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

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Buonocore A, McIntosh RD, Melcher D. Beyond the point of no return: effects of visual distractors on saccade amplitude and velocity. *J Neurophysiol* 115: 752–762, 2016. First published December 2, 2015; doi:10.1152/jn.00939.2015.—Visual transients, such as a bright flash, reduce the proportion of saccades executed, ~60–125 ms after flash onset, a phenomenon known as saccadic inhibition (SI). 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 SI. First, saccades launched between 30 and 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," ~60 ms before 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 postflash, toward the end of SI, 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.

eye movements; saccadic inhibition; main sequence

THE MAIN CHALLENGE FOR THE oculomotor system in a complex environment is to select when and where to move the eyes to land near targets of interest. Saccadic amplitudes range from only a few minutes of arc to >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 ~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, prosaccade, and antisaccade tasks (Reingold and Stampe 2002). Interestingly, an analogous effect has been shown for endogenously triggered microsaccades. "Micro-SI" describes a similar dip in microsaccade rate, ~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 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). At first, it was suggested that the SDT was a constant value, ~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 Loon et al. 2002). More recently, this notion was challenged by Ludwig et al. (2007), by showing that even if the SDT were not influenced by variations in saccadic reaction times (SRTs), 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

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limit, reported in behavioral 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 SDT, 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 (2002, 2004) 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 microsaccades (Hafed and Ignashchenkova 2013; Rolfs et al. 2008). For example, Hafed and Ignashchenkova (2013) reported that the microsaccadic rate was not only reduced, 100 ms after a supplementary stimulus, but also that the spatial character of the persisting microsaccades was sensitive to the location of that stimulus. Their interpretation was that the observed microsaccades reflected an instantaneous "read out" of activations in the oculomotor maps of the superior colliculus (SC), 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 saccade. However, aside from the work of Guillaume (2012) and some observations made by Edelman and Xu (2009), no other studies have made a detailed analysis of the time course of such parameters, following distractors with different characteristics, in a way that is analogous to what has been done for the temporal domain.

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

METHODS

Participants. Nine (*experiment 1*), 10 (*experiment 2*), and 8 (*experiment 3*) volunteers, aged between 18 and 30 yr, 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; protocol approval was given by the University of Trento Research Ethics Committee. 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 cathode ray tube monitor (1,024 × 768 pixels) at 85 Hz (*experiment 1*) or 100 Hz (*experiments 2* and *3*). In all of the experiments, participants were seated, with their head resting on a chin and forehead rest 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 (SR Research, Ottawa, ON, Canada; detection algorithm: pupil and corneal reflex; 1,000 Hz sampling; saccade detection based on 30°/s velocity and 9,500°/s^2 acceleration thresholds; maximum head movement[1] tolerance = 25 mm × 25 mm × 10 mm, horizontal × vertical × 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 his head from the chin rest. In all of the experiments, the background was gray (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* (Fig. 1*A*), after a random interval varying between 500 and 1,200 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 one-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). SRTs 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 one-half of the trials, the four stimuli had the same orientation (vertical or horizontal) and on the other one-half, one of them had a different orientation. At the end of the trial, participants were asked to report if all of 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

[1] An anonymous reviewer raised the issue of whether the amplitude effects that we observe in our results could be artifactual 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-unrestrained setup (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 artifacts of lateralized head movements specific to our experimental manipulation. In *experiments 1* and 2, the stimuli were presented at the center or top/bottom of the screen, where no lateralized response was required to the distractor.

