Saccade-like Behaviour in the Fast-Phases of Optokinetic Nystagmus: An Illustration of the Emergence of Volitional Actions from Automatic Reflexes

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Abstract

As a potential exemplar for understanding how volitional actions emerged from reflexes, we studied the relationship between an ancient reflexive gaze stabilisation mechanism (optokinetic nystagmus) and purposeful eye movements (saccades) that target an object. Traditionally these have been considered distinct (except in the kinematics of their execution) and studied independently. We find that the fast-phases of optokinetic nystagmus (OKN) clearly show properties associated with saccade planning: (1) They are characteristically delayed by irrelevant distractors in an indistinguishable way to saccades (the saccadic inhibition effect); (2) horizontal OKN fast-phases produce curvature in vertical targeting saccades, just like a competing saccade plan. Thus we argue that the saccade planning network plays a role in the production of OKN fast-phases, and question the need for a strict distinction between eye movements that appear to be automatic or volitional. We discuss whether our understanding might benefit from shifting perspective and considering the entire 'saccade' system to have developed from an increasingly sophisticated OKN system.

Introduction

Understanding automaticity and volition

One of the characteristics lying at the heart of human behaviour is the interplay between automatic and volitional control of actions. Consider walking down a street. The action of walking is achieved by very many animal species encompassing a wide range of brain sizes,

and often appears stereotyped, repetitive and highly automated. Yet when you walk down a street puddles are missed, adjustments are made to accommodate raised kerbs, cracked pavement or slippery patches, and your feet generally avoid stepping on unsavoury items. Thus, like so many human behaviours, there are underlying processes showing inherent flexibility and selection (and therefore appearing to be 'volitional') alongside others that appear automatic. A key question is the extent to which such volitional and automatic aspects of behaviour are controlled by separate systems or by one integrated network - do our notions of volition and automaticity only describe useful categories of behavioural output, or do they actually map onto distinct types of neural system?

If we assume distinct neural systems for automatic and volitional processes, then an intuitive analogy is that of an automatic pilot or cruise control system and a human pilot or driver. The cruise control system is sufficient as long as the road and traffic are predictable, but if anything tricky or unusual is perceived, the driver takes over command. Most areas of psychology have been built upon similar fundamental distinctions between actions that appear automatic, inflexible and can be handled by relatively unintelligent neural mechanisms and those that appear consciously willed, which involve intelligent selection between competing decisional alternatives, and require highly sophisticated neural processes (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977, 1984). This dichotomy between 'automatic' and 'voluntary' processes remains embedded in many contemporary articles across a variety of disciplines: for example in spatial attention (Barbot, Landy, & Carrasco, 2012; Chica, Bartolomeo, & Lupiáñez, 2013; Ibos, Duhamel, & Ben Hamed, 2013; Macaluso & Doricchi, 2013; McAuliffe, Johnson, Weaver, Deller-Quinn, & Hansen, 2013; Mysore & Knudsen, 2013; D. T. Smith, Schenk, & Rorden, 2012); temporal attention (Lawrence & Klein, 2013); cognition (Lifshitz, Bonn, Fischer, Kashem, & Raz, 2013); motor cueing (Martín-Arévalo, Kingstone, & Lupiáñez, 2013); reading (Feng, 2012); perception (Pfister,

Heinemann, Kiesel, Thomaschke, & Janczyk, 2012; Spence & Deroy, 2013); social cognition/perception (Laidlaw, Risko, & Kingstone, 2012) or emotion regulation (R. Viviani, 2013). Similarly, 'voluntary' and 'automatic' actions are clearly distinguished in clinical literature, for conditions ranging from deafness (Bottari, Valsecchi, & Pavani, 2012), to Parkinson's disease (D'Ostilio, Cremers, Delvaux, Sadzot, & Garraux, 2013; van Stockum, MacAskill, & Anderson, 2012; van Stockum, MacAskill, Myall, & Anderson, 2013; Vervoort et al., 2013), Huntington's disease (Patel, Jankovic, Hood, Jeter, & Sereno, 2012), autism (Vernazza-Martin, Longuet, Chamot, & Orève, 2013) and mild traumatic brain injury (Zhang, Red, Lin, Patel, & Sereno, 2013).

Presumably volitional actions originally evolved gradually and incrementally out of simpler reflexive behaviour (Post & Leibowitz, 1985; Walls, 1962). By emphasising the apparent differences between automatic and volitional behaviour we may be missing the opportunity to learn about complex 'volitional' systems from studying the more tractable automatic mechanisms; the classic distinction may actually be unhelpful for penetrating the mysteries of volition itself. Some lines of research have begun to emphasise the connections, rather than the distinctions, between processes that appear automatic or volitional, as a means to begin understanding how 'volitional' processes might have developed from 'automatic' ones (for reviews, see McBride, Boy, Husain, & Sumner, 2012; Sumner & Husain, 2008). In this view, the 'driver' is not a distinct mechanism from the 'cruise control' that operates in parallel and occasionally takes command; rather the cruise control system incrementally becomes more sophisticated and able to flexibly handle all the tricky situations. Avoiding puddles is just as much part of 'walking' as is putting one foot in front of the other; the more sophisticated job is not necessarily run by a distinct system.

Eye movements as exemplar effector system

While the interplay between processes that appear automatic or volitional has relevance to many areas of psychology, to study it, one must choose an effector system as an exemplar. Oculomotor control can usefully encapsulate the debate and serve to test specific hypotheses. Oculomotor decisions are the most frequent we make, many of which appear to be volitionally willed, and have been used as models for decisions in general (Carpenter & Williams, 1995; Cutsuridis, Smyrnis, Evdokmds, & Perantonis, 2007; P. L. Smith & Ratcliff, 2004). The underlying machinery of oculomotor behaviour is relatively well understood, partly because oculomotor tasks allow simple, easily controlled and easily implemented paradigms that are also well-suited to primate neurophysiology (Bell, Everling, & Munoz, 2000; Munoz & Everling, 2004; White, Theeuwes, & Munoz, 2011). In this way eye movements are able to link the fields of psychology and neurophysiology.

Moreover intentional eye movements exist alongside gaze stabilisation mechanisms that are paradigmatic exemplars of ancient reflexive behaviour and whose characteristics and neural underpinning has been extensively researched. Intentional foveation and fixation of a specific point is achieved through saccades and smooth-pursuit eye movements; whereas automatic gaze stabilisation is achieved largely through the vestibular-ocular reflex (VOR) and optokinetic nystagmus (OKN). VOR rotates the eye in response to vestibular signals generated by the head's motion (Hess, 2011), while OKN rotates the eye in response to visual motion signals. Both reflexes help negate certain components of movement upon the retina that would otherwise occur during self-motion or when viewing moving scenes, such as out of a moving train window (Bárany, 1907 and Purkinje, 1825, as cited in Bender & Shanzer, 1983; Findlay & Gilchrist, 2003). These reflexive gaze stabilisation mechanisms are assumed to predate the emergence of 'intentional' saccades and pursuit (Post & Leibowitz, 1985), since they are present in every mobile-eyed vertebrate so-far investigated (Walls, 1962), and are present from birth in humans (Distler & Hoffmann, 2011; Luna & Velanova, 2011).

Stare vs. look OKN

OKN consists of two distinct phases: the 'slow phase' is a smooth rotation of the eye in response to a large consistent field of motion on the retina. Each slow phase is followed by a resetting jump serving to reposition the eye in its orbit (the fast-phase). Previous authors have drawn a distinction between OKN that arises from viewing stimuli purposefully and attentively ('look-OKN', which has strong similarities with purposeful pursuit), and that which arises from passively viewing a moving stimulus ('stare-OKN'; Ter Braak, 1936, as cited in Fite, 1968). We focus on stare-OKN, which is considered to be fully automatic (Pola & Wyatt, 1985), occurs reflexively with a latency of about 100ms whenever large field retinal motion occurs (Collewijn, 1972; Gellman, Carl, & Miles, 1990), is characterised by shorter amplitude waveforms with a higher frequency of fast-phases than look-OKN (Cheng & Outerbridge, 1974; Knapp, Gottlob, McLean, & Proudlock, 2008) and (unlike look-OKN) is not associated with activity in cortical areas (Kashou et al., 2010; Konen, Kleiser, Seitz, & Bremmer, 2005; Schraa-Tam et al., 2009). Henceforth when we simply use 'OKN', we refer to stare-OKN unless otherwise stated.

OKN slow phases and pursuit

The slow-phases even of stare-OKN share characteristics with 'voluntary' smooth pursuit; for example they appear to be affected similarly by stimulus velocity and size (Pola & Wyatt, 1985). Furthermore initial smooth pursuit is driven by a largely reflexive initial rapid rise in velocity that some authors have stated is the same mechanism that drives the slow-phases of OKN (Gellman et al., 1990; Pola & Wyatt, 1985). The perceptual system is also similarly affected by OKN slow-phases and smooth pursuit, flashed targets are misperceived in the direction of the eye movement in both cases (Brenner, Smeets, & van den Berg, 2001; Kaminiarz, Krekelberg, & Bremmer, 2007; Tozzi, Morrone, & Burr, 2007), and sensitivity to

chromatic contrast is improved during both smooth pursuit (Schütz, Braun, Kerzel, & Gegenfurtner, 2008) and OKN slow-phases (Schütz, Braun, & Gegenfurtner, 2009).

OKN fast phases and saccades

The focus of this paper will be on the fast-phases of OKN and their relationship with goal-directed, intentional saccades. Both are fast and abrupt in order to minimise the amount of time visual perception is disrupted (Enderle & Wolfe, 1987; Harris & Wolpert, 2006; Ilg, Bremmer, & Hoffmann, 1993). Since 'saccade' simply means abrupt or fast change, the terms 'fast-phase' and 'saccade' have been used interchangeably by some authors when discussing nystagmus (Baloh, Yee, & Honrubia, 1980; Cheng & Outerbridge, 1974). Properties shared by intentional saccades and automatic fast-phases suggest that they may be generated by overlapping neural networks. For example, the distribution of fast-phase intervals is similar to that of both visually evoked and spontaneous saccades (Carpenter, 1993; Roos, Calandrini, & Carpenter, 2008), including a population very short latencies possibly analogous to express saccades (Carpenter, 1994; Fischer et al., 1993), and can be modelled by accumulator models originally designed for saccades (Carpenter & Williams, 1995; Roos et al., 2008). Additionally both saccades and fast phases show the systematic relationship between amplitude and velocity referred to as the 'main sequence' (Bahill, Clark, & Stark, 1975; Guitton & Mandl, 1980; Ron, Robinson, & Skavenski, 1972). The main sequences of saccades and fast-phases are very similar, but not quite identical (Garbutt, Harwood, & Harris, 2001; Kaminiarz, Königs, & Bremmer, 2009), which suggests they are executed by partially overlapping brainstem circuitry (Anastasio, 1997; Bense et al., 2006; Curthoys, 2002; Lueck & Kennard, 1990). Indeed, it is considered likely that this brainstem 'saccade' machinery evolved first for OKN, and purposeful saccades emerged later (Ron et al., 1972; Walls, 1962). The relationship between OKN fast-phases and saccades could

therefore represent an ideal model for understanding how processes that appear volitional developed from automatic reflexes.

Despite this appeal, however, saccades and OKN are normally assumed to be controlled by separate processes (Konen et al., 2005), in line with the traditional dichotomy between actions that appear automatic or volitional. While fast-phases of OKN are assumed to be wholly generated in the brainstem circuitry simply in response to the eye deviation caused by the OKN slow phase (Waddington & Harris, 2012), saccades are known to be planned through a network of higher-level areas involving the superior colliculus, basal ganglia and frontal and parietal cortices (Scudder, 1988). Indeed some models of fast-phase generation (Anastasio, 1997; Curthoys, 2002) are explicitly models of saccade generation (e.g. Scudder, 1988) with top-down input via the SC (superior colliculus) removed, just as a cruise control system lacks an intentional driver. Consistent with this idea, functional imaging has not found activity for stare-OKN fast-phases in the saccade network (Kashou et al., 2010; Konen et al., 2005).

However, if we are to understand purposeful saccades as emerging from automatic OKN fast phases, a shift in perspective may be warranted. Rather than considering the cortico-collicular 'saccade' planning network as being built afresh on top of the 'lower' machinery responsible for OKN, we might consider the cortico-collicular network to have originally developed to gradually increase the sophistication of OKN – specifically, to allow fast phases to land on specific objects, rather than simply resetting the eye in its orbit. Under natural viewing conditions moving observers do not appear to make fast-phases and saccades separately; rather the fast-phases of OKN have target-selecting properties (Moeller, Kayser, Knecht, & Konig, 2004). Indeed, the conceptual distinction between cortico-collicular planned saccades and brainstem-driven OKN may have arisen simply because most saccade studies are conducted with stationary observers and stimulus backgrounds, where no OKN is

elicited, while most OKN studies are conducted with simple moving stimulus fields without any objects of interest or any task that would require targeting saccades (e.g. Collewijn, 1975; Konen et al., 2005).

