Toward a model of microsaccade generation: The case of microsaccadic inhibition

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Microsaccades are one component of the small eye movements that constitute fixation. Their implementation in the oculomotor system is unknown. To better understand the physiological and mechanistic processes underlying microsaccade generation, we studied microsaccadic inhibition, a transient drop of microsaccade rate, in response to irrelevant visual and auditory stimuli. Quantitative descriptions of the time course and strength of inhibition revealed a strong dependence of microsaccadic inhibition on stimulus characteristics. In Experiment 1, microsaccadic inhibition occurred sooner after auditory than after visual stimuli and after luminance-contrast than after color-contrast visual stimuli. Moreover, microsaccade amplitude strongly decreased during microsaccadic inhibition. In Experiment 2, the latency of microsaccadic inhibition increased with decreasing luminance contrast. We develop a conceptual model of microsaccade generation in which microsaccades result from fixation-related activity in a motor map coding for both fixation and saccades. In this map, fixation is represented at the central site. Saccades are generated by activity in the periphery, their amplitude increasing with eccentricity. The activity at the central, fixation-related site of the map predicts the rate of microsaccades as well as their amplitude and direction distributions. This model represents a framework for understanding the dynamics of microsaccade behavior in a broad range of tasks.

Keywords: fixational eye movements, oculomotor control, superior colliculus, saccadic inhibition

Citation: Rolfs, M., Kliegl, R., & Engbert, R. (2008). Toward a model of microsaccade generation: The case of microsaccadic inhibition. *Journal of Vision*, 8(11):5, 1–23, http://journalofvision.org/8/11/5/, doi:10.1167/8.11.5.

Introduction

The visual system is equipped with a toolbox of eye movements to facilitate the acquisition of information. Most notably, saccades (rapid eye movements) bring areas of interest onto the fovea, allowing for high-resolution visual analysis. Fixations of the eyes create the basis of visual perception and they consist of dynamically rich miniature eye movements (Engbert, 2006; Martinez-Conde, 2006): The eyes drift slowly and microsaccades, i.e., small jumps in gaze position, frequently occur (e.g., Ratliff & Riggs, 1950). Since saccades and fixations are considered mutually exclusive behaviors, most current models of saccade generation assume a strong inhibitory interplay of the processes controlling saccades and fixations (e.g., Findlay & Walker, 1999; Munoz & Fecteau, 2002; Trappenberg, Dorris, Munoz, & Klein, 2001). Thus, paradoxically, microsaccades occur during visual fixations, but they should be mutually exclusive with them, if they are viewed as saccades. Are microsaccades inconsistent with fixation or is their occurrence an integral part of it? To approach this question, we

(1) review the literature on likely physiological correlates of microsaccade generation, (2) present two experiments, closely investigating microsaccadic inhibition, a hallmark of microsaccadic behavior, and (3) propose a conceptual model of microsaccade generation based on the insights gained.

On the origin of microsaccades

Although the consequences of microsaccades, e.g., for perception and fixation stability, have been studied extensively (see Martinez-Conde, Macknick, & Hubel, 2004, for a review), little is known about their implementation in the oculomotor system. It is clear, however, that despite their different amplitudes and traditional subdivision into different categories of eye movements, microsaccades and large-scale saccades share a wide range of characteristics. First, both are typically binocular eye movements with almost identical amplitudes and directions in both eyes (Ditchburn & Ginsborg, 1953; Krauskopf, Cornsweet, & Riggs, 1960; Lord, 1951). Second, both fall on the main sequence (Zuber, Stark, &

Cook, 1965), i.e., the relationship between peak velocity and amplitude in these movements follows a linear relationship. Third, both result in a strong elevation of the visual perceptual threshold covering some time around the movement (saccadic suppression; Beeler, 1967; Latour, 1962; Volkmann, 1962; Volkmann, Schick, & Riggs, 1968; Zuber, Crider, & Stark, 1964; Zuber & Stark, 1966). Fourth, it was argued that inter-saccadic intervals in reading have similar distributions as inter-microsaccadic intervals during simple fixation of a letter (Cunitz & Steinman, 1969). Fifth, microsaccades are subject to voluntary control. Their rate can be reduced intentionally (Fiorentini & Ercoles, 1966; Steinman, Cunitz, Timberlake, & Herman, 1967) and very few are produced in high-acuity observational (Bridgeman & Palca, 1980) and finely guided visuomotor tasks (Winterson & Collewijn, 1976). Some subjects may even generate saccades as small as microsaccades voluntarily (Haddad & Steinman, 1973). Sixth, there is a strong relationship between spatial attention and the generation of saccades. Saccades are virtually always preceded by shifts of covert attention (e.g., Deubel & Schneider, 1996; Kowler, Anderson, Dosher, & Blaser, 1995) and both covert attention and saccades have strongly overlapping neurophysiological foundations (e.g., Corbetta et al., 1998; Kustov & Robinson, 1996). A pronounced correlation was also found for covert attention and microsaccades (Engbert & Kliegl, 2003b; Galfano, Betta, & Turatto, 2004; Hafed & Clark, 2002; Laubrock, Engbert, & Kliegl, 2005; Laubrock, Engbert, Rolfs, & Kliegl, 2007; Rolfs, Engbert, & Kliegl, 2004, 2005).

These findings are consistent with the notion that microsaccades and saccades are the product of the same machinery implementing the generation of high-velocity eye movements. However, although repeatedly proposed (Engbert, 2006; Gandhi & Keller, 1999; Munoz, Dorris, Paré, & Everling, 2000; Steinman, Haddad, Skavenski, & Wyman, 1973; Zuber et al., 1965), this hypothesis has rarely been tested explicitly by the means of neurophysiology (exceptions are Van Gisbergen, Robinson, & Gielen, 1981, and Van Gisbergen & Robinson, 1977). Rolfs, Laubrock, and Kliegl (2006), in turn, recently reported behavioral evidence for an interaction of microsaccade and saccade generation. In a delayed response task, they demonstrated (1) that the rate of microsaccades strongly decreases in expectation of the signal to launch a saccade and (2) that the occurrence of microsaccades up to several hundred milliseconds before the go signal resulted in a strong increase in saccade latency.

Because the implementation of normal saccades is comparatively well understood, we may derive predictions about the neural correlates of microsaccades in the oculomotor control system. The generation of saccades relies on a network of different brain areas, involved in the generation of reflexive and voluntary oculomotor behavior (see Munoz & Everling, 2004, for an overview). A central node in that network is the superior colliculus (SC), a layered structure in the dorsal mesencephalon engaged in

the programming and execution of saccadic eye movements (e.g., Munoz et al., 2000). The intermediate and deeper layers of the SC contain motor-related cells the activity of which is strongly correlated with the generation of saccades and visual fixation. These cells constitute a topographically organized motor map coding for saccades to the contralateral visual hemifield. In this map, saccade amplitudes are continuously represented, decreasing from the caudal to the rostral pole of the SC (Robinson, 1972). Neurons distributed throughout the map exhibit an increasing discharge rate prior to and during saccades directed into their response field (Munoz & Wurtz, 1995a; Sparks, Holland, & Guthrie, 1976; Wurtz & Goldberg, 1972). These cells have been labeled saccade neurons (SN). Most cells in the rostral pole, underneath visually driven SC cells representing the fovea, tonically discharge during fixation and pause or decrease firing during most saccades (Munoz & Guitton, 1991; Munoz & Wurtz, 1993a). These cells have been labeled fixation neurons (FN). Thus, FN and SN are active in an antagonistic fashion, a characteristic probably shaped by local inhibitory connections between neurons in the SC motor map as suggested by anatomical (Behan & Kime, 1996; Mize, Jeon, Hamada, & Spencer, 1991) and neurophysiological studies (McIllwain, 1982; Meredith & Ramoa, 1998; Munoz & Istvan, 1998; Olivier, Dorris, & Munoz, 1999).

However, recent evidence suggests that a distinction between FN and SN is misleading for at least three reasons. First, the interactions between FN and SN are the same as between SN and SN elsewhere in the motor map of the SC (Basso & Wurtz, 1997; Munoz & Istvan, 1998). Second, desired gaze position rather than fixed saccade vectors appears to be represented in the motor map of the SC (Bergeron & Guitton, 2000, 2002; Bergeron, Matsuo, & Guitton, 2003; Choi & Guitton, 2006). Finally and in agreement with this notion, FN and SN create a continuum with similar discharge characteristics for gaze-position errors of different amplitudes rather than representing two distinct types of neurons (Bergeron & Guitton, 2002; Krauzlis, Basso, & Wurtz, 1997). Accordingly, Krauzlis et al. (1997) concluded that "there are no fundamental differences between 'buildup cells' in the caudal SC and 'fixation cells' in the rostral SC; both are tuned for particular, albeit different, amplitudes of motor error" (p. 1695).

With the body of evidence provided, we would like to make a case for the hypothesis that microsaccades, occurring involuntary during attempted fixation, are generated by activity in the rostral pole of the SC (see also Gandhi & Keller, 1999; Munoz et al., 2000). Although direct neurophysiological data concerning this hypothesis is not yet available, there are hints pointing to an involvement of the rostral SC in microsaccade generation. First, electrical stimulation of cells in the very rostral pole in the intermediate layers of the monkey SC elicited small saccades with amplitudes well below 1° (Basso, Krauzlis, & Wurtz, 2000; Gandhi & Keller, 1999;

Robinson, 1972). Second, several authors reported that cells in the rostral SC of monkeys, i.e., FN, did not decrease their discharge rate for small-amplitude contraversive saccades (Anderson, Keller, Gandhi, & Das, 1998; Krauzlis, 2003; Krauzlis et al., 1997; Krauzlis, Basso, & Wurtz, 2000; Munoz & Wurtz, 1993a, 1995b). Third, the firing rates of neurons in the rostral SC increased after small displacements of a foveal fixation target (Krauzlis et al., 1997), indicating that even so-called FN encode a gaze-position error. Finally, microsaccades and large saccades are generated by equivalent signals in the saccadic burst generator (Van Gisbergen et al., 1981; see also Van Gisbergen & Robinson, 1977, and Yamazaki, 1968), the final stage of oculomotor processing, which receives the bulk of its input from the SC motor map.

