensure appropriate and flexible decision making in different task contexts?
4. How do pathological changes in the basal ganglia cause deficits in decision making in the patient population?

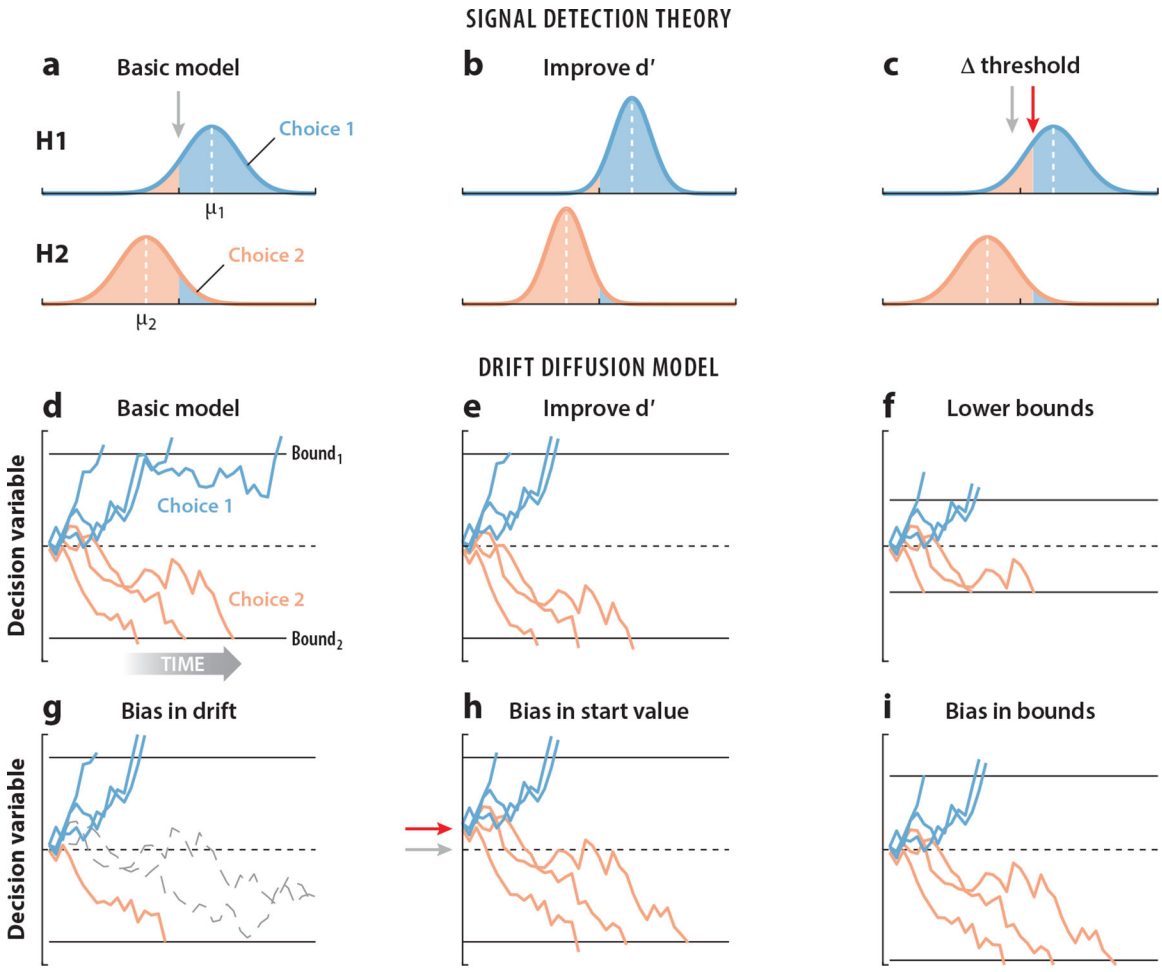

Figure 1.