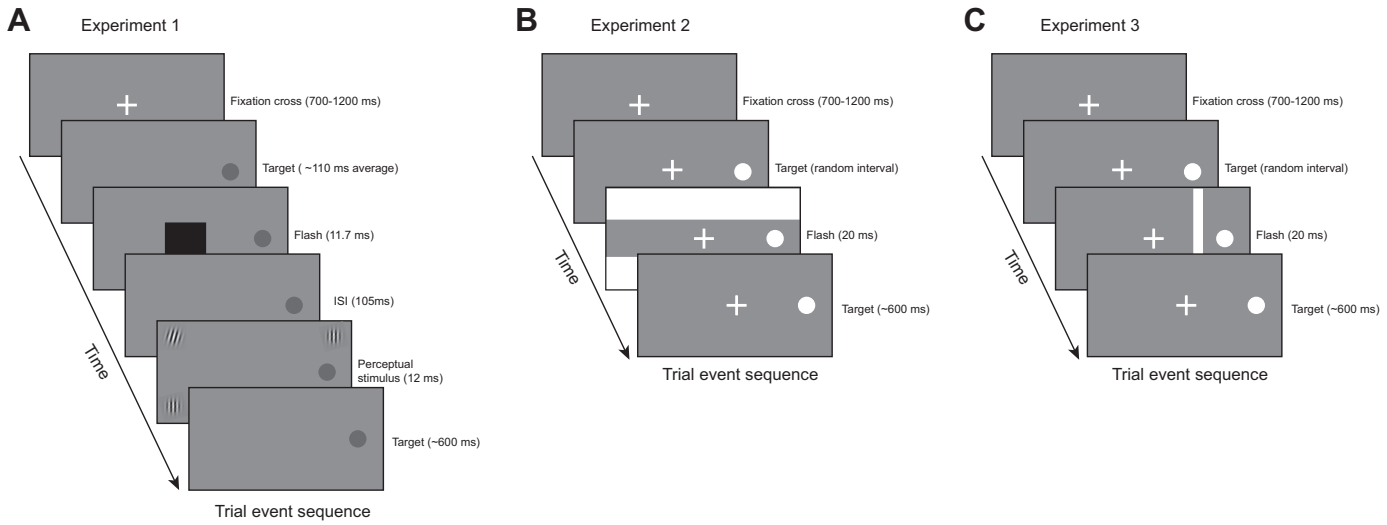

Fig. 1. Experimental designs. *A*: trial sequence of *experiment 1*. Participants were required to maintain fixation and to make a saccade to a red dot (shown as gray dots; 0.5° of visual angle), appearing on the left or on the right side of the fixation cross at 10° of eccentricity. Participants were also instructed to report if 1 out of the 4 briefly presented probes had a different orientation from the others (50% of trials) or if instead, all probes were the same. On Flash trials, a black square (3.5° of visual angle) was presented at fixation for 11.7 ms to elicit saccadic inhibition (SI). Participants were asked to ignore the flash. ISI, interstimulus interval. *B*: trial sequence of *experiment 2*. Participants were instructed to maintain fixation and then to saccade toward a white stimulus presented on the left/right side of the screen at either 4°, 8°, or 12° of visual angle. In Flash trials, 2 white bars were covering 1/3 of the top and 1/3 of the bottom of the screen. Participants were asked to ignore the flash. *C*: trial sequence of *experiment 3*. Similar structure of *experiment 2* but restricted to 1 target eccentricity (10°). In Flash trials, a white bar could be presented either 4° less eccentric than target location (as shown in figure) or 4° more eccentric than target location. In the figure, stimuli are not to scale.

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, one-half with the target on the right and one-half with the target on the left side of the screen. The median SRT from the last 50 of these trials provided an estimate of the expected SRT for that participant for the experimental blocks. In the experimental blocks, target-only (no-flash) trials were intermingled equally with target plus distractor (flash) trials, in which in addition to the target, the black square (3.5°, 2.3 cd/m^2) was flashed at the center of the screen for 11.7 ms. The onset of this central flash varied randomly from ~117 ms before to 11.7 ms after the expected SRT for that participant in steps of 11.7 ms, thereby providing a wide range of distractor delays. Each of the two conditions (flash, no-flash) occurred 64 times per block, shuffled randomly. Each participant completed two sessions of eight experimental blocks on different days for a total of 1,024 trials. Although originally conceived for a different purpose, the experimental design described above provided a rich dataset for an opportunistic exploration of the time-course analysis of saccade kinematics and provided the basic template for the two experiments subsequently designed to investigate these issues further (but did not include the perceptual task and used different distractor locations).

Experiment 2 (Fig. 1*B*) was designed to replicate and extend the observations of the first experiment. The saccadic task was similar to that of *experiment 1*, but changes were introduced to sample a wide range of distractor delays relative to expected saccade onset and to optimize the effect of the visual transient upon the oculomotor response. No perceptual task was presented in either the second or third experiment. After a random interval, varying between 500 and 1,200 ms, a white dot (0.5°, 124 cd/m^2) was displayed at three possible eccentricities (4°, 8°, and 12° of visual angle), with equal probability to the right or to the left of the visual field. As in *experiment 1*, each participant performed a preliminary block of 60 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 stimulus-onset asynchronies (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 1,560 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; Fig. 1*C*). 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

Table 1. *Maximum of SI (Dip Maximum) and latency of the maximum (Dip Latency) for each participant and condition in experiment 1*

Participant	Condition	Dip Maximum	Dip Latency
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
	Means	0.78	85
	SD	0.14	10.3

SI, saccadic inhibition. Means and SD are reported in the bottom rows.

block. Participants completed 2 sessions for 8 blocks in 1 day for a total of 960 trials, with 320 trials per flash condition.

Data screening. We excluded saccades with latencies of <70 ms (*experiment 1*: ~1.2%; *experiment 2*: ~2.5%; *experiment 3*: ~0.73%) or of >500 ms (*experiment 1*: ~2.7%; *experiment 2*: ~0.14%; *experiment 3*: ~0.75%). We also removed saccades with an amplitude <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 *experiments 2* and *3*, we also excluded 2.6% and 1.52% of saccades, respectively, due to blinks.

Analysis of SI. In all the experiments, we performed an analysis of the SRT distributions for all valid trials by following the procedure to calculate the dip ratio of Bompas and Sumner (2011). 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 a 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 (see Fig. 3, *A*, *D*, and *G*). Statistical analysis was performed on the individual parameters extracted from each SI profile across the three experiments and is reported in Tables 1, 2, and 3.

