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Microsaccades inhibition triggered by a repetitive visual distractor is not subject to habituation: Implications for the programming of reflexive saccades

Francesca Bonetti [a], *Matteo Valsecchi* [b] *and Massimo Turatto* [a,*]

[a] *Center for Mind/Brain Sciences, University of Trento, Italy*
[b] *Department of Psychology, University of Bologna, Italy*

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ABSTRACT

The oculomotor capture triggered by a peripheral onset is subject to habituation, a basic form of learning consisting in a response decrement toward a repeatedly presented stimulus. However, it is unclear whether habituation of reflexive saccades takes place at the saccadic programming or execution stage (or both). To address this issue, we exploited the fact that during fixation the programming of a reflexive saccade exerts a robust but short-lasting phasic inhibition in the absolute microsaccadic frequency. Hence, if habituation of reflexive saccades occurs at the programming stage, then this should also affect the microsaccadic frequency, with a progressive reduction of the inhibitory phase. Conversely, if habituation occurs only at the later stage of saccade execution, the no change in the microsaccadic pattern is expected. Participants were repeatedly exposed to a peripheral onset distractor, and when eye movements were allowed, we replicated the oculomotor capture habituation. Crucially, however, when fixation was maintained the microsaccadic response did not change as exposure to the onset progressed, suggesting that habituation of reflexive saccades does not take place at the programming stage in the superior colliculus (SC), but at the later stage of saccade execution in the brainstem, where the competition between different saccades might be resolved. This scenario challenges one of the main assumptions of the *competitive integration model* for oculomotor control, which assumes that competition between exogenous and endogenous saccade programs occurs in the (SC). Our results and interpretation are instead in agreement with neurophysiological studies in non-human primates showing that saccadic adaption, another form of oculomotor plasticity, takes place downstream from the SC.

* *Corresponding author*. Center for Mind/Brain Sciences, University of Trento, Italy.
E-mail address: massimo.turatto@unitn.it (M. Turatto).

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1. Introduction

Although we can deliberately select the target of our eye movements (voluntary saccades), our gaze is sometimes involuntarily captured by salient stimuli (reflexive saccades), especially if they abruptly appear in the visual field, a phenomenon called *oculomotor capture* (Irwin, Colcombe, Kramer, & Hahn, 2000; Theeuwes, Kramer, Hahn, & Irwin, 1998; Theeuwes, Kramer, Hahn, Irwin, & Zelinsky, 1999). The fact that reflexive saccades are automatically engaged by salient stimuli has a high adaptive value, because it allows the organism to give immediate priority of analysis to events potentially relevant for survival. Yet, if the gaze were completely at the mercy of salient stimuli, the oculomotor capture response would become maladaptive, as the eyes would be continuously attracted by the same salient stimulus, albeit irrelevant, repeatedly appearing in the visual field. Hence, the brain must possess some mechanism to suppress, or at least to attenuate, the detrimental influence of repetitive distractors.

In agreement with this possibility, there is evidence that the oculomotor system is progressively less engaged by irrelevant repetitive onset stimuli (Bonetti & Turatto, 2019; Godijn & Kramer, 2008). Interestingly, the oculomotor capture decline conforms to several key features of habituation (Bonetti & Turatto, 2019), a response decrement that results from repeated stimulation and that does not involve sensory or motor fatigue (Thompson, 2009). Hence, while recent studies have proposed that capture can be attenuated by means of strategic top-down suppressive mechanisms that send inhibitory signals to the distractor representation (Gaspelin, Leonard, & Luck, 2017; Gaspelin & Luck, 2018; Sawaki & Luck, 2010), it should be noted that a more automatic-like mechanism of habituation of the orienting response (Sokolov, 1963), based on statistical learning derived from the history of stimulation, could successfully explain the same results. Specifically, according to the *stimulus-model comparator* theory proposed by Sokolov (1963), an antecedent of modern theory of predictive coding (Rao & Ballard, 1999), the brain constantly generates predictions about the incoming sensory input on the basis of a model the "world" derived from the past sensory experience. When there is a match between expectations and sensory input the orienting response evoked by the latter is suppressed, namely habituation is observed. By contrast, when expectations are violated, an orienting response is triggered with the purpose of identifying the new sensory input (Sokolov, 1963). Habituation has been shown to affect different types of components of the orienting response, including cortical, autonomic and somatic changes like the eye movements, with the latter apparently being the firsts to habituate as originally reported by Sokolov himself.

Regardless of whether oculomotor capture is attenuated by habituation-like mechanisms, or by a top-down voluntary suppressive mechanisms, what remains unclear is whether this phenomenon occurs at the saccadic execution stage, or at the earlier stage of saccadic programming. Indeed, the programming and execution of saccades are two functionally distinct processes with different neural substrates. With this regard, a key role in saccadic programming is played, among others, by the SC, a subcortical sensory-motor structure that integrates information from cortical regions such as the frontal eye fields (FEFs), the supplementary eye field (SEF) and the posterior parietal cortex (PPC) (Schall, 1991; White & Munoz, 2012; Wurtz, Basso, Paré, & Sommer, 2000). The execution of saccades, instead, involves a particular network of neurons in the brainstem called "saccadic burst generator" (SBG), which receives commands from the SC to either hold fixation or make a saccade, and generates the corresponding motor signals to drive the extraocular muscles controlling the eye position (Otero-Millan, Macknik, Serra, Leigh, & Martinez-Conde, 2011; Scudder, Kaneko, & Fuchs, 2002; Sparks, 2002).

As for the functional mechanisms controlling the gaze direction, different theoretical models have been proposed. The most prominent one, called *competitive integration model*, postulates that the oculomotor control arises from the integration of competitive motor programs in the same motor map of the SC (e.g., Findlay & Walker, 1999; Godijn & Theeuwes, 2002; Trappenberg, Dorris, Munoz, & Klein, 2001). Notably, activity at one location of the map spreads to neighboring locations, whereas it inhibits activity at distant locations, with a saccade being generated when a threshold of activation is reached at a given location. Furthermore, in agreement with the notion that fixation-related cells are comparable to saccade-related cells, but with a foveal receptive field (Munoz & Wurtz, 1993b, 1993a), the same competitive mechanism, based on lateral inhibition, would also explain the interplay between fixation and saccades. Since fixation location is part of the saccade map, when fixation is actively maintained the central portion of the map (located in the rostral part the of SC) is strongly activated, and the periphery of the map (represented in the caudal part of the SC) inhibited; by contrast, the opposite scenario emerges when a saccade is programmed, with the central part of the map inhibited by the peripheral activity (e.g., Godijn & Theeuwes, 2002).

