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Lateral interactions in the superior colliculus produce saccade deviation in a neural field model

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ABSTRACT

Contrary to human intuition, saccades (rapid eye movements) rarely go directly to their intended destination, but instead typically deviate from the optimal track. Previous studies have demonstrated that saccades may deviate toward or away from irrelevant distractors. Deviation toward distractors is generally explained with theories of "population coding", while deviation away from distractors is believed to be caused by top-down inhibition at the distractor location. With a Mexican-hat shaped lateral interaction kernel, we successfully simulated both deviation toward and away from distractors using a neural field model of the superior colliculus (SC). Our findings suggest that top-down inhibition of the SC is not necessary for the generation of saccade deviations.

1. Introduction

Humans make over 100,000 eye movements every day. While looking at a scene, searching for a target typically involves a series of saccades (rapid eye movements) interspersed by short fixations during which the scene is processed. While awake, humans normally make 3–4 saccades per second. Behavioral and neurophysiological studies of this unique type of eye movement have greatly advanced our understanding of motor control.

1.1. Neural field models of the SC

The superior colliculus (SC), a layered midbrain structure, is a crucial component of the oculomotor system (for a recent review, see White & Munoz, 2011). The intermediate layers of the SC (SCi) contain a topographic motor map that encodes the vector of saccades into the contralateral visual field (Ottes, Van Gisbergen, & Eggermont, 1984; Robinson, 1972; Van Gisbergen, Van Opstal, & Tax, 1987; see Fig. 1A). A similar map also exists in the superficial layers of the SC (SCs), which receives projections from the retina and the visual cortex and is in alignment with the motor map in SCi (Helms, Özen, & Hall, 2004; Isa, Endo, & Saito, 1998; Wurtz & Goldberg, 1972). Many computational models of the SC (e.g., Kopecz & Schöner, 1995; Trappenberg et al., 2001) assume that the lateral connection between neurons in the SC depends on distance, with proximal neurons exciting each other and distal neurons inhibiting each other. This lateral interaction ensures only one locus of activity dominates the motor map when multiple sources of input are present (winner-take-all). Such lateral connectivity in the SC is backed by *in vivo* extracellular recordings in monkeys (Dorris, Olivier, & Munoz, 2007; Meredith & Ramoa, 1998; Munoz & Istvan, 1998; Trappenberg et al., 2001). However, even though a long-range inhibitory connection is prominent in SCs, *in vitro* recordings in rodents indicate that such long-range connections may be absent in SCi (Isa & Hall, 2009).[1] Although the existence of long-range inhibitory connectivity in the SCi is still under debate (see Marino et al., 2011; for a summary of existing findings), neural field models (Amari, 1977; Wilson & Cowan, 1973) with such lateral inhibition have proven to be successful in simulating various saccade-related behaviors and cell recordings (Arai & Keller, 2005; Arai, Keller, & Edelman, 1994; Kopecz & Schöner, 1995; Trappenberg et al., 2001; Wang et al., 2011; Wilimzig, Schneider, & Schoener, 2006).

1.2. Saccade deviation

It is known that the trajectory of saccades may either deviate toward (Becker & Jürgens, 1979; Coren & Hoenig, 1972; Findlay, 1982; Walker et al., 1997; Watanabe, 2001) or away (Doyle & Walker, 2001; McSorley, Cruickshank, & Inman, 2009; Van der Stigchel & Theeuwes, 2005) from a task irrelevant visual distractor (for reviews, see Van der Stigchel, 2010; Van der Stigchel, Meeter, & Theeuwes, 2006; Walker & McSorley, 2008). When deviating toward a visual distractor, saccades typically land close to the distractor

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[1] A recent *in vitro* study showed long-range intrinsic inhibition in the SCi, albeit weak and largely masked by local excitation (Phongphanphanee et al., 2008; cf., Marino et al., 2011).

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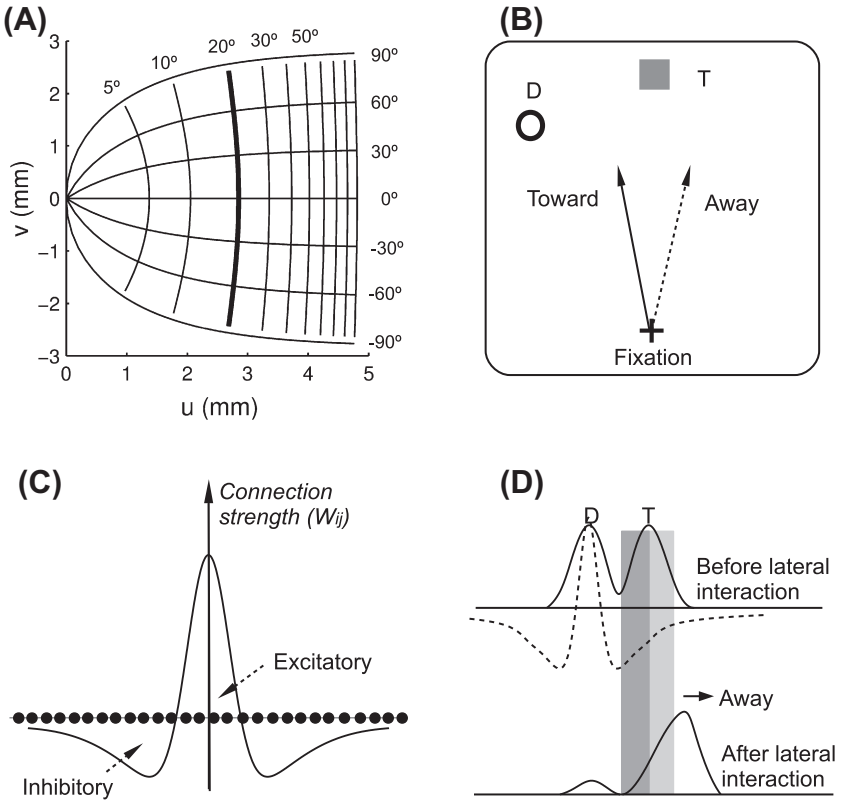

Fig. 1. (A) The topographic motor map in the SC (Van Gisbergen, Van Opstal, & Tax, 1987); u is the anatomical distance (mm) from the rostral pole along the axis representing horizontal position; v is the perpendicular distance where positive and negative values represent the medial and lateral sides of the SC, respectively. The thick black line highlights a SC slice representing 20° saccades in various directions. (B) The typical display setup of a distractor task. The arrows are drawn to show that the initial direction of a saccade may deviate away from, or toward, the distractor. *D*, distractor; *T*, target. (C) The Mexican-hat shaped lateral interaction assumed to be present in the SC. The connection strength (weight) between two neurons (black dots) varies with distance. (D) Schematic explanation of how a Mexican-hat shaped lateral interaction kernel (dashed line in the upper panel) can generate saccades deviating away from the distractor, see text for details.

location (cf., oculomotor capture, Theeuwes et al., 1998), or somewhere between the distractor and target locations (saccade averaging or global effect, see Chou, Sommer, & Schiller, 1999; Coren & Hoenig, 1972; Findlay, 1982). These observations have been attributed to insufficient suppression of distractor related activity (McPeek, 2006; McPeek, Han, & Keller, 2003; Van der Stigchel, Meeter, & Theeuwes, 2006) or population coding (Tipper, Howard, & Jackson, 1997). When positioned in close proximity, the distractor- and target-related activity collaborates, merging into one activity "bubble" centered at an intermediate location (for computational exploration of this idea, see Meeter, Van der Stigchel, & Theeuwes, 2010; Satel et al., 2011; Wilimzig, Schneider, & Schoener, 2006). This explanation is backed by several neurophysiological investigations showing stronger neuronal activity at SC sites representing locations between the target and distractor (Edelman & Keller, 1998; Glimcher & Sparks, 1993; van Opstal & van Gisbergen, 1990). However, up to now, there is no satisfying explanation in the literature for deviation away from distractors. The dominant view of this phenomenon relates deviation away from distractors to a form of top-down inhibition at the distractor location (Van der Stigchel, Meeter, & Theeuwes, 2006). The observation of saccades deviating away from the response field of inactivated SC regions (Aizawa & Wurtz, 1998; Lee, Rohrer, & Sparks, 1988; Quaia et al., 1998) lends some support to this view. However, this top-down inhibition theory was put into doubt by a recent study (White, Theeuwes, & Munoz, 2012), where neuronal activity was recorded in SCi while the monkey performed a distractor task. The distractor–target onset asynchrony (DTOA) was either 400 ms or 0 ms. If deviation away were to be caused by top-down inhibition, during the 400 DTOA, one would expect a reduction of distractor evoked neuronal activity associated with saccades that deviated away from the distractor. However, there was no reduction of activity in this time window. Although not statistically reliable, in this time window, stronger deviation away from distractors was accompanied by stronger distractor evoked SC activity. Importantly, this is the exact opposite pattern predicted by a theory that assumes deviation away from distractors is the result of top-down inhibition.

