Neural Dynamics of the Basal Ganglia during Perceptual, Cognitive, and Motor Learning and Gating

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Abstract

This article summarizes neural models that simulate how the basal ganglia contribute to associative and reinforcement learning, and to movement gating, in multiple brain systems. The first model proposes how the substantia nigra pars compacta (SNc) generates widespread dopaminergic learning signals in response to unexpected rewarding cues, including a circuit for adaptively timed learning using metabotropic glutamate receptor (mGluR)-mediated Ca2+ spikes that occur with different delays in striosomal cells. Similar circuits for spectral timing occur in cerebellum and hippocampus. The TELOS model shows how the substantia nigra pars reticulata (SNr) learns to selectively gate saccadic eye movements or cognitive plans, and how spatially-invariant object categories can activate spatially-variant representations of specific actions. The VITE model proposes how basal ganglia gating controls selection and variable speeds of arm movements. The cARTWORD model explains how prefrontally-controlled basal ganglia gates can explain phonemic restoration, notably how future context can influence how past sounds are consciously heard. The MOTIVATOR model clarifies how the basal ganglia and amygdala coordinate their complementary functions in the learning and performance of motivated acts. The lisTELOS model proposes how sequences of saccades can be learned and performed from an Item-Order-Rank spatial working memory under the control of three parallel basal ganglia loops. Basal ganglia gating in working memory storage, visual imagery, useful-field-of-view, thinking, planning, and Where's Waldo searching are also discussed, as is how its breakdown can lead to hallucinations.

Keywords: basal ganglia, prefrontal cortex, inferotemporal cortex, parietal cortex, frontal eye fields, supplementary eye fields, superior colliculs, amygdala, orbitofrontal cortex, motor cortex, spinal cord, cerebellum, supplementary eye fields**,** saccades, arm movements, Item-Order-Rank working memory, reinforcement learning, adaptive timing, movement gating, visual imagery, spatial attention, thinking, planning, search, hallucinations

1. Introduction

1.1. Linking Brain to Mind with Neural Models: Method of Minimal Anatomies

The rapid development of behavioral and cognitive neuroscience parallels the growing interest in mechanistically linking brain mechanisms to behavioral functions. Expressed in another way, this interest asks: How can a brain gives rise to a mind? How can the classical Mind/Body Problem be solved? The remarkable experimental and theoretical progress in understanding brain *or* mind in the fields of neuroscience and psychology has not often provided clear mechanistic links between them, if only because mind is an emergent property that arises from widespread interactions among multiple brain regions, and experimental methods can probe the detailed structure of such interactions only partially. Yet establishing such a linkage between brain and mind is crucial in any mature theory of how a brain or mind works. Without such a link, the mechanisms of the brain have no functional significance, and the functions of behavior have no mechanistic explanation.

In order to establish such a link with sufficient clarity for it to be scientifically predictive, rigorous mathematical models are needed that can simultaneously describe multiple levels of brain and behavioral organization. A rapidly growing number of such models can now quantitatively simulate the neurophysiologically recorded dynamics of identified nerve cells in known anatomies *and* the behaviors that they control. Many predictions of these models have also been supported by subsequent experiments over the years. In this restricted sense, the Mind/Body Problem is at last starting to be understood.

A particularly successful approach uses a theoretical method that has been systematically developed and applied during the past fifty years (Grossberg, 1999). One begins with scores or even hundreds of parametrically structured *behavioral* experiments in a particular problem domain because the brain has evolved to achieve *behavioral* success. Starting with behavioral data makes sense if one wants to derive a model whose brain mechanisms have been shaped during evolution by behavioral success. Large number of behavioral experiments are needed to rule out many otherwise seemingly plausible answers.

The method uses a large behavioral database to discover novel design principles and mechanisms to explain how an individual, behaving in real time, can generate these data as emergent properties. The minimal mathematical model that can realize these design principles has always looked like part of a brain. Fifty years of modeling have consistently led to the empirical conclusion that brains look the way that they do because they embody the natural computational designs to control an individual's autonomous adaptation to a changing environment in real time. Moreover, this kind of behavior-to-principle-to-model-to-brain theoretical derivation has often disclosed unexpected functional roles of the neural mechanisms that are not clear from neural data alone.

Having made a connection top-down from behavior to brain, one can now use mathematical and computational analysis to disclose what the minimal model, and its variations, can and cannot explain. Using this information, one can now also exert upon the model both top-down constraints from behavior, and bottom-up constraints from brain, to point to one or more additional design principles that are needed to explain even more data. These new design principles and their mechanistic realizations are then consistently assimilated into the model. This process is repeated cyclically, thereby leading by a process of "conceptual evolution" to a series of progressively unlumped models, each consistent with the others, and with an increasing broad explanatory and predictive range, including more neural mechanistic detail. At the present time, although one cannot "derive the entire brain" in one step, an increasing number of these

models can individually explain behavioral, neurophysiological, neuroanatomical, biophysical, and even biochemical data.

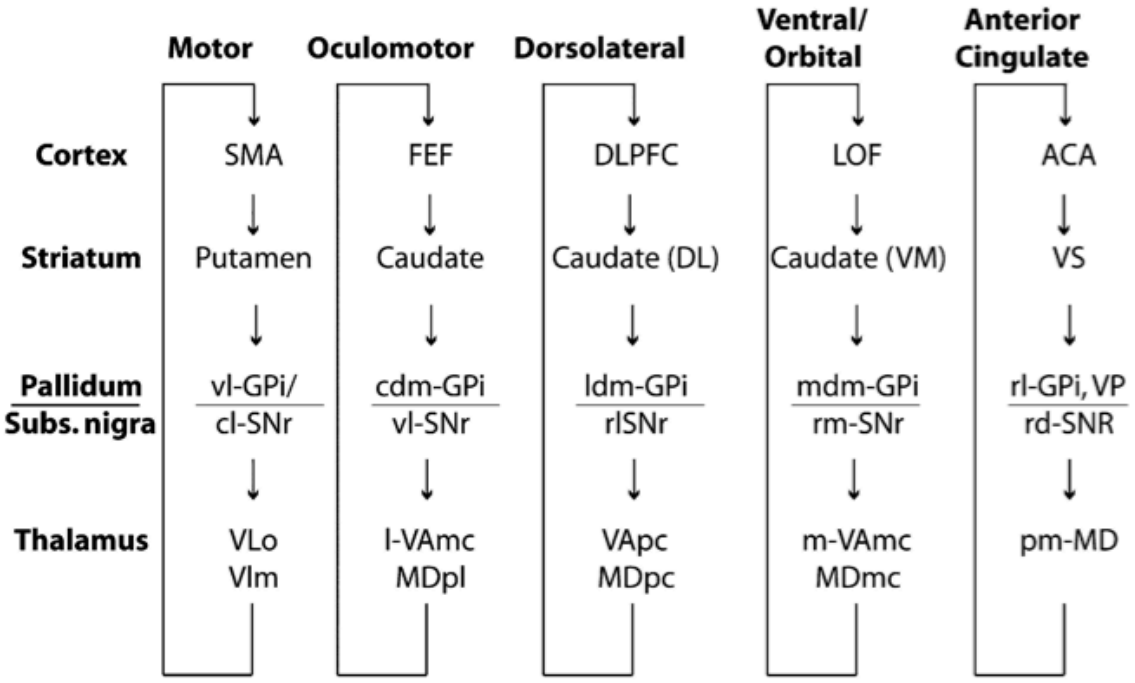


Figure 1. Basal ganglia parallel loops. The dorsal and ventral striatum are differentially connected to discrete prefrontal cortical regions in segregated cortico-striatal circuits, as summarized by Alexander, DeLong, and Strick (1986). The putamen plays a critical role within the so-called motor circuit, while the caudate forms part of the oculomotor, dorsolateral and ventral/orbital circuits. SMA= supplementary motor area, vl-GPi = ventrolateral globus pallidus (internal segment), cl-SNr = caudolateral substantia nigra pars reticulata, VLo = ventrolateral nucleus of thalamus pars oralis, Vlm = ventrolateral nucleus of thalamus pars medialis, FEF = frontal eye fields, cdm-GPi = caudodorsomedial globus pallidus (internal segment), vl-SNr = ventrolateral substantia nigra pars reticulata, l-VAmc = lateral ventral anterior nucleus of thalamus pars magnocellularis, MDpl = parvocellular subnucleus ofmediodorsal nucleus of the thalamus, DLPFC = dorsolateral prefrontal cortex, Caudate (DL) = dorsolateral caudate, Caudate (VM) = ventromedial caudate, mdm-GPi = dorsomedial globus pallidus (internal segment), rm-SNr = rostromedial substantia nigra pars reticulata, m-VAmc = medial ventral anterior nucleus of thalamus pars magnocellularis, MDmc = magnocellular subnucleus of mediodorsal nucleus of the thalamus, ACA = anterior cingulate area, VS = ventral striatum, rl-GPi = rostrolateral globus pallidus (internal segment), VP = ventral posterior nucleus of the thalamus, rd-SNr = rostrodorsal substantia nigra pars reticulata, pm-MD= posteromedial mediodorsal nucleus of the thalamus [Reprinted with permission from Grahn, Parkinson, and Owen, 2009].

1.2. Modeling the Basal Ganglia

The above perspective helps to clarify the challenge facing any theorist who wishes to model the basal ganglia. This is true because the basal ganglia, in addition to comprising multiple subcortical nuclei, are widely interconnected with multiple other brain regions, including the cerebral cortex, thalamus, amygdala, and hippocampus (http://en.wikipedia.org/wiki/Basal_ganglia, http://www.scholarpedia.org/article/Basal_ganglia). Numerous experimental studies have proposed roles for the basal ganglia in processes such as reinforcement learning and action selection, or gating. Figure 1 schematizes how these functions are organized in parallel thalamo-cortical motor, spatial, visual, and affective loops. To understand how these processes work, and what kinds of events are reinforced or selected, one needs models of how all the relevant brain regions interact and how these interactions give rise to the behaviors that they control.

1.3. Complementary Computing and Laminar Computing

What form do neural models of such processes take? This answer is constrained by the discovery of novel computational paradigms whereby advanced brains are organized.

Complementary Computing. Complementary Computing addresses the question: What is the nature of brain specialization? The brain's organization into distinct anatomical areas and processing streams shows that brain processing is indeed specialized. However, much data shows that these streams interact strongly and do not compute their respective functions in the manner of independent modules. *Complementary Computing* (Grossberg, 2000b, 2012) concerns the discovery that pairs of parallel cortical processing streams compute complementary properties in the brain. Each stream has complementary computational strengths and weaknesses, much as in physical principles like the Heisenberg Uncertainty Principle. Each cortical stream can also possess multiple processing stages. These stages realize a *hierarchical resolution of uncertainty*. "Uncertainty" here means that computing one set of properties at a given stage prevents computation of a complementary set of properties at that stage. Complementary Computing proposes that the computational unit of brain processing that has behavioral significance consists of parallel interactions between complementary cortical processing streams with multiple processing stages to compute complete information about a particular type of biological intelligence. For example, it will be reviewed below how the basal ganglia and amygdala compute complementary properties of reinforcement learning, with the basal ganglia helping to control learning in response to unfamiliar and unexpected events and the amygdala helping to control conditioned reinforcement and incentive motivational support for familiar and expected events.

Laminar Computing. Laminar Computing concerns the fact that the cerebral cortex, the seat of higher intelligence in all modalities, is organized into layered circuits (often six main layers) that undergo characteristic bottom-up, top-down, and horizontal interactions. Laminar Computing proposes how variations and specializations of this shared laminar design embody different types of biological intelligence, including vision, speech and language, and cognition (Grossberg, 1999, 2012). Laminar Computing explains how the laminar design of neocortex may realize the best properties of feedforward and feedback processing, digital and analog processing, and bottom-up data-driven processing and top-down attentive hypothesis-driven processing. For example, it will be reviewed below how the basal ganglia interact with prescribed layers of the frontal eye fields and prefrontal cortex to control the learning and performance of individual eye movements and sequences of eye movements.

2. Neural Models for Reinforcement Learning and Action Selection and Planning

Each of the subsequent sections summarizes a model that explains different aspects of how the basal ganglia contribute to associative and reinforcement learning, and to movement gating, in multiple brain systems.

The model in Section 3 proposes how the substantia nigra pars compacta (SNc) generates widespread dopaminergic learning signals in response to unexpected rewarding cues, including a circuit for adaptively timed learning using metabotropic glutamate receptor (mGluR)-mediated Ca2+ spikes that occur with different delays in striosomal cells. This section also notes that similar circuits for such adaptively timed learning, which is called spectral timing, seem to occur at the parallel fiber-Purkinje cell synapses of the cerebellum, where they control adaptively timed movements, and the dentate-CA3 circuits of the hippocampus, where they control adaptively timed motivated attention. The hippocampal adaptive timing circuits go through lateral entorhinal cortex and its hippocampal projections, and include "time cells". These circuits seem to be computationally homologous to circuits for spatial navigation in medial entorhinal cortex and its hippocampal projections, and include grid and place cells.

The TELOS model that is reviewed in Section 4 shows how the substantia nigra pars reticulata (SNr) learns to selectively gate saccadic eye movements or cognitive plans. It also clarifies how spatially-invariant object categories in the What cortical stream can learn to control spatially-selective movement representations in the Where cortical stream.

The VITE model that is reviewed in Section 5 proposes how basal ganglia gating controls selection and variable speeds of arm movement trajectories that are planned in cortical circuits, including trajectories that can cope with obstacles and unexpected perturbations. The FLETE model complements VITE by simulating the spinal cord and cerebellar circuits that enable VITE to generate accurate trajectories that take into account muscle forces and tensions of a multi-joint arm.

The cARTWORD model that is reviewed in Section 6 explains how prefrontally-controlled basal ganglia gates can explain phonemic restoration, notably how future context can influence how past sounds are consciously heard. cARTWORD describes a hierarchy of laminar cortical circuits that are variations of laminar cortical circuits which have also been used to model 3D vision and figure-ground perception, as well as cognitive working memory and list chunking processes. These list chunks represent the most predictive sequences of items that are stored in the working memory at any time. In cARTWORD, the cognitive working memory activates list chunks that represent the most predictive sequences of stored sounds at any given moment. When such a list chunk gets sufficiently active, it opens a basal ganglia gate that enables the entire cortical hierarchy to generate a resonance that represents the consciously heard sequence as it unfolds through time.

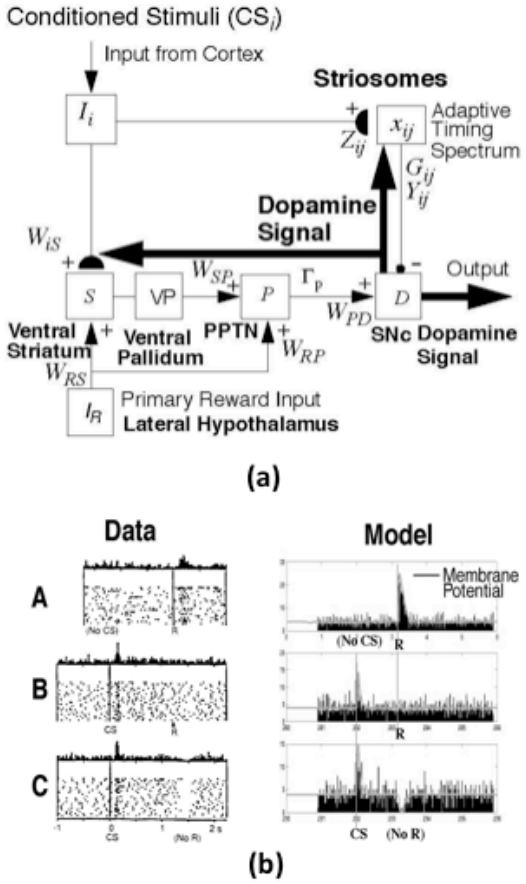
The MOTIVATOR model that is reviewed in Section 7 clarifies how the basal ganglia and amygdala coordinate their complementary functions in the learning and performance of motivated acts. In particular, whereas the basal ganglia generate Now Print dopaminergic signals to drive new learning in response to unexpected rewards, the amygdala is activated by already learned conditioned reinforcers and generates incentive motivational outputs that control motivated attention and performance to acquire valued and familiar goal objects. Of particular importance in MOTIVATOR is the role of inferotemporal-amygdala-orbitofrontal resonances that focus attention upon motivationally salient objects while supporting conscious awareness of emotions.

The lisTELOS model that is reviewed in Section 8 proposes how sequences of saccades

can be learned and performed from an Item-Order-Rank spatial working memory under the control of three parallel basal ganglia loops. Such an Item-Order-Rank working memory model can store sequences of items with multiple repeats in working memory, and is supported both by psychological and neurophysiological data. This Item-Order-Rank working memory is defined by a laminar cortical circuit that is a variant of the cARTWORD cognitive working memory. Variations of the same working memory design have been predicted represent spatial, linguistic, and motor sequences, thereby providing another example of the conceptual and mechanistic unification that Laminar Computing has begun to provide.

Section 9 summarizes how basal ganglia gating may also control working memory storage, visual imagery, useful-field-of-view in spatial attention, thinking, planning, and Where's Waldo searching, as is how its breakdown can lead to hallucinations.

Section 10 notes how complementary processes of spatially-invariant object category learning and motivated attention interact with spatially-variant control of actions. These complementary systems enable the brain to rapidly learn more about a changing world without experiencing catastrophic forgetting, yet to also be able to adapt its spatial and motor representations to efficiently control our changing bodies. The basal ganglia bridge this complementary divide to support learning and gating across the entire brain.