On the basis of psychophysical data and physiological considerations, Rolfs, Kliegl, and Engbert (2008) proposed an extended version of the *competitive integration model* that can also account for the pattern of microsaccadic activity elicited by the occurrence of a salient stimulus in the visual field. Microsaccades are a specific type of fixational eye movements that share some important characteristics with normal saccades, and are considered part of the same eye movement continuum (Martinez-Conde, Otero-Millan, & MacKnik, 2013; McCamy et al., 2012; Otero-Millan, Troncoso, Macknik, Serrano-Pedraza, & Martinez-Conde, 2008). For example, saccades and microsaccades are both binocular eye movements, with the same amplitude and direction in both eyes (Ditchburn & Ginsborg, 1953), and both following the *main sequence* (Zuber, Stark, & Cook, 1965), namely a linear relationship between peak velocity and amplitude. Furthermore, saccades (Deubel & Schneider, 1996; Kowler, Anderson, Dosher, & Blaser, 1995) and microsaccades (Engbert, 2006; Galfano, Betta, & Turatto, 2004; Hafed & Clark, 2002; Laubrock, Engbert, & Kliegl, 2005; Rolfs, Engbert, & Kliegl, 2004) are also similarly affected by spatial attention. According to the Rolfs et al. (2008) model, microsaccades and saccades originate from competitive commands in the same SC motor map. Specifically, activation in the central part of the map, coding for foveal locations, is observed during fixation. Crucially, because of local-excitation mechanisms this activity spreads to neighboring more peripheral

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locations, thus eliciting small-amplitude saccades during fixation, identified as microsaccades. Furthermore, in agreement with previous competitive integration models (Godijn & Theeuwes, 2002; Kopecz, 1995; Trappenberg et al., 2001), activity in the peripheral part of the map, associated with saccadic programming, would inhibit fixational activity in the central part of the map, and vice versa. Thus, the Rolfs et al. (2008) model provides an explanation for the inhibition of microsaccades observed soon after the occurrence (approximately 100 msec later) of a peripheral stimulus in the visual field, a result that since the study of Engbert and Kliegl (2003) has been consistently reported (e.g., Betta & Turatto, 2006; Hafed & Ignashchenkova, 2013; Laubrock et al., 2005). As originally proposed by Engbert (2006), the idea is that the transient increased activation of a peripheral location in the SC motor map, corresponding to the programming of a saccade, has an inhibitory effect on the activity in the rostral pole, which codes for fixation, and consequently the rate of microsaccades, which depends on such activation, drops toward zero (Rolfs et al., 2008).

Because of this functional relation between saccades and microsaccades, one should predict that if habituation of oculomotor capture results from a weakening of the saccadic programming activity in the SC, then the microsaccadic inhibitory response evoked by the peripheral event should become less pronounced as habituation unfolds. Note that the same prediction holds true even if oculomotor capture is progressively reduced by virtue of top-down inhibitory signals applied to the distractor (Gaspelin & Luck, 2018). Ideally, if with practice the activity related to the exogenous saccadic programming vanishes completely, then the microsaccadic inhibitory phase should in parallel disappear too. By contrast, if habituation of oculomotor capture does not take place at the saccadic programming stage, but occurs downstream from the SC, at the saccadic execution stage, then the early microsaccadic inhibition should remain unaffected by the repetitive exposure to the peripheral distractor.

2. Experiment 1

To reveal whether the programming of saccades is subject to habituation, we exploited the fact that, as postulated by existing competitive integration models (e.g., Rolfs et al., 2008), this neural activity should affect the pattern of microsaccades. To this aim, we recorded microsaccadic activity elicited by a peripheral onset stimulus while fixation was maintained. On each trial, the target was a white disk appearing either to the left or to the right (counterbalanced across participants) of a central fixation point. The task was to silently count the number of targets during each block of the experiment. This paradigm ensured that, in addition to the fixation point that remained on the screen for the entire trial, the only transient onset stimulus appearing on the display was the one relevant for the task.

2.1. Methods

Below we report how we determined our sample size, all data exclusions (if any), all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study.

2.1.1. Participants

Twenty-four participants (16 females, mean age = 22.75) of the University of Trento were recruited from the Department of Psychology for course credits or monetary compensation (6 €). They had normal or corrected-to-normal vision and were all naïve as to the purpose of the experiment. Informed consent was obtained from all participants. All the experiments were carried out in accordance with the Declaration of Helsinki, and with the approval of the local institutional ethics committee (Comitato Etico per la Sperimentazione con l'Essere Umano, Università degli Studi di Trento, Italy).

In this and the following experiments our sample size was determined on the basis of our previous study on oculomotor capture (Bonetti & Turatto, 2019).

2.1.2. Apparatus

Stimuli were presented on a 23.6-inch VIEWPixx/EEG color monitor (1920 × 1080, 100 Hz) and generated with a custom made program written in MATLAB and the Psychophysics Toolbox (Pelli, 1997) running on a Dell Precision T1600 machine (Windows 10).

2.1.3. Stimuli and procedure

Each trial (see Fig. 1, Panel A) started with the presentation of a grey central fixation point (diameter of .73°; .75 cd/m^2) on a black background (.07 cd/m^2). After 2500 msec, the target, consisting of a peripheral white disk (diameter of 2°; 45.3 cd/m^2) appeared for 87 msec either on the left or on the right of the fixation point, with an eccentricity of 5°.

For each participant the position of the target was fixed. After the target occurrence, the fixation point remained on the screen for 2500 msec. The next trial started after a blank inter-trial interval of 3000 msec, so that the total length of the trial was about 8 s. Participants were instructed to maintain the gaze on the central point while focusing their attention on the target. The task was to silently count the number of targets and to report it at the end of each block by using the computer keyboard. Since the target appeared in all trials, the number of trials was slightly different in each block to keep participants engaged in the counting task. Hence, there were 48 trials in Block 1, 50 trials in Block 2, and 46 trials in Block 3.

2.1.4. Eye movements recording and microsaccades detection

Eye movements were recorded binocularly using an EyeLink 1000 Desktop Mount system (SR Research, Ontario, Canada), with a sampling rate of 500 Hz and a spatial resolution of less than .01° of visual angle. Each block was preceded by a nine-point calibration procedure, which was repeated during the block if participants' gaze drifts exceeded 1.5°. Microsaccades were detected using a velocity-based algorithm developed by Engbert and Kliegl (2003), and were then analyzed using custom-made scripts in Matlab. The algorithm was applied to 1300-msec epochs of eye-position recording, ranging from 300 msec prior to the presentation of the lateral stimulus to 1000 msec after the stimulus onset. The algorithm defines microsaccades as part of the eye movement trajectory, where velocity (calculated over a moving window of nine samples)