1.3. A unified theory of saccade deviation

The primary purpose of the present study is to explain deviations in saccade *direction*,[2] as frequently studied in a distractor paradigm, with a neural field model of the SC (Trappenberg et al., 2001). A typical display setup in the distractor paradigm is illustrated in Fig. 1B. The participant initiates a saccade to a target while a distractor appears before, after, or simultaneously with the target. The main findings in the literature can be summarized as follows. (a) Saccades may deviate toward or away from task-irrelevant distractors (e.g., Van der Stigchel, Meeter, & Theeuwes, 2007; Van der Stigchel & Theeuwes, 2005; White, Theeuwes, & Munoz, 2012). (b) In contrast to deviation toward distractors, the magnitude of deviation away is typically small, with the initial directional deviation smaller than

[2] The present paper examines the initial deviation of saccade direction only. Studies devoted to the spatiotemporal properties of saccade trajectories, i.e. curvature (e.g., Ludwig & Gilchrist, 2002; McSorley, Haggard, & Walker, 2006, 2009), are not considered here. We assume that downstream mechanisms in the brainstem control the trajectory after a saccade has been triggered.

5° (e.g., Godijn & Theeuwes, 2002). (c) Deviation away from distractors can also be observed when the distractor is presented up to 400 ms before target onset (White, Theeuwes, & Munoz, 2012). (d) Saccade deviations vary with saccade latency (e.g., McSorley, Cruickshank, & Inman, 2009; Mulckhuyse, Van der Stigchel, & Theeuwes, 2009).

To accommodate the above mentioned observations, we propose an intuitive theory of saccade deviation based on two principles. First, the lateral connection between saccade-related neurons in the SC is Mexican-hat shaped (e.g., Arai et al., 1999; Trappenberg et al., 2001). This interaction kernel is characterized by a strong proximal excitation zone, and inhibitory connections at longer distances that weaken as the distance between two neurons increases further (see Fig. 1C). This Mexican-hat shaped lateral interaction has been shown to be effective in reproducing both cell-recordings and saccade behavior in various experimental tasks (Arai et al., 1999; Trappenberg et al., 2001; Wang et al., 2011). In our simulations, we will demonstrate that such Mexican-shaped lateral interaction itself is sufficient to produce both deviation toward and away from distractors. Second, neuronal activity in the SC is modulated by inhibitory projections from the substantia nigra pars reticulata (SNr). Jiang, Stein, and McHaffie (2003) have shown that SNr neurons which project to the ipsilateral SC have relatively small visual receptive fields that are in topographic alignment with their SC target neurons. Previous studies have also shown that a visual signal can temporarily release the tonic SNr inhibitory input to the SC (e.g., Basso & Wurtz, 2002; Handel & Glimcher, 1999; Hikosaka & Wurtz, 1983; Jiang, Stein, & McHaffie, 2003; Joseph & Boussaoud, 1985). This visually elicited release of SNr inhibition (SNr disinhibition) is necessary for our theory to accommodate the observation that saccades may deviate away from distractors presented long before target onset (White, Theeuwes, & Munoz, 2012).

1.3.1. Lateral interactions produce both deviation toward and away from distractors

Our model assumes that the lateral interaction in the SC is Mexican-hat shaped (see Fig. 1C). When the distance between the distractor and target is in the range of lateral collaboration, the distractor and target related activity will merge additively. The merged activity, which encodes the vector of the imminent saccade, will eventually peak at an SC site representing an intermediate location between the target and distractor (the global effect). When the distance between the distractor and target is in the range of lateral competition, as illustrated in Fig. 1D, one side of the target activity (dark shaded area) receives stronger inhibition from the distractor than the other side (light shaded area). In the same vein, the distractor activity also receives uneven inhibition from the target. Thus, target and distractor activities push each other away during lateral interaction and the target related activity will eventually trigger a saccade that deviates away from the distractor.

1.3.2. SNr activity modulates saccade deviation

The above theory can explain deviation away from distractors when the target and distractor are presented at roughly the same time. However, if the DTOA is long, distractor evoked SC activity should have largely decayed by the time of target presentation. Consequently, no visually elicited distractor activity will compete with the target activity, and no deviation away from (or toward) distractors will be observed. To accommodate the observation of deviation away from distractors at a 400 ms DTOA (White, Theeuwes, & Munoz, 2012), SNr disinhibition at the distractor stimulated SC site was included in our simulations (for similar implementation, see Arai & Keller, 2005). We hypothesized that SNr disinhibition is responsible for the sustained discharge

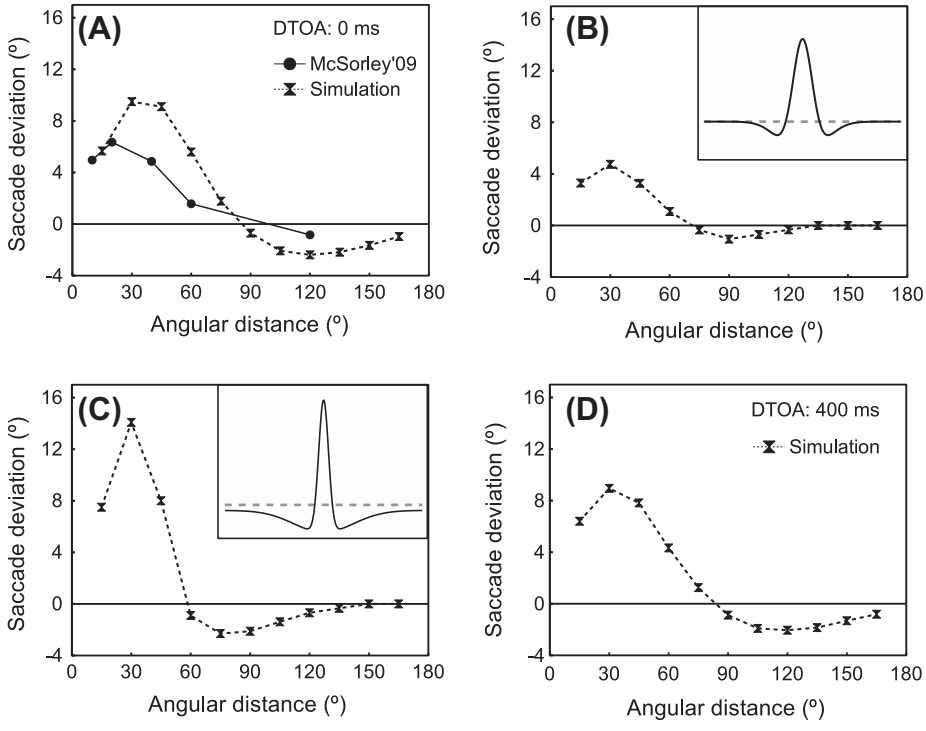

Fig. 2. Saccade deviation varies with distractor–target distance Positive and negative values on the y-axes represent deviation toward and away from distractors, respectively. DTOA = 0 ms in (A–C), DTOA = 400 ms in (D). (A) Results of simulations with a kernel similar to Trappenberg et al. (2001); human data was adapted from McSorley, Cruickshank, and Inman (2009). McSorley, Cruickshank, and Inman (2009) reported deviations in both trajectory (relative to the direct path between fixation and landing position) and landing position (see their Fig. 2a and b). We summed these two measures to estimate the overall directional deviation (averaged over latency bins, see also Fig. 5). (B) Results of simulations with a kernel in which the long-range inhibition was removed, $a = 72$, $b = 24$, $c = 0$, $\sigma_a = 0.5$ mm, $\sigma_b = 1.0$ mm (see the upright corner). (C) Results of simulations with a kernel of small width, $a = 144$, $b = 24$, $c = 6$, $\sigma_a = 0.25$ mm, $\sigma_b = 1.5$ mm (see the upright corner). (D) Results of simulations with SNr disinhibition.

