Beyond the point of no return

Beyond the point of no return: effects of visual distractors on saccade amplitude and velocity

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Abstract

Visual transients, such as a bright flash, reduce the proportion of saccades executed around 60-125 ms after flash onset, a phenomenon known as *saccadic inhibition*. Across three experiments, we apply a similar time-course analysis to the amplitudes and velocities of saccades. Alongside the expected reduction of saccade frequency in the key time period, we report two perturbations of the "main sequence", one before and one after the period of saccadic inhibition. First, saccades launched between 30 to 70 ms following the flash were hypometric, with peak speed exceeding that expected for a saccade of similar amplitude. This finding was in contrast to the common idea that saccades have passed a "point-of-no-return" around 60 ms prior to launching, escaping interference from distractors. The early hypometric saccades observed were not a consequence of spatial averaging between target and distractor locations, as they were found not only following a localized central flash (Experiment 1), but also following a spatially generalized flash (Experiment 2). Second, across experiments, saccades launched at 110 ms post-flash, toward the end of saccadic inhibition, had normal amplitude but a peak speed higher than expected for that amplitude suggesting increased collicular excitation at the time of launching. Overall, the results show that saccades that escape inhibition following a visual transient are not necessarily unaffected, but instead can reveal interference in spatial and kinematic measures.

Keywords: eye movements, saccadic inhibition, main sequence

Introduction

The main challenge for the oculomotor system in a complex environment is to select when and where to move the eyes in order to land near targets of interest. Saccadic amplitudes range from only a few minutes of arc to over 80° of visual angle, and the kinematics of saccadic performance are generally invariant across tasks and people. Perhaps the best example of this regularity is the lawful monotonic relationship between saccadic amplitude and peak speed, called the *main sequence* (Bahill et al. 1975; Collewijn et al. 1988), which holds up to ~60° of visual angle (at which the peak speed saturates at ~500°/s). Another stereotyped feature of the saccadic system is how it responds to sudden transient events. Reingold and Stampe (1999, 2000, 2003, 2004) used a highly salient flashed distractor, and revealed a characteristic "dip" in saccadic frequency beginning as early as 60-70 ms after the flash, with maximal depression around 90 ms, rebounding to normal levels by 120-130 ms. This *saccadic inhibition* (SI) generalized beyond the text-reading and scene-exploration tasks first tested, with distractors having similar effects in gap, overlap, pro-saccade and anti-saccade tasks (Reingold and Stampe 2002). Interestingly, an analogous effect has been shown for endogenously triggered micro-saccades. "Micro-saccadic inhibition" describes a similar dip in micro-saccade rate, about 100 ms after the presentation of a visual cue (Engbert and Kliegl 2003; Hafed and Clark 2002). As for standard SI, this effect was shown to occur with any sensory transient presented during saccadic planning, supporting the idea of generalized inhibitory mechanisms in the oculomotor system (Hafed and Ignashchenkova 2013).

An interesting question concerns the time window during which a saccadic plan is susceptible to interference. The most commonly accepted estimates of *when* a saccade plan can still be modulated have come from double-step tasks, in which

participants have to saccade toward a target that sometimes jumps to a second location after initial presentation. The amplitude of the first saccade varies as a function of the delay between the target jump and the onset of the first saccade. When this delay is short, the eye movement will land at the first target location, but for longer delays the saccade will land at the second target. For intermediate delays, the saccade tends to land in between the two locations (Becker and Jürgens 1979). This amplitude transition function can be used to determine the "point of no return" at which the new input can be no longer affect the motor plan and the saccade will not change its destination, and it is defined by the transition point for the first deviations from the first target position (i.e. the earliest sign of any influence of the second target). This time interval was interpreted as the delay between the afferent signal reaching the first oculomotor structures and the triggering of the eye movement signal to the muscles (Becker 1991).

The period between this point and saccade onset, which Ludwig et al (2007) called "saccadic dead time" (SDT), has been estimated to be as brief as 60 ms (Findlay and Harris 1984; Ludwig et al. 2007). At first, it was suggested that the SDT was a constant value, about 70 ms, similar across different eye movement tasks (Beutter et al 2003; Findlay and Harris 1984; Hooge et al 1996; Ludwig et al 2005; Van Loon et al 2002) and it also represented a critical parameter for models of eye movements (Nuthmann et al 2010; Reichle et al 1998; Van Loo et al 2002). More recently, this notion was challenged by Ludwig et al (2007) by showing that even if the SDT was not influenced by variations in saccadic reaction times it was susceptible to manipulations of the spatial configuration of the two targets. Similarly, Walshe and Nuthmann (2015) showed that the SDT was affected by the type of background used during double-step tasks, approaching a minimum value of 70 ms for uniform scenes

(black background). Nonetheless, the lower limit reported in behavioural studies has not been lower than the 60 ms estimated by Ludwig et al (2007). The onset of SI, 60-70 ms after a visual flash, is thus compatible with the concept of saccadic dead time, implying a generalized temporal boundary before saccadic execution, during which new visual changes, either relevant (double-step) or irrelevant (distractors), cannot influence the impending saccade.

Across multiple studies of SI, Reingold and Stampe reported consistent changes in the timing of saccades, accounting for the SI dip profile, but they did not report any spatial or kinematic changes in the saccades that were launched. More recently, however, there have been clear indications that SI does have some influence on the spatial aspect of saccadic behavior (Buonocore and McIntosh 2012; Edelman and Xu 2009; Guillaume, 2012). Specifically, saccades launched during the period immediately preceding or following the SI dip, induced by a contralateral distractor or a mask covering a large part of the screen and target, have been found to be hypometric (falling short of the target) (Edelman and Xu 2009; Guillaume, 2012). These observations may echo findings made in studies of micro-saccades (Hafed and Ignashchenkova 2013; Rolfs et al. 2008). For example, Hafed and Ignashchenkova (2013) reported that the micro-saccadic rate was not only reduced 100 ms after a supplementary stimulus, but that the spatial character of the persisting micro-saccades was sensitive to the location of that stimulus. Their interpretation was that the observed micro-saccades reflected an instantaneous “read out” of activations in the oculomotor maps of the superior colliculus, affected both by the target and the supplementary stimulus.

Recent literature thus suggests that SI might not be exclusively temporal in nature but may also involve changes in the kinematic and spatial aspects of the

118 saccade. However, aside from the work of Guillaume (2012) and some observations
119 made by Edelman and Xu (2009), no other studies have made a detailed analysis of
120 the time-course of such parameters following distractors with different characteristics,
121 in a way that is analogous to what has been done for the temporal domain.

122 In the present paper we adopted precisely this strategy. In addition to a
123 standard SI analysis, we applied a time-course analysis to study the gain and peak
124 speed of saccades launched at different times following a visual flash. First, we
125 applied this novel analysis to a previously collected dataset (from an unpublished
126 experiment that incorporated SI within a visual discrimination task) that was well
127 suited to this exploration. This exploratory Experiment 1 confirmed that SI could be
128 associated with a modulation of saccadic gain, following a central flash not dissimilar
129 to the transient mask that Guillame (2012) found to affect saccadic amplitude. We
130 followed up this preliminary observation with two experiments designed to more
131 finely measure the subtle changes in the saccade characteristics. In Experiment 2, we
132 used a generalized flash located in the top and bottom of the screen to exclude the
133 possibility that the amplitude effects were related specifically to the spatially localized
134 nature of the central flash. In Experiment 3, we manipulated distractor location, to be
135 more or less eccentric than the target, to test whether saccade hypometria was
136 dependent upon distractor location, as has been suggested for micro-saccades (Hafed
137 and Ignashchenkova 2013), or resulted from a more general inhibitory phenomenon.
138 Across these three experiments, we report a complex interplay between spatial and
139 temporal modulations for distractors interfering at different stages of saccade
140 programming and execution, including during the commonly accepted “saccadic dead
141 time” that is thought to occur after the saccade plan passes a point of no return.
142

Method

Participants

Nine (Experiment 1), ten (Experiment 2) and eight (Experiment 3) volunteers aged between 18 and 30 years participated. All were free from neurological and visual impairments. The experiment was conducted in accordance with the 1964 Declaration of Helsinki, and the guidelines of the University of Trento Research Ethics Committee for behavioral experiments. All participants gave informed written consent and received €7 per testing hour, or course credits.