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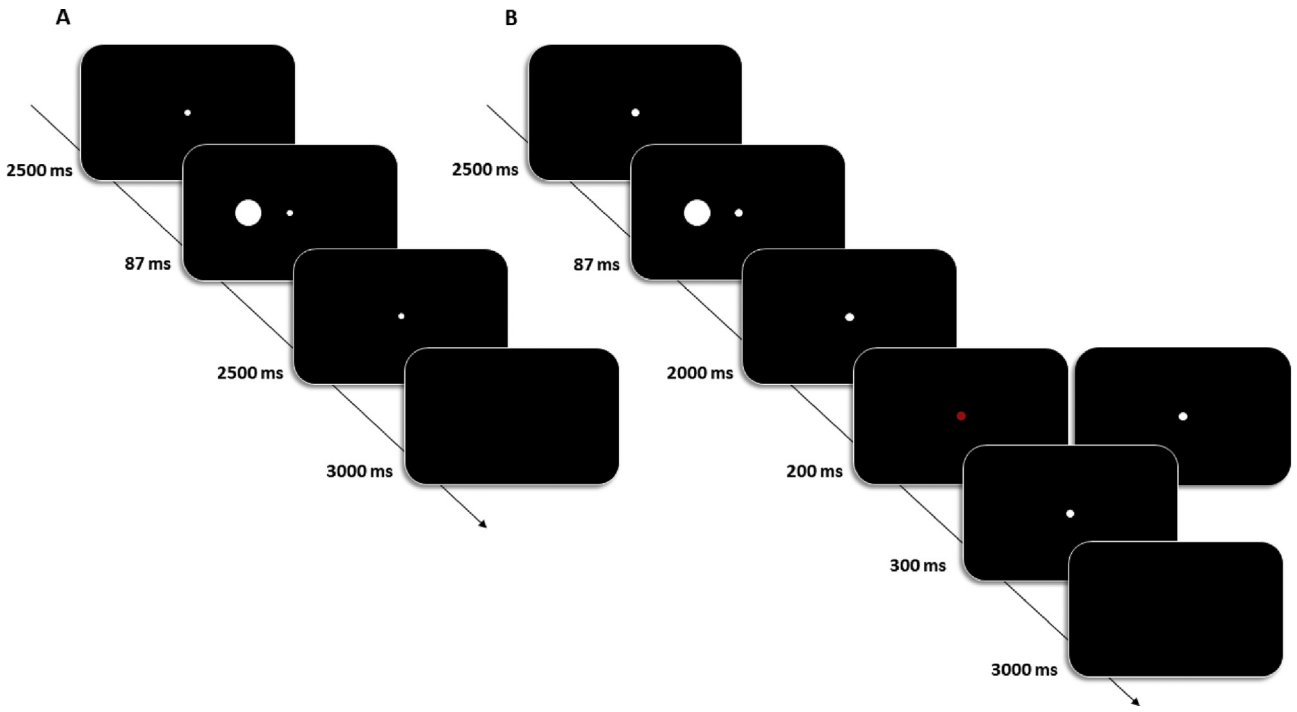

Fig. 1 – Example of stimuli used in the experimental session. Panel A depicts the main events of Experiment 1. A grey central fixation point was presented for 5087 msec, during which a peripheral onset stimulus (a white disk) appeared for 87 msec. The task was to silently count the number of onset stimuli, and to report this number at the end of each block. Panel B depicts the main events of Experiment 2, in which the trial sequence was similar, with some exceptions. 2000 msec after the onset of the peripheral stimulus the central fixation point could turn to red for 200 msec, or alternatively it could remain grey (see Methods for details). The task was to silently count the number of times in which the fixation changed its color, and to report this number at the end of each block. In both experiments, each trial was followed by a blank inter-trial interval of 3000 msec. The total duration of each trial was the same in both experiments (~8 s).

exceeds a relative velocity threshold multiple (λ) of the median SD. We used a relative velocity threshold set to five median-based SDs of the velocity values observed (λ = 5), a minimum temporal threshold of six samples (12 msec, since the sampling rate was set to 500 Hz), and a maximum peak velocity of 300° s^{-1}. Epochs with eye blinks or saccades exceeding 1.5° in amplitude were discarded from analysis. Less than 5% of data was discarded because of this criterion.

2.2. *Results and discussion*

2.2.1. *Accuracy*

In all experiments, the first step was to calculate participants' accuracy in the counting task, which was high in all blocks (96% in Block 1, 98% in Block 2, 95% in Block 3), thus confirming that participants were actively engaged in the task.

2.2.2. *The main sequence*

Before analyzing the data, we checked whether the microsaccadic eye movements that we detected satisfied the velocity–amplitude relationship criterion (Zuber et al., 1965). According to this criterion, a positive correlation, called the *main sequence*, must exist between saccadic amplitude ad saccadic peak velocity. Fig. 2 shows the microsaccadic peak velocities of all participants as a function of the microsaccades amplitudes, for each experiment: in Experiment 1 (Panel A) the linear relationship between amplitude and velocity was strongly positive, as confirmed by a very high correlation coefficient ($r = .94$).

2.2.3. *Absolute frequency of microsaccades*

We then computed the absolute frequency of microsaccades, which was calculated separately for each participant and block of trials, and then averaged across participants. The rate of microsaccades was calculated by convolving the raw microsaccadic frequency with a Gaussian window with 25 msec standard deviation (SD), moving in 1-mses steps. Visual inspection of Fig. 3 reveals the classical pattern of response in the microsaccadic rate, which includes an initial inhibition of the microsaccadic frequency followed by a rebound (Engbert & Kliegl, 2003). The crucial question was whether the inhibition of microsaccades decreased between the first and the last block. To this aim, we decided to compare the frequencies of microsaccades in the 100 msec preceding stimulus onset and in a 100 msec window around the time of overall peak inhibition in Block 1 (108–208 msec post stimulus onset). A two-way repeated-measure analysis of variance (ANOVA) with Block (1 *vs* 3) and ROI (pre-stimulus *vs* peak inhibition) as within-observers factors, yielded a significant effect of ROI: $F(1,23) = 36.258$, $p < .001$, $\eta_p^2 = .612$, and no significant main effect of Block: $F(1,23) = .125$, $p = .727$, $\eta_p^2 = .005$. Crucially, the analysis did not yield a significant two-way

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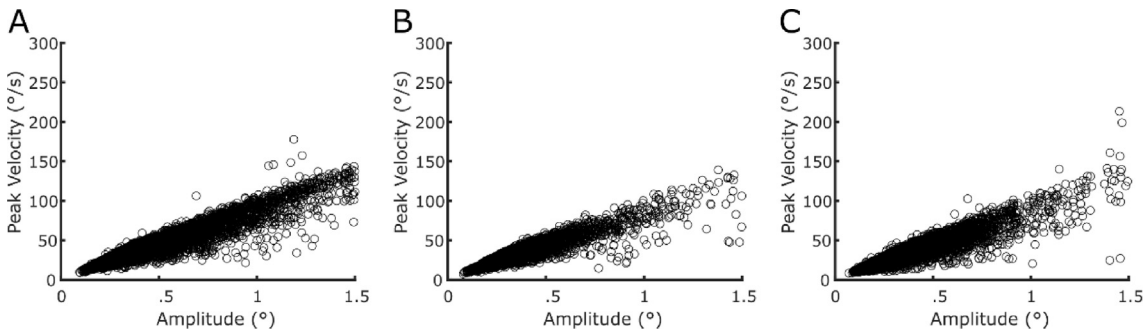

Fig. 2 – Each panel depicts the relation between microsaccades peak velocity and amplitude for all blocks of trials in Experiment 1 (Panel A), and Experiment 2 (Panel B), and for blocks 2–4 in Experiment 3 (Panel C). The plot contains microsaccades from the whole pool of participants (4833 microsaccades in Experiment 1; 5010 microsaccades in Experiment 2; 6751 microsaccades in Experiment 3).