Analysis of saccadic kinematics. The analysis of saccade kinematics focused on 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 larger than one indicating a speed higher than expected and values less than one indicating a speed lower than expected. Therefore, the calculation of normalized peak speed 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 *experiments 1* and *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 first-order polynomial function. In *experiment 2*, we made use of all of the eccentricities to estimate the best main sequence fit using a second-order polynomial function. In Fig. 2, we show one example of fitting for each experiment (Fig. 2, *A–C*) along with the coefficient of determination 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, reaction times were binned using a bin width of 20 ms, and the mean saccadic gain and normalized peak speed were 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: 10–130 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: 10–130 in 20 ms intervals) repeated-

Table 2. *Maximum of SI (Dip Maximum) and latency of the maximum (Dip Latency) for each participant and condition in experiment 2*

Participant	Condition	Dip Maximum	Dip Latency
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
	Means	0.74	78
	SD	0.10	10.1
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
	Means	0.74	79
	SD	0.13	13.4
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
	Means	0.77	77
	SD	0.15	14.7

Means and SD are reported in the bottom rows of each section.

Table 3. *Maximum of SI (Dip Maximum) and latency of the maximum (Dip Latency) for each participant and condition in experiment 3*

Participant	Condition	Dip Maximum	Dip Latency
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
	Means	0.52	74
	SD	0.23	22.5
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
	Means	0.74	71
	SD	0.23	18.4

Means and SD are reported in the bottom rows of each section.

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 sample *t*-tests comparing no-flash with flash conditions at each time bin. With the consideration that adjacent time bins are likely to be correlated, we performed the Benjamini and Hochberg (1995) and the Benjamini and Yekutieli (2001) procedure, controlling the false discovery rate of a family of hypothesis tests. Corrected *P* levels are reported in the text.

RESULTS

Experiment 1—analysis of SI. Overall, we confirmed the main SI effect by showing a strong bimodality in the flash histogram, with the lowest saccadic frequency happening ~90 ms after flash onset. For illustrative purposes, Fig. 3*A* 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 for details of the SI profile calculation). With the use of the parameters extracted from the individual SI profiles, we estimated that an average maximum of 78% of saccades was inhibited at 85 ms after the flash onset, matching well with the timing of SI and micro-SI found in previous experiments (Bompas and Sumner 2011; Buonocore and McIntosh 2008, 2012, 2013; Edelman and Xu 2009; Guillaume 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 [$F(1.6,12.82) = 8.99$; $P < 0.005$; Fig. 3*B*]. During flash trials, we observed a strong decrease in saccadic amplitude (hypometria) for saccades launched 20–80 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: [$t(8) = 3.35$; $P < 0.023$]}. 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. In examining Fig. 3*C*, there is an indication that the violation might be concentrated in a few specific time points after flash onset, during the pre- and postinhibitory period, as observed by Guillaume (2012). Nonetheless, whereas 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 (−1.20°) ms 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. With the consideration that the average no-flash SRTs were ~220 ms for this task, saccades launched 130–150 ms after flash onset corresponded to flash stimuli and 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 buildup 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 presented an intriguing pattern of modulations following distractor interference that confirmed and expanded previous reports of spatial and temporal effects (Buonocore and McIntosh 2012; Edelman and Xu 2009; Guillaume 2012). Nonetheless, whereas reduced saccadic gain was clear during the preinhibitory 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