Apparatus, stimuli and procedure

Stimuli were presented on a 17-inch CRT monitor (1024 x 768 pixels) at 85 Hz (Experiment 1) or 100 Hz (Experiment 2 and 3). In all the experiments, participants were seated with their head resting on a chin and forehead rest in order to reduce head movements. The eyes were horizontally and vertically aligned with the center of the screen at a distance of 60 cm. Eye movements were recorded with the EyeLink 1000 system (detection algorithm: pupil and corneal reflex; 1000 Hz sampling; saccade detection was based on a 30 deg/s velocity and 9500 deg/s^2 acceleration thresholds; maximum head movement[1] tolerance equal to 25 mm by 25 mm by 10 mm - horizontal by vertical by depth respectively). In all three experiments, a five point-calibration on the horizontal and vertical axes was run at the beginning of each session and after three consecutive trial blocks. Additional calibrations were added if the participant moved their head from the chinrest. In all the experiments the background was grey (23.5 cd/m^2). The experimenter started each trial with a drift

correction, after which a tone accompanied the onset of a 0.50° central fixation cross (124 cd/m^2).

In *Experiment 1* (Figure 1A), after a random interval varying between 500 to 1200 ms, a red dot (0.5°, 28.2 cd/m^2) was displayed at 10° of eccentricity, equally often to the right or to the left of fixation. Participants were required to make a saccade to this target as soon as it appeared. Independent of that requirement, in half of the trials, a black square was flashed for 11.7 ms at the center of the display; this square was the "flash" stimulus used to elicit SI in this experiment (see below). Saccadic reaction times (SRT) were recorded as the interval between target onset and the start of the saccade. This first experiment was originally designed for a different purpose and incorporated a perceptual task whereby four Gabor patches (size = 6°; frequency = 0.9 cycle/degree) were presented in the four corners of the monitor for 12 ms, 105 ms after flash (or invisible flash in target only condition) onset. On half of the trials, the four stimuli had the same orientation (vertical or horizontal) and on the other half, one of them had a different orientation. At the end of the trial, participants were asked to report if all the Gabor patches were the same or if one was different. This perceptual element of Experiment 1 is not relevant for present purposes and the results of the perceptual task were analyzed separately in a different unpublished manuscript focusing on saccadic suppression. Critically, the present analyses were restricted to trials in which saccades were launched up to 45 ms after the display of the Gabor patches. Thus, the presence of the perceptual targets was not likely to influence the pattern of results. No perceptual targets were present in the other two experiments reported here.

Participants performed a preliminary block of 64 target-only trials, half with the target on the right and half with the target on the left side of the screen. The

192 median SRT from the last 50 of these trials provided an estimate of the expected SRT
193 for that participant for the experimental blocks. In the experimental blocks, target-
194 only (no-flash) trials were intermingled equally with target plus distractor (flash)
195 trials, in which, in addition to the target, the black square (3.5°, 2.3 cd/m^2) was
196 flashed at the center of the screen for 11.7 ms. The onset of this central flash varied
197 randomly between ~117 ms before to 11.7 ms after the expected SRT for that
198 participant in steps of 11.7 ms, thereby providing a wide range of distractor delays.
199 Each of the two conditions (flash, no flash) occurred 64 times per block, shuffled
200 randomly. Each participant completed two sessions of eight experimental blocks, on
201 different days, for a total of 1024 trials. Although originally conceived for a different
202 purpose, the experimental design described above provided a rich dataset for an
203 opportunistic exploration of the time-course analysis of saccade kinematics, and
204 provided the basic template for the two experiments subsequently designed to further
205 investigate these issues (but which did not include the perceptual task, and utilized
206 different distractor locations).

207 Experiment 2 (Figure 1B) was designed to replicate and extend the
208 observations of the first experiment. The saccadic task was similar to that of
209 Experiment 1 but changes were introduced to sample a wide range of distractor delays
210 relative to expected saccade onset and to optimize the effect of the visual transient
211 upon the oculomotor response. No perceptual task was presented in either the second
212 or third experiment. After a random interval varying between 500 to 1200 ms, a white
213 dot (0.5°, 124 cd/m^2) was displayed at three possible eccentricities (4°, 8° and 12°
214 degrees of visual angle) with equal probability to the right or to the left of the visual
215 field. As in Experiment 1, each participant performed a preliminary block of 60
216 target-only trials (20 for each eccentricity) to determine the median SRT from which

to calculate flash onset. In the experimental blocks, target-only (no-flash) trials were intermingled with target plus distractor (flash) trials. The distractor consisted of two white rectangles (width: ~33°, length: ~8.5°, 124 cd/m^2) covering one third of the top and bottom of the screen (see: Reingold and Stampe (2002) for a similar procedure). The flash was presented for 20 ms. During the course of the experiment, flash onset was varied around the participant-specific median SRT by randomly subtracting one of six possible SOAs, spanning from 20 to 120 ms in steps of 20 ms. In each trial we recorded the SRT and, at the end of the trial, calculated flash-to-saccade delay for that trial by subtracting flash onset from the current SRT. To ensure adequate sampling of saccades in each time bin after flash onset (bin size 20 ms), we kept track of the number of saccades recorded within each time bin and, when any bin reached a threshold of 60 observations, replaced the SOA most closely matching that flash-to-saccade delay with the SOA of the least represented bin. At the end of each block, the median SRT, used to calculate flash onset, was updated with the median of the current block. Overall, we ran 260 trials per condition, i.e. two flash (absent-present) conditions by three target eccentricities (4°, 8° and 12° of visual angle) for a total of 1560 trials. Participants completed two sessions on different days in which the 780 trials were divided in 13 blocks of 60 trials each.

Experiment 3 had a similar procedure to Experiment 2 but only one target eccentricity was used (10° of visual angle) (Figure 1C). As in the other experiments, each participant performed a preliminary block of 30 target-only trials to determine the median SRT from which to calculate flash onset. In the experimental blocks, target-only (no-flash) trials were intermingled with target plus distractor (flash) trials. In distractor trials, the flash was presented for 20 ms and consisted of a white vertical rectangle (width: ~2°, length: ~24°, 124 cd/m^2) either less eccentric (flash-, 6°) or

more eccentric (flash+, 14°) than the target (10°). Flash onset was varied around the per-participant median SRT, which was updated after each block, by randomly subtracting one of four possible SOAs spanning from 30 to 60 ms in steps of 10 ms. This procedure generated a high density distribution within the first 130 ms after flash onset allowing us to strengthen the analysis of amplitude and velocity variations. Overall, we ran 20 trials per flash condition (flash absent, flash- and flash+) for a total of 60 trials per block. Participants completed two sessions for eight blocks in one day for a total of 960 trials, with 320 trials per flash condition.

Data screening

We excluded saccades with latencies of less than 70 ms (Experiment 1: ~1.2%; Experiment 2: ~2.5%; Experiment 3: ~0.73%) or of more than 500 ms (Experiment 1: ~2.7%; Experiment 2: ~0.14%; Experiment 3: ~0.75%). We also removed saccades with an amplitude less than 1° amplitude (Experiment 1: ~0.6%; Experiment 2: 3.4%; Experiment 3: ~2.10%) and saccades made in the wrong direction (Experiment 1: ~0.02%; Experiment 2: ~2.4%; Experiment 3: ~0.05%). In Experiment 2 and 3 we also excluded 2.6% and 1.52% of saccades, respectively, due to blinks.

Analysis of saccadic inhibition

In all the Experiments, we performed an analysis of the SRT distributions for all valid trials by following Bompas and Sumner's (2011) procedure to calculate the "dip" ratio. As a first step, we recoded SRTs relative to flash onset, by subtracting from each SRT the SOA between target and flash. Then, for each participant and condition (Experiment 1: no-flash and flash; Experiment 2 no-flash and flash at each target eccentricity; Experiment 3: no-flash, flash- and flash+), we created percentage

frequency histograms (bin width 4 ms) that were then lightly smoothed using a Gaussian kernel with 24 ms window and 2 ms SD. The smoothed histograms were interpolated to obtain 1 ms precision. To estimate the level of SI, we computed the proportional change for each point in time in the flash distribution relative to the no-flash distribution by using the formula: (no-flash - flash)/no-flash. This operation was performed on both the no-flash and flash condition. In the no-flash condition an "invisible" stimulus was presented using the same time procedure as for the flash condition (for detailed analysis on this procedure see: McIntosh and Buonocore 2014). The magnitude (i.e. maximum of inhibition) and the latency (time to the maximum) of SI were taken in the first 150 ms after flash onset. To visualize the average SI profile, per condition, the individual profiles were then averaged across participants and the 95% confidence interval was computed at each time point (Figure 3A, D and G). Statistical analysis was performed on the individual parameters extracted from each SI profile across the three Experiments are reported in Table 1, 2 and 3.

