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**Book Section:**
Chambers, J.M., Gurney, K. orcid.org/0000-0003-4771-728X, Humphries, M. et al. (1 more author) (2011) Mechanisms of choice in the primate brain: a quick look at positive feedback. In: Seth, A.K., Prescott, T.J. and Bryson, J.J., (eds.) Modelling Natural Action Selection. Cambridge University Press , pp. 390-420. ISBN 9781107000490

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# Mechanisms of choice in the primate brain: a quick look at positive feedback

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## Abstract

The mammalian brain's decision mechanism may utilise a distributed network of positive feedback loops to integrate, over time, noisy sensory evidence for and against a particular choice. Such loops would mitigate the effects of noise and have the benefit of decoupling response size from the strength of evidence, which could assist animals in acting early at the first signs of opportunity or danger. This hypothesis is explored in the context of the sensorimotor control circuitry underlying eye movements, and in relation to the hypothesis that the basal ganglia serve as a central switch acting to control the competitive accumulation of sensory evidence in positive feedback loops representing alternative actions. Results, in support of these proposals, are presented from a systems-level computational model of the primate oculomotor control. This model is able to reproduce behavioural data relating strength of sensory evidence to response time and accuracy, while also demonstrating how the basal ganglia and related oculomotor circuitry might work together to manage the initiation, control and termination of the decision process over time.

## 0.1 Introduction

Whether it's a cheetah deciding whether its prey is veering left or right, a rabbit deciding whether that movement in the bushes is friend or foe, or a poker player wondering if his opponent has a stronger hand, infinitesimally small variations in sensory input can give rise to vastly different behavioural outcomes: the cheetah veers left and not right; the rabbit flees or continues grazing, the card player bets a month's salary or folds. The outcome of such decisions can be critical, even a matter of life or death, which is why there will have been tremendous evolutionary pressure to develop decision-making mechanisms that can extract maximal utility from limited sensory information. In this article, using the oculomotor system as an exemplar, we argue that the vertebrate basal ganglia are one of the results of that evolutionary pressure and explore how these structures tame and exploit positive feedback loops (henceforth PFBLs) within the brain in order to make the most of limited information.

In humans, the usual behavioural outcome arising from a change in our visual environment is that we reorient our gaze in order to investigate that change. Indeed, we typically make rapid, ballistic eye movements, termed saccades, two or three times per second. As one of the most frequent actions we perform, deciding where to look next is therefore one of the most common decisions we make.

We are all familiar with the idea of "taking our time" in order to make the right decision, but how long is long enough? Amid the convoluted anatomy of the primate oculomotor system one can discern a relatively short pathway from the retina to the superior colliculus (SC) and back to the extraocular muscles. The SC responds to visual stimulation in approximately 40ms and electrical stimulation of its deeper layers can trigger a saccade within 20ms (Wurtz and Goldberg, 1989). Consequently, this pathway could, in principle, initiate a saccade in response to a visual stimulus in 60 ms. However, in humans, visually triggered saccades are typically elicited with a response time (RT) of 200ms or more. It would seem then that the brain "takes its time" even when making this most common of decisions.

Curiously, the amount of time an individual takes to decide where to look next is highly variable. When a subject is asked to repeatedly saccade to a light appearing unpredictably in their peripheral vision, the distribution of their RTs is heavily skewed with the majority of responses beginning a few hundred milliseconds after stimulus onset but with a long tail of responses with some taking a second or more (Carpenter and Williams, 1995). Furthermore, the instructions given to a participant in such a study can dramatically affect this distribution. For instance, an emphasis on accuracy tends to shift the distribution towards longer response times, while an emphasis on speed has the opposite effect (Reddi and Carpenter, 2000). Not only are we able to adjust the length of time we take to react to externally cued events, we are, of course, also able to voluntarily move our eyes in order to achieve arbitrary goals, such as reading the words of this article. It would seem, therefore, that deciding where to look next is a non-trivial problem.

The diffusion model (Ratcliff, 1978) is an influential psychological model of decision making that can account for the brain's variable procrastination in reaching decisions. The model assumes that sensory evidence in favour of alternative responses is fundamentally noisy, whether due to the environment (e.g. tall grass obscuring a cheetah's prey), or due to random neural activity in the brain. Key to the model is the idea that the brain accumulates, or integrates, evidence over time in order to mitigate the effects of this noise, only making a decision when the difference in evidence for and against an action reaches a threshold level. The model is able to account for the skewed distribution of RTs obtained in saccadic studies and also provides insight into how the trade-off between speed and accuracy can be controlled by modifying the decision threshold. With a low threshold the model is able to make fast selections but is more likely to make errors due to noise. With a high threshold, the model integrates the evidence for longer and makes fewer errors since there is more time to average out the noise contribution.

Remarkably, under laboratory conditions the integration of evidence has indeed been observed in the brain. The stochastic motion discrimination task (Britten et al., 1993), presents a monkey with a situation not wholly dissimilar to that faced by the hypothetical cheetah described above. The animal is presented with a display containing moving dots, a proportion of which are moving left on some trials and right on others, while the remaining dots move randomly (thus providing environmental noise). The difficulty of the task can be varied by adjusting the motion strength i.e. by changing the relative number of dots moving coherently. The monkey is given a reward for correctly indicating in which direction the majority of dots are moving by making a saccade, in the same direction, to one of two targets flanking the dot display.

The medial temporal (MT) area of visual cortex is stimulated by this task as neurons in this area are highly sensitive to motion. More specifically, each MT neuron is responsive to motion in a particular direction so that their firing rate indicates the extent to which their preferred motion is present in the current visual scene. Consequently, on a trial in which the net flow of dots is to the left, MT neurons that are sensitive to leftward motion have an average firing rate that is higher than that of neurons sensitive to rightward motion (Britten et al., 1993). For the motion discrimination task, therefore, the noisy neural activity in area MT can be thought of as the evidence that the brain has available in order to decide where to look next.

The integration of area MT's evidence appears to occur in downstream oculomotor structures that are implicated in the planning and execution of saccades (Ditterich et al., 2003; Gold and Shadlen, 2007; Schall, 2001). For instance, in the lateral intra-parietal (LIP) area neurons that are able to trigger saccades to the left or right target exhibit a ramp-like build-up of activity as the animal observes the moving dots (Roitman and Shadlen, 2002; Shadlen and Newsome, 2001). Allowing the animal to respond at its own pace, Roitman and Shadlen (2002) demonstrated that both the accuracy and speed of decisions increases with motion strength and that this corresponds with a steeper rise of activity in those LIP neurons with motor fields centred on the target that the animal ultimately saccades to. LIP neurons corresponding to the alternative target also demonstrate an initial increase in firing rate but this is suppressed below baseline rates prior to saccade generation. Taken together, these findings support the idea that LIP neurons represent the accumulation of evidence for and against a particular saccade. This, and the finding that the decision process is completed when LIP neurons reach a threshold firing rate, suggests that the brain utilises a decision algorithm similar to the diffusion model.

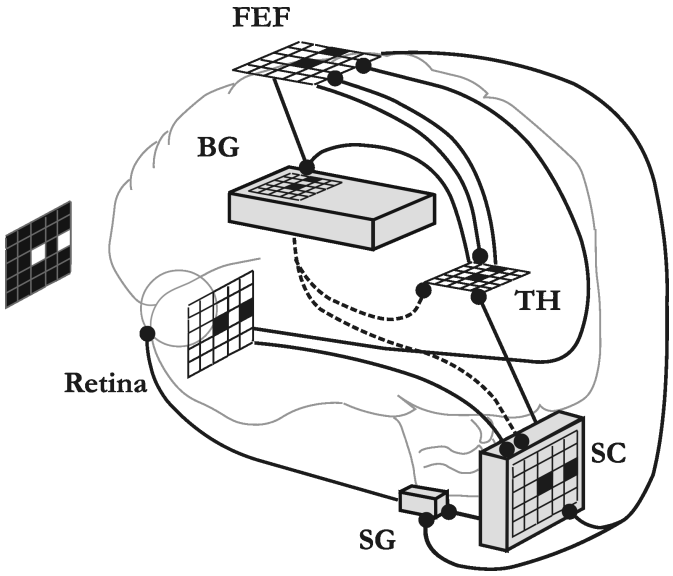


Figure 0.1 Brain areas forming the reactive oculomotor system. SC - superior colliculus; SG - saccadic generator; TH - thalamus; FEF - frontal eye fields; BG - basal ganglia. Solid and dashed lines denote excitatory and inhibitory projections respectively.

### 0.1.1 The oculomotor system

In order to explore how the circuitry of the brain implements a diffusion-model-like decision mechanism we now consider the anatomy of the oculomotor system of which a simplified circuit diagram is illustrated in figure 0.1. In particular, we focus on those areas that are known to be involved in the production of visually-guided saccades, as the model of the oculomotor system we present later is restricted to these areas.