Microsaccadic inhibition

If microsaccade generation is related to activity in the rostral SC, microsaccadic behavior should be sensitive to the same stimulus characteristics as the neuronal activity at the level of the SC motor map. To test this hypothesis, we investigated one of the most robust findings in microsaccade research: microsaccadic inhibition. It was first reported by Engbert and Kliegl (2003b) as the initial part of the microsaccade-rate signature: Shortly after the presentation of a visuospatial, attentional cue, microsaccade rate abruptly dropped to a minimum (microsaccadic inhibition) before showing an enhancement period and a final resettlement at the initial baseline level. The allocation of visuospatial attention is not even necessary to elicit the effect. It was observed for simple display changes (Engbert & Kliegl, 2003b), and even in the absence of visual events, using auditory cues (Rolfs et al., 2005). A very similar effect in response to irrelevant stimuli was reported for large saccades (saccadic inhibition) in a broad range of eye-movement tasks including simple saccade paradigms (Reingold & Stampe, 2002), reading (Reingold & Stampe, 1999, 2000, 2003, 2004; Stampe & Reingold, 2002), visual search (Reingold & Stampe, 1999, 2000, 2004; Stampe & Reingold, 2002), and picture viewing (Graupner, Velichkovsky, Pannasch, & Marx, 2007; Pannasch, Dornhoefer, Unema, & Velichkovsky, 2001).

Most previous studies that reported microsaccadic inhibition remained at a descriptive level. There are no experiments with systematic manipulations of stimulus characteristics to examine the stereotypical effect in detail. Only recently, Engbert (2006) examined the time line of this effect, thereby constraining the number of physiological systems involved in its generation. Compiling a number of studies that reported the microsaccade-rate signature, he concluded that the robust inhibitory part of the signature could only be explained in terms of a very fast subcortical processing circuit, probably involving the direct, retinotectal pathway from the retina to the SC. The enhancement phase, in contrast, which has been found to

be more variable across experiments, appears to be modulated by higher cognitive processes. As shown recently, it may even completely fail to appear, resulting in prolonged inhibition (Valsecchi, Betta, & Turatto, 2007; Valsecchi & Turatto, 2007).

Thus, for microsaccadic inhibition, it has been proposed that the SC assists its implementation (Engbert, 2006; Laubrock et al., 2005; Rolfs et al., 2005). Specifically, Engbert (2006) suggested that a stimulus-related signal results in a transient increase of the mean-field activation in the motor map of the SC. As a result of global inhibition within this map, activity at the rostral pole decreases. As microsaccades are hypothesized to be the result of activity in the rostral pole of the SC, their rate drops off. The present work follows up on these proposals, aiming to improve our understanding of microsaccade generation in the oculomotor system.

In two experiments, we triggered microsaccadic inhibition by presenting uninformative stimuli in the course of a demanding visual discrimination task. A set of measures of inhibition provides a quantitative description of the effect. Two main experimental factors were manipulated, the modality of the irrelevant stimulus (auditory or visual; Experiment 1) and, for visual onsets, their luminance contrast to the background (Experiments 1 and 2). Based on our findings, we derive a conceptual, physiologically inspired model of microsaccade generation. Microsaccades are hypothesized to be the result of fixation-related activity in a map coding for both fixation and saccades. This model provides a consistent theoretical framework for the explanation of the dynamics of the rate and direction of microsaccades that were reported in a variety of tasks. In addition, it generates predictions for the behavior of microsaccades in response to cognitive and sensory input, the interactions of microsaccades and subsequent saccades, and the dynamics of physiological processes in the oculomotor system.

Experiment 1: Luminance vs. color vs. auditory stimuli

Neuronal response latencies in the SC are known to depend considerably on stimulus characteristics. Experiment 1 examined the sensitivity of microsaccadic inhibition to qualitatively different stimuli. First, as revealed by single-cell studies in monkeys and cats, the latency with which auditory input can modulate activity in the SC motor map is much lower than that of visual stimuli, even when visual input directly impinges on the SC via the retinotectal pathway (Jay & Sparks, 1984, 1987; Stein & Meredith, 1993). Second, it has been shown that the retinotectal pathway is "blind" to stimuli that are equiluminant to the background (Schiller & Malpeli, 1977). Therefore, the initial visual response of the

intermediate and deeper layers of the SC reflects luminance differences only, while activity selecting color-defined targets is delayed, probably due to a relay via the extrastriate visual cortex (McPeek & Keller, 2002; Ottes, Van Gisbergen, & Eggermont, 1987).

Thus, if the SC constitutes a major neural substrate of microsaccade generation, microsaccadic inhibition was predicted (1) to occur faster after auditory stimuli than after visual stimuli and (2) to be delayed in response to color-contrast as compared to luminance-contrast stimuli. Experiment 1 tested these predictions.

Methods

Participants

Data were collected in two periods. In a first period, 40 undergraduate and high-school students (27 female) performed one session each. In a second period, 20 undergraduate and high-school students (12 female) completed two sessions each (1 to 13 days apart, 4.6 days on average). Subjects were paid €6 per session or received study credit for their participation. They were 16 to 27 years old (18.9 years on average), had normal or corrected-to-normal vision, reported normal hearing, and were in good health. All experiments were performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and individuals gave their informed consent prior to their participation in the study.

Experimental setup and eye-movement recordings

Participants were seated in a silent and dimly lit room with the head positioned on a chin rest, 50 cm in front of a computer screen. Eye-movement data were recorded using an EyeLink-II system (SR Research, Osgoode, Ontario, Canada) with a sampling rate of 500 Hz and a noiselimited instrument spatial resolution better than 0.01°. Visual stimuli were presented on a 19-inch EYE-Q 650 Monitor (1024 \times 768 resolution or 40° by 30° of visual angle; frame rate 120 Hz) using gray background color. Auditory stimuli were played via Sennheiser HD 520 II headphones. An Apple Power Macintosh G4 computer served to control the experiment; manual responses were recorded via its standard keyboard. The experimental software controlling stimulus display and response collection was implemented in MATLAB (MathWorks, Natick, Massachusetts, USA), using the Psychophysics (Brainard, 1997; Pelli, 1997) and EyeLink (Cornelissen, Peters, & Palmer, 2002) toolboxes.

Procedure

Participants were divided into two groups, each containing a total 30 individuals (20 from the first and 10 from the second data-collection epoch). Both groups performed the same visual discrimination task but were

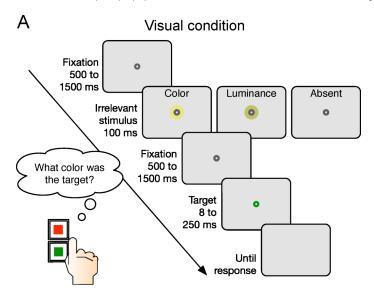
presented with either visual- or auditory-irrelevant stimuli. Aside from the irrelevant stimuli, the procedure was identical for both the visual and the auditory group.

After a key training, linking "red" to the up- and "green" to the down-arrow key, participants performed eight randomly ordered practice trials, introducing the task, and 240 test trials. Before the first and after every 15th test trial, the eye tracker was calibrated (standard 9-point grid) and calibration was validated. On every fifth trial, a drift correction was carried out. Before each test trial, the fixation spot was displayed at the center of the computer screen. Participants began fixating and correct fixation was checked. If gaze position was not detected in a region four times as large as the fixation spot, the experimenter carried out a drift correction and re-started the trial. If the eyes were still not detected within the critical area, the calibration was repeated.

Figures 1A and 1B illustrate the trial sequences used in the visual and auditory conditions, respectively. Participants were required to look at the fixation spot during the whole trial while they performed a visual discrimination task. That is, after a variable period, the dark gray fixation spot shortly changed its color to green or red (equiluminant with the previous gray) and disappeared subsequently. Participants made speeded responses to this target stimulus, indicating which color was displayed. In 120 of 240 trials, task-irrelevant stimuli (yellow rings around the fixation spot in the visual condition; a burst sound in the auditory condition) were interspersed during the fixation period, and participants were told to simply ignore them. A within-subject factor luminance was nested in the visual condition: In 60 trials, the yellow ring was equiluminant to the background and in 60 trials it had luminance contrast (henceforth, colorcontrast and luminance-contrast conditions, respectively). The two types of target stimuli (red or green) replacing the fixation stimulus after that period had equal probability across the 240 trials and within each condition. All kinds of trials were presented in a random order.

The fixation times prior to and following irrelevant stimuli ranged from 500 to 1500 ms of fixation. Irrelevant visual stimuli were presented for 100 ms; irrelevant tones were presented for 82 ms. Thus, fixation periods had a total duration of 1100 to 3100 ms and 1082 to 3082 ms in the visual and auditory conditions, respectively.

Target presentation times adapted to the participants' performance. We aimed to achieve a relatively constant level of about 75% correct responses using the weighted up-down method proposed by Kaernbach (1991): In the first trial of a session, the target stimulus was presented for 12 monitor frames (100 ms). After each correct response (e.g., up-arrow key after a red target), presentation time was reduced by 1 frame (minimum: 1 frame). After each incorrect response, it was increased by 3 frames (maximum: 30 frames). An average performance of 80% correct responses was produced with mean target presentation times of about 4 and 3 frames in the visual and auditory conditions, respectively. The deviation from the



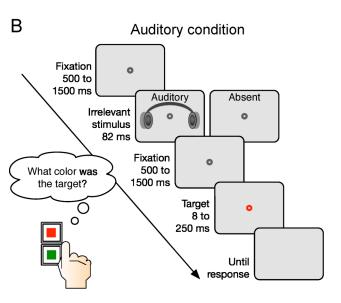


Figure 1. Illustration of trial sequences and participants' visual discrimination task in (A) the visual and (B) the auditory conditions of Experiment 1.

expected performance level of 75% was due to the limitation of stimulus presentation times to units of frames, resulting in ceiling effects of performance, once the minimum presentation time (8.3 ms) was reached.