Analysis of saccadic kinematics

The analysis of saccade kinematics focused upon saccadic gain and normalized peak speed. The first step was to extract these variables for every trial. Saccadic gain is saccade amplitude divided by the target amplitude for that trial, with values greater than one indicating overshoot (hypermetria), and values less than one indicating hypometria (undershoot). Normalized peak speed was the *observed* peak speed divided by the peak speed *predicted* from the observed saccade amplitude on that trial, with values bigger than one indicating a speed higher than expected, and values less than one indicating a speed lower than expected. The calculation of normalized

peak speed therefore included an additional initial step to predict peak speed from the main sequence relationship between saccade amplitude and peak speed. To do so, for each participant separately, we fitted a polynomial function to the *observed* peak speed over the *observed* saccadic amplitude in all no-flash trials, and extracted the polynomial for the best fit according to a least-squares procedure. In Experiment 1 and Experiment 3, there was only one target location, and the spread of observed saccade amplitudes was too small (~2°) to model the entire main sequence function, so we used a 1st order polynomial function. In Experiment 2, we made use of all the eccentricities to estimate the best main sequence fit using a 2nd order polynomial function. In Figure 2 we show one example of fitting for each experiment (panel A, B and C) along with the R^2 for each participant in all of the experiments. Based on these individual fit parameters, we derived the *predicted* peak speed from the observed saccade amplitude in each trial, and used this value to normalize the observed peak speed for that trial.

We then analyzed the time-course of these kinematic variables relative to the flash event inducing SI. For each participant, RTs were binned using a bin-width of 20 ms and the mean saccadic gain and normalized peak speed was calculated for saccades launched within each time bin. For Experiments 1 and 3, the means were entered into separate two (flash: no-flash vs. flash) by seven (bin: 10 to 130 in 20 ms intervals) repeated-measures ANOVAs. In Experiment 2, a two (flash: no-flash vs. flash) by three (eccentricity: 4°, 8° and 12°) by seven (bin: 10 to 130 in 20 ms intervals) repeated-measures ANOVA was performed, with Greenhouse-Geisser adjustments to the degrees of freedom where sphericity was violated. Significant interactions were followed up by a series of paired samples t-tests comparing no-flash versus flash conditions at each time bin. Considering that adjacent time bins are likely

to be correlated, we performed the Benjamini & Hochberg (1995) and the Benjamini & Yekutieli (2001) procedure controlling the false discovery rate (FDR) of a family of hypothesis tests. Corrected p-levels are reported in the text.

Results

Experiment 1 - Analysis of saccadic inhibition

Overall, we confirmed the main SI effect by showing a strong bimodality in the flash histogram, with the lowest saccadic frequency happening around 90 ms after flash onset. For illustrative purposes, Figure 3A shows the average SI profile across participants, expressed as the ratio of inhibited saccades (i.e. delayed) to baseline saccadic frequency for the no-flash condition (see Methods section for details of the SI profile calculation). Using the parameters extracted from the individual SI profiles, we estimated that an average maximum of 78% of saccades were inhibited at 85 ms after the flash onset, matching well with the timing of SI and micro-saccade inhibition found in previous experiments (Bompas and Sumner 2011; Buonocore and McIntosh 2008, 2012, 2013; Edelman and Xu 2009; Guillaume et al. 2012; Hafed and Ignashchenkova 2013; Reingold and Stampe 2002). Individual values for the latency and the magnitude of inhibition were consistent across participants (Table 1).

Experiment 1 - Analysis of saccadic kinematics

For the gain, the repeated-measures ANOVA revealed a main effect of Flash [$F(1,8) = 12.96$; $p < 0.01$] and Bin [$F(2.25,17.99) = 8.29$; $p < 0.005$] but more interestingly, there was a significant interaction between the two factors [$F(1.6,12.82) = 8.99$; $p < 0.005$] (Figure 3B). During flash trials we observed a strong decrease in saccadic

amplitude (hypometria) for saccades launched 20 to 80 ms after flash onset (30 ms bin: [$t(8) = 6.29$; $p < 0.002$]; 50 ms bin [$t(8) = 3.92$; $p < 0.016$]; 70 ms bin [$t(8) = 3.35$; $p < 0.023$]). In order to estimate a possible violation of the main sequence, we analyzed the time-course of the normalized peak speed. We report a significant main effect of Flash [$F(1,8) = 10.00$; $p < 0.01$] and Bin [$F(6,48) = 4.03$; $p < 0.005$] but no interaction between these factors [$F(3.22,25.78) = 2.21$; $p = 0.1$]. The data suggest a general disturbance of the main sequence during flash trials, with peak speed exceeding the value predicted from saccadic amplitude. Looking at Figure 3C, there is an indication that the violation might be concentrated in a few specific time-points after flash onset, during the pre- and post- inhibitory period, as observed by Guillaume (2012). Nonetheless, while these data are suggestive, we were unable to confirm a significant temporal modulation of the main sequence. However, it should be noted that the above was an opportunistic and exploratory analysis of a dataset collected for different reasons. Experiments 2 and 3 directly investigated these trends with more targeted studies that were designed to have greater power to investigate the kinematic changes suggested by Experiment 1.

Experiment 1 - Interim discussion

Taken together, the modulations in saccadic gain and normalized peak speed suggest a general violation of the main sequence. First, a strong saccadic hypometria was observed for saccades launched between 30 (-0.48°) and 70 ms (-1.20°) after flash onset, which was not accompanied by a proportional reduction in peak speed. This pattern of data suggests that saccades launched during this period may have initially been programmed for greater amplitudes, but terminated in-flight following arrival of the distractor signal (Edelman and Xu 2009; Guillaume 2012; Munoz et al. 1996).

There were subsequent, albeit weaker, indications of a second violation toward the end of SI, where the peak velocity tended to exceed that predicted from the main sequence. Considering that the average no-flash SRTs were ~220 ms for this task, saccades launched 130-150 ms after flash onset corresponded to flash stimuli presented only 70 - 90 ms after target onset, thus relatively close in time to the target onset. The presentation of the flash may thus have summed with the build-up of target-related activity, generating an overall increase in the level of SC activation. At the time of saccade launching, this increased activity might have translated as increased velocity.

The exploratory analysis reported above brought up an intriguing pattern of modulations following distractor interference that confirmed and expanded previous reports of spatial and temporal effects (Edelman and Xu 2009; Buonocore and McIntosh 2012; Guillaume (2012). Nonetheless, while reduced saccadic gain was clear during the pre-inhibitory period (Edelman and Xu 2009; Guillaume 2012), the pattern of elevation of normalized peak speed was not so tightly locked to a particular time period; a more powerful experiment may be required to determine these patterns of kinematic variation more definitively. Moreover, in the present experiment we used as the distractor a single, highly localized and central flash that might have interfered with saccadic amplitude during target selection because it was partially interfering with the saccade trajectory, similarly to the mask stimuli used by Guillaume (2012). Instead of causing general inhibition, this less eccentric distractor might have induced smaller saccadic amplitudes via spatial interference, offering an alternative account of the observed hypometria. This could be analogous to observations of micro-saccadic inhibition, whereby the target-flash configuration was found to determine the pattern of amplitudes changes (Hafed and Ignashchenkova 2013; Rolfs et al. 2008).

Thus, to more closely measure the possible violations of the main sequence found in this preliminary dataset, we designed a further experiment to test whether these patterns were robust. First, we increased the power to detect small variations by substantially increasing the number of trials. Second, the timing of the flash was more finely tuned online to each participant's saccadic performance in order to elicit a strong SI in every participant. Third, to minimize the possibility of a direct spatial interference of the distractor as a competing saccadic target, the flash was more spatially generalized across the display, occupying both the top and bottom thirds of the screen (see Reingold and Stampe, 2002). Finally, we extended the range of target eccentricities to better map the main sequence function.

Experiment 2 - Analysis of saccadic inhibition

The parameters extracted from the individual SI profiles (Table 2) were closely similar across the three eccentricities and the maximum inhibition was about 74, 74 and 77 percent for the three eccentricities respectively with a latency of 78, 79 and 77 ms after the flash onset, matching the data from Experiment 1. Neither the magnitude nor the latency of inhibition were significantly different between the three eccentricities [magnitude: $F(2, 18) < 1$; N.S.; latency: $F(1.13, 10.15) < 1$; N.S]. For descriptive purposes, in Figure 3D we report the average profile across the three eccentricities.

