Biologically constrained action selection improves cognitive control in a model of the Stroop task

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The Stroop task is a paradigmatic psychological task for investigating stimulus conflict and the effect this has on response selection. The model of Cohen et al. (1990) has hitherto provided the best account of performance in the Stroop task, but there remains certain key data that it fails to match. We show that this failure is due to the mechanism used to perform final response selection — one based on the diffusion model of choice behaviour (Ratcliff, 1978). We adapt the model to use a selection mechanism which is based on the putative human locus of final response selection, the basal ganglia/thalamo-cortical complex (Redgrave et al., 1999). This improves the match to the core human data and, additionally, makes it possible for the model to accommodate, in a principled way, additional mechanisms of cognitive control that enable better fits to the data. This work prompts a critique of the diffusion model as a mechanism of response selection, and the features that any response mechanism must possess to provide adaptive action selection. We conclude that the consideration of biologically constrained solutions to the action selection problem is vital to the understanding and improvement of cognitive models of response selection.

Keywords: Stroop, response selection, action selection, diffusion model, basal ganglia

Introduction

The Stroop task provides a thoroughly explored experimental framework for investigating cognitive aspects of selection. In this task, subjects have to name the ink colour of word-strings which can themselves spell out the name of a colour. When the ink-colour and the word-name contradict each other response selection is slowed and errorful (compared to conditions where the word-name is neutral or complementary with respect to the ink-colour). This is ‘the Stroop Effect’. A simple reversal of the task, that of reading the word-name and ignoring the ink-colour, does not produce an opposite effect (a ‘reverse Stroop’ effect).

The asymmetrical interaction of the colour-naming and word-naming processes can be interpreted within an automaticity framework (MacLeod, 1991; Posner & Snyder, 1975). Here, word-reading is an ‘automatic’, or ‘overlearnt’, response which is triggered on stimulus presentation and difficult to interrupt, and colour-naming is a controlled process which is not automatic and is liable to interference from word-reading. Variations on the basic Stroop task have been successful in clarifying the nature of automatic processing (Besner & Stolz, 1999; Besner, Stolz, & Boutilier, 1997; Dishon-Berkovits & Algom, 2000; Durgin, 2000).

Here, however, we wish to focus on the Stroop task as defining a process of selection. The Stroop task has a long history of use in the investigation of aspects of response selection at a cognitive level (MacLeod, 1991) and, more recently, at the neural level (MacLeod & MacDonald, 2000). In particular, while early processing of stimulus information is clearly important to an understanding of the Stroop task, the final response uttered on each trial is subject to the constraints imposed by a response or decision mechanism, which must translate internal cognitive states into motor action.

Much progress has been made in investigating decision mechanisms in simple two-alternative choice tasks. Mathematical models of such simple decisions are able to accurately predict the patterns of reaction times and errors across task variations, and there is a considerable history in psychology of their development and refinement (Luce, 1986; Ratcliff & Smith, 2004). More recently it has been possible to connect these models with neurophysiological data (Ratcliff, Cherian, & Segraves, 2003; Reddi, Asrress, & Carpenter, 2003) and with an information theoretic foundation for optimal decision making (Bogacz et al., in press; Bogacz, Usher, Zhang, & McClelland, this issue). These developments promise an exciting period of cross-fertilisation between neurobiological and psychological perspectives on simple decisions (P. Smith & Ratcliff, 2004; Opris & Bruce, 2005; Platt, 2002). The current work investigates how one
instance of this class of model serves selection in a model of the more complex Stroop task.

An additional perspective on decision making is supplied by workers in neuroscience, animal behaviour, ethology and robotics who have defined, and explored solutions to, the problem of action selection: the resolution of conflicts between competing requests for behavioural expression through a final common motor path (Redgrave, Prescott, & Gurney, 1999).

The aim of the present work was to determine whether a biologically plausible model of the putative locus of action selection in humans (the basal ganglia) could work as the response mechanism in a model of a cognitive task (the Stroop task). This is therefore a first step in making links between possible neural substrates for action selection, neural correlates of decision making, and cognitive processes of selection.

Modelling the Stroop Task

In a seminal paper, Cohen, Dunbar, & McClelland, 1990 described a model of processing in the Stroop task and its variations.

The current work focusses on the third element above: the response mechanism and its role in determining overall model behaviour.

The Cohen model matches the basic Stroop data very well (See figure 2). Not only does the model capture the quantitative difference that word-reading is faster than colour-naming, and unaffected by the word information, but it also matches the asymmetry between the size of the interference effect (the slowing of colour-naming due to contradictory word-information) and the facilitation effect (the speeding of colour-naming due to compatibility with the word-information). All the stimulations presented, both of our model and our replication of Cohen’s model, are shown run without added noise, since this does not affect the mean results.