#### The superior colliculus

Retinal ganglion cells project directly to the SC (Schiller and Malpeli, 1977), a multi-layered, midbrain structure, that preserves the spatial organisation of its retinal input. Figure 0.2 shows the basic connectivity of the SC as implemented in the model of Arai et al. (1994) (hereafter referred to as the Arai model) which we have incorporated into our own large-scale model (discussed in the methods section). The superficial layer of the SC relays its phasic retinal input to deeper motor layers, which in turn, send excitatory projections to a set of brainstem nuclei, collectively known as the saccadic generator (SG) circuit, which provide closed-loop control of the eye muscles (Sparks, 2002). The

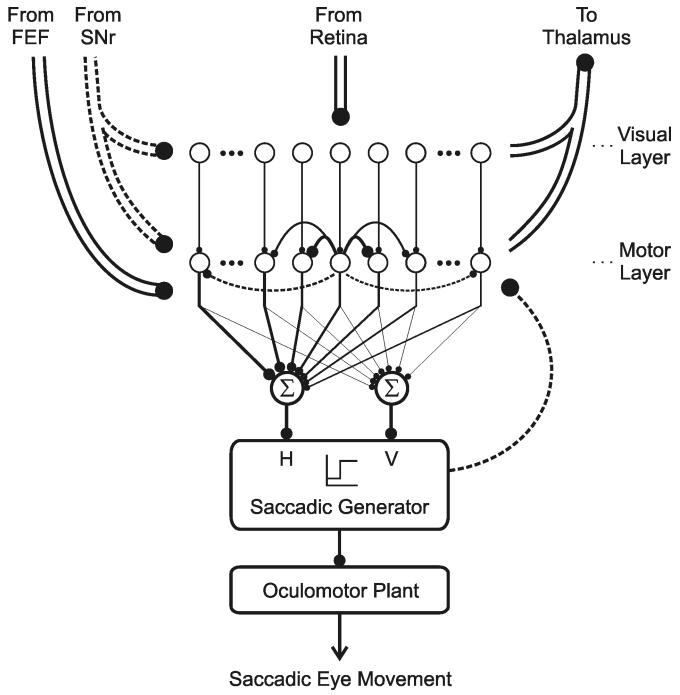


Figure 0.2 A model of the SC based on Arai et al. (1994). Solid and dashed lines denote excitatory and inhibitory projections respectively. Double-lines denote topographic projections. See text for description.

deeper layers of the SC also receive excitatory input from several frontal, visual, auditory, and somatosensory areas of cortex, so that saccades can be triggered voluntarily, or in response to processed visual features, localised noises, or physical contact with the body (Stein, 1993).

### The frontal eye field

Another important source of input to the SC comes from the frontal eye fields (FEF), an area of the frontal lobes implicated in saccade generation. In addition to projecting to the SC, the FEF also project directly to the SG so that a person or monkey with a SC lesion is still able to generate saccades. The FEF has reciprocal connections with both prefrontal and posterior cortices constituting the "where pathway" of visual processing (Ungerleider and Mishkin, 1982). The input it receives from the dorsolateral prefrontal cortex (DLPFC) is implicated in the generation of voluntary saccades, while that from posterior cortices, including LIP, relays information concerning the location of salient

visual targets. The nature of the processing that takes place in the "where pathway" is not important for our purposes (indeed, in our depiction of this circuit in figure 0.1 we have greatly simplified it by showing a direct connection between the retina and the FEF), other than to say that it preserves a retinotopic organisation throughout. The reciprocal connectivity between FEF and the posterior cortices suggests that FEF is both activated by the sensory information fed forward, and able to feedback the results of any frontal processing to those areas supplying the sensory information (see Cisek, this volume, for a similar proposal relating to the reach system). The evolution of build-up activity in LIP observed during the motion discrimination task could, therefore, be partially driven by the FEF.

### The saccadic generator

The inner workings of the SG are beyond the scope of this article, however, one important detail of its operation is key to understanding later discussions. Models of the SG invariably incorporate a population of neurons found in the nucleus raphe interpositus known as the omni-pause-neurons (OPNs) (Langer and Kaneko, 1990). These neurons derive their name from the fact that they exhibit a pause in baseline firing just prior to saccade generation. They are thought to actively inhibit the brainstem neurons that drive changes in eye position and as such OPNs represent the oculomotor system's final gateway, blocking saccades until they themselves are silenced. OPNs are indirectly inhibited by those areas of the SC and FEF which represent potential saccade targets, while they are excited by the foveal regions of these structures (Buttner-Ennever et al., 1999; Gandhi and Keller, 1997, 1999; Stanton et al., 1988; Segraves, 1992). The fixation and saccade regions of the SC and FEF therefore provide the SG with conflicting commands, these being "maintain fixation", and "saccade to a new location" respectively. It is likely then that the relative level of activity in the fixation and saccade regions of the SC determines which of these two behaviours is expressed. Correspondingly Munoz and Istvan (1998) have demonstrated that a decline in fixation activity is concomitant with the build up of target related activity in the SC. This finding suggests that competitive dynamics within the oculomotor system must suppress ongoing fixation activity before a saccade can be generated.

### The visuo-motor response

The visually-guided saccade task is one of the most common paradigms used to probe activity within the oculomotor system. For this task the animal is trained to maintain active fixation of a central stimulus and to then saccade

to a suddenly-appearing target stimulus in peripheral vision. Electrophysiological studies with primates have revealed that neurons in SC, FEF and LIP display remarkably similar patterns of activity during this task (Ferraina et al., 2002). Firstly, Neurons that represent the fovea show a tonic activation while the animal is maintaining fixation, and this appears to be largely endogenous in origin as it is not reliant on a fixation stimulus being present (Munoz and Wurtz, 1993). Secondly, neurons representing target coordinates display increases in activity that are time-locked to target stimulus onset, saccade onset or both - response classes that are respectively referred to as visual, motor or visuo-motor (Figure 0.3; Munoz and Wurtz, 1995).

When animals produce saccades with a short RT it is often hard to discern separate visual and motor peaks although careful analysis of the data reveals it to be present (Sparks et al., 2000). Experiments in which the animal must delay its saccade make distinct peaks much more apparent. Under the delay paradigm the motor component displays a steady build-up of activity not dissimilar to that observed in the motion discrimination task described earlier (Wurtz et al., 2001). Hanes and Schall (1996) demonstrated that the onset of the saccade is time-locked to the instant at which the motor activity in FEF reaches a threshold level, and similar thresholds have been found for LIP (Roitman and Shadlen, 2002) and SC (Pare and Hanes, 2003). Interestingly, as the build-up of motor activity continues towards threshold, there is a concomitant decrease in fixation activity. Recall that under the motion discrimination task it appears that decisions are only completed when there is sufficient difference between the elevated activity of the LIP neurons representing the chosen target and the suppressed activity of those representing the alternative target (Roitman and Shadlen, 2002). Similarly, under the visually-guided saccade paradigm where the animal is making a choice between maintaining fixation and saccading to the target stimulus it would seem that, just as for the motion discrimination task, there is a requirement for a sufficiently large difference in the activity representing the competing alternatives i.e. between fixation- and saccade-related activity. The oculomotor model we present later in this article incorporates the idea that OPNs are responsible for delaying action until this condition is met or, in other words, that the OPNs implement thresholding in the brain's evidence accumulation mechanism for saccadic eye movements.

### 0.1.2 Accumulation by Positive Feedback

Given that the motor component of the visuo-motor response appears to represent the active process of decision making and that it is observed throughout the oculomotor system, it is interesting to consider what neural circuitry un-

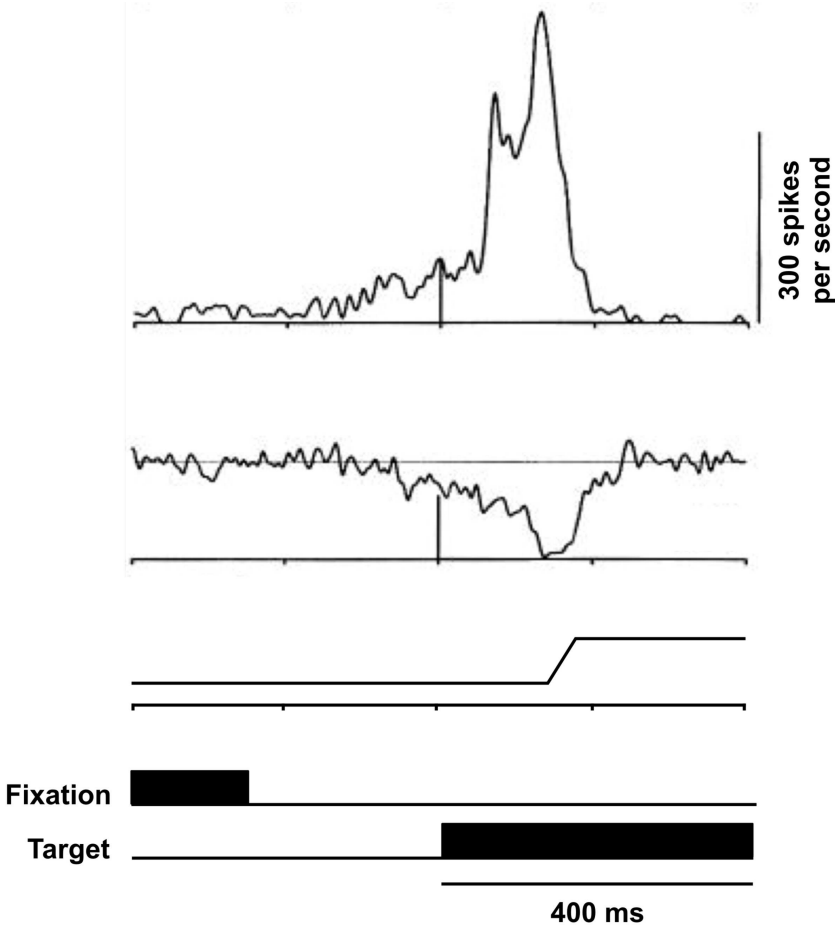


Figure 0.3 Typical target- and fixation-related activity in the intermediate layers of monkey SC recorded during a visually guided saccade. Top trace shows a clear bimodal visuo-motor response in SC motor layer. Middle trace shows fixation-related activity reducing as target-related activity builds-up. Bottom trace shows approximation of eye position for the same period. Data adapted from Munoz & Wurtz., 1995.