Experiment 2 - Analysis of saccadic kinematics

By using the gain as measure of saccadic spatial performance, we found a significant main effect of Flash [$F(1,9) = 13.13$; $p < 0.006$] and Bin [$F(1.943, 17.488) = 5.64$; $p < 0.01$] and again a significant interaction between the two factors [$F(2.361, 21.245) =$

5.6; $p < 0.008$] (Figure 3E). There was no effect of eccentricity, suggesting that these modulations were similar across a range of saccadic amplitudes. The gain was reduced for saccades launched at 30 ms after flash onset [$t(9) = 3.41$; $p < 0.027$] with a minimum value for saccades launched at 50 ms after flash onset [$t(9) = 5.39$; $p < 0.003$], replicating the finding of Experiment 1. To check if these modulations violated the main sequence, we inspected the normalized peak speed. We report a significant interaction between Flash and Bin [$F(6,54) = 4.65$; $p < 0.001$] (Figure 3F). Pair-wise t-test comparisons confirmed a violation exceeding the expected peak speed for saccades launched at 30 ms [$t(9) = 3.41$; $p < 0.0273$]. More anomalously, there was a significant *reduction* in normalized peak speed for saccades launched at 50 ms after the distractor [$t(9) = 4.28$; $p < 0.014$]. Overall, the data from Experiment 2 confirmed and extended the results reported in Experiment 1. We replicated saccadic hypometria during the pre-inhibitory period (Edelman and Xu 2009; Guillaume 2012), associated with a violation of the main sequence and confirmed that this main sequence violation was specific in time. We again saw a qualitative trend toward a second, later rise in the main sequence ratio during the post-inhibitory period, although this trend did not reach statistical significance.

Experiment 2 - Interim discussion

The data from Experiment 2 confirmed that saccades launched during the pre-inhibitory period were truncated in flight, perturbing the main sequence (Edelman and Xu 2009; Guillaume 2012). Additionally, and surprisingly, we also observed a reduction of the normalized peak speed just before the start of inhibition. This finding was unexpected and, at present, we do not have a firm explanation for it. One possibility is that, on entering into the inhibitory period, when the interference is

maximal and the reduction in gain is peaking, saccades may be truncated even *before* achieving peak speed, consequently decreasing the ratio between the predicted and the observed velocities. This would predict that saccades launched in this time period would be associated with a reduced duration, since the truncation would happen so early. To explore this idea, we ran an analysis of saccadic duration, and confirmed a significant reduction specifically for saccades launched at 50 ms after the distractor [$t(9) = 3.58$; $p < 0.0417$], thus coincident with the reduced peak speed. Nonetheless, since this pattern of reduced peak speed was not evident in Experiment 1, more studies are needed to rule out the possibility that this observation was just a chance finding. Finally, we again saw indications, albeit relatively weak, of violations of the main sequence during the post-inhibitory period.

In Experiment 1, we considered that one possible explanation for the reduction in saccadic gain was that the flash-related activation may have interfered directly with the planning of the saccade trajectory; that is, a spatial averaging effect. In Experiment 2, this issue was addressed by placing the flash in the top and bottom third of the screen (Reingold and Stampe, 2002). Nonetheless, one could argue that the "center of gravity" of the flash configuration was still at the center of the screen; according to the micro-saccade inhibition literature, the final read out of the superior colliculus activation after flash presentation could be skewed toward the screen center, predicting hypometria by spatial averaging.

To better test the possibility of a spatial averaging effect we ran Experiment 3 in which the position of the flash relative to the target was either less (flash-) or more (flash+) eccentric than the saccade target. If the hypometria was generated by a general truncation mechanism, we should see the hypometria for both the less and more eccentric flash. On the other hand, if the effect is driven by flash location we

467 should record hypometria for the less eccentric flash and hypermetria for the more
468 eccentric flash.

469 Although Edelman and Xu (2009) tested the effect of distractor location on SI,
470 reporting that flashes appearing at the location of the saccade goal led to "express-
471 like" saccades, rather than SI, no prior study (cf. Guillaume, 2012) has systematically
472 investigated the effect of the flash location relative to saccadic target upon saccadic
473 amplitude and peak speed, leaving this important issue open.

474

475 *Experiment 3 - Analysis of saccadic inhibition*

476 The SI profile in the flash+ condition was smaller compared to the flash- condition
477 (Figure 3G). The analysis performed on the parameters extracted from the individual
478 profiles showed that the maximum inhibition was about 52% (flash+) and 83% (flash-
479) [$t(7) = 6.71$; $p < 0.0005$] with a latency of 74 and 71 ms respectively after the flash
480 onset [$t(7) = 1.26$; N.S.]. Individual parameters for the two conditions are reported in
481 Table 3. These data imply that the eccentricity of the flash, relative to the target, has a
482 strong impact on the level of saccadic inhibition, an interesting observation that has
483 been little explored in prior studies.

484

485 *Experiment 3 - Analysis of saccadic kinematics*

486 For gain, the main effect of Flash was reliable [$F(2,14) = 47.92$; $p < 0.0001$] as was
487 the main effect of Bin [$F(6,42) = 5.12$; $p < 0.001$]. More importantly, as in
488 Experiments 1 and 2, there was a significant interaction between the two factors
489 [$F(12,84) = 16.67$; $p < 0.0001$] (Figure 3H). Follow-up analyses of the Flash by Bin
490 interaction replicated the strong hypometria effect but with different timings for the
491 two conditions. In the flash- condition, the hypometria started for saccades launched

30 ms after flash onset and numerically peaked for saccades launched at 50 and 70 ms after flash onset [Bin 30: $t(7) = 5.30$; $p < 0.0039$; Bin 50: $t(7) = 7.60$; $p < 0.0009$; Bin 70: $t(7) = 4.19$; $p < 0.0095$] (as for Experiment 1 and 2). On the other hand, hypometria was observed, but started much later in the flash+ condition, peaking for saccades launched between 110 to 130 ms after flash onset [Bin 110: $t(7) = 7.1178$; $p < 0.0013$; Bin 130: $t(7) = 5.5058$; $p < 0.0032$]. These pronounced differences of timing allow the possibility that the two types of hypometria might have different origins.

As for the other experiments, the reductions in gain were accompanied by violations of the main sequence. We report a significant main effect of Bin [$F(6,42) = 7.63$; $p < 0.001$] and a significant interaction between Flash and Bin [$F(12,84) = 5.51$; $p < 0.0001$] (Figure 3I). In particular, the violation was present for saccades launched at 30 ms in the Flash- condition [$t(7) = 3.92$; $p < 0.020$] and followed by violations in the post-inhibitory period at 110 ms and 130 ms [$t(7) = 3.03$; $p < 0.044$, $t(7) = 5.79$; $p < 0.005$]. The flash+ condition had only one significant violation point during the post-inhibitory period, at 130 ms [$t(7) = 3.9773$; $p < 0.0374$]. We did not see in any of the conditions a reverse in the violation, as observed in Experiment 2. Nonetheless, looking at the bottom row of Figure 3 it is suggestive that for all the experiments the shape of the normalized peak speed oscillated compared to the steady baseline condition, with higher or lower values alternating within the total time-course. Thus, although the most consistent statistical pattern is for distractor-induced reductions in saccadic gain, with violations of the main sequence in a positive direction (i.e. increased peak speed to amplitude ratios), the qualitative pattern emphasizes that the perturbations of the main sequence may be somewhat unstable in direction as well as degree.

Overall, this pattern of results suggests that the variations in amplitude might be first driven by a truncation mechanism followed by a readout of the superior colliculus map, similarly to what has been reported in the micro-saccade literature (Hafed and Ignashchenkova 2013). The flash- condition showed a clear truncation (hypometria accompanied by relatively high peak speed) stopping the saccade in-flight for motor programs launched 30 ms after flash onset. This was followed up by a strong hypometria (but with appropriately-scaled peak speed), as predicted by saccadic averaging. On the other hand, in the flash+ condition there was no significant evidence of hypometria or increase in peak speed soon after flash onset. Moreover, the kinematics of saccades launched during the SI period were not influenced by the presence of the flash. The very large difference between the two flash conditions indicates that the spatial layout was having an impact on saccadic amplitude in a way compatible to a spatial readout of the superior colliculus map. Nonetheless, contrary to a strict prediction of the read out hypothesis, we do not report any hypermetria for the flash+ condition, but this was probably a simple consequence of the logarithmic compression of the visual map in which more eccentric locations occupy less neural tissue (Ottes et al. 1986; Van Gisbergen et al. 1987).