Incorrect responses released an error feedback; correct responses directly initiated the next trial. After every eighth trial, participants received a performance feedback, to induce a strong focus on the visual discrimination task; it included the participant's evolution of mean latencies of correct responses averaged over blocks of eight trials.

Stimuli

The fixation spot was a ring with a diameter of 0.8° of visual angle in dark gray color (CIE 1931: $Y = 12.2 \text{ cd/m}^2$,

x = 0.268, y = 0.288) and an inset with a diameter of 0.1° , displayed on a light-gray colored background ($Y = 31.8 \text{ cd/m}^2$, x = 0.272, y = 0.290). Target stimuli were identical to the fixation spot with respect to form and luminance; they only differed in color (green: Y = 12.2, x = 0.275, y = 0.375; red: Y = 11.9, x = 0.328, y = 0.301). Visual-irrelevant stimuli were yellow rings with a diameter of 1.6° (luminance-contrast condition: $Y = 18.4 \text{ cd/m}^2$, x = 0.316, y = 0.369; color-contrast condition: $Y = 32.0 \text{ cd/m}^2$, x = 0.319, y = 0.371) transiently surrounding the fixation spot. Auditory-irrelevant stimuli were 70 dBA approximated white noise sounds (background noise: about 35 dBA). The error feedback was a central circle of the actual target color (diameter of 1.6°) surrounding the fixation spot for 300 ms.

Before an experimental session, equiluminant colors were determined on an individual basis using a flicker-fusion method. Participants were instructed to minimize the flickering of two colored spots alternating with 20 Hz by adjusting the luminance of one color. This procedure was performed for the different pairs of colors employed: In both groups, the colors of the target stimuli (green and red) were adjusted to the dark gray of the fixation spot. In addition, in the visual condition, the color of the irrelevant stimulus was adjusted to the background.

Data preparation

Microsaccades were detected using an improved version (Engbert, 2006) of an algorithm proposed by Engbert and Kliegl (2003b). Velocities were computed from successive eye positions recorded in a trial. Microsaccades were detected in 2D velocity space using thresholds for peak velocity (6 SD) and minimum duration (6 ms, or three data samples). We considered only binocular microsaccades, that is, microsaccades detected in both eyes with temporal overlap.

Trials including saccades larger than 1° of visual angle were discarded. Some trials had to be excluded due to data loss during eye-movement recording. To be included in the analyses, a participant had to contribute at least 60 trials with and 60 trials without an irrelevant stimulus. In the visual condition at least 30 trials with color-contrast and 30 trials with luminance-contrast-irrelevant stimuli had to meet the given criteria. In the visual condition, 28 participants contributed 142 to 463 trials to the final data analyses, resulting in a total of 6703 trials (out of 9120 or 73.5%) in which 25554 microsaccades were detected. In the auditory condition, 23 participants contributed 140 to 455 trials to the final data analyses, resulting in a total of 5020 trials (out of 7440 or 67.5%) in which 22083 microsaccades were detected.

Data analysis

Confidence intervals were computed using a simple bootstrapping technique (Efron & Tibshirani, 1993): From

an original sample of N values, 1000 bootstrap samples were generated, each by selecting N values of the original sample (with replacement). We computed the 1.96-fold of the standard deviation of the means of these 1000 bootstrap samples to generate 95% confidence intervals of the mean of the original sample. For within-subject statistical inferences, we removed between-subject variance beforehand, using the procedure proposed by Cousineau (2005).

To compute microsaccade-rate evolutions, we applied a filter, which has been adopted from analyses of neural firing rates (Dayan & Abbott, 2001; see also Engbert, 2006). For each participant, microsaccade events were collapsed across all trials that were contributed in a certain condition (pooled from all sessions performed). Microsaccade onsets t_i were aligned to the onset of the irrelevant stimulus. The series of microsaccadic events of i = 1,2,3...N at times t_i is formalized by

$$\rho(t) = \sum_{i=1}^{N} \delta(t - t_i), \tag{1}$$

where δ denotes Dirac's δ function. Microsaccade rate r(t) was then determined by temporal averaging, i.e.,

$$r(t) = \int_{0}^{\infty} \omega(\tau)\rho(t-\tau)d\tau, \tag{2}$$

applying the window function

$$\omega(\tau) = \alpha^2 \tau \exp(-\alpha \tau). \tag{3}$$

This causal filter $\omega(\tau)$ results in a systematic delay of the computed temporal evolution of microsaccade rate. In detail, r(t) is only influenced by past microsaccadic events and the delay with maximum impact on r(t) strongly depends on the choice of the decay parameter α . To prevent these temporal biases, we shifted the whole function r(t) to finally get

$$\hat{r}(t) = r\left(t + \frac{1}{\alpha}\right),\tag{4}$$

ensuring that the maximum impact on microsaccade rate $\hat{r}(t)$ comes from microsaccades observed at time t. In our analyses, t had a temporal resolution of 1 ms. To transform the rate to units of Hz, thus, $\hat{r}(t)$ was multiplied by 1000 and divided by the number of contributing trials. Finally, to generate mean microsaccade-rate evolution, individual rates were averaged across participants. The decay parameter was set to $\alpha = 1/20$ ms.

For a detailed description of microsaccade-rate evolution and for the determination of experimental effects, we

standardized microsaccade rates in a first step. To this end, we computed a baseline rate by averaging the rate across the last 50 ms before the onset of the irrelevant stimulus. The microsaccade-rate evolution was then normalized to a baseline of 1 Hz by dividing the rate at each point in time by the baseline rate (expected microsaccade rate).

Subsequently, we computed various measures of inhibition (see leftmost panel of Figure 3 for an illustration), most of which were introduced in the work of Reingold and Stampe (2004) on saccadic inhibition. First, the minimum saccadic frequency was located in a time window from 0 to 400 ms. Second, the bottom of the dip was defined as the period at which microsaccade frequency was below the minimum plus 10% of the difference between 1 (the baseline) and the minimum frequency. The center of the bottom of the dip will be referred to as latency to maximum saccadic inhibition L_{max} . Third, the magnitude of inhibition μ (henceforth, magnitude) is 1 minus the frequency at L_{max} . Thus, higher values indicate stronger inhibition. Fourth, we computed the latency to 50% of maximum saccadic inhibition $L_{50\%}$, i.e., the time at which inhibition reached 50% of its magnitude. To this end, a period before L_{max} was determined during which inhibition was in between 1/3 and 2/3 of its magnitude. The center of this period was taken as $L_{50\%}$. To determine the point of return to 50% of magnitude $(L_{50\%r})$, the same was done for the period after $L_{50\%}$. Finally, the duration of inhibition δ (henceforth, duration) is the interval while the inhibition remained above 50% of its magnitude. The results of these analyses are illustrated in Figure 3.

In addition, we propose a new measure—the *shape of inhibition* σ (henceforth, *shape*). This measure is sensitive to the form of the dip of the frequency curve. It is visualized in Figure 3 as the dark portion of the shaded area. The shape is computed as the ratio of areas

$$\sigma = \frac{1}{0.5\mu\delta} \int_{t=L_{50\%}}^{L_{50\%r}} (1 - 0.5 \,\mu - f(t)) dt, \tag{5}$$

which translates into

$$\sigma = \frac{2}{\mu} - 1 - \int_{t=I_{soly}}^{L_{50\%r}} f(t) dt, \tag{6}$$

since $\delta = L_{50\%r} - L_{50\%}$. Thus, the shape has a range of $0 < \delta \le 1$ with higher values describing more bellied curves and lower values covering a range of rather pointed curves.

These measures of inhibition could not be estimated reliably for individual participants because, most of the time, individual microsaccade-frequency evolutions were very noisy. Therefore, we employed a bootstrap method to create a sample of stable frequency curves that is representative of the assumed underlying frequencyevolution population. This algorithm can be used to reliably estimate standard errors in this type of data (Efron & Tibshiriani, 1993). It requires thousand independent bootstrap samples, each consisting of N individual frequency evolutions drawn with replacement from the pool of N observed individual microsaccade-frequency evolutions. That is, in a given bootstrap sample, participants could be included 0 to N times, but over the whole set of replications, a participants' data were included approximately equally often. For each replication, a mean frequency evolution was computed, resulting in 1000 bootstrap frequency evolutions and corresponding measures of inhibition. Means and confidence intervals were computed from these 1000 replications. This procedure was conducted for all conditions under investigation: luminance contrast, color contrast, and auditory. The same 1000 bootstrap samples were used for the two visual conditions, effectively controlling for between-subject variability.

Results

Performance in the task

For the analyses of response times and error rates, we removed trials with response times shorter than 70 ms and longer than 1000 ms. A mixed-model ANOVA with stimulus presence (present vs. absent) and modality group (visual vs. auditory) as independent variables revealed an interaction of these two factors; F(1,49) = 23.84, p < 0.001. In addition, a main effect of stimulus presence was evident; F(1,49) = 15.51, p < 0.001. No main effect of modality was observed; F < 1. Post hoc contrasts revealed that stimulus presence affected response times in the visual modality only (visual: 472 vs. 509, t[27] = 5.43; p < 0.001; auditory: 504 vs. 506 ms, t[22] = 0.47; p = 0.64). On average, participants were 37 ms faster if an irrelevant visual stimulus was presented while waiting for the discrimination target. There was a marginal difference in the errors rates contingent on stimulus presence (F[1,49] =3.95, p = 0.052), but no effect of modality and no interaction of the two factors; Fs < 1.