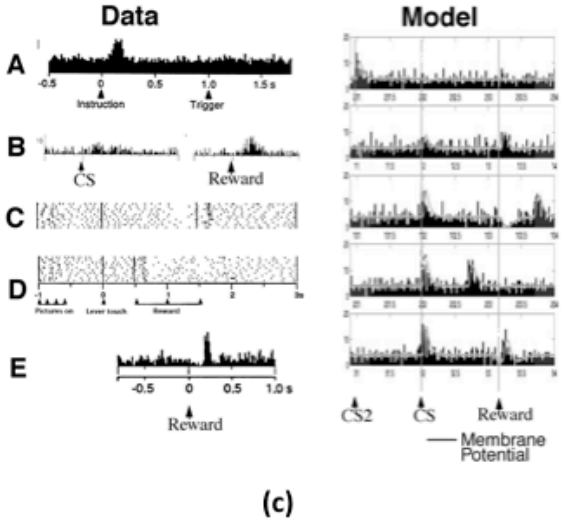


Figure 2. (a) Model circuit for the control of dopaminergic Now Print signals in response to unexpected rewards. Cortical inputs (I_i), activated by conditioned stimuli, learn to excite the SNc via a multi-stage pathway from the ventral striatum (S) to the ventral pallidum, and then on to the PPTN (P) and the SNc (D). The inputs I_i excite the ventral striatum via adaptive weights W_{iS}, and the ventral striatum excites the PPTN via double inhibition through the ventral pallidum, with strength W_{SP}. When the PPTN activity exceeds a threshold GP, it excites the SNc with strength W_{PD}. The striosomes, which contain an adaptive spectral timing mechanism (x_{ij}, G_{ij}, Y_{ij}, Z_{ij}), learn to generate adaptively-timed signals that inhibit reward-related activation of the SNc. Primary reward signals (I_R) from the lateral hypothalamus both excite the PPTN directly (with strength W_{RP}) and act as training signals to the ventral striatum S (with strength W_{RS}) that trains the weights W_{iS}. Arrowheads denote excitatory pathways, circles denote inhibitory pathways, and hemidisks denote synapses at which learning occurs. Thick pathways denote dopaminergic signals. [Reprinted with permission from Brown, Bullock, and Grossberg, 1999.]. (b) Dopamine cell firing patterns: Left: Data. Right: Model simulation, showing model spikes and underlying membrane potential. A. In naive monkeys, the dopamine cells fire a phasic burst when unpredicted primary reward R occurs, such as if the monkey unexpectedly receives a burst of apple juice. B. As the animal learns to expect the apple juice that reliably follows a sensory cue (conditioned stimulus, CS) that precedes it by a fixed time interval, then the phasic dopamine burst disappears at the expected time of reward, and a new burst appears at the time of the reward-predicting CS. C. After learning, if the animal fails to receive reward at the expected time, a phasic depression, or dip, in dopamine cell firing occurs. Thus, these cells reflect an adaptively-timed expectation of reward that cancels the expected reward at the expected time. [The data are reprinted with permission from Schultz et al. (1997). The model simulations are reprinted with permission from Brown et al. (1999).]. (c) Dopamine cell firing patterns: Left: Data. Right: Model simulation, showing model spikes and underlying membrane potential. A. The dopamine cells learn to fire in response to the earliest consistent predictor of reward. When CS2 (instruction) consistently precedes the original CS (trigger) by a fixed interval, the dopamine cells learn to fire only in response to CS2. [Data reprinted with permission from Schultz et al. (1993)]. B. During training, the cell fires weakly in response to both the CS and reward. [Data reprinted with permission from Ljungberg et al. (1992)]. C. Temporal variability in reward occurrence: When reward is received later than predicted, a depression occurs at the time of predicted reward, followed by a phasic burst at the time of actual reward. D. If reward occurs earlier than predicted, a phasic burst occurs at the time of actual reward. No depression follows since the CS is released from working memory. [Data in C and D reprinted with permission from Hollerman and Schultz (1998)]. E. When there is random variability in the timing of primary reward across trials (e.g., when the reward depends on an operant response to the CS), the striosomal cells produce a Mexican Hat depression on either side of the dopamine spike. [Data reprinted with permission from Schultz et al. (1993)]. [Model simulation reprinted with permission from Brown et al. (1999).]

3. Adaptively Timed Reinforcement Learning in Response to Unexpected Rewards

3.1. Balancing fast excitatory conditioning against adaptively timed inhibitory conditioning

This overview begins by reviewing a neural model that proposes how the basal ganglia may use parallel excitatory and inhibitory learning pathways to selectively respond to unexpected rewarding cues, and to thereby trigger widespread dopaminergic Now Print, or reinforcement learning, signals to multiple brain regions (Figure 2a; Brown, Bullock, and Grossberg, 1999). In

particular, humans and animals can learn to predict both the intensities and the times of expected rewards. Correspondingly, the firing patterns of dopaminergic cells within the substantia nigra pars compacta (SNc) are sensitive to both the predicted and the actual times of reward (Ljungberg et al., 1992; Schultz et al., 1993; Mirenowicz and Schultz, 1994; Schultz et al., 1995; Hollerman and Schultz, 1998; Schultz, 1998).

Figures 2 and 3 summarize some of the main neurophysiological properties of these cells along with model simulations of them. Notable among them (Figures 2b and 2c) is the fact that reinforcement learning enables SNc cells to respond immediately to unexpected cues, such as conditioned stimuli (CS), during classical conditioning, but to omit responses in an adaptively timed fashion to expected rewards, such as unconditioned stimuli (US). The model also simulates related anatomical and neurophysiological data about the pedunculo-pontine tegmental nucleus (PPTN), lateral hypothalamus, ventral striatum, and striosomes (Figure 3a). Thus, the responses of SNc cells are themselves altered by the conditioning process, even as they alter how other brain regions process associative learning signals.

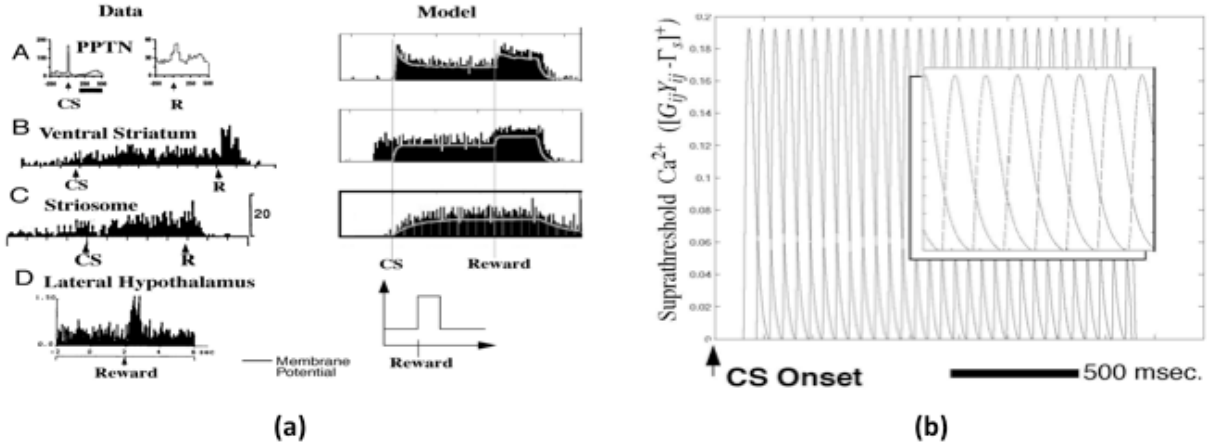


Figure 3. (a) Trained firing patterns in PPTN, ventral striatum, striosomes, and lateral hypothalamus. Left: Data. Right: Model simulations, showing model spikes and underlying membrane potential. A) PPTN cell (cat), showing phasic responses to both CS and primary reward. [Data reprinted with permission from Dormont et al. (1998)]. In the model, phasic signaling is due to accommodation or habituation (Takakusaki et al., 1997), which causes the cell to fire in response to the earliest reward-predicting CS and US reward, but not to subsequent CSs prior to reward. B) Ventral striatal cells show sustained working memory-like response between trigger and a US reward, and a phasic response to the US reward. [Data reprinted with permission from (Schultz et al., 1992)]. C) A ventral striatal cell, predicted here to be a striosomal cell, shows buildup to phasic primary reward response. For the model cell, j = 39. [Data reprinted with permission from (Schultz et al., 1992)]. D) A lateral hypothalamic neuron with a strong, phasic response to glucose reward. [Data reprinted with permission from Nakamura and Ono (1986)] The majority of these neurons fired in response to primary reward but not to a reward-predicting CS. The model lateral hypothalamic input is a rectangular pulse. [Model simulation reprinted with permission from Brown et al. (1999).]. (b) Striosomal spectral timing model and close-up (inset), showing individual timing pulses. Each curve represents the suprathreshold intracellular Ca^{2+} concentration of one striosomal cell. The peaks are spread out in time so that reward can be predicted at various times after CS onset. Learning does this by

strengthening the inhibitory effect of the striosomal cell with the appropriate delays. The model uses 40 peaks, spanning approximately 2 seconds and beginning 100 msec. after the CSs (cf., Grossberg and Schmajuk, 1989). Model properties are robust when different numbers of peaks are used. It is important that the peaks be sufficiently narrow and tightly-spaced to permit fine temporal resolution in the reward-cancelling signal. However, a trade-off ensues in that more timed signals must be used as the time between peaks is reduced. The timed signals must not begin too early after the CS, or they will erroneously cancel the CS-induced dopamine burst. The 100 msec. post-CS onset delay prevents this from happening. [Reprinted with permission from Brown et al. (1999).]

The neural model depicted in Figure 2a proposes how two parallel learning pathways from limbic cortex to the SNc work together to control adaptively timed SNc conditioning. One pathway controls excitatory conditioning through the ventral striatum, ventral pallidum, and PPTN. This pathway learns to generate CS-activated excitatory SNc dopamine bursts as conditioning proceeds (Figure 2bA). The other pathways controls adaptively timed inhibitory conditioning through the striosomes, thereby learning to prevent dopamine bursts in response to predictable reward-related signals. The net effect on SNc output bursting depends upon the balance of excitatory and inhibitory signals that converge upon these cells. When expected rewards are received, the excitatory and inhibitory signals are balanced, so that SNc cells do not fire (Figure 2bB). On the other hand, if an expected reward is not received, then striosomal inhibition of SNc that is unopposed by excitation results in a phasic drop in dopamine cell activity (Figure 2bC).

3.2. Spectral adaptively timed inhibitory conditioning by Ca^{2+} and mGluR

The adaptively timed inhibitory learning is proposed to arise from the population response of an intracellular spectrum of differently timed responses (Figure 3b). The differently timed responses are proposed to arise from metabotropic glutamate receptor (mGluR)-mediated Ca^{2+} spikes that occur with different delays in striosomal cells. A dopaminergic burst that co-occurs with a Ca^{2+} spike is proposed to potentiate inhibitory learning at that delay.

The model's mechanism for realizing adaptively timed inhibitory conditioning is proposed to be a variation of a mechanism of adaptively timed learning that is found in several brain regions. This mechanism is called *spectral timing* because it relies upon the population response of a spectrum of differently timed cells or cell sites. The Spectral Timing model proposes an answer to a perplexing problem: How do brains generate responses that are adaptively timed over hundreds of milliseconds or even seconds, when individual neuronal cell potentials respond on a time scale that is orders of magnitude faster? The model proposes that a gradient of Ca^{2+} responses within the mGluR system accomplishes this feat (Fiala, Grossberg, and Bullock, 1996), and that this is an ancient discovery by evolution that has been utilized in cellular tissues outside the brain as well.

3.3. Spectrally timed learning in basal ganglia, hippocampus, and cerebellum

Accordingly, the Spectral Timing model has been used to explain and simulate several different types of data that exhibit adaptively timed learning, including both normal and abnormal adaptively timed behaviors. The normal behaviors include reinforcement learning, motivated attention, and action, via circuits involving basal ganglia, hippocampus (Grossberg and Merrill, 1992, 1996; Grossberg and Schmajuk, 1989), and cerebellum (Fiala, Grossberg, and Bullock, 1996). In particular, a spectrally timed circuit through dentate-CA3 hippocampal circuits is proposed to control adaptively-timed motivated attention via incentive motivational signals that

are proposed to subserve the Contingent Negative Variation (CNV) event-related potential. A spectrally timed circuit through cerebellar (parallel fiber)-(Purkinje cell) synapses is proposed to control adaptively timed responding via mechanism of learned long-term depression (LTD). Abnormal adaptive timing due to cerebellar lesions, or in autistic individuals, may cause actions to be prematurely released in a context-inappropriate manner that can prevent them from receiving normal social rewards (Grossberg and Seidman, 2006; Grossberg and Vladusich, 2011; Sears, Finn, and Steinmetz, 1994).

It should also be emphasized that spectral timing is not the only mechanism whereby the brain can cause responses to be delayed over significant time intervals. Cognitive working memories also have this property, and have been modeled by laminar prefrontal cortical circuits (Grossberg and Pearson, 2008); see Section 8. One signature of spectral timing is a Weber Law property, also called scalar timing (Gibbon, Church, and Meck, 1984), whereby longer delays coexist with greater variance in the response distribution through time. A spectrum of adaptively timed "time cells" have been discovered using neurophysiological recordings in the hippocampus (MacDonald et al., 2011). These cells exhibit the predicted Weber law property.

3.4. Neural relativity: Space and time in the entorhinal-hippocampal system

Another interesting feature of the spectral timing story concerns the fact that the hippocampus processes spatial as well as temporal information. This observation raises the question: Why are both space and time both processed in the hippocampus? The fact of this convergence is consistent with data and hypotheses about a possible role of hippocampus in episodic learning and memory, since episodic memories typically combine both spatial and temporal information about particular autobiographical events; e.g., Eichenbaum and Lipton, 2008. Grid cells in the medial entorhinal cortex (Hafting et al., 2005) and place cells in the hippocampal cortex (O'Keefe and Dostrovsky, 1971) together play a key role in the representation of space in the entorhinal-hippocampal system and how it controls both spatial navigation and episodic memory. Multiple scales of entorhinal grid cells can cooperate in a self-organizing map to learn place cell receptive fields (Grossberg and Pilly, 2014; Pilly and Grossberg, 2013). These multiple scales form along a dorsoventral spatial gradient in the entorhinal cortex such that grid cells have increasingly large spatial scales (that is, larger spatial intervals between activations in a hexagonal grid) in the ventral direction. Grid cells with several different spatial scales along the dorsoventral gradient can cooperate to form place cells that can represent spaces much larger than those represented by individual grid cells, indeed place cells capable of representing the lowest common multiple of the grid cell scales that activate them (Gorchetchnikov and Grossberg, 2007; Pilly and Grossberg, 2012)

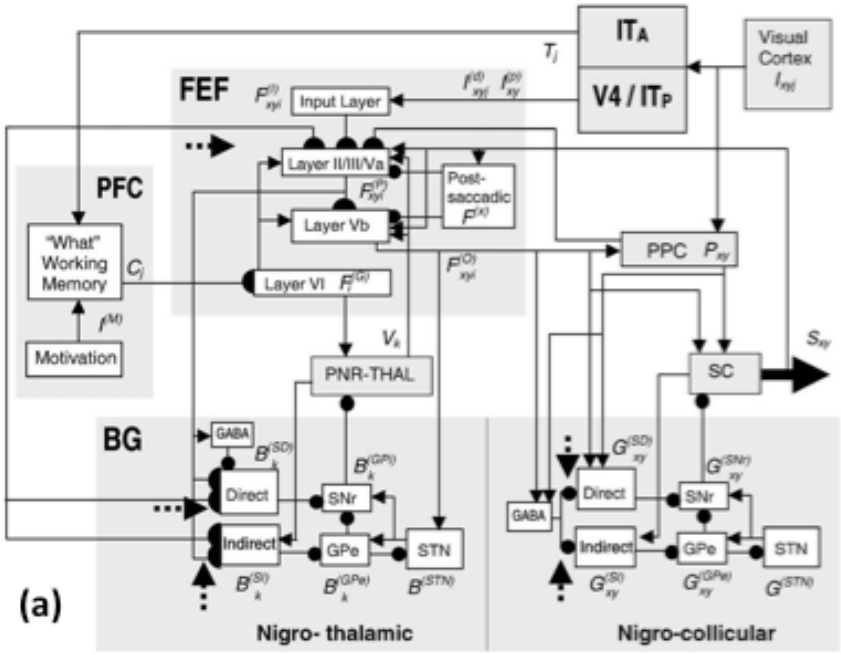
This background indicates the similarity in how the entorhinal–hippocampal system deals with both time and space. In the case of temporal representation by Spectral Timing, a spectrum of small time scales can be combined to represent much longer and behaviorally relevant temporal delays. In the case of spatial representation by grid cells, a spectrum of small grid cell spatial scales can be combined to represent much larger and behaviorally relevant spaces through place cells. This homology has led to the name Spectral Spacing for the mechanism whereby grid cells give rise to place cells.

The Spectral Timing model reflects the part of entorhinal–hippocampal dynamics that is devoted to representing objects and events, and includes lateral entorhinal cortex. The Spectral Spacing model reflects a complementary part of entorhinal–hippocampal dynamics that is devoted to representing spatial representations, and includes medial entorhinal cortex. Both of these processing streams are joined in the hippocampus to support spatial navigation as well as

episodic learning and memory (Eichenbaum and Lipton, 2008).

This proposed homology between spatial and temporal representations is supported by rigorous mathematical modeling and data simulations. Grossberg and Pilly (2012, 2014) have developed the Spectral Spacing model to show that neural mechanisms which allow a dorsoventral gradient of grid cell spatial scales to be learned are formally the same as mechanisms that enable a gradient of temporal scales to control adaptive timing in the Spectral Timing model (Grossberg and Merrill, 1992, 1996; Grossberg and Schmajuk, 1989). Grossberg and Pilly (2012, 2014) were forced into this mechanistic homology in order to be able to quantitatively simulate challenging data about parametric properties of grid cells along the dorsoventral gradient. Thus, it may be that space and time are both in the hippocampus because they both exploit a shared set of computational mechanisms. The phrase ‘‘neural relativity’’ tries to celebrate this predicted homology of spatial and temporal properties of the entorhinal–hippocampal system.

