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The Basal Ganglia viewed as an Action Selection Device

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Abstract

The *action selection* problem describes the task of resolving conflicts between the different functional systems that can control behavior. This paper reviews the role of the *basal ganglia* (BG) summarising evidence that they function within the vertebrate brain architecture as a specialized action selection device. There is a rich connectivity within the BG whose function is not well understood. We outline a new computational model of BG intrinsic pathways which demonstrates that these circuits could allow the BG to implement clean switching between competing functional systems.

1 The role of the basal ganglia in action selection

An important task for the vertebrate nervous system is the resolution of conflicts between functional units that are physically separated within the brain but are in competition for common resources. For instance, the neural systems involved in tasks such as feeding, drinking, and escape, are located at widely distributed sites and at multiple levels of the neuraxis, yet are in competition for the use of the same effector mechanisms. The task of resolving such conflicts has been the subject of much research in ethology and artificial intelligence (see [1,2]) where it is termed the *action selection* problem. We have argued in [2] that the requirement for effective action selection favors the evolution of centralised switching devices, and that in the vertebrate brain, the *basal ganglia,* a group of functionally-related, central brain structures, have evolved to fill this role. This paper briefly reviews neuroscientific evidence for the involvement of the BG in action selection and outlines a new computational model of BG intrinsic circuitry viewed as implementing a switching device.

The principal components of the primate basal ganglia are the *striatum*, the *globus pallidus (GP)*, and the *subthalamic nucleus (STN)* in the forebrain, and the *substantia nigra (SN)* in the midbrain. The globus pallidus contains two separate areas which are termed the internal and external segments *(GPi* and *GPe).* Homologous structures (though often with different names) are found in the nervous systems of other vertebrate classes [3]. The BG are illustrated in a schematic drawing of a generalized mammalian brain in figure 1. The proposal that the BG performs action selection in the vertebrate brain is not a radical perspective on BG function but rather derives from a growing consensus that a key function of these structures is

to enable desired actions and to inhibit undesired, potentially competing, actions (see [2,4] for review). This literature suggests the following view of the functional architecture of the BG. Activity relating to 'bids' for access to common resources (e.g. muscle groups) appears to be continuously projected to the input side of the BG from relevant functional sub-systems in the midbrain and forebrain of the animal. This activity may form the 'common currency' in which the relative salience of competing requests can be effectively compared. Internal circuitry within the BG then determines a 'winner' whose contact with the output mechanisms is specifically disinhibited. The following briefly summarizes some of the key findings in support of this proposal, further details and supporting evidence are described in [2].

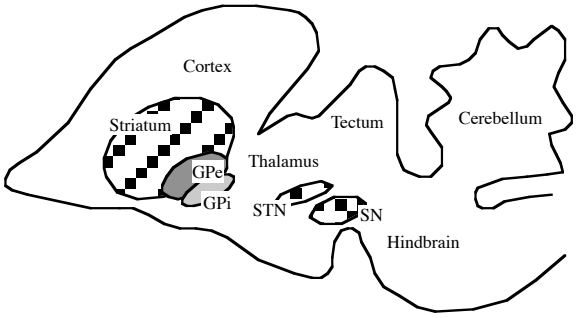

Figure 1: Schematic diagram of a saggital section through a generalized mammalian brain showing the principal BG structures, adapted from [3].

Anatomical evidence shows that cortical and midbrain sensorimotor systems, plus several of the forebrain limbic structures, communicate directly with motor and pre-motor mechanisms in the brainstem and spinal cord. However, these systems also project, usually via a collateral (split) pathway, to the striatum, the main input center of the BG (see figure 2), this branch could allow them to enter into a competition for control of motor outputs hosted within the BG. Afferents from a wide range of sensory and motivational systems also arrive at BG input neurons. These connections could allow both extrinsic and intrinsic factors to enter into a "vast machinery" of context-specific filters in the striatum [4], influencing the strength of rival bids, and hence the currently preferred course of action. The input connectivity of the BG therefore indicates that it is well placed to resolve the problem of selecting an appropriate action for a given circumstance.

The principal output structures of the BG are the SN and GPi. Neurons in both these structures are tonically active and inhibitory and project to all the different sensorimotor systems that are in contact with the striatum. This tonic inhibition acts as a brake on the target systems thereby denying them access motor circuitry. Signals emanating from the striatum inhibit the inhibitory BG output centers so *disinhibiting* selected systems (see [2, 4, 5] for review, and see figure 2 for an illustration of this double-inhibitory pathway). In the absence of such signals there can be no voluntary movement. The BG thus seems to hold a 'veto' over midbrain and forebrain systems that seek access to the motor outputs which is relinquished, for a selected action, through the mechanism of *disinhibition*.

Projection lines through the various sub-components of the basal ganglia appear to be largely organized into segregated parallel 'channels'. This segregation is maintained in the disinhibitory output projections. Behavioral studies indicate that although the architecture of these channels is similar throughout most of the BG, different areas are functionally heterogeneous. For instance, restricted lesions at different locations in the striatum effect different actions such as forelimb manipulation, biting and gait. This would suggest that the circuitry in these local areas in the striatum may primarily be used to resolve conflicts between competitors bidding for incompatible uses of specific groups of muscles. More generally, each local group of parallel circuits may be competing for a single output mechanism thereby forming a single, multi-way 'switch'. If this interpretation is correct then the BG may provide an array of similar switching devices.