Theoretical frameworks for studying perceptual decision making. (*a*) Illustration of the signal detection theory. The top and bottom curves indicate the likelihood functions for each of the two alternatives. The gray arrow indicates the observation value that corresponds to a decision threshold with a likelihood ratio of 1. Filled areas indicate observations that would lead to either choice 1 (*blue*) or choice 2 (*orange*). Note that the orange-filled area for the top curve and the blue-filled area for the bottom curve correspond to incorrect choices. (*b*) Improving the d′ (sharpening the likelihood functions) can reduce incorrect choices. (*c*) Changing the decision threshold biases the choice. (*d*) Illustration of the drift diffusion model. The decision variable (DV) trajectories are shown for three trials of each choice. A decision is committed when the DV reaches one of the two bounds. (*e*) Improving the d′ can increase the drift rates for both choices, leading to more accurate choices with shorter decision time. (*f*) Lowering the bounds symmetrically can reduce decision time but may also lead to more errors. (*g*) When fictive evidence for choice 1 is added at each time point, the resulting bias in drift rate leads to more and faster choice 1 and fewer and slower choice 2. The gray traces represent trials when a decision is not reached in the time window. (*h*) A positive offset added to the DV leads to more and faster choice 1 and fewer and slower choice 2. (*i*) Changing the bounds asymmetrically also biases the decision process.

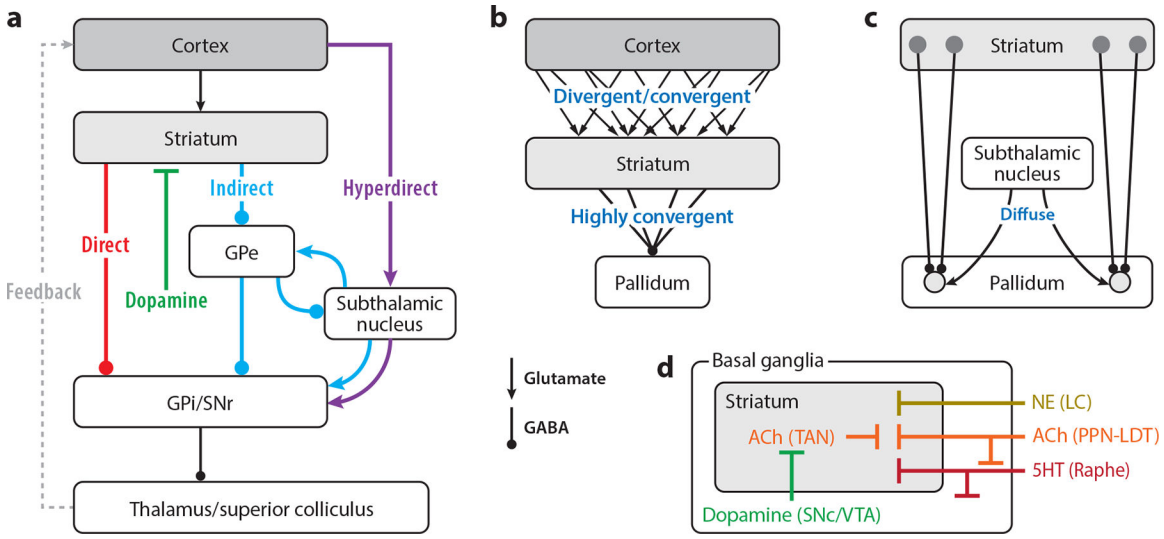

Figure 2.

The basal ganglia pathway. (*a*) A simplified diagram of the cortico-basal ganglia-thalamus/SC loop. (*b*) The basal ganglia pathway is highly convergent. (*c*) The STN has diffuse projections to pallidal output neurons. (*d*) The basal ganglia nuclei, especially the striatum, receive multiple types of neuromodulatory inputs. Abbreviations: 5HT, serotonin; ACh, acetylcholine; GPe, external segment of the globus pallidus; GPi, internal segment of the globus pallidus; LC, local coeruleus; NE, norepinephrine; PPN-LDT, pedunculopontine nucleus-laterodorsal tegmental complex; SC, superior colliculus; SNc, substantia nigra pars compacta; SNr, substantia nigra pars reticulata; STN, subthalamic nucleus; TAN, tonically active neurons; VTA, ventral tegmental area.