In summary, spectrally timed learning seems to play multiple roles in learning to control motivated attention and action. Its role in the basal ganglia thus seems to illustrate a brain design that has been exploited to control multiple types of adaptively timed behaviors.



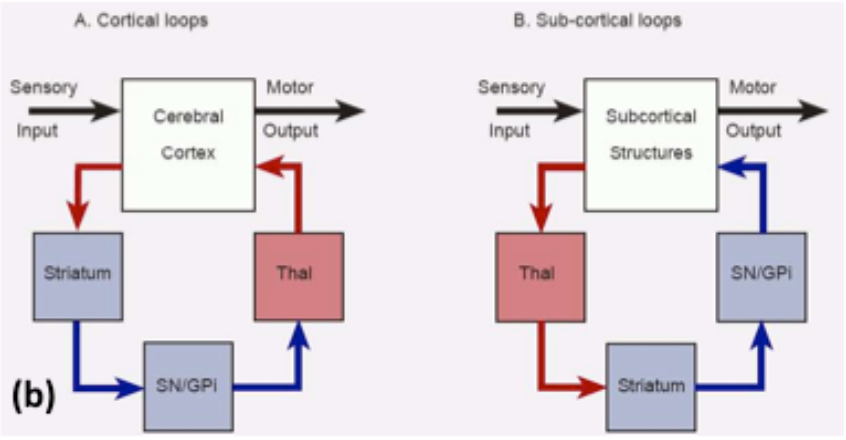


Figure 4. (a) TELOS model macrocircuit showing how layers of the frontal eye fields (FEF) interact with several brain regions, including the basal ganglia (BG), superior colliculus (SC), GABA-ergic striatal interneurons (GABA-SI), external (lateral) segment of the globus pallidus (GPe), internal (medial) segment of the globus pallidus (GPi), anterior inferotemporal cortex (ITa), posterior inferotemporal cortex (ITp), prestriate cortical area V4 (V4), posterior parietal cortex (PPC), prefrontal cortex (PFC), substantia nigra pars reticulata (SNr), subthalamic nucleus (STN), pallidal-(GPi) or nigral-(SNr) receiving zone of the thalamus (e.g., mediodorsal, ventral anterior, and ventral lateral pars oralis nuclei) (PNR-THAL). Separate gray-shaded blocks highlight the major anatomical regions whose roles in planned and reactive saccade generation are treated in the model. Excitatory connections are shown as arrowheads, inhibitory connections as ballheads. Filled semi-circles denote cortico-striatal and cortico-cortical pathways whose connection weights can be changed by learning. Such learning is modulated by reinforcement-related dopaminergic signals (dashed arrows) that are generated from SNc, as described in Figure 2a and the surrounding text. In the FEF block, Roman numerals I–VI label cortical layers; Va and Vb, respectively, are superficial and deep layer V. Further symbols are variable names in the mathematical model. Subscripts *xy* index retinotopic coordinates, whereas subscript *i* denotes an FEF zone wherein a plan is learned and that is gated by an associated BG channel. All variables for FEF activities use the symbol F. Processed visual inputs $I_{xy}^{(p)}$ and $I_{xyj}^{(d)}$ emerging from visual areas including V4 and ITp feed into the model FEF input cells and affect activations $F_{xyi}^{(I)}$. Connections that carry such inputs are predicted to synapse on cells in layer III (and possibly layers II and IV). Visual input also excites the PPC, P_{xy}; and ITa, T_j: A PFC motivational signal $I^{(M)}$ arouses PFC working memory activity C_i, which in turn provides a top-down arousal signal to model FEF layer VI cells, with activities $F_i^{(G)}$. The FEF input cell activities $F_{xyi}^{(I)}$ excite FEF planning cells $F_{xyi}^{(P)}$, which are predicted to reside in layers III/Va (and possibly layer II). Distinct plan layer activities represent alternative potential motor responses to input signals, e.g. a saccade to an eccentric target or to a central fixation point. FEF layer VI activities $F_i^{(G)}$ excite the groups/categories of plans associated with gated cortical zones *i* and associated thalamic zones *k*. The BG decide which plan to execute and send a disinhibitory gating signal that allows thalamic activation *Vk,* which excites FEF layer Vb output cell activities $F_{xyi}^{(O)}$ to execute the plan. The model distinguishes a thalamus-controlling BG pathway (Kemel et al., 1988), whose variables are symbolized by *B*, and a colliculus-controlling pathway, whose variables are symbolized by *G*. Thus, the striatal direct (SD) pathway activities B_k^{SD} and $G_{xy}^{(SD)}$, respectively, inhibit GP_i activities $G_k^{(BP_i)}$ and *SNr* activities $G_{xy}^{(SN_r)}$ which, respectively, inhibit thalamic activities V_k and collicular activities S_{xy}. As further specified in Figure 3a below, if the FEF saccade plan matches the most salient sensory input to the PPC, then the BG disinhibit the SC to open the gate and generate the saccade. However, if there is conflict between the bottom-up input to PPC and the top-down planned saccade from FEF, then the BG-SC gate is held shut by feedforward striatal inhibition (note BG blocks labeled GABA) until the cortical competition resolves. When a plan is chosen, the resulting saccade-related FEF output signal $F_{xyi}^{(O)}$ activates PPC, the STN and the SC (S_{xy}). The SC excites FEF postsaccadic cell activities $F_{xyi}^{(X)}$, which delete the executed FEF plan activity. The STN activation helps prevent premature interruption of plan execution by a subsequent plan or by stimuli engendered by the early part of movement.

[Reprinted with permission from Brown et al. (2004).]. (b) Cortical and subcortical sensorimotor loops through the basal ganglia. A. For cortico-basal ganglia loops, the position of the thalamic relay is on the return arm of the loop. B. In the case of all sub-cortical loops, the position of the thalamic relay is on the input side of the loop. Predominantly excitatory regions and connections are shown in red while inhibitory regions and connections are blue. Tonic basal ganglia inhibition gates shut the activation of targeted cells. Abbreviations: Thal, thalamus; SN/GP, substantia nigra/globus pallidus. [Reprinted with permission from P. Redgrave, Basal ganglia, Scholarpedia, 2(6):1825].

4. Associative and reinforcement learning of eye movements

4.1. Eye movements as a model system for understanding movement and cognition

The circuit in Figure 2a generates Now Print reinforcement learning signals that regulate associative learning in multiple brain regions. The TELOS model (Figure 4a; Brown, Bullock, and Grossberg, 2004) was developed to illustrate how this widespread Now Print signal can be used to learn several different types of saccadic eye movement behaviors. Eye movements were chosen as a good explanatory target for this modeling task because, first, behavioral and neurophysiological data are abundant for this kind of behavior and, second, eye movements are an excellent brain system for understanding how sensory modalities, like vision and audition, control motor actions. In addition, it is known that the parietal attention circuits that are used to command eye movement target positions are also used to command arm movement target positions (Andersen et al., 1997; Deubel and Schneider, 1996). Thus such a model can be adapted to control the targeting of arm movements as well.

This task is facilitated by the availability of detailed neural models both of eye movement control (e.g., Gancarz and Grossberg, 1998, 1999; Grossberg and Kuperstein, 1989; Grossberg et al., 1997; Grossberg, Srihasam, and Bullock, 2012; Srihasam, Grossberg, and Bullock, 2009) and arm movement control (e.g., Bullock, Cisek, and Grossberg, 1998; Bullock and Grossberg, 1988, 1991; Contreras-Vidal, Grossberg, and Bullock, 1997; Grossberg and Paine, 2000). Finally, some eye movements can be made to remembered positions in space, and sequences of planned eye movements can be learned; see Section 8. Thus this system also provides a useful window into higher-order cognitive brain processes, and how they interact with sensory and motor processes.

4.2. How does the brain "know before it knows"? Gating reactive and planned behaviors

The TELOS model proposes detailed mechanistic solutions to several basic problems in movement control: How does the brain learn to balance between reactive and planned movements? How do recognition and action representations in the brain cooperate to launch movements toward valued goal objects: How does the brain learn to switch among different movement plans as it is exposed to different combinations of scenic cues and timing constraints?

Rapid reactive movements are needed to ensure survival in response to unexpected dangers. Planned movements, that involve focused attention, often take longer to select and release. How does the brain prevent reactive movements from being triggered prematurely in situations where a more slowly occurring planned movement would be more adaptive? If this could not be achieved, then reactive movements could always preempt the occurrence of more appropriate context-selective planned movements, and indeed could prevent them from ever being learned.

This requirement leads to a second critical role of the basal ganglia, in addition to its role in selectively responding to unexpected rewards in SNc and broadcasting Now Print signals across

the brain to learn the contingencies that have caused the unexpected event. This critical role concerns how the basal ganglia select context-appropriate movement plans and actions using *movement gates*. Such a movement gate can, for example, prevent a reactive movement from being launched until the planned movement can effectively compete with it.

All movement gates that are controlled by the basal ganglia tonically inhibit movement commands (Figure 4b). When a specific gate is inhibited, the cells that control the corresponding movement command can be activated. Thus, the brain needs to keep each movement gate active until it can be inhibited to release the corresponding plan or action. The successive inhibitory connections illustrated in Figure 4b accomplish this. The substantia nigra pars reticulata (SNr) regulates this sort of gating process. In particular, outputs from the basal ganglia provide GABA-ergic inhibitory gating of their target structures. In the primate saccadic circuit, cells in the SNr tonically inhibit the superior colliculus (SC), but pause briefly to allow the SC to generate a saccade to a selected target location (Hikosaka and Wurtz, 1983, 1989). Lesions in this system can release a 'visual grasp reflex' (Guitton, Buchtel, and Douglas, 1985); namely, impulsive orienting to any visually salient object. Ancient vertebrate species, such as frogs, already had basal ganglia (Marin et al., 1998). Indeed, lesions of the basal ganglia projection to the optic tectum, the SC homolog in frogs, impair the frog's ability to orient selectively (Ewert, Schurg-Pfeiffer, and Schwippert, 1996).

These gates solve he following challenging problem: When a sensory cue occurs, such as an extrafoveal flashing light on the retina, the fastest response would be an orienting response to look at it. For this to happen, the cue needs to open the appropriate basal ganglia gate to enable the reactive movement to occur. However, if the cue is a discriminative cue to do a different action, especially an action that requires rapid execution, then the reactive response is not adaptive. However, it may take longer to fully process the cue to determine its adaptive conditional response than it would to activate the reactive response. How does the brain know that a plan is being elaborated, even before it is chosen, so that the reactive gate can be kept shut? How does the brain "know before it knows"? In particular, how does the brain prevent a reactive movement command from opening its gate before a planned movement command is ready to open a different gate, yet also allow a reactive movement command to open its gate as rapidly as possible when no planned movement command is being selected?

The TELOS model (Figure 4a) was developed to explain and simulate how the brain may achieve this sort of balance between reactive and planned movements as it controls the learning and performance of saccadic eye movements. The acronym TELOS (TElencephalic Laminar Objective Selector) is inspired by the ancient Greek word telos for goal, end, or completion of a plan.

4.3. Frontal–parietal resonance codes plan choice and leads to planned gate opening

According to TELOS, the brain ''knows before it knows'' in the following way: The model predicts how the distribution of excitation and inhibition that converges on the basal ganglia when a plan is being elaborated keeps the reactive gate closed (Figure 5a). Before the appropriate movement plan is selected, there can be multiple bids converging on the basal ganglia to open one or another movement gate. It is this competition between different reactive and planned representations that keeps the reactive movement gate closed. When a movement plan is finally chosen, there is agreement between cells in the frontal eye fields (FEF) and the parietal cortex representations of target position (Figure 5aD). This agreement changes the excitatory–inhibitory balance and enables excitatory feedback to become activated between FEF and the parietal cortex.

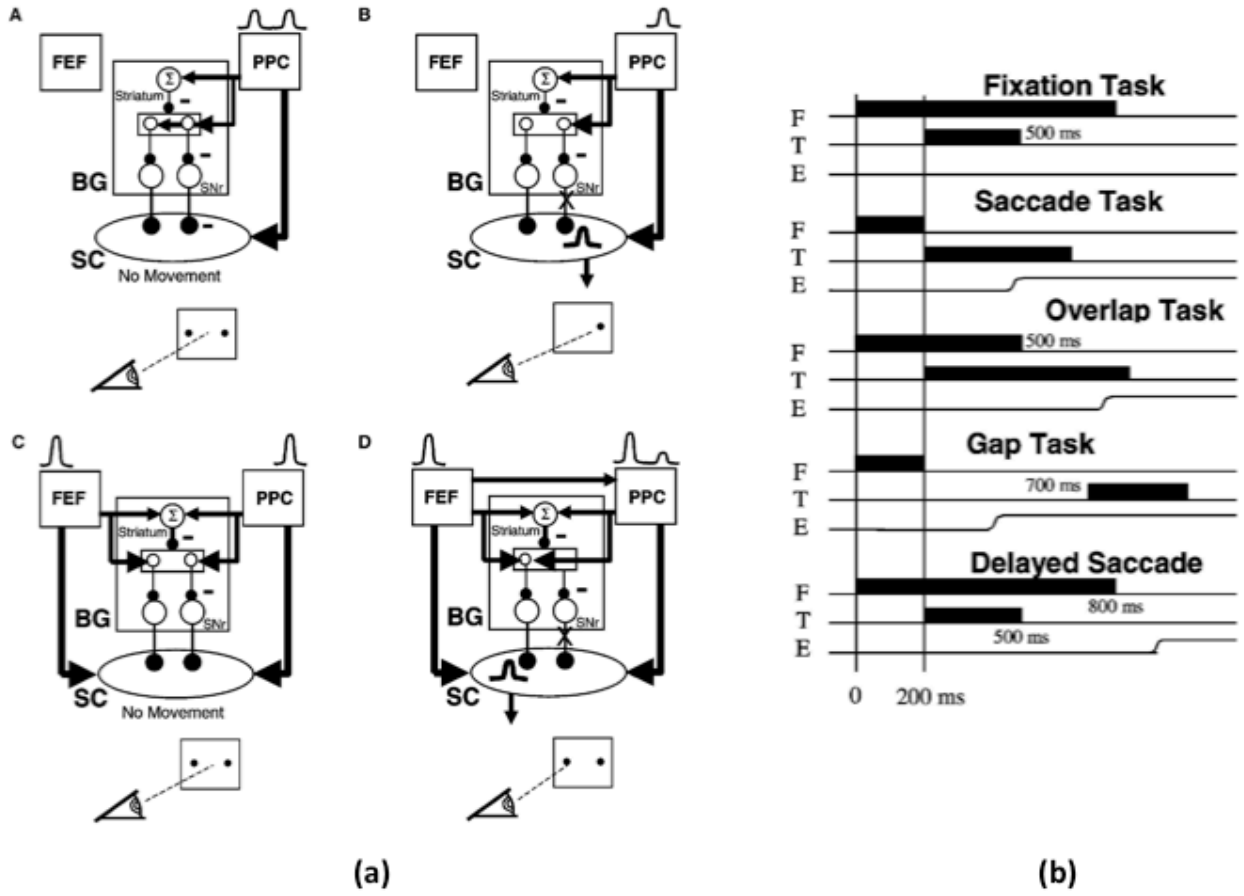


Figure 5. (a) Cortical and striatal processes in location-specific gating of the superior colliculus (SC) by the basal ganglia (BG), leading to a resonance between the frontal eye fields (FEF) and the posterior parietal cortex (PPC) when a target location is selected. A. When multiple stimuli exist as potential saccade goals, the corresponding PPC representations specifically excite striatal spiny projection neurons (SPNs; shown in the rectangle within the BG rectangle) and non-specifically excite feedforward inhibitory interneurons (labeled with a capital sigma) via corticostriatal projections. If more than one saccade plan is active, then striatal feedforward inhibition from all active plans prevents any one plan from activating its corresponding striatal SPNs to open the BG gate. This is because the pooled inhibitory input to each SPN can overwhelm the specific excitatory input. Therefore the SC is not released from inhibition from the SNr, and movement is prevented while conflicting cortical plan activities remain unresolved. B. Targets compete in the PPC via inhibitory interactions. When competition resolves so that the movement plan is unambiguous, the PPC's excitatory input to striatal SPNs eventually exceeds striatal feedforward inhibition, which wanes as competing plans lose activation and stop convergent excitation of striatal inhibitory interneurons. The output signals from the winning SPN inhibit the SNr, thereby opening its normally closed gate, which disinhibits part of the SC map. C. If the FEF plans a saccade goal that differs from the location of a strong visual stimulus, the competing frontal and parietal activities collectively drive striatal feedforward inhibition to keep the BG gate shut until the conflict resolves. D. As the frontal cortex imposes its saccade goal on the parietal cortex, the competition between saccade goals resolves, enabling a FEF-PPC

resonance to develop, and allowing the selected BG gate to open, thereby enabling the chosen saccadic command to be released. Note: The absence of an icon for FEF activity in B. indicates not that FEF would be inactive in case B., but only that FEF contains no plan contrary to PPC in case B. [Reprinted with permission from Brown et al. (2004).]. (b) Oculomotor tasks of Hikosaka et al. (1989). Black bars indicate intervals of visual stimulus presentations and the trace labeled E gives the horizontal component of eye position (line of gaze). In the *fixation task*, the subject must maintain gaze on the fixation point, F, despite a brief display of a distracter target, T, at a different locus. In the *saccade task*, the subject must make a pro-saccade from the fixation point to the target, which appears at a different locus, just as the fixation point shuts off. In the *overlap task* (similar to a GO/NOGO task), the target and the fixation point are displayed in overlapping intervals. A pro-saccade to the target is rewarded only if generated after the fixation point shuts off. The *gap task* imposes a delay between the offset of the fixation point and the onset of the target. The gap task target appears at a consistent location across trials, and the subject learns to make an anticipatory pro-saccade to the target location during the gap between fixation light offset and target onset. The *delay task* requires the subject to remember the location of a briefly-flashed target and later foveate it. [Adapted with permission from Hikosaka et al., 1989, p. 781]. The TELOS model in Figure 4a learned and performed all these tasks.

