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A competitive integration model of exogenous and endogenous eye movements

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Abstract We present a model of the eye movement system in which the programming of an eye movement is the result of the competitive integration of information in the superior colliculi (SC). This brain area receives input from occipital cortex, the frontal eye fields, and the dorsolateral prefrontal cortex, on the basis of which it computes the location of the next saccadic target. Two critical assumptions in the model are that cortical inputs are not only excitatory, but can also inhibit saccades to specific locations, and that the SC continue to influence the trajectory of a saccade while it is being executed. With these assumptions, we account for many neurophysiological and behavioral findings from eye movement research. Interactions within the saccade map are shown to account for effects of distractors on saccadic reaction time (SRT) and saccade trajectory, including the global effect and oculomotor capture. In addition, the model accounts for express saccades, the gap effect, saccadic reaction times for antisaccades, and recorded responses from neurons in the SC and frontal eye fields in these tasks.

Keywords Eye movements · Saccades · Superior colliculus · Frontal eye fields · Competitive integration

During active vision, our eyes constantly scan the environment using saccadic eye movements. Saccades are the fast movements of the eyes made to bring the fovea onto an object for detailed visual analysis (Becker 1989; Leigh and Zee 1999). Which object is the target of a saccade seems to depend on the interaction between two types of signals: endogenous signals reflecting the intentions, goals and beliefs of the observer, and exogenous signals representing the properties of the stimulus environment. When an observer intentionally selects only those objects required for the task at hand, selection is said to occur in an endogenous, voluntary, goal-directed manner. When specific properties present in the visual field determine selection independent of the observer's goals and beliefs, selection is said to occur in an exogenous, involuntary, stimulus-driven manner.

Studies employing the oculomotor capture paradigm (Godijn and Theeuwes 2002; Irwin et al. 2000; Theeuwes et al. 1998) have demonstrated that endogenous and exogenous signals can be in competition. In this paradigm, observers have to make an endogenous (top-down) saccade toward a uniquely colored target (so-called singleton) while ignoring the sudden onset of a distractor. In many instances, the eyes go to the distractor before a corrective saccade brings them to the target. On those trials, the endogenous signal to move the eyes to the target was disrupted by the exogenous signal generated by the visual onset.

It is generally assumed that the interaction between endogenous and exogenous signals takes place in a saccade map. Many theories assume that the site of this saccade map is the intermediate layers of the superior colliculi (SC)[1] (Godijn and Theeuwes 2002; Munoz et al. 2000; Trappenberg et al. 2001), perhaps together with other oculomotor areas (e.g., the frontal eye fields, Munoz and Schall 2003). The SC integrate input from many cortical areas, such as the frontal eye fields (FEF), the supplementary eye fields, the posterior parietal cortex, dorsolateral prefrontal cortex (DLPFC), and occipital visual areas (Lock et al. 2003; Lui et al. 1995; Munoz

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[1] There are two superior *colliculi*, one in each hemisphere, coding for saccades in the opposite hemifield.

2002). They send the result of this integration process to the cerebellar and brainstem premotor circuitry where the eye movement is programmed (Moschovakis 1996).

The SC do not simply code the motor command for an eye movement. SC activity seems to not represent particular saccades, but to denote desired gaze displacements in oculocentric coordinates (Bergeron et al. 2003; Freedman and Sparks 1997; Freedman et al. 1996; Soetedjo et al. 2002). This means that the location of activity in SC determines which target is going to be foveated, but not how this is brought about (e.g., via a saccade, combined eye and head movements, or smooth pursuit, Krauzlis et al. 2004).

Here, we present a computational model of the eye movement system, based on the idea that competitive integration of endogenous and exogenous information takes place within the SC and other areas. The main goal of the model is to account, in one framework, for findings in the eye movement paradigms used in experimental psychology. Although we focus on behavioral findings, the model can also account for electrophysiological findings in the paradigms discussed. There have been previous models with similar aims (Kopecz 1995; Trappenberg et al. 2001). The main advance of the current model over those are that we implemented the SC as a two-dimensional saccade map (instead of the common 1-D map), and connect the SC to a brainstem saccade generator. This makes it possible to analyze the interaction between signals in space and time, and to simulate saccade trajectories and endpoints. We will show that the idea of competitive integration of signals in SC helps to understand many behavioral findings in saccade research, while also being in keeping with much electrophysiological evidence from recordings in the FEF and SC.

1 The model

1.1 Structure of the model

Our model contains four modules: the FEF, the SC, the brainstem saccade generator, and the cerebellum. FEF was modeled as a set of ten model neurons. Each had a "movement field," an area of the visual field to which it codes saccades, corresponding to either a possible target location, or to the location of the fixation cross. Our brainstem model was based on the seminal work of Van Gisbergen et al. (1981) and Becker and Jürgens (1990). Van Gisbergen et al. (1981) showed how the brain stem might translate a desired horizontal eye movement into a motor command to the eye muscle. Becker and Jürgens (1990) proposed a way to couple two such mechanisms, one coding for horizontal and one for vertical saccades, to produce oblique saccades.

As constructing a large two-dimensional SC map with a sufficient number of model neurons would have made the simulations unwieldy, we settled on an architecture in which we only explicitly simulated neurons on eight radial axes across each collicus (see Fig. 1a). The output of other neurons was extrapolated from these axes. Each axis consisted of 250 model neurons, all of which coded for saccades with a common angle. This brings the total neuron number for SC to 4000. One axis covered the outer rim of the colliculi, containing model neurons with receptive fields on the vertical meridian (this axis was shared by both colliculi, as in both the outer rim codes for the same, i.e., vertical, saccades). Another axis went through the center of the SC, with model neurons with receptive fields on the horizontal meridian. The other six axes were arranged at maximal distances in between these two, with neighboring axes coding for saccades that were 22.5° apart. This is not the approach normally taken in neural network models of the SC, which tend to model the SC as a square.

1.1.1 Model neurons

Model neurons in each layer all had the same parameter values. It has been proposed that the SC contains three classes of saccade neurons (Munoz and Wurtz 1995a). Fixation neurons, found in the rostral part of the SC, fire strongly while the eyes remain fixated. The other two classes, together called saccade neurons, are found in the caudal part of the SC. They fire whenever a saccade is made to a certain location in retinotopic space. Buildup neurons are characterized by a slow buildup of activity before a saccade, in contrast to burst neurons, which are silent until right before a saccade. However, later evidence suggested that the dichotomy between buildup and burst neurons is not strict, and it may be more appropriate to consider them as a continuum (Anderson et al. 1998; Munoz and Wurtz 1995a). With parameter values chosen here, model neurons in SC showed some buildup of activity before a saccade, and would fall in the middle of the spectrum from buildup to burst neurons. Fixation neurons, meanwhile, may be seen as saccade neurons coding for a saccade of zero amplitude, and that is how they are treated here.

Model neurons, both in the SC and the FEF, were leaky integrators, with a continuous firing rate that was a function of their membrane potential. Membrane potential and firing rate were simulated in time steps of 1 ms. Membrane potential V of model neurons in FEF and SC was a function of inputs, previous membrane potential, and lateral interactions. The change in V at every 1-ms time step was calculated with a leaky integrator membrane update function:

$$\Delta V = \frac{-(V_r - V) + e_{\text{net}}(V_e - V) + i_{\text{net}}(V_i - V) + i_{\text{lat}}(V_i - V)}{\tau} \quad (1)$$

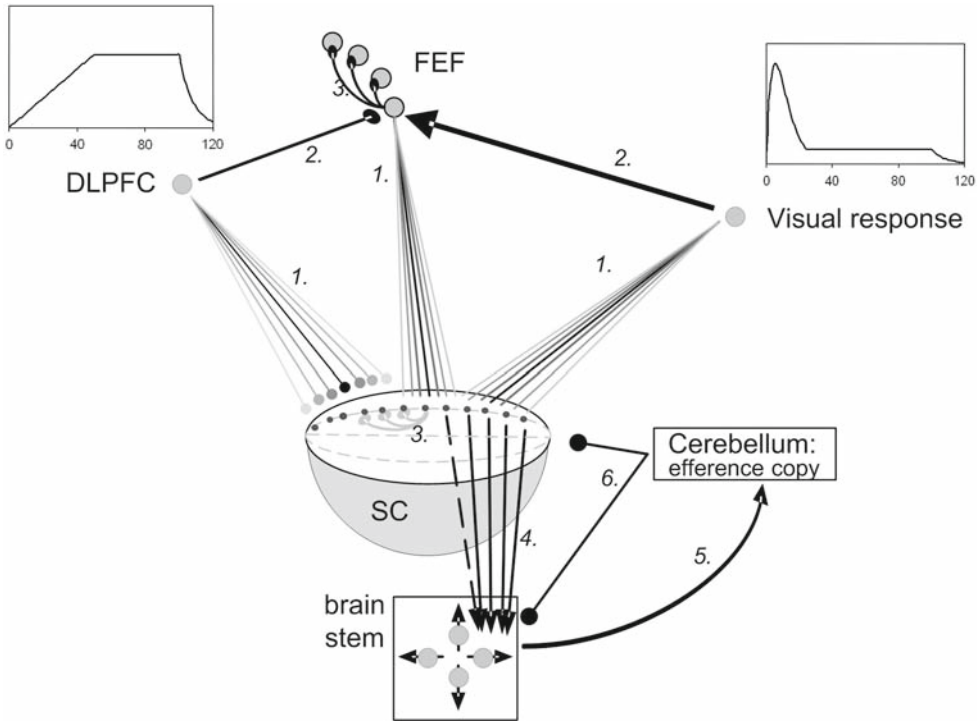

Fig. 1 Model architecture. The model contains four modules: the frontal eye fields (FEF), the SC, the brainstem saccade plant, and the cerebellum. The FEF consists of ten neurons, the SC of 4,000 neurons arranged on eight axes (*black nodes* drawn on one axis; only five axes drawn for clarity). Two types of input project to the FEF and the SC: visually evoked signals from posterior cortical areas and signals related to task demands from the DLPFC. DLPFC signals are inhibitory in SC, and can be both inhibitory and excitatory in FEF. The time course of both inputs is shown inside boxes. The SC receives input from FEF neurons which project to SC neurons with similar visual fields. Each FEF model neuron included in the model has a motor field that corresponds to one potential stimulus location. FEF neurons project to the SC. Weights on these connections follow a Gaussian distribution, with strong connections to SC neurons with the same field, and progressively weaker ones to SC neurons with progressively more different motor fields. Visual and DLPFC inputs are distributed using the same connection scheme (connections indexed with 1.). The same inputs are sent to the FEF neuron with the right movement field (connections indexed with 2.). In both the FEF and in the SC neurons inhibit one another with uniform inhibitory connections (indexed with 3.). The activity of SC neurons is decomposed into vertical and horizontal components and summed, which are relayed to four brainstem saccade systems coding for the four cardinal directions an eye movement can make (connections indexed with 4.). Activity of the brainstem saccade plant is relayed to the eye, but also to the cerebellum where an efference copy is calculated of the saccade (indexed by 5.). The efference copy is fed back to the brainstem saccade plant. During its calculation, the cerebellum also sends a uniform inhibition to SC neurons (connection indexed with 6)

In this and other equations, indexes for the neuron and the time step will be left out unless they are essential. e_{net} and i_{net} are the excitatory and inhibitory inputs from outside the layer to the neuron, respectively, and i_{lat} is lateral inhibition. V_e is the reversal potential of Na^+, V_i that of Cl^- and, V_r that of the leak current (+30, −90, and −75 mV, respectively). These reversal potentials and V were rescaled to the interval $[-1, 7]$ for computational convenience (reversal potentials became +7, −1, and 0). The time constant τ was set to 25 ms in the SC. It was set to a higher value of 50 ms for FEF model neurons as neurons in FEF tend to change their firing rate gradually, suggesting integration of inputs over time.