derlies it. Models seeking to address this question have largely concentrated on the cortical microcircuitry (Usher and McClelland, 2001; Ditterich et al., 2003; Wang, 2002). Of these, the model proposed by Wang (2002) provides the most biologically plausible account of how populations of cortical neurons might accurately integrate sensory evidence by exploiting recurrent excitatory connections between neighbouring neurons. Arai et al. (1994) also offered lo-

cal recurrent excitation as the most likely explanation for the build-up of motor activity observed in SC prior to a saccade. However, inspection of figure 0.1 reveals that the oculomotor system contains at least two additional positive feedback loops: SC-TH-FEF-SC, and FEF-TH-FEF (TH = thalamus) (Sommer and Wurtz, 2004; Haber and McFarland, 2001) formed *between* oculomotor areas. Given this interconnectivity, it seems likely that the build-up of motor activity observed throughout the oculomotor system arises through the combination of PFBLs formed between neighbouring neurons within each oculomotor area, and by PFBLs formed by the long-range projections between these areas.

To understand the way in which positive feedback can be used to perform integration, consider the block diagram shown in figure 0.4a which shows a simple rise-to-threshold mechanism with blocks $f$, $b$ and $m$, representing neural populations, which for the purpose of this discussion can be thought of as leaky integrators (Arbib, 2003), with an output limited to a minimum firing rate of zero, and a maximum of $y_{max}$. A salience signal $c$ representing the sensory and/or motivational "evidence" supporting an action, is fed into a closed loop formed by blocks $f$ and $b$, the output of which is passed to block $m$, which provides the motor signal $y^m$, that drives the action. Block $m$ also receives an inhibitory signal $\theta$ (assumed constant), which acts as a threshold to ensure that no action is produced until the output of the closed loop $y^f$ exceeds a critical value. This architecture is loosely based on the oculomotor system (as shown in figure 0.1), with the single loop formed by $f$ and $b$ representing the combined effect of SC-SC, SC-TH-FEF-SC, and FEF-TH-FEF loops, and $\theta$ representing the threshold effect of the omni-pause neurons in the saccadic generator circuit. Accordingly, the signal $\beta$ represents the inhibitory influence of the BG on these loops, the effect of which we shall consider shortly. We first consider the effect of the gains $w^{fb}$, and $w^{bf}$, which represent the synaptic weights of the projection from $f$ to $b$ and from $b$ to $f$ respectively. The closed loop gain $G$, of the sub-system formed by $f$ and $b$ is given by

$$G = w^{fb} w^{bf} \tag{0.1}$$

Figure 0.4b shows the response of the system in figure 0.4a, to a step change in salience of $\Delta c$, for different values of $G$. For $G > 1$, $y^f$ is unstable and grows exponentially before saturating at $y_{max}$, so that action is guaranteed provided the selection threshold $\theta$ is less than $y_{max}$. In this situation activity in the loop is self-sustaining, so that even when the salience signal returns to zero, the output of $f$ remains saturated. For $G < 1$, $y^f$ is stable and has an equivalent open-loop gain of $1/(1-G)$, so that the final value of $y^m$ is not guaranteed to reach saturation, but instead depends on the size of the salience signal $c$. Under

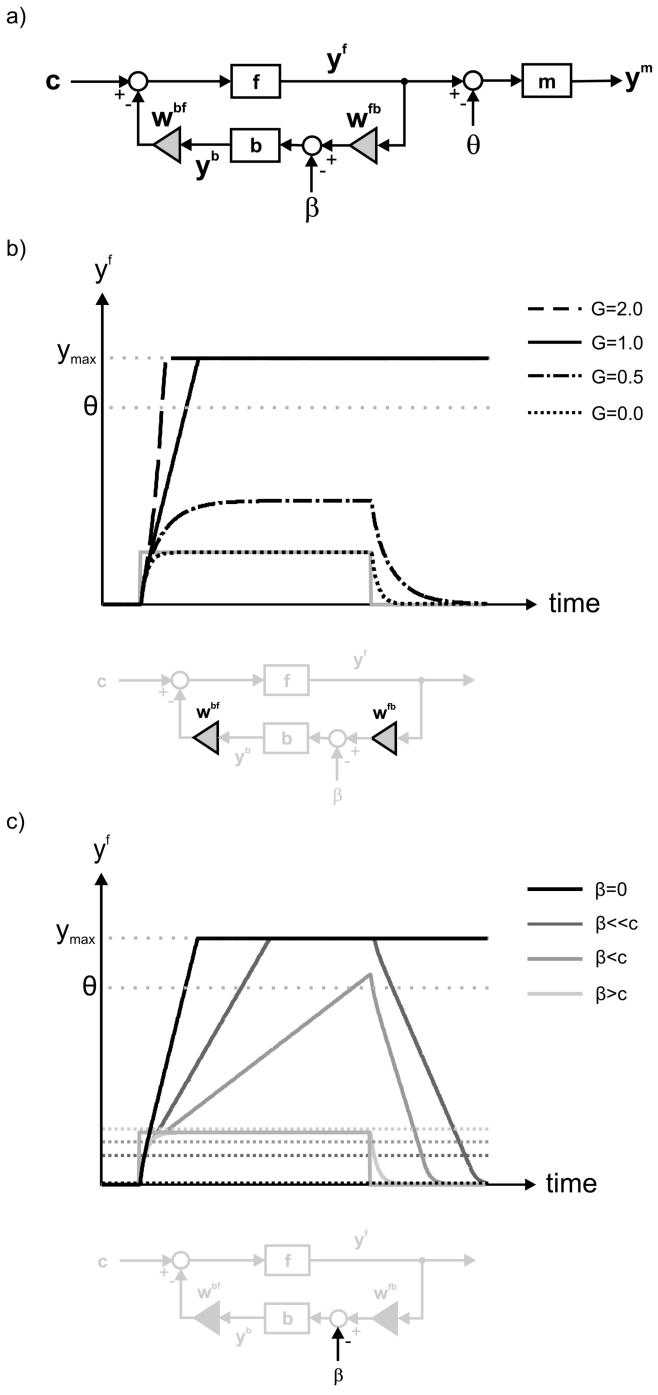


Figure 0.4 a) A simple behavioural control system incorporating positive feedback. b) The effect of varying the closed loop gain $G$. dashed line: G=2; solid line: G=1; dash-dot line: G=0.5; dotted line: G=0. c) The effect of varying the level of loop inhibition $\beta$. dashed line: $\beta = 0$; solid line: $\beta \ll \Delta c$; dash-dot line: $\beta < \Delta c$; dotted line: $\beta \geq \Delta c$. See text for details.

this condition, the output of $f$ tracks the salience signal, returning to zero when the salience signal does so.

With $G = 1$ the model exhibits the interesting behaviour of marginal stability, for which $y^f$ increases linearly [1] before reaching saturation. Recall that the diffusion model requires the temporal integration of evidence. With $G = 1$ this system approximates an ideal integrator and, as such, represents a way in which a pair of neurons, whose membrane voltages decay on a millisecond timescale, might accurately integrate information over the hundreds of milliseconds typically taken to make decisions. The circuit also makes clear another potential benefit that positive feedback can add to a selection system, namely the ability to raise weak sensory (and motivational) salience signals to the level required to elicit action. Unchecked, this amplification will cause even the weakest of salience signals to trigger its corresponding behaviour, so that a system like this will seldom be at rest. This may upon first consideration sound rather inefficient, however, ethological models suggest such a scheme underlies animal behaviour. As Roeder (1975) points out:

> animals are usually 'doing something' during most of their waking hours, especially when in good health and under optimal conditions.

One potential benefit that arises from this tendency to act, is that problems are dealt with before they become unmanageable. For instance, in the absence of any other deficits, a mildly hungry animal will set about finding, and consuming food, thus ensuring that its hunger is sated before its energy levels become dangerously low. Accordingly, McFarland (1971) has shown that a hypothetical model of action selection incorporating positive feedback, is able to account for animal feeding patterns. The oculomotor system could also be described as being unnecessarily active, however, orienting towards even weakly salient objects might provide an animal with an unexpected opportunity, or give it sufficient forewarning to avoid impending danger. By guaranteeing that motor signals reach saturation, positive feedback also acts to decouple the magnitude of a response from the magnitude of the salience signal driving it so that, for instance, a saccade's metrics (e.g., speed, duration) are largely independent from the properties of the stimulus that triggered it.