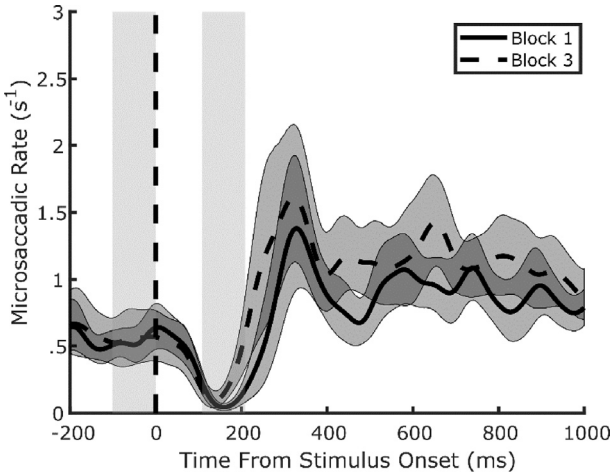

Fig. 3 – Time course of absolute microsaccadic frequency in response to the occurrence of the peripheral onset in Experiment 1 (Block 1 *vs* 3). The plots were created by convolving the frequency of microsaccades with a Gaussian filter of 25 msec SD. The *vertical dashed line* indicates the onset of the peripheral stimulus, whereas the *vertical grey areas* delimit the time windows (pre-stimulus baseline and maximum inhibition) which were used for statistical testing. *Shaded error areas* represent 95% inter-subject confidence intervals of the mean absolute microsaccadic frequency calculated through bootstrapping. Notice that in both blocks a clear inhibition of microsaccades is observed.

interaction $F(1,23) = 1.019$, $p = .323$, $\eta_p^2 = .042$, indicating that the strength of microsaccadic inhibition was largely unchanged between the first and third block.

The results of Experiment 1 thus provided no indication of habituation in the absolute frequency of microsaccades during the inhibitory phase, which seems to suggest that the programming of the saccade generated by the onset of the lateralized target was not subject to habituation either. However, one may note that habituation of microsaccades did not emerge because the peripheral onset was task relevant, and it is well established that habituation is weaker for significant stimuli than for stimuli that are irrelevant to the organism (McSweeney & Murphy, 2009; Steiner & Barry, 2011, 2014; Verbaten, Woestenburg, & Sjouw, 1980). In particular, the study of Verbaten et al. (1980) investigated the possibility that, compared to task-irrelevant stimuli, task-relevant onsets may induce a stronger orienting response (OR), which is more resistant to habituation. The authors found that the skin conductance response (SCR), which is an index of OR, was larger and had a lower habituation rate in response to task-relevant than to task-irrelevant stimuli. Furthermore, it has been shown that the relevance of the stimulus being processed affects also the frequency of microsaccades (Valsecchi, Betta, & Turatto, 2007; Valsecchi & Turatto, 2009). For example, Valsecchi et al. (2007) revealed a difference in the absolute frequency of microsaccades between the active and passive condition, namely between the condition in which participants had to silently count the number of stimuli, and the condition in which the stimuli were passively viewed.

Hence, the next experiment examined whether habituation of the microsaccadic inhibition could emerge when the peripheral onset was task irrelevant.

3. Experiment 2

We used the same paradigm as Experiment 1, with the important exception that the lateral disk was task-irrelevant, as participants were asked to perform a task on the central fixation point.

3.1. Methods

3.1.1. Participants

Twenty-four participants (16 females, mean age = 23.66) of the University of Trento were recruited from the Department of Psychology for course credits or monetary compensation (6 €). They had normal or corrected-to-normal vision and were all naïve as to the purpose of the experiment. Informed consent was obtained from all participants.

3.1.2. Apparatus

As in Experiment 1.

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3.1.3. *Stimuli and procedure*

As in Experiment 1, except that, on each trial, after the disappearance of the lateral onset disk, the fixation point remained on the screen for other 2500 msec, during which it could turn to red for 200 msec, or alternatively it could remain gray (see Fig. 1, Panel B). Participants were instructed to maintain the gaze and attention on the central fixation point. The task was to silently count the number of times in which the fixation point turned to red, and to report this number at the end of each block by using the computer keyboard. Since the number of the fixation color changes varied across blocks (ranging from five to eight), the number of trials (50) was the same in each block.

3.1.4. *Eye movements recording and microsaccades detection*

As in Experiment 1.

3.2. *Results and discussion*

3.2.1. *Accuracy*

The accuracy of participants was high in all blocks (100% in Block 1, 98% in Block 2, 97% in Block 3), thus confirming that participants were really focused on counting the number of times in which the central fixation point became red.

3.2.2. *The main sequence*

Fig. 2 (Panel B) shows the relation between microsaccadic peak velocities and amplitude for all participants in the three blocks of trials of Experiment 2. The relation was strongly positive, and confirmed by a very high correlation coefficient ($r = .93$).

3.2.3. *Absolute frequency of microsaccades*

Absolute frequency of microsaccades is depicted in Fig. 4. As in Experiment 1, visual inspection of Fig. 4 reveals a microsaccadic inhibition, which in Block 1 peaked again at exactly 158 msec after the stimulus onset, then followed by an increment of the microsaccadic rate (~350 msec after the stimulus onset). A two-way ANOVA with Block (1 *vs* 3) and ROI (pre-stimulus *vs* peak inhibition) as within-observers factors, yielded the same pattern of significance as in Experiment 1: a significant effect of ROI: $F(1,23) = 33.178$, $p < .001$, $\eta_p^2 = .591$, no significant main effect of Block: $F(1,23) = 3.536$, $p = .073$, $\eta_p^2 = .133$, and no two-way interaction: $F(1,23) = 3.536$, $p = .073$, $\eta_p^2 = .133$. Notice that the results for the main effect of Block and for the interaction are identical because the absolute microsaccadic rate in the peak inhibition window, i.e., the total number of saccades detected, was exactly the same in the two blocks, so that the difference between the rates in the pre-stimulus ROI drives both the effect of block and the interaction. Also notice that the rate is slightly higher in the pre-stimulus ROI for the third block, so that, if anything, the interaction would even point to more inhibition at the end of the experiment.

The second experiment was conducted to see whether in Experiment 1 habituation of microsaccades did not occur because the peripheral onset stimulus was task-relevant. However, no evidence of habituation emerged even when the onset was completely task-irrelevant as in the present experiment. Although previous studies have clearly shown that the reflexive saccades elicited by a similar onset are subject to a robust habituation (Bonetti & Turatto, 2019), a lack of habituation in the microsaccadic activity supports the interesting possibility that the progressive attenuation of the oculomotor capture phenomenon does not involve the stage of saccadic programing. However, since the current paradigm and the one used to show habituation of exogenous saccades (Bonetti & Turatto, 2019; Godijn & Kramer, 2008), are not identical, before we accept the conclusion that saccade programming is not subject to habituation we decided to conduct a final experiment, in which we directly addressed, in the same pool of participants, and with the same stimuli, both habituation of saccades (Block 1) and microsaccades (Blocks 2–4).