In addition to matching the fundamental data, the model gives an implementational definition of automaticity: automaticity arises from greater strength of processing. In a connectionist framework this means stronger weightings between stimulus and response (as in the Cohen model), or additional connections between modules involved in stimulus response translation (as in other connectionist models of Stroop processing, Phaf, Vanderheijden, & Hudson, 1990; Zhang, Zhang, & Kornblum, 1999). Either way the implication is that there is no sharp dichotomy between ‘automatic’ processes and ‘controlled’ processes, and, additionally, that other quantitative differences, such as response time differences, arise out of this single fundamental mechanistic difference.

A plausible alternative theory of Stroop processing — and of automatic processing in general — is that more automatic processes are those in which pre-response processing is faster. This theory suggests that Stroop interference is due to the response evoked by the (contradictory) word element of the stimulus arriving at some response bottleneck earlier, creating slower selection of the opposite (and correct) response when it arrives there (we can posit that in a con-
Figure 2. The fundamental pattern of reaction times in the basic Stroop tasks. There are two tasks — word-naming (filled circles) and colour-naming (unfilled squares). Within each task there are three possible conditions; in the congruent condition the word and colour agree, in the conflict condition the word and the colour disagree, in the control condition the irrelevant element is neutral with respect to the target. Empirical data, (a), from Dunbar & MacLeod (1984) for which standard error bars are shown, and simulation data, (b), replication of simulation by Cohen et al (1990).

The experimental data are shown in figure 3, panel (a). By convention SOAs which involve the to-be-ignored element being presented first are labelled negative. Clearly no amount of headstart for colour-information (i.e. at negative SOAs) allows it to interfere with word-reading (Glaser & Glaser, 1982), demonstrating that the automaticity of word-reading is not a consequence of enhanced speed of processing. For colour-naming, the word element causes interference if it appears at any point before colour processing is finished (up to 300 ms after the appearance of the colour element — close to the asymptotic limit for reaction times). Additionally, the appearance of the word before the colour always causes interference, however long the subject is given to accommodate to the presence of the word. This, and other results which contradict the automaticity as speed-of-processing account (Dunbar & MacLeod, 1984), leave the automaticity as strength-of-processing account more preferable (this is not to say that strength-of-processing accounts do not imply that automatic processes will be faster than controlled processes — they do — rather they merely assert that speed of processing is a byproduct of a more fundamental distinction between the two types of processes rather than being causative in itself).

The original simulation data for the SOA manipulation within the Cohen model are shown in figure 3, panel (b). The Cohen model simulates the correct relative ordering of the reaction times in all conditions with respect to the empirical data. Cohen et al. (1990) note some discrepancy between their simulation and the model — firstly, that in the simulations colour information does interfere with word reading, albeit marginally, and that, secondly, the influence of word...
information on colour reading is not reduced but increases for SOAs before -200 ms. These discrepancies would not contradict the empirical data, and hence a strength of processing account, if the size of these effects was limited to that shown over the range originally tested by Cohen et al. (1990). The size of the interference and facilitation effects are not, however — as Cohen et al. (1990) suggest — asymptotic with increasingly negative SOA (as shown below, figure 4). Given this, it is of concern that the primary model of the automaticity-based account of Stroop processing, the Cohen model, is not able to simulate the primary data which falsifies the speed-of-processing account but instead produces a pattern of reaction times which would, if true, appear to validate a speed-of-processing account.

**Limitations of the Cohen model with SOA are failures of response selection**

Our replication of Cohen et al.’s (1990) model shows that, beyond the range of data of SOA values they originally present, the trends visible in the original data continue so that model behaves inconsistently with the strength-of-processing account and consistently with the experimentally disproved speed-of-processing account of automaticity in the Stroop task (see figure 4).

Consider the change in the simulated reaction times as SOA gets more negative — as the to-be-ignored element of the stimulus appears increasingly before the to-be-responded-to element. For the colour-naming task in the conflict condition the model RT increases as the word element slows selection based on the colour. Eventually, beyond -1300ms, the word is presented early enough to prompt a response on its own. This response will be an incorrect one, since in the conflict condition the word is opposite to the colour. RTs now start to decrease with increasingly negative SOAs because the reaction time is defined as the time between the onset of the to-be-responded-to stimulus element and the occurrence of selection. So RT eventually falls below zero because selection occurs before the onset of the colour (this is highlighted in figure 4 by the point at which the RT lines cross the dotted line representing zero on the RT axis). If the word is congruent to the colour information then there is comparable interference, but this reveals itself as a speeding of the correct response (which likewise falls below zero RT beyond -1500ms). Note that the time between the onset of the irrelevant element and selection is constant, so beyond the point at which the irrelevant element is determining selection the rate in the decrease in reaction time becomes a function of the decrease in SOA, not of changes in model output.

For the same fundamental reasons, in the word-naming task the conflict and congruent conditions diverge in the same way (albeit over a longer time span, the point at which word-reading times fall below zero SOA is not shown here). Thus, the model behaves in accordance with the experimentally disproved speed-of-processing account: presenting colour information ahead of word information creates a reverse Stroop effect — colour information interferes with word-reading. This is surprising, not least because the stated purpose of the model was to validate a strength of processing account.