This mutually reinforcing excitatory feedback develops into a synchronous resonance (Grossberg, 2012) that is predicted to signal consistency between a finally selected movement plan and the parietal representation of the corresponding attended target location. When this happens, the balance of excitation and inhibition enables the appropriate basal ganglia movement gate to open and release the context-appropriate action. Buschman and Miller (2007) have reported such prefrontal–parietal resonances during movement control, and Pasupathy and Miller (2004) have reported different time courses of activity in the prefrontal cortex and basal ganglia that are consistent with how basal ganglia-mediated gating of prefrontal cortical plans may be learned.

4.4. Spatially-invariant object categories control spatially directed actions

In further support of this proposal, TELOS model simulations emulate how SNc dopaminergic reward and non-reward signals guide monkeys to learn and perform saccadic eye movements in fixation, single saccade, overlap, gap, and delay (memory-guided) saccade tasks (Figure 5b). After learning occurs, model cell activation dynamics quantitatively simulate, and predict functional roles for, the dynamics of seventeen types of identified neurons during performance of these tasks.

Movements towards valued goal objects cannot be made until the goal objects are recognized and movement directions specified. To achieve efficient object recognition, the What cortical processing stream builds object representations that become increasingly invariant under changes in object view, sizes, and positions at higher cortical areas. In particular, these representations become significantly ‘positionally invariant’, or independent of the retinotopic position or size of the object (Bar et al., 2001; Sigala and Logothetis, 2002; Tanaka, Saito, Fukada, and Moriya, 1991). Indeed, recent neural models have clarified how such invariant object categories may be learned and recognized in the anterior regions of the inferotemporal cortex (ITa) as a result of suitable interactions between the What and Where cortical streams (Cao, Grossberg, and Markowitz, 2011; Fazl, Grossberg, and Mingolla, 2009; Foley, Grossberg, and Mingolla, 2012; Grossberg, 2009; Grossberg, Markowitz, and Cao, 2011).

In addition to overcoming the crippling combinatorial explosion of memory and search requirements that would have occurred if every variation in an object's appearance forced learning of a different recognition code, such invariant representations are sufficiently compact to facilitate their learned association with reinforcement and motivational mechanisms, such as those supported by the amygdala (Aggleton, 1993; Barbas, 1995; Baxter et al., 2000; DeDoux, 1993; Schoenbaum et al., 2003). Positive feedback between the invariant representations and the amygdala, as part of an inferotemporal-amygdala-orbitofrontal resonance (see Section 7), enable the brain to focus motivated attention upon the representations of valued goal objects (Chang, Grossberg, and Cao, 2014; Grossberg, 1972a, 1972b, 1975, 1982); see Section 7.

Nothwithstanding the possible pleasures of Platonically contemplating a valued goal object, such contemplation is insufficient to ensure survival in the forest primeval, let alone in a modern society. Indeed, even to discover and learn what objects may have value, it is also necessary to also be able to physically engage them by moving and reaching towards them. Given that the recognition codes that are attentively amplified by motivational signals are often independent of position, the brain then faces the challenging problem of computing how to move to the position of an object after it is attended and recognized. The invariant object categories are learned within the What cortical stream. The Where cortical processing stream elaborates the representations of object spatial position and direction that are needed to compute motor commands.

The TELOS model proposes how interactions across the What and Where processing streams overcome their computationally complementary informational deficiencies (Grossberg, 2000) to generate movements towards recognized objects. In the model, the pathways from ITa and ITp to FEF (Figure 4a) mediate the associative linkage between invariant object categories and positionally-sensitive motor representations. Indeed, cells in ITp are sensitive to simple features falling within particular retinotopic loci (Kobatake and Tanaka, 1994; Tanaka et al., 1991; Komatsu and Ideura, 1993), whereas the position-invariant cells in ITa are sensitive to objects regardless of their specific retinotopic locus (Gross et al., 1985; Tanaka et al., 1991). This linkage, combined with the inferotemporal-amygdala-orbitofrontal resonance that focuses attention upon valued goal objects, has been used to propose a solution to the Where's Waldo problem, or how to search for a valued goal object in a cluttered scene (Chang, Grossberg, and Cao, 2014).

Data about the anatomical projections from feature-sensitive areas such as ITp, when combined with physiological evidence on the emergence of feature selectivity in FEF neurons when features are consistently rewarded (Bichot, Schall, and Thompson, 1996), support this anatomical linkage, as well as the model hypothesis that dopaminergic Now Print signals from SNc (Figures 2a and 4a) regulate reward-guided learning mediated by weight changes in the IT to FEF pathways. In particular, reward-related dopaminergic signals modulate learning in both the striatum of the basal ganglia and the frontal cortex (Gaspar, Bloch, and Le Moine, 1995; Schultz, 1998).

The trained system allows or prevents movements, according to their appropriateness (Bullock and Grossberg, 1991; Crosson, 1985; Hikosaka and Wurtz, 1983; Mink, 1996; Mink and Thach, 1993; Redgrave, Prescott, and Gurney, 1999). Indeed, it is not enough to recognize and move towards an object. An animal or human needs to know when to move towards or away from an object and when not to do so, depending on reward contingencies. In addition, when confronted with the same scene, an animal may respond differently depending on its changing needs, such as eating food if hungry, or drinking water if thirsty. The model explains how the

brain learns and remembers many plans that involve different sets of discriminative and scheduling constraints, and how it switches among them as needed. These design and circuit details go beyond the scope of the current review.

5. Basal ganglia gating of variable-speed arm movements: Synergy, synchrony, and speed

5.1. VITE model of arm trajectory formation

The basal ganglia control the gating of all phasic movements, including both eye movements and arm movements. Arm movements, unlike eye movements, can be made at variable speeds that are under volitional basal ganglia control. Arm movements realize the Three S's of Movement Control; namely, Synergy, Synchrony, and Speed: Specific combinations of muscle groups can be combined into a movement *synergy*, whereby the bound muscles can move *synchronously*, in equal time, to a target position at variable *speed*s. The simplest model of arm movement trajectory formation with these properties is the Vector Integration to Endpoint, or VITE, model (Figure 6a; Bullock and Grossberg (1988). To make such a movement, a representation of where the arm is now (its *present position vector*) is subtracted from a representation of where we want the arm to move (its *target position vector*), thereby computing a *difference vector* that represents the direction and distance of movement needed to attain the target. After moving to the target, the target and present positions agree, so the difference vector is zero. In other words, this sort of matching is inhibitory.

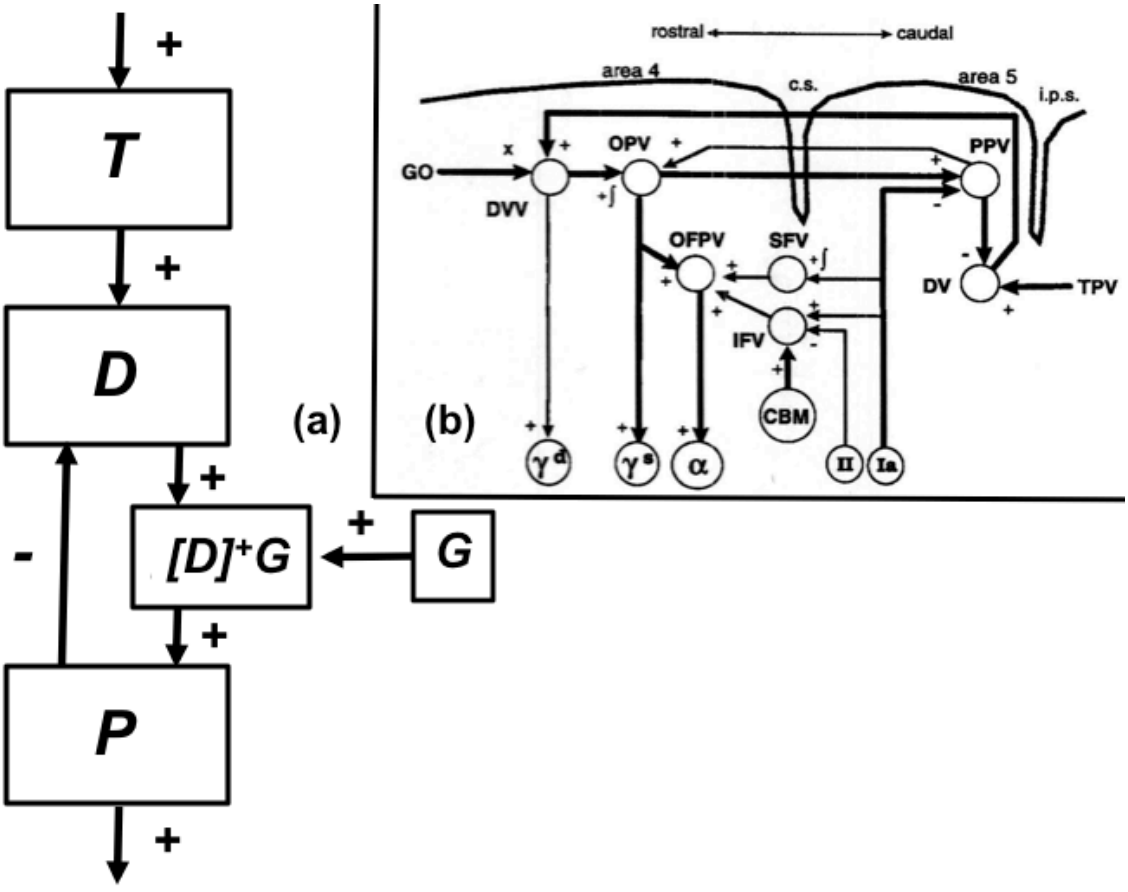


Figure 6. (a) Vector Integration To Endpoint circuit (Bullock and Grossberg, 1988) for control of movement trajectories. *T* is the target position vector, *P* the outflow present position vector, *D*

the difference vector, and *G* the volitional GO signal that multiplies, or gates, *D*. See text for details. (b) Cortical circuit mode of VITE interactions that can compensate for obstacles and variable loads on the arm during trajectory formation. Thick connections represent the kinematic feedback control aspect of the model, with thin connections representing additional compensatory circuitry. GO = scaleable basal ganglia gating signal; DVV = desired velocity vector; OPV = outflow position vector; OFPV outflow force-plus-position vector; SFV = static force vector; IN = inertial force vector; CBM = assumed cerebello-cortical input to the IFVstage; PPV = perceived position vector; DV = differencevector; TPV = target position vector; γ^d = dynamic gamma motoneuron; γ^s = static gamma motoneuron; α = alpha motoneuron; la = type la afferent fiber; II = type II afferent fiber (position error feedback); c.s. = central sulcus; i.p.s. = intraparietal sulcus. The symbol + represents excitation, - represents inhibition, x represents multiplicative basal ganglia gating, and $+\int$ represents integration. See Bullock, Cisek, and Grossberg (1998) for details. [Reprinted with permission from Bullock, Cisek, and Grossberg (1998).]

5.2. Variable-speed arm movements due to variable-size GO signals

To better understand how this works, note that the difference vector is volitionally gated, or multiplied, by a basal ganglia GO signal (Figure 6a) that determines when and how fast the movement will occur (Bullock and Grossberg, 1988; Bullock, Cisek, and Grossberg, 1998). When both the GO signal and the difference vector are positive, their product is integrated by the present position vector, causing the present position vector to approach the target position vector. When both vectors are the same, the movement stops.

The cells with non-zero activities in the target position vector control the muscle groups that are included in the currently active synergy. A zero GO signal does not move the arm at all, whereas a progressively larger GO signal enables it to move at increasingly fast speeds. It is because the GO signal multiples the difference vector that all muscles within the synergy contract synchronously and reach the position represented by the target position vector at the same time.

5.3. Motor-equivalent reaching and arm movements given perturbations and obstacles

The VITE model has been extended in several directions. One extension is to the Direction-to-Rotation Effector Control Transform, or DIRECT, model of motor-equivalent reaching (Bullock, Grossberg, and Guenther, 1993), which clarifies how accurate, single-synergy, reaches can be made on the first try, under visual guidance, with a tool or with clamped joints. DIRECT suggests how learning reaching coordinates in space using a Piagetian circular reaction automatically enables the ability, or affordance, to touch a target in space with a tool. A variant of DIRECT, called the Directions-Into-Velocities-of-Articulators, or DIVA, model, has been used to simulate data about motor-equivalent speech articulator movements during speech production (Guenther, 1995; Guenther, Ghosh, and Tourville, 2006). Another VITE extension (Figure 6b) describes the cortical circuits that enable arm movements to be made in the presence of unexpected perturbations and obstacles (Bullock, Cisek, and Grossberg, 1998). This elaboration enables the quantitative simulation of neurophysiological data about the dynamics of multiple identified cell types in cortical areas 4 and 5.

Models like VITE focuses primarily on Platonic aspects of movement planning and trajectory formation, although for VITE to cope with unexpected perturbations and obstacles, feedback to the cortex from subcortical processes, such as alpha and gamma motoneurons, is also

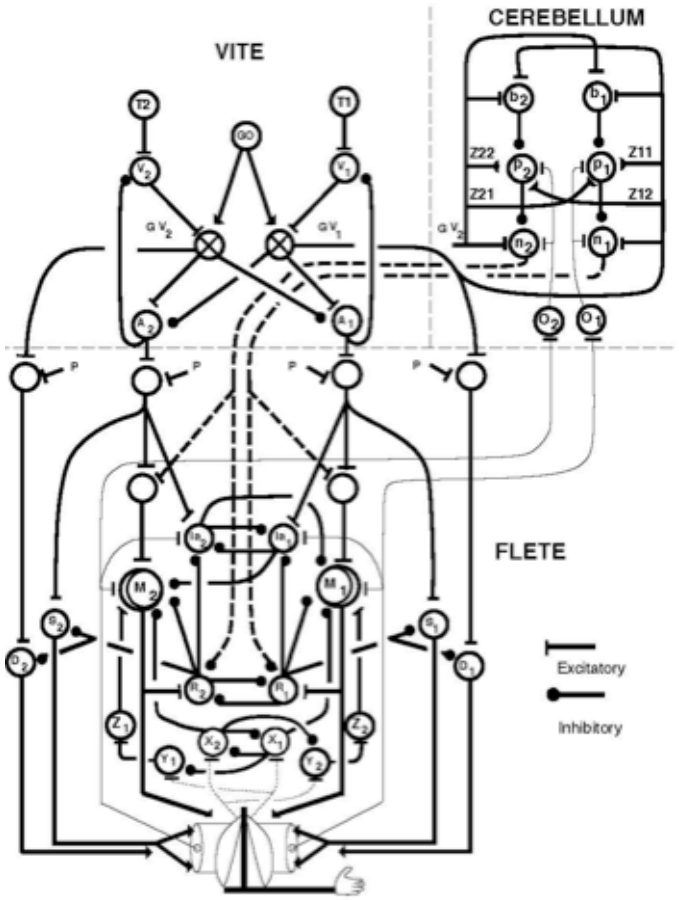


Figure 7. Model circuit for neuromuscular control system. Upper-left part: The VITE model for variable-speech synergy formation and trajectory generation. Lower part: The FLETE model of the opponently organized spino-muscular system. Dotted lines show feedback pathways from sensors embedded in muscles. The two lateral feedback pathways arise in spindle organs sensitive to muscle stretch and its first derivative. The two medial feedback pathways arise in Golgi tendon organs sensitive to muscle force. Signals T_1 and T_2 specify the target position vector; signals A_1 and A_2 specify the desired position vector; signals V_1 and V_2 specify the difference vector; signal $GO = G$ is the basal ganglia GO signal that controls movement selection and speed; signals GV_1 and GV_2 specify the desired velocity vector; and signal P scales the level of coactivation. Upper-right part: Feedforward cerebellar model computes transient inverse-dynamic signals that excite motoneurons and modulate the gain in spinal circuits. Key: b = basket cells; p = Purkinje cells; n = nucleus interpositus cells; O = inferior olive; CF = climbing fibers from inferior olive to Purkinje cells; and z = long-term memory weights. Paths ending in filled dots are inhibitory; all others are excitatory. [Reprinted with permission from Contreras-Vidal, Grossberg, and Bullock, 1997.]

modeled (Figure 6b). The Factorization of Length and Tension, or FLETE, model (Figure 7) complements VITE by using cerebellar and spinal circuits to compensate for the forces and tensions that are needed to accurately move real arms along commanded trajectories. These cerebellar and spinal circuits of FLETE interact with the thalamo-cortico-basal ganglia circuits of VITE, and with opponent muscle groups in the arm, in order to plan and execute arm movement

trajectories using multi-joint arms (Bullock and Grossberg, 1991; Contreras-Vidal, Grossberg, and Bullock, 1997).

In all these arm control models, the gating effects of the basal ganglia on movement are represented with a simple GO signal. A similar simplification has been sufficient to explain an important gating role of the basal ganglia on speech perception.