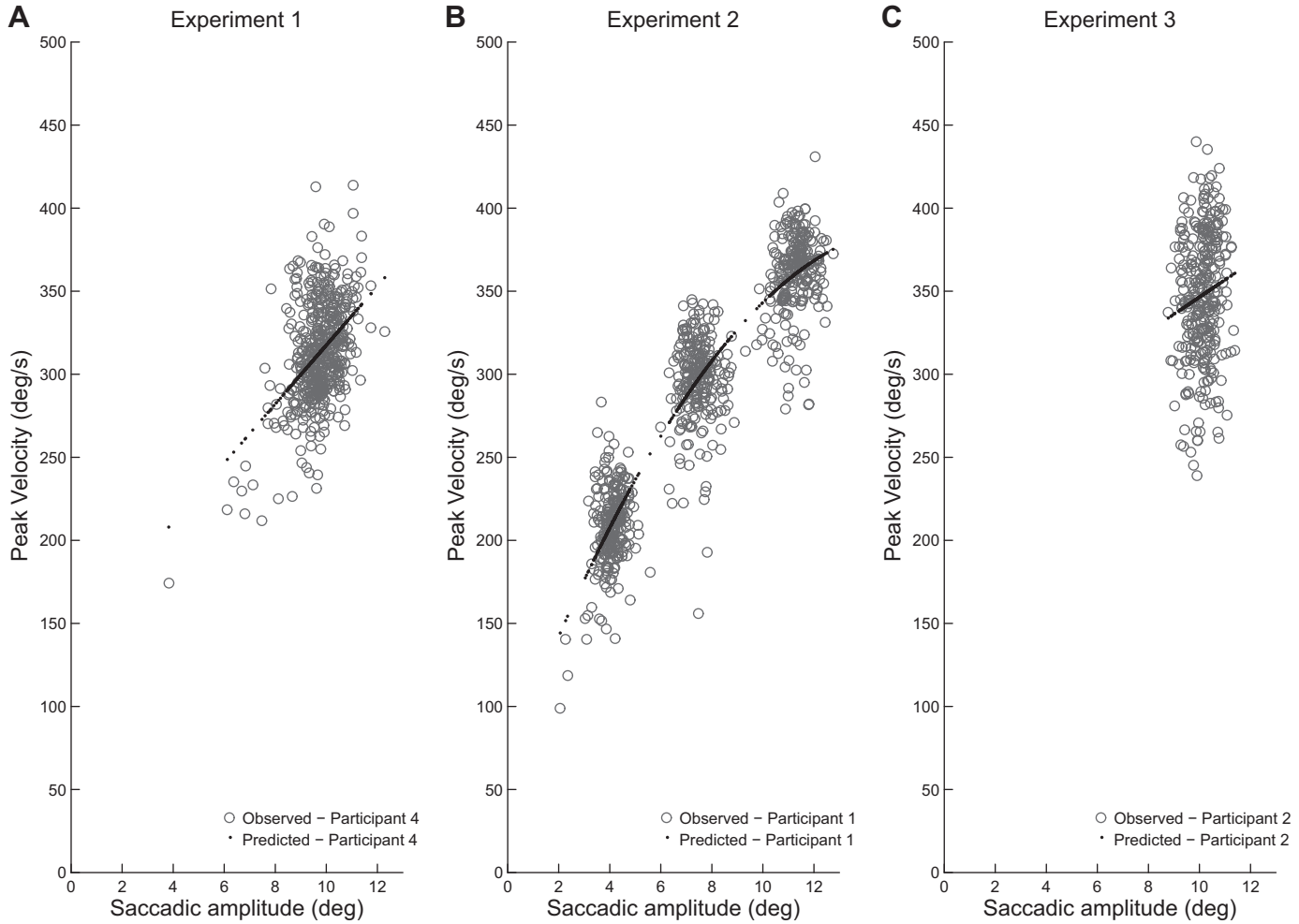

Fig. 2. Example of saccadic main sequence fit. Three observers with similar main sequence values were chosen, 1 for each experiment, and plotted with their respective fits. *A*: *experiment 1*; *B*: *experiment 2*; *C*: *experiment 3*. The empty gray dot symbols show each observation in the no-flash trials, whereas the solid black dots represent velocities predicted from the corresponding amplitudes, based on the individual fit. *Experiment 1* (*A*) and *experiment 3* (*C*) had only 1 eccentricity (10°), and the fitted function is a first-order polynomial. *Experiment 2* (*B*) had a range of eccentricities (4°, 8°, and 12°), and the fitted function is a second-order polynomial. Coefficient of determination for each experiment and participant is reported as follows: *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.

of micro-SI, whereby the target-flash configuration was found to determine the pattern of amplitudes changes (Hafed and Ignashchenkova 2013; Rolfs et al. 2008).

Thus to measure more closely 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 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 map the main sequence function better.

Experiment 2—analysis of SI. The parameters extracted from the individual SI profiles (Table 2) were closely similar across the three eccentricities, and the maximum inhibition was ~74%, ~74%, and ~77% 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 was significantly different among the three eccentricities [magnitude: $F(2, 18) < 1$, not significant (N.S.); latency: $F(1.13, 10.15) < 1$, N.S.]. For descriptive purposes, in Fig. 3*D*, we report the average profile across the three eccentricities.

Experiment 2—analysis of saccadic kinematics. With the use of the gain as a 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 [$F(2.361, 21.245) = 5.6$; $P < 0.008$; Fig. 3*E*]. 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