A final interesting observation is that we also recorded hypometric saccades in the flash+ condition, but *following* the inhibitory period. The hypometria was also accompanied by an increased peak speed indicating that these saccades were programmed for the correct target location but subsequently felt short, leading to a violation of the main sequence. It is important to note that these saccades were ones that would have been re-instated or reprogrammed, so the reported effect is not the same as the hypometric saccades recorded during the pre-inhibitory period. This late hypometria is more similar to the one reported by Guillaume (2012) with masking

542 stimuli covering either the entire screen (full mask) or only the portion of the screen
543 where the target was displayed (half mask). Similarly to our findings, Guillaume also
544 observed an increase in peak speed for these reinstated saccades, as in our Experiment
545 1, 2 and 3 (where we did not record a gain reduction). One possibility might be that
546 the later spatial effects are generated by cortico-tectal feedback from areas such as the
547 frontal eye field and the lateral intraparietal cortex inhibiting the SC and truncating
548 the saccade at a later processing stage.
549

550 General conclusions

551 In three experiments, we flashed a visual transient at a range of times relative to a
552 target-directed saccade, at different positions: either at fixation, at the top and bottom
553 of the screen or at a location on the target axis more or less eccentric than the target.
554 In all cases, once the data were aligned temporally to the onset of the flash, a
555 distinctive pattern of variation in saccadic behavior was revealed both in *time* and
556 *space*.

557 First, we replicated the well-known temporal inhibitory effect of the flash (SI:
558 Reingold & Stampe, 1999, 2002) on the initiation of saccades, with a maximal
559 decrease in saccadic frequency varying from 53 to 83 percent across experiments, and
560 the latency of maximum inhibition ranging from 77 to 86 ms. The decrease in
561 saccadic frequency began as early as 60 ms, recovering by 110 ms after the flash.
562 These timings are compatible with the idea that the triggering mechanism of a saccade
563 cannot be changed beyond a point-of-no-return around 60 ms before launching
564 (Reingold and Stampe 2002), and thus with the concept of a "saccadic dead time"
565 applied to this pre-launch period (Findlay and Harris 1984; Ludwig et al. 2007). In
566 passing, we also made a new observation (Experiment 3) that SI magnitude, but not

latency, was strongly affected by flash eccentricity, with greater inhibition for nearby distractor locations. This result, although not a focus of our paper, carries the interesting suggestion that eccentricity is more influential upon SI than distance from target (since in our experiment the flash was equally distant from the target in both the flash+ and flash- conditions).

Our major interest was in the kinematic character of saccades launched following a flashed distractor, and here we focused on saccade amplitude (gain) and its relation with peak speed (main sequence relation). In all Experiments, we observed a strong hypometria for saccades launched a mere 20 ms after the flash, extending to saccades launched up to 80 ms after the flash. The maximum reduction in gain was ~12% in Experiment 1, ~5% (considering all target eccentricities together) in Experiment 2, and ~15% in Experiment 3. Interestingly, the hypometric saccades were not always accompanied by the correspondingly lower peak speed expected from the main sequence. These perturbations of the main sequence were time specific in both Experiments 2 and 3, and maximal for saccades launched around 30 ms after flash onset. A second peak of relative increase in the peak speed was visible for saccades launched around 110 and 130 ms after the flash, in this case unaccompanied by an increase in saccadic gain. Taken together, the data show a complex violation of the main sequence around the onset and offset of the SI dip that develops over time, oscillating with higher or lower values compared to the steady baseline condition.

One hypothesis to account for the early perturbation of the main sequence during the pre-inhibitory period (reduced gain without reduced peak speed) would be that a saccade already in flight was suddenly interrupted by flash onset (see also: Edelman and Xu 2009; Guillaume 2012), creating hypometric saccades with peak speeds appropriate to the originally intended target. In Experiment 2, we additionally

592 observed a decrease of the normalized peak speed compared to baseline toward the
593 end of this early period of perturbation, suggesting that in some circumstances the
594 saccades might have been truncated prior to achieving the peak speed expected for
595 that amplitude. The most striking aspect of these data is saccadic modulation for
596 distractors presented a mere 30 ms before execution, and thus 30 ms before the
597 earliest inhibition of saccade launching. This demonstrates interference from
598 distractors presented during "saccadic dead time" (Ludwig et al. 2007; Weber et al.
599 1992), in which the saccadic program is past the "point of no return" (Reingold &
600 Stampe, 2002), and should be impervious to further visual stimulation. It may indeed
601 be that no changes were implemented to the saccade program itself, but that these
602 very late distractors may have acted to modify the saccade in-flight. Our result
603 confirms that this terminal phase of saccade preparation, immediately prior to
604 launching, despite being immune to reprogramming, may still be permeable to
605 distractor interference during saccade execution, beyond the point of no return.

606 The late phase of kinematic perturbation, around the offset of the SI dip, had a
607 rather different character. We found a pattern of elevated peak speeds without a
608 significant change in saccadic amplitude, except for the flash+ condition in
609 Experiment 3. This late phase of perturbation was visible in all three experiments, but
610 was statistically weak, reaching significance only in Experiment 3. One speculation is
611 that this reflects something about saccades being recovered, or reprogrammed
612 following inhibition, as if these inhibited saccades required an additional impetus to
613 escape the inhibitory effect that resulted in a higher peak speed. Alternatively, the
614 presentation of the flash, temporally close to the target onset for this time period,
615 might have summed up with the target activity leading to an increase in the level of
616 SC activation. These saccades might have remained spatially accurate rather than

being hypermetric because of the feedback loop that controls the saccades within the brainstem (Sparks 2002). Saccades can maintain amplitude information and vary duration/velocity to compensate for external perturbation, such as in the interrupted saccades paradigm (Keller and Edelman, 1994). One hypothesis could be that about 100 ms after flash onset the processing of saccadic amplitude was well advanced so that amplitude/direction were already specified by the activity at the saccadic goal (Anderson, Keller, Gandhi and Das 1998). Nonetheless, the sudden activation of other superior colliculus neurons summed up with the ongoing process, resulting in a "global higher activity" at the time of saccade launching, that we recorded as increased velocity, as predicted by the "dual coding" (Sparks and Mays 1990) and vector summation hypotheses (Goossens and Van Opstal 2006; Van Opstal and Goossens 2008). Further replication work and modeling of the activity within the superior colliculus layers would be required before advancing any strong functional interpretation of this late perturbation of the main sequence.

Alternatively, according to Guillaume (2012) the second modulations are related to the activity induced by the flash in cortical areas sending inhibitory signal to the SC. This second mechanism would interrupt saccades similarly to the early mechanisms, hence generating the modulations observed also in the kinematics. Our data do not fully support the view that the late modulations mimic the truncation mechanism observed soon after flash onset, as in Guillaume 2012, since aside from the flash+ condition in Experiment 3, we did not record late hypometric saccades. On the other hand, we do agree that cortico-tectal feedback, especially from the frontal eye field, might modulate the motor program during the post-inhibitory period and have an impact on the spatial parameters of the saccade.

641 In terms of neurophysiology, given its wide generality across tasks and its
642 short latency, SI has been conceptualized as a low-level interference in the early
643 stages of visual processing and it has been modeled in terms of activity within the
644 intermediate and deep layers of the superior colliculus (Bompas and Sumner 2011).
645 Target and flash onsets generate a burst of activation in the superior colliculus
646 oculomotor map. Following the burst, buildup neurons coding for spatially separated
647 target/flash locations (e.g., Everling et al. 1999; Dorris et al. 1997; Munoz and Wurtz
648 1995a) start interacting through lateral inhibition (Olivier et al. 1999). If the flash is
649 central, or not too eccentric, additional stimulation from fixation neurons and/or direct
650 activation of the omnipause neurons might strongly interfere with the completion of
651 the motor program (Gandhi and Keller 1997). In order for a saccade to be inhibited,
652 flash-related interference must begin prior to the "point-of-no-return" at which the
653 saccade-related motor burst is unstoppable (Reingold and Stampe 2002). The latest
654 point in time that a distractor onset can still inhibit saccade execution is determined by
655 the time necessary for visual information to reach the intermediate superior colliculus
656 and to influence motor structures, estimated around 35-47 ms after visual stimulation
657 (Rizzolatti et al. 1980). This timing closely matches the first variation in saccade
658 kinematics, affecting saccades launched around 30 ms after flash onset. Accordingly,
659 in a number of neurophysiological studies with single cell recording from the nucleus
660 raphe interpositus it has been reported that omnipause neurons respond to a light pulse
661 as they do to electrical stimulation, stopping the saccade in flight (Evinger et al 1982).
662 We propose that the early phase of hypometria recorded in the present experiment
663 might have been induced mainly by the sudden activation of the omnipause network
664 subsequent to flash presentation. Another possibility would be that the sudden visual
665 burst elicited by the irrelevant flash interferes with saccade programming to the point

666 that activity for the flash suddenly reaches threshold, favoring interruption of the
667 current saccadic plan, similarly to the mechanisms that generates express saccades
668 (Edelman and Keller 1996).