Do OKN fast phases show evidence of saccade planning and competition?

It is well established that saccades show properties associated with the fast-phases of OKN (e.g. a similar main sequence [Garbutt et al., 2001]). This has previously been postulated to be because the saccade network commandeers the lower-level brainstem machinery that produce OKN fast-phases (Ron et al., 1972; Walls, 1962). However, if saccades arose from a gradual increase in the sophistication of the optokinetic system as opposed to the evolution of a separate, higher-level network, then a strong prediction is that OKN fast-phases should show most of the same properties as saccades; critically, properties associated with planning in the cortico-collicular network, not just properties such as the main sequence associated with the brainstem. If behaviours that appear volitional are generated by the same system that produces behaviours that appear automatic, not only should volitional behaviours show similar 'mechanical' properties to reflex behaviours (because they use the same effector machinery, as already established), but also the lower-level automatic behaviour should show properties associated with the higher-level 'volitional' behaviour. Thus an integrated system would propose that OKN fast-phases should have the potential to take on properties normally associated with saccades, and show evidence of saccade-like planning and competition.

Here, we use two simple behavioural phenomena readily observable in saccades. In 'saccadic inhibition', irrelevant distractor stimuli that appear during the saccade planning period cause a precisely time-locked population of saccades to be inhibited, leaving a characteristic dip in latency distribution when time-locked to distractor onset, as shown in Figure 1 (Buonocore & McIntosh, 2008, 2012, 2013; Edelman & Xu, 2009; Reingold &

Stampe, 1999, 2002, 2004). This is thought to occur because the distractor stimulus automatically elicits competing activity in saccade-planning areas such as the superior colliculus, which inhibits the rise-to-threshold of saccade-related activity in a competitive neural map (Reingold & Stampe, 2002). The first experiment therefore investigated whether fast-phases show the same characteristic effect, which would suggest that they are subject to sensory activity in the 'saccade' network.

In a second experiment we tested whether fast-phases can act as competitive saccades. According to previous work, when two saccades are programmed in parallel they elicit two sites of activity on 'maps' in the saccade planning network, representing the two saccade end-points, that interact with one another to produce a saccade that is curved (McPeek, Han, & Keller, 2003; McPeek & Keller, 2000). These competing sites of activity are usually thought to exist in the superior colliculus (SC) as shown in Figure 2 (Port & Wurtz, 2003; Walton, Sparks, & Gandhi, 2005), though recent evidence suggests that some forms of curvature might actually originate not in the SC, but in higher cortical areas (White et al., 2011). Either way, if OKN can cause curvature in saccades, this would suggest that activity representing the OKN fast phases must exist in the 'saccade' network and interact with saccade planning.

Experiment 1 (Saccadic Inhibition)

'Saccadic inhibition' is a robust phenomenon whereby irrelevant distractor stimuli inhibit a population of saccades that would otherwise have occurred around 90ms later (Buonocore & McIntosh, 2008; Edelman & Xu, 2009; Reingold & Stampe, 1999, 2000, 2002, 2003, 2004). Figure 1 (reproduced from Bompas and Sumner, 2011) illustrates the characteristic dip in saccade latency distributions when time-locked to distractor onset. Typically, the dip onset is approximately 70ms, reaching a maximum at approximately 100ms. Saccadic inhibition is thought to arise because irrelevant distractor stimuli automatically drive activity in saccade-

processing areas such as the superior colliculus, which then delays the rise to threshold of saccade-related build-up activity through mutual inhibition. This has the effect of reducing the number of saccades occurring shortly after the distractor, causing the dip in the distribution of saccade latencies. Note that the dip begins for saccades that would have occurred 70ms after the distractor; saccades with shorter latencies escape the distractor's influence. Experiment 1 investigated whether irrelevant distractors would elicit a dip in OKN fast-phase distributions, as they do for saccades.

Materials and Methods

Participants

Eight observers participated in Experiment 1, all reporting normal vision. Four were female and age range was 23-28 years. One participant was an author on this paper. All participants gave informed consent and were fully aware of the purpose of the experiment. All procedures had been approved by the School of Psychology, Cardiff University Ethics Committee.

We chose the approach of obtaining many samples in relatively few participants because saccadic inhibition is a remarkably robust phenomenon. Reingold and Stampe (2004) have previously noted that between participant-variability in saccadic inhibition is very low; they showed that it did not matter whether one used individual or pooled data for analysing the dips in the latency distribution. As would be expected, the effect sizes in previous studies using saccadic inhibition have been very high (N = 64 reported effects, mean $r = 0.82$, SD = 0.11) (Buonocore & McIntosh, 2012, 2013; Reingold & Stampe, 2000, 2002, 2003, 2004; Stampe & Reingold, 2002).

Materials

Stimuli were rendered using OpenGL software running on a Raedeon 9800 Pro graphics card and displayed (gamma corrected) through rear projection using a Sony Multiscan projector (VPH 1272QM) onto a large screen (208×156cm) at a refresh rate of 72Hz. The screen had an embedded Fresnel lens, which collimated light evenly throughout the display. Only the central 'green' cathode ray tube of the projector was used and a 0.9 neutral density filter was placed over the projector to lower its luminance. Other than the presented stimuli, the lab was completely dark.

Eye-movements were recorded at a rate of 1000Hz using an SR Eyelink 2000 eye-tracker mounted on a chin and forehead rest 140cm from the screen. Calibration was performed before each experimental block by having observers fixate nine dots in a 3×3 array, each separated by 10°. Between each trial a drift correct was conducted - participants fixated a dot (diameter 0.6°) presented in the centre of the screen for 300ms, and any discrepancy between recorded and actual location of the eye was then corrected for off-line.

Stimuli and Procedure

The effect of distractor stimuli was investigated for two eye-movement conditions: standard targeting saccades (figure 3A) or OKN fast-phases (figure 3B). Each condition was conducted on a separate day, with four participants conducting the standard targeting saccade condition first.

The methods used for the standard targeting saccade condition closely resembled those of Reingold and Stampe (2002). A central fixation point (radius = 0.3°, brightness = 1.24 cd/m^2) was displayed in the centre of the screen for 800 or 1200ms, whereupon it stepped 8° either to the left or the right (see figure 3A). We chose to use two alternative fore-periods in order to exactly match the methods of Reingold and Stampe (2002). Previous studies have used random duration fore-periods (Buonocore & McIntosh, 2008) and fixed-

duration fore-periods (Bompas & Sumner, 2011) and have not found divergent results. Furthermore, for saccadic inhibition, latency is measured with respect to distractor onset, which was randomised. On 50% of trials a distractor stimulus was presented for 30ms, consisting of two bars (1.24 cd/m^2) that filled the screen from ±18° vertically outwards. The bars were more peripheral than those used by Reingold and Stampe (2002) in order to accommodate the requirements of the OKN stimuli described below (in a separate experiment we investigated the influence of eccentricity by repeating the saccade condition with bars placed at ±7°). At the start of each block, 50 baseline trials were run without distractors so that the mean saccadic latency of each observer could be measured. Distractors in subsequent trials were then triggered 50-150ms prior to this value (which was updated throughout the experiment using the 50 preceding no-distractor trials), thus ensuring that the expected dip would fall within the distribution of saccades. Observers completed two blocks, each consisting of 50 baseline trials followed by 400 trials, of which half contained a distractor stimulus. Between each block the lights were turned on and participants given the opportunity to rest.

For the OKN condition, nystagmus was elicited by presenting observers with a band (16° high, 73° wide) of coherently moving random dots (radius = 0.3°, brightness = 0.1cd/m^2, density of 0.5 dots/deg^2, speed 32°/sec, either to the left or right). Between each trial a blank screen of brightness 0.38cd/m^2 was displayed for five seconds to stop participants from dark-adapting during the experiment, which might have allowed them to perceive the external stationary features of the room, as this can disrupt OKN. Participants were instructed not to track any particular dot in the display, but at the same time to not let the band of dots become blurred. To allow other stimuli to be presented at specific points in the OKN waveform, on-line detection of fast-phases was achieved using a velocity criterion of 92°/sec.

Participants viewed the band of OKN-eliciting dots until 15 fast-phases had been detected. Distractor stimuli were then flashed for 30ms every 6-8 detected fast-phases, at a random time between 85 and 235ms following fast-phase detection (see figure 3B). This procedure continued for 30 seconds, and constituted one trial. Forty-five of these trials were conducted per block, and there were five blocks in the experiment. The OKN condition therefore produced approximately 2000 distractor stimulus onsets per participant.

Data Analysis

Eye-movements were analysed off-line using Matlab software (version 2010a, Mathworks Inc.). Eye traces were first smoothed using a Gaussian filter (SD = 16Hz). Targeting saccades were then detected using a velocity criterion of 100°/sec, with the start of the saccade taken to be the time that the velocity first rose above 20°/sec. Fixation was detected when the eye did not deviate by more than 0.3° over a 100ms period. Fast-phases of the OKN waveform were identified using a combination of acceleration (location of zero-crossing), eye-velocity (average velocity across the fast-phase of at least 60°/sec), local minima and maxima of position, and direction of motion (fast-phases nearly always travel against stimulus motion). All trials were visually checked by the experimenter.

In order to plot and calculate the metrics of saccadic inhibition, latency distributions need to be plotted with respect to distractor onset, not target onset (Stampe & Reingold, 2002). This in turn requires a method to create comparison distributions for the no-distractor trials that have no distractor upon which to time-lock. For the saccade condition, we therefore placed a ‘phantom’ distractor in each no-distractor trial. The phantom distractor onset had the same timing as the previous distractor trial. In the OKN condition a similar procedure was used: phantom distractors were placed in OKN slow phases that did not have actual distractors, based on the timing of the most recent actual distractor trial relative to its

preceding fast-phase. Distributions of saccade onsets and fast phase onsets were taken with a bin-size of 1 ms (this being the temporal resolution of the eye-tracker), and were smoothed using a Gaussian filter with a standard deviation of 20ms (from the analysis described in Bompas & Sumner, 2011). The distraction ratio (*(baseline –distractor distribution)/ baseline*) was calculated (as in Bompas & Sumner, 2011; Reingold & Stampe, 2004). The onset of 'saccadic inhibition' was taken when the distraction ratio first rose above 2% (following Bompas & Sumner, 2011).

Results

OKN had a mean resetting fast-phase frequency of 3.07Hz (SD = 0.36Hz), this is a strong indication our experiment elicited reflexive OKN, sometimes referred to as 'stare OKN' (Cheng & Outerbridge, 1974; Freeman & Sumnall, 2005; Kolarik, Margrain, & Freeman, 2010).

The results in Figure 4 show that irrelevant distractor stimuli cause a dip in the latency distributions of OKN fast-phases for every participant (left-hand panels). OKN fast-phases therefore display the 'saccadic inhibition' effect. Pooled data distributions (right-hand panels of Figure 4) reveal that our experiment found the standard dip in saccadic conditions (top and bottom), and also that a dip was present in the OKN fast-phase condition (middle).

The mean onset time of the dip for the saccadic condition (18° distractors) was 96 ms (SD = 9 ms; see blue symbols in Figure 4), which is comparable to the mean onset time for the OKN condition (mean = 94 ms, SD = 18 ms, $t(7) = -0.28$, $p = 0.79$, $r = 0.11$). For both OKN and saccades with 18° distractors, mean dip onsets were later than those for saccades with 7° distractors ($t(7) = 4.08$, $p = 0.005$, $r = 0.84$; $t(7) = 4.78$, $p = 0.002$, $r = 0.87$), presumably due to the more peripheral distractors (which have never been tested in previous literature). For saccades with 7° distractors the mean dip onset was 72ms (SD = 11 ms), in

line with previous literature (Bompas & Sumner, 2011; Edelman & Xu, 2009; Reingold & Stampe, 2002).

The amplitude of the dip is expressed as the percentage of saccades or fast-phases inhibited at the dip maximum point. There is no indication that the inhibition effect is smaller in amplitude for OKN than for saccades with the same distractors; in fact it is larger, with the distractor inhibiting an average 88% (SD = 9%) of fast-phases at its peak, whereas only 65% (SD = 15%) of saccades were inhibited at its peak ($t(7) = 3.39$, $p = 0.012$, $r = 0.79$). Note that the mean of individual dip amplitudes is larger than the dip amplitude in the pooled data distribution shown in Figure 4 owing to slight variability in the dip maximum point across participants (represented by horizontal red error bars in the pooled data distributions of Figure 4; note that this error bar represents the full range, not the SE or SD that were too small to plot clearly). Being larger, the dip peak is also later, since the peak depends on the amplitude, given that the onset time is fixed by sensory and motor delays in and out of the oculomotor system (see Bompas & Sumner, 2011). Note that superficial differences in the breadth of the dips and the distribution around the dips between OKN fast-phases and saccades are expected from the different shapes in the baseline distributions (dashed lines, Figure 4). Therefore the most relevant measure is the timing of the dip onset.