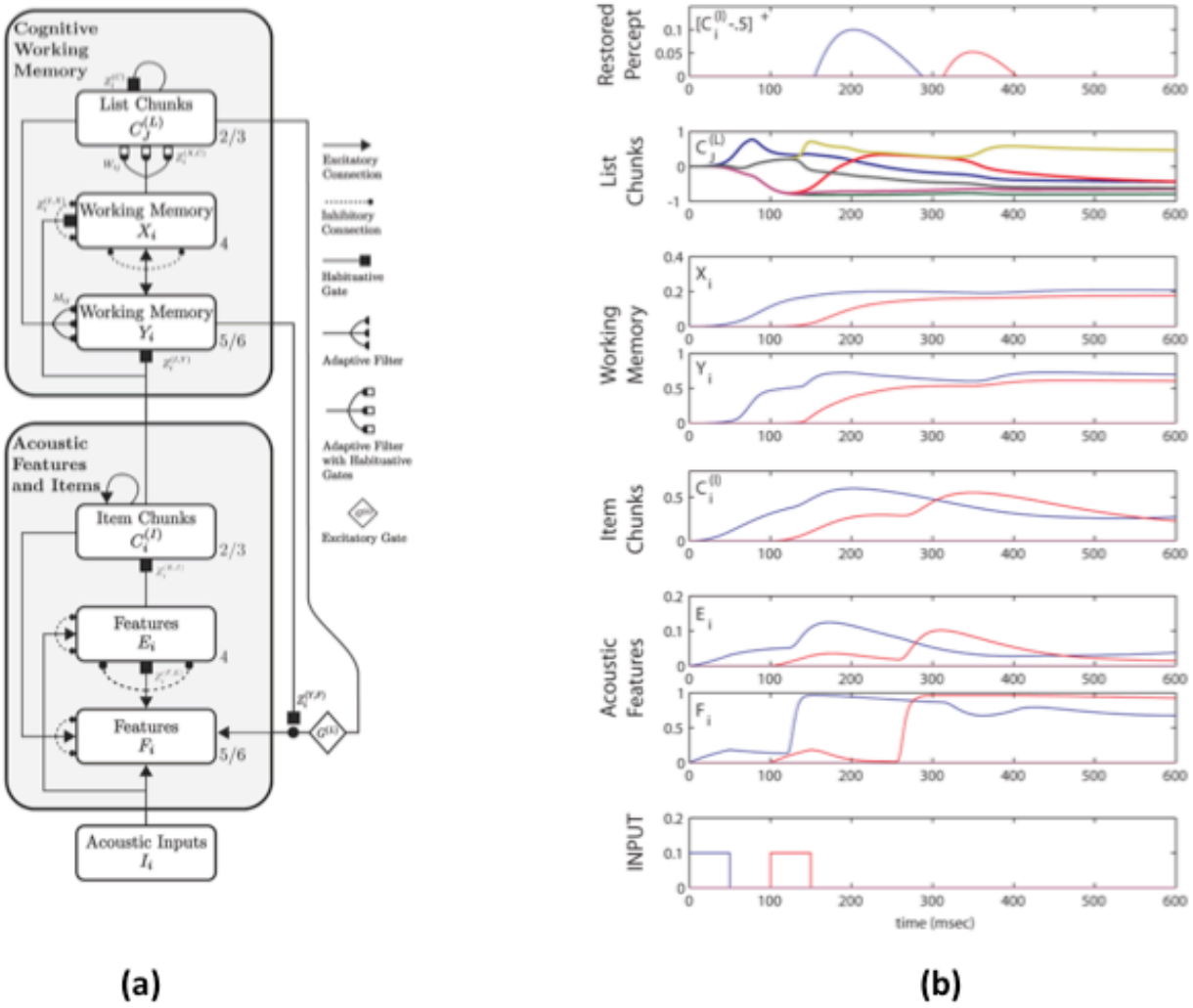


Figure 8. (a) Macrocircuit of the cARTWORD model. This macrocircuit shows a hierarchy of cortical levels that help to explain several of the processes that enable speech and language perception. Each level is organized into laminar cortical circuits, wherein deep layers (6 and 4) are responsible for processing and storing inputs, and superficial layers (2/3) are proposed to group distributed patterns across these deeper layers into unitized, or chunked, representations. The lowest level is responsible for processing acoustic features (cell activities F_i and E_i) and items (cell activities $C_i^{(I)}$), whereas the higher level is responsible for storing of sequences of acoustic items in working memory (activities Y_i and X_i), and representing these stored sequences of these items as unitized, context-sensitive representations by list chunks

(activities $C_J^{(L)}$). The list chunks are selected and stored in short-term memory by a masking field, which is a multiple-scale, self-similar, recurrent on-center off-surround network. The top-town pathway from the list chunks in cognitive working memory to the acoustic feature level schematizes the role of the basal ganglia. When a list chunk or chunks gets sufficiently active (and is thus most predictive of the current working memory context), it generates an output signal that acts like an excitatory gating signal $G^{(L)}$, which enables the top-down modulatory feedback from the cognitive working memory to amplify the attended featural patterns and thereby trigger a system-wide resonance between all the processing levels. This excitatory gating signal is a simplified representation of the kind of disinhibitory process whereby the basal ganglia enable cortico-cortical processing loops to resonate, as in Figure 4a. (b) Network dynamics in response to a sequence of three inputs presented "1- -3" (bottom row, with '1' shown in blue and '3' in red), with a 50 msec silence duration interval. See text for details. [Reprinted with permission from Grossberg and Kazerounian (2011).]

6. Basal ganglia gating of speech perception

6.1. cARTWORD model, resonant wave, conscious speech, and phonemic restoration

Interactions between the frontal cortices and the basal ganglia arise across several different modalities of intelligence, including more cognitive processes such as the control of consciously heard speech and language. The conscious ARTWORD (cARTWORD) model of Grossberg and Kazerounian (2011) illustrates how such gating helps to control the consciously heard temporal order of speech that is influenced by contextual cues that may occur after the heard formants (Figure 8a). cARTWORD describes how the laminar circuits within a hierarchy of cortical processing stages may interact to generate such a conscious speech percept. Earlier modeling work showed how variations of this circuit design may be used to explain and predict challenging psychophysical and neurobiological data about 3D vision, figure-ground perception, and visual object recognition (e.g., Cao and Grossberg, 2005; Fang and Grossberg, 2009; Grossberg and Versace, 2008; Grossberg and Yazdanbakhsh, 2005), and about cognitive working memory and list chunking (Grossberg and Pearson, 2008; Silver et al., 2011); see Section 8. This unity of processing clarifies how variations of a shared laminar neocortical design across modalities enable the brain to compute multiple types of biological intelligence, and illustrates the paradigm of Laminar Computing.

cARTWORD further develops the hypothesis that conscious speech percepts are emergent properties that arise from resonant states of the brain (Grossberg, 1978, 1986, 2003; Grossberg, Boardman, and Cohen, 1997; Grossberg and Myers, 2000). Such a resonance develops when bottom-up signals that are activated by environmental events interact with top-down expectations, or prototypes, that have been learned from prior experiences. The top-down expectations carry out a matching process that selects those combinations of bottom-up features that are consistent with the learned prototype while inhibiting those that are not. In this way, an attentional focus concentrates processing on those feature clusters that are deemed important on the basis of past experience. The attended feature clusters, in turn, reactivate the cycle of bottom-up and top-down signal exchange. This reciprocal exchange of signals equilibrates in a resonant state that binds the attended features together into a coherent brain state. Such resonant states, rather than the activations that are due to bottom-up processing alone, are proposed to be the brain events that regulate fast and stable learning of speech and language, and that give rise to conscious speech and language percepts. Indeed, I have predicted that "conscious speech is a resonant wave" and that "silence is a temporal discontinuity in the rate at which the resonance

develops" (Grossberg, 2003).

The feedback dynamics of these resonances enable the brain to incorporate both past and future contextual information, often acting over hundreds of milliseconds, into the processing of speech and language, without destroying the correct temporal order of consciously heard words. Such contextual disambiguation is necessary to understand speech and language during the multi-speaker noisy environments that are characteristic of real-life speech and language experiences. The fact that conscious speech percepts are influenced by cues occurring hundreds of milliseconds before or after the heard formants challenges classical concepts about the functional units of speech perception and recognition. In order for such contextual influences to have an effect on speech perception, sequences of speech items are temporarily stored in a working memory.

A classical example of a percept in which future context disambiguates consciously heard speech is *phonemic restoration* (Samuel, 1981a, 1981b; Warren, 1970, 1984; Warren and Obusek, 1971; Warren and Sherman, 1974; Warren and Warren, 1970). cARTWORD explains and computationally simulates how a hierarchy of laminar cortical processing stages, gated by the basal ganglia, can explain this and related speech percepts wherein conscious percepts depend upon contextual information (Figures 8a).

The following example of phonemic restoration illustrates the conceptual issues. Suppose broadband noise replaces the phonemes /v/ and /b/ in the words delivery and deliberation, respectively. Despite the initially ambiguous initial portion of these words ('deli-'), if the broadband noise is immediately followed by 'ery' or 'eration', listeners hear the /v/ or /b/ as being fully intact and present in the signal. Such experiences show that top-down lexical influences contribute to the formation of conscious speech percepts.

Several challenging conceptual issues are raised by this and related examples. First, why is the noise in "deli-noise-[ery/eration]" not heard before the last portion of the word is even presented? This may be explained by the fact that, if the resonance has not developed fully before the last portion of the word is presented, then this portion can influence the expectations that determine the conscious percept.

Second, how does the expectation convert the noise in "deli-noise-[ery/eration]" into a percept of [/v/-/b/]? This occurs due to the top-down matching process that selects expected feature clusters for attentive processing while suppressing unexpected ones. In the "deli-noise-[ery/eration]" example, spectral components of the noise are suppressed that are not part of the expected consonant sound.

Attentive selection during phonemic restoration and other speech and language percepts is not merely a process of symbolic inference. Indeed, it directly influences phonetic percepts. For example, if a reduced set of spectral components is used in the noise, then a correspondingly degraded consonant sound is heard (Samuel, 1981a, 1981b).

Third, how do future events influence past events without smearing over all the events that intervene? In particular, if the /v/ or /b/ in "delivery/deliberation" is replaced by silence, how is the silence perceived as silence despite the fact the disambiguating cue would have influenced the percept were these phonemes to be replaced by noise? Here again the nature of the top-down matching process is paramount. Top-down attentive matching process is *modulatory*; it can prime, sensitize, and select feature components that are consistent with its prototype, but it cannot create something out of nothing.

Fourth, how can sharp word boundaries be perceived even if the sound spectrum that represents the words exhibits no silent intervals between them? cARTWORD illustrates the

hypothesis (see the review in Grossberg, 2003) that silence will be heard between words whenever there is a temporal break between the resonances that represent the individual words. In other words, just as conscious speech is a resonant wave, silence is a discontinuity in the rate at which this resonant wave evolves.

The top-down attentive matching that selects context-appropriate sounds is controlled by recognition categories that are sensitive to particular combinations of sequences of speech items through time. These categories are also called *list chunks* (see Grossberg and Pearson (2008) for a review). List chunks are selected and stored in short-term memory by a multiple-scale, self-similar, on-center off-surround network that is called a *masking field* (Cohen and Grossberg, 1986, 1987; Grossberg, 1978a). A masking field can select the list chunks that are mostly strongly supported by the sequence of items currently stored in the working memory. The multiple spatial scales that are represented in a masking field enable list chunks to be selected that are sensitive to item sequences of different length. The self-similar property of the masking field enables list chunks that represent longer sequences to inhibit list chunks that represent shorter sequences. This property also helps to explain data such as the word superiority effect and the Magical Number Seven of George Miller (Cohen and Grossberg, 1986; Grossberg, 1986; Grossberg and Pearson, 2008).

These facts lead to the fifth issue: How does the brain know how to wait until the most active, and thus predictive, combination of list chunks is chosen to release the top-down attentive signals that will select and resonate with the syllable, word, or sentence sounds that will be consciously heard? Here is where the basal ganglia play a critical role in the model. In particular, basal ganglia gating enables future phonetic contexts to have enough time to help choose the list chunks that will become sufficiently active to open a basal ganglia gate. Moreover, gate opening, as illustrated in Figure 8a, enables the entire hierarchy of processing stages—acoustic features, acoustic items, stored sequences of these items in working memory, and list chunks—to resonate during a conscious speech percept. Including representations can include the distinctions of acoustic features in the emerging resonance enables conscious percepts to include acoustic distinctions that are used to understand language.

6.2. Adaptive Resonance Theory, language learning, and the stability-plasticity dilemma

The name cARTWORD derives from the fact that this model is an example of Adaptive Resonance Theory, or ART. ART has predicted that the attentive resonance and matching processes that support phonemic restoration are necessary ones to enable speech and language to be learned quickly without forcing the non-selective, or catastrophic, forgetting of previously learned memories (Grossberg, 1978, 1986, 2003). Indeed, the need to solve this *stability-plasticity dilemma* occurs in many perceptual and cognitive processes.

It has elsewhere been mathematically proved that the properties of this top-down attentive matching process, called the ART Matching Rule, are necessary to enable fast learning without catastrophic forgetting (Carpenter and Grossberg, 1987). The ART Matching Rule proposes how a top-down, modulatory on-center, off-surround network controls the read-out of learned top-down expectations and attentional focusing, as well as the dynamical stabilization of both bottom-up and top-down learned memories (see Section 9). Because of the role of the off-surround, or competition, in attentional focusing, this process is sometimes described as "biased competition" (e.g., Desimone, 1998; Kastner and Ungerleider, 2001).

Due to the need to solve the stability-plasticity dilemma in all perceptual and cognitive processes, the ART Matching Rule for top-down attentional matching seems to occur in other perceptual modalities, notably vision (Bhatt, Carpenter, and Grossberg, 2007; Carpenter and

Grossberg, 1987; Gove, Grossberg, and Mingolla, 1995). Reviews of supportive perceptual and neurobiological data, ART models that describe the mathematical form of the ART Matching Rule, and the predicted link between attentive matching, resonance and learning, can be found in Grossberg (2012), Grossberg and Versace (2008), and Raizada and Grossberg (2003).

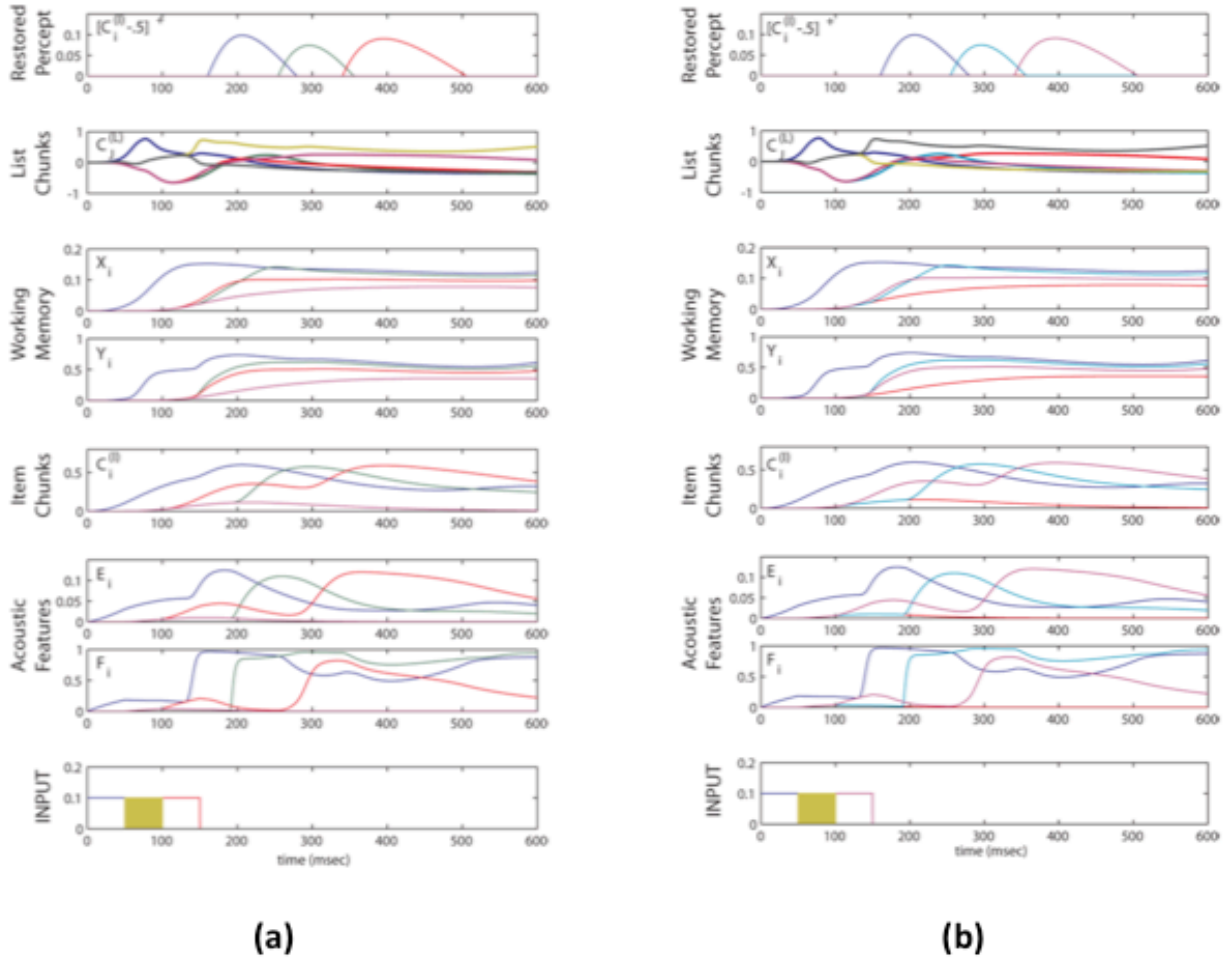


Figure 9. (a) Network dynamics in response to a sequence of three inputs presented **"**1- * -3" where '*****' denotes noise as presented for 50 msec in place of any phoneme ('1' is shown in blue, '*' is shown as a filled yellow pulse, and '3' is shown in red). See text for details of how the excised item '2' is restored. (b) Network dynamics in response to the sequence **"**1- * -5**"**, where ***** again denotes noise ('1' is shown in blue, '*' is shown in yellow, and '5' is shown in purple). See text for details of how the excised item '4' is restored. [Reprinted with permission from Grossberg and Kazerounian (2011).]