### 0.1.3 Competition in the oculomotor system

Much of the research into the neurobiology of decision making has focussed on LIP and recent models of decision making are consistent with the idea that this area is responsible for decision making. Under these proposals populations

[1] after fast transients related to the neural time constant have settled

of neurons representing alternative actions compete with each other through mutual- (Usher and McClelland, 2001), feed-forward- (Ditterich et al., 2003) or pooled-inhibition (Wang, 2002). Despite these architectural differences it has been demonstrated empirically (Ratcliff and Smith, 2004) and analytically (Bogacz et al., 2006) that all three architectures can implement the diffusion process if appropriate parameters are selected.

Despite this, there is reason to suspect that LIP is not the sole seat of oculomotor decision making. As described in our review of the oculomotor system above, the ramp-like rise to threshold of motor activity observed in LIP is also observed within the FEF and SC, two areas that, like LIP, receive input from areas of extrastriate cortex (including area MT), and are able to elicit saccades. Lesions studies have revealed considerable redundancy amongst these structures. LIP lesions having relatively little effect on oculomotor function (Li et al., 1999). Lesions to either FEF or SC produce more profound deficits (Dias and Segraves, 1999; Schiller and Chou, 1998) but only a dual lesions of both FEF and SC can cause a permeant loss of function (Schiller et al., 1980).

One possible interpretation of this apparent redundancy is that each of the oculomotor areas has some intrinsic capacity for action selection. More specifically, it may be the case that, as has been suggested for LIP, both FEF and SC have a local micro-circuit capable of independently implementing the diffusion process. If oculomotor decisions are computed in this distributed fashion then it would suggest that participating structures must coordinate with each other in order to ensure that conflicting motor commands are not issued to the brainstem.

An alternative interpretation the oculomotor system's redundancy is that decisions are not computed in a distributed fashion but, rather, centrally by a dedicated selection mechanism. Redgrave et al. (1999) have argued that a centralised architecture is superior to a distributed architecture in terms of connectivity and metabolic efficiency.

To understand why this is the case consider, for instance, the mutual inhibition model of (Usher and McClelland, 2001). In this model, neurons representing saccades to alternative locations compete with each other via reciprocal inhibitory connections. While there is certainly evidence of reciprocal inhibition within cortex (Windhorst, 1996), if neurons representing saccades to all visual coordinates are to compete with each other, then neurons in every part of the retinotopic map in LIP would have to be connected to those in every other part. Evidence for sufficiently long-range inhibitory connectivity is lacking, and this is perhaps unsurprising given that such many-to-many connectivity would be a costly method of facilitating competition in terms of developmental overhead and metabolic consumption. This cost is compounded if similar connectivity

is also necessary within FEF and SC, as it would presumably have to be under a distributed selection architecture. For these reasons we feel it is unlikely that the brain implements selection in this distributed fashion.

Redgrave et al. (1999) suggested that the basal ganglia might constitute a centralised selection mechanism that offers a more efficient method of selecting between alternative actions. Under this proposal, structures which generate potentially conflicting motor commands send "bids for action" to a central arbitrator, which chooses amongst them and signals this choice back to the bidding structures. The idea that the BG are involved in action selection is a recurring theme in the literature (Mink, 1996; Kropotov and Etlinger, 1999) and forms the basis of a unifying hypothesis of BG function that incorporates known anatomy and physiology (Prescott et al., 1999; Redgrave et al., 1999).

Anatomical and functional evidence also support this role for the BG within the oculomotor system. FEF and TH both project to the input nuclei of the BG with retinotopic projections (Hikosaka et al., 2000; Harting et al., 2001), so that the SC-SC, SC-TH-FEF-SC, and FEF-TH-FEF positive feedback loops identified earlier can each provide either direct or indirect bids to the BG. Also, the substantia nigra pars reticulata (SNr) - one of the output nuclei of the BG - provides strong tonic inhibition to TH and SC so that the BG can impose choices upon the same positive feedback loops. Indeed, this inhibitory output is known to pause prior to saccade initiation (Hikosaka et al., 2000) suggesting that the BG are acting to gate the build-up of saccade-related activity within the oculomotor system.

Having established that connectivity between the BG and oculomotor structures conforms to the expectations of a centralised selection scheme, we now consider the computation performed by the BG. Gurney et al. (2001) suggest that the intrinsic connectivity of the BG implements a form of feed-forward selection network. Figure 0.5 shows their computational model (hereafter referred to as the Gurney model), and provides a description of how intrinsic BG processing achieves signal selection. A key assumption is that the topography of BG inputs is preserved throughout the BG nuclei so that competing actions are represented by activity in distinct channels. The extent to which a channel is selected is determined by the difference between its input salience and the sum of all other input saliences. The calculation takes place in SNr, where diffuse excitatory input from the sub-thalamic nucleus (STN) effectively provides the sum of channel activity, and focused inhibitory input from D1 striatal cells provides a measure of individual channel activity. The diffuse STN projection allows inter-channel communication, so that input to a given BG channel acts to raise the level of inhibition outputted from all other channels. Thus, the growth rate of motor activity in a BG controlled PFBL, will depend not only

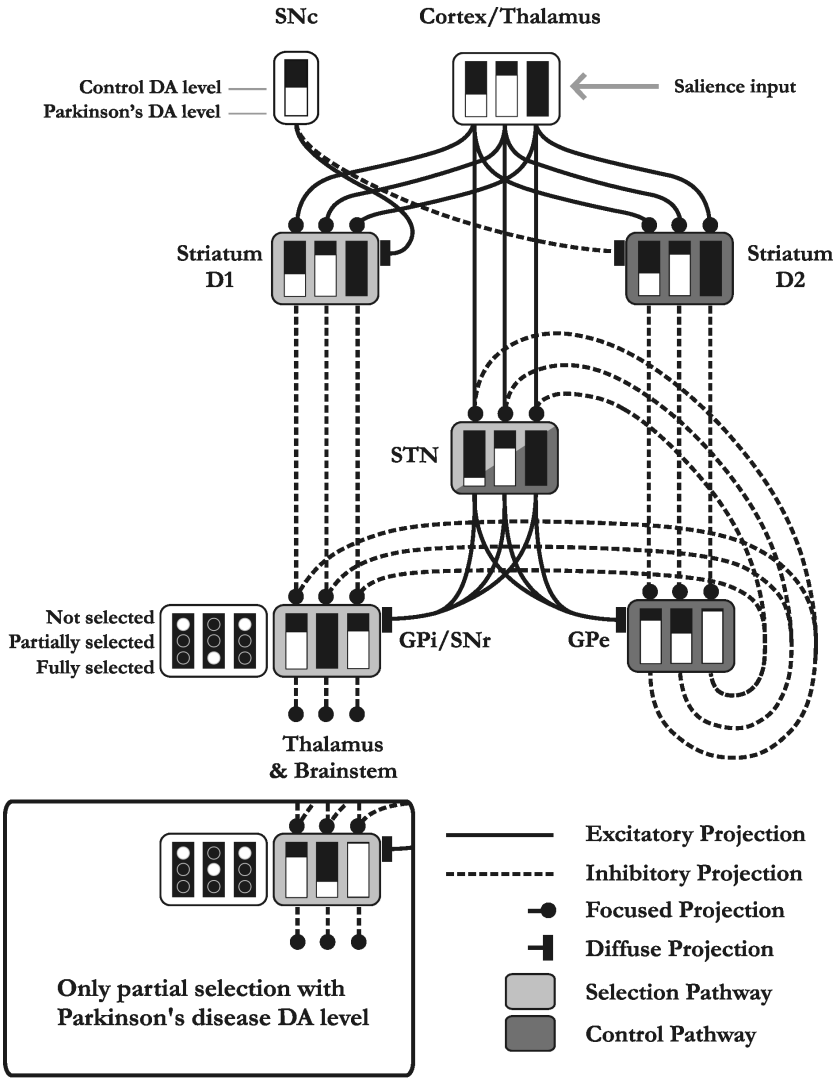


Figure 0.5 The intrinsic BG model of Gurney et al.[2001b], assumes that duplicate salience input is sent to the sub-thalamic nucleus (STN) and striatum, which is further sub-divided in two groups of cells classified by the type of dopamine (DA) receptor they express (D1 and D2). The globus pallidus internal segment (GPi) and substantia nigra pars reticulata (SNr) - which together form the output nuclei of the BG - send inhibitory projections back to thalamus and to motor nuclei in the brainstem (e.g., the SC). Spontaneous, tonic activity in the STN guarantees that this output is active by default, so that all motor systems are blocked. Gurney et al., identify two separate functional pathways within the BG. The selection pathway is responsible for disinhibiting salient actions: salience input to a channel activates D1, which then inhibits GPi/SNr thus silencing inhibitory output in the channel. The diffuse projection from STN to GPi/SNr means that all channels receive an increased excitatory drive. This is offset in the most active channel by the inhibitory input from D1, but goes unchecked in less active channels thus acting to block unwanted actions. The control pathway defined by Gurney et al., incorporates the globus pallidus external segment (GPe), and provides capacity-scaling by ensuring that STN activity does not become excessively high when multiple channels have non-zero salience, thus assuring full disinhibition of the winning channel irrespective of the number of competing channels. Because the striatal input to the control and selection pathways utilise different DA receptors, changes in tonic DA levels affect them differentially. Consequently, when DA is reduced to PD-like levels, the balance between the two pathways is disturbed resulting in residual inhibition on the selected channel (inset).

on the sensory input driving it, but also on the activity in other BG controlled loops. So that, for instance, in the visually-guided saccade paradigm, activity in a loop corresponding to the fixation coordinate will affect activity in a loop corresponding to the target coordinate.