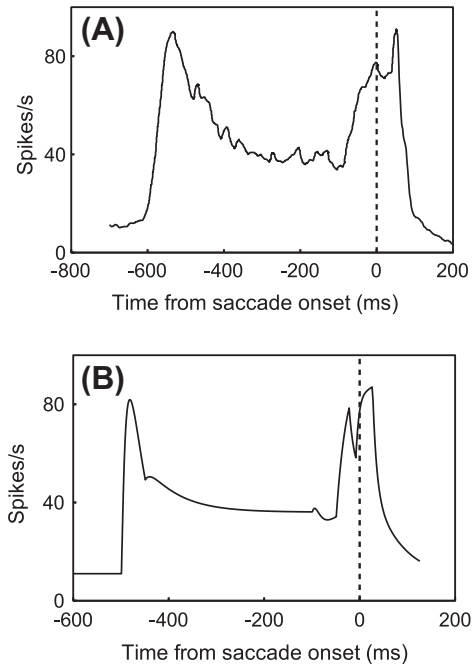

Fig. 3. SNr modulates distractor evoked SC neuronal activity. (A) Distractor evoked SC activity (aligned to saccade onset), adapted from White, Theeuwes, and Munoz (2012) with permission. (B) Simulated neuronal activity with SNr disinhibition.

following the initial SC response to the distractor (see White, Theeuwes, & Munoz, 2012; Fig. 7B), see Fig. 3A. Thus, the distractor elicited activity will be able to interact with target-related activity over a relatively long time period.

2. Model architecture and parameters

The present study used a 1-dimensional neural field model of the SC (Trappenberg et al., 2001). We chose to use a 1-dimensional model because deviation away from distractors is a relatively small behavioral effect, usually less than 5° (e.g., Godijn & Theeuwes, 2004), so to reflect subtle changes in deviations away in response to experimental manipulations (e.g., Mulckhuyse, Van der Stigchel, & Theeuwes, 2009), a model with high spatial resolution is needed. However, a high resolution 2-dimensional model poses an impractically high computational load onto readily available hardware. Also, because saccade deviation is frequently measured in terms of saccade direction (e.g., Port & Wurtz, 2003; Van der Stigchel, Meeter, & Theeuwes, 2006), and because in some studies the target and distractor are presented at the same eccentricity (e.g., McPeek, Han, & Keller, 2003; McSorley, Haggard, & Walker, 2009; Theeuwes & Godijn, 2004), a simplification was made by using a population of nodes (n = 1000) on a straight line to roughly represent 5 mm of SCi tissue, which encodes saccades with 20° eccentricity in various directions (thick black line in Fig. 1A). Because the iso-eccentricity meridian of 20° approximates a straight line, the distance error introduced by this later simplification is negligible.

The connection strength (w_{ij}) between two nodes i and j was defined in Eq. (1), with parameters a = 72, b = 24, c = 2.4, σ_a = 0.6 mm, σ_b = 1.8 mm, unless specified otherwise. These parameters were the same as those used by Trappenberg and colleagues (Satel et al., 2011; Trappenberg et al., 2001; Wang et al., 2011), except that the amount of long-range inhibition was reduced (from c = 6.5) to reflect the fact that there may be only weak long-range inhibitory connections in the SCi (Phongphanphanee et al., 2008). The internal dynamics of a node i, $u_i(t)$, is described in Eq. (2), where w_{ij} is the connection strength between node i and node j, $r_j(t)$ is the discharge level of node j, $I_i^{exo,endo}$ is an excitatory input to node i, I_i^{snr} is an inhibitory SNr input to node i, and τ = 10 ms is a time constant. A sigmoid gain function (Eq. (3)) is used to relate the discharge level of a node i to its internal state, where β = 0.08.

$$w_{ij} = a \cdot \exp\left(\frac{-(x_j - x_i)^2}{2\sigma_a^2}\right) - b \cdot \exp\left(\frac{-(x_j - x_i)^2}{2\sigma_b^2}\right) - c \quad (1)$$

$$\tau \frac{du_i(t)}{dt} = -u_i(t) + \sum_j w_{ij} r_j(t) + I_i^{exo,endo}(t) + I_i^{snr}(t) \quad (2)$$

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t))} \quad (3)$$

$$I_k^{exo,endo} = e \cdot \exp\left(\frac{(x_k - x_i)^2}{2\sigma_e^2}\right) \quad (4)$$

$$I_k^{snr} = -R_{max} + R \cdot \exp\left(\frac{(x_k - x_i)^2}{2\sigma_{snr}^2}\right) \quad (5)$$

As in previous studies (Kopecz, 1995; Kopecz & Schöner, 1995; Marino et al., 2011; Trappenberg et al., 2001), the excitatory input was dissected into exogenous (visual) and endogenous (presumably a saccade decision signal from higher cortical areas) inputs. These two inputs were used to represent the SCs, and higher cortical areas, respectively. The latency of the exogenous (*exo*) input (i.e., the time it takes to reach the SC) was estimated to be 70 ms (Dorris, Paré, & Munoz, 1997), while the latency of endogenous (*endo*) input was estimated to be 120 ms. Both exogenous and endogenous inputs were assumed to have a Gaussian spatial shape (see Eq. (4)). In our simulations, the width of exogenous and endogenous inputs were fixed at σ_{exo} = 0.5 mm and σ_{endo} = 1.2 mm, which are close to cell-recording based estimations (Marino et al., 2011). The strength of exogenous and endogenous inputs were fixed at e_{exo} = 30 and e_{endo} = 15 in our simulations unless specified otherwise; these values were also chosen to approximate cell-recordings (White, Theeuwes, & Munoz, 2012) and regular saccade latencies. The exogenous input was set to decay exponentially (with a time constant of 10 ms), while endogenous input was sustained until the onset of the saccade (cf., Satel et al., 2011; Wang et al., 2011). In addition to excitatory inputs, every node received a constant inhibitory input from the SNr (Eq. (5)), R_{max} = 16, which can be released by a visual stimulus (Hikosaka & Wurtz, 1983).

Once the activity of a node reached a threshold of 80% of its maximum discharge rate, a trigger was assumed to be sent to the saccade burst generator in the brainstem, eliciting a saccade in its encoded direction. In our simulations, a 25-ms efferent delay is used to calculate saccade latency.

3. Simulation results

3.1. Simulation 1: saccade deviation and distractor–target distance

The primary prediction of our theory is that saccade deviation varies with distractor–target distance. Simulations were performed to demonstrate this prediction. In these simulations, the angular distance between the target and the distractor was varied between 15° and 165° and the DTOA was set to 0 ms. As shown in Fig. 2A, our model produced deviation toward a distractor when it was close to the target, and deviation away from a distractor when it was distal to the target. It is clear that our model simulations closely resemble the human data of McSorley, Cruickshank, and Inman (2009).