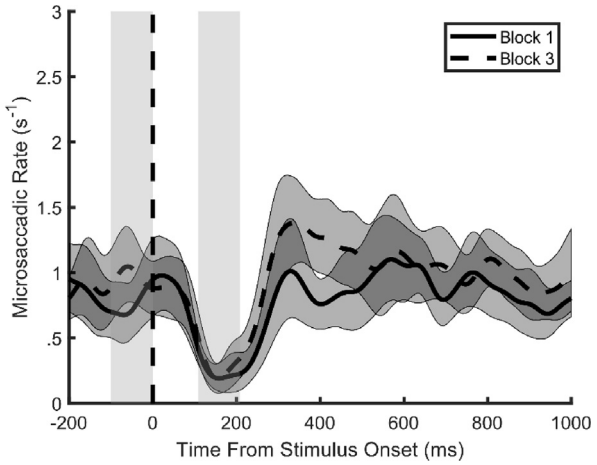

Fig. 4 – Time course of absolute microsaccadic frequency in response to the occurrence of the peripheral onset in Experiment 2 (Block 1 *vs* 3). The plots were created by convolving the frequency of microsaccades with a Gaussian filter of 25 msec SD. The *vertical dashed line* indicates the onset of the peripheral stimulus, whereas the *vertical grey areas* delimit the time windows (pre-stimulus baseline and maximum inhibition) which were used for statistical testing. *Shaded error areas* represent 95% inter-subject confidence intervals of the mean absolute microsaccadic frequency calculated through bootstrapping. Notice that in both blocks a clear inhibition of microsaccades is observed.

4. Experiment 3

The first block of the present experiment was designed to replicate previous evidence of habituation of oculomotor capture elicited by a peripheral task irrelevant onset. For this reason, participants were asked to make a saccade, as fast and as accurate as possible, toward a target stimulus, while in some trials an additional peripheral onset, serving as distractor, appeared on the screen. By contrast, from the second block onward (Blocks 2–4) the task was identical to that used in Experiment 2, and allowed us to replicate the lack of habituation in microsaccades found in previous experiments,

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but this time elicited by the same onset stimulus used in Block 1.

Hence, the present experiment differed from the previous two in the following main features: (a) the duration of the peripheral onset stimulus was increased from 87 msec to 1000 msec; (b) in the first block the display consisted of four grey disks arranged around the fixation point, one of which turned to green to indicate the goal of the endogenous saccade; (c) in the first block the frequency of the peripheral onset was lowered from 100% to 30%; (d) the peripheral onset position was randomly changed between two possible locations (at clock position 3 or 9). These changes were necessary to make the paradigm as much similar as possible to the one used in our previous study (Bonetti & Turatto, 2019), in which we documented habituation of oculomotor capture.

4.1. Methods

4.1.1. Participants

Twenty-four participants (19 females, mean age = 21.25) of the University of Trento were recruited from the Department of Psychology for course credits or monetary compensation (6 €). They had normal or corrected-to-normal vision and were all naïve as to the purpose of the experiment. Informed consent was obtained from all participants.

4.1.2. Apparatus

As in Experiments 1 and 2.

4.1.3. Stimuli and procedure

Block 1. Each trial (see Fig. 5, Panel A) started with the presentation of a grey fixation point (diameter of .73°; .75 cd/m^2) on a black background (.07 cd/m^2), surrounded by four equidistant grey disks (diameter of 1.2°, .62 cd/m^2) placed on an imaginary circle (at clock positions 1, 5, 7 and 11) with a radius of 5° around the central fixation point. After 2500 msec one of the disks turned green (17 cd/m^2) while the others remained grey. The unique green disk was the saccadic target, and on each trial its position was randomly assigned in one of the four possible locations. Participants were asked to make a saccade toward the target disk as fast as possible. In the first thirty trials there was no distractor, whereas starting from Trial 31, an onset white disk (diameter of 2°; eccentricity of 5°; 45.3 cd/m^2) could appear (30% frequency) simultaneously with the target in one of two possible locations (at clock position 3 or 9). If a saccade was detected before the target occurrence, an error message appeared on the screen, and the trial was aborted and then restarted. This allowed us to ensure that each saccade toward the target or the distractor started from the central fixation point. Each trial was followed by a blank inter-trial interval of 3000 msec. The total length of the trial was about 8 s.

Blocks 2–4. The stimuli (see Fig. 5, Panel B) were identical to those used in the first block, except that no target was presented, namely none of the four grey disks turned to green. In addition, in a small proportion of trials the fixation point turned to red for 200 msec. Participants were instructed to maintain fixation on the central fixation point throughout the whole trial, and were informed that the peripheral onset would have appeared with a frequency of 100%, in the same positions of Block 1. After the disappearance of the peripheral onset, the fixation point remained on the screen for 2500 msec, during which it could turn to red for 200 msec. Each trial was followed by a blank inter-trial interval of 3000 msec. The total length of the trial was about 8 s. As in Experiment 2, the participants' task was to silently count the number of times in which the fixation point turned to red, and to report this number at the end of each block by using the computer keyboard.

4.1.4. Eye movements recording and microsaccades detection

As in Experiments 1 and 2.

4.2. Results and discussion

4.2.1. Block 1 – saccades

4.2.1.1. Oculomotor capture. In Block 1 the oculomotor capture was defined as the percentage of participants that, on each trial, erroneously made the first saccade toward the distractor. We considered only saccades with an amplitude larger than 1° of visual angle, and whose starting position was within 1.5° from the central fixation point. If the endpoint of the saccade had an angular deviation of less than 15° of arc from the center of either the target or the distractor (i.e., if the saccadic endpoint was within a 30° cone which extended from fixation to the center of the stimulus, the saccade was classified as landed on that particular stimulus). To test whether the oculomotor capture decreased with practice, we divided the 15 distractor-present trials into three bins of five trials each. The results depicted in Fig. 6 showed that the percentage of oculomotor capture diminished as exposure to the distractor progressed (for similar results see, Bonetti & Turatto, 2019; Godijn & Kramer, 2008), a pattern substantiated by a repeated measure ANOVA with Bin (from 1 to 3) as factor, which resulted significant, $F(2, 46) = 7.854$, $p = .001$, $\eta_p^2 = .255$.

4.2.1.2. Saccade latency. The saccade latency analysis, conducted only on Block 1, was meant to detect any indirect effect of the distractor on the saccades correctly landing on the target location. Previous studies have indeed consistently shown that the latency of the saccades landing on the target are increased or decreased by the presence of a distractor appearing in a position near or far from the target (Bonetti & Turatto, 2019; Godijn & Theeuwes, 2002; Walker, Deubel, Schneider, & Findlay, 1997). To this aim, we measured the latency of the saccades directed toward the target. Trials in which the saccade latency was either shorter than 80 msec or longer than 800 msec were excluded from the analysis. Due to this outliers-detection criterion, less than 5% of the trials were discarded from the analyses. On average, in the first block the saccadic latencies were shorter in the distractor-absent trials (M = 326 msec) than in the distractor-present trials (M = 381 msec), $t(29) = -4.45$, $p < .001$, $d = -.909$ (see Fig. 7). In order to analyze the time course of the latency of the saccades directed toward the target, we divided the 15 distractor-present trials into three 5-trial bins, and we conducted a repeated measures ANOVA with Bin (from 1 to 3) as factor, which resulted significant, $F(2, 46) = 18.097$, $p < .001$, $\eta_p^2 = .440$ (see Fig. 7). This means that the latency of the saccades correctly landing on the target, when the distractor was