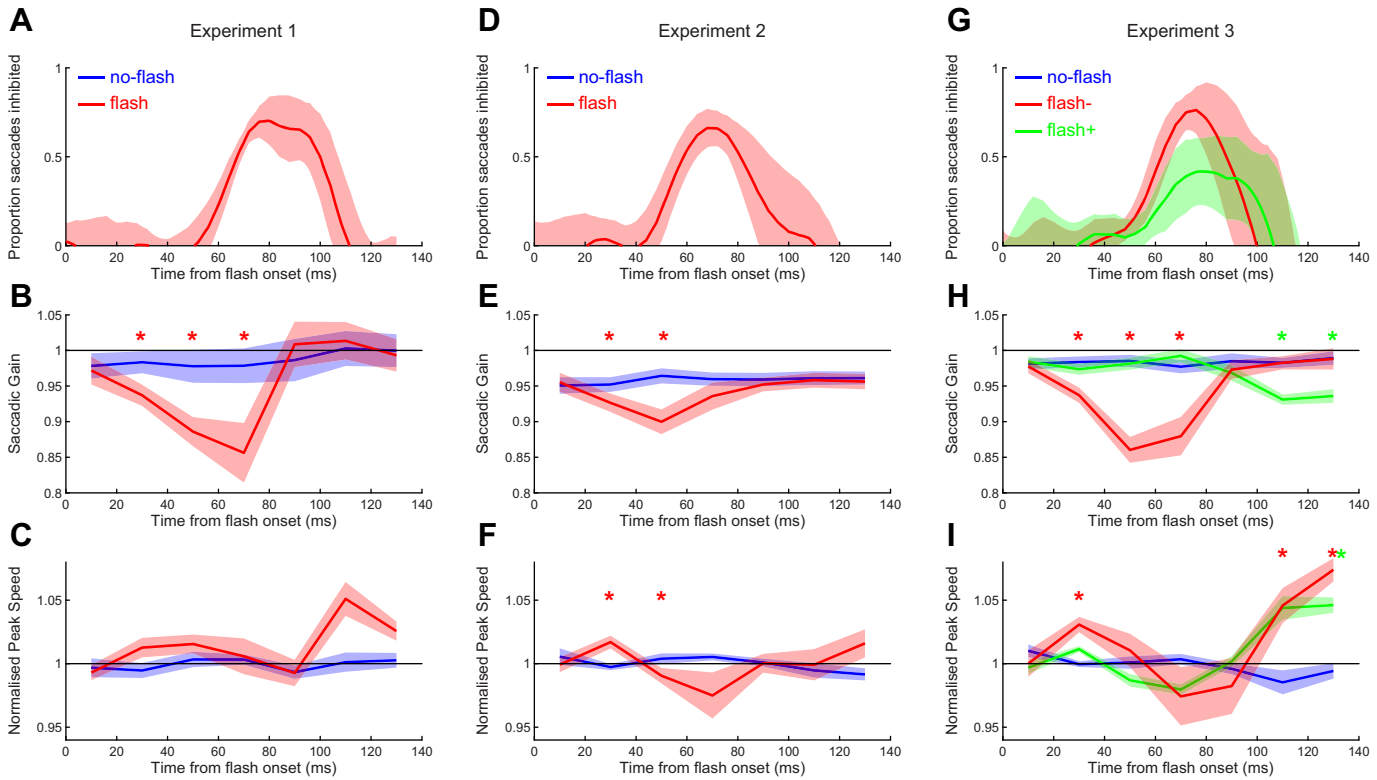

Fig. 3. SI, saccadic gain, and normalized peak speed in *experiments 1–3*. *A*, *D*, and *G*: average SI profiles with 95% confidence interval (shaded area). In *experiment 2*, the SI profile was averaged also across the 3 eccentricities, since we did not find any statistical difference among the 3 conditions. In *experiment 3*, red represents flash− and green flash+ conditions (same convention in *H* and *I*). Variation in saccadic gain (*B*, *E*, and *H*) and normalized peak speed (*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 (false discovery rate corrected). Shaded areas represent the SE.

[$F(6,54) = 4.65$; $P < 0.001$; Fig. 3*F*]. 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 preinhibitory 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 postinhibitory period, although this trend did not reach statistical significance.

Experiment 2—interim discussion. The data from *experiment 2* confirmed that saccades launched during the preinhibitory 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 postinhibitory 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-SI literature, the final read out of the SC 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 were generated by a general truncation mechanism, then 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 should record

hypometria for the less-eccentric flash and hypermetria for the more-eccentric flash.

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

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

Experiment 3—analysis of saccadic kinematics. For gain, the main effect of flash was reliable [$F(2,14) = 47.92$; $P < 0.0001$], as was the main effect of bin [$F(6,42) = 5.12$; $P < 0.001$]. More importantly, as in *experiments 1* and *2*, there was a significant interaction between the two factors [$F(12,84) = 16.67$; $P < 0.0001$; Fig. 3*H*]. Follow-up analyses of the flash-by-bin interaction replicated the strong hypometria effect but with different timings for the 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 *experiments 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 [$F(12,84) = 5.51$; $P < 0.0001$; Fig. 3*I*]. 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 postinhibitory period at 110 and 130 ms [$t(7) = 3.03$, $P < 0.044$; $t(7) = 5.79$, $P < 0.005$, respectively]. The flash+ condition had only one significant violation point during the postinhibitory 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, in looking at Fig. 3, *C*, *F*, and *I*, it is suggestive that for all of the experiments, the shape of the normalized peak speed oscillated compared with 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 SC map, similarly to what has been reported in the microsaccade 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 SC 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 preinhibitory period. This late hypometria is more similar to the one reported by Guillaume (2012), with masking stimuli covering either the entire screen (full mask) or only the portion of the screen where the target was displayed (half mask). Similarly to our findings, Guillaume (2012) also observed an increase in peak speed for these reinstated saccades, as in our *experiments 1–3* (where we did not record a gain reduction). One possibility might be that the later spatial effects are generated by corticotectal feedback from areas, such as the frontal eye field and the lateral intraparietal cortex, inhibiting the SC and truncating the saccade at a later processing stage.