Thus the presence of an irrelevant distractor causes a dip in the latencies of OKN fast-phases just as it does for saccades. The onset of this dip for both OKN and saccades is later than has been previously reported when the distractor stimuli were presented at a greater eccentricity than in prior studies. Presenting the distractor stimuli more centrally (as is done in established experiments) results in earlier dip onsets that are comparable to previously published results (Bompas & Sumner, 2011; Edelman & Xu, 2009; Reingold & Stampe, 2002). The common effect that a distractor has on both fast-phases and saccades suggests that they are generated with shared mechanisms. In Experiment 2 we probed this relationship

further by investigating whether another well-known saccadic effect, namely the curvature produced by competing saccade plans, occurred for targeting saccades when made in the presence of OKN fast-phases.

Experiment 2 (Saccade Curvature)

If two saccades are programmed in parallel, this induces activity coding for two competing saccade-end points that have been recorded as being represented in the superior colliculus (McPeek & Keller, 2001, 2002; Port & Wurtz, 2003). The interaction between the two sources of activity can result in a saccade that is curved, such that the trajectory of a saccade executed due to one locus of activity deviates towards the other site of activity (McPeek et al., 2003; McPeek & Keller, 2000). Figure 2A illustrates how the parallel planning of a horizontal fast-phase and a near-vertical saccade might theoretically elicit activity in two discrete locations in the SC, or more generally in the saccade planning network.

To test this hypothesis we asked observers to make vertical saccades during horizontal OKN (Figure 5A and 5B). At the point at which the vertical saccade is executed, there may be activity already in the oculomotor system planning a horizontal fast-phase, as illustrated in Figure 5B. If this occurs, we would expect these sites of activity to interact and thus the targeting saccade to deviate in the direction of the fast-phase (see Figure 2A). We refer to this direction of curvature as the 'competition-predicted' direction.

The vertical saccades were aimed at a bar that stretched across the width of the screen. We did not use a point as the saccade target because it is known that saccades can curve to correct for target displacements (Findlay & Harris, 1984). By using a bar there is less need for the oculomotor system to make online horizontal adjustments to the saccade trajectory so that it lands on target; thus we can better isolate any curvature attributable to competing activity elicited by fast-phases. Potentially observers could attempt to land on some

identifiable point on the bar, such as its centre. But any online correction would create curvature in the opposite direction to the fast-phase because the eye is displaced in the slow-phase direction. Thus, any curvature produced by in-flight correction is opposite to the 'competition-predicted' direction, and so could only obscure the 'competition-predicted' curvature, rather than be confounded with it.

Further evidence that any curvature in the targeting saccade depends upon fast phases, rather than other aspects of the design, can be gleaned from the timing of saccades relative to the fast phases. Curvature due to interaction between the two types of eye movement would be expected to peak at around the time of the fast phase, and be reduced for saccades elicited farther in time from the fast phase.

Materials and Methods

Participants

Eight observers participated in Experiment 2, five of whom were female and age range was 22-28 years. None of the participants were authors of this paper. All reported normal vision. Six observers had previously participated in Experiment 1. All participants gave informed consent, and all procedures were vetted by the Ethics Committee for the School of Psychology, Cardiff University. Similarly to Experiment 1, we took the approach of numerous observations for relatively few participants because we did not expect the low-level effect of saccade curvature to differ much between subjects; previous studies using oculomotor competition to elicit curvature show large effect sizes (N = 53 reported effects, mean $r = 0.80$, SD = 0.15) (Doyle & Walker, 2002; Hermens, Sumner, & Walker, 2010; McSorley, Haggard, & Walker, 2004, 2009; Nummenmaa & Hietanen, 2006; Theeuwes, Olivers, & Chizk, 2005; Van der Stigchel, Meeter, & Theeuwes, 2007; Van der Stigchel &

Theeuwes, 2005, 2006; van Zoest, Van der Stigchel, & Barton, 2008; Walker, McSorley, & Haggard, 2006; White et al., 2011)

Stimuli and Procedure

OKN was elicited using the same band of dots as in Experiment 1. Participants viewed the band of OKN-eliciting dots until a bar appeared above or below the dots, to which the participants were instructed to make a targeting-saccade (see Figure 5A). The targeting-saccade stimulus was triggered after 11, 12 or 13 detected fast-phases, plus a variable delay of 110-300ms. On 25% of trials (randomly determined) the band of dots did not move to allow baseline measures of targeting saccades without concomitant OKN. Target onset in baseline trials could not be yoked to an OKN waveform, therefore onset time was calculated as if fast-phases had occurred three times a second (Cheng & Outerbridge, 1974; Freeman & Sumnall, 2005; Kolarik et al., 2010).

The target stimulus was a horizontal line stretching across the width of the screen and positioned ±10° from centre of the screen, line height = 0.3°, brightness = 1.24cd/m^2. It was displayed for 50ms. The band of OKN-eliciting dots remained on the screen for 14ms following the target stimulus, meaning the targeting saccade was conducted in the dark. It has been shown that OKN will continue for around a second following extinguishing of all stimuli (Gellman & Fletcher, 1992; Leigh & Zee, 1999). A period of 1000ms was therefore allowed for the targeting saccade, followed by an inter-trial interval of 300ms. The experiment was split into 10 blocks of 40 trials each, and no more than five blocks were completed in a single day.

Data Analysis

Eye-movements were analysed as in Experiment 1. To express the magnitude of saccade curvature, the amplitude and direction of all saccade trajectories were first normalised. A second-order polynomial was then fitted to each saccade trajectory, and the coefficient of the quadratic term was taken to directly represent the magnitude of curvature (following Ludwig & Gilchrist, 2002).

Results

During OKN, we found 54.6% of the targeting saccades had curvature in the competition-predicted direction (i.e. saccades that curved in the direction of the fast-phase). However, given that saccades are rarely exactly straight (P. Viviani, Berthoz, & Tracey, 1977), Figure 6 plots the amplitude of curvature in OKN compared to the baseline condition for both group and individual data. All participants showed larger curvature when deviations were in the competition-predicted direction, compared to baseline or deviations that were not in the competition-predicted direction (see Figure 6A). Seven of our eight participants also showed an increase in the magnitude of curvature from baseline for those deviations that were not competition-predicted (Figure 6B).

Saccades that curved in the direction predicted by competition from OKN fast-phases showed significantly greater deviation than those that did not curve in the competition-predicted direction ($t(7) = -4.28$, $p = 0.004$, $r = 0.85$); they were also significantly more curved than the mean unsigned curvature found in the baseline (no OKN) condition ($t(7) = -6.73$, $p < 0.001$, $r = 0.93$). Hence, the greatest amount of curvature we found was in the direction predicted by an interaction between the fast-phase and saccade planning. The increase from baseline for those saccades that curved against the competition-predicted direction (i.e. those predicted by online correction) was also significant ($t(7) = -3.03$, $p =$

0.019, $r = 0.75$); which may reflect some degree of on-line correction of the saccade towards a point on the target line.

Beyond the simple comparison of curvature in the competition-predicted direction and against it, we predicted an association of curvature with timing. Figure 7A illustrates the activity in build-up neurons associated with saccades (red) and hypothesised activity for fast phases (blue) during four cycles of OKN. The profiles are based on actual cell recordings from Munoz and Wurtz (1995) by tracing recorded neural activity shown in their Figure 2. No adjustment has been made to their temporal dynamics, except to remove the initial visual burst seen for target-evoked saccades (the sharp rise in the red profiles) from the putative fast-phase activity; we have not modelled mutual inhibition. If fast phases are programmed like saccades, activity for them would rise to threshold in the saccade network repeatedly at a rate of about 3Hz. Meanwhile, activity for the vertical saccade would rise in response to the onset of each target stimulus (marked by the grey vertical line in Figure 7A).

The illustrated activity associated with the first targeting saccade in Figure 7A comes about half way between two fast phases and so is least likely to be affected by them. The second saccade comes just after a fast phase; thus its planning overlaps considerably with that of a fast phase, and would incur greater interaction with the fast phase activity. We would also expect a saccade that came just before a fast-phase to be influenced by it, but since the fast phase then does not occur due to cessation of optokinetic stimulation, and OKN is not regular enough to predict exactly when it would have occurred, we have to rely on saccades that follow fast phases to test the hypothesis that saccades initiated near the time of fast phases are most likely to interact with fast-phase planning and thus to curve in the 'competition-predicted' direction.

Consistent with the prediction, Figure 7B shows that the majority of targeting saccades made shortly after an OKN fast-phase (analogous to target-onset (ii) in Figure 7A) deviated in the direction of the fast-phase (competition-predicted direction). This effect then decayed away to a point where targeting saccades were equally likely to deviate in the competition-predicted direction or not. This would be analogous to a saccade to target onset (i) in Figure 7A. This accounts for the fact that overall only a small majority (54.6%) of targeting saccades deviated in the competition-predicted direction. There is a trend showing that the proportion curving in the competition-predicted direction rises again for longer latencies – i.e. when we might expect that the next fast phase is imminent. This is also consistent with our prediction, but since the fast-phase does not actually occur it is harder to be as sure that this rise is due to fast-phase activity. Furthermore the amount of data for the longest delays is inevitably small (represented by the area of the circles in Figure 7B), which makes testing the prediction that curvature is associated with an imminent fast-phase difficult.

The presence of saccade curvature during OKN with deviations in the direction of the fast-phase (especially if the saccade is initiated at around the time of a fast-phase) provides evidence that OKN fast-phases can indeed act like competitive saccades. Thus the generation of saccades cannot be independent from the activity of fast-phases, for fast-phases to have a demonstrable effect on the behaviour of saccades they must share common neural networks.

Discussion

Our results show first that distractor stimuli inhibit OKN fast-phases in the same characteristic way that they inhibit standard, targeting saccades; and second, that targeting saccades are curved in the direction of fast-phases programmed at the same time. This saccade-like behaviour in the fast-phases of OKN implies a functional overlap in the

programming of fast-phases and saccades in the cortico-collicular network where saccadic inhibition and curvature are thought to originate. The functional overlap is therefore not just in the brainstem execution circuitry.

This proposal compliments the evolutionary perspective that saccades developed from fast-phases in the first place, through development of top-down influence over the phylogenetically older fast-phase generating systems (Ron et al., 1972; Walls, 1962). However, we would argue for a shift in perspective: the OKN fast-phase machinery is not commandeered by a separate cortico-collicular 'saccade' network; rather, the cortico-collicular 'saccade' network is not originally a 'saccade' network at all (as viewed for laboratory saccades made by a stationary observer). We speculate that the cortico-collicular network incrementally developed as a means to gradually increase the sophistication of OKN fast-phases.

It is difficult to empirically test whether the oculomotor system is organised as a single unified system or whether there are separate, but interconnected, specialist mechanisms for generating saccades and fast-phases. However, we believe that given the converging evidence, it is more parsimonious to assume the existence of a single system. If saccades were generated by a specialist module built atop the existing optokinetic system, it seems unlikely that the lower system would show properties associated with the higher system. A single network is also a more parsimonious path for evolutionary development to follow. We believe that such development of the oculomotor system could serve as a more general example for how behaviours that appear volitional can emerge from the increasing sophistication of automatic reflexes.

The Relationship between Reflexive and Voluntary Eye-Movements

These experiments can aid our understanding of how stimulus-driven (reflexive) eye-movements interact with consciously-willed (volitional) eye-movements. Some studies have suggested that reflexive and volitional saccades are programmed by distinct populations and engage in a first-past-the-post winner-takes-all race, without directly interacting (Theeuwes, Kramer, Hahn, & Irwin, 1998; Theeuwes, Kramer, Hahn, Irwin, & Zelinsky, 1999; Walker & McSorley, 2006). Conversely others have argued for a competitive integration model where reflexive and volitional eye-movements exist on a common motor map and inevitably influence one another (Godijn & Theeuwes, 2002).

Here we have shown that OKN fast-phases demonstrably interact with targeting saccades. This implies 'reflexive' and 'volitional' eye-movements do indeed exist on a common motor map and lends support to competitive integration models However previous experiments have studied the interaction using saccades driven in different ways (Godijn & Theeuwes, 2002), but drawing an unambiguous distinction between voluntary and reflexive saccades can be difficult. OKN fast-phases are more clearly considered a low-level, reflexive eye-movement, thus we can conclude more unequivocally that there is no distinct segregation between actions that appear reflexive and volitional.