Here we trace this flaw to the response mechanism used in the model. Cohen’s model of Stroop processing explicitly draws on the choice behaviour literature (Luce, 1986) and adopts an exact analogue of the diffusion model (Ratcliff, 1978; Ratcliff & Smith, 2004) to resolve the response selection problem presented by the Stroop task. In the diffusion model the momentary balance of evidence regarding the two possible responses at each point in time is used to adjust a running total. The momentary balance of evidence is defined by the strength of evidence in favour of one response minus the strength of evidence in favour of the other. At each time step the change in the running total is drawn from a Normal distribution with a mean defined by the balance of evidence (in this case, this is the difference between the output units of the connectionist front end). When this total, which reflects the accumulated evidence, crosses either a positive threshold (indicating selection of one response) or a negative threshold (indicating selection of the other response) selection occurs. The diffusion model has been shown to be an analytically tractable form of several connectionist models.
of decision making, and an optimal decision algorithm for a
two-choice decision situation (Bogacz et al., in press) where
either desired accuracy or time-to-decision is specified (obvi-
ously these two mutually constrain each other). Further, po-
tential neurobiological correspondences to the evidence ac-
cumulation processes of the diffusion model have been iden-
tified (Gold & Shadlen, 2000; Ratcliff et al., 2003; Reddi et
al., 2003).

The diffusion model response mechanism takes the out-
puts of the connectionist ‘front-end’ of the Cohen model as
inputs. Because the model, like all connectionist models,
works on graded signals there is always some input due to
the to-be-ignored stimulus, even if this is very small due to
the attentional inhibition. In the case of the colour-naming
task, it is integral to the model’s function that some influence
of the word-element of the stimulus survives attentional se-
lection and comes to influence the response stage. Without
this feature the basic effect of Stroop interference would not
be present. However, in SOA conditions, this influence of
the to-be-ignored element may accumulate indefinitely. This
affects selection time to an extent proportional to the time
it is presented multiplied by the strength of evidence con-
voyed. So arbitrarily small amounts of evidence can provoke
erroneous selection if presented for long enough, or they can
massively slow correct selection (because accumulated evi-
dence for the opposite response must be overcome).

The fact that Cohen et al.’s (1990) model involves a re-
sponse mechanism is ignored in textbook treatments of the
model (Ellis & Humphreys, 1999; Sharkey & Sharkey,
1995; O’Reilly & Munakata, 2000) and even overlooked in
Cohen et al.’s own analysis of the function of the model
(Cohen et al., 1990). This reflects, we argue, a regrettable,
but not untypical, neglect of the action selection problem in
psychology. Reinforcing this view, we have recently shown
how, contrary to the original account of Cohen et al. (1990),
it is the response mechanism, not the neuronal transfer func-
tion, which generates the important differences in reaction
times between conditions (Stafford & Gurney, 2004), and it
is the response mechanism which explains the asymmetry in
the magnitudes of the interference and facilitation effects in
the Cohen model (a matter about which there has been some
debate, MacLeod & MacDonald, 2000).

In summary, our investigation of evidence accumulation
as a mechanism of selection in the Cohen model of the Stroop
task will have implications for theories of selection in gen-
eral. The core element in this investigation is to show how a
more biologically realistic response mechanism — a model
of action selection in the basal ganglia — overcomes the de-
iciencies noted here.

The basal ganglia and thalamic complex as a response
mechanism in a cognitive task

The basal ganglia are a set of sub-cortical nuclei that have
been implicated in a range of motor and cognitive functions
(Brown, Schneider, & Lidsky, 1997). Recently we have pro-
vided a unified account of basal ganglia function by hypothe-
sising that they are a key element in resolving the action se-
lection problem by serving as a central ‘switch’ or arbitrar-
between action requests (Redgrave et al., 1999). Anatomically
this is plausible because the basal ganglia receive widespread
input from all over the brain, including many areas of the
cortex (Parent & Hazrati, 1993) and subcortex (McHaffie,
Stanford, Stein, Coizet, & Redgrave, 2005). Outputs from
the basal ganglia project back, directly or indirectly, to their
input targets, forming closed anatomical loops (Alexander &
Crutcher, 1990; McHaffie et al., 2005). For loops including
cortex, this occurs indirectly via thalamus. We focus, first,
on those aspects of our decision circuitry that make use of
the basal ganglia alone.