6.3. Simulations of phonemic restoration

Figures 8b and 9 illustrate how cARTWORD simulates percepts of phonemic restoration in the consciously heard temporal order, even when the sound that disambiguates the utterance occurs after the noise. Figure 8b depicts the model's dynamics in response to a sequence of three inputs presented "1- -3" (bottom row, with '1' shown in blue and '3' in red), with a 50 msec silence

duration interval between '1' and '3'. The plots in rows 2 and 3 from the bottom, show the response of the acoustic feature layers F_i and E_i . The fourth plot from the bottom shows the activities $C_i^{(I)}$ of the acoustic item category cells. The activities Y_i and X_i of cells in the cognitive working memory layers (shown in the fifth and sixth plots from the bottom) respond to the incoming activity from the acoustic item layer. The seventh plot from the bottom shows the response of list chunk activities $C_J^{(L)}$ in the masking field in response to the evolving pattern of activity in working memory. When one or more list chunks gets sufficiently active, it opens the basal ganglia gate that will enable the resonance to unfold throughout the network. The singleton list chunks coding for "1", "2" and "3" are shown in blue, green and red, respectively, and the list chunks coding for "1-2-3" and "1-4-5" are shown in yellow and black, respectively. The top plot shows the resonant activity across the acoustic item layer, and exhibits a temporal break between the super-threshold activity of item cells '1' (blue trace) and '3' (red trace), corresponding to the silence perceived by listeners under these presentation conditions.

Figure 9a depicts model dynamics in response to a sequence of three inputs presented **"**1- * -3" where **'*'** denotes noise that is presented for 50 msec in place of any phoneme ('1' is shown in blue, '*' is shown as a filled yellow pulse, and '3' is shown in red). The bottom row shows presentation of the inputs, and the next two rows again show the response of the acoustic feature layers F_i and E_i . The fourth plot from the bottom shows the activities $C_i^{(I)}$ of the acoustic item category cell activities. The activities Y_i and X_i in the cognitive working memory layers, in response to the inputs from the acoustic item cells, are shown in the fifth and sixth plots from the bottom. The seventh plot from the bottom shows the response of list chunk activities $C_J^{(L)}$ in the masking field in response to the evolving pattern of activity in working memory. The singleton list chunks coding for "1", "2" and "3" are shown in blue, green and red respectively, and the list chunks coding for "1-2-3" and "1-4-5" are shown in yellow and black, respectively. Once the list chunk coding for "1-2-3" (the yellow trace) wins the competition with the "1-4-5" chunk (the black trace) upon unambiguous presentation of the acoustic item '3' at 100 msec, feedback from the chunk cells allows for the selection of and amplification of the components of noise consistent with its learned expectations, namely the excised acoustic item '2' in working memory. Basal ganglia gated feedback from the selected list chunk then drives acoustic features and items in such a way that the resonant wave across these items (shown in the top plot) continuously progresses across '1', '2', and then '3' (blue, green and red traces, respectively), indicating that the excised item '2' has been restored.

Figure 9b shows that the backward-in-time restoration is specific to the item that disambiguates the utterance. In particular, this simulation shows the network dynamics in response to the sequence **"**1- * -5**"**, where ***** again denotes noise ('1' is shown in blue, '*' is shown in yellow, and '5' is shown in purple). The only difference between this simulation and that of Figure 9a is the final item of the sequence, '5', which serves as future contextual information with respect to the excised phoneme, '4', which is to be restored. Rather than selection of the "1-2-3" list chunk (shown in yellow in the seventh plot from the bottom), presentation of the acoustic item '5' allows the "1-4-5" list chunk (shown in black) to win the competition across the masking field layer. Feedback from this chunk allows the selection and amplification of the components of noise consistent with its learned expectations, namely '4', whose activity is shown in cyan in the working memory activities of Y_i and X_i . The feedback

from working memory to acoustic features again causes the super-threshold activity in the acoustic item layer (shown in the top plot) to exhibit a resonant wave from '1', to '4', and then to '5' (blue, cyan, and magenta traces respectively), indicating that the excised item '4' has indeed been restored.

These simulations illustrate how that the restoration occurs in response to inputs arriving after the noise and, just as the restoration examples with 'delivery' and 'deliberation', completes the intervening sound in a context-appropriate way.

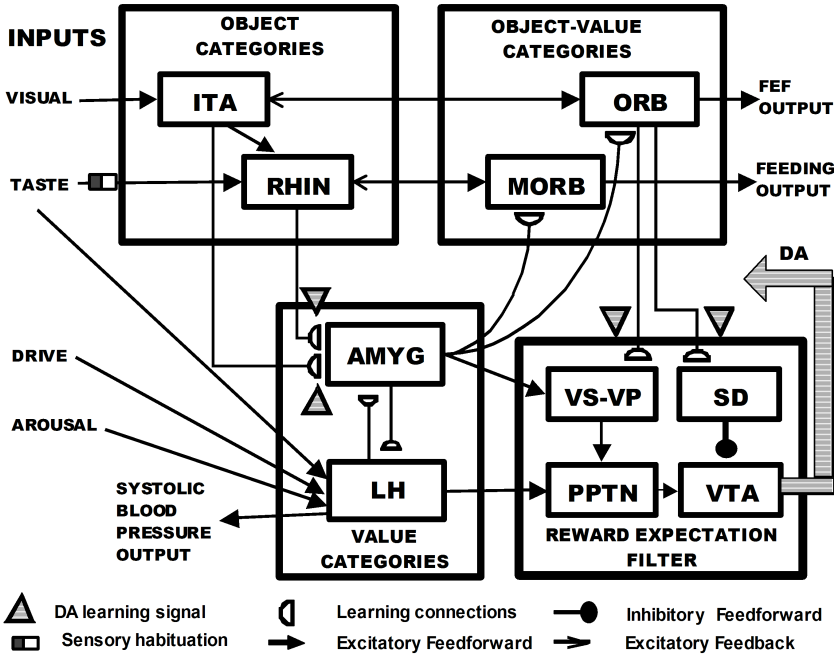


Figure 10. Overview of MOTIVATOR model: Brain areas in the MOTIVATOR circuit can be divided into four regions that process information about conditioned stimuli (CSs) and unconditioned stimuli (USs). (a) Object Categories represent visual or gustatory inputs, in anterior inferotemporal (ITA) and rhinal (RHIN) cortices; (b) Value Categories represent the value of anticipated outcomes on the basis of hunger and satiety inputs, in amygdala (AMYG) and lateral hypothalamus (LH); (c) Object-Value Categories resolve the value of competing perceptual stimuli in medial (MORB) and lateral (ORB) orbitofrontal cortex; and (d) the Reward Expectation Filter in the basal ganglia detects the omission or delivery of rewards using a circuit that spans ventral striatum (VS), ventral pallidum (VP), striosomes of the striatum, the pedunculopontine nucleus (PPTN) and midbrain dopaminergic neurons of the SNc/VTA (substantia nigra pars compacta/ventral tegmental area). The circuit that processes CS-related visual information (ITA, AMYG, ORB) operates in parallel with a circuit that processes US-related visual and gustatory information (RHIN, AMYG, MORB). The model captures systematic changes in processing of the same stimuli at different times, due to processes of learned category formation, sensory habituation, satiation or deprivation of particular rewarding outcomes, CS-US associative learning, and violations of expectations based on learned regularities. Model outputs modulate saccadic choice and reaction time, and blood pressure changes. [Reprinted with permission from Dranias, Grossberg, and Bullock (2008).]

7. Complementary roles of basal ganglia and amygdala in reinforcement learning

7.1. MOTIVATOR model

The TELOS model (Sections 3 and 4) illustrates how the basal ganglia, notably SNc, may generate dopaminergic Now Print signals in response to unexpected rewarding events. This proposal does not explain how previously rewarded, and currently valued, behaviors can be carried out under familiar and expected circumstances. The amygdala works together with the basal ganglia to support the complementary roles of new learning in response to unexpected reinforcing events and motivated performance in response to already conditioned cues, respectively. The MOTIVATOR model (Figure 10; Dranias, Grossberg, and Bullock, 2008; Grossberg, Bullock, and Dranias, 2008) proposes how key aspects of this interaction take place. MOTIVATOR is an acronym for Matching Objects To Internal VAlues Triggers Option Revaluations.

MOTIVATOR describes cognitive-emotional interactions between higher-order sensory cortices and an evaluative neuraxis composed of the hypothalamus, amygdala, orbitofrontal cortex, and basal ganglia. Given a conditioned stimulus (CS), the model amygdala and lateral hypothalamus interact to calculate the expected current value of the subjective outcome that the CS predicts, constrained by the current state of deprivation or satiation. The amygdala codes value categories (Aggleton, 1993; LeDoux, 1993) that relay the expected value information to object-value categories in the orbitofrontal cortex (Barbas, 1995; Baxter et al., 2000; Schoenbaum et al., 2003). These object-value categories also receive inputs from object categories in the anterior inferotemporal cortex, while medial orbitofrontal cells receive gustatory inputs from rhinal cortex. Both object and value information are needed to vigorously activate orbitofrontal cells. The activations of these orbitofrontal cells code the subjective values of objects. These values guide behavioral choices.

The model basal ganglia detect errors in CS-specific predictions of the value and timing of rewards. As in TELOS, excitatory inputs from the pedunculopontine nucleus interact with timed inhibitory inputs from model striosomes in the ventral striatum to regulate dopamine burst and dip responses from cells in the SNc and ventral tegmental areas. Learning throughout the brain is strongly modulated by these dopaminergic signals. Once conditioned, the amygdala can receive learned conditioned reinforcer signals from sensory cortices, such as the inferotemporal and rhinal cortices, and convey learned incentive motivational signals to the orbitofrontal cortex (Figure 8a).

Using these mechanisms, MOTIVATOR proposes mechanistic answers to the following kinds of questions: What brain processes allow an animal to use cues to quickly assess the options in its environment and estimate their values relative to the animal's current needs? How are strong needs ignored when the environment affords no opportunity for their satisfaction? How are normally attractive and highly available options ignored for a time after the needs that they consummate have been satisfied? In particular, MOTIVATOR simulates data about the conditioning of cues that predict specific outcomes in a task setting, the automatic revaluation of conditioned reinforcers following food-specific satiety, and motivational and emotive influences on decision processes, reaction time, response vigor, and blood pressure. Revaluation refers to the observation that motivational shifts can alter the vigor of conditioned responses (Dickinson and Balleine, 2001; Corbit and Balleine, 2005).

7.2. Basal ganglia learning affects sensory-amygdala-orbitofrontal motivated performance

MOTIVATOR unifies and further develops the Cognitive-Emotional-Motor, or CogEM, model

of cognitive-emotional learning and performance (Grossberg, 1971, 1972a, 1972b, 1975, 1982, 1984; 2000c; Grossberg and Gutowski, 1987; Grossberg and Levine, 1987; Grossberg, Levine and Schmajuk, 1987; Grossberg and Merrill, 1992; Grossberg and Schmajuk, 1987) and the TELOS model of how an animal learns to balance reactive vs. planned behaviors through learning based on reward expectation and its disconfirmation (Brown, Bullock, and Grossberg, 1999, 2004). The CogEM model focused on how affective brain regions, such as the lateral hypothalamus and amygdala, interact with sensory and cognitive areas, such as inferotemporal cortex and orbitofrontal cortex. In particular, an inferotemporal-amygdala-orbitofrontal resonance focuses motivated attention upon currently valued objects, as it also supports core consciousness and "the feeling of what happens" (Damasio, 1999). Indeed, the heuristic model that Damasio (1999) derives from his clinical data has the same form as the CogEM model, but uses different terminology for its processing stages. As reviewed in Sections 3 and 4, the TELOS model focused on how the basal ganglia regulate attention and reinforcement-based learning in thalamocortical systems. MOTIVATOR clarifies how both amygdala and basal ganglia processes interact to control reward-based processes.

Here is a more detailed summary of how MOTIVATOR proposes that these brain regions interact: Visual inputs activate view-invariant representations of visual objects in the anterior inferotemporal cortex (ITA). Gustatory cortex relays the taste properties salty, sweet, umami, and fatty to rhinal cortex (RHIN) and to gustatory-responsive lateral hypothalamic cells (LH_gus). RHIN cells also receive ITA inputs, and can thereby code gustatory-visual properties of food rewards. Endogenous drive and arousal inputs project to lateral hypothalamic input cells (LH_in). LH_in cells represent the homeostatic state of the animal by reporting fat, salt, amino acid, and sugar levels. LH_gus cells correlate gustatory tastes with corresponding homeostatic features and excite lateral hypothalamic output cells (LH_out), which project to amygdala (AMYG) cells that categorize distributed patterns of activity across LH_out states, and thus represent value categories. The LH-AMYG network computes the net subjective outcome associated with a consummatory act. It thereby defines a neural representation of US (unconditioned stimulus) reward value. Because the AMYG also receives conditionable CS-activated signals from ITA and RHIN, it can mediate CS-US learning. Given a CS, the AMYG and LH interact to calculate the expected current value of the subjective outcome that the CS predicts, given the current state of deprivation or satiation for that outcome. The AMYG relays the expected value information via incentive motivational signals to ITA-recipient orbitofrontal (ORB) and RHIN-recipient medial orbitofrontal (MORB) cells, whose activations code the relative subjective values of objects. These values guide behavioral choices.

The model basal ganglia (BG) detect errors in CS-specific predictions of the value and timing of rewards. Striosomes (SD) of the ventral striatum (VS) prevent predicted rewards from generating SNc/VTA responses by inhibiting dopamine cells in the SNc/VTA with adaptively timed signals (Figures 3 and 15). Inputs from the LH_gus and the ventral striatum (VS) excite the pedunculopontine nucleus (PPTN/LDT) whenever a conditioned (CS) or unconditioned (US) rewarding cue occurs. Cells in the PPTN/LDT, in turn, excite dopamine cells in the SNc/VTA.

When inhibitory signals from the SD and excitatory signals from the PPTN/LDT mismatch, a dopamine dip or dopamine burst may occur. A dopamine burst occurs in the SNc/VTA when an unexpected rewarding CS or US is presented. When an unexpected rewarding cue is presented, SD cells are unable to relay anticipatory inhibitory signals to the SNc/VTA and reward-related excitation is relayed from the PPTN/LDT to dopaminergic cells in the SNc/VTA, eliciting a dopamine burst. When an expected reward is omitted, a dopamine dip

occurs. In this case, a rewarding CS is presented and SD cells send an adaptively timed inhibitory input to the SNc/VTA at the expected time of reward. When US presentation is omitted, dopaminergic SNc/VTA cells never receive a reward-related excitatory signal from the PPTN/LDT and are instead transiently suppressed by inhibitory signals from the SD (Figure 2b).

Model simulations reproduce discharge dynamics of known cell types, including signals that predict saccadic reaction times and CS-dependent changes in systolic blood pressure. Learning in cortical and striatal regions is strongly modulated by dopamine, whereas learning between the AMYG and LH_out cells is not.

7.3. Influences of amygdala and orbitofrontal lesions on learning and behavior

In addition, interactions of the BG and AMYG with sensory and ORB cortices enable the model to replicate the complex pattern of spared and impaired behavioral and emotional capacities seen following lesions of the amygdala and orbitofrontal cortex (Grossberg, Bullock, and Dranias, 2008). For example, experimental data show that the ability of a conditioned stimulus to act as a conditioned reinforcer is impaired following amygdala lesions (Hatfield et al., 1996; Setlow et al., 2002a). Experiments also reveal that, if the CS is trained prior to the amygdala lesion being made, the ability of the CS to function as a conditioned reinforcer and to induce secondary conditioning is intact. This preserved function relies on pathways through the ventral striatum (Setlow et al., 2002b). In the model, US-specific drive-value category cells in the amygdala project to the ventral striatum, providing teaching signals for inputs from the ORB (Figure 15). When the model is trained prior to amygdala lesions, connections between the orbitofrontal cortex and US-specific ventral striatal cells learn to reflect US value and compensate for the loss of the amygdala. Recovery of second-order conditioning occurs because this pretraining establishes a learned pathway from the ORB to the ventral striatum that enables the CS to trigger a dopamine burst.

8. Item-order-rank working memory and basal ganglia gating of behavioral sequences

8.1. Basal ganglia control of sequential learning and performance of saccades

Although the TELOS model included some properties of the prefrontal cortex (PFC, Figure 4a), including its ability to store salient plan representations in short-term memory using recurrent shunting on-center off-surround networks (Grossberg, 1973), its model PFC did not include a sequential working memory; namely, a network capable of temporarily storing in short-term memory a sequence of items and their temporal order. However, intelligent behavior depends upon the capacity to think about, plan, execute, and evaluate sequences of events. Whether we learn to understand and speak a language, solve a mathematics problem, cook an elaborate meal, or merely dial a phone number, multiple events in a specific temporal order must somehow be stored temporarily in working memory. As event sequences are temporarily stored, they are grouped, or chunked, through learning into unitized plans, or list chunks, and can later be performed at variable rates under basal ganglia volitional control either via imitation or from a previously learned plan. See Grossberg and Pearson (2008) for simulations of how such variable-speech sequential performance can be controlled.

The cARTWORD model does include a sequential working memory that temporarily stores a sequence of acoustic items in working memory as they are unitized through learning into list chunks. However, the effects of basal ganglia gating in cARTWORD were expressed in the simplest way, and did not attempt to explain how different parts of the basal ganglia gate the release of different components of sequentially organized behaviors. The lisTELOS model (Silver et al., 2011) does offer this kind of detailed explanation of basal ganglia dynamics during the learning and performance of sequences of saccadic eye movements.

8.2. Item-Order-Rank working memories store sequences using activity gradients

Lashley (1951) suggested that items are temporarily stored in working memory (WM) within spatially separable neural populations, thus transforming the temporal problem of serial order into a spatial problem. Grossberg (1978a, 1978b) developed a neural model of WM through which a temporal stream of inputs could be stored as an evolving spatial pattern before being performed sequentially during rehearsal. In such an Item-and-Order WM, individual nodes, which represent cells or cell populations, represent *list items*, and the order in which the items were presented is stored by an *activity gradient* across these nodes. A *primacy gradient* achieves performance in the correct temporal order. In a primacy gradient, the first item in the sequence is represented by the cell(s) with the highest activity, and subsequent items are stored with progressively less activity. A rehearsal wave, that is volitionally controlled by the basal ganglia, opens gates that enable read-out of these stored activities when it is time to reproduce the sequence. The cell with the highest activity is read out first and self-inhibits its WM representation, an example of an *inhibition of return*, thereby preventing perseverative performance of this item. This process is repeated until the entire sequence is reproduced and there are no active nodes in the WM.