A Role for the Superior Colliculus in OKN Fast-Phases?

We believe that the behavioural effects observed in these experiment may help elucidate some of the neural pathways responsible for the fast-phases of OKN, which currently are far less well-known than those pathways that generate the slow-phase (Waddington & Harris, 2012). One possibility is that the saccade-like effects observed with fast-phases may be attributable to the superior colliculus (SC). This is because both of the behavioural tasks we employed have strong links to processing in the SC. The onset of the saccadic inhibition effect is highly consistent with the sum of the conduction times from stimulus onset to the

SC, and from SC activity to executed saccade (around 60-90ms, Reingold & Stampe, 2000, 2002). Furthermore, sub-threshold stimulation of the SC affects saccades in the same way as distractor stimuli do (Dorris, Olivier, & Munoz, 2007). Additionally, saccadic inhibition is an emergent property of SC models (Bompas & Sumner, 2011; Engbert, 2012).

Dip onsets were no different for saccades and fast-phases with 18° distractors, implying a common mechanism underlying the effect. However dip onsets for both occurred later than has been previously reported (Bompas & Sumner, 2011; Reingold & Stampe, 2002), which we believe is due to the eccentricity of our distractors. It is already well-known that the dip onset depends upon the characteristics of the distractor stimulus; this is assumed to reflect changes in the temporal dynamics of SC processing (Buonocore & McIntosh, 2012; Pannasch, Dornhoefer, Unema, & Velichkovsky, 2001; Reingold & Stampe, 2004; Stampe & Reingold, 2002). Accordingly, dip onsets elicited by different distractors are predicted to reflect the systematic differences in saccadic latency if those distractor stimuli are used as targets (Bompas & Sumner, 2011). Since there is a gradual increase in saccade latency as target eccentricity increases beyond 2° (Bell et al., 2000; Kalesnykas & Hallett, 1994), we expect dip latency to increase with the eccentricity of distractors. This is what we found.

It is possible that the saccadic inhibition effect stems from more than one locus. For example sudden visual transients have been shown to affect activity in omnipause neurones as well as the SC (Boehnke & Munoz, 2008; Everling, Paré, Dorris, & Munoz, 1998; Munoz, Dorris, Paré, & Everling, 2000); as such the crucial interactions may be between SC, omnipause neurones or other brainstem circuitry. Similarly, a component of saccadic inhibition may reflect inhibitory influences from FEF and basal ganglia that impinge upon the SC. However, the data from Experiment 1 indicate that all loci involved in saccadic inhibition are also involved in OKN fast phases - if only a subset were relevant for OKN, we would expect the inhibition effect to be smaller. In fact it was larger than for saccades.

Curvature has also been associated with the SC. During curved saccades, two sites of activity have been observed in the SC (McPeek & Keller, 2002; Port & Wurtz, 2003) and stimulation of the SC elicits saccade curvature towards the stimulated site (McPeek et al., 2003; McPeek & Keller, 2000). Conversely, inactivation of areas of SC using muscimol causes deviations away from the inactivated area (Aizawa & Wurtz, 1998; although see also White et al., 2011). We furthermore observed that the majority of targeting saccades executed shortly after an OKN fast-phase were curved in the direction of that fast-phase, and that after 100ms this effect decayed away to the point at which saccades curved roughly equally in and against the direction of OKN fast-phases. This is in line with what is known about the time course of SC activity and curvature: for two sites of activity in the SC to elicit saccade curvature they must occur in close temporal proximity (Noto & Gnadt, 2009).

The conclusion that OKN fast-phases involve SC processing is in line with observations that activity has been recorded in the SC during OKN fast-phases (Schiller & Stryker, 1972), and that stimulation of the SC can induce nystagmus-like movements (Bergmann, Costin, Gutman, & Chaimovitz, 1964; Straschill & Rieger, 1973). Furthermore the SC is ideally situated to engage in fast-phase related processing, as it has substantial connections between reticular formation areas known to be involved in the generation of the fast-phase of nystagmus (Cohen, Matsuo, Fradin, & Raphan, 1985; Grantyn & Grantyn, 1976; Hikosaka & Kawakami, 1977; Kitama, Ohki, Shimazu, Tanaka, & Yoshida, 1995). We do not claim that the SC is the main site in which fast-phases are generated, as ablation of the SC has little influence on basic fast-phases elicited in standard nystagmus paradigms (Albano & Wurtz, 1982; Pierrot-Deseilligny, Rosa, Masmoudi, Rivaud, & Gaymard, 1991; Schiller, True, & Conway, 1980). However this does not preclude the SC from having a strong functional involvement. For example, whist the FEF and posterior parietal cortex are strongly linked to higher-level saccadic processing, they are not necessary for saccades themselves to

be executed (Lynch, 1992; Lynch & McLaren, 1989).While brainstem burst and pause neurones are the *minimum* neural substrate required for fast-phase generation, connections to higher-level areas such as the SC may modulate fast-phases (Curthoys, 2002).

Putative Role of 'Higher-Level' Processing in Fast-Phases

Our conclusion that the saccade network is integrated with the OKN network does not rely on our observed interactions stemming from the SC. Whilst it is most parsimonious to assume an SC locus, as this is closest to the structures already associated with OKN, there is no reason to rule out contribution from the rest of the network including frontal and parietal cortex. Key features of the 'SC' models accounting for saccadic inhibition also mirror properties of the FEF. Furthermore, recent neurophysiological evidence suggests that these cortical regions can also be a source of saccade curvature, since in a paradigm commonly used to elicit curvature, no curvature-related activity was detected in the SC throughout most of the delay period where it might have been expected (White et al., 2011). As already pointed out, if saccadic inhibition arises from multiple parts of the network, then Experiment 1 implies that OKN fast phases must be susceptible to all these loci, since the inhibition effect was actually larger not smaller than for saccades.

The putative influence of cortical areas upon OKN has so far been limited to the delivery of visual information to, and between subcortical areas. For example, binocular connections mediated via the cortex are assumed to be the mechanism that allows monocular OKN symmetry to develop (Distler & Hoffmann, 1992; Lewis, Maurer, Chung, Holmes-Shannon, & Van Schaik, 2000; Schor, Narayan, & Westall, 1983). Furthermore OKN slow-phase gain and symmetry can be affected by ablation of the SC (Flandrin & Jeannerod, 1981) or the visual cortex (Montarolo, Precht, & Strata, 1981). However, as far as we know, the notion that cortical saccade generating areas could modify OKN fast-phases in a task-relevant

manner, and could be just as relevant to the control of OKN fast-phases as they are for saccades, has never been postulated before. There are potential functional benefits for processing fast-phases at a higher-level. We suspect that this could allow fast-phases to take on flexible, goal-directed behaviour. Under natural viewing conditions moving observers do not appear to make fast-phases and saccades separately; rather the fast-phases of OKN have target-selecting properties (Moeller et al., 2004). Some models of fast-phase generation (Anastasio, 1997; Curthoys, 2002) are explicitly models of saccade generation (e.g. Scudder, 1988) with top-down input from the SC removed. Adding the cortico-collicular network back into the model would account for how fast phases can also target specific stimuli.

We speculate that cortico-collicular areas are silent during OKN only in experimental lab conditions where there are no interesting objects in the visual scene for targeting (Kashou et al., 2010; Konen et al., 2005). In such experiments fast-phases of OKN do not have to be directed to any particular stimuli; without any task-relevant objects in the visual field, the fast-phases act simply to reset the position of the eye following slow-phases, in similar vein to the fast-phases of VOR, that by definition are not made to any visible stimulus. Nevertheless, we would still postulate that VOR would be integrated with higher-level processes that appear volitional. For example the gain of the VOR slow-phase is significantly improved by imagining a stationary, earth-fixed target while in complete darkness, and gains are significantly reduced by imagining a target that moves with head motion (Barr et al., 1976; Melvill-Jones, 1994; Melvill-Jones, Berthoz, & Segal, 1984). For VOR to be modified in a non-visual, goal-directed way such as this shows that it is able to take on flexible behaviour associated with ‘higher-level’ processing that could be analogous to the ability of OKN fast-phases to take on goal-directed, saccade-like behaviour.

Furthermore, ‘higher-level’ connections between saccadic and fast-phase processing could enable targeting saccades to integrate displacements of the eye due to OKN. This

would be crucial to enable the accurate foveation of targets during self-motion, in a similar way to the necessary coordination between vestibular-ocular reflexes and saccades that exists to allow accurate eye-head gaze shifts (Cullen, Huterer, Braidwood, & Sylvestre, 2004; Cullen & Roy, 2004; Jürgens, Becker, & Rieger, 1981).

Summary

We have shown that OKN fast-phases can both demonstrate and affect behavioural phenomena strongly associated with saccades. This suggests that fast-phases and saccades have more than just a superficial similarity stemming from shared peripheral motor circuitry; they are also subject to some of the same pre-processing. On the basis of our data, it appears that cortico-collicular saccade network is functionally involved in the modulation of OKN fast-phases. Our findings therefore provide further evidence that automatic and volitional actions are more strongly integrated than is often thought, and builds upon work that suggests there is no sharp dichotomy between automatic, inflexible movements and voluntary, adaptive movements (McBride et al., 2012). Co-ordination between 'volitional' target-selecting and 'reflexive' gaze-stabilizing systems would allow a moving observer to most efficiently act in rich visual scene. In this view, we have a potential illustration for how an automatic system incrementally develops to exhibit behaviours that appear volitional.

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.

Albano, J. E., & Wurtz, R. H. (1982). Deficits in eye position following ablation of monkey superior colliculus, pretectum, and posterior-medial thalamus. *Journal of Neurophysiology, 48*(2), 318-337.

Anastasio, T. J. (1997). A burst-feedback model of fast-phase burst generation during nystagmus. *Biological Cybernetics, 76*(2), 139-152. doi: 10.1007/s004220050327

Bahill, A. T., Clark, M. R., & Stark, L. (1975). The main sequence, a tool for studying human eye movements. *Mathematical Biosciences, 24*(3–4), 191-204. doi: 10.1016/0025-5564(75)90075-9

Baloh, R. W., Yee, R. D., & Honrubia, V. (1980). Optokinetic nystagmus and parietal lobe lesions. *Annals of Neurology, 7*(3), 269-276. doi: 10.1002/ana.410070311

Barbot, A., Landy, M. S., & Carrasco, M. (2012). Differential effects of exogenous and endogenous attention on second-order texture contrast sensitivity. *Journal of Vision, 12*(8), 1-15. doi: 10.1167/12.8.6

Barr, C. C., Schultheis, L. W., & Robinson, D. A. (1976). Voluntary, non-visual control of the human vestibulo-ocular reflex. *Acta Oto-laryngologica, 81*(3-6), 365-375. doi: 10.3109/00016487609119974

Bell, A. H., Everling, S., & Munoz, D. P. (2000). Influence of stimulus eccentricity and direction on characteristics of pro- and antisaccades in non-human primates. *Journal of Neurophysiology, 84*(5), 2595-2604.

Bender, M. B., & Shanzer, S. (1983). History of optokinetic nystagmus. *Neuro-Ophthalmology, 3*(2), 73-88. doi: 10.3109/01658108309009722

Bense, S., Janusch, B., Vucurevic, G., Bauermann, T., Schlindwein, P., Brandt, T., . . . Dieterich, M. (2006). Brainstem and cerebellar fMRI-activation during horizontal and vertical optokinetic stimulation. *Experimental Brain Research, 174*(2), 312-323. doi: 10.1007/s00221-006-0464-0

Bergmann, F., Costin, A., Gutman, J., & Chaimovitz, M. (1964). Nystagmus evoked from the superior colliculus of the rabbit. *Experimental Neurology, 9*(5), 386-399. doi: 10.1016/0014-4886(64)90073-1

Boehnke, S. E., & Munoz, D. P. (2008). On the importance of the transient visual response in the superior colliculus. *Current Opinion in Neurobiology, 18*(6), 544-551. doi: 10.1016/j.conb.2008.11.004

Bompas, A., & Sumner, P. (2011). Saccadic inhibition reveals the timing of automatic and voluntary signals in the human brain. *Journal of Neuroscience, 31*(35), 12501-12512. doi: 10.1523/JNEUROSCI.2234-11.2011

Bottari, D., Valsecchi, M., & Pavani, F. (2012). Prominent reflexive eye-movement orienting associated with deafness. *Cognitive Neuroscience, 3*(1), 8-13. doi: 10.1080/17588928.2011.578209

Brenner, E., Smeets, J. B. J., & van den Berg, A. V. (2001). Smooth eye movements and spatial localisation. *Vision Research, 41*(17), 2253-2259. doi: 10.1016/S0042-6989(01)00018-9

Buonocore, A., & McIntosh, R. D. (2008). Saccadic inhibition underlies the remote distractor effect. *Experimental Brain Research, 191*(1), 117-122. doi: 10.1007/s00221-008-1558-7

Buonocore, A., & McIntosh, R. D. (2012). Modulation of saccadic inhibition by distractor size and location. *Vision Research, 69*, 32-41. doi: 10.1016/j.visres.2012.07.010

Buonocore, A., & McIntosh, R. D. (2013). Attention modulates saccadic inhibition magnitude. *Quarterly Journal of Experimental Psychology, 66*(6), 1051-1059. doi: 10.1080/17470218.2013.797001

Carpenter, R. H. S. (1993). Distribution of quick-phase intervals in optokinetic nystagmus. *Ophthalmic Research, 25*(2), 91-93. doi: 10.1159/000267270

Carpenter, R. H. S. (1994). Express optokinetic nystagmus. In A. F. Fuchs, T. Brandt, U. Buttner & D. Zee (Eds.), *Contemporary Ocular Motor and Vestibular Research: A Tribute to David A. Robinson* (pp. 185-187). Stuttgart: Georg Thieme Verlag.