The basal ganglia and action selection

Our model of the circuitry intrinsic to the basal ganglia
is drawn directly from our earlier work (Gurney, Prescott,
& Redgrave, 2001a). This, in turn is based on the known
anatomy and physiology of the vertebrate basal ganglia,
shown in figure 5a and described in detail in several recent
reviews (e.g. Mink, 1996; Y. Smith, Bevan, Shink, & Bol-
am, 1998). The main input nuclei of the basal ganglia are
the striatum and the subthalamic nucleus (STN). The STN
is the only source of excitation within the basal ganglia. In
primates, the major output nuclei are the internal segment
of the globus pallidus (GPI), and substantia nigra pars reticulata
(SNr). These nuclei provide extensively branched GABAcere-
gic efferents to functionally related zones of the ventral thala-
mus (which in turn projects back to the cerebral cortex), the
midbrain and hind-brain areas critical for movement (e.g.
Kha et al., 2001). The external segment of the globus pal-
lidus (GPe) is an intrinsic source of inhibition within the basal
ganglia. Two separate striatal populations have been identi-
fied (Gerfen & Young, 1988): (i) a population that contains
the neuropeptides substance P and dynorphin, and preferen-
tially expresses the D1 subtype of dopamine receptors; and
(ii) a population that contains enkephalin and preferentially
expresses the D2 subtype of dopamine receptors. In most
accounts of basal ganglia anatomy, the D1-preferential pop-
ulation is usually associated with projections to SNr and GPe
alone, while its D2 counterpart is associated with projections
to GPe (Gerfen et al., 1990).

The basic assumption underlying our model was that the
brain is processing, in parallel, a large number of sensory,
cognitive and motivational streams or ‘channels’, each of
which may be requesting/promoting different actions to be
A further assumption was that, implicit in the representation of each action, there is an encoding of its salience or propensity to be selected for execution. In our model, we assumed that channel salience had already been extracted from phasic excitatory input by processes in the basal ganglia input nuclei. The input to the model, therefore, was simply the scalar-valued salience of each channel. The basal ganglia output is inhibitory and tonically active. Selection then occurs via selective disinhibition of target structures (Chevalier & Deniau, 1990) which include (as well as thalamus) premotor areas of the brainstem. Once inhibition has been released in this way, the corresponding behaviour is enacted. In summary then, large salience signal inputs at striatum and subthalamic nucleus (STN) select for low signal outputs at the entopeduncular nucleus (EP) and substantia nigra pars reticulata (SNr).

We used the computational premise of selection to guide our interpretation of basal ganglia anatomy in functional terms. One architectural feature that may be invoked in this respect is the diffuse excitation from STN to its targets — GP, SNr/EP (Parent & Hazrati, 1993, 1995) — in combination with more focused inhibition from striatum to the same nuclei. This constitutes an off-centre, on-surround network that can perform a selection function, as noted by Mink and Thach (1993). However, this simple scheme is ambiguous as it stands since it is not clear what function the GP serves as an ‘output layer’ in a feedforward net. We resolved this problem by observing that, while selection could be performed in principle by the complex of striatum (D1), STN and SNr/EP alone, the relative levels of excitation and inhibition required to achieve this function were only obtained (and indeed guaranteed) by the inhibition supplied by GP. We therefore hypothesized that the GP acts within a control pathway (comprising striatum (D2), STN and GP) as a source of control signals for the selection pathway (striatum (D1), STN, SNr/EP). The new functional architecture described above (Gurney et al., 2001a) is shown in figure 5b. Note that it is quite different from the prevailing ‘direct/indirect’ pathway scheme of Albin et al (1989), and hypothesises a different role for GPe from that posited by Hazy, Frank & O’Reilly (this issue) and Frank, Scheres & Sherman (this issue).

The resulting model (Gurney, Prescott, & Redgrave, 2001b) was able to successfully select and switch between channels based on their input salience. In addition, the model allowed dopaminergic modulation of basal ganglia function in ways compatible with disorders of dopamine function (e.g Parkinson’s disease). While, the role of dopamine is not discussed here, we note that the model is rich enough, in principle, to account for data derived from studies with relevant population we needed to consider was, therefore, the set of neurons responsible for a single channel within each of the basal ganglia nuclei.

Figure 5. Basal ganglia anatomy and functional architecture
a) basal ganglia anatomy used as the basis for the model,
b) new functional architecture for basal ganglia (Gurney, Prescott & Redgrave, 2001a) used in the current work. See text for details.
including the thalamic complex

As noted above, the basal ganglia sits in a wider anatomical context comprising closed loops of cortex-basal ganglia-thalamus-cortex. In previous work, we modelled such loops by embedding the basal ganglia model (described above) into a loop incorporating motor and somatosensory cortex (Humphries & Gurney, 2002). In that instance, there are well understood anatomical relations between these cortical areas, basal ganglia, and specific nuclei within thalamus. In the current work, the specific areas of cortex associated with word reading and colour processing are not well understood. We therefore adopt a simplified version of the model in (Humphries & Gurney, 2002) by using only a single cortical area (figure 6).

Further, whereas in the somatosensory/motor loop the thalamic nucleus is identified as the ventrolateral (VL) thalamus (Price, 1995), here it is left non-specific and is labelled ‘Thalamus’ in figure 6. A component common to both the original and simplified scheme is the thalamic reticular nucleus (TRN) which sends diffuse inhibition to Thalamus. The extended thalamo-cortical model retains the channel-based scheme of the basal ganglia model and reciprocal connections between thalamus and TRN imply the latter acts as a distal lateral inhibition mechanism for the former. Input to the model comes from other cortical areas and constitutes an initial representation of salience.