Such an Item-and-Order WM is also sometimes called *competitive queuing* (CQ; Houghton, 1990), and many models have adapted this scheme. Both psychophysical and neurophysiological data have confirmed its predicted properties. For example, psychophysical experiments have shown that latency data from error trials can be best explained by models that use a primacy gradient and self-inhibition; e.g., Farrell and Lewandowsky (2004). Electrophysiological recordings from PFC have, moreover, shown that the temporal order of items in a sequence of stored motor commands is stored using their relative activity levels, and that these activities are reset by self-inhibition as each motor command is executed; e.g. Averbeck, Chafee, Crowe, and Georgopoulos (2002, 2003).

In addition to these Item-and-Order properties, the activity of PFC neurons for a given list item is sometimes modulated by the rank, or position, of that item within the sequence, and error data imply utilization of rank information in serial recall (see Silver et al. (2011) for a review).

The LIST PARSE model of working memory and list chunking (Grossberg and Pearson, 2008) proposed how laminar circuits in PFC represent these two types of processes. Grossberg and Pearson (2008) also suggested how rank-order coding may be incorporated into the activity gradients within an Item-and-Order WM to represent item repeats at arbitrary list positions. They suggested, in particular, that the PFC rank information is derived from representations of numerosity in posterior parietal cortex (PPC; Grossberg and Repin, 2003). Silver et al. (2011) built upon this heuristic proposal to rigorously model a prefrontal *Item-Order-Rank* model of WM storage and performance. This WM was used to quantitatively simulate neurobiological data about rank-order coding in a spatial WM in PFC as it interacts with multiple brain regions, including SC, PPC, PFC, FEF, and the supplementary eye fields (SEF), all regulated by the basal ganglia, in order to learn and perform sequences of saccadic eye movements. This neural architecture is called the lisTELOS model (Figure 11a) to acknowledge that it unifies and extends properties of both the LIST PARSE and TELOS models.

8.3. All working memories are variations of the same circuit design

Grossberg (1978a, 1978b) derived Item and Order working memories from two postulates: the LTM Invariance Principle and the Normalization Rule. The LTM Invariance Principle makes

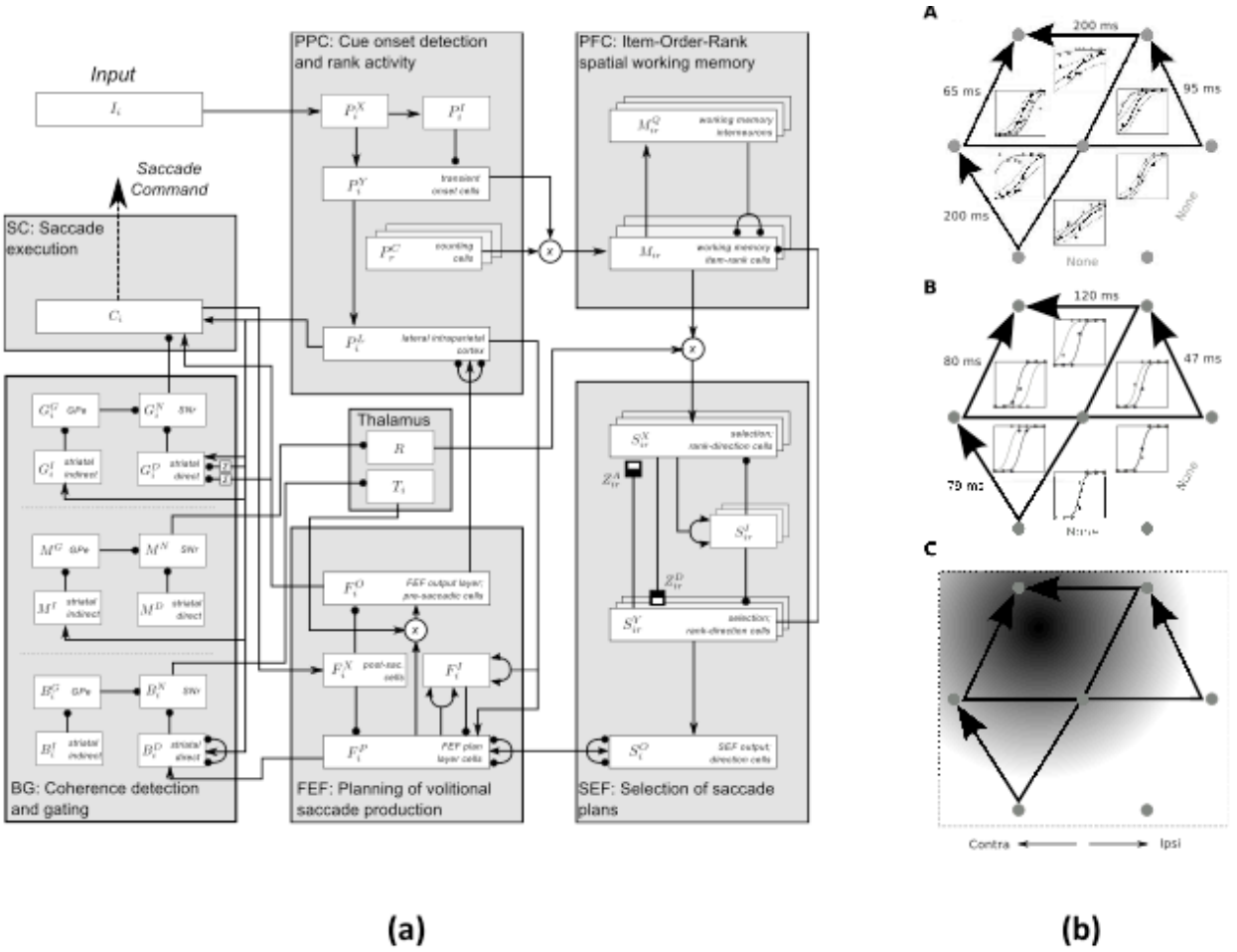


Figure 11. (a) The lisTELOS Item-Order-Rank spatial working memory and performance model. Each gray box represents a brain region within which fields of cells, represented by white inset boxes, share similar functional roles. Arrowheads denote excitatory connections between cells, and filled circles represent inhibitory connections. Curved branches at the ends of connections represent one-to-many fan-out connections that impact all other cells in the field. Half-filled boxes at the ends of connections represent habituative gates which exhibit activity-dependent changes in synaptic efficacy. White circles containing a multiplication sign (×) represent multiplicative interaction between two signals. Boxes containing a sigma (Σ) represent the sum of outputs from all cells in the field that gave rise to the projection. Stacked field representations denote populations of rank-sensitive cells. See Figure 12 for more details about how three basal ganglia loops contribute to the learning and performance of saccadic eye movement sequences by the model. [Reprinted with permission from Silver et al. (2011).]. (b) Evidence that prefrontal cortex and SEF embody spatial representations of saccadic target locations. A. Microstimulation causes saccade trajectories to converge. The bias observed for each of the six pairs of adjacent cues (insets) can be used to identify the saccade trajectory rendered more likely by microstimulation (arrows). Note that the saccadic trajectories converge toward the upper left target. B. Model simulations reproduce the convergence effect. C. In model simulations, microstimulation habituates synapses according to a two-dimensional Gaussian function centered over the microstimulation site. Saccade trajectories following

microstimulation tend to climb this gradient. [Data adapted with permission from Histed and Miller (2006). Simulation reprinted with permission from Silver et al. (20]

precise the idea that there is no point in storing novel sequences of events in working memory if the brain cannot learn to unitize the sequences for future skillful performance. This Principle claims that working memories are *designed* to enable such stable list chunking to occur. In particular, it demands that all working memories enable a novel superset list chunk (up to a certain maximal length) to be learned without forcing catastrophic forgetting of familiar subset chunks. For example, the LTM Invariance Principle ensures that a novel superset word like MYSELF can be learned without forcing forgetting of the familiar subwords MY, SELF, and ELF. As a result, as new items are stored through time in working memory, subset list chunks can continue to activate their familiar list chunks until they are inhibited by contextually more predictive superset list chunks; e.g., until MY is supplanted by competition from MYSELF through time. The *learning* of chunk MY within its bottom-up filter is not undermined, but the *current activation* of the chunk MY can be inhibited by MYSELF.

The Normalization Rule assumes that the *total activity* of the working memory network has a maximum that is (approximately) independent of the total number of actively stored items. In other words, working memory has a *limited capacity* and activity is redistributed, not just added, when new items are stored.

Two properties are needed for these postulates to hold:

(1) In order not to force recoding of sublists (like MY) as superlists of them (like MYSELF) are stored in working memory, activities of items in working memory tend to preserve their *relative* activations, or *ratios*, throughout the time that they are stored in working memory, even if the storage of new items through time might change the absolute amount of activity with which each item is stored. This property enables the adaptive filter that converts the distributed pattern of stored items into list chunks (see Figure 8a) to activate already learned list chunks in response to their sublists in working memory.

(2) Novel superlists (like MYSELF) must be able to activate the chunking network despite the salience of already learned sublists (like MY), so that learning of a new list chunk with which to represent the novel superlist can occur. This is accomplished by using a masking field at the list chunk level, as in the cARTWORD model.

How can brain evolution be smart enough to discover the laws of something so seemingly sophisticated as a working memory? Remarkably, Item and Order and Item-Order-Rank working memories that satisfies the LTM Invariance Principle and Normalization Rule can be realized by a ubiquitous kind of neural network: an on-center off–surround network whose cells obey the shunting, or membrane, equations of neurophysiology and which interact via *recurrent* on-center off-surround connections. Recurrent shunting on-center off-surround networks are ubiquitous in the brain (Grossberg, 1973). Recurrence is needed because the positive feedback from a cell population to itself in the recurrent on-center stores the evolving input pattern, while the recurrent competition contrast-normalizes the stored activities across the network. The shunting, or multiplicative, properties of the membrane equations, combined with the on-center off-surround interactions, enable the network to compute *ratios* of cell activities across the network, as is required by the LTM Invariance Principle, even as they normalize the total activity across the network, as required by the Normalization Rule.

Because all working memories need to obey the LTM Invariance Principle and the Normalization Rule, similar working memory circuits were predicted to store spatial, linguistic,

and motor sequences (Grossberg, 1978a). The cARTWORD and lisTELOS models and their data explanations provide supportive evidence for this prediction for the cases of linguistic and spatial working memories. See Grossberg (1978) for a review of additional supportive experimental evidence.

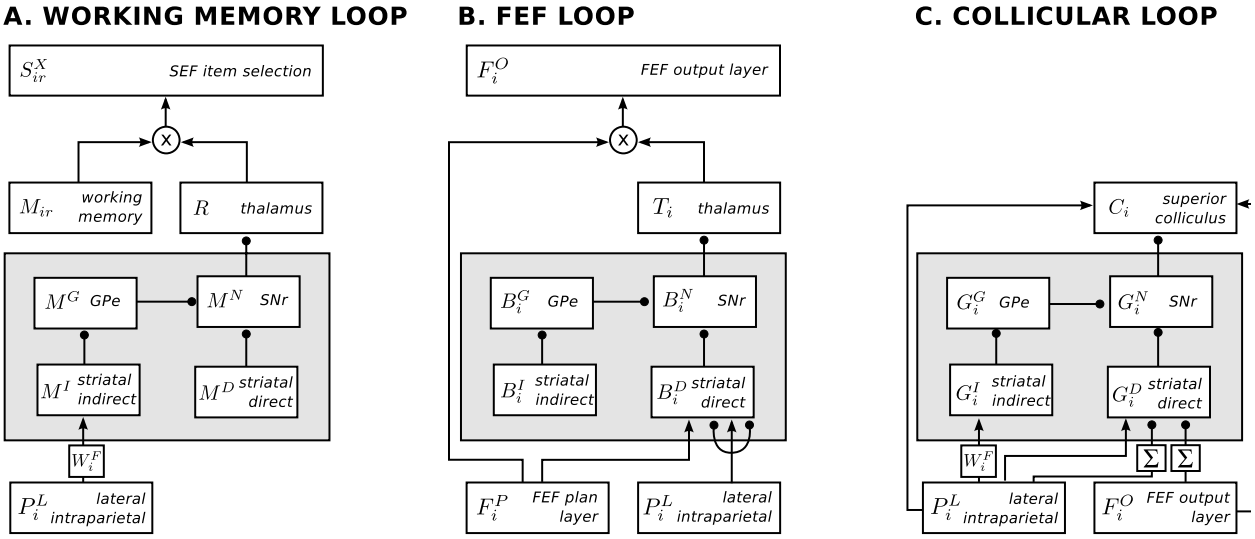


Figure 12. The lisTELOS model explains how three loops through the basal ganglia contribute to saccadic performance. Each loop projects to a separate thalamic or collicular population (cf., Figure 1), modulating the population's excitability and thereby controlling the flow of information from one model stage to another. A. The left panel represents the working memory loop through the BG, which is responsible for controlling the flow of information from working memory cell activities M_{ir}, to the SEF selection cell activities S_{ir}^X. B. The FEF loop controls the flow of plan signals from FEF plan layer cell activities F_i^P to FEF output layer cell activities F_i^O. C. The collicular loop controls excitation of SC cell activities C_i, by FEF output cell activities F_i^O, and LIP cell activities P_i^L. See text for details. [Reprinted with permission from Silver et al. (2011).]

8.4. Supplementary eye fields select saccadic targets from sequences stored in spatial WM

LisTELOS proposes how item representations may be chosen from WM by the SEF, an oculomotor area in dorsomedial frontal cortex (Schlag and Schlag-Rey, 1987) which is heavily interconnected with the PFC (Barbas and Pandya, 1987; Huerta and Kaas, 1990) and which also exhibits rank-related activity (Berdyyeva and Olson, 2009; Isoda and Tanji, 2002, 2003). SEF is thus anatomically and physiologically well-suited to interact with a rank-selective WM. Its role in the selection of saccadic targets is consistent with many data, reviewed in Silver et al. (2011). For example, patients with lesions in what was at the time called the supplementary motor area (Gaymard, Pierrot-Deseilligny, and Rivaud, 1990; Gaymard, Rivaud, and Pierrot-Deseilligny, 1993) have mostly intact performance for visually-guided saccades, antisaccades, and single memory-guided saccades, but greatly degraded performance for *sequences* of memory-guided saccades. In addition, activation of SEF during sequential saccade tasks has been observed with positron emission tomography (Petit et al., 1996) and during a functional magnetic resonance

imaging study (Heide et al., 2001) whose authors concluded that "the supplementary eye field essentially controls the triggering of memorized saccade sequences."

The competence of the lisTELOS model was tested by simulating data collected from several different paradigms, including visually-guided and memory-guided saccade tasks and several sequential saccade tasks, notably the immediate serial recall (ISR) task. The model is also compatible with known anatomical data, and reproduces behavioral and electrophysiological data under a variety of conditions, including those in which SEF activity is perturbed by microstimulation (Histed and Miller, 2006; Yang, Heinen, and Missal, 2008). These last data provide particularly strong support for the concept of a spatial Item-Order-Rank working memory due to the manner in which microstimulation may alter the temporal order, but not the target positions, that are acquired by the sequential saccadic eye movements (Figure 11b).

8.4. Basal ganglia regulation of saccade sequence learning and performance

To explain learning and performance of eye movement sequences, and by extension other kinds of movement sequences, the lisTELOS model simulates in considerable cellular detail how three loops through the basal ganglia (BG; Middleton and Strick, 2000) control the flow of information between model areas (Figure 12). Each of these loops is based on the BG implementation used in the TELOS model (Figure 4a). As reviewed in Section 4, in TELOS, consistent with hypotheses of other researchers (Alexander and Crutcher, 1990; Bullock and Grossberg, 1988, 1991; Gancarz and Grossberg, 1999; Grossberg, Roberts, Aguilar, and Bullock, 1997; Hikosaka and Wurtz, 1983; Mink, 1996), the BG are responsible for controlling the selective release of a movement through a gating process. Eye movements are initiated when consistent saccade plans in FEF and PPC occur, thereby changing the balance of excitation and inhibition impinging on the BG in favor of selective gate opening, and triggering a frontal-parietal resonance that embodies a system consensus about a chosen saccadic command (Figure 9D). By ensuring that these areas reach consensus before allowing saccade generation, the BG avoid various problems such as premature execution of reactive saccades when a planned saccade is appropriate, or simultaneous execution of multiple saccade plans, as sometimes occurs in the form of saccadic averaging (Lee, Rohrer, and Sparks, 1988; Ottes, Van Gisbergen, and Eggermont, 1984). Thus, in addition to unifying processes of numerosity in PPC, spatial WM storage in PFC, and saccade selection in SEF, the model elaborates how the BG selectively gate the release of a saccadic movement when frontal-parietal resonance occurs.

BG gate opening in the model relies on opposing forces between the direct and indirect pathways (Figures 4a and 11a; Brown et al., 2004; Frank, 2005; Frank, Loughry, and O'Reilly, 2001; Mink, 1996). The direct and indirect pathways begin with two distinct populations of γ-aminobutyric acid (GABA) releasing medium spiny projection neurons (MSPNs) in the striatum, the input nucleus of the BG. These pathways differentially express D1 and D2 receptors (Gerfen et al., 1990; Surmeier, Ding, Day, Wang, and Shen, 2007). In particular, MSPNs in the direct pathway send projections directly to the globus pallidus internal segment (GPi) and the substantia nigra pars reticulata (SNr), which serve as output nuclei of the BG. Cells in GPi/SNr are GABAergic and tonically inhibit cells in the thalamus or SC (Bullock and Grossberg, 1991; Hikosaka and Wurtz, 1983; Horak and Anderson, 1984). Activation of direct pathway MSPNs inhibits GPi/SNr cells, and thereby disinhibits cells downstream from the tonic GPi/SNr signal.