Microsaccade rate

The dynamics of microsaccade rate $\hat{r}(t)$ in response to the irrelevant stimuli were determined along the lines described in the Methods section. The results are plotted in Figure 2. Both the visual and the auditory stimuli produced strong microsaccadic inhibition. Figure 2 also displays raster plots, in which each line shows a participant's microsaccade data from 30 randomly chosen trials (the number of trials was kept constant to facilitate

comparison); these plots clearly reveal that the effect is very stable across observers.

Figure 3 displays microsaccade-frequency evolutions, which were used to estimate various measures of inhibition using the procedure described in the Methods section. Table 1 displays means (and confidence intervals) of theses measures; mean differences (±95% confidence intervals) will be given in the text. Let us first consider the effects of luminance on microsaccadic inhibition. The latency of inhibition was strongly modulated by the

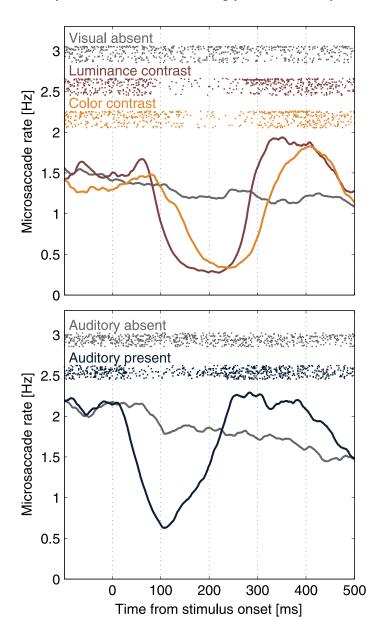


Figure 2. Microsaccade rate in the visual and auditory conditions. The line plots display microsaccade-rate evolution averaged across participants. The raster plots show corresponding individual microsaccade data from 30 trials per condition, randomly chosen for each participant. Each line represents one participant, each dot corresponds to a microsaccade observed at the corresponding point in time.

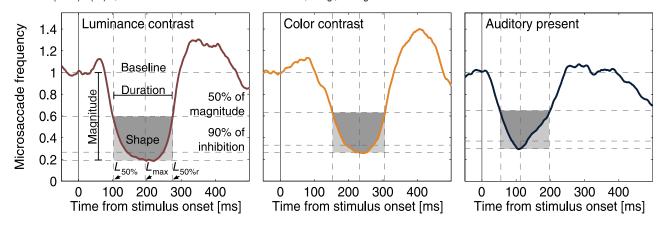


Figure 3. Microsaccadic inhibition in response to irrelevant stimuli in Experiment 1. Microsaccade frequency is a standardized representation of the microsaccade rate. Latencies ($L_{50\%}$ = latency to 50% of maximum inhibition; L_{max} = latency to maximum inhibition; $L_{50\%r}$ = latency to return to 50% of maximum inhibition), duration, magnitude, and shape of microsaccadic inhibition are illustrated in the first panel.

luminance of the irrelevant stimulus; luminance-contrast stimuli resulted in faster inhibition than color-contrast stimuli. This difference in latencies between the two conditions was 50 ± 21 ms as measured by $L_{50\%}$ and 37 ± 22 ms as measured by L_{\max} . In contrast, the strength of inhibition as measured by its duration, its magnitude, and its shape was not reliably affected by the luminance manipulation (confidence intervals of differences included zero).

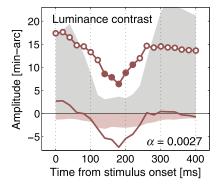
In the auditory condition, a very short latency of inhibition was observed as measured by $L_{50\%}$ (53 ms) and L_{max} (110 ms). Overall, the latency of inhibition was shorter in the auditory than in the visual conditions. $L_{50\%}$ differed by 49 \pm 14 and $L_{\rm max}$ differed by 87 \pm 16 ms comparing the auditory with the luminance-contrast condition; latency measures in the auditory condition differed even stronger from the color-contrast condition ($L_{50\%}$: 99 ± 22 ms; L_{max} : 124 ± 23 ms). In contrast, the strength of inhibition as measured by its duration and its magnitude did not differ significantly between the visual and auditory conditions; if anything, the duration was somewhat shorter in the auditory as compared to the luminance-contrast condition (34 \pm 35 ms). However, the shape was clearly more pointed in the auditory as compared to both the luminance-contrast (0.18 ± 0.10) and the color-contrast (0.12 ± 0.10) conditions. This finding suggests a higher sensitivity of this measure to the total amount of inhibition as compared to the magnitude and duration of inhibition, at least in the present set of conditions.

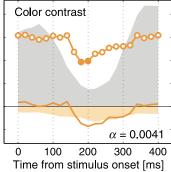
Microsaccade amplitude

Figure 4 illustrates the temporal evolution of microsaccade amplitudes (maximum of the distances between any two positions on a microsaccade's trajectory) locked to the onset of the irrelevant stimuli as well as corresponding statistical inference tests. Colored lines with circular markers show mean amplitudes, averaged across subjects, and were determined using a ±40 ms moving boxcar window. We determined significant deviations using a permutation method that we previously applied to microsaccade-direction evolutions (Rolfs et al., 2005). Surrogate amplitude evolutions were created, representing the null hypothesis that no amplitude modulation was induced by the presentation of the irrelevant stimulus. To this end, amplitudes of all microsaccades that a participant generated in the time window presented in Figure 4 were permuted. That is, the overall distribution of microsaccade amplitudes remained unchanged (as did the rate evolution), but any single amplitude that was measured was assigned to a randomly chosen microsaccadic event in the data set (without replacement). This was done for each participant and an average surrogate amplitude evolution

Condition	L _{50%} [ms]	L _{max} [ms]	Duration δ [ms]	Magnitude μ [proportion]	Shape σ [proportion]
Luminance contrast	102 (±11)	197 (±14)	176 (±22)	0.81 (±0.12)	0.78 (±0.08)
Color contrast	152 (±19)	234 (±21)	154 (±29)	0.75 (±0.09)	0.71 (±0.08)
Auditory present	53 (±9)	110 (±8)	142 (±27)	0.70 (±0.09)	0.59 (±0.05)

Table 1. Means ($\pm 95\%$ confidence intervals) of the various measures of inhibition, as estimated from the bootstrapping technique (see text for details) for Experiment 1. *Note*: $L_{50\%}$ = latency to 50% of maximum inhibition; L_{max} = latency to maximum inhibition.





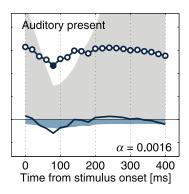


Figure 4. Analysis of microsaccade-amplitude evolution in response to irrelevant stimuli in Experiment 1. Colored lines with circular markers depict mean microsaccade amplitude in time windows of ± 40 ms. Gray background profiles illustrate mean microsaccade rates computed for the same time windows. Colored lines without markers show deviations of mean microsaccade amplitude from surrogate data that represent the null hypothesis that amplitudes was not modulated by the presentation of the irrelevant stimulus (areas in light colors give 95% confidence intervals plotted as deviations from 0; see text for details). Alpha levels, adjusted using the false-discovery-rate procedure (Benjamini & Hochberg, 1995), are given in each panel. Filled markers highlight significant decreases in mean microsaccade amplitude.

was computed as for the original data. This procedure was repeated 1000 times, resulting in 1000 surrogate amplitude evolutions. From these, means and 95% confidence intervals were computed. For each condition, Figure 4 shows these confidence intervals as filled areas (light colors) deviating from zero; zero represents the mean of the surrogate data. Deviations of the original microsaccade-amplitude evolution from the mean surrogate evolution are overlaid as colored lines (without markers). When this line leaves the confidence bands, strong deviations were observed. To compensate for multiple testing, the alpha level for inferential tests was adjusted using the false-discovery-rate procedure (Benjamini & Hochberg, 1995). Filled markers highlight significant deviations and alpha levels are given for each condition in the corresponding panels.

A strong decrease in mean amplitude was found for the two visual conditions. A similar effect, though weaker, was observed after auditory stimuli. The light gray background profiles in Figure 4 illustrate the rate of microsaccades in the time windows used for mean amplitude computation. It is evident that the decrease in microsaccade amplitude closely followed the time course of microsaccadic inhibition in each condition. Significant deviations in microsaccade amplitude were evident in time windows of strongest inhibition, that is, from 140 ± 40 to 220 ± 40 ms in the luminance-contrast, from 180 ± 40 to 200 ± 40 ms in the color-contrast, and at 80 ms in the auditory condition.

Discussion

We have argued that the latency of microsaccadic inhibition should be sensitive to the properties of the triggering stimulus. While luminance-contrast stimuli may exert influence on the SC motor-map directly via retinotectal connections, this pathway is blind to color contrast (Schiller & Malpeli, 1977). The delayed presence of a color-related signal in the SC (McPeek & Keller, 2002) was hypothesized to express itself in a delayed microsaccadic inhibition effect. Our data confirm this prediction. Color-contrast input delayed microsaccadic inhibition by 37 to 50 ms on average (depending on the measure) as compared to the luminance-contrast input.