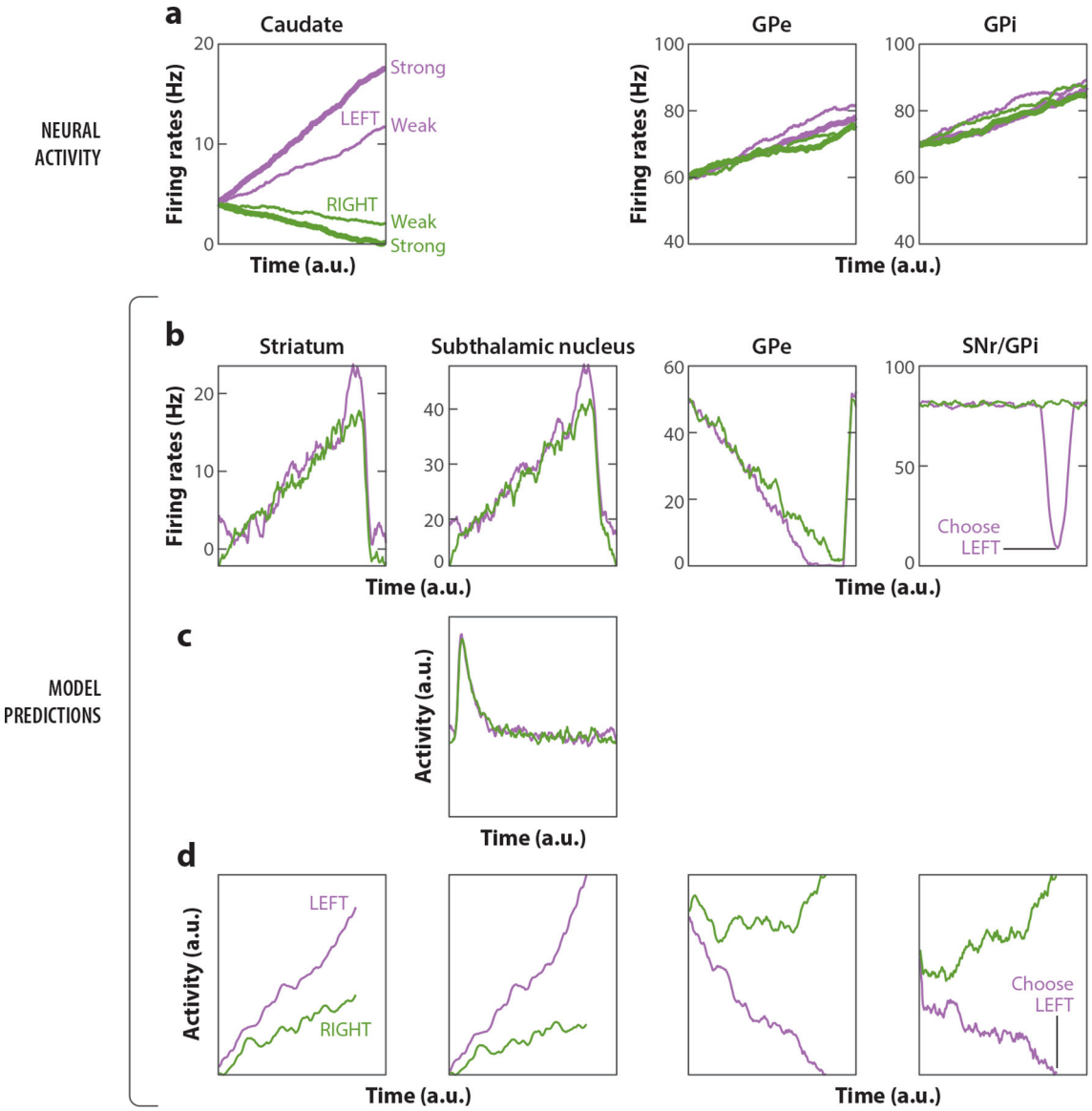

Figure 3.

Model-predicted and observed neural activity patterns in the BG. (*a*) Illustration of observed single-neuron activity patterns in the caudate (based on Ding & Gold 2010), GPe, and GPi (based on Thura & Cisek 2017). Note that GPi neurons can show choice- and evidence-independent upward or downward ramping activity. (*b*) Illustrations of predicted striatal, GPe, STN, and SNr activity patterns during a perceptual decision task, based on the model described byWei et al. (2015). A choice is made when SNr activity is sufficiently suppressed. (*c*) Illustration of predicted STN activity patterns, based on the model described by Ratcliff & Frank (2012). (*d*) Illustrations of predicted striatal, GPe, STN, and pallidal output activity, based on the model described by Bogacz & Gurney (2007). Abbreviations: BG, basal ganglia; GPe, external segment of the globus pallidus; GPi, internal segment of the globus pallidus; SNr, substantia nigra pars reticulata; STN, subthalamic nucleus.