Carpenter, R. H. S., & Williams, M. L. L. (1995). Neural computation of log likelihood in the control of saccadic eye movements. *Nature, 377*(6544), 59-62. doi: 10.1038/377059a0

Cheng, M., & Outerbridge, J. S. (1974). Inter-saccadic interval analysis of optokinetic nystagmus. *Vision Research, 14*(11), 1053-1058. doi: 10.1016/0042-6989(74)90201-6

Chica, A. B., Bartolomeo, P., & Lupiáñez, J. (2013). Two cognitive and neural systems for endogenous and exogenous spatial attention. *Behavioural Brain Research, 237*(0), 107-123. doi: 10.1016/j.bbr.2012.09.027

Cohen, B., Matsuo, V., Fradin, J., & Raphan, T. (1985). Horizontal saccades induced by stimulation of the central mesencephalic reticular formation. *Experimental Brain Research, 57*(3), 605-616. doi: 10.1007/bf00237847

Collewijn, H. (1972). Latency and gain of the rabbit's optokinetic reactions to small movements. *Brain Research, 36*(1), 59-70. doi: 10.1016/0006-8993(72)90766-4

Collewijn, H. (1975). Oculomotor areas in the rabbit's midbrain and pretectum. *Journal of Neurobiology, 6*(1), 3-22. doi: 10.1002/neu.480060106

Cousineau, D. (2005). Confidence intervals in within-subject designs: A simpler solution to Loftus and Masson's method. *Tutorials in Quatitative Methods for Psychology, 1*(1), 42-45.

Cullen, K. E., Huterer, M., Braidwood, D. A., & Sylvestre, P. A. (2004). Time course of vestibuloocular reflex suppression during gaze shifts. *Journal of Neurophysiology, 92*(6), 3408-3422. doi: 10.1152/jn.01156.2003

Cullen, K. E., & Roy, J. E. (2004). Signal processing in the vestibular system during active versus passive head movements. *Journal of Neurophysiology, 91*(5), 1919-1933. doi: 10.1152/jn.00988.2003

Curthoys, I. S. (2002). Generation of the quick phase of horizontal vestibular nystagmus. *Experimental Brain Research, 143*(4), 397-405. doi: 10.1007/s00221-002-1022-z

Cutsuridis, V., Smyrnis, N., Evdokmds, L., & Perantonis, S. (2007). A neural model of decision-making by the superior colicullus in an antisaccade task. *Neural Networks, 20*(6), 690-704. doi: 10.1016/j.neunet.2007.01.004

D'Ostilio, K., Cremers, J., Delvaux, V., Sadzot, B., & Garraux, G. (2013). Impaired automatic and unconscious motor processes in Parkinson's disease. *Scientific Reports, 3*. doi: 10.1038/srep02095

Distler, C., & Hoffmann, K. P. (1992). Early development of the subcortical and cortical pathway involved in optokinetic nystagmus: the cat as a model for man? *Behavioural Brain Research, 49*(1), 69-75. doi: 10.1016/s0166-4328(05)80195-0

Distler, C., & Hoffmann, K. P. (2011). The optokinetic reflex. In S. P. Liversedge, I. D. Gilchrist & S. Everling (Eds.), *The Oxford Handbook of Eye Movements* (pp. 65-83). Oxford: Oxford University Press.

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

Doyle, M., & Walker, R. (2002). Multisensory interactions in saccade target selection: Curved saccade trajectories. *Experimental Brain Research, 142*(1), 116-130. doi: 10.1007/s00221-001-0919-2

Edelman, J. A., & Xu, K. Z. (2009). Inhibition of voluntary saccadic eye movement commands by abrupt visual onsets. *Journal of Neurophysiology, 101*(3), 1222-1234. doi: 10.1152/jn.90708.2008

Enderle, J. D., & Wolfe, J. W. (1987). Time-Optimal Control of Saccadic Eye Movements. *Biomedical Engineering, IEEE Transactions on, BME-34*(1), 43-55. doi: 10.1109/tbme.1987.326014

Engbert, R. (2012). Computational modeling of collicular integration of perceptual responses and attention in microsaccades. *Journal of Neuroscience, 32*(23), 8035-8039. doi: 10.1523/jneurosci.0808-12.2012

Everling, S., Paré, M., Dorris, M. C., & Munoz, D. P. (1998). Comparison of the discharge characteristics of brain stem omnipause neurons and superior colliculus fixation

neurons in monkey: Implications for control of fixation and saccade behavior. *Journal of Neurophysiology, 79*(2), 511-528.
Feng, G. (2012). Is there a common control mechanism for anti-saccades and reading eye movements? Evidence from distributional analyses. *Vision Research, 57*(0), 35-50. doi: 10.1016/j.visres.2012.01.001
Findlay, J. M., & Gilchrist, I. D. (2003). *Active Vision: The Psychology of Looking and Seeing*. Oxford: Oxford University Press.
Findlay, J. M., & Harris, L. R. (1984). Small saccades to double-stepped targets moving in two dimensions. In A. G. Gale & F. W. Johnson (Eds.), *Theoretical and Applied Aspects of Eye Movement Research* (pp. 71-78). Amsterdam: Elseveir.
Fischer, B., Weber, H., Biscaldi, M., Aiple, F., Otto, P., & Stuhr, V. (1993). Separate populations of visually guided saccades in humans: Reaction times and amplitudes. *Experimental Brain Research, 92*(3), 528-541. doi: 10.1007/BF00229043
Fite, K. V. (1968). Two types of optomotor response in the domestic pigeon. *Journal of Comparative and Physiological Psychology, 66*(2), 308-314. doi: 10.1037/h0026373
Flandrin, J. M., & Jeannerod, M. (1981). Effects of unilateral superior colliculus ablation on oculomotor and vestibulo-ocular responses in the cat. *Experimental Brain Research, 42*(1), 73-80. doi: 10.1007/bf00235731
Freeman, T. C. A., & Sumnall, J. H. (2005). Extra-retinal adaptation of cortical motion-processing areas during pursuit eye movements. *Proceedings of the Royal Society B: Biological Sciences, 272*(1577), 2127-2132. doi: 10.1098/rspb.2005.3198
Garbutt, S., Harwood, M. R., & Harris, C. M. (2001). Comparison of the main sequence of reflexive saccades and the quick phases of optokinetic nystagmus. *British Journal of Ophthalmology, 85*(12), 1477-1483. doi: 10.1136/bjo.85.12.1477
Gellman, R. S., Carl, J. R., & Miles, F. A. (1990). Short latency ocular-following responses in man. *Visual Neuroscience, 5*(02), 107-122. doi: 10.1017/S0952523800000158
Gellman, R. S., & Fletcher, W. A. (1992). Eye position signals in human saccadic processing. *Experimental Brain Research, 89*(2), 425-434. doi: 10.1007/bf00228258
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. doi: 10.1037//0096-1523.28.5.1039
Grantyn, A. A., & Grantyn, R. (1976). Synaptic actions of tectofugal pathways on abducens motoneurons in the cat. *Brain Research, 105*(2), 269-285. doi: 10.1016/0006-8993(76)90425-x
Guitton, D., & Mandl, G. (1980). A comparison between saccades and quick phases of vestibular nystagmus in the cat. *Vision Research, 20*(10), 865-873. doi: 10.1016/0042-6989(80)90067-x
Harris, C., & Wolpert, D. (2006). The main sequence of saccades optimizes speed-accuracy trade-off. *Biological Cybernetics, 95*(1), 21-29. doi: 10.1007/s00422-006-0064-x
Hermens, F., Sumner, P., & Walker, R. (2010). Inhibition of masked primes as revealed by saccade curvature. *Vision Research, 50*(1), 46-56. doi: 10.1016/j.visres.2009.10.008
Hess, B. J. (2011). Vestibular response. In S. P. Liversedge, I. D. Gilchrist & S. Everling (Eds.), *The Oxford Handbook of Eye Movements* (pp. 45-64). Oxford: Oxford University Press.
Hikosaka, O., & Kawakami, T. (1977). Inhibitory reticular neurons related to the quick phase of vestibular nystagmus - Their location and projection. *Experimental Brain Research, 27*(3), 377-396. doi: 10.1007/bf00235511

Ibos, G., Duhamel, J. R., & Ben Hamed, S. (2013). A functional hierarchy within the parietofrontal network in stimulus selection and attention control. *Journal of Neuroscience, 33*(19), 8359-8369. doi: 10.1523/jneurosci.4058-12.2013

Ilg, U. J., Bremmer, F., & Hoffmann, K.-P. (1993). Optokinetic and pursuit system: A case report. *Behavioural Brain Research, 57*(1), 21-29. doi: 10.1016/0166-4328(93)90057-W

Jürgens, R., Becker, W., & Rieger, P. (1981). Different effects involved in the interaction of saccades and the vestibulo-ocular reflex. *Annals of the New York Academy of Sciences, 374*(1), 744-754. doi: 10.1111/j.1749-6632.1981.tb30915.x

Kalesnykas, R. P., & Hallett, P. E. (1994). Retinal eccentricity and the latency of eye saccades. *Vision Research, 34*(4), 517-531. doi: 10.1016/0042-6989(94)90165-1

Kaminiarz, A., Königs, K., & Bremmer, F. (2009). The main sequence of human optokinetic afternystagmus (OKAN). *Journal of Neurophysiology, 101*(6), 2889-2897. doi: 10.1152/jn.00114.2009

Kaminiarz, A., Krekelberg, B., & Bremmer, F. (2007). Localization of visual targets during optokinetic eye movements. *Vision Research, 47*(6), 869-878. doi: 10.1016/j.visres.2006.10.015

Kashou, N. H., Leguire, L. E., Roberts, C. J., Fogt, N., Smith, M. A., & Rogers, G. L. (2010). Instruction dependent activation during optokinetic nystagmus (OKN) stimulation: An FMRI study at 3 T. *Brain Research, 1336*, 10-21. doi: 10.1016/j.brainres.2010.04.017

Kitama, T., Ohki, Y., Shimazu, H., Tanaka, M., & Yoshida, K. (1995). Site of interaction between saccade signals and vestibular signals induced by head rotation in the alert cat: functional properties and afferent organization of burster-driving neurons. *Journal of Neurophysiology, 74*(1), 273-287.

Knapp, C. M., Gottlob, I., McLean, R. J., & Proudlock, F. A. (2008). Horizontal and vertical look and stare optokinetic nystagmus symmetry in healthy adult volunteers. *Investigative Ophthalmology & Visual Science, 49*(2), 581-588. doi: 10.1167/iovs.07-0773

Kolarik, A. J., Margrain, T. H., & Freeman, T. C. A. (2010). Precision and accuracy of ocular following: influence of age and type of eye movement. *Experimental Brain Research, 201*(2), 271-282. doi: 10.1007/s00221-009-2036-6

Konen, C. S., Kleiser, R., Seitz, R. J., & Bremmer, F. (2005). An fMRI study of optokinetic nystagmus and smooth-pursuit eye movements in humans. *Experimental Brain Research, 165*(2), 203-216. doi: 10.1007/s00221-005-2289-7

Laidlaw, K. E. W., Risko, E. F., & Kingstone, A. (2012). A new look at social attention: Orienting to the eyes is not (entirely) under volitional control. *Journal of Experimental Psychology-Human Perception and Performance, 38*(5), 1132-1143. doi: 10.1037/a0027075

Lawrence, M. A., & Klein, R. M. (2013). Isolating exogenous and endogenous modes of temporal attention. *Journal of Experimental Psychology-General, 142*(2), 560-572. doi: 10.1037/a0029023

Leigh, R. J., & Zee, D. S. (1999). *The Neurology of Eye Movements* (3rd ed.). New York: Oxford University Press.