The firing rate was a linear function of the membrane potential, with a threshold for firing applied of −67.5 mV (recoded to +0.5):

$$f = [V - \theta]^+ \tag{2}$$

1.1.2 Input to FEF and the SC

Many brain areas project to FEF and the intermediate layer of the SC. To constrain our modeling, we focus on two inputs (see Fig. 1): visual input, e_{v}, and DLPFC input, e_{dl} and i_{dl} for excitation and inhibition, respectively. In FEF, e_{net} of Eq. 1 was a function of e_{v}, and e_{dl}. To simulate saturating synapses and interactions between stimuli, these excitatory inputs were squashed to net input, e_{net}:

$$e_{\text{net}} = 1 - 1/e^{(e_v + e_{\text{dl}})/c} \tag{3}$$

Constant c was equal to 0.5. In FEF net inhibition i_{net} was equal to i_{dl}. The SC received two excitatory inputs: visual

input e_v and FEF input e_{fef}; e_{net} was defined as in Eq. 3 with as inputs e_v and e_{fef}, and with $c = 4$. The SC received inhibitory input from DLPFC, i_{dl}, and from the cerebellum, i_{cer} (only during saccades; see below); i_{dl} and i_{cer} summed to i_{net}.

1.1.2.1 Visual inputs The first input, e_v, represents visually evoked signals reaching both FEF and the SC from posterior cortical areas. Cortical area V1 has fast projections to FEF (i.e., through area MT, Ungerleider and Desimone (1986)) and to the intermediate layers of the SC (Schiller et al. 1979). Early visual responses are remarkably similar in many areas of the occipital lobe (Gawne and Martin 2002), although the onset of the responses may differ (Schmolesky et al. 1998). We therefore inferred that bundling their visually evoked responses into one visual input would be a valid simplification.

This visual input, e_v, was assumed to be only excitatory. Its time course was taken from electrophysiological recordings from occipital cortex (Gawne and Martin 2002). Neural responses in occipital areas to a stimulus consist of a strong transient response that fades quickly, with thereafter a lower persistent response if the stimulus remains in the receptive field. At offset of the stimulus, neural firing decreases approximately exponentially (Gawne and Martin 2002). These features were captured in a function with three parts (see inset in Fig. 1a): a strong transient modeled as a double exponential, a persistent firing level modeled as a continuous function, and an offset modeled as exponential decay. The strength of visual inputs to neuron j in FEF and SC, e_v, is given below as a function of the time, $t - t_{on}$, in milliseconds since the time step t_{on} that the visual inputs arrive in the SC and FEF. During presentation of the stimulus, the function is the maximum of the transient (Eq. 4a) and the sustained signal (Eq. 4b):

$$e_v(t, j) = S_v R(l_v, l_j) b \left(e^{-\frac{t-t_{on}}{\tau_1}} - e^{-\frac{t-t_{on}}{\tau_2}} \right) \quad \text{if } b \left(e^{-\frac{t-t_{on}}{\tau_1}} - e^{-\frac{t-t_{on}}{\tau_2}} \right) > 1 \tag{4a}$$

$$e_v(t, j) = S_v R(l_v, l_j) \quad \text{if } b \left(e^{-\frac{t-t_{on}}{\tau_1}} - e^{-\frac{t-t_{on}}{\tau_2}} \right) \leq 1 \tag{4b}$$

Here S_v is the strength of the stimulus (equal to 1 for all target stimuli, and taken from a normal distribution with $\mu = 1.0$ and $\sigma = 0.1$ for distractor stimuli). $R(l_v, l_j)$ is a function of the position of the neuron, l_j, in SC or FEF, and the location with maximal input for the stimulus, l_v; it can vary from 1 when $l_j = l_v$ to 0 (see below). b is a scaling constant equal to 100. Time constants τ_1 and τ_2 had values of 5 and 6 ms, respectively. The onset of visual input in SC and FEF, t_{on}, was set at 70 ms after the stimulus is presented in the outside world, approximately equal to V1 response onset in monkey (Schmolesky et al. 1998).

After offset of the stimulus, the strength of the signal decayed exponentially to 0:

$$e_v(t) = S_v R(l_{max}, l_j) e^{-\frac{t-t_{off}}{\tau_3}} \tag{5}$$

Here, $t - t_{off}$ is the time after offset of visual input (70 ms after stimulus offset in the outside world); the time constant τ_3 equaled 25 ms.

1.1.2.2 DLPFC The second input consists of signals from the dorsolateral prefrontal cortex (DLPFC). The DLPFC is known to be responsive to task demands, with a high proportion of neurons changing their activity as a function of task. They may, for example, respond to targets and not to distractors, independent of how the target is defined on a particular trial (Everling et al. 2006). Moreover, such DLPFC neurons seem to discriminate between targets and distractors very early in the trial (Everling et al. 2006). In saccadic tasks, up to 40% of DLPFC neurons show activity related to the task (Tinsley and Everling 2002). This includes neurons that are active during fixation and neurons that seem to prepare for a saccade. As revealed with fMRI, preparation for an antisaccade activates DLPFC more strongly than any other brain region (Brown et al. 2007). Neuropsychological evidence also suggests that this area plays an important role in the control of voluntary eye movements. Damage to the DLPFC results in problems with the suppression of unwanted saccades (for a review see, Pierrot-Deseilligny et al. 2005) and impairment of the accuracy of memory-guided saccades (Funahashi et al. 1993).

The DLPFC projects densely to FEF (Pierrot-Deseilligny et al. 2005) and to the intermediate and deep layers of the SC (Goldman and Nauta 1976; Johnson and Everling 2006; Yeterian and Pandya 1991). Several findings suggest that the latter connection is primarily inhibitory (Lynch and Tian 2006). Most prominently, Johnson and Everling (2006) found that in an antisaccade task, DLPFC neurons with a confirmed connection to the ipsilateral colliculus tended to fire most strongly when antisaccades were made to the ipsilateral side, i.e., to the side that was coded for by the opposite colliculus. Johnson and Everling (2006) concluded that DLPFC neurons projecting to the SC are mostly involved in inhibiting prosaccades. They speculated that such neurons project either to fixation neurons in the rostral SC, or to interneurons in more caudal areas that contain the saccade neurons. There are also indirect connections from DLPFC to the SC, via the basal ganglia and the Substantia Nigra pars reticulara (SNr) (Hikosaka et al. 2006; Hikosaka et al. 1993; Jayaraman et al. 1977). Since SNr neurons are tonically active and are GABAergic, it is generally thought that SNr delivers a constant inhibition to saccade neurons to help maintain fixation. Consistent with this data, in the model connections from the DLPFC to SC are inhibitory. Model connections from the

DLPFC to the FEF, on the other hand, are both excitatory and inhibitory. There is little data to support this assumption. Long-range projections originating from the cortex tend to glutamatergic (Rockland 1997), suggesting a preponderance of excitation; however, DLPFC inputs may target interneurons in FEF, as they seem to do in the SC.

In addition to inhibiting some saccades, we hypothesized that DLPFC neurons are also involved in the preparation for specific saccades. If the location of an upcoming saccade is known, saccades to it are faster than if the target can appear at multiple locations (Hackman 1940). Moreover, within the SC anticipatory activity is seen at the location of the upcoming target. This activity increases with the likelihood that a saccade to the location has to be made (Dorris and Munoz 1998). Here, we propose that one way in which the brain prepares for a saccade to a particular location is through a disinhibition of SC neurons coding for that location. This may occur through direct DLPFC input to the SC, but a more likely pathway is via the basal ganglia and SNr. We base this idea on two findings. First, if targets appear at a predictable moment on a predictable location, humans will make many anticipatory saccades. Damage to the DLPFC reduces the number of such saccades significantly for all target positions (Pierrot-Deseilligny et al. 2005). Second, SNr neurons coding for a certain location pause when a memory saccade must be made to that location (Basso and Wurtz 2002). Indeed, SNr has been hypothesized to play a role in target selection by disinhibiting a specific region of the SC (Jiang et al. 2003). However, SNr neurons were shown not to be sensitive to the number of potential target locations in a visual search paradigm (while saccadic reaction time (SRT) is), which suggests that another pathway is also responsible for anticipatory activity (Basso and Wurtz 2002). This pathway, we suggest, is the excitatory one from DLPFC to FEF implemented in our model.

The form of DLPFC signals, both inhibitory and excitatory, was based on recordings by Tinsley and Everling (2002). Activity related to fixation decays over the course of some 100 ms after fixation offset. Activity related to the preparation of a saccade seems to build up approximately linearly over time and then to plateau. Accordingly, DLPFC input to SC and FEF consisted of three parts: a rise phase, a constant phase, and an offset phase (see inset in Fig. 1a). During the constant phase, e_{dl} and i_{dl} were equal to $S_{\mathrm{dl}} * R(l_{\mathrm{dl}}, l_j)$, where S_{dl} is the strength of DLPFC input and $R(l_{\mathrm{dl}}, l_j)$ the function also used above, of neuron position l_j and the location of maximal input, l_{dl}. The offset phase was the same as for visual inputs, with S_{v} in Eq. 5 replaced by S_{dl} and the time constant set to 150 ms. During the rise phase, after onset of DLPFC inputs at t_{dl}, DLPCF inputs rose linearly to S_{dl} in 50 ms. In the first 50 ms after t_{dl}, e_{dl} was thus equal to (and the same held for and i_{dl}):

$$e_{\mathrm{dl}} = \frac{(t - t_{\mathrm{dl}})}{50} S_{\mathrm{dl}} R(l_{\mathrm{dl}}, l_j) \tag{6}$$

The onset of DLPFC inputs, t_{dl} trailed onset of visual inputs, t_{on}, by a fixed 30 ms. The onset of DLPFC input buildup is thus 100 ms after stimulus presentation, and its duration is 50 ms. Although other values might also be defensible, these seem to be a reasonable estimate given neural firing profiles reported by Tinsley and Everling (2002), and onset of firing data given by Schmolesky et al. (1998).