Returning to the simple model shown in figure 0.4a, we now consider the effect of the inhibitory input $\beta$, which represents the effect of SNr inhibition upon oculomotor PFBLs. Figure 0.4c shows the response of the system to a step change in salience of $\Delta c$, for different values of $\beta$ and with the loop weights set to give ideal integration ($G = 1$). When the inhibitory input to the loop is greater or equal to the salience signal i.e., $\beta \geq \Delta c$, the positive feedback is effectively disabled because the input to $b$ is zero or less. Consequently, the system behaves like a first order system, with its output settling at the level of its input. Under these circumstances, action is not guaranteed and will depend upon the magnitude of the salience signal $c$. For $\beta < \Delta c$ the feedback becomes active as soon as $y^f$ exceeds $\beta$, causing a linear increase in $y^f$ with a rate determined by the difference $\Delta c - \beta$, thus guaranteing that $y^m$ reaches $y_{max}$, and overcomes the selection threshold. The inhibitory input also provides a means of overcoming the self-sustaining property of the loop, causing activity to decays linearly at a rate, again determined by $\Delta c - \beta$, when the salience signal returns to zero. From this it is clear that $\beta$ acts as both a threshold for activation of the PFBL, and a rate controller for the evolution of activity in the loop. Or, in other words, $\beta$ determines whether "evidence accumulation" is initiated, is able to scale the rate of accumulation, and can help passively terminate the accumulation process by removing accumulated evidence.

Having explored the properties of a single PFBL under inhibitory control, the remainder of this chapter examines the behaviour of multiple PFBLs in the context of a computational model of the primate oculomotor system. As pictured in figure 0.1, this system can be thought of as a set of parallel loops, like those in figure 0.4, each one corresponding to a different spatial coordinate. A key difference, according to the approach taken here, is that each loop's $\beta$ input is determined by the competitive dynamics of the BG. The model described below is a revised version of the oculomotor system model proposed by Chambers (2007) and hereafter referred to as the Chambers model. This model was previously shown to be able to reproduce data from several visually-guided experimental paradigms. Here we will demonstrate that a simplified version of the model can also reproduce data from a "noisy" two-alternative, forced-choice task similar to the motion discrimination task reviewed earlier. Specifically, we will demonstrate that the model is able to reproduce appropriate RT distributions and error rates, and demonstrate a relationship between RT and

the strength of sensory evidence relative to noise levels. We will also show that plasticity within the BG could provide a means of adaptively controlling the accumulation process. Before presenting these results we provide a brief overview of the original model (see Chambers, 2007, for a full description) together with details of the modifications made for the purposes of the current study.

## 0.2 Methods

The Chambers model simulates, from perception to action, the full sensorimotor competency of visually guided saccade generation. More specifically, the model simulates an experimental display, the retina, the SC (based on the model of Arai et al., 1994) (figure 0.2), FEF, TH, the BG (based on the model of Gurney et al., 2001)(figure 0.5), the SG (based on the model of Gancarz and Grossberg, 1998), and the eyeball and its musculature.

The model explicitly tests the "central switch" hypothesis of Redgrave et al. (1999) as the BG is the only structure in the model able to inhibit the build-up of motor activity i.e. reciprocal inhibition within cortex and the SC is not modelled. The BG is modelled using a 2-dimensional version of the Gurney et al. model (described in the preceding section), which receives one-to-one excitatory projections from FEF and TH and sends a one-to-one inhibitory projection to SC and back to TH.

The model also tests the hypothesis that a distributed network of PFBLs acts in concert to integrate evidence for and against specific saccades. Local positive feedback is modelled within SC via reciprocal excitatory connectivity between neighbouring neurons (with weights reducing with distance as modelled by Arai et al., 1994; Arai and Keller, 2005), long-range positive feedback loops are modelled by one-to-one projections between SC, TH, and FEF.

The Chambers model also tests the idea that the SG is involved in managing the accumulation process. The model incorporates the biologically-plausible model of the SG proposed by Gancarz and Grossberg (1998), which converts the spatially distributed representation of a saccade target, as found in SC and FEF, into the appropriate temporal signals necessary to drive the extra-ocular musculature. In the Chambers model the activity of the OPNs is determined by activity in the FEF and SC: foveal activity acts to increase OPN output, thus preventing saccades, while activity in the periphery inhibits the OPNs thus facilitating saccade generation. Under this interpretation of the anatomy, the OPNs are therefore ultimately responsible for setting the threshold for action within the oculomotor system.

Finally, the Chambers model also incorporates evidence suggesting that the SG provides negative feedback to the SC as a saccade is generated (Soetedjo et al., 2002; Goossens and Van Opstal, 2000). It is suggested that this inhibitory signal provides a means of actively resetting the decision mechanism by removing previously accumulated evidence.

For the current study, several changes were made to the Chambers model in order to simplify its interpretation and speed its execution time. First, the original model reproduced the log-polar representation of visual space found throughout the oculomotor system. Here we have removed this and simply represent visual space linearly in order to simplify the interpretation of results. Second, the Gancarz and Grossberg (1998) model of the SG was not included. Instead, we approximate the behaviour of this model by taking the centroid of combined SC and FEF activity just prior to saccade generation. Furthermore, we assume that saccades are made instantaneously, thus removing the need to simulate the dynamics of the eyeball. One aspect of the Gancarz model that is retained, however, is the inclusion of OPNs. These are modelled as a single leaky integrator excited and inhibited respectively by the foveal, and peripheral representations of FEF and SC. The centroid of saccade-related activity is sampled, and a saccade generated, when OPN activity reaches zero, and is not sampled again for a simulated refractory period of 100 ms (this prevents the eye from being continually repositioned despite the OPN remaining at zero for a short duration). At the same time that the centroid is taken, an inhibitory signal is injected into the build-up layer of the SC, simulating the feedback from the SG to the SC.

It has previously been demonstrated that the Chambers model can reproduce behavioural data from several visually-guided experimental paradigms (Chambers, 2007). In this chapter we seek to demonstrate that the model can also reproduce data from a "noisy" two-alternative, forced-choice task similar to the motion discrimination task reviewed earlier. It was not possible to test the oculomotor model with the motion discrimination task as it lacks a representation of area MT and is, as a consequence, unable to simulate tasks that require the subject to make a discrimination based on stimulus motion.

We instead simulate an alternative paradigm which requires the subject to make a discrimination based on stimulus luminance. Under this paradigm, which has been utilised by Ludwig et al. (2005), the subject is first presented with a central fixation stimulus, which is abruptly extinguished and replaced with two spatially separated target stimuli. The subject is required to saccade to the brighter of the two targets. The luminance of the targets varies randomly over the course of the trial, with values being drawn from a normal distribution. The distributions used for each target have the same variance but different

means. Task difficulty can be adjusted by altering the difference in mean luminance relative to the power of the noise or, in other words, by adjusting the signal to noise ratio.

In order to explore the selection capabilities of this system and its similarity to the diffusion model, we investigate its behaviour under 4 conditions:

- the control condition: the dimmer target has a mean luminance that is 95% that of the brighter target
- a high luminance condition: the luminance of both targets is increased from the control value by 10%
- a high contrast condition: the luminance of the brighter target is increased from the control value so that the dimmer target has a luminance that is 90% that of the brighter target
- a low weight condition: the targets have the same mean luminance as the control condition, but the model's cortico- and thalamo-striatal weights are reduced to 90% of their control value

We simulate the luminance discrimination task by generating a 2-dimensional array that represents the world, a sub-region of which is inputted into a retinal model. The sub-region that is sampled depends on the current simulated eye position, which is initially set to be at the centre of the world-array where the fixation stimulus is also located. The retinal model is a two layer network, with one layer that responds phasically to luminance increases and one which produces a tonic output proportional to luminance level. Both retinal layers project to the FEF layer, while only the phasic layer projects to the superficial layer of SC (which in turn relays that input to the deep layer of SC). These projections both introduce a 50ms delay to simulate delays introduced by retinal processing and axonal propagation.

A random number generator provides input into the FEF layer of the model in order to simulate the combined effects of environmental and neural noise. This noise source is temporally filtered using a low pass Butterworth filter in order to decouple the power spectrum of the noise from the simulation frequency of the model.

Each experimental condition is simulated for 400 trials with a sampling frequency of 400 Hz. A trial consists of 2 seconds of simulated activity. The fixation stimulus is presented from 50ms to 600ms, and is then exchanged for the target stimuli which remains on for the remainder of the trial (figure 0.6a).

In each trial, if the endpoint of a generated saccade is within +/- $2^o$ of the target with the higher mean luminance it is considered to be a correct response. Trials producing saccades that land elsewhere, or that fail to produce a saccade at all, are considered to have produced incorrect responses.

## 0.3 Results

### 0.3.1 Accumulation dynamics in the oculomotor model

We first review the operation of the model during a single trial of the luminance discrimination task in order to highlight what each part of the modelled anatomy contributes to the decision process. Figure 0.6 shows typical model activity during a trial conducted under the control condition. This trial highlights the effect of noise on the decision process as the model erroneously selects the target with the lower mean luminance.