Lewis, T. L., Maurer, D., Chung, J. Y. Y., Holmes-Shannon, R., & Van Schaik, C. S. (2000). The development of symmetrical OKN in infants: quantification based on OKN acuity for nasalward versus temporalward motion. *Vision Research, 40*(4), 445-453. doi: 10.1016/S0042-6989(99)00190-X

Lifshitz, M., Bonn, N. A., Fischer, A., Kashem, I. F., & Raz, A. (2013). Using suggestion to modulate automatic processes: From Stroop to McGurk and beyond. *Cortex, 49*(2), 463-473. doi: 10.1016/j.cortex.2012.08.007

Ludwig, C. J. H., & Gilchrist, I. D. (2002). Measuring saccade curvature: A curve-fitting approach. *Behavior Research Methods Instruments & Computers, 34*(4), 618-624. doi: 10.3758/bf03195490

Lueck, C. J., & Kennard, C. (1990). Eye-movement control and nystagmus. *Current Opinion in Ophthalmology, 1*(5), 476-483. doi: 10.1097/00055735-199001050-00008

Luna, B., & Velanova, K. (2011). Development from reflexive to controlled eye movements. In S. P. Liversedge, I. D. Gilchrist & S. Everling (Eds.), *The Oxford Handbook of Eye Movements* (pp. 621-642). Oxford: Oxford University Press.

Lynch, J. C. (1992). Saccade initiation and latency deficits after combined lesions of the frontal and posterior eye fields in monkeys. *Journal of Neurophysiology, 68*(5), 1913-1916.

Lynch, J. C., & McLaren, J. W. (1989). Deficits of visual attention and saccadic eye movements after lesions of parietooccipital cortex in monkeys. *Journal of Neurophysiology, 61*(1), 74-90.

Macaluso, E., & Doricchi, F. (2013). Attention and predictions: control of spatial attention beyond the endogenous-exogenous dichotomy. *Frontiers in Human Neuroscience, 7*. doi: 10.3389/fnhum.2013.00685

Marino, R. A., Rodgers, C. K., Levy, R., & Munoz, D. P. (2008). Spatial relationships of visuomotor transformations in the superior colliculus map. *Journal of Neurophysiology, 100*(5), 2564-2576. doi: 10.1152/jn.90688.2008

Martín-Arévalo, E., Kingstone, A., & Lupiáñez, J. (2013). Is "Inhibition of Return" due to the inhibition of the return of attention? *Quarterly Journal of Experimental Psychology, 66*(2), 347-359. doi: 10.1080/17470218.2012.711844

McAuliffe, J., Johnson, M., Weaver, B., Deller-Quinn, M., & Hansen, S. (2013). Body position differentially influences responses to exogenous and endogenous cues. *Attention, Perception, & Psychophysics, 75*(7), 1342-1346. doi: 10.3758/s13414-013-0553-7

McBride, J., Boy, F., Husain, M., & Sumner, P. (2012). Automatic motor activation in the executive control of action. *Frontiers in Human Neuroscience, 6*, 82. doi: 10.3389/fnhum.2012.00082

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. doi: 10.1152/jn.00657.2002

McPeek, R. M., & Keller, E. L. (2000). Competition between saccadic goals in the superior colliculus or frontal eye fields results in curved saccades. [Meeting Abstract]. *Society for Neurosceince Abstracts, 26*, 291.

McPeek, R. M., & Keller, E. L. (2001). Short-term priming, concurrent processing, and saccade curvature during a target selection task in the monkey. *Vision Research, 41*(6), 785-800. doi: 10.1016/s0042-6989(00)00287-x

McPeek, R. M., & Keller, E. L. (2002). Superior colliculus activity related to concurrent processing of saccade goals in a visual search task. *Journal of Neurophysiology, 87*(4), 1805-1815. doi: 10.1152/jn.00501.2001

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

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

Melvill-Jones, G. (1994). On the role of a goal in motor control. In A. F. Fuchs, T. Brandt, U. Buttner & D. Zee (Eds.), *Contemporary Ocular Motor and Vestibular Research: A Tribute to David A. Robinson* (pp. 472-479). Stuttgart: Georg Thieme Verlag.

Melvill-Jones, G., Berthoz, A., & Segal, B. (1984). Adaptive modification of the vestibulo-ocular reflex by mental effort in darkness. *Experimental Brain Research, 56*(1), 149-153. doi: 10.1007/bf00237451

Moeller, G. U., Kayser, C., Knecht, F., & Konig, P. (2004). Interactions between eye movement systems in cats and humans. *Experimental Brain Research, 157*(2), 215-224. doi: 10.1007/s00221-004-1835-z

Montarolo, P. G., Precht, W., & Strata, P. (1981). Functional organization of the mechanisms subserving the optokinetic nystagmus in the cat. *Neuroscience, 6*(2), 231-246. doi: 10.1016/0306-4522(81)90059-2

Munoz, D. P., Dorris, M. C., Paré, M., & Everling, S. (2000). On your mark, get set: Brainstem circuitry underlying saccadic initiation. *Canadian Journal of Physiology and Pharmacology, 78*(11), 934-944. doi: 10.1139/y00-062

Munoz, D. P., & Everling, S. (2004). Look away: The anti-saccade task and the voluntary control of eye movement. *Nature Reviews Neuroscience, 5*, 218-228. doi: 10.1038/nrn1345

Munoz, D. P., & Wurtz, R. H. (1995). Saccade-related activity in monkey superior colliculus. I. Characteristics of burst and buildup cells. *Journal of Neurophysiology, 73*(6), 2313-2333.

Mysore, S. P., & Knudsen, E. I. (2013). A shared inhibitory circuit for both exogenous and endogenous control of stimulus selection. *Nature Neuroscience, 16*(4), 473-U143. doi: 10.1038/nn.3352

Noto, C. T., & Gnadt, J. W. (2009). Saccade trajectories evoked by sequential and colliding stimulation of the monkey superior colliculus. *Brain Research, 1295*, 99-118. doi: 10.1016/j.brainres.2009.07.069

Nummenmaa, L., & Hietanen, J. K. (2006). Gaze distractors influence saccadic curvature: Evidence for the role of the oculomotor system in gaze-cued orienting. *Vision Research, 46*(21), 3674-3680. doi: 10.1016/j.visres.2006.06.004

Pannasch, S., Dornhoefer, S. M., Unema, P. J. A., & Velichkovsky, B. M. (2001). The omnipresent prolongation of visual fixations: saccades are inhibited by changes in situation and in subject's activity. *Vision Research, 41*(25–26), 3345-3351. doi: 10.1016/s0042-6989(01)00207-3

Patel, S. S., Jankovic, J., Hood, A. J., Jeter, C. B., & Sereno, A. B. (2012). Reflexive and volitional saccades: Biomarkers of Huntington disease severity and progression. *Journal of the Neurological Sciences, 313*(1–2), 35-41. doi: 10.1016/j.jns.2011.09.035

Pfister, R., Heinemann, A., Kiesel, A., Thomaschke, R., & Janczyk, M. (2012). Do endogenous and exogenous action control compete for perception? *Journal of Experimental Psychology-Human Perception and Performance, 38*(2), 279-284. doi: 10.1037/a0026658

Pierrot-Deseilligny, C., Rosa, A., Masmoudi, K., Rivaud, S., & Gaymard, B. (1991). Saccade deficits after a unilateral lesion affecting the superior colliculus. *Journal of Neurology, Neurosurgery & Psychiatry, 54*(12), 1106-1109. doi: 10.1136/jnnp.54.12.1106

Pola, J., & Wyatt, H. J. (1985). Active and passive smooth eye movements: Effects of stimulus size and location. *Vision Research, 25*(8), 1063-1076. doi: 10.1016/0042-6989(85)90094-X

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. doi: 10.1152/jn.01151.2002

Post, R. B., & Leibowitz, H. W. (1985). A revised analysis of the role of efference in motion perception. *Perception, 14*(5), 631-643. doi: 10.1068/p140631

Reingold, E. M., & Stampe, D. M. (1999). Saccadic inhibition in complex visual tasks. In W. Becker, H. Deubel & T. Mergner (Eds.), *Current Oculomotor Research: Physiological and Psychological Aspects*. New York: Plenum Press.

Reingold, E. M., & Stampe, D. M. (2000). Saccadic inhibition and gaze contingent research paradigms. In A. Kennedy, R. Radach, D. Heller & J. Pynte (Eds.), *Reading as a Perceptual Process* (pp. 119-145). Oxford: North-Holland.

Reingold, E. M., & Stampe, D. M. (2002). Saccadic inhibition in voluntary and reflexive saccades. *Journal of Cognitive Neuroscience, 14*(3), 371-388. doi: 10.1162/089892902317361903

Reingold, E. M., & Stampe, D. M. (2003). Using the saccadic inhibition paradigm to investigate saccadic control in reading. In J. Hyona, R. Radach & H. Deubel (Eds.), *Mind's Eye: Cognitive and Applied Aspects of Eye Movement Research* (pp. 347-360). Amsterdam: Elsevier Science Publishers.

Reingold, E. M., & Stampe, D. M. (2004). Saccadic inhibition in reading. *Journal of Experimental Psychology-Human Perception and Performance, 30*(1), 194-211. doi: 10.1037/0096-1523.30.1.194

Ron, S., Robinson, D. A., & Skavenski, A. A. (1972). Saccades and the quick phase of nystagmus. *Vision Research, 12*(12), 2015-2022. doi: 10.1016/0042-6989(72)90055-7

Roos, J. C. P., Calandrini, D. M., & Carpenter, R. H. S. (2008). A single mechanism for the timing of spontaneous and evoked saccades. *Experimental Brain Research, 187*(2), 283-293. doi: 10.1007/s00221-008-1304-1

Schiller, P. H., & Stryker, M. (1972). Single-unit recording and stimulation in superior colliculus of the alert rhesus monkey. *Journal of Neurophysiology, 35*(6), 915-924.

Schiller, P. H., True, S. D., & Conway, J. L. (1980). Deficits in eye movements following frontal eye-field and superior colliculus ablations. *Journal of Neurophysiology, 44*(6), 1175-1189.

Schneider, W., & Shiffrin, R. M. (1977). Controlled and automatic human information processing: I. Detection, search, and attention. *Psychological Review, 84*(1), 1-66. doi: 10.1037/0033-295X.84.1.1

Schor, C. M., Narayan, V., & Westall, C. (1983). Postnatal development of optokinetic after nystagmus in human infants. *Vision Research, 23*(12), 1643-1647. doi: 10.1016/0042-6989(83)90178-5

Schraa-Tam, C. K. L., van der Lugt, A., Smits, M., Frens, M. A., van Broekhoven, P. C. A., & van der Geest, J. N. (2009). Differences between smooth pursuit and optokinetic eye movements using limited lifetime dot stimulation: a functional magnetic resonance imaging study. *Clinical Physiology and Functional Imaging, 29*(4), 245-254. doi: 10.1111/j.1475-097X.2009.00858.x

Schütz, A. C., Braun, D. I., & Gegenfurtner, K. R. (2009). Chromatic contrast sensitivity during optokinetic nystagmus, visually enhanced vestibulo-ocular reflex, and smooth pursuit eye movements. *Journal of Neurophysiology, 101*(5), 2317-2327. doi: 10.1152/jn.91248.2008

Schütz, A. C., Braun, D. I., Kerzel, D., & Gegenfurtner, K. R. (2008). Improved visual sensitivity during smooth pursuit eye movements. *Nature Neuroscience, 11*(10), 1211-1216. doi: 10.1038/nn.2194