In addition, we found much shorter latencies of microsaccadic inhibition after auditory as compared to visual stimuli. This is in agreement with physiological data showing that signals reach the SC motor map earlier on average when they originate from auditory rather than visual stimulation (Jay & Sparks, 1984, 1987; Stein & Meredith, 1993). As the intensity of the auditory stimuli was not systematically varied with respect to the flashes in the present experiment and no attempt was made to obtain subjectively matched stimulus intensities for the two modalities, a direct comparison of microsaccadic inhibition evoked by auditory- and visual-irrelevant stimuli may appear to be inconclusive. Note, however, that all stimuli were clearly above threshold and held constant across the experiment with respect to their physical properties. Therefore, we can still derive a strong argument based on published neuronal conduction delays: Auditory stimuli evoked microsaccadic inhibition after extremely short latencies (53 ms on average, as measured by $L_{50\%}$). This latency is too short to be produced by visual input because the lower physiological limit for an impact of visual input on oculomotor behavior is in the range of 60 to 70 ms (Reingold & Stampe, 2002). These estimates are based on a series of single-cell-recording studies in monkeys: First, for bright flashed stimuli, Rizzolatti, Buchtel, Camarda, and Scandolara (1980) reported visual latencies of 35 to 47 ms in the superficial layers of the SC. Second, transmission delays to the intermediate layers of the SC range from 5 to 10 ms (Lee, Helms, Augustine, & Hall, 1997). Finally, the time after which suprathreshold stimulation of the intermediate SC evokes a saccadic response is at least 20 ms (Robinson, 1972). While the final motor delay of 20 ms may be independent of stimulus modality, input delays are not. In contrast to visual input, sound-induced signals often reach the intermediate SC within 30 ms or less (Jay & Sparks, 1984, 1987). Thus, a latency of 50 ms (as observed in the present study) for microsaccadic inhibition is physiologically plausible in response to auditory stimuli, but according to current knowledge impossible after visual input. We conclude that the SC is a likely candidate for the implementation of microsaccadic inhibition. Our additional finding that microsaccade amplitude strongly decreased in the course of microsaccadic inhibition is predicted by a model that associates microsaccade generation with activity in the rostral part the SC motor map (see General discussion section).

Our results complement previous work that showed that the enhancement phase in microsaccade rate, which often follows microsaccadic inhibition, is delayed for color-contrast stimuli as compared to luminance-contrast stimuli (Valsecchi & Turatto, 2007). The study also provides quantitative support for our result that auditory attentional cues produce faster microsaccadic inhibition than visual cues in an earlier study (compare Figures 2 and 3 in Rolfs et al., 2005).

Experiment 2: Varying luminance contrast

Experiment 1 yielded large latency differences of microsaccadic inhibition for different stimulus qualities. Recent work has shown, however, that visually induced neuronal activity in the SC motor map also depends on the intensity of the stimulus. Specifically, Bell, Meredith, Van Opstal, and Munoz (2006) demonstrated that the latency of stimulus-induced neuronal discharges in the intermediate layers of the SC is a function of the stimulus' luminance contrast to the background. For high-contrast stimuli, neuronal response latencies were shorter than for low-contrast stimuli. Thus, Bell et al. (2006) established a correlation between the latency of neuronal activity in the SC and the latency of saccades generated in response to stimuli of different luminance contrast (see also Boch, Fischer, & Ramsperger, 1984; Kingstone & Klein, 1993; McPeek & Schiller, 1994; Reuter-Lorenz, Hughes, & Fendrich, 1991).

In Experiment 2, we tested whether the latency of microsaccadic inhibition also varies with luminance contrast. Shorter latencies were expected for high-contrast stimuli, longer latencies for low-contrast stimuli. We used the same task as in Experiment 1 and varied

stimulus luminance in three steps: high, medium, and low contrast

Methods

Participants

Twenty-seven undergraduate and high-school students (22 female) were paid €7 or received study credit for their participation in the experiment. They were 18 to 35 years old (22.5 years on average), had normal or corrected-to-normal vision, reported normal hearing, and were in good health.

Experimental setup and eye-movement recordings

Experimental setup and eye-movement recording was identical to that used in Experiment 1, except that visual stimuli were presented on a 22-inch iiyama HM204DT CRT (46° by 34° of visual angle).

Procedure

Participants performed the same visual discrimination task described in Experiment 1. In all 240 trials, task-irrelevant stimuli were interspersed during the fixation period, and participants were told to simply ignore them. High-contrast, medium-contrast, and low-contrast stimuli were presented in 80 trials each; their order was randomized across the experiment.

Stimuli

The stimuli used in Experiment 2 were identical to those used in the visual condition of Experiment 1, except for their colors and luminances. The fixation spot was dark gray (CIE 1931: $Y = 3.43 \text{ cd/m}^2$, x = 0.264, y = 0.294), displayed on a mid-gray colored background ($Y = 16.0 \text{ cd/m}^2$, x = 0.270, y = 0.293). Target stimuli were identical to the fixation spot with respect to luminance; they only differed in color (green: Y = 3.37, x = 0.272, y = 0.397; red: Y = 3.35, x = 0.323, y = 0.303). Visual-irrelevant stimuli were gray to white rings with a diameter of 1.6° (high-contrast condition: $Y = 65.3 \text{ cd/m}^2$, x = 0.273, y = 0.291; medium-contrast condition: $Y = 22.8 \text{ cd/m}^2$, x = 0.270, y = 0.292; low-contrast condition: $Y = 16.3 \text{ cd/m}^2$, x = 0.269, y = 0.292), transiently surrounding the fixation spot.

Data preparation and analysis

Data were prepared and analyzed as described for Experiment 1, except that a participant had to contribute at least 40 valid trials in each of the three conditions (high, medium, and low contrast) to be included in the analyses. Twenty-five participants contributed 131 to 236 trials to

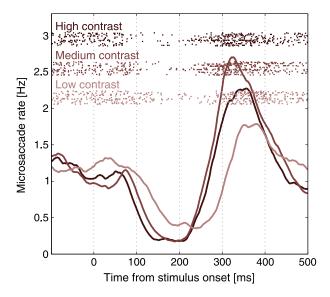


Figure 5. Microsaccade rate in the three conditions of Experiment 2. The line plots display microsaccade-rate evolution averaged across participants. The raster plots show corresponding individual microsaccade data from 30 trials per condition, randomly chosen for each participant. Each line represents one participant, each dot corresponds to a microsaccade observed at the corresponding point in time.

the final data analyses, resulting in a total of 5128 trials (out of 6000 or 85.5%) in which 15251 microsaccades were detected.

Results

Performance in the task

Response times did not differ between the three irrelevant-stimulus conditions (high contrast: 523 ± 7 ms; medium contrast: 524 ± 5 ms; low contrast: 528 ± 8 ms). The same was true for the errors rates (high contrast: 78.0 ± 1.4 ms; medium contrast: 79.0 ± 1.7 ms; low contrast: 76.8 ± 1.8 ms).

Microsaccade rate

In Figure 5, microsaccade rate $\hat{r}(t)$ is plotted as a function of time from the onset of the irrelevant stimulus. All stimuli produced strong microsaccadic inhibition. The

raster plots in the same figure demonstrate that the effect is very stable across observers.

Table 2 displays means (and confidence intervals) of the various measures of inhibition, computed as described for Experiment 1; mean differences will be given in the text. The latency of inhibition was strongly modulated by the luminance contrast of the irrelevant stimulus. The higher the contrast was, the shorter was the latency of microsaccadic inhibition. This difference in latencies between the high- and low-contrast conditions was 41 ± 21 ms as measured by $L_{50\%}$ and 44 \pm 32 ms as measured by L_{max} . The duration of inhibition was somewhat shorter in the medium-contrast than in the high-contrast condition $(27 \pm 22 \text{ ms})$, but only marginally so as compared to the high-contrast condition (30 \pm 33 ms); high- and lowcontrast conditions did not differ (3 \pm 26 ms). The magnitude of inhibition was significantly smaller in the low-contrast as compared to the medium-contrast condition (0.15 \pm 0.13), but only marginally so as compared to the high-contrast condition (0.14 \pm .16). Finally, differences in the shape of inhibition were negligible.

Microsaccade amplitude

Figure 6 shows the analysis of the temporal evolution of microsaccade amplitudes locked to the onset of the irrelevant stimulus in the three conditions. Plots and inferential statistics were computed as for Experiment 1 (Figure 4). A significant decrease (highlighted by filled markers) in the mean amplitude was found for the high-and medium-contrast conditions in the time windows from 140 ± 40 to 180 ± 40 ms, but not for the low-contrast condition. Light gray background profiles in Figure 6 illustrate the rate of microsaccades in the time windows used for mean amplitude computation. As in Experiment 1, significant decrease in microsaccade amplitude coincided with strongest microsaccadic inhibition.

Discussion

The latency of microsaccadic inhibition clearly depended on the luminance contrast of the triggering stimulus. High-contrast stimuli resulted in fast microsaccadic inhibition. The latency of inhibition was somewhat longer after medium-contrast stimuli. The longest

Condition	L _{50%} [ms]	L_{max} [ms]	Duration δ [ms]	Magnitude μ [proportion]	Shape δ [proportion]
High contrast Medium contrast Low contrast	97 (±13)	178 (±12)	154 (±21)	0.82 (±0.13)	0.79 (±0.12)
	115 (±17)	187 (±12)	127 (±24)	0.83 (±0.10)	0.71 (±0.13)
	138 (±17)	222 (±29)	157 (±20)	0.69 (±0.12)	0.75 (±0.20)

Table 2. Means ($\pm 95\%$ confidence intervals) of the various measures of inhibition, as estimated from the bootstrapping technique (see text for details) for Experiment 2. *Note*: $L_{50\%}$ = latency to 50% of maximum inhibition; L_{max} = latency to maximum inhibition.

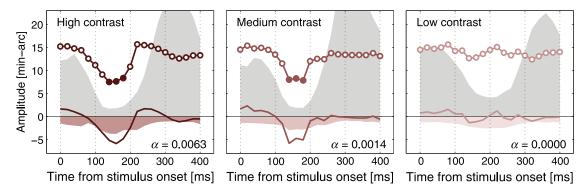


Figure 6. Analysis of microsaccade-amplitude evolution in response to irrelevant stimuli in Experiment 2. Same organization as for Figure 4. Colored lines with circular markers depict mean microsaccade amplitude in time windows of ± 40 ms. Gray background profiles illustrate mean microsaccade rates computed for the same time windows. Filled markers highlight significant decreases in mean microsaccade amplitude.

latencies of inhibition were found for low-contrast stimuli. Moreover, in the latter condition, the magnitude of inhibition was smaller.