A critical assumption concerns the locus of variability. We assume that there is relatively little variance in the strength of visual signals (i.e., as long as the eyes remain open, a stimulus with a given intensity will generate approximately the same response in early visual areas). On the other hand, we assume sizeable variation in the strength of DLPFC signals from one trial to the next. In effect, variance in DLPFC signals is the main determinant in the model of SRT. Underlying is the idea, also suggested by others (de Jong 1999) that preparation for a trial is variable, and that the extent of preparation determines whether an observer is able to react swiftly to a stimulus or not. With regard to the eye movement system, there is a wealth of data that suggests that activity preceding the onset of the saccade can predict its speed (e.g., Connolly et al. 2005; Dorris et al. 1997; Ipata et al. 2006).

1.1.2.3 FEF FEF neurons also project to SC (e.g., Huerta et al. 1986). Responses in FEF are somewhat mixed. They are clearly responsive to task demands, which is shown for example by different responses to targets and distractors (Bichot and Schall 2002), and different responses in the pro- and antisaccade tasks (Everling and Munoz 2000). However, purely visual responses also have been reported (Bruce and Goldberg 1985), and early firing is not always different for targets and distractors (Bichot and Schall 2002). All these responses are present in FEF cells that project to the SC (Sommer and Wurtz 2001).

In the model, input from FEF to SC, e_{fef}, was twice the sum of the firing rate of all FEF neurons j on time step $t - d$ (d being a synaptic delay of 10 ms), $f_{j,t-d}$, multiplied by $R(l_{\mathrm{fef}}, l_j)$, a function of the location of the SC neuron and the location receiving maximal input from the FEF neurons (l_{fef}):

$$e_{\mathrm{fef}} = 2 \sum_j f_{j,t-d} R(l_{\mathrm{fef}}, l_j) \tag{7}$$

1.1.2.4 Topology of inputs The ten FEF neurons projected to SC model neurons with similar visual fields. Visual, DLPFC, and FEF inputs to SC show a coarse topology, in that cortical cells tend to project to many neighboring neurons in SC (Funahashi et al. 1990; Komatsu and Suzuki 1985; Lock et al. 2003; Sommer and Wurtz 2001). This implies that

collicular neurons have relatively broad visual fields, which is indeed the case (Dorris et al. 2007; Ottes et al. 1986; Schiller et al. 1980). The spatial shape of the inputs to SC was a Gaussian around an SC location receiving maximum input $l_{\max}$. At location l_j of neuron j its strength was:

$$R(l_{\max}, l_j) = e^{-\mathrm{dist}(l_{\max}, l_j)^2/c} \tag{8}$$

Here, dist(x, y) is the Euclidian distance in mm (which translates into distance in model neurons divided by 100), and c is a constant equal to 0.245 with unit $^1/_{\mathrm{mm}}$. This function is 1 at location $l_{\max}$ and drops to zero if l_j is far away from $l_{\max}$. Both the equation and the parameter values were taken from Trappenberg et al. (2001), who based their Gaussian kernel on electrophysiological recordings.

In FEF, all neurons were assumed to correspond to one position at which a visual stimulus could appear. $R(l_{\max}, l_j)$ was equal to 1 for that neuron, and equal to 0 for all others.

1.1.3 Lateral interactions

In several models, most prominently that of Trappenberg et al. (2001), lateral interactions between intermediate layer collicular neurons were assumed to have a Mexican hat function, with short-range excitation and long-range inhibition. Little evidence for such interactions exists, however. In a study of lateral interactions within the intermediate layers of the SC, Munoz and Istvan (1998) found mostly inhibitory interactions, with lateral excitation only between contralateral fixation zones (they found two saccade cells excited by ipsilateral stimulation, but could not establish a spatial gradient in the interactions). We therefore chose the simplest scheme consistent with the evidence, which is lateral inhibition between all modeled SC neurons with uniform strength. Lateral inhibitory interactions were also assumed in FEF. There is little evidence to date on whether inhibitory interactions exist in FEF, but such interactions are common in the neocortex (Braitenberg and Schüz 1991).

Using t as an index for the time step, lateral inhibition was a function of the firing rates, $f_{j,t-d}$, of all other model neurons in the layer d time steps earlier (d, modeling synaptic delays, was set to 3 ms):

$$i_{\mathrm{lat}} = s \sum_j f_{j,t-d} \tag{9}$$

The strength of lateral inhibition s was set to 0.01 in SC, and 0.9 in FEF (as there are many more model neurons in SC lateral inhibition was actually about as strong in SC as in FEF).

1.1.4 Saccade initiation and trajectory

To generate saccades from SC activity, we implemented a model of the brainstem saccade generator that accounts well for both saccade characteristics and brainstem recordings (Becker and Jürgens 1990; Van Gisbergen et al. 1981). The Van Gisbergen et al. (1981) model describes how a drive from outside the brainstem leads to a burst of firing in brainstem burst neurons that, via intermediary steps, control the muscles of the eye. Firing of the burst neurons is reduced via a feedback loop that relies on an efference copy of the saccade, an internal copy of the movement of the eyes computed from brainstem output. This efference copy is subtracted from the outside drive, leading to a smaller and smaller drive to the burst neurons the closer the saccade is to completion. In effect, burst neurons choke themselves off when the saccade is near completion.

Becker and Jürgens (1990) proposed an extension of this one-dimensional model to a two-dimensional model that can account for oblique saccades. They duplicated the model of Van Gisbergen et al. (1981) to create a horizontal and vertical components of saccades. These together determine saccade direction. This is in keeping with evidence that long-lead burst generator neurons (LLBNs), the main recipients of SC input, code mainly for saccades in the vertical and horizontal directions (Hepp and Henn 1983), with oblique saccades resulting from an activation of LLBNs coding for horizontal and for vertical saccades. The horizontal and vertical components interact via inhibitory connections. We implemented this, using parameter values given by Van Gisbergen et al. (1981) in Table 3 ('Normal' line, but with $b_{\mathrm{m}} = 1300$ as in their Fig. 11D), and the coupling factor c given by Becker and Jürgens (1990).

To link this brainstem model to the SC, we made five assumptions:

- Activity in the SC sums to a horizontal and a vertical drives to the brainstem system.
- A saccade is generated whenever the drive to either the horizontal or the vertical EBNs crosses a threshold.[2]
- The length of the saccade is determined by a weighted average of activity in the SC.
- Activity within the SC continues to influence the direction of the saccade while it is programmed and executed.
- The efference copy is computed in the cerebellum.

The first assumption is that activity in the whole SC is summed into a horizontal and a vertical drives. To compute

[2] The movement may also be achieved by combinations of eye and head movements. Bergeron et al. (2003); Soetedjo et al. (2002). We only implement eye movements because most relevant research has been done on head-fixed participants.

these we made use of the fact that SC neurons were placed on radial axes. All neurons on axis a coded for eye movements in the same direction Φ_a. (there were eight axes per colliculus at regular intervals, so that Φ_a could take values from 0° to 337.5° in increments of 22.5°, where 0° refers to a straight saccade to the right). We summed activation of all neurons on an axis, then broke it into vertical and horizontal components. These, summed, were the horizontal (H) and vertical (V) drives to brain stem:

$$H = \sum_a \cos(\Phi_a) \sum_{j \in a} f_j \tag{10a}$$

$$V = \sum_a \sin(\Phi_a) \sum_{j \in a} f_j \tag{10b}$$

Here, a ranges over all axes and j over all neurons on axis a.

The second assumption follows other models of saccade initiation (Findlay and Walker 1999). What seems to determine the exact onset of a saccade is a sharp decrease in the output of brainstem omnipause neurons, which fire at high frequencies during fixation. What causes this decrease is not known—one obvious source, a drop in drive from SC fixation neurons, is ruled out by the fact that fixation neuron activity does not correlate very well with omnipause pausing (Everling et al. 1998), and by evidence that omnipause neurons are silenced not by a drop in excitation but by glycine-mediated inhibition (Kanda et al. 2007). The saccade-related burst in SC is an obvious source of this inhibition, although it is as yet unclear how this burst translates into inhibition of omnipause neurons (for more evidence that the burst in SC is related to saccade initiation see Hanes and Schall 1996; Munoz and Schall 2003; Soetedjo et al. 2002). In the model, a saccade was initiated if either H or V crossed a threshold of 35 (for rightward or upward movements) or −35 (for leftward or downward movements).

The third assumption that a weighted average of activity in the SC determines the length of the saccade was a heuristic one (see Goossens and Van Opstal (2006) for a more sophisticated proposal for how SC activity could set the length of saccades). The model of Becker and Jürgens (1990) takes as input a desired eye displacement vector with a certain direction, Φ, and length, r. To compute both in a simple way from activity in the SC, we computed direction Φ as $\tan^{-1}(V/H)$. The desired length of the saccade, r, was computed from the optimal eccentricity for each model neuron given its position on the collicular map. r was set to the average of these eccentricities, weighted by firing rate:

$$r = \frac{\sum_J \left[A\sqrt{e^{2u/B_u} - 2e^{u/B_u}\cos(v/B_v) + 1} \right] f_j}{\sum_j f_j} \tag{11}$$

The formula between brackets is the inverse given by Van Gisbergen et al. (1987) of the formula of Ottes et al. (1986). It takes as inputs u, the distance in mm of neuron j from the rostral pole, and v the distance from the midline of the colliculus. Constant A, the rostral-to-caudal size of the colliculus, was set to 4 mm, B_u to 1.4 mm, and B_v to 1.8 mm/rad (taken from Van Gisbergen et al. 1987).

The fourth assumption listed above may be the most controversial: we follow Walton et al. (2005) in assuming that while saccades are executed, the desired saccade vector as computed from SC activity is constantly updated. This took the form that the desired eye displacement vector (r, Φ) is updated on every time step for as long as the saccade lasts. Large shifts in SC activity during a saccade can thus redirect the saccade to another point. Such an assumption seems justified in the face of evidence that saccades can be redirected mid-flight (Amador et al. 1998), and that saccades can be curved under the influence of for example distractors in the visual field (Ludwig and Gilchrist 2003; McPeek et al. 2003; Port and Wurtz 2003; Van der Stigchel and Theeuwes 2005; Walker et al. 1997). We do not claim that the SC determines the exact trajectory of a saccade, since there is enough evidence that it does not (Bergeron et al. 2003; Goossens and Van Opstal 2000; Quaia et al. 1998). Instead, we suggest that through continuous input to the brainstem LLBNs, the SC influences the trajectory. This assumption is consistent with existing models of the brainstem saccade machinery. In those models (Gancarz and Grossberg 1998; Goossens and Van Opstal 2006; Lefevre et al. 1998; Quaia et al. 1998), SC outputs keep featuring in the computations of the weighted average during the generation of a saccade. Thus changes in SC input to the brainstem (Gancarz and Grossberg 1998) or the cerebellum (Lefevre et al. 1998) should result in a changed saccade endpoint.