The original somatosensory/motor loop model displayed enhanced selection capabilities in several respects when compared with the model of the basal ganglia alone (Humphries & Gurney, 2002). Further, using these models in robot controllers has shown that their selection behaviour is of sufficient efficiency and sophistication to be behaviourally adequate in realistic environments (Girard, Cuzin, GuilJot, Gurney, & Prescott, 2003; Prescott, Montes Gonzalez, Gurney, Humphries, & Redgrave, 2006). Details of these models are to be found in (Gurney et al., 2001a; Humphries & Gurney, 2002) and also in the annotated code which is provided in the accompanying electronic supplementary material.

Combining the Cohen model with the basal ganglia response mechanism.

It is natural to ask if the extended thalamo-cortical-basal ganglia model, viewed as a decision mechanism, can perform appropriate selection in a cognitive task. The model was developed with a view to accounting for action selection in the domain of systems neuroscience with no intention, originally, of being used to generate reaction time data. The model’s ability to account for such data would therefore serve to validate it further, and open up further possibilities for investigating biologically plausible response mechanisms in the study of cognition.

The rationale for constructing the connectionist Cohen model and our systems neuroscience model are quite different. The Cohen model is a minimal connectionist model designed to test a high-level hypothesis about automatic and controlled processing. On the other hand, our basal ganglia models are biologically constrained, respecting the known anatomy of the target circuits, and were designed to test the hypothesis that those specific circuits could support action selection. Further, whereas learning is a key component of the Cohen model, it does not figure in our models of basal ganglia and thalamus.

There are, however, sufficient points of contact between the two models to allow them to be joined in a unified scheme. Thus, the model in figure 6 is built out of standard leaky integrator neurons (Arbib, 1995) — a feature that it shares with the Cohen model — so that they both utilise a common signal representation denoting neuronal population responses.

The reaction-time behaviour of the model is read from the output units of basal ganglia. Recall that these represent neuron populations providing tonic (continuous background) inhibition to motor targets, and that selection occurs on those channels whose inhibitory output is sufficiently reduced. Reaction time is then interpreted as the time to selection, which is the time from stimulus onset to reduction of basal ganglia output on the selected channel to some threshold value. Moreover, we suppose that this selection threshold may be greater than zero. Although a zero output would demonstrate unequivocal selection, it is unrealistic to suppose that a pop-
Figure 7. Architecture of the combined model. Initial inputs are processed by the connectionist ‘front-end’ of the Cohen model as described in Cohen et al. (1990). Outputs representing the evidence in favour of the two possible responses are interpreted as initial salience inputs to the cortical component of the thalamo-cortical-basal ganglia model in figure 6. The basal ganglia outputs determine which response occurs (the output channel which first passes a selection threshold) and the reaction time (the time required to reach that threshold).

Figure 8. Comparing (a) empirical and (b) simulation reaction times when using the basal ganglia model as the response mechanism for the basic Stroop. Empirical data is from Dunbar & MacLeod (1984), for which standard error bars are shown.

Simulations I: matching basic empirical data

The combined model successfully replicates the basic (colour-naming) Stroop task, and the word-reading variation (see figure 8). This shows that the model is capable of performing basic selection in a cognitive task, and of producing realistic reaction time values.

The ability to realistically model learning phenomena is a key benefit of connectionist models. The combined model mimics the power-law function of learning (figure 9), just as the original Cohen model does (note that no learning takes place in the response mechanism component in either model). This demonstrates that the learning dynamic captured by the connectionist front-end is not interfered with by the use of the basal ganglia response mechanism; graded changes in the signals from the front end are converted into control and learning. Only one minor change is required to this ‘front-end’ to make it compatible with the basal ganglia model response mechanism. The output units of the original Cohen model have resting values of 0.5, the midpoint of their output range which lies in the interval [0, 1]. This is inconsistent with our new interpretation of these signals as salience values, since it indicates that all possible responses have moderately strong saliences at rest. In the combined model, the resting values of the front-end are set to 0.1, indicating a weakly salient input to the basal ganglia (small changes in weight initialisation are also required as a consequence of this manipulation; for details see Stafford, 2003).

In all other respects the combined model is exactly as published by Cohen et al (1990), except with the basal ganglia model replacing evidence accumulation as the method of final response selection. The basal ganglia thalamo-cortical model used is exactly as published elsewhere (Gurney et al., 2001b; Humphries & Gurney, 2002).
appropriately graded changes in reaction times.

Figure 9. The model conforms to the power law of practice (Logan, 1988). Both axis use a log scale. Simulation results are shown as dots. The simple regression for the data is shown as a straight line and follows the form \( \log(\text{Processing Time}) = 2.65 - 0.46 \log(\text{Epochs}) \). \( R^2 = 0.948 \).