Scudder, C. A. (1988). A new local feedback model of the saccadic burst generator. *Journal of Neurophysiology, 59*(5), 1455-1475.
Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review, 84*(2), 127-190. doi: 10.1037/0033-295X.84.2.127
Shiffrin, R. M., & Schneider, W. (1984). Automatic and controlled processing revisited. *Psychological Review, 91*(2), 269-276. doi: 10.1037/0033-295X.91.2.269
Smith, D. T., Schenk, T., & Rorden, C. (2012). Saccade preparation is required for exogenous attention but not endogenous attention or IOR. *Journal of Experimental Psychology-Human Perception and Performance, 38*(6), 1438-1447. doi: 10.1037/a0027794
Smith, P. L., & Ratcliff, R. (2004). Psychology and neurobiology of simple decisions. *Trends in Neurosciences, 27*(3), 161-168. doi: 10.1016/j.tins.2004.01.006
Spence, C., & Deroy, O. (2013). How automatic are crossmodal correspondences? *Consciousness and Cognition, 22*(1), 245-260. doi: 10.1016/j.concog.2012.12.006
Stampe, D. M., & Reingold, E. M. (2002). Influence of stimulus characteristics on the latency of saccadic inhibition. In J. Hyona, D. P. Munoz, W. Heide & R. Radach (Eds.), *Brain's Eye: Neurobiological and Clinical Aspects of Oculomotor Research* (Vol. 140, pp. 73-87). Amsterdam: Elsevier Science B.V.
Straschill, M., & Rieger, P. (1973). Eye movements evoked by focal stimulation of the cat's superior colliculus. *Brain Research, 59*(0), 211-227. doi: 10.1016/0006-8993(73)90262-x
Sumner, P., & Husain, M. (2008). At the edge of consciousness: Automatic motor activation and voluntary control. *Neuroscientist, 14*(5), 474-486. doi: 10.1177/1073858408314435
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. doi: 10.1111/1467-9280.00071
Theeuwes, J., Kramer, A. F., Hahn, S., Irwin, D. E., & Zelinsky, G. J. (1999). Influence of attentional capture on oculomotor control. *Journal of Experimental Psychology-Human Perception and Performance, 25*(6), 1595-1608. doi: 10.1037//0096-1523.25.6.1595
Theeuwes, J., Olivers, C. N. L., & Chizk, C. L. (2005). Remembering a Location Makes the Eyes Curve Away. *Psychological Science, 16*(3), 196-199. doi: 10.1111/j.0956-7976.2005.00803.x
Tozzi, A., Morrone, M. C., & Burr, D. C. (2007). The effect of optokinetic nystagmus on the perceived position of briefly flashed targets. *Vision Research, 47*(6), 861-868. doi: 10.1016/j.visres.2006.10.022
Van der Stigchel, S., Meeter, M., & Theeuwes, J. (2007). Top-down influences make saccades deviate away: The case of endogenous cues. *Acta Psychologica, 125*(3), 279-290. doi: 10.1016/j.actpsy.2006.08.002
Van der Stigchel, S., & Theeuwes, J. (2005). Relation between saccade trajectories and spatial distractor locations. *Cognitive Brain Research, 25*(2), 579-582. doi: 10.1016/j.cogbrainres.2005.08.001
Van der Stigchel, S., & Theeuwes, J. (2006). Our eyes deviate away from a location where a distractor is expected to appear. *Experimental Brain Research, 169*(3), 338-349. doi: 10.1007/s00221-005-0147-2
van Stockum, S., MacAskill, M. R., & Anderson, T. J. (2012). Impairment of voluntary saccades and facilitation of reflexive saccades do not co-occur in Parkinson's disease. *Journal of Clinical Neuroscience, 19*(8), 1119-1124. doi: 10.1016/j.jocn.2011.10.014

van Stockum, S., MacAskill, M. R., Myall, D., & Anderson, T. J. (2013). A perceptual discrimination task results in greater facilitation of voluntary saccades in Parkinson's disease patients. *European Journal of Neuroscience, 37*(1), 163-172. doi: 10.1111/ejn.12033

van Zoest, W., Van der Stigchel, S., & Barton, J. J. S. (2008). Distractor effects on saccade trajectories: a comparison of prosaccades, antisaccades, and memory-guided saccades. *Experimental Brain Research, 186*(3), 431-442. doi: 10.1007/s00221-007-1243-2

Vernazza-Martin, S., Longuet, S., Chamot, J. M., & Orève, M. J. (2013). Emotions and voluntary action: What link in children with autism? *Behavioural Brain Research, 251*(0), 176-187. doi: 10.1016/j.bbr.2013.05.049

Vervoort, G., Nackaerts, E., Mohammadi, F., Heremans, E., Verschueren, S., Nieuwboer, A., & Vercruysse, S. (2013). Which aspects of postural control differentiate between patients with Parkinson's Disease with and without freezing of gait? *Parkinsons Disease*. doi: 10.1155/2013/971480

Viviani, P., Berthoz, A., & Tracey, D. (1977). The curvature of oblique saccades. *Vision Research, 17*(5), 661-664. doi: 10.1016/0042-6989(77)90143-2

Viviani, R. (2013). Emotion regulation, attention to emotion, and the ventral attentional network. *Frontiers in Human Neuroscience, 7*. doi: 10.3389/fnhum.2013.00746

Waddington, J., & Harris, C. M. (2012). Human optokinetic nystagmus: A stochastic analysis. *Journal of Vision, 12*(12), 1-17. doi: 10.1167/12.12.5

Walker, R., & McSorley, E. (2006). The parallel programming of voluntary and reflexive saccades. *Vision Research, 46*(13), 2082-2093. doi: 10.1016/j.visres.2005.12.009

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.

Walls, G. L. (1962). The evolutionary history of eye movements. *Vision Research, 2*(1–4), 69-80. doi: 10.1016/0042-6989(62)90064-0

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. doi: 10.1152/jn.01199.2004

White, B. J., Theeuwes, J., & Munoz, D. P. (2011). Interaction between visual- and goal-related neuronal signals on the trajectories of saccadic eye movements. *Journal of Cognitive Neuroscience, 24*(3), 707-717. doi: 10.1162/jocn_a_00162

Zhang, M. R., Red, S. D., Lin, A. H., Patel, S. S., & Sereno, A. B. (2013). Evidence of cognitive dysfunction after soccer playing with ball heading using a novel tablet-based approach. *Plos One, 8*(2). doi: 10.1371/journal.pone.0057364

Figures

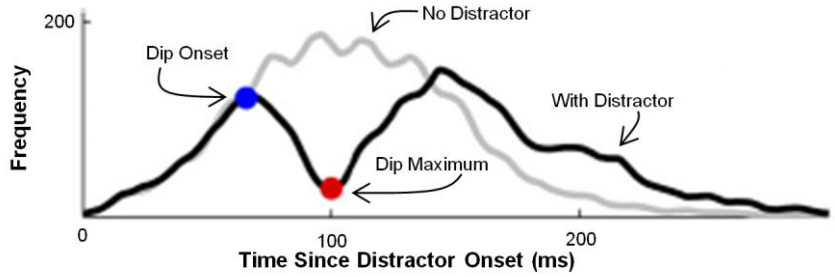

Figure 1. The saccadic inhibition effect in a single observer reproduced from Bompas and Sumner (2011, Fig 4). A distractor stimulus is briefly flashed before a saccade is executed to a suddenly appearing target. Plotting the time between distractor flash and saccade initiation reveals a characteristic dip in the distribution of saccade initiation times (black line). This dip reveals that distractors delay saccades that would otherwise have occurred around 70-150ms later (distributions were taken with a bin size of 4ms, lightly smoothed using a Gaussian kernel with a 5ms window and 1ms SD, and interpolated to 1ms precision).

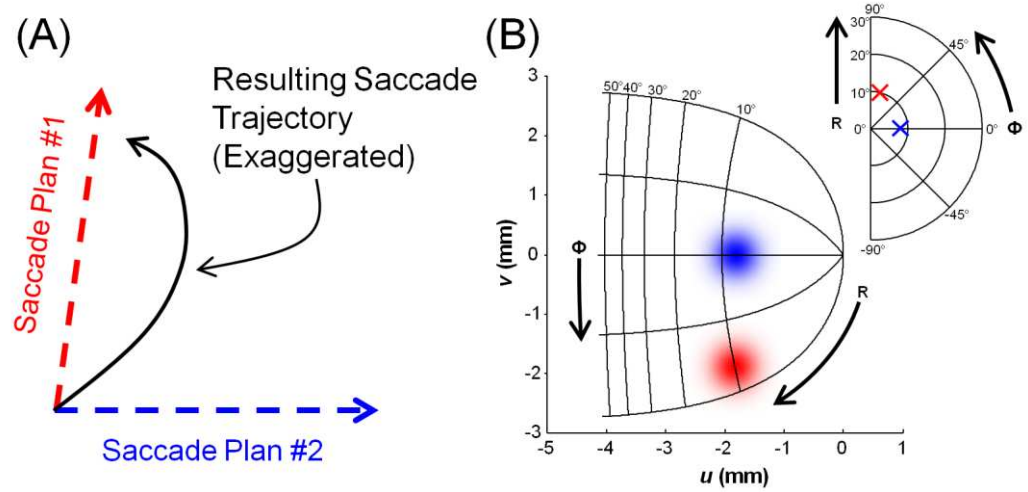

Figure 2. ***A*****,** The parallel planning of an oblique and a horizontal saccade can result in the execution of a single, curved saccade that deviates toward the competing end point. This is thought to occur due to competing sites of activity in the superior colliculus, shown in panel ***B*****.** Sites of activity in the superior colliculus are illustrated for the horizontal saccade of 8°, and the oblique saccade of 10° (insert shows the oblique saccade [red] and horizontal saccade [blue] endpoints in retinal co-ordinates; formulas to convert retinal co-ordinates to SC locations taken from Marino, Rodgers, Levy, & Munoz, 2008).

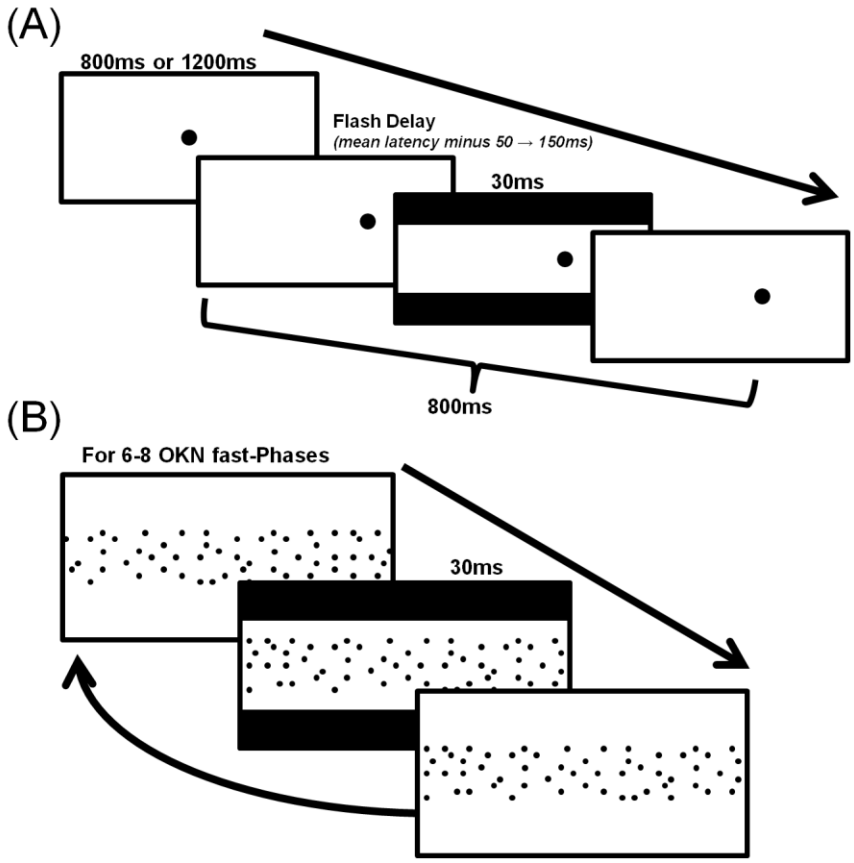

Figure 3. Illustrated procedure for testing 'saccadic inhibition' for standard targeting saccades (A) and OKN fast-phases (B). ***A***, Participants made a saccade when the dot stepped left or right, while on 50% of trials irrelevant bars (black in illustration) flashed before the saccade was made (Reingold & Stampe, 2002). ***B***, OKN was induced by passively viewed random dots moving left or right while irrelevant bars flashed intermittently in order to assess their effect on OKN fast-phases.