#### Initiation

Figure 0.6 shows activity in a sub-set of model layers from 200ms prior to target onset. There are a number of things to note about this period, and that immediately following target onset. First, as can be seen from figures 0.6b and c, noise in the target channels prior to target onset is not integrated thus ensuring that the system does not make spontaneous saccades in response to noise. This resistance to noise is due to the inhibitory output from SNr acting upon TH and SC, which ensures that the net input to these structures is negative thus preventing positive feedback dynamics from being initiated by the SC-SC, FEF-TH-FEF and SC-TH-FEF-SC PFBLs upon which the model's accumulation dynamics rely.

Recall that the Gurney et al. (2001) BG model generates a baseline inhibitory output in the absence of salient input so that, in effect, downstream structures have a brake applied by default. Also recall that, when the BG model does have salient input, the level of SNr output is increased in losing channels (figure 0.5). Prior to the target onset the BG selects the fixation channel (as this is the only channel with external input) and, as a consequence, all other channels, including the target channels, receive above baseline inhibition. This selection is evident from the slight reduction in SNr activity in the fixation channel (figure 0.6d) relative to that in the target channels (figures 0.6b and c). The reduction in fixation channel SNr activity is relatively small owing to a manipulation we made to simulate the influence of prefrontal cortex upon the oculomotor BG. We explain this in the following section.

At 650ms into the trial, retinal input corresponding to target onset reaches the FEF layer (figures 0.6b and c) injecting a "pulse-step" waveform of input into FEF and thus the model's system of PFBLs. The initial phasic burst is sufficiently large to overcome the effect of SNr inhibition on TH and SC, thus allowing the accumulation dynamic to be initiated. The "pulse" also acts to rapidly establish a "beach-head" of accumulated evidence in the system's

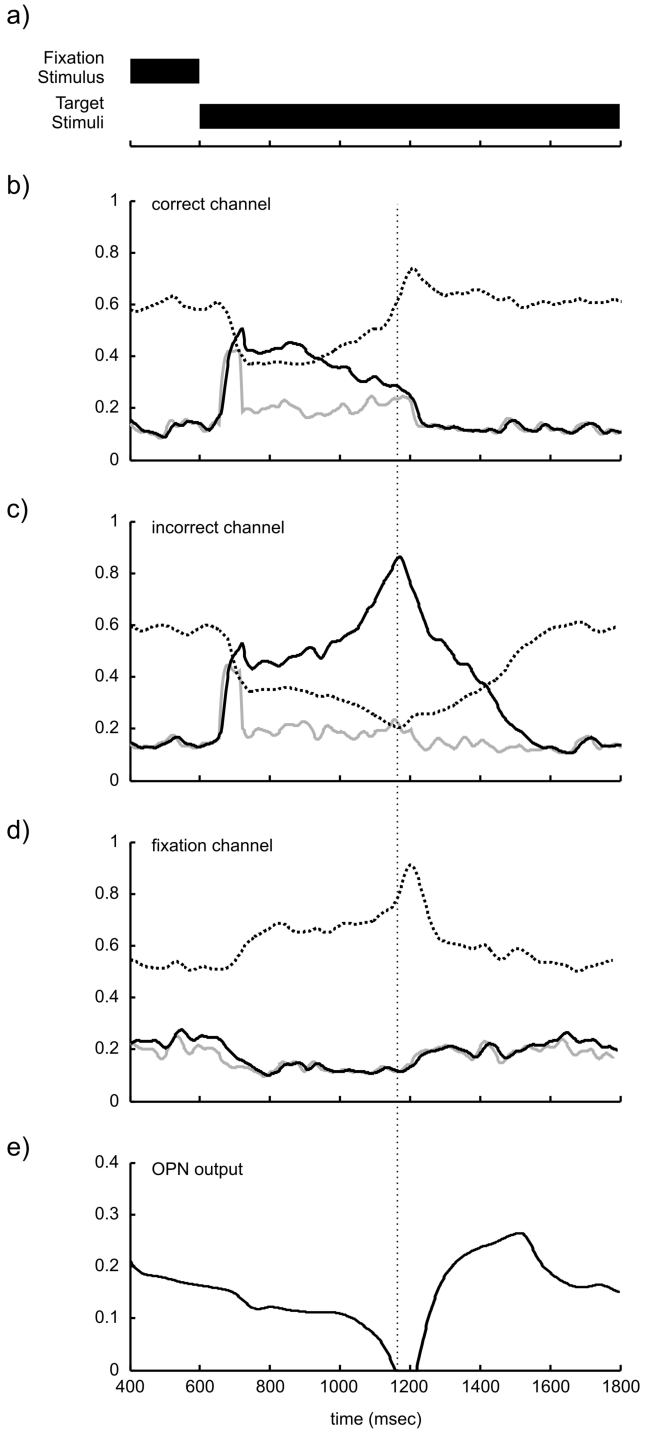


Figure 0.6 Model activity for a trial resulting in the selection of the incorrect target under the control condition. a) Experimental timing; fixation onset occurred 350ms prior to the period shown; fixation offset and dual target onset was simultaneous; b) output of neurons within the channel corresponding to the correct target - that is neurons with a receptive field centred on the correct target; for this and the following 2 panels, the solid and broken black lines correspond to output from FEF, and SNr respectively; the grey line corresponds to sensory input supplied to FEF i.e. the combination of retinal input and the noise source; c) neural output from the incorrect target channel; d) neural output from fixation channel; e) output from the OPNs. The vertical dropline denotes the instant at which a saccade to the incorrect target was initiated.

PFBLs which subsequently feeds into the BG and causes a corresponding reduction in SNr activity thus enabling the integration of the "step" over time.

One interpretation of this scheme, is that the burst of activity generated by stimulus onset is a form of interrupt signal, signalling that there is a stimulus that may warrant breaking from ongoing fixation. With this interpretation in mind, it is interesting to note that when a predictable stimulus onset acts to distract an animal from obtaining reward on a saccadic task, the corresponding phasic burst of activity (as observed in superficial SC) becomes attenuated over the course of several trials (Goldberg and Wurtz, 1972). It is likely that this attenuation prevents the accumulation of sensory evidence corresponding to the distracter, thus diminishing its ability to trigger a saccade. Later we will propose that the BG, in conjunction with prefrontal cortex, may be involved in this pre-attentive habituation.

**Competitive accumulation**

Under the control condition, the contrast between targets is low compared to the level of noise. As a result of this the accumulated activity in each target's channel is very similar post target onset (figures 0.6b and c). Correspondingly, the level of SNr activity applied to each channel is also similar, so that the BG grant neither channel a significant advantage over the other. Initially then, the rate of accumulation is mainly dependent on the strength of evidence for each target, as provided by tonic retinal input. However, as a result of the noise in the system, accumulated evidence in favour of the incorrect target takes an early lead in the particular trial shown in the figure. The BG circuitry responds to this increase in the incorrect target's channel activity by reducing SNr activity further for this channel, while increasing that to all other channels.

As a result of the change in relative SNr activity the BG imposes a bias on the accumulation process that favours evidence in the leading channel over that of losing channels. In other words, the system shows a primacy effect, favouring early evidence over that which comes later. In the trial shown, the accumulated evidence in the correct target's channel, despite having the higher mean input, is not able to overtake that in the incorrect target's channel which leads to an increase in this BG mediated bias. At approximately 950ms the SNr input to the correct target's channel increases to such a level that the net input to that channel's accumulator circuit is negative, thus causing activity therein to decline. Conversely, SNr input to the incorrect target's channel continues to decrease, accelerating the rate of evidence accumulation in favour of making an erroneous saccade. This separation of signals is consistent with the findings of Roitman and Shadlen (2002) using the motion discrimination task. In the following sections we suggest that the increase in decision signal contrast that

results from this separation is key to both the correct programming of saccadic movements and to facilitating rapid learning within the BG.

The BG mediated competition between channels is also able to account for the mutual exclusivity between target and fixation activity reported by Roitman and Shadlen (2002). Prior to target onset, the fixation channel is the most active channel although, as fig 0.6d shows, the accumulation dynamic has not significantly amplified the input signal in this channel. The model was manipulated in order to prevent accumulation in the fixation channel, as early experiments, in which buildup was permitted, produced unrealistically prolonged RTs because residual fixation activity competed strongly with burgeoning target activity. Accumulation was prevented by providing additional drive to the fixation channel's D2 pathway in BG, which has the effect of increasing SNr output for that channel. This is consistent with earlier work (Chambers and Gurney, 2008) which demonstrated a mechanism via which associative areas of PFC might manipulate the behaviour of motor systems by top-down inputs to motor striatum. Our manipulation therefore represents the effect of frontal associative systems having learnt to restrict the effective salience of fixation stimuli relative to novel peripheral onsets.

**Selection, enaction and accumulator reset**

In our interpretation of the oculomotor anatomy, the OPNs represent the final barrier to action and, thus, indirectly determine when the decision process is over. Figure 0.6e shows the activity in the OPNs over the course of the trial. Recall that fixation activity in FEF and SC excites the OPNs while target-related activity inhibits them. At 1150ms the sum of target-related activity is sufficiently large compared to fixation activity that the OPNs are silenced and the saccade generation process is commenced.