The SOA task reveals that using the basal ganglia as a response mechanism provides a superior fit to the data than when using the original response mechanism (figure 3, panel (c)). Within the original range of the SOA values the simulation data more closely matches the empirical data. Running the model at extended SOA values (figure 10) confirms that reaction times using the basal ganglia response mechanism are stable. At negative SOAs the salience output caused by the to-be-ignored element of the stimulus is not sufficient to cause selection. Thus, using the basal ganglia response mechanism, the model makes the correct selection at all SOA values. In addition the amount of interference and facilitation it causes is limited. This is reflected in the stabilisation of reaction times at SOAs below -400 ms.

simulations II: dynamic attentional inhibition

Providing the model with stability under SOA conditions makes possible further model manipulations which bring the model up to date, in a principled way, with developments in our understanding of automatic processing and cognitive control. It has been suggested that selection in the Stroop task is dynamically controlled by a process that monitors for conflicts (located in the anterior cingulate cortex) and increases attentional control in response (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004). Two additional simulations presented here demonstrate that using the basal ganglia model as the response mechanism allows the use of dynamic-attentional modulation to enhance the match to the empirical data.

Here, we do not propose an account of conflict monitoring, nor tie it to any specific anatomical location. Instead we implement solely the essential feature that the appearance of the to-be-ignored element provokes, after some delay, an increase in the attentional inhibition acting on it. The length of the delay used here is 100ms which accords well with the time-scale of phenomenon such as negative priming (see May, Kane, & Hasher, 1995, for a review) and neurophysiological recordings of activity suppression due to attentional processes (Chelazzi, Duncan, Miller, & Desimone, 1998). See Usher & McClelland (2001) for a discussion of the time-course of activity during choice selection.

The implementation of attentional modulation in our model is achieved in the following way. After 100 (simulated) milliseconds the inhibition on the relevant hidden units of the Cohen model is increased in magnitude from the default value of \(-4\) to \(-4.9\), the value used by Cohen et al. (1990) in their simulations of the SOA task (at all values of the SOA). Thus dynamic attentional modulation is a modification of the mechanism that already exists in the model for implementing attentional selection, using parameters that have already been established. The parameterisation of the attentional modulation could have been finessed but we sought to test the validity of the idea without such ad-hoc modifications.

Figure 11, shows the simulation results for the model with this dynamic attentional modulation. Reaction times in the colour-naming conflict condition now peak around the 0ms
SOA point, and flatten-off at a lower level, as occurs in the empirical results (figure 3, panel (a)) — this is an improvement over both the Cohen model and the combined model without dynamic attentional modulation. This simulation both solves the stability problem and matches the peak and decline in reaction times that the empirical data shows.

In contrast, with dynamic attentional modulation the original Cohen model does not successfully match the empirical data (see figure 12). Because the stability problem is not resolved, the to-be-ignored stimulus element still provokes erroneous selection at long enough SOAs, and causes unrealistic amount of response-time interference before that.

Discussion

Our primary result is that a neurobiologically plausible model of action selection allows the successful simulation of reaction times in the Stroop task, despite the fact that the model construction was structurally and functionally guided by quite different principles. Although the front-end of the model was explicitly designed to do Stroop processing it is the response mechanism which is responsible for converting signal outputs into reaction times. Structurally, the model was constrained by the known functional neuroanatomy of the basal ganglia; functionally, it was a quantitative interpretation of our action selection hypothesis (Redgrave et al., 1999). The basal ganglia model was not explicitly designed to simulate reaction times, nor was it constrained by human cognitive performance, yet when processing outputs from the front-end of Cohen et al’s (1990) model it has advantages over the diffusion model, which was explicitly designed to simulate reaction times, in simulating reaction times.

why the basal ganglia model successfully simulates reaction times

The model captures the basic Stroop (figure 8) and learning (figure 9) phenomena because, for moderately sized saliences, selection time is based on the relative difference between the to-be-selected salience and the competing salience (if any). To understand the emergence of reaction time differences in the basal ganglia model, consider figure 13. Panel (a) shows traces (directly from the simulation) of the output signals corresponding to the correct response, in a control and a conflict condition; these signals cross the selection threshold and therefore produce a behavioural response. Note that the output signal in the conflict condition falls to a lower level than in the control condition. It is this final level to which the signal drops which defines the rate at which the signal drops and hence the time to selection. The final signal level is in turn, dependent on the relative difference between the to-be-selected salience and the competing salience (if any).

The schematic diagram of signal time courses (figure 13, panel (b)) clarifies the way in which final equilibrium output governs selection and reaction time. The rate of decrease of the output signal has the same relation to time-to-selection as the drift rate (strength-of-evidence) does to mean reaction time.

Figure 11. Simulation SOA data when using the basal ganglia model as the response mechanism and with the addition of ‘dynamic attentional modulation’

Figure 12. Simulation SOA data when using the original Cohen model with the diffusion model as the response mechanisms with the addition of ‘dynamic attentional modulation’
activation
time
(a)
(b)
conflict
control
conflict
non-selected
selection threshold
stimulus onset
time
stimulus onset
time

Figure 13. Selection in the basal ganglia (a) output signals from example runs of the model in the conflict (solid line) and control (dotted line) conditions (b) schematic illustration of the way in which final equilibrium output governs selection and reaction time. A selected signal with fast RT (solid line), a selected signal with larger RT (dotted line) and a non-selected signal (dot-dash line) are shown.

time in the diffusion model. However, because the rate of decrease in the basal ganglia model is ultimately determined by the final output signal resting level, selection does not always occur. In particular small saliences - which might result from a to-be-ignored stimulus - do not drive the output down beyond the selection threshold.