Trappenberg et al.'s (2001) lateral interaction parameters were optimized to approximate cell-recordings and saccade latencies in a visual distractor task. Those parameters were used in our model because the present study also explored the effects of visual distractors, albeit in the spatial domain (i.e., saccade deviations). Previously, this interaction kernel has been criticized for including long-range inhibitory connections (Isa & Hall, 2009). To reflect recent neurophysiological observations that indicate weak long-range connections in the SCi (Phongphanphanee et al., 2008), only a small amount of long-range inhibition ($c = 2.4$) was included in our lateral interaction kernel (see Fig. 1C). It is important to note that our explanation of saccade deviation away from distractors does not depend on intrinsic long-range inhibition in the SC. Deviation away from distractors, albeit weaker, can still be produced when only short-range inhibition is included in the interaction kernel (see Fig. 2B).

As in McSorley, Cruickshank, and Inman (2009), our model produced deviation away from distractors only when the distractor–target distance was greater than 90°, while in the literature deviation away from distractors is frequently observed when the distractor–target distance is only 45° (e.g., Mulckhuyse, Van der Stigchel, & Theeuwes, 2009; White, Theeuwes, & Munoz, 2012). The discrepancy is probably due to the relatively large width of our interaction kernel. By reducing the width of the interaction kernel, our model was able to produce robust deviation away from distractors with a small distractor–target distance (60°), see Fig. 2C. It is also possible that this discrepancy between studies is caused by other mechanisms contributing to saccade deviations, with further empirical study needed to clear up this issue.

3.2. Simulation 2: saccade deviation and SNr disinhibition

Another observation in the literature is that deviation away from distractors can be caused by distractors presented long before target onset (White, Theeuwes, & Munoz, 2012). Cell-recordings reported in White, Theeuwes, and Munoz (2012; see Fig. 3A) shed some light on the mechanism(s) behind this observation. White, Theeuwes, and Munoz (2012) found that, rather than a quick decay, the distractor evoked neuronal activity sustained until target onset (see Dorris et al., 2002; Fecteau & Munoz, 2005; for similar observation of sustained SC activity evoked by task irrelevant visual stimuli). It is possible that this sustained distractor activity competed with target evoked activity and drove saccades away from distractors. A likely source of this sustained distractor activity is SNr disinhibition.

Early work by Hikosaka and Wurtz (1983) showed that most SNr neurons reduce their level of discharge in response to a visual stimulus (see also Basso & Wurtz, 2002; Joseph & Boussaoud, 1985). This visually evoked SNr response is initially strong and then decays to a weaker level where it is sustained (Basso & Wurtz, 2002); the response field of such a SNr response is relatively focal (Hikosaka & Wurtz, 1983; Jiang, Stein, & McHaffie, 2003). For simplicity, we have assumed that the distractor evoked a SNr response (disinhibition) ($R = 4$, $\sigma_{snr} = 0.3$ mm) which had a latency of 120 ms (Hikosaka & Wurtz, 1983) and lasted for 500 ms. Hikosaka and Wurtz (1983) also found that the visual response of some SNr neurons was strong, saccade-locked, and had a fairly large response field (see also Basso & Wurtz, 2002). Thus, we have further assumed that the target evoked a strong and broad SNr response ($R = 16$, $\sigma_{snr} = 1.8$ mm) shortly (15 ms) before saccade onset. In our simulations, the distractor–target distance here was set to 45°, as in White, Theeuwes, and Munoz (2012).

As shown in Fig. 3, the simulated neuronal activity (Fig. 3B) resembled distractor evoked SC activity recorded in the monkey SC (White, Theeuwes, & Munoz, 2012; see Fig. 3A). Following the initial visual response (the first peak), there was a sustained discharge caused by SNr disinhibition (Fig. 3B). When the target appeared, the quick rise of the target visual response caused a dip in the distractor evoked activity. The target related endogenous input and the saccade-locked SNr disinhibition were responsible for the final peaks in the simulation.

With the DTOA set to 400 ms, we also varied the distractor–target distance between 15° and 165° in our simulations. The pattern of results (see Fig. 2D) was similar to those obtained in Simulation 1 (see Fig. 2A), which provides a prediction for future studies. Note that, in those simulations, the amount of deviation depended on the magnitude of distractor evoked SNr disinhibition (see simulations reported in the next section).

3.3. Simulation 3: saccade deviation and saccade latency

As frequently reported in the literature, saccade deviation varies with saccade latency (McSorley, Cruickshank, & Inman, 2009; McSorley et al., 2006; McSorley, Haggard, & Walker, 2009; Mulckhuyse, Van der Stigchel, & Theeuwes, 2009; but see Walker, McSorley, & Haggard, 2006; White, Theeuwes, & Munoz, 2012). This observation has been regarded as evidence for the top-down inhibition theory of deviation away from distractors. The idea here is that because top-down inhibition takes time to develop and reach the SC (strong) deviation away from distractors is only observed when saccade latency is long (Van der Stigchel, 2010; Van der Stigchel, Meeter, & Theeuwes, 2006). We suggest that this observation can also be explained in terms of lateral competition in the SC; that is, factors that affect the relative activation level at the target and distractor locations cause saccade latency and deviation to co-vary. These factors include, but are not limited to, the relative strength of the distractor input and, in the presence of long DTOAs, the magnitude of the distractor evoked SNr disinhibition. When the distractor is close to the target, due to short-distance collaboration, stronger activity at the distractor location would reduce latency and increase deviation toward distractors. However, when the distractor is distal to the target, stronger activity at the distractor location would increase both latency and deviation away from distractors. This idea was supported by simulation results presented in Fig. 4. For close distractors, as the distractor input (Fig. 4A) or the distractor evoked SNr disinhibition (Fig. 4B) increases, saccade latency decreases and deviation toward distractors increases. For distal distractors, as the distractor input (Fig. 4C) and distractor evoked SNr disinhibition (Fig. 4D) increases, both saccade latency and deviation away from distractors increases.

The above proposal can explain the observation that deviation toward distractors decreases (e.g., McSorley, Cruickshank, & Inman, 2009) and deviation away from distractors increases (e.g., Mulckhuyse, Van der Stigchel, & Theeuwes, 2009) with saccade latency. However, it is unclear why a transition from toward to away appears as saccade latency increases (e.g., McSorley et al., 2006; see also Fig. 5A).[3] For most studies that have explored the relationship between saccade deviation and saccade latency, a common feature is that temporal gaps between fixation offset and target onset were introduced to manipulate saccade latencies. Previous behavioral studies have shown that temporal gaps reduce saccade latency, referred to as the gap effect (Reuter-Lorenz, Hughes, & Fendrich, 1991; Saslow, 1967). Monkey neurophysiology suggests that the gap effect could be attributed to top-down preparatory input during the gap period which raises the baseline level of activation in the SC (e.g., Dorris, Olivier, & Munoz, 2007; Dorris, Paré, & Munoz, 1997;

[3] McSorley et al., (2006) reported the magnitude of curvature in saccade trajectories, rather than deviation in saccade direction. We contacted Eugene McSorley and asked for their data. Our analysis of their raw data showed that the pattern of results reported in their paper, i.e., deviation toward changed into deviation away as latency increased, was also present in the initial saccade directions.