The SG model generates a saccade to the centroid of summated FEF and SC activity (approximating the behaviour of the more biologically plausible Gancarz model) and so it is critical to accurate target acquisition that the BG competitive dynamics produce a clear peak of accumulator activity centred on the chosen target, while suppressing activity elsewhere as seen in figures 0.6b and c. We model saccades as an instantaneous shift in eye position. The visual consequence of this shift is that the target stimuli move to different locations on the retina. For the trial shown, the saccade was accurate and so the incorrect target is now at the centre of the retina.

Post-selection it is critical that accumulated evidence is removed otherwise the system will continue to generate a staircase of saccades with the same relative displacement as the first (an effect that is observed when SC is driven continuously by micro-stimulation; Breznen et al., 1996). As fig 0.6c shows,

activity in the selected channel does indeed begin to decay after saccade generation. The model discards accumulated evidence both passively and actively. As the simple system shown in figure 0.4c shows, removal of excitatory input can lead to passive decay of activity. An eye movement moves the target stimuli to a different part of the retina which, owing to the zero-luminance background used in the featured experiment, causes the target channels to lose their excitatory input. This reduction can be seen in the input trace in figures 0.6b and c, and the increase in fixation channel activity resulting from target acquisition can be seen in figures 0.6d.

In a natural scene there is every possibility that during, and immediately after, a saccade there *will* be salient input at the retinal coordinate the saccade target previously occupied. Consequently, *active* suppression of accumulated evidence is required in order to guarantee that accumulated evidence will be removed. One approach the oculomotor system appears to utilise is the active blocking of visual input whilst the eye is moving, a phenomenon known as "saccadic suppression" (Thiele et al., 2002). Another active method is the robust negative feedback from the SG to the SC which effectively eliminates the SC-TH-FEF-SC and SC-SC feedback loops. The current model is tuned so that the combined effect of its distributed positive feedback loops approximates a single loop with a closed loop gain of unity (as shown in figure 0.4c). Consequently, active suppression of two of these loops reduces the effective gain below unity so that the system loses its ideal integrator properties. We modelled both of forms of active suppression as a brief burst of inhibition applied to the retina and the SC at the instant that a saccade is triggered.

**Summary**

The preceding sections have shown that the model is able to cleanly select between multiple options (albeit incorrectly in the given example) when provided with physiologically plausible inputs. Furthermore, the model illustrates that the accumulation dynamic observed throughout the oculomotor system can be reproduced through the inhibitory control of a distributed positive feedback network. Also, although not modelled, feedback to LIP, from FEF, could in principle induce a similar pattern of activity in that area, consistent with observations. Key control issues not addressed by abstract mathematical models such as initiation thresholds, and reset mechanisms, have been shown to have physiological correlates in the guise of baseline SNr output and brainstem feedback respectively. We now consider the affect of sensory evidence and internal processing on the decision process.

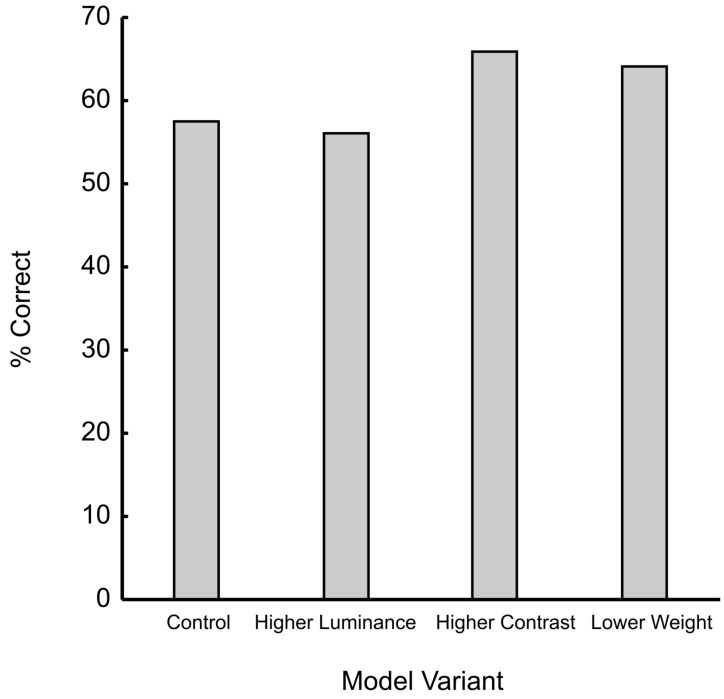


Figure 0.7 Percentage of trials that produced a saccade to the correct target for each of the experimental conditions.

### 0.3.2 Accuracy, response time and the effect of learning

Figure 0.7 shows how successful the model was in selecting the brightest target under the 4 experimental conditions. These results demonstrate that while increases in absolute target luminance do nothing to increase the model's accuracy, increases in the contrast between stimuli does. This is consistent with findings from the motion discrimination task in which increased motion contrast gives rise to increased response accuracy (Roitman and Shadlen, 2002). That increased stimulus contrast improves accuracy is perhaps unsurprising as it arises as a natural consequence of competitive dynamics. A less intuitive finding is that lowering cortico- and thalamo-striatal weights can produce a similar accuracy improvement to that achieved by increased contrast. This manipulation reduces the efficacy with which accumulated evidence within a given channel is able to request a reduction in the SNr activity applied to it. Because SNr levels are higher for a given level of accumulated evidence,

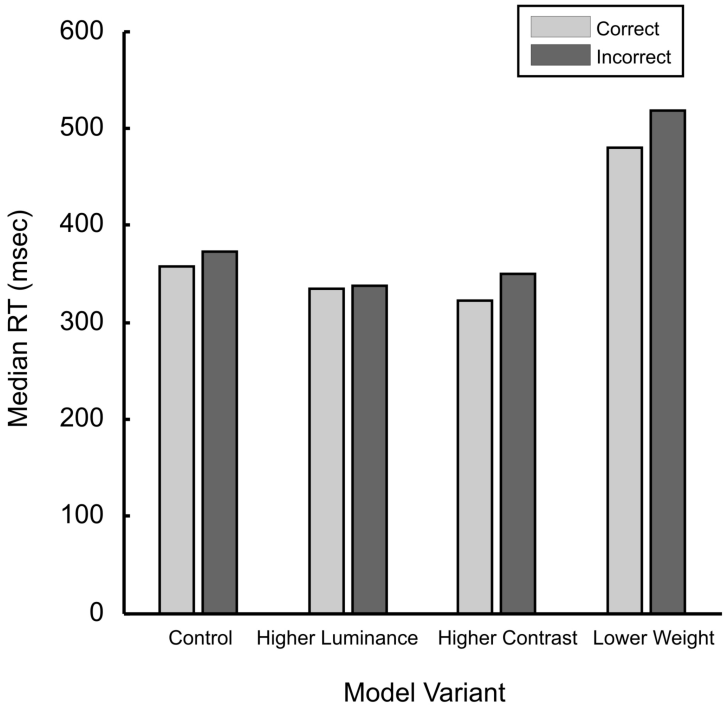


Figure 0.8 Median response time for correct and incorrect trials for each of the experimental conditions.

under this condition, the accumulation dynamic progresses more slowly (as illustrated in figure 0.4c). By being forced to "take its time" in this way, the accumulation process is better able to average out the effects of noise and in so doing reduces the error rate. This result highlights the potential role that striatal plasticity may play in modulating the dynamics of decision making.

Figure 0.8 shows how the median RT of correct and incorrect trials varies between experimental conditions. These results highlight that, while lowering cortico- and thalamo-striatal weights produced a similar increase in accuracy to increased target contrast, it comes at the cost of prolonged RT, the same trade-off observed when subjects voluntarily elect to increase their response accuracy in a saccadic task. The results in figure 0.8 also reveal that, under all experimental conditions, the RT of incorrect trials is longer than that of correct trials, which is consistent with findings from the motion discrimination task (Roitman and Shadlen, 2002). This property of the model arises from the fact