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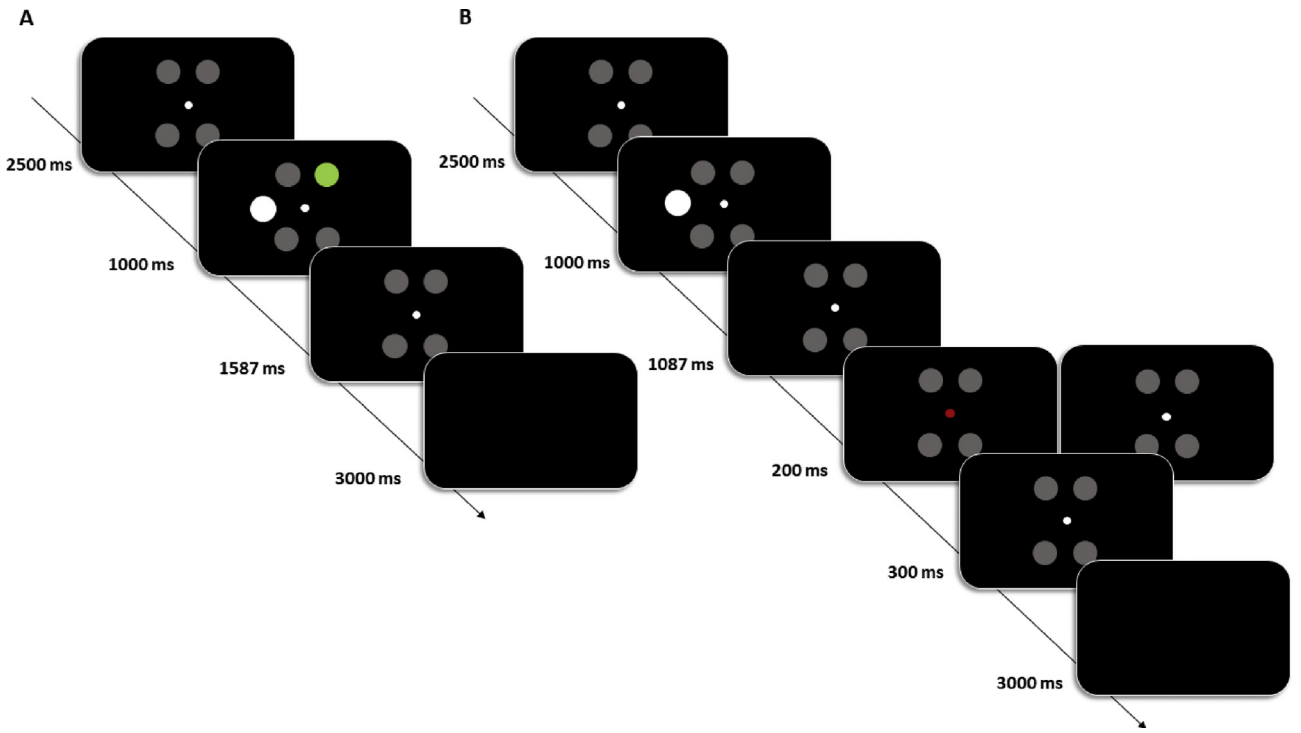

Fig. 5 – Example of stimuli used in Experiment 3. Panel A depicts the main events of Block 1. A grey central fixation point (central filled disk) was presented for 2500 msec, surrounded by four grey disks. After that, one of the grey disks turned to green for 1000 msec and served as saccadic target, while the others remained grey. At the same time, on 30% of trials an additional white disk was added to the display, in one of two possible locations (at clock positions 3 or 9), and served as distractor. The task was to make a saccade as fast and as accurate as possible toward the target stimulus. Panel B depicts the main events of Blocks 2–4, in which the trial sequence was similar, with some exceptions: the target stimulus never appeared (i.e., none of the grey disks turned to green); after the disappearance of the distractor the central fixation point could turn to red for 200 msec, or alternatively it could remain grey. On each block, the central fixation point turned to red in a variable number of trials (from five to eight). The task was to silently count the number of times in which the fixation point turned to red, and to report this number at the end of each block. In all blocks (Panels A and B), each trial was followed by a blank inter-trial interval of 3000 msec. The total duration of each trial was about 8 s.

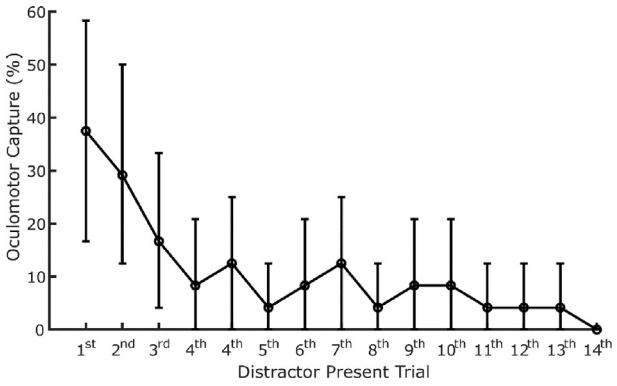

Fig. 6 – The figure shows the percentage of oculomotor capture triggered by the peripheral onset distractor, as a function of trial number, in the first block of Experiment 3. On the *x-axis*, only distractor present-trials are depicted, and each marker represents the amount of oculomotor capture in a single distractor-present trial (from the 1st to the 15th). Bars represent bootstrapped 95% confidence intervals.

presented, decreased significantly following the repeated exposure to the distractor. We will delve into the implication of this result in the *General discussion* section.

4.2.2. *Block 2–4 – microsaccades*

4.2.2.1. ACCURACY. The accuracy of participants in Blocks 2–4 was high (99% in Block 2, 99% in Block 3 and 97% in Block 4), thus confirming that they were focused on the counting task.

4.2.2.2. THE MAIN SEQUENCE. Fig. 2 (Panel C) shows that the linear relation between microsaccades amplitude and peak velocity was strongly positive, and this was confirmed by the correlation coefficient ($r = .91$).

4.2.2.3. ABSOLUTE FREQUENCY OF MICROSACCADES. Absolute frequency of microsaccades, is depicted in Fig. 8. Visual inspection of Fig. 8 reveals the typical inhibition-rebound microsaccadic pattern for both blocks, although there seems to be a general trend towards higher frequency in block 4. This was confirmed by a two-way ANOVA with Block (2 *vs* 4) and ROI (pre-stimulus *vs* peak inhibition, in this case 119–219 msec post-stimulus onset) as within-observers factors, yielded significant main effects of both ROI:

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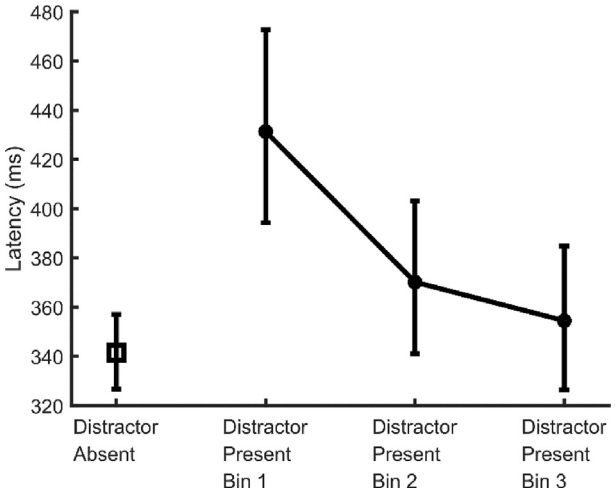

Fig. 7 – The figure shows the mean latency of saccades directed toward the target, as a function of the distractor presence/absence. The square marker depicts the mean saccadic latency when no distractor was presented (i.e., in the first 30 trials of Block 1), whereas the connected points depict the mean saccadic latency in the distractor-present trials, divided into three consecutive 5-trial bins. Bars represent bootstrapped 95% confidence intervals.