These findings parallel earlier studies on saccade generation. In detail, the latency of express saccades (saccades with extremely short latencies) is much longer in response to low-contrast stimuli as compared to highcontrast targets. In fact, the distribution of saccade latencies was typically shifted toward longer latencies when lower target contrasts were used in simple saccade tasks (Boch et al., 1984; Kingstone & Klein, 1993; McPeek & Schiller, 1994; Reuter-Lorenz et al., 1991). Like microsaccadic inhibition, express saccades are probably generated by a low-level oculomotor circuit, involving the SC (e.g., Bell et al., 2006; Dorris, Paré, & Munoz, 1997; Munoz & Wurtz, 1992; Schiller, Sandell, & Maunsell, 1987). Hence, the results of Experiment 2 corroborate the hypothesis that the SC is involved in the generation of microsaccades.

General discussion

We examined the effect of irrelevant stimuli on microsaccade statistics. Irrelevant auditory and visual stimuli elicited a strong drop in microsaccade rate, i.e., microsaccadic inhibition. Experiment 1 showed that the effect had a similar magnitude and duration but a lower latency for luminance-contrast as compared to color-contrast flashes. Auditory stimuli triggered extremely fast inhibition, again of similar magnitude but with a different shape. Experiment 2 showed that the latency of visually induced microsaccadic inhibition is a function of the luminance contrast of the triggering stimulus, higher contrast levels resulting in faster inhibition. In addition, in all but one of the conditions tested here, mean microsaccade amplitudes decreased significantly in the course of microsaccadic inhibition. These findings are in

agreement with the hypothesis that microsaccadic inhibition is related to a fast oculomotor circuitry involving the SC (Engbert, 2006; Laubrock et al., 2005; Rolfs et al., 2005). In line with the evidence reviewed in the Introduction, we propose that the SC motor map constitutes the final arena for the generation of microsaccades and the implementation of microsaccadic inhibition. Then, downstream the SC motor map, microsaccades are executed by the saccadic burst generating circuit (Van Gisbergen et al., 1981; Van Gisbergen & Robinson, 1977).

Inspired by knowledge about neurophysiological processes in the SC, we will outline our current understanding of how microsaccades are created in a motor map commonly coding for microsaccades and saccades. This model provides mechanisms of microsaccade generation accounting for the findings reported here and elsewhere. In two final sections, we will consider alternative potential mechanisms of microsaccade generation and discuss the relation of microsaccadic and saccadic inhibition.

A conceptual model of microsaccade generation

Many models of oculomotor control hold the view that the interplay of saccades and fixations is the result of continuous integration of motor plans, competing for expression (e.g., Godijn & Theeuwes, 2002; Kopecz, 1995; Kopecz & Schöner, 1995; Munoz & Fecteau, 2002; Trappenberg et al., 2001). In these models, the plan to hold fixation is represented at the central location of a motor map. Non-central locations in the map encode saccades, their amplitudes increasing with eccentricity. Depending on the location activated, suprathreshold activity in that map generates either a saccade or fixation. Endogenous and exogenous inputs modify the distribution of activity in the map; local excitation and global inhibition shape its internal dynamics (Amari, 1977). These models are able to account for many different

behavioral anomalies, for instance the decrease of saccade latencies in the gap task (e.g., Munoz & Fecteau, 2002). However, none of them was ever used to account for microsaccade statistics in simple oculomotor tasks, nor did they incorporate a mechanism for the generation of spontaneous saccades during attempted fixation.

In line with these previous models and based on the physiological considerations in the Introduction, we propose that microsaccades and saccades are the result of competing motor plans represented in a common motor map. A schematic of such a map is depicted in Figure 7A. Following previous models of saccade generation, local excitation and global inhibition govern the dynamics of activity in the map: neighboring locations in the map activate each other while distant sites reciprocally interact. Thus, endogenous (voluntary, task-related) and exogenous (automatic, stimulus-driven) input to the map will modulate the distribution of activity both locally and globally. Saccade metrics are encoded topographically in the map; the motor plan to hold fixation is represented in the map's center (labeled 0 in Figure 7A); the plan to generate a saccade is represented in its periphery, saccade amplitudes increasing with eccentricity. Suprathreshold activity at a certain site in the map will result in the expression of the corresponding behavior (thresholds are depicted by dashed lines in Figure 7).

Thus, during fixation, activity is focused around the central part of the motor map, which receives input from the foveal region of the retina. Critically, we propose that, due to local-excitation mechanisms, this activity spreads to slightly peripheral locations. As this activity passes threshold, small-amplitude saccades may intrude while the system is in the state of steady fixation. The model holds

that the distribution and the dynamics of suprathreshold activity mediate the rate as well as the metrics of generated microsaccades. Thus, while random factors such as noise, which is inherent to physiological systems, will play a major role in determining the metrics of a particular microsaccadic event, the average distribution of microsaccade amplitudes and directions across many trials of similar stimulation will reflect the corresponding average distribution of suprathreshold activity in the motor map.

Accordingly, we propose that microsaccade rate is high for high levels of fixation-related activity and increases when this activity rises; microsaccade rate is low for low levels of fixation-related activity (Figure 7B, top panel) and decreases when this activity drops off. Second, the range of microsaccade amplitudes observed during fixation is dictated by the width of the hill of suprathreshold activity (Figure 7B, middle panel). Third, biases in the location of the hill of activity will result in biases in microsaccade direction (Figure 7B, bottom panel). These biases in activity will mainly have three sources:

- 1. an input signal representing the desired gaze position (e.g., a fixation spot) that is displaced due to fixation errors,
- 2. interactive influences from other locations in the motor map,
- 3. spatially correlated noise in the activity of the motor map.

During fixation, activity may be above threshold at any time. Therefore, a temporal trigger mechanism is needed to account for the rate of microsaccades typically

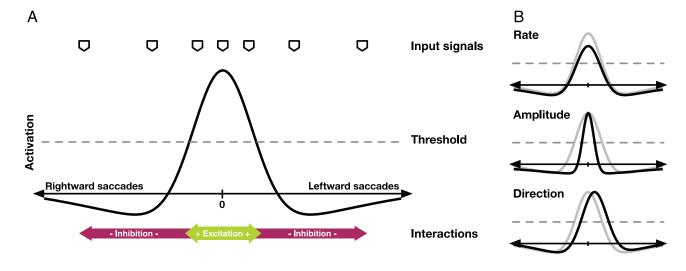


Figure 7. Outline of an activation-map model of microsaccade generation. (A) Microsaccades are the result of suprathreshold activity at the central part of a motor map. The distribution of this activity dictates the resulting microsaccadic behavior. (B) Three examples are shown to illustrate how different activity distributions (black lines) result in different microsaccadic behavior. The distribution of activity from (A) is plotted as a baseline for comparison (gray lines). Activity in the top panel results in a lower rate of microsaccades. Activity in the middle panel produces a more narrow microsaccade-amplitude distribution. Activity in the bottom panel results in an excess of microsaccades directed rightward.

observed during fixation. Various approaches to this issue may be considered. First, noise in the activity of the motor map might play a critical role for the initiation of microsaccades, as it will sometimes result in a shift of the balance of activity in favor of locations representing small saccades. Second, Engbert and Mergenthaler (2006) suggested that the trigger signal depends on the variability of visual input signals. If slow drift movements of the eyes do not suffice, microsaccades occur to enhance retinal image slip. One might also assume an autonomous timing mechanism for the generation of saccades (Richter, Engbert, & Kliegl, 2008). The dynamics of this mechanism might be subject to random variation and depend on current oculomotor needs. Thus, after the passage of some time following a saccadic eye movement, a new movement will be initiated. Alternatively or in addition to that, microsaccades might only be observed if the current gaze fixation error exceeds a critical value. Therefore, very small microsaccades might not be observed because the fixation error created does not reach a critical level (cf., Krauzlis et al., 1997). Note that the core of our model

does not depend on how the saccade-initiation process is implemented. In any case, the metrics of a triggered microsaccade depend on the current distribution of activity in the motor map.

Explaining microsaccadic inhibition

Figures 8A and 8B show schematics of microsaccaderate and amplitude evolutions, respectively, as they were observed in the present study. According to the model proposed here, a decrease in microsaccade rate results from a drop of activity at the central part of the motor map. In the case of microsaccadic inhibition, inhibitory processes relating to the onset of a visual or auditory transient cause this drop of activity. The stimulus results in enhanced activity at some location in the saccadic motor map and competes with activity at the fixationrelated site. Using the format of presentation introduced in Figure 7, Figure 8C shows how activity at the central pole of a saccade map decreases with time and how, consequently, fewer microsaccades may be generated. At the

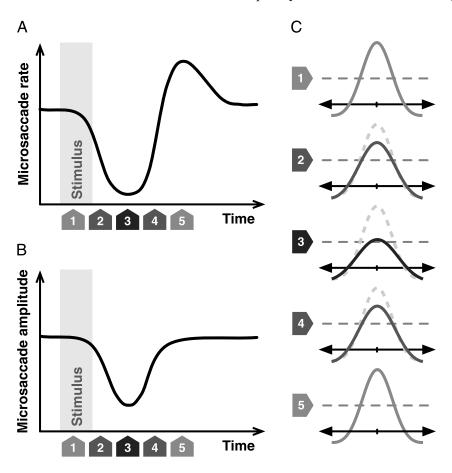


Figure 8. Schematics of (A) microsaccade rate and (B) amplitude evolutions are depicted along with (C) the process causing these effects as explained by the model of microsaccade generation (see Figure 7 for descriptions). Five states of the map are shown, highlighting five points in time during microsaccadic inhibition (identified by numbered arrows in all panels). As a result of competitive activation elsewhere in the motor map (not shown), activity at the center decreases. Consequently, less microsaccades may be generated. During strong inhibition, only small microsaccade amplitudes may be generated. The subsequent rise of activity may result in an excess of microsaccade generation. Dashed gray lines represent the baseline activity (top panel) for comparison.

same time, this model predicts that inhibition affects first of all microsaccades with large amplitudes, as corresponding activity is most likely to fall short of threshold. During microsaccadic inhibition, therefore, a greater portion of microsaccades will have small amplitudes because the peak of activity is located at the center of the map. Put differently, during strong microsaccadic inhibition, only the very central parts of the saccade map remain activated above threshold (see third panel in Figure 8C). As a consequence and in line with the data presented here, mean microsaccade amplitude decreases when inhibition increases. Indeed, there was a smaller effect on microsaccade amplitude in the auditory condition of Experiment 1 and no measurable effect in the low-contrast condition of Experiment 2. These conditions differed from others in the observed strength of inhibition, as measured by a more pointed shape (auditory condition) and a lower magnitude of inhibition (low-contrast condition). We think that weaker inhibition is related to a decreased efficiency of these stimuli in driving inhibitory mechanisms in the motor map. In the low-contrast condition, this may be explained by the weaker input signal associated with the stimulus. In the auditory condition, stimuli were played to both ears via headphones, that is, without any spatial information. As a consequence, they induced an enhanced, but unspecifically distributed mean-field activity in the motor map, less efficiently driving spatial competition mechanisms.