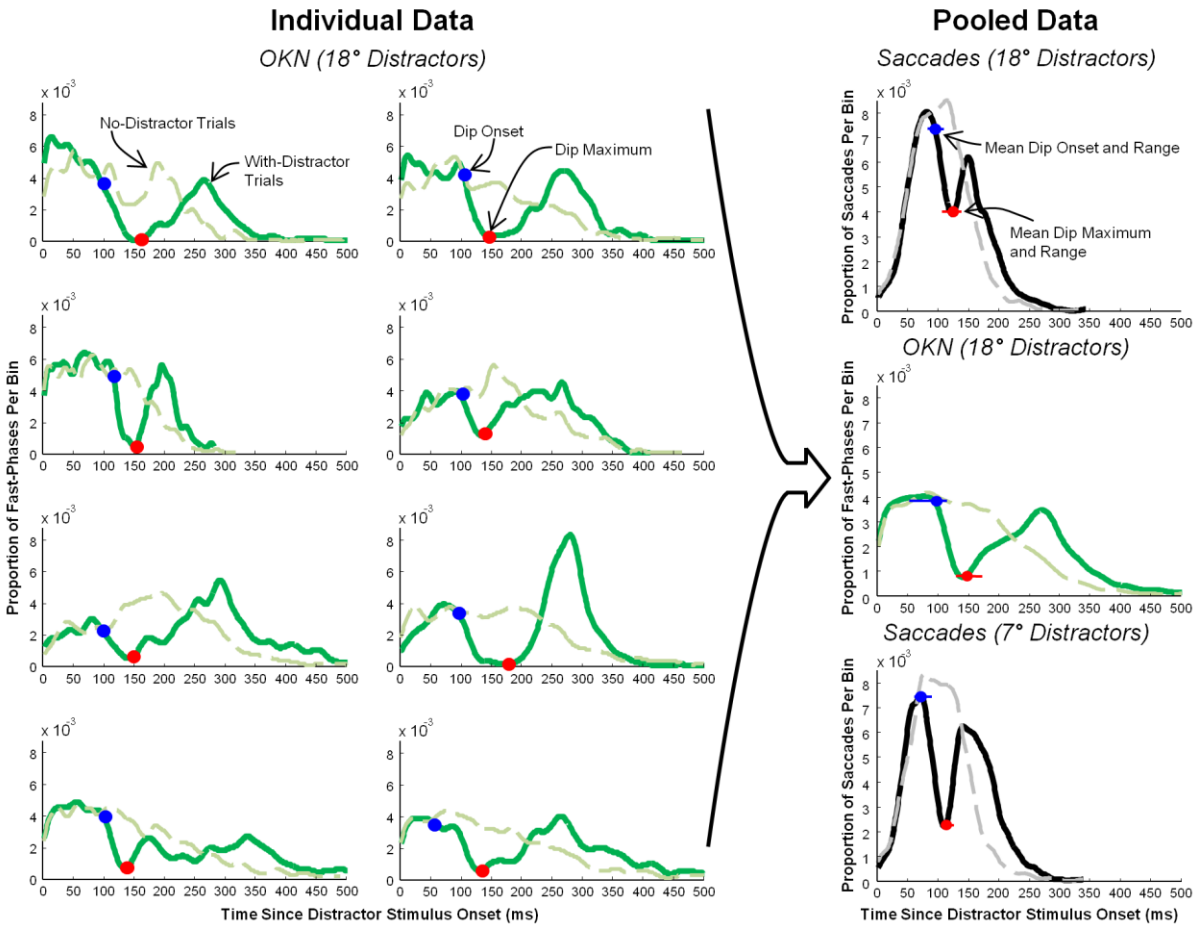

Figure 4. Individual data (left-hand panels) of OKN fast-phase latencies with respect to distractor onset for trials with (solid) and without (dashed) flashed distractor stimuli (see analysis section of Methods for how no-distractor distributions are created). The saccadic inhibition effect occurs for OKN fast-phases in all participants. Pooled data distributions (right-hand panels) reveal that a dip in the latency distribution occurs for OKN fast-phases (green) just as it does for targeting saccades. Blue circles represent the mean dip onset, and the horizontal blue error-bars represent the numerical range of dip onsets across participants. Red circles and error-bars represent the mean dip maximum, and the range of dip maxima respectively.

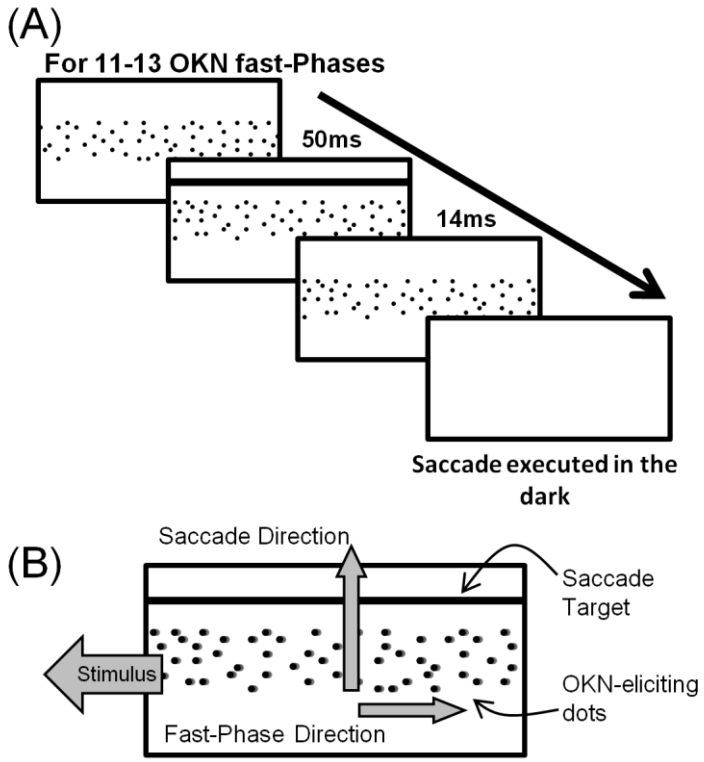

Figure 5. Illustration of method for eliciting curvature from an OKN fast-phase. ***A*,** OKN was induced by a horizontally moving band of random dots. A saccade target line was presented above or below these dots following 11-13 fast phases. The participant was simply required to lift or lower their gaze to the vertical location of the line. ***B*,** At the time of the saccade we hypothesise there could be two motor commands programmed in parallel, the targeting saccade (vertical component) and an OKN fast-phase (horizontal component).

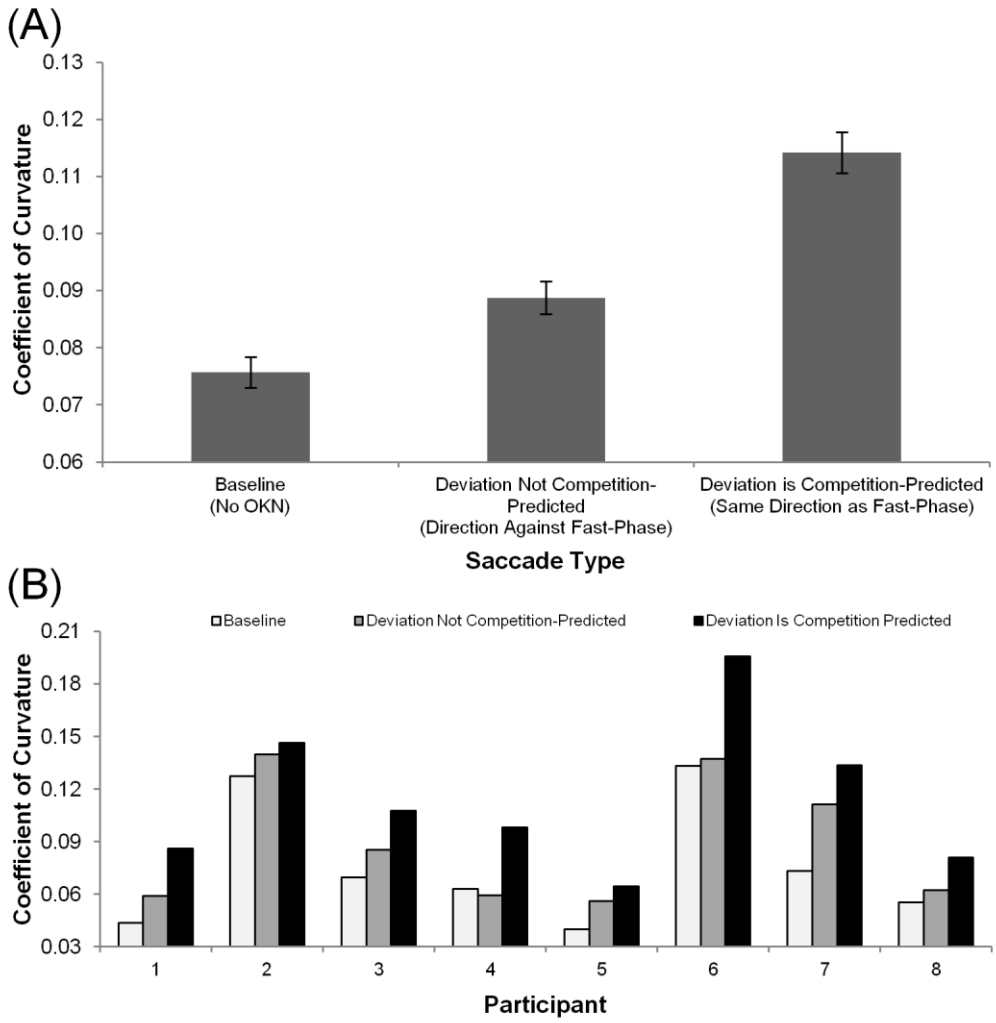

Figure 6. ***A,*** Competition-predicted saccades that deviate in the direction of the OKN fast-phase (rightmost bar) had significantly larger trajectory deviations than those deviating against the fast-phase, which were not competition-predicted (middle bar); or baseline saccades that are initiated from fixation (leftmost bar). Non-competition-predicted saccades that do not curve in the direction of the fast-phase (middle) show a smaller, yet significant increase in trajectory deviations than baseline. Left and rightward curvature is combined for each condition. Error bars show ± 1 x standard error with variance attributed to individual differences partialled out in line with Cousineau's (2005) method. ***B*****,** Individual data reveals that all participants show the largest deviations for those saccades that curve in the competition-predicted direction (black bars).

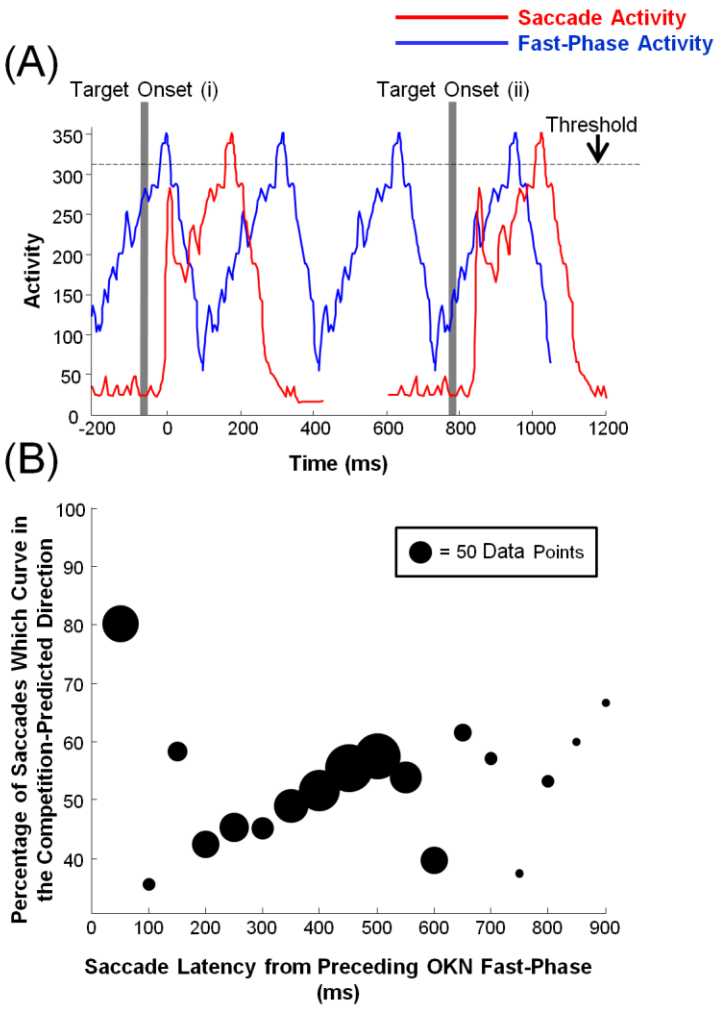

Figure 7. ***A*****,** Hypothetical rise to threshold for build-up neurones in SC for both OKN fast-phases (blue line) and targeting saccades (red line) (build-up activity constructed from actual neurophysiological SC recordings by Munoz & Wurtz, 1995). Target onset (i) results in a targeting saccade activity that rises when there is relatively little fast-phase-related activity; however target onset (ii) results in targeting saccade activity that rises when there is concurrent fast-phase activity. ***B*****,** shows that for targeting saccades made in temporal proximity to a fast-phase, the majority deviate in the direction of the fast-phase. There is a possibility that the proportion of saccades deviating in the direction of fast-phases rises once again for longer delays where an imminent fast phase is likely.