669 On the other hand, when the transient is presented between 60 to 130 ms
670 before the start of the saccade, the consequences would be expected to be mainly
671 temporal, with a high percentage of inhibited saccades, and the reported hypometria
672 during this phase may reflect the spatial read-out of the SC map. These long lasting
673 inhibitory processes might be driven mainly by lateral inhibition (Buonocore and
674 McIntosh 2008; Olivier et al. 1999; Reingold and Stampe 2002) and reflect
675 competition during target selection processes rather than a sudden truncation of the
676 motor plan.

677 An alternative view, inspired by the micro-saccadic literature, would instead
678 suggest that distractor onset might induce a phase reset. One mechanism that has been
679 proposed to account for the reduction in micro-saccade generation is that the new
680 visual information could generate a countermanding process, cancelling the upcoming
681 micro-saccade in order to initiate a new one (Hafed and Ignashchenkova 2013).
682 Similar processes have been documented for standard saccades within the superior
683 colliculus (Parè and Hanes 2003) and are also compatible with the timings estimated
684 by modeling of SI using competing motor commands (Bompas and Sumner 2011;
685 Trappenberg et al 2001). The stimulus configuration would skew the superior
686 colliculus activity so that saccades would follow the final readout of the superior
687 colliculus activity, predicting modulations in the kinematic parameters similar to
688 those reported here. From the data at hand, we favor the hypothesis that the early
689 hypometria was the consequence of a more general mechanism probably involving
690 the sudden onset of the omnipause neurons network or the activation of burst neurons.

691 Finally, reprogrammed saccades that are launched in the post inhibitory period might
692 have been influenced by extra excitation of the SC map induced by the flash that was
693 temporally close to the target onset.

694 We conclude that distractor effects have broader influences than previously
695 recognized, which can be expressed both in time and space depending on the stage of
696 saccade preparation or execution with which the distractor interferes. Spatial and
697 kinematic effects arise earlier than outright inhibition of the saccade, whilst, more
698 speculatively, saccades reprogrammed after inhibition might exhibit subtly altered
699 kinematics, characterized by increased speed. The point-of-no-return does not put a
700 saccade beyond the reach of distractors; saccades that escape temporal inhibition may
701 instead show changes in velocity, amplitude or both.

Footnotes

1. An anonymous reviewer raised the issue of whether the amplitude effects that we observe in our results could be artefactual to head movements. It is important to note that eye movements of this magnitude (lower than 20° of visual angle) are normally accomplished without head movement even in a head un-restrained setup (see Freedman 2008; Fuller 1992). Moreover, it would be very hard to come to a principled account for the exact pattern of gain modulation found here, in particular in the target plus flash conditions, as artefacts of lateralized head movements specific to our experimental manipulation. In Experiment 1 and 2, the stimuli were presented either at the center or top/bottom of the screen, where no lateralized response was required to the distractor.

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857 ## Figure captions.

858

859 **Figure 1.** Experimental designs. Panel A. Trial sequence of Experiment 1.
860 Participants were required to maintain fixation and to make a saccade to a red dot
861 (0.5° of visual angle) appearing on the left or on the right side of the fixation cross at
862 10° of eccentricity. Participants were also instructed to report if one out of the four
863 briefly presented probes had a different orientation from the others (50% of trials) or
864 if instead all probes were the same. On Flash trials, a black square (3.5° of visual
865 angle) was presented at fixation for 11.7 ms in order to elicit SI. Participants were
866 asked to ignore the flash. Panel B. Trial sequence of Experiment 2. Participants were
867 instructed to maintain fixation and then to saccade toward a white stimulus presented
868 on the left/right side of the screen at either 4, 8 or 12 degrees of visual angle. In Flash
869 trials, two white bars were covering the 1/3 of the top and 1/3 of the bottom of the
870 screen. Participants were asked to ignore the flash. Panel C. Trial sequence of
871 Experiment 3. Similar structure of Experiment 2 but restricted to one target
872 eccentricity (10 degrees). In Flash trials, a white bar could be presented either 4
873 degrees less eccentric than target location (as showed in figure) or 4 degrees more
874 eccentric than target location. In all figures stimuli are not in scale.

875

876 **Figure 2.** Example of saccadic main sequence fit. Three observers with similar main
877 sequence values were chosen, one for each Experiment, and plotted with their
878 respective fits. Panel A depicts Experiment 1, panel B Experiment 2 and pane C
879 Experiment 3. The empty blue dot symbols show each observation in the no-flash
880 trials while the full black dots represent velocities predicted from the corresponding
881 amplitudes, based on the individual fit. Experiment 1 (panel A) and Experiment 3

(panel C) had only one eccentricity (10 degrees) and the fitted function is a 1st order polynomial. Experiment 2 (panel B) had a range of eccentricities (4, 8 and 12 degrees) and the fitted function is a 2nd order polynomial. R^2 for each experiment and participant are reported below.

Experiment 1: 0.15; 0.02; 0.61; 0.22; 0.09; 0.16; 0.05; 0.50; 0.20.

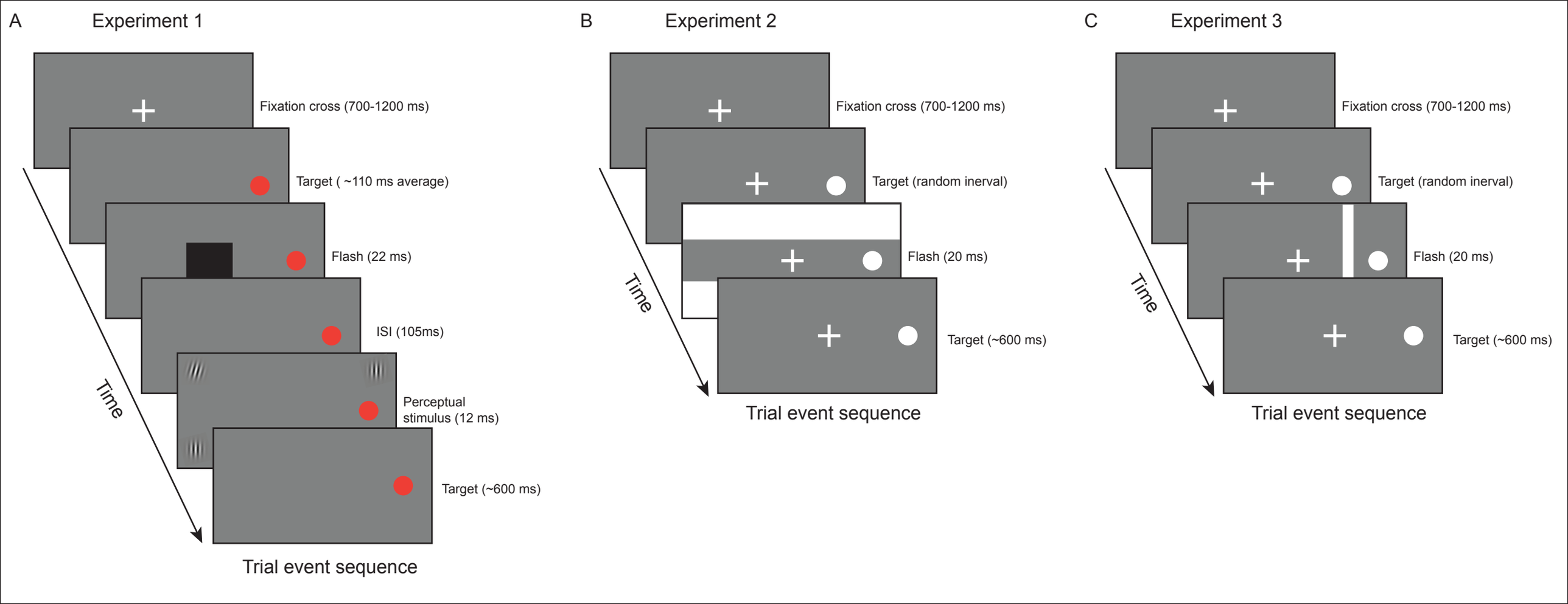
Experiment 2: 0.85; 0.75; 0.66; 0.70; 0.76; 0.76; 0.72; 0.84; 0.81; 0.92.