The latency at which the threshold is reached cannot be directly compared to saccade latencies, as there is an afferent delay between burst activity within SC and saccade initiation of about 25 ms (Munoz and Wurtz 1995b). Adding this delay to the time at which the threshold is reached yields latencies that can be compared to observed saccade reaction times.

1.1.4.1 Role of the cerebellum In Van Gisbergen et al.'s (1981) model an efference copy of the saccade, denoted by E, is subtracted from the drive to the brainstem saccade generator. Such an efference copy was already proposed by von Helmholtz in the nineteenth century, but it has proven hard to find neurons computing an efference copy of saccades. Optican and coworkers (Lefevre et al. 1998; Quaia et al. 1998) have proposed that the cerebellum computes what is in effect an efference copy through a spatial code. Although this idea is by no means uncontroversial, we concur that the cerebellum is a likely location for the efference copy (our fifth assumption above). The cerebellum is assumed to inhibit the brainstem saccade generator in proportion to E, the part of the saccade already made, in this way generating the

feedback loop that is part of the Van Gisbergen et al. (1981) model. Moreover, the cerebellum is also assumed to inhibit the SC in proportion to E. Indeed, evidence exists that the caudal fastigial nucleus of the cerebellum, the area proposed by Lefevre et al. (1998) to hold the efference copy, sends inhibitory projections to SC saccade neurons (Guillaume and Pelisson 2001) with a synaptic delay d of 10 ms. Thus, i_{cer}, the last input to the SC introduced above, is an inhibition of the whole SC present whenever a saccade is made:

$$i_{\text{cer}} = 0.1\dot{E}_{t-d} \tag{12}$$

1.1.5 Simulation setup

Before discussing the different paradigms used to test the model, we first introduce the general setup during a simulated trial and the input parameters used in all simulations. Every trial starts with a fixation point in the middle of the visual field. The presence of the fixation stimulus is simulated by two components: one is generated by the visual signal of the presence of the fixation point (strength S_v of 0.25 in arbitrary units). The other is endogenous DLPFC input to FEF representing the top-down focus on the fixation point (strength S_{dl} sampled from a normal distribution clipped at 0 with mean 0.3 and standard deviation, s.d., of 0.2). Once the fixation point is removed, visual and DLPFC responses at the fixation location start diminishing after 70 and 100 ms, respectively.

During the whole simulation, DLPFC inhibits all SC neurons except those coding for fixation with a strength of $(1 - R) * 0.4$. Here, R is equal to the weight from an input at fixation as defined in Eq. 8; since R goes from 1 to 0, $1 - R$ is 0 at the most rostral SC location, increasing to 1 further out on the axes. In paradigms in which the location of the upcoming target is known, an anticipatory DLPFC input is subtracted from this inhibition at that location with a strength S_{dl} sampled from a normal distribution clipped at 0 with mean of 0.3 and s.d. of 0.2. The same input is given to FEF. In paradigms in which the location of the target is not known (e.g., visual search paradigms) there are no such inputs.

At the presentation of a target or distractor, a visual signal was generated at the location of the stimulus (strength S_v sampled from a normal distribution with mean 1 and s.d. = 0.1 for distractor and S_v was a fixed 1 for targets; as mostly the *relative* strength of stimuli is important, varying all strengths would be overkill). All stimuli were presented at locations halfway fixation and the end of the saccade map. As SC visual fields have a well-known logistic warping relative to outside space (Ottes et al. 1986), this location is equivalent to approximately 10° of eccentricity. Targets were always presented on the horizontal axis; distractors could be presented on the remaining axes. Targets and distractors were distinguished through modulations of DLPFC-activity: the distractor location, and not the target location, was inhibited with a strength S_{dl} sampled from a normal distribution with mean 0.25 and s.d. 0.1.

In most paradigms, observers have to make an eye movement toward a target. In antisaccade paradigms, the target signals that an eye movement has to be made to some other location, usually at the opposite side of fixation. In antisaccade paradigms, DLPFC inputs were set to be negative at the location of the target to inhibit a saccade toward the target (strength S_{dl} sampled from a normal distribution with mean 1 and s.d. 0.2), and positive at the location to which an eye movement has to be made (strength S_{dl} sampled from a normal distribution with mean 0.65 and s.d. 0.1).

In all simulations, top-down signals could vary in strength. To capture the effect of these variations on saccades, we sampled from the range of each parameter at regular intervals of 0.05, and ran each simulation for all combinations of parameter values. The full distribution of outcomes was then reconstructed by multiplying each outcome with the probability with which the underlying combination of values occurred.

2 Results

2.1 Knowing the target location

If an observer in an eye movement study knows where the next target will appear, saccades to that target are faster than when the location is unknown in advance. In the model, the first situation is equivalent to having an anticipatory DLPFC signal at the location of the upcoming target, while the second is equivalent to having no such signal. In the first situation, SRTs are some 30 ms lower than in the second (see Table 1), reproducing the behavioral finding (Dorris and Munoz 1998; Hackman 1940). This effect was much larger for paradigms with a distractor, which is not surprising but has not been tested directly yet.

2.2 Gap effect

The *gap effect* refers to the reduction in saccade latency when the fixation stimulus disappears before the onset of the target, compared to when the fixation remains present (Bekkering et al. 1996; Kingstone and Klein 1993; Saslow 1967). The gap period then denotes the time between the offset of fixation and the presentation of the target. This robust reduction in latency is independent of advance knowledge of the location of the saccade target (Walker et al. 1995). It can be partly reproduced by presenting an auditory signal to announce the target, while the fixation stimulus remains present (Kingstone and Klein 1993; Reuter-Lorenz et al. 1991), indicating that there are two components to the gap effect. First,

Table 1 Saccadic latencies in ms (SRTs, mean and SD) produced by the model in the paradigms studied in this article, always for the situation in which the position at which the target would appear was known in advance, or was unknown (e.g., in visual search)

Paradigm	Target pos. known		Target pos. unknown	
Standard saccade	206	(38)	241	(35)
Gap 100 ms	153	(27)	185	(18)
Gap 200 ms	123	(25)	167	(9)
Warning signal	186	(25)	215	(17)
Antisaccade (no gap)	393	(67)	390	(53)
Antisaccade (gap)	380	(82)	382	(55)
Distractor (close)	207	(55)	295	(59)
Distractor (far)	225	(48)	328	(55)

For comparison with real saccade latencies, a 25 ms. afferent delay was added to the time at which SC activity reached threshold

both the removal of the fixation stimulus and the tone function as a warning signal by providing temporal information about the target appearance (Ross and Ross 1980; Ross and Ross 1981). The second component, not produced by the tone, is related to the visual offset. We equate the first component with an offset of the DLPFC input at the fixation point, the second with an offset of visual signals.

In our simulations, we compared a no-gap condition in which fixation offset and target onset occurred simultaneously, with gap conditions in which we introduced a gap between fixation and target onset of either 100 or 200 ms. In the gap no stimulus was present. Then a target stimulus appeared on the horizontal axis on the left of the fixation location.

In our model, the average SRT in the 100-ms gap condition is 153 ms after target presentation, whereas in the no-gap condition the threshold is reached 206 ms after appearance of the target. This implies a gap effect of 53 ms, roughly the effect size observed in behavioral studies (e.g., Shafiq et al. 1998). When the gap period is extended to 200 ms the gap effect increases to 83 ms (see Table 1), in line with Saslow (1967) who showed that longer gap periods results in increasing gap effects. We further simulated a non-visual warning signal that announces the upcoming target by removing DLPFC input at fixation. Because the fixation point remains visible, visual input at fixation remains constant (with corresponding lateral inhibition). In line with behavioral results (Kingstone and Klein 1993; Reuter-Lorenz et al. 1991) this results in a smaller gap effect than when the fixation stimulus is removed (20 vs. 53 ms).

2.2.1 Express and anticipatory saccades

An important feature in the gap paradigm is the bimodal distribution of saccade latencies. The fastest saccades in this distribution are called *express saccades* (Fischer and Boch 1983; Fischer and Ramsperger 1984), which have extremely short latencies as compared to 'normal' eye movements (±100–120 vs. ±150–250 ms). Our model produces a sizeable number of express saccades in gap conditions (around 50% with a 200-ms gap; see Fig. 2).

At very high levels of anticipatory input, some anticipatory saccades were seen, defined as saccades that are initiated before the stimulus has been presented (see Fig. 2). With the parameter values chosen here, these saccades were only seen in the 200-ms gap condition.

2.3 Activity profile of SC model neurons

Figure 3 shows the mechanisms that cause the gap effect in the model. In Fig. 3a, the arrow denotes the time step at which the offset of the fixation point starts affecting SC activity. This offset reduces activation at the fixation location in the SC saccade map, as was reported by Dorris and Munoz (1995). As a result, the lateral inhibition in the SC map is reduced. When the target stimulus arrives, this results in stronger activity of saccade neurons at the target location, also shown in Fig. 3a, than when the fixation stimulus is not removed. At the target location, activity of model saccade neurons shows two consecutive peaks: a first peak when the stimulus is presented, and a second peak that results in a saccade. This is also seen in the SC (e.g., Fig. 3c). The first peak is the result of the strong transient activity seen throughout the visual system with a visual onset (e.g., Gawne and Martin 2002). The second peak, usually referred to as the *motor burst*, results when visual and FEF input combine at the target location. Since FEF neurons take some time to reach a high level and since the pathway over FEF involves one more synapse, this second peak is delayed relative to the first visual one.