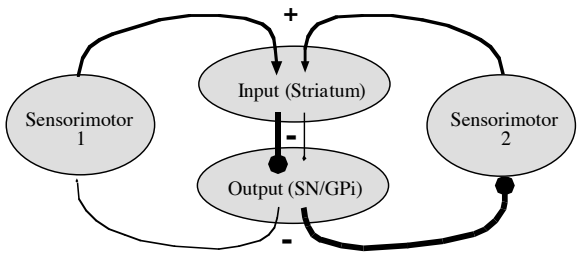

Figure 2: Functional diagram of the principal hypothesized selection mechanism. Sensorimotor systems project to the striatum, the main BG input structure. Intrinsic striatal circuitry resolves the selection competition in favor of the strongest competitors (here 1) and selectively inhibits neurons in SN/GPi switching off their tonic inhibitory control of winning sensorimotor systems whilst maintaining or increasing inhibition on losers (here 2). (Thicker lines indicate stronger excitatory or inhibitory links.)

In [2] we summarize evidence that various aspects of behavior selection and switching are effected by neurochemical or neurophysiological interventions in BG structures. The BG are also implicated in a number of human brain disorders including Parkinson's disease, Huntingdon's Disease, and Tourette's syndrome, whose symptoms may be interpretable as resulting from the failure, or inappropriate operation, of selection mechanisms. BG structures are important in instrumental conditioning and in various forms of sequential learning suggesting that they are appropriately designed for adaptive tuning of selection mechanisms. It has recently been suggested that the theory of temporal difference learning in actor-critic mechanisms could be used to understand the learning architectures embedded in the BG (see [6]). This suggests the prospect of a fruitful interaction between work on artificial reinforcement learning systems and the understanding of adaptive BG processes.

The BG have been implicated in a wide range of processes that includes aspects of motor control, perception, learning, and memory (see [2, 4, 6]). BG involvement in so many diverse functions suggests to us that it may play a similar function in multiple domains—that is, selecting between competitors that require access to some limited resource be it motor, cognitive, or memorial.

2 A new model of intrinsic basal ganglia function

A number of computational models of BG function, at both the cellular and circuit level, have been investigated (see [6]), however, there are as yet few models that capture the distinctive neurodynamics of BG circuits while mimicking their behavioral functions [5]. Our current research is directed at developing models of exactly this sort, and, as a first step, we have constructed a system-level simulation of the mechanisms operating within a single BG selection circuit.

There is a rich connectivity within the BG whose function is not clear. Our initial work has resulted in a simulation in which different functional components of the intrinsic BG circuit are modeled as leaky integrator units. Our investigations of the behavior and dynamical properties of this model are beginning to provided valuable insights into the possible role of each of the component pathways. A full quantitative description of the model and simulation results will be published elsewhere [7], here we briefly outline some of our principal findings.

As shown in figure 3, our model is composed of two functional subsystems, one which performs the selection process *per se* (the *selector* sub-system*),* and another which adaptively controls the former (the *adaptive controller).*

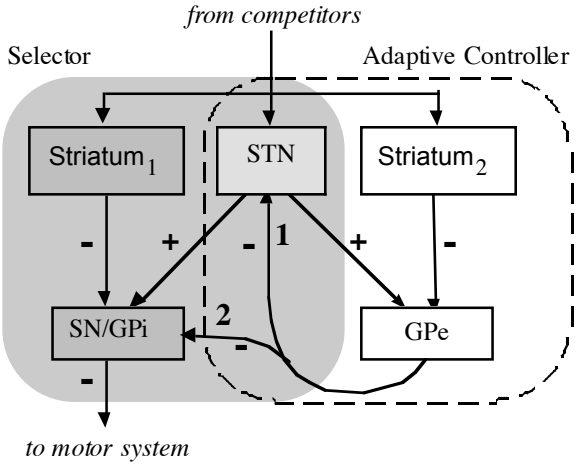

Figure 3: Functional model of the intrinsic circuitry of a single basal ganglia 'switch'. The gray area encloses the components of the *selector* subsystem, while the dotted line encloses the components of the *adaptive controller* subsystem (note the subthalamic nucleus belongs to both).

The selector sub-system resolves the competition for specific output mechanisms by providing off-centre, on-surround activation of SN/GPi (the BG output structures). Excitatory input to a population of neighbouring neurochemically defined striatal neurons (*striatum1* in the diagram) encodes the salience of competing bids. Activated striatal neurons directly inhibit neurons in SN/GPi thereby providing the off-centre effect. Through a second input pathway, the subthalamic nucleus (STN) provides diffuse excitation to SN/GPi. This pathway acts as the on-surround, ensuring the inhibition of losing and inactive competitors. Within the striatum the

contrast between stronger and weaker competitors is further enhanced through recurrent reciprocal inhibition.