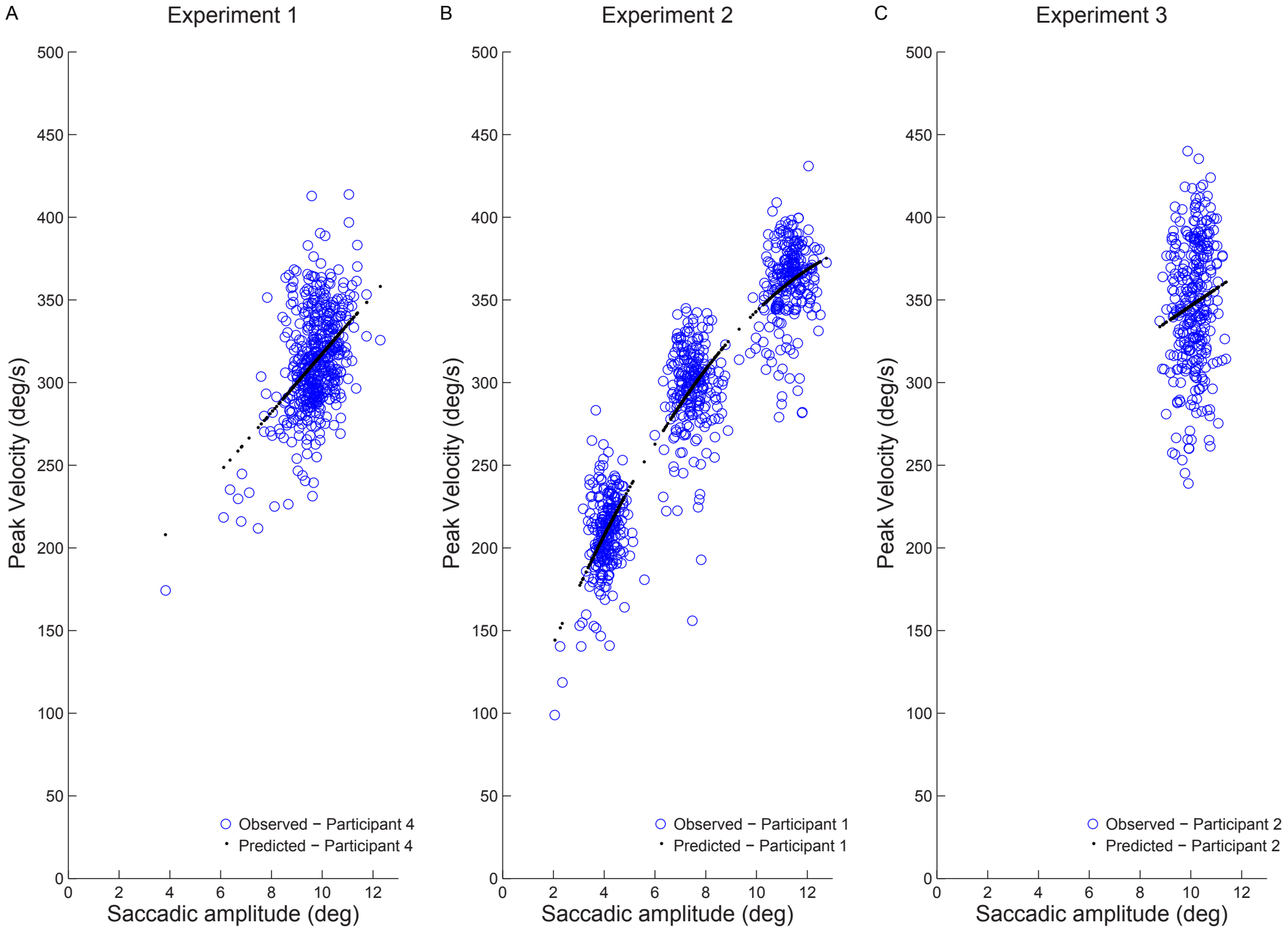
Experiment 3: 0.21; 0.02; 0.04; 0.51; 0.09; 0.25; 0.19; 0.02.

Figure 3. SI, saccadic gain and normalised peak speed in Experiment 1, 2 and 3. Panel A, D and G. Average SI profiles with 95% CI (shaded area). In Experiment 2, the SI profile was averaged also across the three eccentricities since we did not find any statistical difference among the three conditions. In Experiment 3, red represent flash- and green flash+ conditions (same convention in plot H and I). Variation in saccadic gain (panel B, E and H) and normalised peak speed (panel C, F and I) for target only (blue) and target plus flash (red and red/green in Experiment 3) trials. Data are binned in 20 ms intervals. Time on the x-axis is relative to flash onset; x-axis values thus represent the temporal lead of the flash relative to the observed launching of the saccade. Asterisks indicate significant differences between the no-flash and flash conditions (FDR corrected). Shaded areas represent the standard error of the mean.

Authors contribution

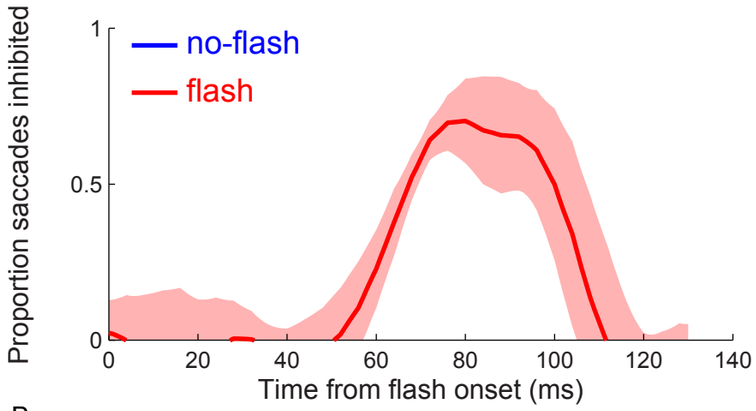
Antimo Buonocore and David Melcher designed Experiment 1, and all three authors designed Experiments 2 and 3. Antimo Buonocore carried out data collection and data analysis. Interpretation of the data was done equally by the three authors. Antimo Buonocore drafted the manuscript and Robert McIntosh and David Melcher provided critical revisions. All authors approved the final version of the manuscript for submission.


A
Experiment 1
Fixation cross (700-1200 ms)
Target (~110 ms average)
Flash (22 ms)
ISI (105ms)
Perceptual stimulus (12 ms)
Target (~600 ms)
Time
Trial event sequence
B
Experiment 2
Fixation cross (700-1200 ms)
Target (random inerval)
Flash (20 ms)
Target (~600 ms)
Time
Trial event sequence
C
Experiment 3
Fixation cross (700-1200 ms)
Target (random inerval)
Flash (20 ms)
Target (~600 ms)
Time
Trial event sequence

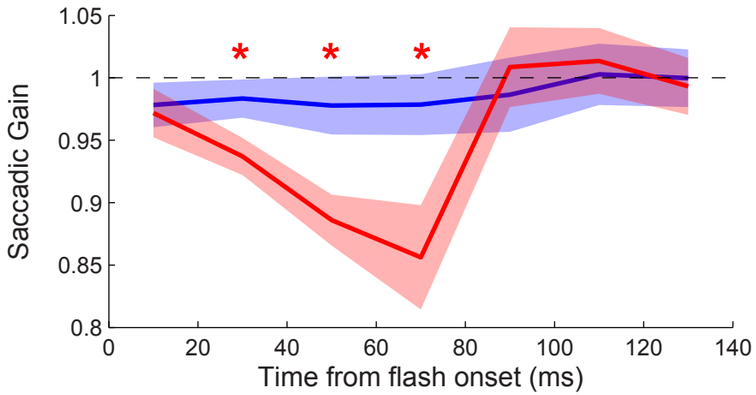

A
Experiment 1
B
Experiment 2
C
Experiment 3
Peak Velocity (deg/s)
Saccadic amplitude (deg)
0
50
100
150
200
250
300
350
400
450
500
0
2
4
6
8
10
12
Observed − Participant 4
Predicted − Participant 4
Observed − Participant 1
Predicted − Participant 1
Observed − Participant 2
Predicted − Participant 2