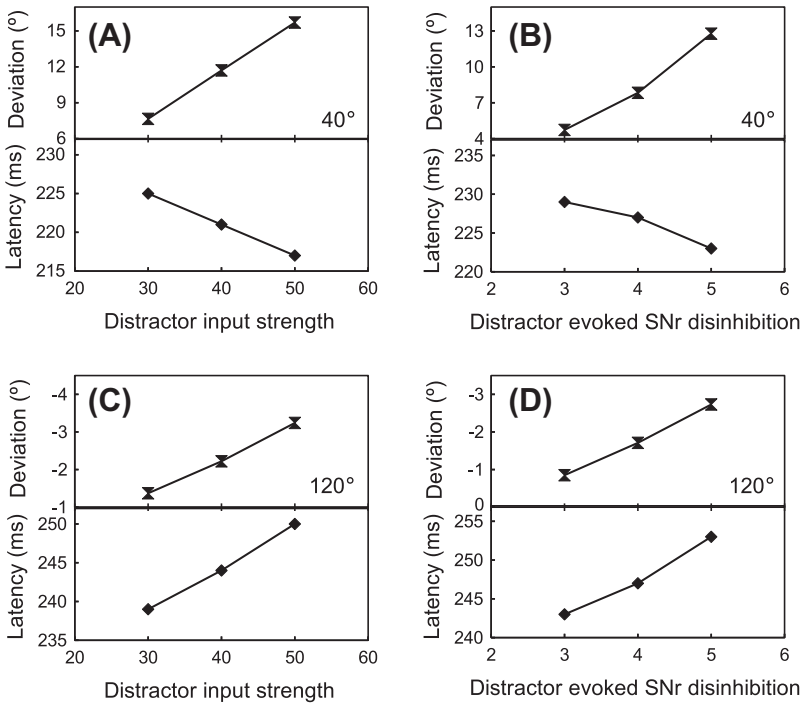

Fig. 4. The relative distractor input strength (A and C) and the magnitude of distractor-evoked SNr disinhibition (B and D) cause saccade latency and deviation away to co-vary. In those simulations, the exogenous target input strength was fixed at $e = 40$, the distractor–target distance was varied between 40° (A and B) and 120° (C and D); for the SNr simulations, DTOA was set to 400 ms.

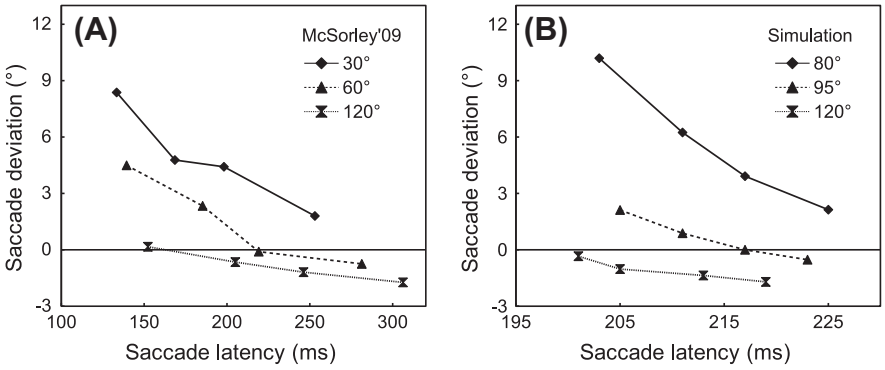

Fig. 5. Temporal gaps cause saccade deviation and saccade latency to co-vary. (A) Human data adapted from the McSorley, Cruickshank, and Inman (2009). (B) Simulation results. The angular distance between the distractor and target are presented in the legend.

Marino et al., 2011).[4] This top-down preparatory input reflects cognitive factors such as temporal or spatial expectation of the target (e.g., Paré & Munoz, 1996). In our simulations, we assumed that a broad endogenous preparatory input ($\sigma_e = 2.4$ mm), which linearly increase with time (0.03/ms), arrived at the SC 0, 50, 100 or 150 ms before target onset. As shown in Fig. 5, our model reproduced the pattern of results reported in McSorley, Cruickshank, and Inman (2009), including the transition from deviation toward distractors to deviation away from distractors in their 60° (distractor–target angular distance) condition. However, extensive simulations demonstrated that this pattern of results could only be produced when the preparatory input was sent to a location in between the target and the distractor. The exact spatial and temporal dynamics of top-down preparatory input is largely unknown (but see Marino et al., 2011). It is possible that our simulations, as presented in Fig. 5B, have explored a parameter space similar to McSorley, Cruickshank, and Inman (2009).

It is important to note that, as shown in our simulations (Fig. 5B) and in McSorley, Cruickshank, and Inman (2009; see Fig. 5A), the transition from deviation toward distractors to deviation away from distractors also depends on distractor–target distance. The transition only occurred in the 60° condition of McSorley, Cruickshank, and Inman (2009) and in the 95° condition of our simulation. When the distractor and target are presented at a distance where the global effect is typically observed, although deviation toward distractors is reduced as saccade latency increases (Fig. 5A), no deviation away from distractors has been reported in the literature.

4. General discussion

Contrary to human intuition, saccades almost never go directly to the target, but instead they usually deviate from the optimum

[4] It is possible that these responses are also contributed to by the decreasing of fixation activity at the rostral pole, which globally suppresses neuronal activity in the caudal SC (e.g., Dorris & Munoz, 1995).

track to their intended destination. This phenomenon is most prominent when the saccade target is accompanied by a visual distractor. Previous studies have demonstrated that saccades may deviate toward or away from a visual distractor. In the literature, deviation toward a distractor is generally explained with lateral interaction or "population coding" that is implemented in the SC, however, the underlying mechanism(s) of deviation away from distractors is still under debate. Many scholars speculated that deviation away from distractors may have a very different neural implementation, i.e., top-down inhibition at the distractor location (for reviews, see Van der Stigchel, Meeter, & Theeuwes, 2006; Walker & McSorley, 2008).

Rather than assuming separate mechanisms for deviation toward and away from distractors, the present study proposed that both phenomena can be produced by the lateral interactions within the SC. This theory has two simple principles. First, the lateral interaction within the SC is Mexican-hat shaped (Trappenberg et al., 2001); when the distractor and target are in the range of lateral competition, they push themselves away from each other (see Fig. 1D). Second, the tonic SNr inhibition to the SC can be temporarily released by visual stimulation (Hikosaka & Wurtz, 1983). The later principle can explain the observation of deviation away from distractors when distractors are presented before target onset (White, Theeuwes, & Munoz, 2012) by causing long-lasting activity in the collicular map in response to the distractor.

4.1. Previous modeling work on saccade deviation

Recent work by Arai and Keller (2005) and Meeter, Van der Stigchel, and Theeuwes (2010) also explored saccade deviation with neural field models, but none of them reported successful simulations of deviation away from distractors. The interaction kernel used by Arai and Keller (2005, p. 24) was similar to the one used in simulations presented in Fig. 2B, however, the spatial resolution of their 2-dimensional model was low (20×20 nodes for one SC). As mentioned before, deviation away from distractors is a small behavioral effect, so it is possible that the low spatial resolution of their model led to undetected deviation away from distractors. The lateral interaction kernel used by Meeter, Van der Stigchel, and Theeuwes (2010) was Gaussian-shaped and a saccade vector was determined by the average vector rather than the peak of a winning activity in the network. According to our theory of deviation away from distractors (see Fig. 1D), this model cannot produce such deviations without external mechanisms.

Even though our model produced both deviation toward and away from distractors without the help of external (top-down) inhibition, the present study does not preclude the possibility that some observations of deviation away from distractors are contributed to by active inhibition to the SC. Previous studies showed that reversible inactivation of an SC region can cause saccades to deviate away from the response field of the inactivated region (Aizawa & Wurtz, 1998; Lee, Rohrer, & Sparks, 1988; Quaia et al., 1998). By suppressing node activity in the inactivated region, a similar result has been produced in a neural field model of the SC (Badler & Keller, 2002) using a lateral interaction kernel similar to the one used in the present study. This suggests that external inhibition inducing deviation away from distractors is computationally feasible, and one possible source of this external inhibition is the SNr (White & Munoz, 2011). Previous studies have shown that some SNr neurons increase rather than decrease their level of discharge around the time of saccade onset (Handel & Glimcher, 1999; Jiang, Stein, & McHaffie, 2003), so the SNr modulates SC activity through both enhancing and releasing of tonic inhibition.