The *adaptive controller* sub-system is structurally similar to the *selector* but is based around a different neurochemically-defined cell population in the striatum (*striatum2),* and provides an indirect link to BG output structures via the globus pallidus external segment (*GPe*). Two likely functions of this subsystem are, via pathway *1* in figure 3, to make the selection circuit robust to variation in the number of competitors and their relative levels of support; and, via pathway *2*, to act as a gain control on the output signal strength of competitors. A further function of the adaptive controller could be to enhance the high frequency response of the system thereby allowing faster switching.

Simulation results illustrating the operation of the switching mechanism and the effect of 'lesioning' pathway 1 are illustrated in figure 4. The top row of graphs (1-3) illustrates the normal operation of the BG switching mechanism (figure 3) for three model competitors with different 'salience' strengths and onset times. In each graph the solid line indicates the activity of the excitatory input to the striatum and STN (the salience signal), and the dotted line the activity of the inhibitory BG output from SN/GPi to the motor system. Competitors with output close to zero are selected, those with high output are suppressed. Competitor 1 (salience=0.6, onset=1) is activated first and is selected, 3 (salience=0.5, onset=2) fires next but has lower salience than 1 and is therefore not selected, 2 (salience=0.8, onset=3) is activated last but has the highest salience and is therefore selected while 1 and 3 are suppressed. The BG output signals indicate reasonably clean switching between competitors, in other words, the selection competition is resolved rapidly and decisively in favor of the strongest competitor. This simulation run is repeated in the bottom row of graphs (1*–3*) with the inhibitory GPe–STN link removed (pathway 1 in figure 3). Clean switching is compromised (winners are not effectively disinhibited) as the 'lesioned' network is inappropriately sensitive to the number of active competitors and their relative levels of support (see, for instance, the increase in inhibitory output to competitor 1 at t=2 when 3 becomes active).

Aside from input driven effects, the influence of the adaptive control pathway can be modified in the biological setting by changing levels of the neuromodulator dopamine. Dopamine enhances the response of striatal cells projecting directly to BG output structures *(striatum1)* while suppressing those that project indirectly via GPe *(striatum2),* it therefore appears to alter the balance between the different BG intrinsic pathways. This effect has been incorporated into the model and it appears that increased dopamine has the potential to make the current selection more vulnerable to alternative competitors; in effect dopamine is capable of dynamically modulating the sensitivity of the switch. Parkinson's disease is associated with abnormally low levels of striatal dopamine, simulating this deficit within a BG model such as our own could therefore improve our understanding this disorder.

The simulation has been the subject of mathematical analyses which make explicit its functional and parametric dependencies, and will facilitate comparisons with other models. The analytic approaches that we are currently using rely on approximating the nonlinear output characteristics of each subsystem in a piecewise-linear scheme. In the spirit of our system-level investigation, our focus is not on the

details of neural non-linearities but rather, in describing enough of the system's gross properties to capture its main consequences. This should allow an examination of the dynamics under small signal changes which, although of interest in its own right has a further significance within our general framework. Specifically, the transient characteristics of the signals generated by the model (e.g. the output traces in figure 4) can be thought of as a 'fingerprint' for the underlying architecture. Comparing this fingerprint with that indicated by the neurophysiological data should provide a further means for evaluating and refining our model.

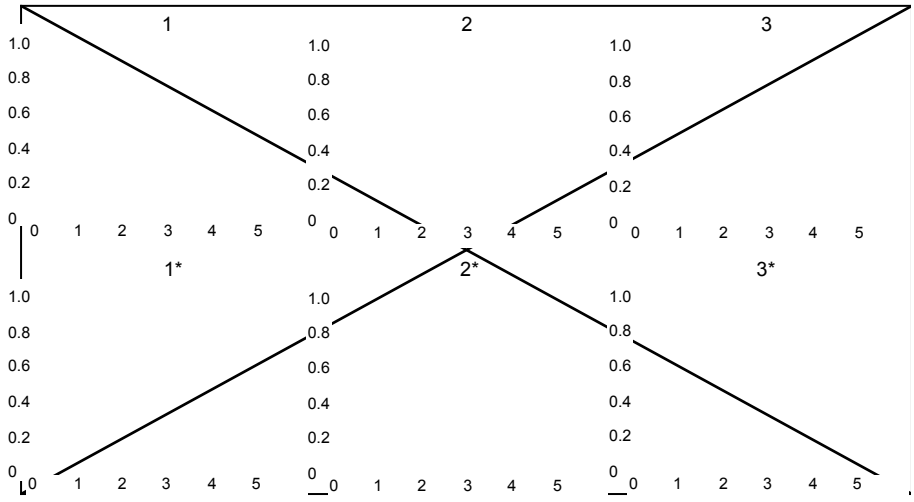

Figure 4: Simulation results. The solid line indicates excitatory input to the BG (salience) and the dotted line inhibitory BG output to the motor system. Neural response is constrained to lie between 0 and 1 on the y-axis. The time-scale on the x-axis is notional.

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