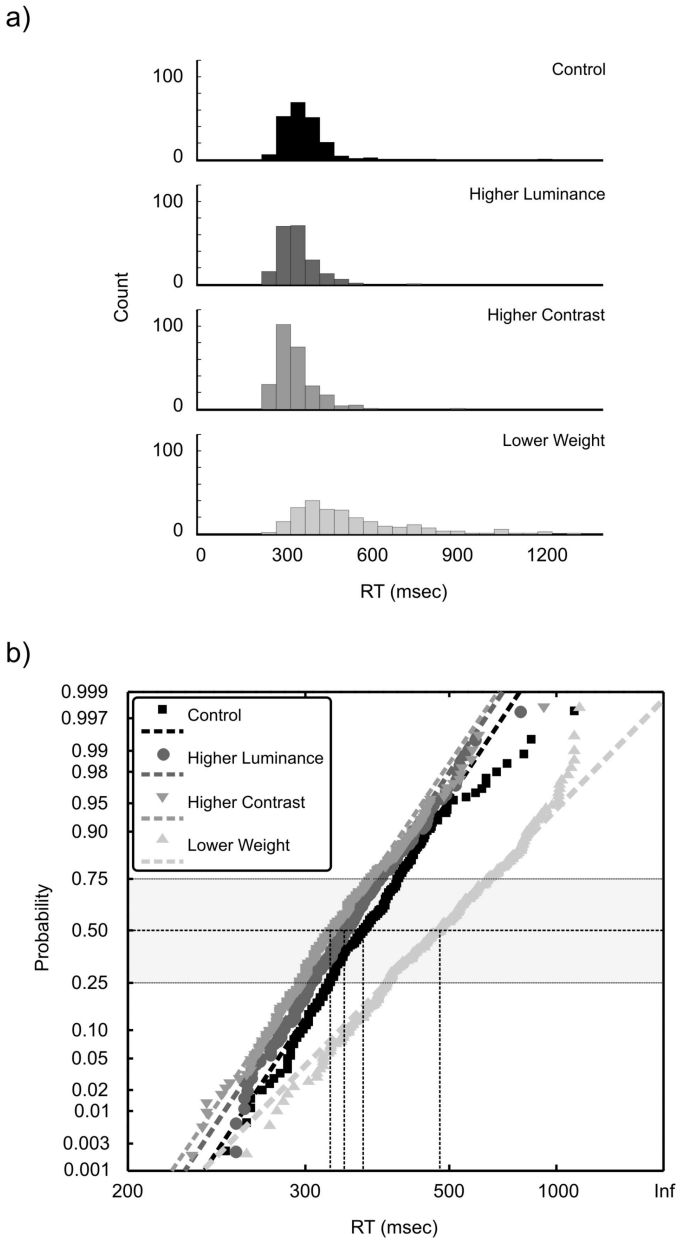


Figure 0.9 Response time distributions for correct trials under each experimental condition a) histograms of RT distribution for each condition; b) reciprobit plots of RT distribution for each condition. See text for explanation.

that on error trials (such as that shown in figure 0.6) the losing channel actually has the higher mean input. This means that, despite having a greater SNr input, the accumulated evidence in the losing channel can grow at a similar rate to that in the lead channel. This increased level of competition prolongs the selection process as it restricts the ability of the BG to further increase the contrast in SNr output.

Figure 0.9 shows the distribution of RTs achieved under each experimental condition for correct trials only. Figure 0.9a represents the RT data as histograms and clearly shows that the distributions of RT under each condition each exhibit a rightward skew as discussed in section 0.1. It is also clear that the distribution for the lower weight condition is significantly more skewed than that of any other condition.

Although intuitive to understand, it is hard to compare distributions represented as histograms. In Figure 0.9b we therefore show the RT distributions as a reciprobit plot. This type of plot is most commonly used to compare RT data with the assumptions of the LATER model of Carpenter (1981) which models decisions making as a race to threshold between evidence accumulators that do not inhibit each other. Although our model differs considerably from the LATER model, it is useful to try and characterise the RT distribution produced by our model using the relatively simple LATER framework.

Firstly, the fact that each condition's plotted results form straight lines (for the inner quartiles at least) indicates that the reciprocal of RT has a normal distribution, indicating that the RT skew exhibited under each condition is consistent with a linear rise to threshold. Secondly, the distributions for the higher luminance and higher contrast conditions appear to be leftwards shifted versions of that for the control condition indicating that the mean rate of evidence accumulation is increased under these conditions. Finally, the fact that the distribution for the lower weight condition is both rightwards shifted and of reduced gradient, indicates that the mean rate of evidence accumulation is lower under this condition, but also that the total amount of evidence to be accumulated is increased i.e. the distance between the initial evidence level and the threshold for action is increased.

## 0.4 Discussion

In this chapter we have demonstrated that the oculomotor anatomy, when viewed as a parallel array of, largely-independent, BG-controlled PFBLs, appears to implement a decision mechanism with properties similar to the diffusion model. Further, we have shown that the BG (as conceptualised by Gurney et al., 2001)

are able to arbitrate between alternative actions represented by accumulated sensory evidence, whilst also providing a threshold for the initiation of the accumulation process and a means of resetting accumulated evidence once an action has been initiated. Finally, we have demonstrated that changes in synaptic weights within the striatum (the BG input nucleus) are able to adjust the system's RT/accuracy tradeoff.

As described in section 0.1 there are several computational models that ascribe observed accumulation dynamics to the cortical microcircuitry (Usher and McClelland, 2001; Ditterich et al., 2003; Wang, 2002). The evidence we have presented in this article does not rule out the possibility that cortical circuitry fulfils an arbitration role, but does serve to highlight the possibility that this function might be performed centrally, by the BG. This view is consistent with other models that highlight the role of BG in controlling the build-up of motor activity in PFBLs (Arai et al., 1994; Grossberg and Pilly, 2008). We now seek to highlight two key advantages that the BG may offer as a centralised selection architecture.

### 0.4.1 Potential advantages of centralised selection by the basal ganglia

#### Algorithm refinement

As described above, the oculomotor model presented in this article has properties in common with the diffusion model. It can be demonstrated that, for two-alternative forced-choice tasks, that the diffusion model is mathematically equivalent to an optimal statistical test known as sequential probability ratio test (SPRT) (Wald, 1947). The equivalent optimal statistical test for decisions involving more than two alternatives is called the *multihypothesis* sequential probability ratio test (MSPRT) (Baum and Veeravalli, 1994). Bogacz and Gurney have demonstrated that the intrinsic connections of the BG can be interpreted as a minimal neural implementation of the MSPRT algorithm (Bogacz and Gurney, 2007, and Bogacz, this volume). Thus, while it may, in principle, be possible to optimally select between two alternatives using the cortical micro-circuit, there is evidence that the specialised architecture of the BG may be best suited to resolving such competitions where there are more than two alternatives. One advantage of separating out this specialised selection function from cortex, may be that cortical specialisations are able to evolve without affecting the optimality of decision making while, at the same time, all modalities requiring decision making, benefit from evolutionary improvements to the BG.

In their model of BG, Bogacz and Gurney (2007) made the simplifying as-

sumption that evidence accumulation occurred independently from the BG i.e. accumulators feed integrated evidence into the BG but are not, in turn, affected by it. The model we present here therefore differs from that of Bogacz and Gurney in that the output of the BG inhibits the PFBLs that feed into it. This change affects the relative importance of sensory evidence supplied over the course of the decision making process.

The diffusion model (and MSPRT) treats all evidence equally throughout the decision process so that evidence arriving just prior to action selection has the same influence on the decision process as the earliest evidence. Our model, in contrast, does not treat all evidence equally because losing accumulators are inhibited (by BG output) to a greater extent than the lead channel, so that as evidence accumulation in the lead channel approaches the selection threshold, losing channels must supply evidence at an every increasing rate if they are to reverse the decision. In other words, whereas the diffusion model (and MSPRT) chooses between actions based on the quantity of evidence alone, the oculomotor system, as we have interpreted it, chooses based upon evidence and ongoing commitment to an action i.e. as accumulated evidence increases, commitment to the leading decision starts to dominate with evidence from losing channels having a reduced influence. The policy implemented by the model therefore values conviction over accuracy.

It may be that our model has more in common with a variant of the diffusion process proposed by Busemeyer and Townsend (1993), which includes a term that is related to the current value of accumulated evidence. Support for an evidence inequality in decision making comes from the work of Ludwig et al. (2005) who tested human subjects using the same luminance discrimination paradigm used in this article. These authors found that the initial 100ms of stimulus presentation had the greatest influence upon the participants ultimate decision with later information having little or no effect.

**Adaptive learning**

In addition to their candidate role as the vertebrate brain's "central switch" (Redgrave et al., 1999), there is good evidence to suggest that the basal ganglia play a critical role in reward-based learning (Hollerman et al., 2000) so that they are perhaps better thought of as an "*adaptive* central switch". In this article we have demonstrated that striatal efficacy can affect accumulation dynamics and hence the RT/accuracy trade-off implemented. Consistent with this role is the fact that striatum receives convergent input from both sensory cortex and most areas of the pre-frontal cortex (PFC), suggesting that the "context-aware" PFC is able to directly influence action selection. This begs the question: what constitutes evidence? In the oculomotor system, for instance, dorsolateral pre-

frontal cortex (DLPFC) provides excitatory input to FEF and oculomotor BG (see Johnston and Everling, 2008, for review), suggesting that "endogenous evidence" in DLPFC could augment, or even act as a substitute for "exogenous evidence" from sensory cortices. This might lead to faster selection times for visible targets or the generation of purely voluntary eye movements to locations for which there is no sensory evidence.

In addition to being involved in decisions to act, it may be that PFC, through its influence on BG, is able to control decisions not to act. Certain tasks require that the subject withhold a response that they would ordinarily elicit, and it would appear that the BG provide a means of blocking habitual behaviour when necessary. Using a model derived from the architecture presented in this article, we have recently explored the role of the "indirect pathway" (involving D2-type medium spiny neurons) in *inaction* selection (Chambers and Gurney, 2008). This work sought to demonstrate how PFC can, via a cortico-striatal projection, learn to either selectively facilitate or block the accumulation of sensory evidence by exploiting PFC neurons that have an asymmetrical influence on the D1- and D2-type neurons present in a given channel. The model is able to successfully reproduce results from the non-match to sample task used by Hasegawa et al. (2004) for which success relies on the participant overriding the "habitual" tendency to attend to a primed location.

## Acknowledgments

This research was supported by the EPSRC Doctoral Training Award scheme, and by the EPSRC-funded REVERB project (EP/C516303/1). The authors are grateful to members of the Adaptive Behaviour Research Group at the University of Sheffield for their advice and comments.

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