4.2. Neural signature of saccade deviations

Several recent studies have explicitly explored the neural signature of saccade deviations in the SC (McPeek, Han, & Keller, 2003; White, Theeuwes, & Munoz, 2012) and the frontal eye fields (FEF) (McPeek, 2006). White, Theeuwes, and Munoz (2012) tested a DTOA of 400 ms and recorded distractor evoked neuronal activity in the SCi. The relatively long DTOA allowed a test of the top-down inhibition theory of deviation away from distractors. If deviation away is caused by top-down inhibition at the distractor location, one would expect a decrease of distractor evoked activity before target onset. However, no such reduction was observed for saccades that deviated away from the distractor, as compared to those deviated toward the distractor. Instead, differences in neuronal activity for saccades that deviated away from and toward distractors were only observed in a small perisaccade time window, starting about 20 ms before saccade onset (for a similar observation in FEF, see McPeek, 2006). It is unclear whether this perisaccade neuronal difference is responsible for saccade deviations. Taking into account an efferent delay (20–30 ms), this neuronal difference emerges after, rather than before, saccade decisions are made in the SC. However, electrical stimulation delivered to the SC in this time window appears to be effective in eliciting deviation toward the response field of the stimulated SC site (McPeek, Han, & Keller, 2003).

4.3. Deviation away from attended, memorized and expected locations

In addition to irrelevant visual distractors, saccades may also deviate away from attended (Sheliga, Riggio, & Rizzolatti, 1994, 1995; Sheliga et al., 1995, 1997), remembered (Theeuwes, Olivers, & Chizk, 2005) and expected (Van der Stigchel & Theeuwes, 2005) locations. These observations may also have a connection to SNr. The SNr is a major output station of the basal ganglia and it receives inhibitory projections from the caudate nucleus (CD). Previous studies have reported enhanced CD neuronal activity related to attention, memory and expectation (for a review, see Hikosaka, Takikawa, & Kawagoe, 2000). It is possible that attending, memorizing or expecting a (distractor) location enhances CD activity, inhibits SNr, and leads to a spontaneous discharge in the SC. As demonstrated in our simulations, such spontaneous SC activity caused by SNr disinhibition will interact with target-related activity to produce saccade deviations.

4.4. Limitations of the present work

Although our model was successful at simulating a wide range of experimental findings on saccade deviations, several limitations of the model need to be acknowledged. First, as our model represents a single collicular slice, only oblique saccades were simulated in the present work, as opposed to vertical saccades that are commonly explored in behavioral experiments. Second, the present paper focuses on the SC activity up to saccade onset and thereby models only the initial saccade directions. It is possible that the SC activity affects the trajectory of a saccade after its initiation (e.g., Meeter, Van der Stigchel, & Theeuwes, 2010; Walton, Sparks, & Gandhi, 2005), but how SC activity interacts with the brainstem to drive saccades was not considered in the present paper. Third, the present model assumes that saccade metrics are determined by the peak of a winning SC activity. This is not identical to the often proposed schemes of vector averaging or vector summation of SC activity to define the motor command (Gandhi & Katnani, 2011). Nevertheless, it is not expected that an exploration of those decoding mechanisms would yield very different results, as in our model the activity at the time of saccade onset is primarily centered around the target node. Finally, although the 1-dimension

simplification of our model provides an accurate representation for stimuli interacting at similar eccentricity, the model cannot explore the dynamics of the interactions at different eccentricities. Similarly, the contribution of fixation neurons at the rostral pole has not been considered in our simulations. A 2-dimensional model would be needed to obtain more accurate simulation of behavioral and neurophysiological observations.

4.5. Testable predictions

Our simulations have produced several predictions that can be tested in future behavioral and neurophysiological studies.

(1) A connection between deviation and distractor–target distance has been suggested by several studies which showed stronger deviations away for distractors present in the same field as the target than for those presented in the opposite field (e.g., Doyle & Walker, 2001; McSorley, Haggard, & Walker, 2004). To our knowledge, McSorley, Cruickshank, and Inman (2009) is the only study that systematically varied the angular distance between the distractor and the target, up to 120°. The relationship between saccade deviation and distractor–target distance, as presented in Fig. 2A, warrants further empirical explorations.

(2) Our simulations showed that both deviation toward and away from distractors can be produced even with a relatively long DTOA (Fig. 2D). This prediction needs further empirical verification. In addition, the simulations predicted that, with a long DTOA, the amount of saccade deviation varies with the magnitude of SNr disinhibition (Fig. 4B and D). This prediction calls for neurophysiological investigations of SNr neuronal activity in a distractor paradigm.

(3) Marino et al. (2011) demonstrated that the source, size and number of inputs to the SC may interact to produce seemingly unexplainable behavioral observations. The present study has shed some light on how the timing of stimulus onset and fixation offset would interact in this parameter space. Our simulations determined that the localization of top-down preparatory activity, as elicited by temporal gaps, was vital for successful simulation of the behavioral results of McSorley, Cruickshank, and Inman (2009). Further exploration of the spatial and temporal characteristics of top-down preparatory responses in the SC is needed.

5. Conclusion

The present study introduced a unified theory of saccade deviation based on the lateral interactions in the SC. This theory was tested with simulations in a neural field model. Model simulations suggest that generally assumed external (top-down) inhibition to the SC is not necessary to elicit saccades which deviate away from task irrelevant distractors. Nevertheless, as demonstrated in previous behavioral studies, top-down factors, such as expectancy, memory and attention, do affect saccade deviations. Behavioral, neurophysiological and computational investigations of these factors are strongly encouraged.

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References

Aizawa, H., & Wurtz, R. H. (1998). Reversible inactivation of monkey superior colliculus. I. Curvature of saccadic trajectory. *Journal of Neurophysiology, 79*(4), 2082–2096.

Amari, S. (1977). Dynamics of pattern formation in lateral-inhibition type neural fields. *Biological Cybernetics, 27*(2), 77–87. http://dx.doi.org/10.1007/BF00337259.

Arai, K., Das, S., Keller, E. L., & Aiyoshi, E. (1999). A distributed model of the saccade system: Simulations of temporally perturbed saccades using position and velocity feedback. *Neural Networks, 12*(10), 1359–1375. http://dx.doi.org/10.1016/S0893-6080(99)00077-5.

Arai, K., & Keller, E. L. (2005). A model of the saccade-generating system that accounts for trajectory variations produced by competing visual stimuli. *Biological Cybernetics, 92*(1), 21–37. http://dx.doi.org/10.1007/s00422-004-0526-y.

Arai, K., Keller, E. L., & Edelman, J. A. (1994). Two-dimensional neural network model of the primate saccadic system. *Neural Networks, 7*(6–7), 1115–1135. http://dx.doi.org/10.1016/S0893-6080(05)80162-5.

Badler, J. B., & Keller, E. L. (2002). Decoding of a motor command vector from distributed activity in superior colliculus. *Biological Cybernetics, 86*(3), 179–189.

Basso, M. A., & Wurtz, R. H. (2002). Neuronal activity in substantia nigra pars reticulata during target selection. *Journal of Neuroscience, 22*(5), 1883–1894.

Becker, W., & Jürgens, R. (1979). An analysis of the saccadic system by means of double step stimuli. *Vision Research, 19*(9), 967–983. http://dx.doi.org/10.1016/0042-6989(79)90222-0.

Chou, I., Sommer, M., & Schiller, P. (1999). Express averaging saccades in monkeys. *Vision Research, 39*(25), 4200–4216.

Coren, S., & Hoenig, P. (1972). Effect of non-target stimuli upon length of voluntary saccades. *Perceptual and Motor Skills, 34*(2), 499–508.

Dorris, M. C., Klein, R. M., Everling, S., & Munoz, D. P. (2002). Contribution of the primate superior colliculus to inhibition of return. *Journal of Cognitive Neuroscience, 14*(8), 1256–1263. http://dx.doi.org/10.1162/089892902760807249.

Dorris, M. C., & Munoz, D. P. (1995). A neural correlate for the gap effect on saccadic reaction times in monkey. *Journal of Neurophysiology, 73*(6), 2558–2562.