It is because the basal ganglia model is designed to operate continuously that it has equilibrium final states. Thus, in the idealised situation of unchanging inputs, all patterns of input, eventually produce unchanging output states. In particular, for some patterns of input, the final output state indicates that no action is selected. In more realistic situations, with noisy input, the basal ganglia-thalamo-cortical model is stable to small transient fluctuations in salience (Humphries & Gurney, 2002). It is with small saliences, and when dealing with successive rather than simultaneous inputs, that the advantages of using a selection mechanism that has non-selection equilibrium states is revealed. Both of these cases are revealed by comparison of the SOA simulations (figures 4 and 10).

weaknesses of the diffusion model

Our simulations illustrate a situation in which simple evidence accumulation is a non-adaptive choice process. The failure of the Cohen model on the SOA simulations is because of a model feature which is neither trivial nor irrelevant. The empirical existence of the basic Stroop interference effect demonstrates that response activation from the to-be-ignored word element of the stimulus must, at least to some extent, ‘break through’ any initial attentional inhibition. This activity, arriving at the response mechanism before the response activation of the colour element, is enough, in the Cohen model, to cause selection. The erroneous selection produced at long SOAs shows that a response mechanism must not make selections based on inconsequentially low inputs.

The Cohen model evidence accumulation mechanism has no minimal threshold on inputs, and no decay of accumulated evidence. This means that there are no equilibrium states and it is constantly being driven to enforce selection, no matter how long this takes. By extension, the diffusion model, the general form of the evidence accumulation mechanism used, contains no capacity for not making a selection. This is a serious flaw. At a minimum it indicates that the context within which the diffusion model of selection is used cannot be ignored or assumed.

alternative solutions

We have considered how the choice of response mechanism affects performance in simulation of the Stroop task. Other mechanisms for matching the core empirical data could be envisaged. Cohen & Huston (1994) adapt the model of Cohen et al (1990) to provide a better match to the SOA data. They do this by removing the diffusion model response mechanism entirely and having selection triggered by activation on the output units crossing a fixed threshold. This solves the problem of selection by arbitrarily small activations, since they do not reach the selection threshold.

This approach allows a fit to the data, but the removal of an explicit response mechanism raises some additional questions. Cohen & Huston’s (1994) model, in this respect, bears
some similarity to the Usher & McClelland (2001) model of perceptual choice. Both models use a single network for processing stimuli and for selecting responses using a simple threshold. Bogacz et al (this issue) have provided extensions and discussions of the optimality of the Usher & McClelland model. This model considers mechanisms of choice comprised of neuron-like elements but removed from a realistic cognitive or biological architecture. Although models without explicit response mechanisms can fit behavioural data (Cohen & Huston, 1994) or be shown to make optimal decisions (Usher & McClelland, 2001, Bogacz et al, this issue) two issues remain unaddressed. Firstly, which neural structures implement the model? Secondly, how is the optimal decision making provided by the model adaptively controlled?

Our approach has been to consider action representation and response selection separately, as in the original Cohen model, and to provide an account of response selection based upon the basal ganglia, as the proposed vertebrate solution to the selection problem. The benefits of using a centralised rather than distributed selection mechanism are discussed in Prescott et al (1999). Amongst these benefits is the greater theoretical ease of coordinating between multiple competing neural loci — both in terms of lower wiring cost and in the ability to centrally mediate the equivalent of thresholds.

**benefits of the basal ganglia model**

The simulation of the SOA paradigm highlights two properties which the basal ganglia as a selection mechanism brings to the combined model to improve the possible account of the data. The first, as already discussed, is the lack of incorrect selection for arbitrarily small saliences. The second is the limit on the maximum possible influence of concurrently or consecutively active inputs. Priming of response times, whether positive or negative, occurs because activity on other channels alters the basal ganglia output signals, at a sub-selection level, thereby affecting the time it takes for outputs to drop below the selection threshold. A similar process occurs in the diffusion model, but accumulated evidence is not limited — and can ultimately lead to incorrect selection (as discussed above). Figure 14 shows the geometry of selection interference in both response mechanisms. In the diffusion model (figure 14, panel (a)) the increase in reaction time due to a preceding to-be-ignored stimulus is a function of the size of that signal multiplied by time — the longer the to-be-ignored stimulus is presented, the greater the size of the interference effect. If the to-be-ignored stimulus is presented for long enough and the accumulated evidence reaches the selection threshold then an incorrect response is made. In the basal ganglia model (figure 14, panel (b)) the amount of increase in reaction time due to a to-be-ignored stimulus is solely a function of the magnitude of the salience that the to-be-ignored stimulus provokes. Because, as discussed above, the basal ganglia model has equilibrium final states, some of which do not indicate selection, the rise in the output signal associated with the correct response is limited and does not increase with time after a certain point. The increase in reaction time result is commensurately limited, and thus the correct response is selected efficiently.