As the activity evoked by the fixation point slowly decreases after its offset, introducing longer gap periods results in lower activity at fixation point and therefore less lateral inhibition of neurons coding for saccades. This in turn leads, after target onset, to a faster buildup of target-related activity in SC, and thus to faster saccades. In the 200-ms gap condition, the decrease in lateral inhibition is large enough to

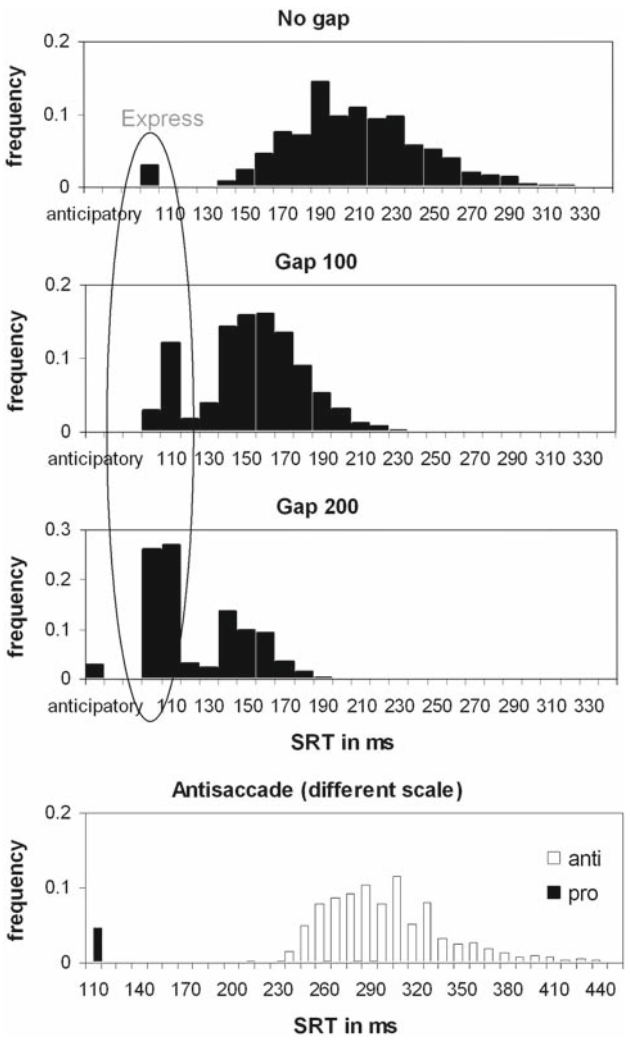

Fig. 2 Saccadic reaction time (SRT) distributions, in bins of 10 ms, for the no-gap, 100-ms gap, 200-ms gap, and antisaccade conditions. Saccades below 125 ms inside the *circle* are regarded express saccades. The model produces more express saccades in the 200-ms gap condition than the 100-ms gap and no-gap conditions. It produces some anticipatory responses (defined as responses before the stimulus arrives) in the 200-ms gap condition. In the antisaccade simulation, it produces very fast prosaccades and some slow ones, with antisaccades being slower

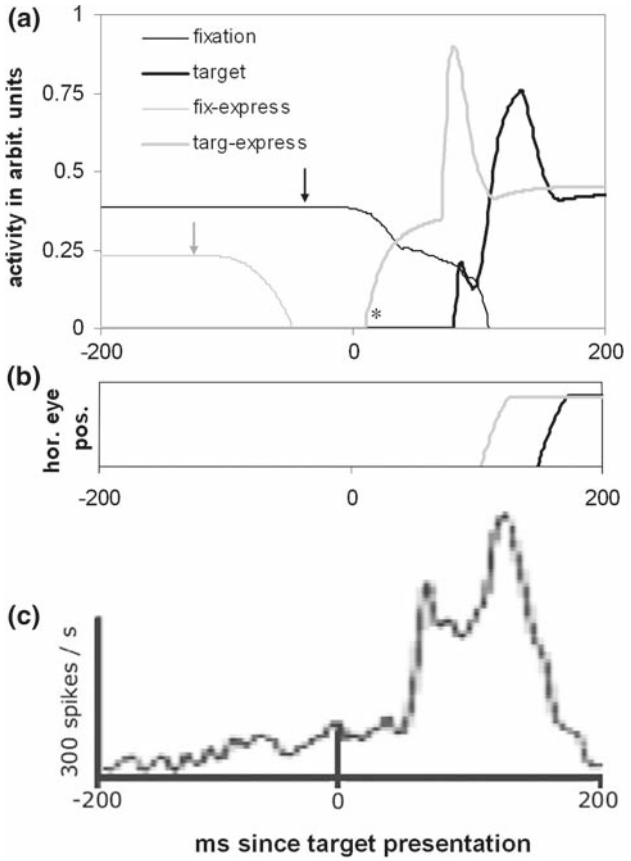

Fig. 3 **a** Activity at rostral fixation location and target location in the SC during two saccades. The *black lines* were generated with a gap between fixation offset and target onset of 100 ms and parameters at their mean values (DLPFC signal at fixation: 0.3; anticipation signal: 0.3). Time point zero corresponds to the time of target presentation. There is an initial peak of visual activity 70 ms after the stimuli are presented, and then a sustained increase of activity that results in a saccade. *Gray lines* give results for a trial in which the model generates an express saccade (gap of 200 ms, parameter values: DLPFC signal at fixation: 0, anticipation signal: 0.2). In this trial, there is a buildup of activity before visual signals arrive at around 70 ms. The start of the buildup activity is indicated with an *asterisk*. **b** Horizontal eye position in the saccades for which SC activity is plotted in panel (**a**), colors as in (**a**). **c** Recording from a SC build-up neuron in a 200-ms gap condition. The neuron shows a build up of activity during the gap, then a peak of visual activity, followed by a second peak of motor-related activity (Munoz and Wurtz 1995a, from their Fig. 5D; reproduced with permission)

sometimes lead to express saccades (gray lines, see below). These activation patterns reflect typical electrophysiological recordings in the SC, as can be seen in the example in Fig. 3c, which shows responses of buildup cells in the SC (Munoz and Wurtz 1995a). Also the drop in activity seen in fixation neurons in SC is mirrored in our model, though the drop tends not to be as drastic in the brain as in our model (Dorris et al. 1997).

Express saccades were found in trials in which there was a relatively small DLPFC input to fixation neurons, and/or a relatively large anticipatory signal at the target location. Both result in less-than-average firing of fixation neurons at the start of the trial, and some buildup of activity in SC neurons coding for the target location (see Fig. 3a for an example of buildup activity leading to an express saccade, and Fig. 3c for an example in the brain). This buildup of activity allows the visual peak in SC activity to reach the threshold, resulting in a very fast saccade. The bimodality of saccade latencies is thus an emergent characteristic of the model (see for similar accounts Dorris et al. 1997; Trappenberg et al. 2001).

Figure 3b shows the simulated eye movement resulting from the SC activation shown in Fig. 3a. Plotted is the horizontal eye displacement (the vertical component was zero in this simulation). The speed and endpoint of the

express and regular saccade are virtually identical, as they are controlled by the brainstem saccade generator. Next to determining the saccade, brainstem activity also inhibits activity in the superior colliculus via cerebellar feedback. This results in the drop in activity seen in Fig. 3a after the initial burst.

Figure 3a also shows that after the motor burst, activity at the target location does not fall back to low values (as they do in the brain) but remain relatively high. This reflects the fact that inputs to the SC remain constant in the model while the saccade is being made and after it. In the brain, suppression of visual activity during saccades may take away input to the SC, but this was not simulated. However, the remaining drive does explain a finding that is somewhat puzzling without it. Current injection into the rostral fixation zone of the SC can stop a saccade (Munoz et al. 1996), presumably because the activation of fixation neurons acts as a brake. With injection durations below 100 ms, both the burst and the saccade resumed after current injection stopped, resulting in a burst with about the normal number of spikes in it and a saccade with a normal amplitude (see Fig. 4b). Our model reproduced these findings to a surprising degree: setting activity of model SC fixation neurons (i.e., those in the middle of the axes) artificially at a level of 3 (−30 mV) resulted in an interrupted SC burst and an interrupted saccade that both resumed after activation of fixation neurons was stopped. The only mismatch between simulation and data was that in the model the saccade went slightly *backward* during the break. This is not likely to occur in reality, but in the model results from the fact that fixation neurons also code for saccades of very small amplitude; activating these thus effectively sends a signal to the brain stem that a very small saccade is needed. Nevertheless, the broad match between data and simulation validates our assumption that the burst of activity in SC does not end because of passive dissipation or because cortical drive falls away, but because of active inhibition during the saccade. Whether this inhibition is delivered via the cerebellum (as we have assumed) or is, for example, generated within the SC remains an open question.

Figure 4 shows that interrupted saccades had normal amplitude. This occurs because the brainstem saccade generator is driven by the mismatch between the desired gaze displacement, given by the SC input, and the efference copy of the movement computed by the cerebellum. Since at the interruption half of the saccade has already been made, the saccade after interruption only has the amplitude needed for the eyes to land at the right endpoint. This mechanism may also explain the seminal results of Sparks and Mays (1980; 1983). They stimulated the SC right before a saccade to an already obscured target. They found that the eye movement caused by the stimulation was corrected with a very fast saccade to the now-obscured target location. This would also result in our model because any eye movement created by stimulating the SC is integrated into the efference copy. After

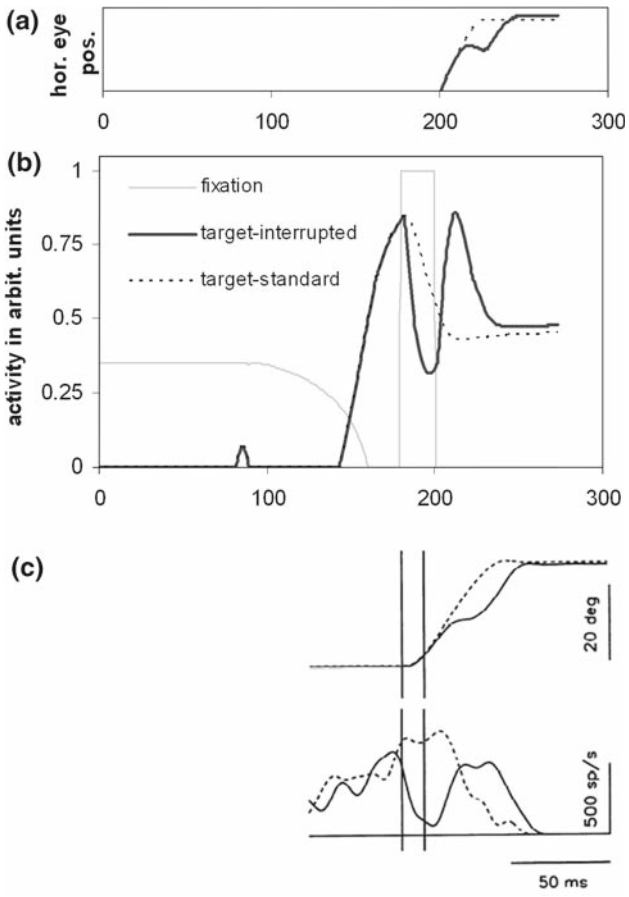

Fig. 4 **a** Horizontal eye position during a normal and an interrupted saccade generated by the model (vertical eye position did not change during the saccade). Both saccades are generated in a no-gap simulation with DLPFC signals at standard values (fixation: 0.3, anticipation: 0.3). During the interrupted saccade (but not the standard saccade) a current injection at the rostral fixation zone for 20 ms was simulated by setting the activity of central fixation neurons to 3. **b** Activity of rostral fixation model neurons and of saccade neurons at the target location for the normal and interrupted saccades. Fixation neuron activity is clipped at 1 in the figure for better visibility. **c** Eye position and neural firing in at SC target position during a standard and an interrupted saccade in the study of Munoz et al. (1996, from their Fig. 5D), reproduced with permission

stimulation, the brainstem saccade generator would get an input equal to the mismatch between the efference copy and the target location as indicated by SC activity. In effect, the brainstem saccade generator would thus automatically correct for the error created by the stimulation. Of course, such a result is dependent on the assumption that the efference copy is not reset immediately after a saccade. The data of both Sparks and Mays (1983) and Munoz et al. (1996) point to an interval of some 100 ms after saccade onset in which the efference copy is intact—after that, saccades do not seem to resume anymore, but to be superseded by new saccades.