Dorris, M. C., Olivier, E., & Munoz, D. P. (2007). Competitive integration of visual and preparatory signals in the superior colliculus during saccadic programming. *The Journal of Neuroscience, 27*(19), 5053–5062. http://dx.doi.org/10.1523/JNEUROSCI.4212-06.2007.

Dorris, M. C., Paré, M., & Munoz, D. P. (1997). Neuronal activity in monkey superior colliculus related to the initiation of saccadic eye movements. *The Journal of Neuroscience, 17*(21), 8566–8579.

Doyle, M., & Walker, R. (2001). Curved saccade trajectories: Voluntary and reflexive saccades curve away from irrelevant distractors. *Experimental Brain Research, 139*(3), 333–344. http://dx.doi.org/10.1007/s002210100742.

Edelman, J. A., & Keller, E. L. (1998). Dependence on target configuration of express saccade-related activity in the primate superior colliculus. *Journal of Neurophysiology, 80*(3), 1407–1426.

Fecteau, J. H., & Munoz, D. P. (2005). Correlates of capture of attention and inhibition of return across stages of visual processing. *Journal of Cognitive Neuroscience, 17*(11), 1714–1727. http://dx.doi.org/10.1162/089892905774589235.

Findlay, J. M. (1982). Global visual processing for saccadic eye movements. *Vision Research, 22*(8), 1033–1045. http://dx.doi.org/10.1016/0042-6989(82)90040-2.

Gandhi, N. J., & Katnani, H. A. (2011). Motor functions of the superior colliculus. *Annual Review of Neuroscience, 34*(1), 205–231. http://dx.doi.org/10.1146/annurev-neuro-061010-113728.

Glimcher, P. W., & Sparks, D. L. (1993). Representation of averaging saccades in the superior colliculus of the monkey. *Experimental Brain Research, 95*(3), 429–435.

Godijn, R., & Theeuwes, J. (2002). Programming of endogenous and exogenous saccades: Evidence for a competitive integration model. *Journal of Experimental Psychology: Human Perception and Performance, 28*(5), 1039–1054. http://dx.doi.org/10.1037/0096-1523.28.5.1039.

Godijn, R., & Theeuwes, J. (2004). The relationship between inhibition of return and saccade trajectory deviations. *Journal of Experimental Psychology: Human Perception and Performance, 30*(3), 538–554.

Handel, A., & Glimcher, P. W. (1999). Quantitative analysis of substantia nigra pars reticulata activity during a visually guided saccade task. *Journal of Neurophysiology, 82*(6), 3458–3475.

Helms, M. C., Özen, G., & Hall, W. C. (2004). Organization of the intermediate gray layer of the superior colliculus. I. Intrinsic vertical connections. *Journal of Neurophysiology, 91*(4), 1706–1715. http://dx.doi.org/10.1152/jn.00705.2003.

Hikosaka, O., Takikawa, Y., & Kawagoe, R. (2000). Role of the basal ganglia in the control of purposive saccadic eye movements. *Physiological Reviews, 80*(3), 953–978.

Hikosaka, O., & Wurtz, R. H. (1983). Visual and oculomotor functions of monkey substantia nigra pars reticulata. I. Relation of visual and auditory responses to saccades. *Journal of Neurophysiology, 49*(5), 1230–1253.

Isa, T., Endo, T., & Saito, Y. (1998). The visuo-motor pathway in the local circuit of the rat superior colliculus. *The Journal of Neuroscience, 18*(20), 8496–8504.

Isa, T., & Hall, W. C. (2009). Exploring the superior colliculus in vitro. *Journal of Neurophysiology, 102*(5), 2581–2593. http://dx.doi.org/10.1152/jn.00498.2009.

Jiang, H., Stein, B. E., & McHaffie, J. G. (2003). Opposing basal ganglia processes shape midbrain visuomotor activity bilaterally. *Nature, 423*(6943), 982–986. http://dx.doi.org/10.1038/nature01698.

Joseph, J. P., & Boussaoud, D. (1985). Role of the cat substantia nigra pars reticulata in eye and head movements. I. Neural activity. *Experimental Brain Research, 57*(2), 286–296.

Kopecz, K. (1995). Saccadic reaction times in gap/overlap paradigms: A model based on integration of intentional and visual information on neural, dynamic fields. *Vision Research, 35*(20), 2911–2925. http://dx.doi.org/10.1016/0042-6989(95)00066-9.

Kopecz, K., & Schöner, G. (1995). Saccadic motor planning by integrating visual information and pre-information on neural dynamic fields. *Biological Cybernetics, 73*(1), 49–60. http://dx.doi.org/10.1007/BF00199055.

Lee, C., Rohrer, W. H., & Sparks, D. L. (1988). Population coding of saccadic eye movements by neurons in the superior colliculus. *Nature, 332*(6162), 357–360. http://dx.doi.org/10.1038/332357a0.

Ludwig, C. J. H., & Gilchrist, I. D. (2002). Measuring saccade curvature: A curve-fitting approach. *Behavior Research Methods, Instruments, & Computers, 34*(4), 618–624.

Marino, R. A., Trappenberg, T. P., Dorris, M., & Munoz, D. P. (2011). Spatial interactions in the superior colliculus predict saccade behavior in a neural field model. *Journal of Cognitive Neuroscience, 24*(2), 315–336. http://dx.doi.org/10.1162/jocn_a_00139.

McPeek, R. M. (2006). Incomplete suppression of distractor-related activity in the frontal eye field results in curved saccades. *Journal of Neurophysiology, 96*(5), 2699–2711. http://dx.doi.org/10.1152/jn.00564.2006.

McPeek, R. M., Han, J. H., & Keller, E. L. (2003). Competition between saccade goals in the superior colliculus produces saccade curvature. *Journal of Neurophysiology, 89*(5), 2577–2590. http://dx.doi.org/10.1152/jn.00657.2002.

McSorley, E., Cruickshank, A., & Inman, L. (2009). The development of the spatial extent of oculomotor inhibition. *Brain Research, 1298*, 92–98. http://dx.doi.org/10.1016/j.brainres.2009.08.081.

McSorley, E., Haggard, P., & Walker, R. (2004). Distractor modulation of saccade trajectories: Spatial separation and symmetry effects. *Experimental Brain Research, 155*(3), 320–333. http://dx.doi.org/10.1007/s00221-003-1729-5.

McSorley, E., Haggard, P., & Walker, R. (2006). Time course of oculomotor inhibition revealed by saccade trajectory modulation. *Journal of Neurophysiology, 96*(3), 1420–1424. http://dx.doi.org/10.1152/jn.00315.2006.

McSorley, E., Haggard, P., & Walker, R. (2009). The spatial and temporal shape of oculomotor inhibition. *Vision Research, 49*(6), 608–614. http://dx.doi.org/10.1016/j.visres.2009.01.015.

Meeter, M., Van der Stigchel, S., & Theeuwes, J. (2010). A competitive integration model of exogenous and endogenous eye movements. *Biological Cybernetics, 102*(4), 271–291. http://dx.doi.org/10.1007/s00422-010-0365-y.

Meredith, M. A., & Ramoa, A. S. (1998). Intrinsic circuitry of the superior colliculus: Pharmacophysiological identification of horizontally oriented inhibitory interneurons. *Journal of Neurophysiology, 79*(3), 1597–1602.

Mulckhuyse, M., Van der Stigchel, S., & Theeuwes, J. (2009). Early and late modulation of saccade deviations by target distractor similarity. *Journal of Neurophysiology, 102*(3), 1451–1458. http://dx.doi.org/10.1152/jn.00068.2009.

Munoz, D. P., & Istvan, P. J. (1998). Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *Journal of Neurophysiology, 79*(3), 1193–1209.

Ottes, F. P., Van Gisbergen, J. A. M., & Eggermont, J. J. (1984). Metrics of saccade responses to visual double stimuli: Two different modes. *Vision Research, 24*(10), 1169–1179. http://dx.doi.org/10.1016/0042-6989(84)90172-X.