This is an example of the general ‘clean switching’ property which has been identified as a desirable feature of any selection mechanism (Redgrave et al., 1999). A response mechanism needs to work in real-time, continuously, dealing with the successive selection of actions and interruption of old actions by new. The SOA paradigm illustrate just one situation where human action selection demonstrates clean switching. The benefits the basal ganglia model brings to modelling the Stroop task demonstrates the value of considering the constraints of natural action selection within cognitive models.

**Figure 14.** Interference in (a) the diffusion model and (b) the basal ganglia model response mechanisms. Signals in the diffusion model represent the accumulated evidence in favour of two possible responses, selection is indicated by crossing either the positive or negative evidence thresholds. Signals in the basal ganglia represent the activity on the to-be-selected action channel, selection is indicated by activity on that channel dropping below the selection threshold. Solid lines show signals subject to interference from a preceding input, dashed lines show signals without this competition. The signal courses for the early or later appearance of the to-be-responded-to stimulus are shown (indicated by points *1* and *2* respectively), and the corresponding size of the interference effects is indicated.
the diffusion model in the context of action selection

That the evidence accumulation response mechanism, on the other hand, has only one type of final state (that of selecting an action) and it continuously moves towards this state, has implications for the diffusion model as a model of response selection. The diffusion model embodies the inevitable progression towards selection because all inputs are integrated into a running total of activity, without any decay of that activity. This ‘perfect integration’ is actually a requirement of the proof that the diffusion model performs optimally (P. Smith & Ratcliff, 2004; Bogacz et al., in press), at least for a restricted class of choices. The simulation of the SOA experiments reveals that selection by perfect integration can be unadaptive in at least some circumstances. This particular case of the general problem of clean-switching illustrates that adaptive action selection involves criteria beyond those which have been used to define decision-optimality (i.e. criteria beyond those pertaining to the kind of simple choices which have hitherto been the main focus of analysis of choice behaviour). This is not to say that the diffusion model, or diffusion-like processes, are inappropriate for selection. Indeed it has recently been proposed that the basal ganglia architecture is able to perform optimal decision making in a manner akin to the diffusion model, but between multiple alternatives (see Bogacz & Gurney, in press). The diffusion model reflects an optimal way of integrating information if the possible choices are defined, the sources of evidence static and if the point at which the choice process begins is a given. Our claim is only that evidence accumulation and the diffusion model alone cannot provide a full account of adaptive action selection. For this wider problem mechanisms are required which signal the appropriate initialisation of the accumulation process, and which reset it or effectively overcome previous accumulation of evidence. The basal ganglia thalamo-cortical model provides a first step towards the integration of the decision-optimal diffusion model into the wider context of adaptive action selection.

summary and future work

This work validates our model against the basic Stroop phenomena. Use of the basal ganglia model as the response mechanism improves the fit that can be made to the empirical data and highlights necessary features response mechanisms should contain, the lack of which was overlooked in the previous account by Cohen et al (1990). Utilising an adaptive, action-selection based response mechanism in the model of Stroop task, allows the principled addition to the model of dynamic attentional modulation (Botvinick et al., 2001, 2004). Use of the basal ganglia model also extends the account of Stroop processing to connect with the neurobiology of selection.

From a wider perspective, there is a ‘theoretical purity’ to testing models outside of the domain that they were developed in. First, the basal ganglia model, while not designed to account for RTs, successfully managed to do so. Secondly the biologically grounded model of the basal ganglia also deals appropriately with signals provided by a more abstract connectionist model of a cognitive task. This depended on a common signal interpretation at the interface between the two model components in terms of population rate codes. We therefore suggest that this offers a useful tactic in any high level cognitive modelling that would enable the gradual replacement of abstract model components with more biologically realistic counterparts. Note, however, that this approach does not undermine the principled use of connectionist modelling in quantitative testing of cognitive hypotheses. Thus, in our present context, the model proposed by Cohen et al (1990) was a test of the hypothesis that the Stroop effect could be accounted for in a framework in which the ‘strength of processing’ devoted to a perceptual or cognitive process determined its status as more or less automatic (or controlled) in relation to other processes. Our work does not challenge the validation of this particular hypothesis since the ‘front-end’ of the model still tests it perfectly adequately.

Finally, the ability of the model to deal with an arbitrary number of inputs will provide opportunities for future modelling investigations of additional selection paradigms, and making connection to the possible underlying neurobiology enriches the account possible of Stroop processing. In particular, we anticipate that the existing provision for dopaminergic modulation of signal processing in our model will allows future tests of the model against various pathologies, such as schizophrenia. Our account will also need to be broadened to account for learning within the basal ganglia. Developing a full account of the interaction of plasticity with decision making will be an important test of all existing models of action selection.

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