2.4 Antisaccades

In the antisaccade paradigm, a visual onset is presented and observers have to make an eye movement away from the onset location to the mirror position on the opposite side of fixation (Everling and Fischer 1998; Hallet 1978; Munoz and Everling 2004). Typically, antisaccades have longer latencies than saccades toward an onset. Moreover, observers frequently make erroneous saccades toward the onset location, an error referred to as an erroneous prosaccade. Successful performance on the antisaccade task thus seems to require two steps: top-down inhibition of a reflexive saccade to the onset location, and then executing a voluntary eye movement to the mirror location of the onset. A prosaccade in this paradigm can therefore be seen as a failure to inhibit the reflexive saccade.

As discussed above, both neuropsychological (Guitton et al. 1985; Pierrot-Deseilligny et al. 2005) and electrophysiological findings (Johnson and Everling 2006) have suggested an important role for the DLPFC in inhibiting the prosaccade. Inhibition from the DLPFC to the SC and FEF stops reflexive saccades, allowing a second DLPFC signal to produce the antisaccade. Without sufficient inhibition activity in the SC related to the onset reaches threshold before the correct position can be endogenously activated, producing an erroneous prosaccade.

In our antisaccade simulation, an onset is presented on the horizontal axis. Inhibition is then applied to this location and the DLPFC activates the correct location. This results in antisaccades that are about 50 ms slower in our model than visually guided saccades (see Table 1). A typical observation in the antisaccade task is that the size of the gap effect is reduced compared to prosaccades (Reuter-Lorenz et al. 1995). Our model replicates this finding, producing a gap effect in antisaccades of 18 ms. In our model, the gap effect results from a diminished lateral inhibition in the gap condition. In the antisaccade paradigm, lateral inhibition is driven by activity not only at fixation, but also at the onset location. This reduces the impact of a diminished activity at fixation in the gap condition.

In our model, erroneous prosaccades result when inhibition is too small to stop the visual onset of the target from producing a saccade; such visually produced saccades tend to occur only close to target onset (see Fig. 2). These prosaccades are thus mostly very fast saccades: 4.5% of saccades in the antisaccade paradigm were fast erroneous prosaccades (9.7% in the antisaccade paradigm with gap). In most antisaccade studies, erroneous prosaccades are made on 10–20% of trials, and indeed tend to be the fastest saccades (e.g.,Bell et al. 2000; Everling et al. 1999; Kristjánsson et al. 2004; Mokler and Fischer 1999). Observers usually also produce some prosaccades with a normal latency. These were rare in the model with chosen parameter values, but did occur (<0.1%).

Exploratively, we also looked at the effect of varying stimulus strength on pro- and antisaccades. By plotting the latency of pro- and antisaccades as a function of stimulus strength, one can derive what the model predicts when contrast of the target stimulus changes. Figure 5 shows that, while prosaccades become faster when the target has a higher strength, antisaccades are slowed down. This is in line with findings from Kristjánsson et al. (2004). These authors also found that for very low-contrast targets, both pro- and antisaccades became slower. This finding is also reproduced, qualitatively albeit not quantitatively, by our model (see left-hand side of Fig. 5).

The necessity of the inhibition component was determined by running a simulation without inhibition. This simulation

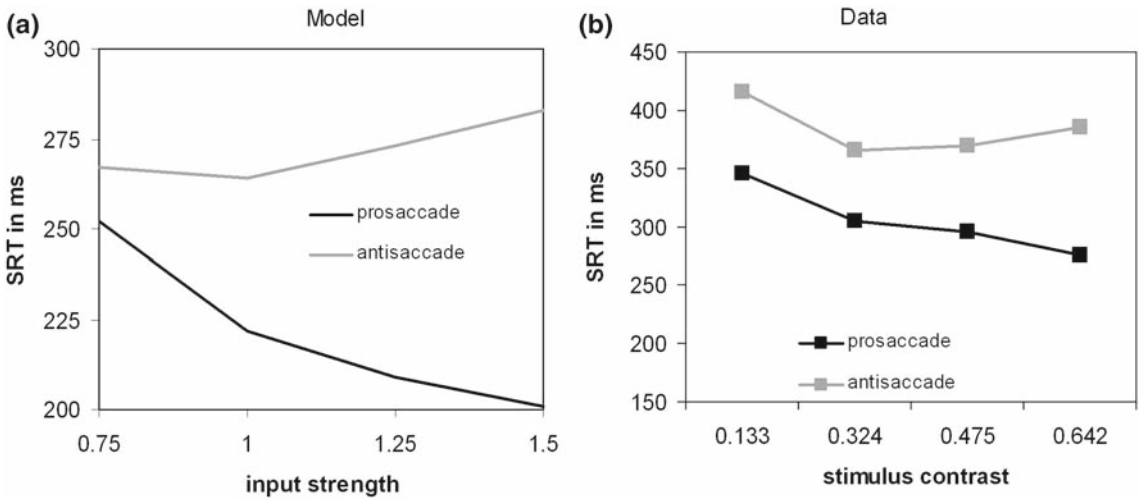

Fig. 5 **a** Saccade latencies in the model for pro- and antisaccades as a function of input strength. **b** Experimentally observed latencies of pro- and antisaccades as a function of stimulus contrast. Redrawn from the data of Kristjánsson et al. (2004)

showed that the lack of inhibition resulted in mostly erroneous prosaccades to the onset location. When no prosaccade was made, antisaccades tended to be very slow as the resolution of competition between the pro- and antisaccade goal took very long. The inclusion of inhibition at the site of the visual stimulus is therefore crucial within the model.

2.5 Distractors

In the paradigms discussed so far only one stimulus was presented. Another set of phenomena has been observed when two visual stimuli are presented after fixation removal. In the standard double onset paradigm one of the presented stimuli is the target and the other is the distractor (e.g., Ottes et al. 1985; Walker et al. 1997). This distinction is based on the instructions given in the experiment, which implies that top-down activation marks the visual element to which the participant has to execute a saccade.

In our simulation, both onsets are presented simultaneously. Targets are presented on the horizontal axis, distractors on one of the other axes at equal eccentricity. Distractors are presented on either an axis positioned 22.5° away from the horizontal (close distractors) or positioned at 67.5° (far distractors). The strength of the visual signal of the two stimuli is on average the same (though with some variance in the strength of distractor stimuli to simulate visual noise). Inputs from DLPFC do differentiate between the two, as we assume that distractor locations are inhibited. This allows competition between the two saccade goals to be resolved. Moreover, we looked at a situation in which the observer knows where the target will be presented, and at a situation in which the observer does not know beforehand where it will occur (i.e., a visual search situation).

An important factor in the double-onset paradigm is the distance between the two elements. When the two stimuli are far apart saccade latency is increased relative to a single target condition, a finding known as the remote distractor effect (Levy-Schoen 1969). The increase in saccade latency disappears when the two onsets are placed within 20° or 30° of angular distance (Walker et al. 1997). In this situation, the eyes often land on the intermediate location, an effect first described by Coren and Hoenig (1972) and referred to as the *global effect* (Findlay 1982).

2.5.1 Saccadic latencies and response profiles

In the model, close distractors neither speed nor slowed saccades relative to the no-distractor standard saccade. With far distractors (angle from target 67.5°), saccade latencies are increased (20 ms when target location is known; see Table 1). The increase in latency is the same for all distractor locations outside a zone of ±50°. This pattern corresponds to what is observed in behavioral experiments: an increase in latency

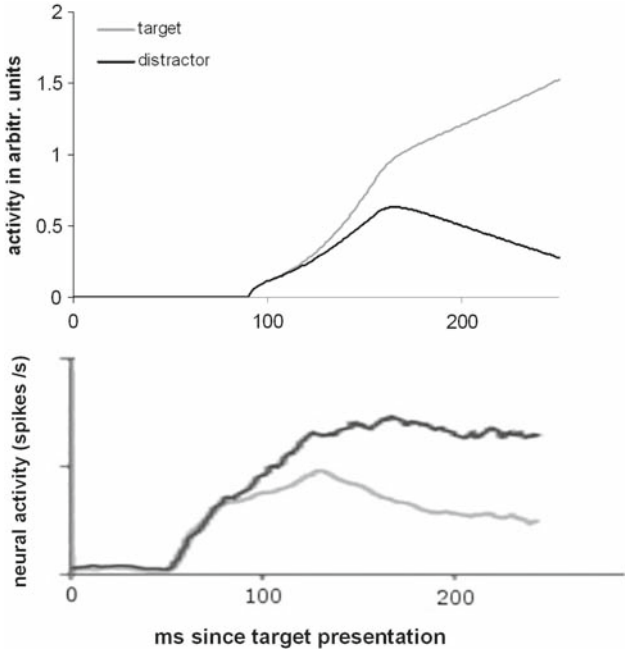

Fig. 6 FEF activity for target and distractor during resolution of competition, for the model (*top*) and in the macaque monkey (*bottom*) (Bichot and Schall 2002, from their Fig. 8A). In both, activity starts out the same for target and distractor, but activity at the distractor location decreases halfway the buildup. *Bottom* panel reproduced with permission

with far distractors which is independent of distance (Walker et al. 1997). For both kinds of distractors, SRTs were much longer when the model was not told where the target would occur, akin to the situation in visual search.

Although inhibition from DLPFC at the distractor position eventually resolves competition between the target and distractor, activity at the distractor location results in lateral inhibition that slows activation buildup at the target location in FEF and SC (as has been found by Basso and Wurtz 1998). Figure 6 shows the activity for targets and distractors in the FEF. It can be seen that the response starts out the same for target and distractor, but whereas activity for targets builds up to a maximum, activity at distractor locations decreases again halfway in the buildup. This is similar to what is observed in electrophysiological recordings of the FEF (Bichot and Schall 2002). Figure 7 shows resulting activation in the SC just before a saccade is made: there is a strong peak of activity at the location coding for the target, with a small and diminishing second peak at the location of the (far) distractor.

Again, the necessity of the inhibition component was investigated by running a simulation without inhibition of the distractor location. In this simulation, competition between target and distractors was generally not resolved. In our model, inhibition is thus crucial for fast responding in the presence of distractors.