DISCUSSION

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

First, we replicated the well-known temporal inhibitory effect of the flash [SI: see Reingold and Stampe (1999, 2002)] on the initiation of saccades, with a maximal decrease in saccadic frequency varying from 53 to 83% across experiments and the latency of maximum inhibition ranging from 77 to 86 ms. The decrease in saccadic frequency began as early as 60

ms, recovering by 110 ms after the flash. These timings are compatible with the idea that the triggering mechanism of a saccade cannot be changed beyond a point of no return, ~60 ms before launching (Reingold and Stampe 2002), and thus with the concept of an SDT applied to this prelaunch period (Findlay and Harris 1984; Ludwig et al. 2007). In 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 ~30 ms after flash onset. A second peak of relative increase in the peak speed was visible for saccades launched ~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 with the steady baseline condition.

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

The late phase of kinematic perturbation, around the offset of the SI dip, had a rather different character. We found a pattern of elevated peak speeds without a significant change in saccadic amplitude, except for the flash+ condition in *experiment 3*. This late phase of perturbation was visible in all three experiments but was statistically weak, reaching significance only in *experiment 3*. One speculation is that this reflects something about saccades being recovered or reprogrammed following inhibition, as if these inhibited saccades required an additional impetus to escape the inhibitory effect that resulted in a higher peak speed. Alternatively, the presentation of the flash, temporally close to the target onset for this time period, might have summed up with the target activity, leading to an increase in the level of SC activation. These saccades might have remained spatially accurate rather than being hypermetric because of the feedback loop that controls the saccades within the brain stem (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 ~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 et al. 1998). Nonetheless, the sudden activation of other SC 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 SC 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 an inhibitory signal to the SC. This second mechanism would interrupt saccades similarly to the early mechanisms, hence generating the modulations also observed 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 corticotectal feedback, especially from the frontal-eye field, might modulate the motor program during the postinhibitory period and have an impact on the spatial parameters of the saccade.

In terms of neurophysiology, given its wide generality across tasks and its short latency, SI has been conceptualized as a low-level interference in the early stages of visual processing, and it has been modeled in terms of activity within the intermediate and deep layers of the SC (Bompas and Sumner 2011). Target and flash onsets generate a burst of activation in the SC oculomotor map. Following the burst, build-up neurons coding for spatially separated target/flash locations (Dorris et al. 1997; Everling et al. 1999; Munoz and Wurtz 1995) start interacting through lateral inhibition (Olivier et al. 1999). If the flash is central or not too eccentric, then additional stimulation from fixation neurons and/or direct activation of the omnipause neurons might strongly interfere with the completion of the motor program (Gandhi and Keller 1997). For a saccade to be

inhibited, flash-related interference must begin before the point of no return, at which the saccade-related motor burst is unstoppable (Reingold and Stampe 2002). The latest point in time in which a distractor onset can still inhibit saccade execution is determined by the time necessary for visual information to reach the intermediate SC and to influence motor structures, estimated 35–47 ms after visual stimulation (Rizzolatti et al. 1980). This timing closely matches the first variation in saccade kinematics, affecting saccades launched ~30 ms after flash onset. Accordingly, in a number of neurophysiological studies with single-cell recording from the nucleus raphe interpositus, it has been reported that omnipause neurons respond to a light pulse, as they do to electrical stimulation, stopping the saccade in flight (Evinger et al. 1982). We propose that the early phase of hypometria, recorded in the present experiment, might have been induced mainly by the sudden activation of the omnipause network subsequent to flash presentation. Another possibility would be that the sudden visual burst elicited by the irrelevant flash interferes with saccade programming to the point that activity for the flash suddenly reaches threshold, favoring interruption of the current saccadic plan, similarly to the mechanisms that generates express saccades (Edelman and Keller 1996).

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

An alternative view, inspired by the microsaccadic literature, would instead suggest that distractor onset might induce a phase reset. One mechanism that has been proposed to account for the reduction in microsaccade generation is that the new visual information could generate a countermanding process, canceling the upcoming microsaccade to initiate a new one (Hafed and Ignashchenkova 2013). Similar processes have been documented for standard saccades within the SC (Parè and Hanes 2003) and are also compatible with the timings estimated by modeling SI using competing motor commands (Bompas and Sumner 2011; Trappenberg et al. 2001). The stimulus configuration would skew the SC activity so that saccades would follow the final readout of the SC activity, predicting modulations in the kinematic parameters similar to those reported here. From the data at hand, we favor the hypothesis that the early hypometria was the consequence of a more general mechanism, probably involving the sudden onset of the omnipause neurons network or the activation of burst neurons. Finally, reprogrammed saccades that are launched in the postinhibitory period might have been influenced by extra excitation of the SC map induced by the flash that was temporally close to the target onset.