Paré, M., & Munoz, D. P. (1996). Saccadic reaction time in the monkey: Advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *Journal of Neurophysiology, 76*(6), 3666–3681.

Phongphanphanee, P., Marino, R., Kaneda, K., Yanagawa, Y., Munoz, D. P., & Isa, T. (2008). *The lateral interaction in the superficial and intermediate layers of the mouse superior colliculus slice*. Abstract presented at the Society for Neuroscience, Washington, DC.

Port, N. L., & Wurtz, R. H. (2003). Sequential activity of simultaneously recorded neurons in the superior colliculus during curved saccades. *Journal of Neurophysiology, 90*(3), 1887–1903. http://dx.doi.org/10.1152/jn.01151.2002.

Quaia, C., Aizawa, H., Optican, L. M., & Wurtz, R. H. (1998). Reversible inactivation of monkey superior colliculus. II. Maps of saccadic deficits. *Journal of Neurophysiology, 79*(4), 2097–2110.

Reuter-Lorenz, P. A., Hughes, H. C., & Fendrich, R. (1991). The reduction of saccadic latency by prior offset of the fixation point: An analysis of the gap effect. *Perception and Psychophysics, 49*(2), 167–175. http://dx.doi.org/10.3758/BF03205036.

Robinson, D. A. (1972). Eye movements evoked by collicular stimulation in the alert monkey. *Vision Research, 12*(11), 1795–1808. http://dx.doi.org/10.1016/0042-6989(72)90070-3.

Saslow, M. G. (1967). Effects of components of displacement-step stimuli upon latency for saccadic eye movement. *Journal of the Optical Society of America, 57*(8), 1024–1029.

Satel, J., Wang, Z., Trappenberg, T. P., & Klein, R. M. (2011). Modeling inhibition of return as short-term depression of early sensory input to the superior colliculus. *Vision Research, 51*(9), 987–996. http://dx.doi.org/10.1016/j.visres.2011.02.013.

Sheliga, B. M., Craighero, L., Riggio, L., & Rizzolatti, G. (1997). Effects of spatial attention on directional manual and ocular responses. *Experimental Brain Research, 114*(2), 339–351. http://dx.doi.org/10.1007/PL00005642.

Sheliga, B. M., Riggio, L., Craighero, L., & Rizzolatti, G. (1995). Spatial attention-determined modifications in saccade trajectories. *Neuroreport, 6*(3), 585–588.

Sheliga, B. M., Riggio, L., & Rizzolatti, G. (1994). Orienting of attention and eye movements. *Experimental Brain Research, 98*(3), 507–522. http://dx.doi.org/10.1007/BF00233988.

Sheliga, B. M., Riggio, L., & Rizzolatti, G. (1995). Spatial attention and eye movements. *Experimental Brain Research, 105*(2), 261–275. http://dx.doi.org/10.1007/BF00240962.

Theeuwes, J., & Godijn, R. (2004). Inhibition-of-return and oculomotor interference. *Vision Research, 44*(12), 1485–1492. http://dx.doi.org/10.1016/j.visres.2003.09.035.

Theeuwes, J., Kramer, A. F., Hahn, S., & Irwin, D. E. (1998). Our eyes do not always go where we want them to go: Capture of the eyes by new objects. *Psychological Science, 9*(5), 379–385. http://dx.doi.org/10.1111/1467-9280.00071.

Theeuwes, J., Olivers, C. N. L., & Chizk, C. L. (2005). Remembering a location makes the eyes curve away. *Psychological Science, 16*(3), 196–199. http://dx.doi.org/10.1111/j.0956-7976.2005.00803.x.

Tipper, S. P., Howard, L. A., & Jackson, S. R. (1997). Selective reaching to grasp: Evidence for distractor interference effects. *Visual Cognition, 4*(1), 1–38. http://dx.doi.org/10.1080/713756749.

Trappenberg, T., Dorris, M., Munoz, D., & Klein, R. (2001). A model of saccade initiation based on the competitive integration of exogenous and endogenous signals in the superior colliculus. *Journal of Cognitive Neuroscience, 13*(2), 256–271.

Van der Stigchel, S. (2010). Recent advances in the study of saccade trajectory deviations. *Vision Research, 50*(17), 1619–1627. http://dx.doi.org/10.1016/j.visres.2010.05.028.

Van der Stigchel, S., Meeter, M., & Theeuwes, J. (2006). Eye movement trajectories and what they tell us. *Neuroscience and Biobehavioral Reviews, 30*(5), 666–679. http://dx.doi.org/10.1016/j.neubiorev.2005.12.001.

Van der Stigchel, S., Meeter, M., & Theeuwes, J. (2007). The spatial coding of the inhibition evoked by distractors. *Vision Research, 47*(2), 210–218. http://dx.doi.org/10.1016/j.visres.2006.11.001.

Van der Stigchel, S., & Theeuwes, J. (2005). Relation between saccade trajectories and spatial distractor locations. *Cognitive Brain Research, 25*(2), 579–582. http://dx.doi.org/10.1016/j.cogbrainres.2005.08.001.

Van Gisbergen, J. A. M., Van Opstal, A. J., & Tax, A. A. M. (1987). Collicular ensemble coding of saccades based on vector summation. *Neuroscience, 21*(2), 541–555. http://dx.doi.org/10.1016/0306-4522(87)90140-0.

van Opstal, A. J., & van Gisbergen, J. A. (1990). Role of monkey superior colliculus in saccade averaging. *Experimental Brain Research, 79*(1), 143–149.

Walker, R., Deubel, H., Schneider, W. X., & Findlay, J. M. (1997). Effect of remote distractors on saccade programming: Evidence for an extended fixation zone. *Journal of Neurophysiology, 78*(2), 1108–1119.

Walker, R., & McSorley, E. (2008). The influence of distractors on saccade target selection: Saccade trajectory effect. *Journal of Eye Movement Research, 2*(3), 1–13.

Walker, R., McSorley, E., & Haggard, P. (2006). The control of saccade trajectories: Direction of curvature depends on prior knowledge of target location and saccade latency. *Perception & Psychophysics, 68*(1), 129–138. http://dx.doi.org/10.3758/BF03193663.

Walton, M. M. G., Sparks, D. L., & Gandhi, N. J. (2005). Simulations of saccade curvature by models that place superior colliculus upstream from the local feedback loop. *Journal of Neurophysiology, 93*(4), 2354–2358. http://dx.doi.org/10.1152/jn.01199.2004.

Wang, Z., Satel, J., Trappenberg, T. P., & Klein, R. M. (2011). Aftereffects of saccades explored in a dynamic neural field model of the superior colliculus. *Journal of Eye Movement Research, 4*(2), 1–16.

Watanabe, K. (2001). Inhibition of return in averaging saccades. *Experimental Brain Research, 138*(3), 330–342.

White, B. J., & Munoz, D. P. (2011). The superior colliculus. In S. Liversedge, I. Gilchrist, & S. Everling (Eds.), *Oxford handbook of eye movements* (pp. 195–213). New York, NY: Oxford University Press.

White, B. J., Theeuwes, J., & Munoz, D. P. (2012). Interaction between visual- and goal-related neuronal signals on the trajectories of saccadic eye movements. *Journal of Cognitive Neuroscience, 24*(3), 707–717. http://dx.doi.org/10.1162/jocn_a_00162.

Wilimzig, C., Schneider, S., & Schoener, G. (2006). The time course of saccadic decision making: Dynamic field theory. *Neural Networks, 19*(8), 1059–1074. http://dx.doi.org/10.1016/j.neunet.2006.03.003.

Wilson, H. R., & Cowan, J. D. (1973). A mathematical theory of the functional dynamics of cortical and thalamic nervous tissue. *Biological Cybernetics, 13*(2), 55–80. http://dx.doi.org/10.1007/BF00288786.

Wurtz, R. H., & Goldberg, M. E. (1972). Activity of superior colliculus in behaving monkey. III. Cells discharging before eye movements. *Journal of Neurophysiology, 35*(4), 575–586.