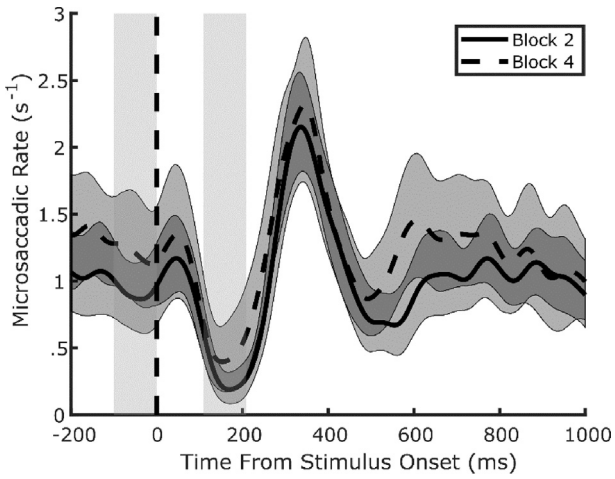

Fig. 8 – Time course of absolute microsaccadic frequency in response to the occurrence of the peripheral onset in Experiment 3 (Block 2 *vs* 4). The plots were created by convolving the frequency of microsaccades with a Gaussian filter of 25 msec SD. The *vertical dashed line* indicates the onset of the peripheral stimulus, whereas the *vertical grey areas* delimit the time windows (pre-stimulus baseline and maximum inhibition) which were used for statistical testing. *Shaded error areas* represent 95% inter-subject confidence intervals of the mean absolute microsaccadic frequency calculated through bootstrapping. Notice that in both blocks a clear inhibition of microsaccades is observed.

$F(1,23) = 33.895$, $p < .001$, $\eta_p^2 = .596$, and Block: $F(1,23) = 6.164$, $p = .021$, $\eta_p^2 = .211$, but no two-way interaction: $F(1,23) = 2.149$, $p = .156$, $\eta_p^2 = .085$. Once again, this indicates that, albeit within the context of a generally increased ms frequency, the inhibition of microsaccades is still equally strong at the end of the experiment.

5. General discussion

Despite exogenous saccades elicited by a peripheral onset distractor are subject to habituation, a key question is whether this form of plasticity takes place at the saccadic execution stage, or involves the earlier stage of saccadic programming. To address this issue, we exploited the fact that the programming of reflexive saccades triggered by a peripheral onset generates an initial phasic drop in the microsaccadic frequency (Engbert, 2006). Hence, because saccades and microsaccades are thought to arise from competing motor plans in the peripheral and central part of the SC motor map (e.g., Rolfs et al., 2008), if habituation of reflexive saccades occurs at the stage of saccadic programming then this should impact the rate of microsaccades. Specifically, any habituation (i.e., weakening) of the saccadic programming activity should be accompanied by a progressive attenuation of the initial microsaccadic inhibition.

The results confirmed that the reflexive saccadic response triggered by an irrelevant onset is subject to a rapid habituation (Bonetti & Turatto, 2019), whereas, quite surprisingly, the inhibitory microsaccadic response elicited by the same stimulus is not. Since there is compelling neurophysiological and psychophysical evidence to believe that the microsaccadic response is modulated by the saccadic programming activity (e.g., Engbert, 2006; Engbert & Kliegl, 2003; Rolfs et al., 2008), this suggests that habituation of reflexive saccades does not arise from changes at the oculomotor programming stage (Experiment 3), neither when the onset stimulus is relevant (Experiment 1), nor when it is irrelevant (Experiment 2).

That the reflexive saccadic and microsaccadic responses are differently affected by habituation is relevant for our understanding of the neural machinery underlying oculomotor control in humans. Indeed, irrespective of whether the oculomotor capture reduction is achieved by means of habituation mechanisms (Sokolov, 1963), or via a strategic top-down distractor inhibition mechanisms (Gaspelin et al., 2017; Gaspelin & Luck, 2018; Sawaki & Luck, 2010), the fact that the saccadic programming stage does not seem to be affected by the repeated exposure to the distractor is a key finding that challenges one of the main assumptions of the *competitive integration model* for oculomotor control (Godijn & Theeuwes, 2002; Kopecz, 1995; Trappenberg et al., 2001). Specifically, the model holds that the competition between different input signals (exogenous and endogenous) is integrated and resolved at the level of saccadic programming within the motor map in the SC. According to the model, any inhibitory modulation of unwanted exogenous saccades occurs in the SC map that generates the motor programs. Two inhibitory mechanisms are assumed to operate in the SC motor map: one, based on lateral-inhibition, rapidly reduces the exogenous activation elicited by a salient stimulus; the other, location specific, provides a direct inhibition on the spatial coordinates of a salient distractor, thus favoring the programming (and execution) of a saccade toward the target

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location. As a result of the inhibition of the distractor location in the saccade map, the mean vector of activity of the saccade directed toward the target would be shifted away from the distractor coordinates, which would cause a curvature in the opposite direction in the saccadic trajectory (Sheliga, Riggio, & Rizzolatti, 1994, 1995; Doyle & Walker, 2001; Godijn & Theeuwes, 2002; Van der Stigchel, 2010). However, on the reasonable assumption that the competitive integration model proposed by Rolfs et al. (2008) is correct, if distractor inhibition were implemented at the level of saccadic programming in the SC, then the suppressive effect of this programming activity on fixational activity, namely on microsaccades, should diminish progressively. Contrary to this prediction, we did not find any reduction in the microsaccadic inhibitory phase as a function of exposure to the onset distractor, a result that was quite unexpected regardless of whether one favors the habituation explanation, as we do, or the involvement of other types top-down inhibitory mechanisms. At present it appears difficult to reconcile our results with those of studies showing a curvature in the target saccades caused by the distractor, which are taken as evidence that the different input signals are integrated at the level of saccadic programming in the SC. One possibility to reconcile the two positions is to assume that also the saccadic curvature caused by a peripheral distractor arises from the inhibition of the corresponding oculomotor command in the brainstem SBG.

However, on the basis of our findings, we favor the hypothesis that the competition between exogenous and endogenous saccades is resolved at the stage of saccade execution in the brainstem SBG, which integrates signals from different neural structures (SC, FEFs, cerebellum) involved in the control of eye movements (Fuchs, Kaneko, & Scudder, 1985; Scudder et al., 2002). Hence, the type of oculomotor plasticity revealed by habituation (also see, Bonetti & Turatto, 2019; Godijn & Kramer, 2008) might have occurred downstream from the SC, likely in the brainstem SBG or in the cerebellum.