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

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

Author contributions: A.B., R.D.M., and D.M. conception and design of research; A.B. performed experiments; A.B. analyzed data; A.B., R.D.M., and D.M. interpreted results of experiments; A.B. prepared figures; A.B., R.D.M., and D.M. drafted manuscript; A.B., R.D.M., and D.M. edited and revised manuscript; A.B., R.D.M., and D.M. approved final version of manuscript.

REFERENCES

Anderson RW, Keller EL, Gandhi NJ, Das S. Two-dimensional saccade-related population activity in superior colliculus in monkey. *J Neurophysiol* 80: 798–817, 1998.

Bahill A, Clark M, Stark L. The main sequence, a tool for studying human eye movements. *Math Biosci* 24: 191–204, 1975.

Becker W. Saccades. In: *Vision and Visual Dysfunction*, edited by Carpenter RH. Boca Raton, FL: CRC, 1991, p. 95–137.

Becker W, Jürgens R. An analysis of the saccadic system by means of double step stimuli. *Vision Res* 19: 967–983, 1979.

Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J R Statist Soc B* 57: 289–300, 1995.

Benjamini Y, Yekutieli D. The control of the false discovery rate in multiple testing under dependency. *Ann Stat* 29: 1165–1188, 2001.

Beutter BR, Eckstein MP, Stone LS. Saccadic and perceptual performance in visual search tasks. I. Contrast detection and discrimination. *J Opt Soc Am A Opt Image Sci Vis* 20: 1341–1355, 2003.

Bompas A, Sumner P. Saccadic inhibition reveals the timing of automatic and voluntary signals in the human brain. *J Neurosci* 31: 12501–12512, 2011.

Buonocore A, McIntosh DR. Attention modulates saccadic inhibition magnitude. *Q J Exp Psychol (Hove)* 66: 1051–1059, 2013.

Buonocore A, McIntosh DR. Modulation of saccadic inhibition by distractor size and location. *Vision Res* 69: 32–41, 2012.

Buonocore A, McIntosh DR. Saccadic inhibition underlies the remote distractor effect. *Exp Brain Res* 191: 117–122, 2008.

Collewijn H, Erkelens CJ, Steinman RM. Binocular co-ordination of human vertical saccadic eye movements. *J Physiol* 404: 183–197, 1988.

Dorris MC, Pare M, Munoz DP. Neuronal activity in monkey superior colliculus related to the initiation of saccadic eye movements. *J Neurosci* 17: 8566–8579, 1997.

Edelman JA, Keller EL. Activity of visuomotor burst neurons in the superior colliculus accompanying express saccades. *J Neurophysiol* 76: 908–926, 1996.

Edelman JA, Xu KZ. Inhibition of voluntary saccadic eye movement commands by abrupt visual onsets. *J Neurophysiol* 101: 1222–1234, 2009.

Engbert R, Kliegl R. Microsaccades uncover the orientation of covert attention. *Vision Res* 43: 1035–1045, 2003.

Everling S, Dorris MC, Klein RM, Munoz DP. Role of primate superior colliculus in preparation and execution of anti-saccades and pro-saccades. *J Neurosci* 19: 2740–2754, 1999.

Evinger C, Kaneko CR, Fuchs AF. Activity of omnipause neurons in alert cats during saccadic eye movements and visual stimuli. *J Neurophysiol* 47: 827–844, 1982.

Findlay JM, Harris LR. Small saccades to double-stepped targets moving in two dimensions. In: *Theoretical and Applied Aspects of Eye Movement Research*, edited by Gale AG, Johnson F. Amsterdam: Elsevier, 1984, p. 71–78.

Freedman EG. Coordination of the eyes and head during visual orienting. *Exp Brain Res* 190: 369–387, 2008.

Fuller JH. Head movement propensity. *Exp Brain Res* 92: 152–164, 1992.

Gandhi NJ, Keller EL. Spatial distribution and discharge characteristics of superior colliculus neurons antidromically activated from the omnipause region in the monkey. *J Neurophysiol* 78: 2221–2225, 1997.

Goossens HH, Van Opstal AJ. Dynamic ensemble coding of saccades in the monkey superior colliculus. *J Neurophysiol* 95: 2326–2341, 2006.

Guillaume A. Saccadic inhibition is accompanied by large and complex modulations when induced by visual backward masking. *J Vis* 12: 5, 2012.

Hafed ZM, Clark JJ. Microsaccades as an overt measure of covert attention shifts. *Vision Res* 42: 2533–2545, 2002.

Hafed ZM, Ignashchenkova A. On the dissociation between microsaccade rate and direction after peripheral cues: microsaccadic inhibition revisited. *J Neurosci* 33: 16220–16235, 2013.