Two things must be noted here. First, a concurrent decrease in microsaccade rate and amplitude could also be explained in terms of enhanced fixation of gaze and a corresponding decrease of saccade-related activity (cf., Rolfs et al., 2005). In the framework of our model, microsaccade amplitude would then decrease because activity collapses around the fixation-related center of the map. We do not believe that this is the case, however, as in this scenario, a decrease in microsaccade rate should always be accompanied by a decrease in mean microsaccade amplitude. Despite a strong microsaccadic-inhibition effect, this was not the case in the low-contrast condition of Experiment 2. Still, it is possible that both mechanisms are at work in different situations (e.g., after foveal vs. peripheral stimulation). Importantly, both hypotheses predict that microsaccades are generated by fixationrelated activity in the map. Second, as mentioned in the Introduction, a decrease in mean amplitude is not necessarily predicted if the microsaccade-rate modulation is triggered by a spatial stimulus, e.g., a cue. In this case, mean microsaccade direction is biased (Engbert & Kliegl, 2003b; Galfano et al., 2004; Hafed & Clark, 2002; Laubrock et al., 2005, 2007; Rolfs et al., 2004, 2005), which might indicate a shift of the activity distribution in the saccadic motor map toward higher amplitudes. Indeed, Gowen, Abadi, Poliakoff, Hansen, and Miall (2007) did not find a decrease in microsaccade amplitude during microsaccadic inhibition in response to peripheral or spatially informative central attentional cues. Conversely, after neutral stimuli presented centrally or bilaterally in the periphery, their data suggest a decrease in mean amplitude at the time of microsaccadic inhibition. Unfortunately, the authors did not test the statistical significance of this specific contrast.

Further behavioral predictions

According to the proposed model, microsaccade statistics provide us with a behavioral correlate of the dynamics of neural activity at the central part of the motor map in which saccade generation is accomplished. Therefore, in turn, the proposed model generates a number of qualitative predictions concerning the dynamics of microsaccadic behavior in response to endogenous and exogenous inputs to the motor map, the interactions of microsaccades and saccades, and the correlations between different features of microsaccadic behavior, i.e., rate, amplitude, and direction.

First, microsaccade rate should reflect the process of fixational disengagement. Fixational disengagement can be induced by visual offsets as in the gap task (Saslow, 1967). It is also accompanying the preparation of saccadic eye movements. In these cases, microsaccade rate and amplitude should decrease during saccade preparation. Indeed, microsaccade rate decreases in expectation of the go signal in a delayed saccade task (Rolfs et al., 2006). If competing activity is spatially asymmetric with respect to the map's center, fixation-related activity should be biased in its location, accounting for direction-specific enhancements of microsaccade direction observed in spatial-cueing experiments (e.g., Engbert & Kliegl, 2003b; Galfano et al., 2004; Laubrock et al., 2005, 2007; Rolfs et al., 2004, 2005). In turn, in tasks, in which a high level of fixation-related activity is needed (e.g., countermanding saccade tasks, go/ nogo tasks, or anti-saccade tasks), microsaccade rates should be comparably high. These predictions need to be tested.

Second, microsaccades are predicted to interact with subsequent saccades as they result from suprathreshold fixation-related activity that competes with activity at saccade-related sites. Consequently, saccadic latencies should be longer in cases in which a microsaccade is observed at the time at which a saccade is required as compared to cases in which no microsaccades are observed in the same time window. This hypothesis has recently been confirmed (Rolfs et al., 2006). In addition, we predict from our model that a delay of subsequent saccades should be largest for large microsaccades, as their occurrence is most likely if fixation-related activity is very high. This hypothesis could also be confirmed (Rolfs, Laubrock, & Kliegl, 2008).

Third, although the rate, direction, and amplitude of microsaccades result from different characteristics of the activity distribution, several dependencies are predicted. First, alterations in microsaccade rate are likely accompanied by concurrent changes in the distribution of microsaccade amplitudes, as a strong decrease of suprathreshold fixation-related activity will likely result in shrinkage of the amplitude distribution (see also Figure 8).

Indeed, a correlation between microsaccade rate and amplitude has already been reported (Martinez-Conde, Macknik, Troncoso, & Dyar, 2006). An exception to this general rule may be observed if fixation-activity is high but focuses narrowly around the center of the motor map. In this case, microsaccade rate will be high, however, producing a narrow amplitude distribution. We think that the shape of the activity distribution is mainly determined by anatomical local and global interconnections in the motor map, a characteristic that may vary between individuals but clearly less so within a given individual. Therefore, we suspect that microsaccade rate and amplitude should in general covary as long as fixation-related activity is centered in the motor map. If, however, fixation-related activity is spatially biased toward one side (e.g., after the presentation of a peripheral flash), larger microsaccade amplitudes (for a particular direction) will be favored. In fact, as gaze-position errors increase, the rate of microsaccades may increase (Krauzlis et al., 1997), a putative account why biases in microsaccade direction often coincide with enhanced microsaccade rates (see Engbert, 2006, for an overview). In the case of a biased activity distribution, thus, microsaccade rate and amplitude do not necessarily covary.

Predicted response latencies of SC neurons for the present set of stimuli

We proposed that the metrics of microsaccades are implemented in the SC motor map. On this assumption and on the assumption that downstream the SC motor map, microsaccadic behavior is merely executed, we follow a reviewer's request and estimate neuronal onset latencies in the SC motor map for the set of stimuli used in this study based on the observed latency of microsaccadic inhibition; we predict this latency to be $L_{\rm SCpred} = L_{50\%} - \tau_{\rm mot}$, where $\tau_{\rm mot}$ is the motor delay taken up by the final processing stages.

To estimate τ_{mot} , we draw on published physiological data. Sparks et al. (1976) found that bursts of activity in SC motor neurons preceded saccades by 20 ms. Munoz and Wurtz (1993b) found that strong electrical stimulation of rostral SC neurons perturbed saccades after 13 ms on average; Schiller and Stryker (1972) reported a range of 20 to 30 ms. There are some indications that the delay depends on stimulus intensity. Robinson (1972) reported that the delay between suprathreshold electrical stimulation of neurons in the SC motor map and the onset of the resulting saccadic eye movements ranged from 20 to 60 ms, depending on stimulation strength. Bell et al. (2006), in turn, examined onset latencies of visually responsive SC neurons after low- and high-contrast luminance stimuli. Latencies differed by 27 ms on average in a gap task; the minimum latency of visually triggered saccades differed by 35 ms. The small difference between these values suggests that stimulus intensity may have an effect on the motor delay, but it was not reliable in that study. To represent potential influences of stimulus strength, we settled for a small range of τ_{mot} , i.e., 15, 20, and 25 ms for high, medium, and low-contrast conditions of Experiment 2, respectively. For the auditory and color-contrast conditions, $\tau_{\rm mot}$ was set to 20 ms. Table 3 provides an overview of the observed latency of microsaccadic inhibition, $L_{50\%}$, and the predicted neuronal latency of stimulus-induced activity in the SC motor map, L_{SCpred} , for our six stimulus conditions.

These predictions may be compared to values actually reported in the neurophysiological literature. In Table 3, we provide some ranges of neuronal response latencies that have been observed for stimulus variations similar to ours in single-cell recordings of the SC ($L_{\rm SCrep}$). We only considered studies of the monkey brain, where latencies are usually about 10 to 25 ms shorter than in humans (e.g., Fischer & Ramsperger, 1984). Taking this into account, the predicted values fall in the range of latencies observed in physiological studies in all cases. Note, however, that

Condition	L _{50%}	$ au_{mot}$	L_{SCpred}	L _{SCrep} (range)	References
Experiment 1					
Luminance contrast	102 (±11)	15	77 (±11)	43 to 100	Jay87
				60 to 66	Bel06
Color contrast	152 (±19)	20	132 (±19)	>98	McP02
Auditory present	53 (±9)	20	33 (±9)	22 to 67	Jay87
				40 to 60	Jay84
Experiment 2					-
High contrast	97 (±13)	15	82 (±13)	43 to 100	Jay87
	, ,		, ,	60 to 66	Bel06
Medium contrast	115 (±17)	20	95 (±17)		
Low contrast	138 (±17)	25	113 (±17)	83 to 102	Bel06

Table 3. Neuronal response latencies in SC visuomotor neurons predicted from the latency of microsaccadic inhibition observed for each of the different stimulus conditions in Experiment 1 and Experiment 2. All values are given in ms. *Note*: $L_{50\%}$ = latency to 50% of maximum inhibition; τ_{mot} = motor delay; L_{SCpred} = predicted neuronal onset latency; L_{SCrep} = range of neuronal onset latency in SC sensory-motor cells for stimulus condition similar to those examined here (if available); reported in references Bell et al. (2006; Bel06), Jay and Sparks (1984, 1987; Jay84, Jay87), and McPeek and Keller (2002; McP02). Ranges of L_{SCrep} represent means $\pm 1SD$.

the neurophysiological studies and ours differed with respect to experimental and analytical techniques, stimuli used, species examined, prior training and behavioral state of the subjects, etc. Therefore, in our study, we put strong emphasis on relative measures, i.e., the presence and direction of latency differences across experimental conditions. A direct test of absolute neuronal response latencies along the pathways involved in microsaccade generation will require the combined acquisition of eye movements and physiological data.