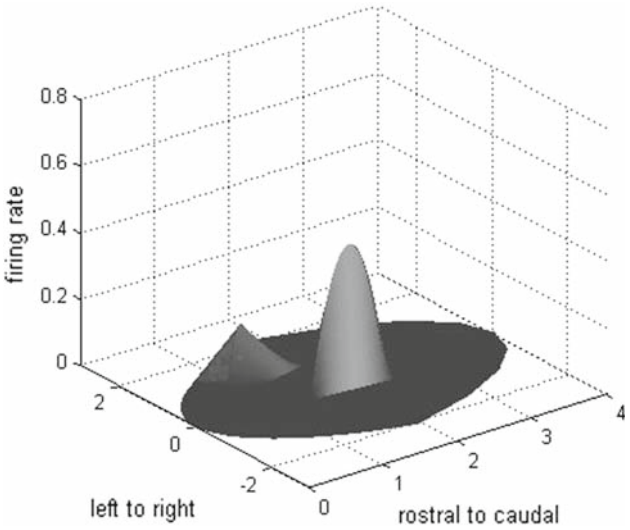

Fig. 7 Firing rate for model neurons across one superior colliculus during a far distractor paradigm. The target is presented at the horizontal meridian, coded for by model neurons on the midline of the colliculus. A far distractor is presented at the same eccentricity but a 67.5 angle with the target. Shown is activity on time step 220, right before the saccade threshold is reached with chosen parameter values (all at their mean values, i.e., DLPFC input at fixation 0.3, at target location 0.3, inhibition at distractor location 0.2)

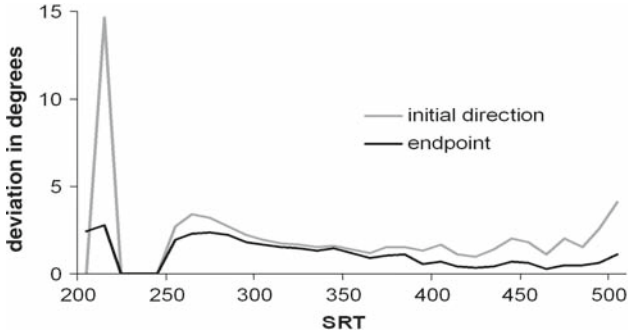

Fig. 9 Average deviation of the saccade from a straight saccade to the target location in the close distractor paradigm, as a function of saccade latency (SRT). Shown is the angle, in degrees, that the saccade makes with a straight saccade to the target both for its initial direction and its endpoint. A straight saccade to the distractor would have an angle of 22.5°

2.5.2 Saccade trajectories

To investigate whether the model also reproduces the global effect, we investigated the saccade trajectory for each saccade that resulted from a concrete target and distractor visual strength. Figure 8 shows a sample of saccades produced by the model in the close distractor condition. Some saccades go straight to the saccade, but many verge toward the distractor. This replicates the global effect. Figure 9 shows average deviation from a straight saccade in degrees in the close distractor paradigm, as a function of SRT. Positive values indicate an initial direction and endpoint that is more in the direction of the distractor (which itself was at 22.5°). Fast saccades tended to go to the center in between the target and distractor or even straight to the distractor: the initial direction of express saccades, with an SRT below 220 ms, was more toward the distractor than toward the target. With increasing SRT, saccades being directed more and more toward the target.

For far distractors, a few saccades curved toward the distractor, but most did not or only negligibly so. Average deviation toward the distractor at saccade onset was 2.7° for close distractors, but only 0.06° for far distractors. Some saccades

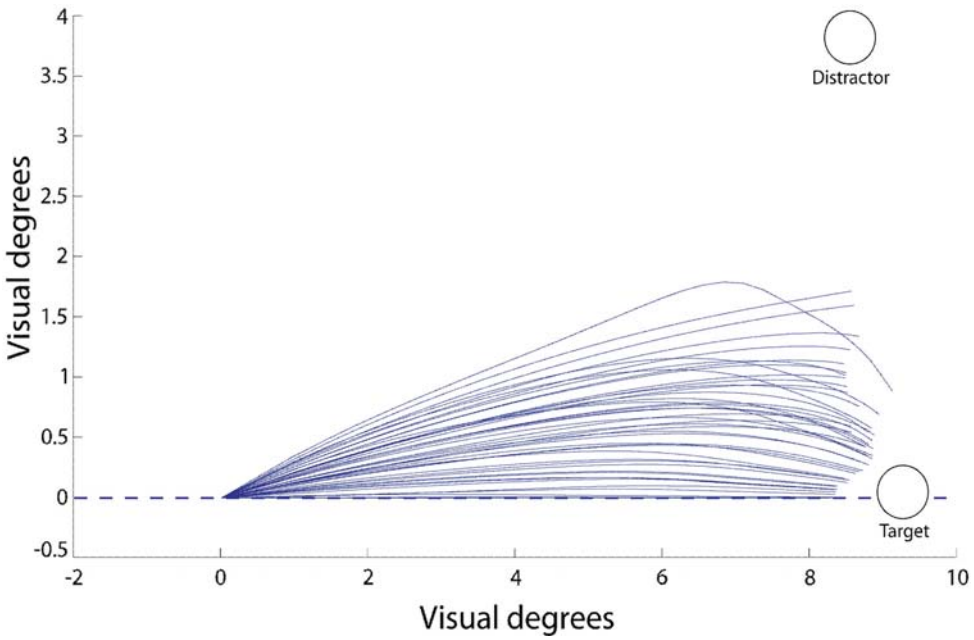

Fig. 8 Sample saccade trajectories, generated in conditions when the distractor is presented close to the target. Saccades start at the origin and then go to the target or distractor (location of the target and distractor in visual space also indicated)

deviating toward far distractors are typically found in non-human primates (McPeek and Keller 2001), but in humans saccades often deviate away from far distractors. We will return to this issue in the discussion.

In the model, saccades to locations in between target and close distractor locations are those that are launched before competition between the target and distractor location is resolved (e.g., Fig. 7). At both SC locations, there is a peak of activity that drives the saccade toward the endpoint coded by the peak locations, and the result is a saccade directed in between the two locations. This explanation is supported by evidence that the largest neural activity during averaging saccades remained at the sites of the two visual stimuli (Edelman and Keller 1998).

Saccades in which this occurred were those in which activity at the distractor location was relatively strong, and where the "center of gravity" of the activity was thus shifted toward the distractor. Implicit in the model is thus a 'center of gravity' account, which claims that the saccade endpoint is based on the relative salience in the visual field (Coren and Hoenig 1972). Several behavioral findings support such an account: the eyes typically land closer to the most probable target location (He and Kowler 1989), to an onset with greatest luminance (Deubel et al. 1984) and the largest size (Findlay 1982).

As can be seen in Fig. 9, saccades with the strongest deviations toward the distractor tend to be faster saccades. Many slower saccades occur when competition between saccade goals has already been resolved. This is in line with behavioral evidence that the global effect occurs more frequently in saccades with short latencies (Godijn and Theeuwes 2002; Ottes et al. 1985; Van der Stigchel and Theeuwes 2005).

Activity at the distractor location can also be so large that the saccade is launched toward the distractor location and not the target location. This would be a case of oculomotor capture, saccades that seem "captured" by a salient distractor. This occurred in our simulations: some saccades were directed toward distractor locations (this explains part of the deviation toward distractors seen in the far distractor paradigm); this was only the case in visual search situations (i.e., when there was no anticipatory activity at the target location) and when visual activity at the distractor location was stronger than at the target location. Indeed, paradigms in which capture is found tend to use distractor stimuli that are visually more salient than the target (Godijn and Theeuwes 2002; Irwin et al. 2000; Theeuwes et al. 1998); moreover, capture in search paradigms is not found when participants know where to expect the target (Yantis and Jonides 1990).

2.5.3 Curved and turnaround saccades

As can be seen in Fig. 9, the deviation in the direction of the distractor was for many saccades larger at the start of the saccade than at its end. Many saccade trajectories thus curve back toward the target location.

In the model, curving saccades are produced not by a separate saccade correction system, but by the ongoing resolution of competition: the longer the competition lasts, the more the distractor location is inhibited. Activity therefore tends to be biased more and more toward the target location while a saccade is being executed. This produces saccades that curve back to the target location, as seen with some saccades in Fig. 8. In those cases, activity starts out stronger at the distractor location, but within the duration of the saccade activity at the target location wins the competition. This produces saccades that first go in the direction of the distractor, but then turn back. In these saccades, high activity is initially found in SC at the distractor location, with a later shift toward the target location. This has indeed been observed using electrophysiological recordings by Port and Wurtz (2003). Moreover, consistent with experimental data (e.g., Godijn and Theeuwes (2002)) such saccades tend to be relatively fast.

3 Discussion

We have presented a model of the SC that reproduces findings from eye movements paradigms used in cognitive psychology and neuroscience: the gap paradigm, antisaccade paradigm, and distractor paradigm. In all the three, our model reproduces basic findings: faster saccades with a gap between fixation offset and target onset, or with a warning signal, the bimodal latency distribution and express saccades seen in gap conditions, slower antisaccades, the reduced gap effect in antisaccades, a slowing of saccades when far distractors are presented, the global effect, saccades that are curved toward a distractor location, and turnaround saccades. But it also reproduces some less evident findings in an emergent fashion:

- "center of gravity" effects in saccade trajectories
- that deviating saccades tend to be faster than non-deviating ones in distractor paradigms
- that antisaccades become slower with increasing stimulus contrast
- that the gap effect is reduced in antisaccade paradigm
- that saccades that deviate toward a distractor often curve back toward the target
- that saccades resume and reach their destination after being interrupted through current injection in the SC fixation zone.

Model inputs were based on findings from electrophysiological studies. Resulting neuronal activity in FEF and SC reproduces electrophysiological recordings for those

paradigms in which these are available. The current model thus shows that behavioral and neurophysiological results in these paradigms can be accounted for within a common framework, namely competitive integration across a saccade map. It shows what can be explained with this relatively simple framework, but it also shows what cannot be explained, which we will return to later.

3.1 Comparison to previous models

Saccade generation has been the topic of many computational models. Several models of what is often called the brainstem saccade generator use the SC as a top layer, which sends inputs to more elaborately implemented brain stem and cerebellar structures (Gancarz and Grossberg 1998; Lefevre et al. 1998; Optican 1995; Quaia et al. 1998). Several others have focused on SC or FEF dynamics (Gancarz and Grossberg 1999; Heinzle et al. 2007; Kopecz 1995; Trappenberg et al. 2001). These SC/FEF models and the brainstem generator models are complementary more than that they are in competition. The former models focus on how different input lead to a peak in activity and interface more with cognitive research. Most of the latter models take an activity peak in SC as a given, and model how a saccade is generated on the basis of that peak. They interface more with basic neurophysiology.

Our model of the SC is indebted to the model by Trappenberg et al. (2001) which is on its turn an elaboration of the Kopecz model (1995; Kopecz and Schoner 1995). These models accounted for several phenomena that were also reproduced by our model (gap effect, basics of close and far distractor effects). However, our model is in several ways exceeds them. First, previous models focus on saccade initiation, whereas here both the initiation and the trajectories of saccades were simulated. Moreover, the use of one dimension limited previous models to simulating paradigms with all stimuli on the horizontal meridian, whereas our model can simulate paradigms in which objects are presented at arbitrary locations in space. This allowed the model to account for characteristics observed in saccade trajectories.