Two lines of evidence support this conclusion. First, when there is a competition between maintaining fixation and executing a saccade, two different activations emerge from the SC, a rostral one related to fixation, and a caudal one related to the movement of the eyes. However, such competition would not resolved at the programming stage within the SC, but rather at the execution stage in the brainstem SBG (Fuchs et al., 1985; Scudder et al., 2002). More specifically, the competition would arise between the omnipause neurons (OPNs), which maintain fixation, and long-lead burst neurons (LLBNs), which are active during a saccade. As put forward by Otero-Millan and colleagues "… the mutually inhibitory circuit between OPNs and LLBNs, driven by the SC, is a likely candidate for the mechanism that normally triggers and suppresses saccades and microsaccades" (Otero-Millan et al., 2011, p. 111). In a similar fashion, one could argue that also the competition between the execution of reflexive and voluntary saccades would not be resolved within the SC, but rather that it would involve a competitive interaction between different populations of LLBNs within the SBG. Therefore, habituation of oculomotor capture could be caused by a progressive decrease in the LLBNs neural activity controlling the reflexive saccade, whereas the LLBNs neural activity coding the saccade directed toward the target would remain unaltered. It also follows that as habituation of reflexive saccades develops, the competition between the two populations of LLBNs is resolved faster in favor of the saccade directed toward the target, a prediction in agreement with the decrease in the latency of endogenous saccades that we have documented (Experiment 3, Block 1; also see, Bonetti & Turatto, 2019).

The second line of evidence that supports our proposal comes from lesions and single-cell recording studies in monkeys, which strongly indicate that *saccade adaptation*, a form of oculomotor plasticity, takes place downstream from the SC (Melis & Van Gisbergen, 1996; Optican & Robinson, 1980). The standard saccade-adaptation paradigm consists in shifting the original position of a saccadic visual target while the saccade is being made, which typically results in an initial undershooting or overshooting of the saccade. However, as training progresses an adaptive process is observed, so that the saccade gain decreases or increases according to the inward or outward direction of the intrasaccadic target step. The fact that such saccadic adaptation is also found for saccades evoked via electrical stimulation of the SC, suggested that such corrective process occurs downstream from this structure, and specifically in the cerebellum, which sends its corrective signals to the brainstem SBG, where oculomotor programs coming from the SC and the FEFs are integrated (Dean, Mayhew, & Langdon, 1994; Melis & Van Gisbergen, 1996). Crucially, in agreement with this hypothesis, while the metric of the saccade changes during the adaptation paradigm, movement related activity in the SC is not modified, but continues to code for the initial target location (Frens & Van Opstal, 1997; Quessy, Quinet, & Freedman, 2010). Furthermore, that the adaptation process might occur downstream from the SC is also suggested by the observation that the effects of short-term saccadic adaptation on visually guided saccades transfers to saccades evoked through electric stimulation (Edelman & Goldberg, 2002). Thus, overall, neurophysiological studies in monkeys, and neuroimaging experiments in humans (Desmurget et al., 1998), concur in showing that the saccadic programming activity in the SC is not affected by saccadic adaptation (but see, Takeichi, Kaneko, & Fuchs, 2007). The SC thus seems to send unvarying commands to both the brainstem SBG and the cerebellum, where saccadic plasticity would take place (Dean et al., 1994; Optican & Robinson, 1980).

Although our results and interpretation fit nicely with this scenario, it should be made clear that our conclusion, namely that the suppression of reflexive saccades does not take place at the programming stage in the SC, rests on the assumption that the programming of saccades does affect the microsaccadic response, namely that the two types of oculomotor activities are functionally related. Hence, while arguments in favor of the existence of such functional links seem quite convincing (Engbert, 2006; Engbert & Kliegl, 2003; Rolfs et al., 2008), yet if this were not the case our conclusion would not be warranted. For example, it has been suggested that direct visual input to the OPNs is responsible for transiently inhibiting the production of microsaccades and for interrupting the execution of those that have already been triggered, resulting in abnormal ratios of peak velocity to microsaccade amplitude

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(Buonocore et al., 2017). Interestingly, the visual responses in OPNs are not simply mirroring the activity of fixation neurons in the SC (Everling, Paré, Dorris, & Munoz, 1998), but they might act as a somewhat independent "pause" system, separate from the SC "go" system. According to this hypothesis, habituation of saccadic programming could still take place in the SC, but it would not be visible in the microsaccadic inhibitory phase because the latter would be mediated by a "pause" system controlled directly by the OPNs. The obvious limitation of this explanation is that, as far as we know, there is no clear evidence of visual pathways that reach the OPNs completely bypassing higher-order structures such as the SC and the FEFs.

Finally, a further implication of our findings concerns the long-lasting debate about the relation between attention shifts and eye movements. According to one view, the two forms of orienting are tightly coupled but independent (e.g., Hunt & Kingstone, 2003b, 2003a; Posner & Petersen, 1990), with separate neural networks involved in the control of covert and overt attention shifts (Smith & Schenk, 2012). An opposite view is proposed by the influential *Premotor Theory of Attention* (PTA), which claims that spatial attention and eye movements are functionally equivalent, such that a shift of attention would result from the programming of an oculomotor command (Rizzolatti, Riggio, Dascola, & Umiltá, 1987), with covert and overt orienting also sharing common neural substrates (de Haan, Morgan, & Rorden, 2008; Moore & Fallah, 2001). However, several neurophysiological studies with monkeys performing visual search tasks have severely challenged the PTA by identifying in the FEFs populations of visual neurons whose activity reflects target selection processes, but not saccadic programming (e.g., Murthy, Thompson, & Schall, 2001; Thompson & Bichot, 2005; Thompson, Biscoe, & Sato, 2005). This distinction has been confirmed by anatomical studies showing pools of neurons in the FEFs projecting to extrastriate visual cortical areas, whereas different ones that project to the SC (e.g., Pouget et al., 2009). In agreement with this view, we note that the exogenous orienting of attention elicited by both a peripheral onset and a feature singleton is subject to habituation (De Tommaso & Turatto, 2019; Pascucci & Turatto, 2015; Turatto, Bonetti, Chiandetti, & Pascucci, 2019; Turatto, Bonetti, & Pascucci, 2018; Turatto, Bonetti, Pascucci, & Chelazzi, 2018; Turatto & Pascucci, 2016; also see; Codispoti, De Cesarei, Biondi, & Ferrari, 2016), whereas the programming of a reflexive saccade toward a repetitive onset does not seem to be affected by habituation. Therefore, this distinct pattern of results also challenges the main tenant of the PTA, namely that the programming of a saccade is equivalent to the orienting of attention, otherwise both should be equally subject to habituation. Hence, when the different results emerging from experiments on habituation of attention and microsaccades are considered altogether, they support the view according to which eye movements programming and attention shifts are independent functions controlled by independent mechanisms (e.g., Smith & Schenk, 2012; Thompson et al., 2005).

To conclude, the microsaccadic activity that we recorded in response to a recurrent peripheral onset suggests that habituation of reflexive saccades, or any other suppressive signal that reduces oculomotor capture, does not take place at the oculomotor programming stage in the SC; on the contrary, and in agreement with neurophysiological studies on short-term saccadic adaptation, this form of plasticity is likely to be expressed downstream from the SC.

Open practices

The study in this article earned Open Materials and Preregistered badges fortransparent practices. Materials and data for the study are available at https://zenodo.org/record/3799054#.XrKtmagzbic.

Acknowledgments

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Data, materials, and code of analyses, have been archived in the following public repository: https://zenodo.org/record/3799054#.XrKtmagzbic.

No part of the study procedure and analyses was preregistered prior to the research being conducted.

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