Other potential neurophysiological origins of microsaccade generation

The results reported here and in previous work are consistent with a model that associates microsaccade generation with activation of the rostral pole of the SC. However, other accounts could, of course, be offered. Indeed, different physiological processes were proposed that could potentially result in the production of microsaccades.

According to one alternative explanation, microsaccades are spurious events caused by a transient lack of activation in omnipause neurons (OPN) in the saccadic burst generator circuit in the brainstem (Ashe, Hain, Zee, & Schatz, 1991; Zee & Robinson, 1979). OPN serve a gatekeeper function for the generation of saccades (e.g., Bergeron & Guitton, 2002). Consequently, short-term dropouts in their tonic activation may indeed trigger small saccadic eye movements that would be interrupted as soon as OPN activity revives. We do not think, however, that OPN malfunction is the origin of microsaccades during normal fixation. As has been pointed out earlier (Abadi & Gowen, 2004), interrupted saccades would likely exhibit an abnormal velocity profile, i.e., higher peak velocities. This is clearly not the case for microsaccades, which perfectly fall on the main sequence (Zuber et al., 1965). A key difference between firing patterns of OPN and FN in the rostral SC is that OPN do not reduce their level of activity during the period of no foveal stimulation in the gap task (Everling, Paré, Dorris, & Munoz, 1998). Future studies of microsaccade-rate dynamics in a gap paradigm (e.g., Rolfs & Vitu, 2007; Saslow, 1967) may thus help disentangle the two accounts.

A further potential origin of microsaccades is subthreshold activation of saccade-related neurons in the caudal SC, i.e., neurons that generate large saccades if activated above threshold (see Rolfs et al., 2004). This proposal was considered by Gowen and Abadi (2005) but could not be substantiated by their analyses. From a physiological perspective, there is only one hint (known to the authors) that this kind of activity could be related to microsaccade production. In a side note, Carello and Krauzlis (2004) remarked that after applying sub-threshold microstimulation to cells at caudal sites in the intermediate and deeper layers of the SC, small saccades (amplitudes of

0.5 to 1°) were evoked in 10% of the trials. Note, however, that a model that puts microsaccade generation to the rostral site of the SC may also explain this finding. We speculate that the distribution of rostral activity was shifted as a consequence of peripheral stimulation. As the likelihood of generating a saccade increases with eccentricity in the rostral pole (Krauzlis et al., 1997), microsaccades could have been triggered at short latencies.

Still a number of further neurophysiological origins of microsaccade production are conceivable and we certainly cannot exclude the possibility that other topographically organized brain areas (e.g., in particular the frontal eye fields) are involved in the generation of microsaccades and the implementation of microsaccadic inhibition. To the contrary, we want to emphasize that the core principles of this model of microsaccade generation and the mechanisms proposed to underlie microsaccadic inhibition are, in general, independent of a particular physiological equivalent. The central role of the SC in the generation of involuntary saccades and the compatibility of this account with the wealth of data presented here and elsewhere, however, strongly suggests that activation of the rostral SC is at least involved in the generation of microsaccades, if not their major determinant. Therefore, we consider it most parsimonious to propose that this structure is the physiological analogue of the motor-map model developed here.

Relation to saccadic inhibition

As already noted in the literature (Engbert, 2006; Engbert & Kliegl, 2003a, 2003b; Laubrock et al., 2005; Rolfs et al., 2005), the appearance of the microsaccaderate signature is evocative of a phenomenon commonly referred to as saccadic inhibition. It describes the effect of irrelevant transients on statistics of saccades that was observed in a broad range of eye-movement tasks, including simple saccade paradigms (Reingold & Stampe, 2002), reading (Reingold & Stampe, 1999, 2000, 2003, 2004; Stampe & Reingold, 2002), visual search (Reingold & Stampe, 1999, 2000, 2004; Stampe & Reingold, 2002), and picture viewing (Graupner et al., 2007; Pannasch et al., 2001). In a typical saccadic-inhibition paradigm, short flashes are presented in the course of an oculomotor task. The main effect that arises from these paradigms is a strong, knee-jerk decrease in the frequency of (normal) saccades forming a dip, time-locked to the flash. This saccadic inhibition occurs as early as 60-70 ms after flash onset. Therefore, it was thought to be an oculomotor reflex related to inhibitory processes in low-level oculomotor structures, i.e., the SC.

Thus, the phenomena of saccadic and microsaccadic inhibition as well as their proposed locus of implementation are very similar, indicating that both effects are probably essentially the same, but originate in different fields of research. However, one major difference between the studies on saccadic inhibition and those investigations

reporting microsaccade-rate evolution is that in the latter case the examined stimuli were always task-relevant, i.e., they carried information relevant to the task at hand. In the present study, much effort was spent to engage performers in the visual discrimination task, thus, to have them ignore the irrelevant stimuli. First, the task itself was very demanding; low-contrast target stimuli were presented for extremely short intervals. Second, irrelevant stimuli were not indicative for the current choice of the discrimination target. Third, they were little predictive for the time of target occurrence. That is, the discrimination target could appear at any time in a range of 500 to 1500 ms after the presentation of an irrelevant stimulus. In Experiment 1, a stimulus was not presented (and, thus, not expected) in 50% of the trials. Finally, continuous feedback on visual-discrimination performance was given to the participant, encouraging a strong focus on the instructed task. Despite all this, strong microsaccadic inhibition was observed. Thus, the present results bring microsaccadic and saccadic inhibition closer together.

Moreover, we demonstrated differential effects of stimulus properties on the time-course of the inhibition of microsaccades. Similar findings can be found in the literature on saccadic inhibition. As has been documented by Stampe and Reingold (2002; see also Stampe, 1999), saccadic inhibition is sensitive to the luminance of the transient event. Inhibition in response to strong changes in the luminance of the visual display had a lower latency than did transients with identical luminance. In the present study, the same pattern of results was evident for the impact of luminance on the inhibition of microsaccades.

For auditory input, the findings in saccadic-inhibition literature are inconsistent. Reingold and Stampe (2004) argued that saccadic inhibition is an optomotor reflex of the oculomotor system, which is sensitive to visual input only. In their paradigm, auditory input (2000 Hz beeps presented for 33 ms) did not affect the rate of saccades during reading. Unfortunately, the authors did not report the volume of the auditory stimuli used, however, frequency and duration of the used beeps suggest that stimulus intensity might not have been sufficient to trigger saccadic inhibition. In fact, neurons in the deep layers of the SC that are sensitive to auditory input might code for different stimulus intensities in terms of response magnitude rather than latency (Perrault, Vaughan, Stein, & Wallace, 2003). Using (possibly more salient) auditory stimuli, Graupner et al. (2007) and Pannasch et al. (2001) also found saccadic inhibition for auditory stimuli. In Pannasch et al.'s study, participants were presented with either of two types of distractors during fixations in a free-picture-viewing task: a small black spot in the visual periphery or a 1000-Hz tone played through loudspeakers. As compared to the effect elicited by visual distractors, saccadic inhibition was weaker in the auditory condition. In addition, it had a shorter latency. Specifically, transient visual events were associated with a drop in the probability of saccades with a latency of 100 ms after distractor onset, whereas auditory distractors reduced the rate of saccades 80 ms after stimulus onset. Graupner et al. (2007) reported similar effects using comparable tones (1500 Hz beeps at 70 dB played for 75 ms). These results are consistent with the time-courses of microsaccadic inhibition reported here. Pannasch et al. (2001) argued that any new event entering a sensory channel would produce an orienting reflex, which expresses itself in a decreased saccade rate. They concluded that saccadic inhibition appears not to be an optomotor reflex but instead arises from a more general process.

The extremely low response time of the oculomotor system in saccadic inhibition in combination with its sensitivity to low-level properties of the stimulus pushed investigators to propose that the SC is the primary candidate for mediating the effect. It was subject to discussion, however, how saccadic inhibition is implemented within that structure (see Reingold & Stampe, 2002). First, it might result from increased activity of saccade-related sites in the SC motor map, thereby eliminating potentially existing peaks of activity in the motor map. Second, the stimulus-induced activity might enhance fixation-related activity, which, in turn, decreases the likelihood of saccadic activity to reach a necessary threshold. Here, we proposed that microsaccades are a behavioral consequence of activity in fixation-related sites in saccadic motor maps and showed that microsaccade rate strongly declines in response to irrelevant input. The study of microsaccade statistics in tasks exploring saccadic inhibition might help to clarify which mechanisms constitute this effect.

Conclusion

With an increasing interest in microsaccades and their function for perception and oculomotor control, it becomes more important to understand their implementation in the circuitry of saccade generation. Our results are compatible with the view that microsaccades result from fixation-related activity in the central part of a motor map encoding saccades of all amplitudes on different sites of this map. A likely neuronal counterpart of this model is the motor map in the SC. Thus, the model provides a blueprint for the computational implementation of microsaccades and their interactions with regular saccades. It affords behavioral predictions as well as predictions about the associated neurophysiological dynamics.

Acknowledgments

We thank Eyal Reingold for his contributions in the planning stage of the experimental research and two anonymous reviewers for their helpful comments on earlier versions of this manuscript. This research was supported by Deutsche Forschungsgemeinschaft (Grants KL955/3 and /6).

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Commercial relationships: none.

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Footnote

¹Supplementary analyses showed that a benefit in response times after irrelevant visual stimuli increased with the length of the stimulus-target interval (STI). Only in the visual condition, increasing STIs were accompanied by a decrease in microsaccade rate. Because microsaccades occurring at the time of target presentation significantly increased response times (possibly due to saccadic-suppression effects associated with microsaccades), the lower rate of microsaccades in the late time interval, may explain the faster responses.

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