We made an attempt to model in more detail input signals to the SC, and to model additional areas of the saccade system, notably the FEF. Gancarz and Grossberg (1999) presented a model that did include links between parietal and frontal areas, FEF, SC, and the brainstem saccade generator. The focus of this model was learning in the cerebellum in saccadic adaptation paradigms, and it was not applied to many of the paradigms simulated here. This makes it difficult to compare that model to the current one. One striking difference, however, is that the Gancarz and Grossberg (1999) model proposes that each pathway through network of brain areas involved in saccades underlies a different set of saccades, whereas we propose that the same connections, e.g., those between FEF and SC, are involved in all kinds of saccades. This, we think, is more in line with the evidence as, for example, FEF seems to be active independent of what kind of saccade is made (Bichot and Schall 2002; Everling and Munoz 2000).

Our translation of SC activity into input to the brainstem saccade generator is consistent with previous models. The idea that saccades are directed to a weighted average of SC activity was already explored by Van Gisbergen et al. (1987), Van Opstal and Van Gisbergen (1990), and Arai and Keller (2005), among others. The idea that activity in SC also codes the length of the saccade is embedded in many models of the brainstem saccade generator. Van Opstal and Van Gisbergen (1990) and Gancarz and Grossberg (1998) both code the length of the saccade in the strength of the signal from SC to the brainstem saccade generator. This would be possible in our model as well, but we did not explore it because in our model the drive from SC also prompts saccade initiation. A stronger drive with longer saccades would lead to the prediction that SRTs are shorter for longer saccades, which they are not outside of the fixation zone (Dafoe et al. 2007). Another proposal is that of Quaia et al. (1998) and Lefevre et al. (1998), who assume that SC relays the length of the saccade to the cerebellum, which then controls the end of the saccade via an explicit stop signal. Our idea that activity in SC continues to influence the saccade generator during the saccade was already explored by Walton et al. (2005). They show that if SC activity at one stimulus location is cut off during the saccade, a curved saccade results. Our model shows how such an activity profile could come about in SC.

Saccades are initiated in our model by activity in SC driving the brainstem saccade generator across a threshold. Collicular burst neurons are assumed to inhibit brainstem omnipause neurons, which demobilize the eyes when no saccades are to be made. Brown et al. (2004), on the other hand, place more emphasis on the basal ganglia as a gate—saccades in their model only result if GABAergic neurons in the Substantia Nigra-pars reticularis are inhibited by, indirectly, cortical inputs. The role of the basal ganglia has not been addressed in great detail here, but two facts speak against an exclusive gating role for the basal ganglia. First, visual saccades are not influenced by stimulation of the SNr, the main output region of the basal ganglia to the saccade system (Basso and Liu 2007). Second, it is hard to imagine artificial stimulation of the SC having an influence on the basal ganglia, but such stimulation does lead to saccades (Robinson 1972).

3.2 Novelties and predictions

A critical assumption in the simulations is that top-down inputs are not only excitatory, but can also inhibit saccades to specific locations. Location-specific inhibition has been

proposed by others (e.g., Godijn and Theeuwes 2002; Tipper et al. 2001), but has not yet been implemented in a computational model. Trappenberg et al. do assume a negative exogenous input at stimulus offset, but this is quite different from the inhibition proposed here. In Trappenberg et al. (2001) the inhibition is exogenous, immediate, and automatic. In our model, inhibition is endogenous, task-related and occurs only after some time. Moreover, it is location-specific: not all saccade neurons in the SC are inhibited in the same way; those coding for the location of a distractor or of the stimulus in an antisaccade paradigm are singled out for inhibition. Although this is also the case in the Trappenberg model, others (e.g., Johnson and Everling 2006) have suggested that inhibition might stretch over, for example, one of the two colliculi.

Our arguments for location-specific inhibition stem from behavioral findings. For example, the trajectory of a saccade to a target is influenced by the exact location of a distractor (Van der Stigchel et al. 2007). This seems to imply that distractor-evoked inhibition is sensitive to the location of the distractor. Physiological and anatomical evidence (Johnson and Everling 2006; Krauzlis et al. 2004; McPeek and Keller 2002a; Ottes et al. 1987) is consistent with both location-specific and broad inhibition. For example, Johnson et al. (2006) found that, during an antisaccade task, DLPFC neurons sent input that was assumed to be inhibitory to the colliculus in which prosaccades were programmed. Their methods did not allow them, however, to ascertain how widely these inputs were spread over the colliculi. Hasegawa et al. (2004), however, found neurons in DLPFC (and to a lesser extent in FEF) that became active when a saccade to a specific object had to be suppressed. These neurons would be ideal for the implementation of location-specific inhibition as proposed here. However, where these neurons projected to was not investigated.

In the current article, location-specific inhibition lies at the basis of our simulations of antisaccades, of distractor effects, and of the curvature of saccades back to the target stimulus. This leads to two sets of predictions:

- If location-specific inhibition is interrupted, either through a general blockade of GABA transmission in the SC or through DLPFC lesions, far distractors should slow saccades to a larger degree, curved saccades should become less common, and prosaccade errors in the antisaccade paradigm more common. Currently only the latter part has been verified in DLPFC lesion studies (Guitton et al. 1985; Pierrot-Deseilligny et al. 2005).
- In the antisaccade and distractor paradigms, activity at the onset or at distractor locations should diminish faster than is the case for stimuli that are presented while the observer must simply maintain fixation. This might be tested in electrophysiological studies.

Another novelty is that activity within the SC continues to influence the direction of the saccade while it is programmed and executed. Although Walton et al. (2005) already explored mathematically how this assumption could generate curved saccades, our model is the first in which SC activity continues to be updated by the model during the execution of a saccade. Redirected saccades are then caused by the shift in SC activity during the saccade. We do not posit that saccade trajectories are completely determined by the SC, but suggest that through a continuously evolving activity, the SC influences the trajectory. It is now generally accepted that many mid-flight corrections involve brainstem mechanisms and the cerebellum (Lefevre et al. 1998; Moschovakis 1996). These mechanisms do not account, however, for saccades in which a trajectory reveals mid-flight changes of goals, such as redirected saccades (Amador et al. 1998). Our proposal addresses these saccades as opposed to minor mid-flight corrections, and also addresses other more cognitive influences on saccade trajectories.

A critical result related to this assumption is that when saccades end in between two targets, the main activity in the SC map is at the two target locations in the SC, and not at the end point of the saccade. Data from Edelman and Keller (1998) seem to support this idea. It is possible that their results only apply to express saccades, although the authors argue against this possibility.

Our assumption of SC-influenced trajectories leads to two more predictions:

- stimulation of SC locations during a saccade should change its trajectory in a predictable way
- during saccades that land halfway between a target and a distractor (the *global effect*), activity in the SC map at target and distractor locations should approximately be balanced.

3.3 Imperfections and directions for future research

There are also notable imperfections to our model. Several connections known to exist were not implemented in the model, as were several brain areas that are known to be involved in the generation of eye movements. By not including these areas, we are making the tacit assumption that these are less important than the areas and connections in the model for normal saccade generation. Here, we discuss the plausibility of this assumption for areas and connections involved:

- One connection not included is how the SC efferents back to FEF. Including this connection would not fundamentally alter the model, as in the current setup the target of the saccade is chosen simultaneously in FEF and SC. It might, however, lead to a closer fit between neural

responses in the model SC and FEF, and the responses in those brain areas.

- The FEF projects to the brainstem saccade plant both directly and via the cerebellum. These connections are not sufficient to elicit saccades: FEF stimulation that is normally sufficient to elicit a saccade does not do so when SC is inactivated (Hanes and Schall 2001). The direct connections may mostly be involved in maintaining fixation, as they target omnipause neurons that are involved in fixating (Segraves 1992). The indirect projections via the cerebellum may play a role in correcting saccades that are off-target.
- The cerebellum may be involved in midflight corrections of saccade trajectories (Lefevre et al. 1998; Moschovakis 1996). It may also be involved in unlearning systematic biases in saccade trajectories (e.g., Catz et al. 2008). Our model provides an alternative account for some corrections seen in saccade trajectories, but is not incompatible with an additional role for cerebellum.
- Both the secondary eye fields (SEF) and the lateral intraparietal area (LIP) project to the SC (Munoz 2002). In both, neural responses seem to represent both visual stimulation, as well as saccade-related activity (e.g., Ferraina et al. 2001; Pouget et al. 2005), a combination also seen in FEF activity. It may thus be that these areas have a similar role as the FEF in supporting saccade target selection.
- The basal ganglia may play a more complex role than they do in our model, in which they are one of the routes through which DLPFC inhibits the SC. In fact, the basal ganglia also receive input from FEF, and show a collection of responses that are not implemented in our model (Basso and Liu 2007).
- In LIP, FEF and in many other cortical areas projecting to the SC, a "predictive remapping" process takes place (e.g., Duhamel et al. 1992). Already before a planned saccade, stimuli activate neurons in whose receptive field they will fall *after* the saccade. This might result in input to SC neurons coding for that new location, something we have not implemented in our model. However, when two saccades are made in close succession, activity related to the second saccade goal is present, during the first saccade, at the location of the second goal *before* the first saccade (McPeek and Keller 2002b). This is not suggestive of a strong remapping of activity before saccades occur.

Another imperfection of the model is our rather sketchy implementation of DLPFC signals. Neurons in DLPFC have been shown to exhibit saccade-related activity. Moreover, DLPFC is known to be involved in implementing task demands. Our proposal has been that this occurs, in the case of saccades, through inhibiting task-incompatible saccade plans as well as the planning of purely endogenous saccades (e.g., antisaccades). Our implementation of these signals has been as simple as possible, as there is little data available to constrain the translation of DLPFC activity into effects on the saccade system. Most notably, we have not included any variance in the timing of the signals. This underlies the relative paucity in the model of slow erroneous prosaccades. Such prosaccades, with latencies comparable to those of antisaccades, would result if on some trials inhibition is later than on others.

A failing is the inability of the model to produce saccades that deviate away from far distractors, as seen with human observers. The trajectory of such saccades typically starts in a direction toward the target but skewed away from the distractor, with the trajectory curving back to the target position later in the saccade (i.e., Doyle and Walker 2001; Van der Stigchel et al. 2007). Such saccades are typically found in human observers in paradigms in which a distractor is presented far from the target stimulus, or in which attention is directed toward a location at an angle with the target location. Notably, such deviations have never been observed in nonhuman primates. This suggests that in such deviations away a mechanism operates that is either specific to human observers, or that is too cumbersome for nonhuman primates to regularly engage in. We have recently argued that deviations away are typically found in saccades under strong top-down control (Van der Stigchel et al. 2006). It may be that this recruits additional inhibition from DLPFC, in a way that skews activity in the SC saccade map away from the distractor. Although there is some evidence for such an account (Van der Stigchel et al. 2006), it remains somewhat speculative. We intend to investigate precise saccade trajectories in more detail in future research.

4 Conclusion

Eye movements and the corresponding neural mechanisms are one of the most heavily researched areas. Our model is one of several that claim that saccade goals result from competitive integration of signals in the SC. However, our model goes beyond other accounts, by showing how such a framework can account for saccadic latencies in many paradigms, and how it accounts for the trajectories of saccades. It also makes explicit what is missing, most notably a more thorough treatment of the role of DLPFC in saccade generation.

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