A

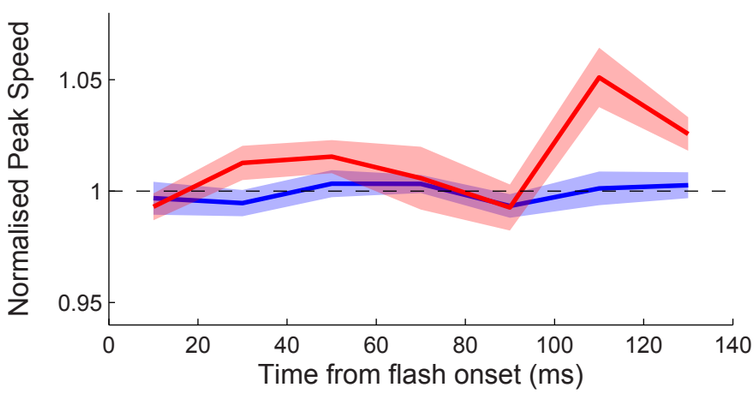
Experiment 1



B

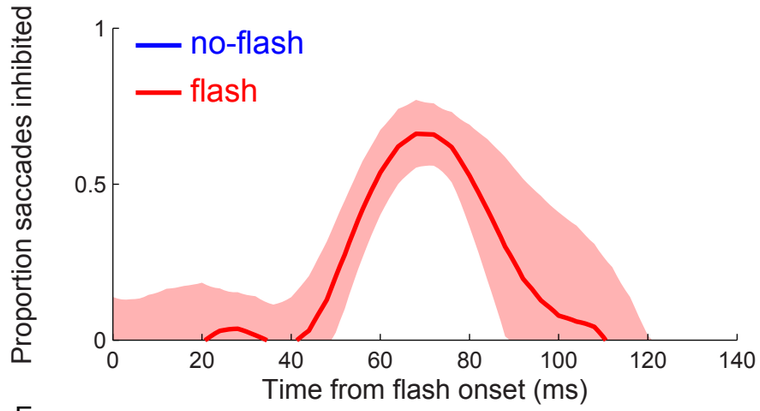


C

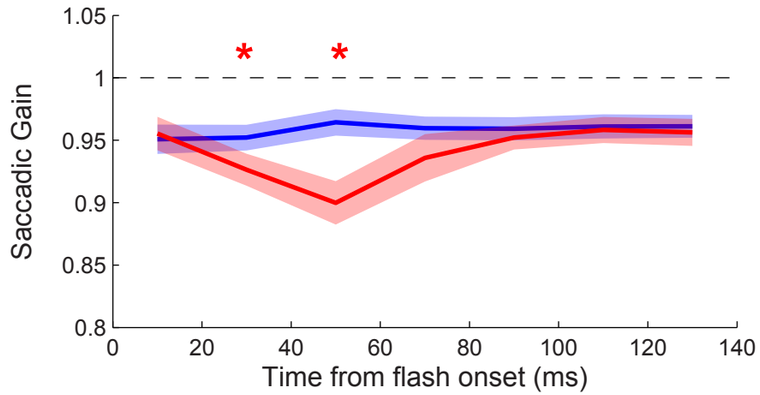


D

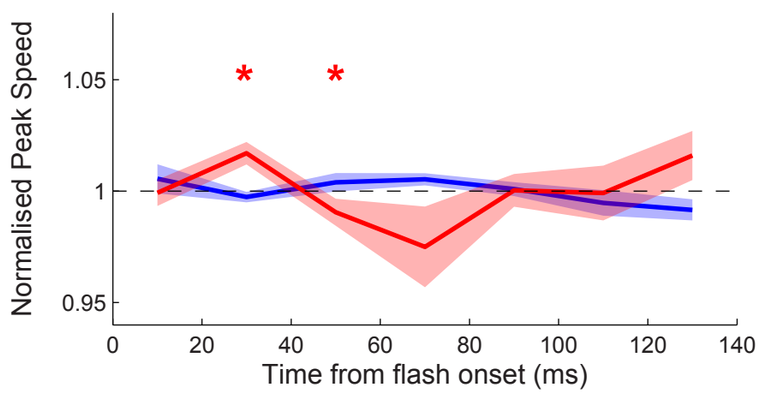
Experiment 2



E

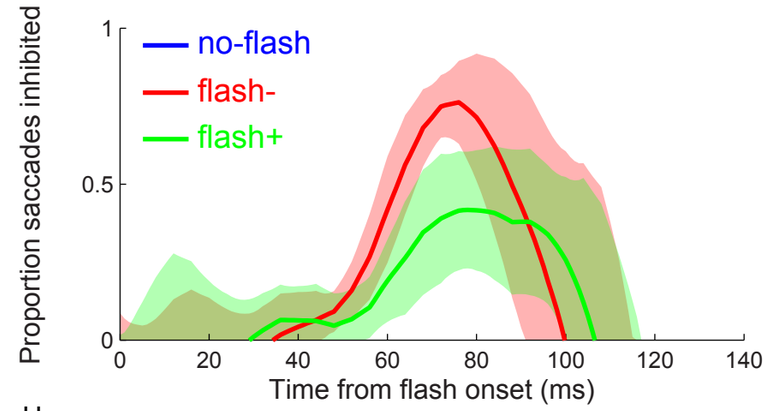


F

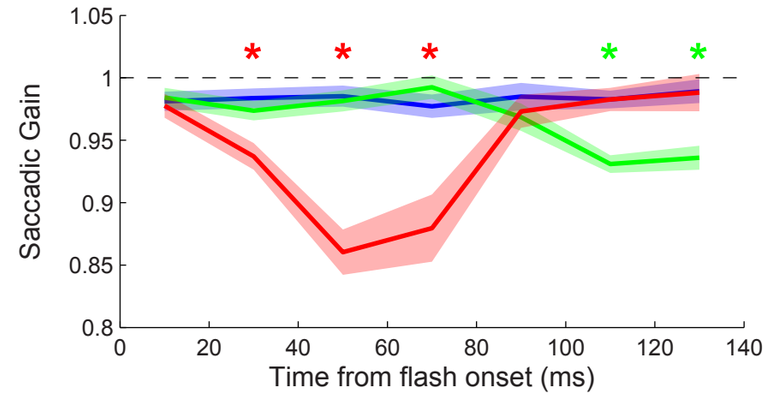


G

Experiment 3



H



I

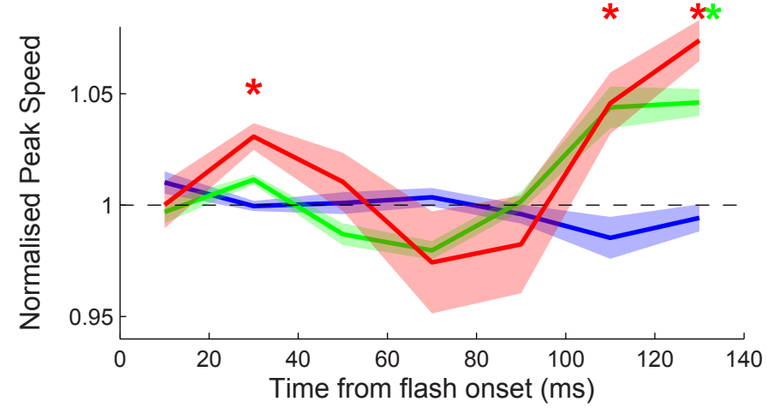


Table 1. Maximum of SI (Dip maximum) and latency of the maximum (Dip latency) for each participant and condition in Experiment 1. Mean and S.D. are reported in the bottom row.

Experiment	Participant	Condition	Dip maximum	Dip latency
1	1	Central flash	0.90	81
	2		0.68	77
	3		0.84	85
	4		0.82	77
	5		0.64	73
	6		0.62	81
	7		0.97	97
	8		0.95	101
	9		0.61	97
		Mean	**0.78**	**85**
		S.D.	**0.14**	**10.3**

Table 2. Maximum of SI (Dip maximum) and latency of the maximum (Dip latency) for each participant and condition in Experiment 2. Mean and S.D. are reported in the bottom row.

Experiment	Participant	Condition	Dip maximum	Dip latency
2	1	Target 4°	0.85	81
	2		0.90	65
	3		0.70	77
	4		0.63	89
	5		0.64	69
	6		0.88	89
	7		0.73	73
	8		0.73	65
	9		0.62	93
	10		0.71	77
		Mean	**0.74**	**78**
		S.D.	**0.10**	**10.1**
2	1	Target 8°	0.82	89
	2		0.90	65
	3		0.60	109
	4		0.74	73
	5		0.50	81
	6		0.88	89
	7		0.79	73
	8		0.79	65
	9		0.60	77
	10		0.75	73
		Mean	**0.74**	**79**
		S.D.	**0.13**	**13.4**
2	1	Target 12°	0.96	85
	2		0.89	65
	3		0.53	109
	4		0.91	73
	5		0.68	73
	6		0.91	93
	7		0.85	69
	8		0.75	65
	9		0.55	65
	10		0.71	69
		Mean	**0.77**	**77**
		S.D.	**0.15**	**14.7**

Table 3. Maximum of SI (Dip maximum) and latency of the maximum (Dip latency) for each participant and condition in Experiment 3. Mean and S.D. are reported in the bottom row.

Experiment	Participant	Condition	Dip maximum	Dip latency
3	1	flash+	0.71	89
	2		0.29	93
	3		0.41	69
	4		0.66	93
	5		0.39	73
	6		0.36	73
	7		0.86	81
	8		0.57	73
		Mean	**0.52**	**74**
		S.D.	**0.23**	**22.5**
3	1	flash-	0.91	85
	2		0.81	73
	3		0.69	69
	4		1.00	89
	5		0.64	69
	6		0.78	73
	7		0.97	77
	8		0.88	81
		Mean	**0.74**	**71**
		S.D.	**0.23**	**18.4**