Indirect pathway MSPNs inhibit cells in the nearby globus pallidus external segment (GPe) which, in turn, inhibit the GPi/SNr output nuclei. Thus, exciting indirect pathway MSPNs disinhibits GPi/SNr cells. The resulting increased activity of GPi/SNr inhibits SC or thalamic cells. As a result, the indirect pathway acts in opposition to the direct pathway: Direct pathway

activation excites cells in thalamus or SC, whereas indirect pathway activation inhibits them. These opposing processes of disinhibition and inhibition realize BG gating.

Working memory loop and gate. Each of the three parallel BG loops gate a separate process. The BG WM loop (Figure 12A) controls signalling from PFC WM cell activities M_{ir} to SEF selection cell activities S_{ir}^X through a thalamic rehearsal gate *R* (see Section 7.2). LIP cell activities P_i^L activate MSPN activities M^I of the indirect pathway using hard-coded connection weights W_i^F. The model hereby responds selectively to the presence of a fixation cue by inhibiting indirect pathway GPe cell activities M^G and thereby disinhibiting SNr cell activities M^N. The resulting increased SNr activity keeps the WM rehearsal gate *R* closed, thereby restricting the flow of information into SEF.

When the fixation point is removed, LIP cell activities P_i^L no longer excite MSPNs, and the rehearsal gate *R* opens, thereby allowing SEF cell activities S_{ir}^X to be activated by WM cell activities M_{ir}. In the absence of any additional fixation cues, this gate remains open, enabling each saccadic plan to be successively selected and to activate downstream areas, such as FEF and SC, to generate the corresponding saccade. Direct pathway MSPN activities M^D maintain constant activity so that, in the absence of indirect pathway activity, the WM rehearsal gate *R* is open.

Frontal eye fields loop and gate. The second BG loop, the FEF loop (Figure 12B), controls the flow of information between the FEF plan layer cell activities F_i^P and the FEF output layer cell activities F_i^O. The thalamic gate T_i controlled by this loop remains closed until FEF plan layer cell activities F_i^P and LIP cell activities P_i^L represent a consistent plan as part of a frontal-parietal resonance. Once the regions contain consistent saccade plans, they excite direct pathway cell activities B_i^D which inhibit SNr cell activities B_i^N, thereby disinhibiting the thalamic cell activities T_i. Once disinhibited, thalamic cell activity, combined with FEF plan layer activity, activate FEF output layer cell activities F_i^O. The FEF output layer then is ready to excite a corresponding saccade plan in further stages of the model, but cannot do so until a second BG gate is opened. Indirect pathway MSPN activities B_i^I and GPe cell activities B_i^G provide a constant source of inhibition to SNr cell activities B_i^N to ensure that only consistent FEF and LIP activity, resulting in strong direct pathway activity, is able to release thalamic activity T_i from inhibition.

Superior colliculus loop and gate. The third gate controls outputs from the SC (Figure 12C) and receives inputs from both FEF output layer cell activities F_i^O and LIP cell activities P_i^L, with special emphasis placed on the central region of the visual field where fixation cues are present, as in the WM loop. A fixation cue at the center of the visual field selectively activates the collicular loop indirect pathway MSPN activities G_i^I, which inhibit GPe cell activities G_i^G, then disinhibit SNr cell activities G_i^N, which in turn inhibit colliculus cells with activities C_i. While a fixation cue is on, it is difficult for FEF or LIP to excite the activities G_i^D of direct pathway MSPNs enough to overcome activity in the indirect pathway. If no fixation cue is on, and the saccadic plans in FEF and LIP are consistent, this third gate opens, which allows FEF

and LIP to excite SC cell activities C_i, thereby leading to a saccadic movement that is consistent with the selected plan.

The three BG loops are critical for holding the model in a state of preparedness as information important for guiding its future responses is being presented, and detecting the task conditions which signal that it is time to utilize the stored information to drive behavior. This process depends largely on the presence and absence of the fixation point. When a fixation cue is present, the rehearsal and collicular gates are held shut and task-relevant cues are simply stored in memory. Once the fixation point is removed, SEF can select saccade targets from WM and excite corresponding representations in FEF. Provided the selected saccade plan is not inconsistent with any external cues represented in LIP, the FEF and collicular BG loops open their gates and allow plan signals to flow to SC, which generates the response.

The above mechanisms propose how an Item-Order-Rank spatial working memory can be used to represent arbitrary spatial sequences, and suggests how three distinct BG gates enable SEF to select spatial targets from WM and excite corresponding representations in downstream oculomotor areas such as SC that are responsible for saccade production.

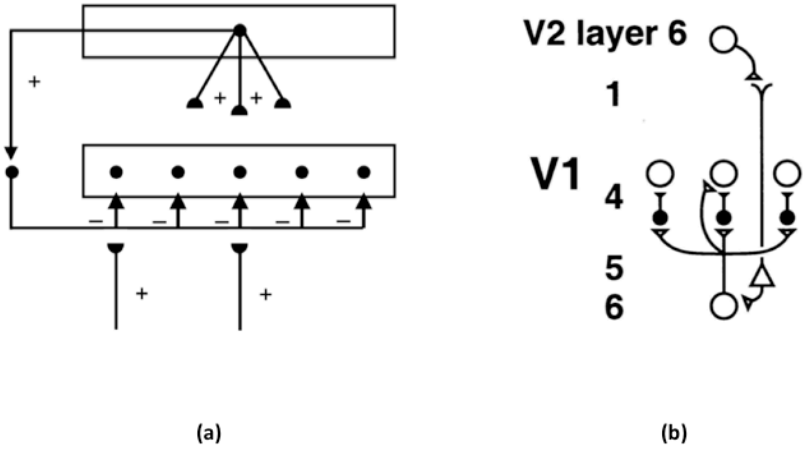


Figure 13. (a) The ART Matching Rule is achieved by a top-down, modulatory on-center, off-surround network. The excitatory on-center (plus signs) encodes a learned prototype in its adaptive weights, or long-term memory traces (hemidisks). This prototype learns from bottom-up inputs, which can fully activate targets cells when top-down signals are off. The inhibitory off-surround (minus signs) is balanced against the on-center so that top-down signals, by themselves, are modulatory, and cannot fully activate their target cells. When both bottom-up and top-down signals are active, only the cells in the top-down on-center that are also receiving bottom-up inputs can fire. Other cell activities are inhibited. A volitional signal from the basal ganglia can disrupt the top-down excitatory-inhibitory balance to enable top-down signals, by themselves, to cause suprathreshold activation. (b) Model circuit for how the ART Matching Rule is realized within the laminar circuits of visual cortical areas V1 and V2. Similar circuits are proposed to occur in other sensory and cognitive cortical areas. Open circles and triangles denote excitatory cells and pathways, respectively; closed black circles and triangles denote inhibitory cells and pathways, respectively. A *folded feedback* circuit carries top-down attentional signals from layer 6 of V2 to layer 4 of V1 via an on-center off-surround pathway from layer 6 to 4 of V1. Corticocortical feedback axons from layer 6 in V2 tend to terminate in layer 1 of V1 (Salin and Bullier, 1995, p. 110) where they can, for example, excite apical

dendrites of layer 5 pyramidal cells whose axons send collaterals into layer 6. From layer 6, the feedback is then "folded" back into the feedforward flow of information from layer 6 to 4 of V1 via an on-center off-surround pathway (Bullier et al., 1996). See Grossberg (2012) and Raizada and Grossberg (2003) for a more complete model of how this circuit is embedded within the bottom-up, horizontal, and top-down (both intracortical and intercortical) interactions within visual cortex.

9. Basal ganglia gating of perceptual and cognitive processes

9.1. From top-down attentional priming to suprathreshold activation

Many other brain processes can also be gated by the basal ganglia, whether automatically or through conscious volition. Several of these gating processes seem to regulate whether a top-down process subliminally primes or fully activates its target cells. As noted in Section 5.1, the ART Matching Rule enables the brain to dynamically stabilize learned memories using top-down attentional matching. Such attentional matching is realized by variants of a top-down, modulatory on-center, off-surround network (Figure 13a) that enables a top-down expectation to prime, or sensitize, the target cells in its on-center without fully activating them. It seems, however, that many of these attentional processes may be gated by the basal ganglia to enable the top-down priming to be converted into suprathreshold activation.

Phasic volitional signals can shift the balance between excitation and inhibition to convert the top-down modulatory on-center into a driving excitatory input that can cause suprathreshold activation. In the ART Matching Rule laminar circuit in Figure 13b, this gating action can either weaken the inhibitory effect of the off-surround, say by inhibiting the inhibitory interneurons in layer 4, or by further disinhibiting the excitatory on-center, say via cells in layer 5; cf. the gating of FEF laminar circuits in the TELOS model (Figure 4a).

9.2. Visual imagery, thinking, planning, and searching

Such a volitionally-mediated shift enables top-down expectations, even in the absence of supportive bottom-up inputs, to cause conscious experiences of imagery and inner speech, and thereby to enable visual imagery, thinking, and planning activities to occur. Thus, the ability of volitional signals to convert the modulatory top-down priming signals into suprathreshold activations provides a great evolutionary advantage to those who possess it.

Such a competence is also important when the brain tries to search for a valued goal object in a cluttered scene; that is, to solve the Where's Waldo problem. As noted in Section 4.4, the reciprocal ART-learned connections between spatially-variant recognition categories in cortical area ITp and spatially-invariant categories in ITa, combined with the inferotemporal-amygdala-orbitofrontal resonance that focuses motivated attention upon valued goal objects, has been used to propose a solution to the Where's Waldo problem, or how to search for a valued goal object in a cluttered scene (Chang, Grossberg, and Cao, 2014). This solution uses the top-down attentional priming by the ART Matching Rule from orbitofrontal cortex to ITa. By itself, such a prime cannot drive its ITa category to suprathreshold activity levels. Volitionally opening the corresponding basal ganglia gate, just as in the triggering of visual imagery, allows the motivationally amplified orbitofrontal object-value categories to fully activate their target invariant ITa object categories, which in turn can subliminally prime consistent ITp categories, again by the ART Matching Rule. When a bottom-up input from Waldo combines with such a prime at the ITp category that represents Waldo's location, this ITp category can become supraliminally activated, and inhibit less activated ITp categories. It can also activate the corresponding position in parietal cortex, which in turn can drive an eye movement towards

Waldo's location. Thus, basal ganglia gating can also enable motivated searches to occur.

9.3. From phasic to tonic gate opening: hallucinations

What happens, however, if volitional control of such priming signals is lost? During a mental disorder like schizophrenia, it is proposed that the phasic volitional signal may become tonically hyperactive. As a result, top-down sensory expectations can generate conscious experiences that are not under the volitional control of the individual who is experiencing them. The net effect is a hallucination. Since the top-down expectations learn prototypes that incorporate the critical features that are used to bind sensory features into conscious experiences, these hallucinations, just like the imagery and inner speech that are generated under normal conditions, are sufficient to generate conscious experiences with vivid personal content. Such hallucinations derive from the critically important ability to learn quickly throughout life without experiencing catastrophic forgetting, along with the consequent ability to learn expectations that focus attention upon important objects. These abilities provide the computational context in which basal ganglia gating can control imagination, thinking, and planning. The fact that these circuits may occasionally get out of balance and cause hallucinations may be viewed as one of the evolutionary costs of our ability to be human. See Grossberg (2000a) for additional discussion of such hallucinations and supportive data.

9.4. Working memory storage and the useful-field-of-view of spatial attention

The LIST PARSE model of working memory (Section 8.2) is realized by an on-center off-surround recurrent network whose on-center is modulatory except when sequential lists of items are being stored in working memory. LIST PARSE proposes, moreover, that this on-center off-surround network occurs in the deeper layers of ventrolateral prefrontal cortex, precisely the location where basal ganglia volitional signals can convert the modulatory on-center into a driving on-center that enables list items to be stored in working memory. A termination of this gating signal then allows the list to be cleared from working memory. This proposal needs to be further tested experimentally.

Another example where basal ganglia gating may influence performance concerns the span of spatial attention, also called the useful-field-of-view. In particular, the distributed ARTSCAN (dARTSCAN) model (Foley, Grossberg, and Mingolla, 2012) suggests how the span of spatial attention may be varied in a task-sensitive manner via learned or volitional signals that are mediated by the basal ganglia. Spatial attention may be focused on one object (unifocal) to control invariant object category learning, or spread across multiple objects (multifocal) to regulate useful-field-of-view, thereby raising the question of how the span of spatial attention is regulated. Individual differences in detection rate of peripheral targets in useful-field-of-view tasks are instructive, and are illustrated by the improved performance of experienced video game players over non-video game players (Green and Bavelier, 2003, 2007). These differences have been explained by dARTSCAN model (Foley, Grossberg, and Mingolla, 2012) as being due to the way in which volitional basal ganglia signals, or learned prefrontal-to-basal ganglia signals, may control the gain for gating the balance between excitation and inhibition in parietal and prefrontal cortex that helps to control the span of spatial attention in these cortical areas. The computer simulations of Foley, Grossberg, and Mingolla (2012) simulated the video game player advantage by assuming that they experienced a lower inhibitory gain.

10. Complementary systems for self-stabilizing expertise in a changing body

The above examples illustrate how the basal ganglia can influence learning and performance across brain systems in the What and Where cortical streams that obey computationally complementary laws (Section 1.3; Figure 14). For example, volitional GO signals control the

selection of motor synergies and the speeds with which they execute arm movement trajectories in the VITE model and its variants (Figures 6 and 7). The VITE model simulates *inhibitory matching* between a present position vector, or where the arm is now, and its target position vector, or where the arm wants to move. When the difference vector between the present and target position vectors equals zero, the movement stops. Corresponding to such inhibitory matching, motor systems that obey VITE-like dynamics also experience *mismatch learning* that calibrates the gains of the vectors that are matched so that the difference vector equals zero when the target and present position vectors represent the same position in space.

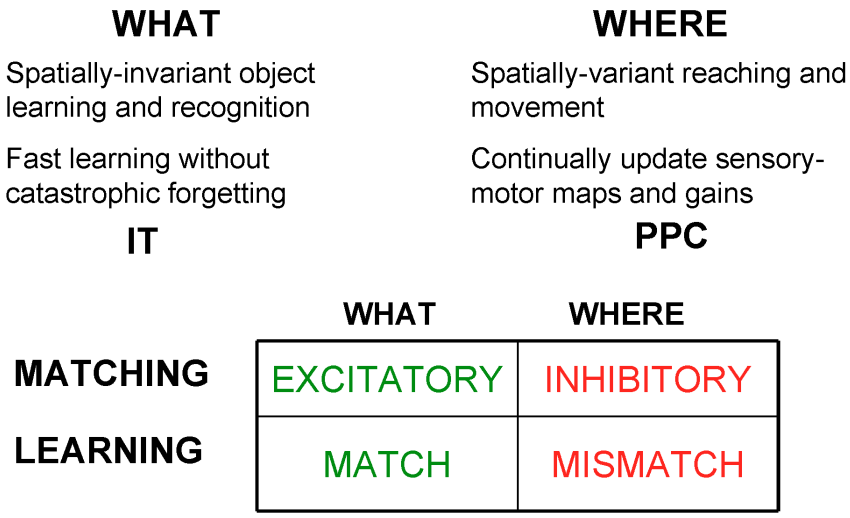


Figure 14. Complementary What and Where cortical processing streams for spatially-invariant object recognition and spatially-variant spatial representation and action, respectively. Perceptual and recognition learning use top-down excitatory matching and match-based learning that achieves fast learning without catastrophic forgetting. Spatial and motor learning use inhibitory matching and mismatch-based learning that enable rapid adaptation to changing bodily parameters. IT = inferotemporal cortex, PPC = posterior parietal cortex. See text for details. [Reprinted with permission from Grossberg (2009).]

Models that experience such vector-based mismatch learning are called Vector Associative Map, or VAM, models or adaptive VITE, or aVITE, models (Gaudiano and Grossberg, 1991, 1992). Such mismatch learning is susceptible to catastrophic forgetting. However, catastrophic forgetting is a good property for learning the spatial maps and sensory-motor gains that control movements in the Where cortical stream. In particular, it would be maladaptive to remember for life the maps and gains whereby our brains controlled our infant limbs. Continual recalibration of maps and gains enables us to efficiently control our changing bodies.

In contrast, perceptual and cognitive systems that obey the ART Matching Rule (Carpenter and Grossberg, 1987, 1991; Grossberg, 2012) experience *excitatory matching* (Figure 13) that can gain-amplify and synchronize cell responses that are part of a bottom-up and top-down matching event. Corresponding to such excitatory matching, ART systems undergo *match based learning* that helps to solve the stability-plasticity dilemma, so that perceptual and cognitive systems can cumulatively learn more about the world, notably invariant object recognition categories within the What cortical stream, without undergoing catastrophic forgetting.

These differences between What and Where stream processing also clarify key properties of conscious experience. For example, the ART prediction that ‘‘all conscious states are resonant states’’ has been elaborated into a classification of the resonances that support different conscious experiences (see Grossberg (2012) for a review), including those supporting declarative memory. This prediction also clarifies why spatial and motor, also called procedural, processes are unconscious: the inhibitory matching process that supports spatial and motor processes cannot lead to resonance.

In summary, perceptual/cognitive processes often use ART-like excitatory matching and match-based learning to create self-stabilizing memories of objects and events that enable us to achieve increasing expertise as we learn more about the world. Complementary spatial/motor processes often use VAM-like inhibitory matching and mismatch-based learning to continually update spatial maps and sensory–motor gains to compensate for bodily changes throughout life. Together these complementary predictive and learning mechanisms create a self-stabilizing perceptual/cognitive front end for intelligently manipulating the more labile spatial/motor processes that enable our changing bodies to act effectively upon a changing world. How the basal ganglia evolved to bridge across, and help to coordinate, these computationally complementary competences to support multiple learning and movement gating processes is an intriguing question for future research.

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