Hooge IT, Erkelens CJ. Control of fixation during a simple search task. *Percept Psychophys* 58: 969–976, 1996.

Keller EL, Edelman JA. Use of interrupted saccade paradigm to study spatial and temporal dynamics of saccadic burst cells in superior colliculus in monkey. *J Neurophysiol* 72: 2754–2770, 1994.

Ludwig CJ, Gilchrist ID, McSorley E. The remote distractor effect in saccade programming: channel interactions and lateral inhibition. *Vision Res* 45: 1177–1190, 2005.

Ludwig CJ, Mildinhall JW, Gilchrist ID. A population coding account for systematic variation in saccadic dead time. *J Neurophysiol* 97: 795–805, 2007.

McIntosh RD, Buonocore A. Saccadic inhibition can cause the remote distractor effect, but the remote distractor effect may not be a useful concept. *J Vis* 14: 15, 2014.

Munoz DP, Waitzman DM, Wurtz RH. Activity of neurons in superior colliculus during interrupted saccades. *J Neurophysiol* 75: 2562–2580, 1996.

Munoz DP, Wurtz RH. Saccade-related activity in monkey superior colliculus. I. Characteristics of burst and buildup cells. *J Neurophysiol* 73: 2313–2333, 1995.

Nuthmann A, Smith TJ, Engbert R, Henderson JM. CRISP: a computational model of fixation durations in scene viewing. *Psychol Rev* 117: 382–405, 2010.

Olivier E, Dorris MC, Munoz DP. Lateral interactions in the superior colliculus, not an extended fixation zone, can account for the remote distractor effects. *Behav Brain Sci* 22: 694–695, 1999.

Ottes FP, Van Gisbergen JA, Eggermont JJ. Visuomotor fields of the superior colliculus: a quantitative model. *Vision Res* 26: 857–873, 1986.

Paré M, Hanes DP. Controlled movement processing: superior colliculus activity associated with countermanded saccades. *J Neurosci* 23: 6480–6489, 2003.

Reichle E, Pollatsek A, Fisher D, Rayner K. Toward a model of eye movement control in reading. *Psychol Rev* 105: 125–157, 1998.

Reingold EM, Stampe DM. Saccadic inhibition and gaze contingent research paradigms. In: *Reading as a Perceptual Process*, edited by Kennedy A, Radach R, Heller D, Pynte J. Amsterdam: Elsevier, 2000, p. 119–145.

Reingold EM, Stampe DM. Saccadic inhibition in complex visual tasks. In: *Current Oculomotor Research: Physiological and Psychological Aspects*, edited by Becker W, Deubel H, Mergner T. New York: Plenum, 1999, p. 249–255.

Reingold EM, Stampe DM. Saccadic inhibition in reading. *J Exp Psychol Hum Percept Perform* 30: 194–211, 2004.

Reingold EM, Stampe DM. Saccadic inhibition in voluntary and reflexive saccades. *J Cogn Neurosci* 14: 371–388, 2002.

Reingold EM, Stampe DM. Using the saccadic inhibition paradigm to investigate saccadic control in reading. In: *The Mind's Eye: Cognitive and Applied Aspects of Eye Movement Research*, edited by Hyona J, Radach R, Deubel H. Amsterdam: Elsevier, 2003, p. 347–360.

Rizzolatti GG, Buchtel HA, Camarda RR, Scandolara CC. Neurons with complex visual properties in the superior colliculus of the macaque monkey. *Exp Brain Res* 38: 37–42, 1980.

Rolfs M, Kliegl R, Engbert R. Toward a model of microsaccade generation: the case of microsaccadic inhibition. *J Vis* 8: 1–23, 2008.

Sparks DL. The brainstem control of saccadic eye movements. *Nat Rev Neurosci* 3: 952–964, 2002.

Sparks DL, Mays LE. Signal transformations required for the generation of saccadic eye movements. *Annu Rev Neurosci* 13: 309–336, 1990.

Trappenberg TP, Dorris MC, Munoz DP, Klein RM. A model of saccade initiation based on the competitive integration of exogenous and endogenous signals in the superior colliculus. *J Cogn Neurosci* 13: 256–271, 2001.

Van Gisbergen JA, Van Opstal AJ, Tax AA. Collicular ensemble coding of saccades based on vector summation. *Neuroscience* 21: 541–555, 1987.

Van Loon EM, Hooge IT, Van den Berg AV. The timing of sequences of saccades in visual search. *Proc Biol Sci* 269: 1571–1579, 2002.

van Opstal AJ, Goossens HH. Linear ensemble-coding in midbrain superior colliculus specifies the saccade kinematics. *Biol Cybern* 98: 561–577, 2008.

Walshe RC, Nuthmann A. Mechanisms of saccadic decision making while encoding naturalistic scenes. *J Vis* 15: 21, 2015.

Weber H, Aiple F, Fischer B, Latanov A. Dead zone for express saccades. *Exp Brain Res* 89: 214–222, 1992.