Spatial primes produce dissociated inhibitory effects on saccadic latencies and trajectories

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Abstract

In masked priming, a briefly presented prime can facilitate or inhibit responses to a subsequent target. In most instances, targets with an associated response that is congruent with the prime direction speed up reaction times to the target (a *positive compatibility effect*; PCE). However, under certain circumstances, slower responses for compatible primes are obtained (a *negative compatibility effect;* NCE). NCEs can be found when a long pre-target delay is used. During the delay, inhibition is assumed to take place, and therefore an effect on saccade trajectories may also be expected. In a previous study, we found the effects of inhibition on response times and trajectories to be dissociated, but this experiment varied the timing of several aspects of the stimulus sequence and it is therefore unclear what caused the dissociation. In the present study, we varied only one aspect of the timing, but replicated the dissociation. By varying just the pre-target delay, we found a PCE for a short delay, and an NCE for a long delay, but saccade trajectories deviated away from prime directions in both conditions. This suggests dissociated inhibitory effects of primes on response times and saccade trajectories.

Keywords: negative compatibility effect, saccade, priming, inhibition

1. Introduction

Visual cues such as arrows presented at fixation have been shown to influence manual and saccadic responses to subsequent target stimuli by producing faster reaction times when the two are at corresponding spatial locations (Kuhn & Benson, 2007; Posner, 1980). These cues can affect responses to the target even when presented for a very brief time, rendering the cue almost invisible. This phenomenon is known as masked priming, in which the cue is referred to as a 'prime'. In most instances, primes with an associated response compatible with the target direction produce faster response times to the target. However, under certain conditions, congruent primes have been shown to slow responses to the target (Eimer & Schlaghecken, 1998). This 'negative compatibility effect' (NCE) has been replicated numerous times (Eimer, 1999; Klapp & Hinkley, 2002; Schlaghecken & Eimer, 2002) and appears to be predominant under conditions of low prime visibility, but can also appear in situations where prime detection is above chance levels (Schlaghecken et al., 2007; Sumner, Tsai, Yu, & Nachev, 2006).

In typical priming, the prime firstly automatically activates a congruent response. When a congruent target is then presented, it benefits from the response preparation of the prime, resulting in faster response times for congruent prime-target combinations. For incongruent combinations, the automatic activity for the prime needs to be inhibited, before the correct target response is initiated, which slows responses. This pattern of response times is the positive compatibility effect (PCE). Evidence for this can be seen in EEG measures such as those associated with response preparation (e.g. lateralised readiness potential), which show an early response to the prime, followed by a later, stronger response to the target (Kopp et al., 1996).

The opposite response time pattern found in NCEs can be explained with one additional assumption - that the automatic activity following the prime is suppressed over

time to a level below baseline. This assumption is made by the motor inhibition hypothesis (Eimer & Schlaghecken, 1998; Sumner, 2007). When a prime-congruent target is presented during this suppression of prime-related activity, it will lead to slower responses than an incongruent, and even a neutral prime. So, with longer intervals between the prime and the target, where the activity associated with the prime is suppressed, we expect to find an NCE. This below-baseline suppression is supported by the finding that, when given a free choice of whether to execute left or right responses, participants generally choose the response opposite to the inhibited prime despite being unaware of such behaviour (Klapp & Hinkley, 2002).

Positive compatibility effects (PCEs) are generally found when the overall temporal inter-stimulus interval (ISI) between prime offset and target onset is 0-60 ms (Sumner, 2007). It is at a longer ISI of 100-200ms that NCEs begin to appear with performance benefits for incongruent trials. Primes that take a different form to traditional arrows, such as circles with rightward or leftward gaps, are capable of producing NCEs, but tend to begin functioning in this way at longer exposure times, arguably due to eliciting weaker motor activation in the first place (Schlaghecken et al., 2007). Although differing in levels of effect, NCEs are robustly found across varying conditions, and much support exists for the motor inhibition model (Schlaghecken et al., 2007; Sumner, 2007; Vainio, 2011). The inhibitory processes that produce NCEs may be generated at two different levels – they could be self-generated by the motor system itself, or could be reactions to stimuli within the task (Boy et al., 2008; Jaśkowski, 2008).

Some models attempt to explain NCEs without invoking inhibition. According to the object-updating account (Lleras & Enns, 2004), the structure presented after the prime, but before the target (the mask) is essential. Only masks with a task-relevant structure in terms of target identity can produce an NCE. Task relevant features present in the mask are assumed to update the motor system following prime-related automatic activation, producing

preparatory activity for an alternative response, resulting in faster responses to incongruent targets. The object-updating account therefore suggests that reaction time benefits for incongruent trials in NCEs may not be caused by inhibition of the prime response, but instead by positive priming for the alternative response by a task-relevant mask. A number of experiments have shown that sequences using task-irrelevant masks produce PCEs rather than NCEs despite successful reduction of prime visibility. This lack of an influence of masks that do not have target-related features is attributed to the masks not updating the motor system with preparation for the alternative response (Lleras & Enns, 2004). In this context, it is important to note that the task-relevance of a mask may not only depend on geometric and structural similarities between target and mask (Jaśkowski & Przekoracka-Krawczyk, 2005; Lleras & Enns, 2006), but also on spatial and temporal similarities (Lleras & Enns, 2006).

These accounts have been useful in revealing alternative conditions under which NCEs can be obtained, but motor inhibition appears to be the main source of the NCE when task-irrelevant masks (e.g. random line masks) are used (Sumner, 2008). The inhibitory processes underlying the NCE are still not fully understood however and further evidence is required. One approach to improving understanding of the inhibitory processes involved is to examine other behavioural responses associated with motor inhibition, and compare the resulting effects. One such phenomenon is that of curved saccade trajectories. Saccade trajectories have been shown to provide a sensitive measure of response inhibition (Sheliga et al., 1994; Doyle & Walker, 2001; McSorley et al., 2006; Van der Stigchel & Theeuwes, 2005; Van der Stigchel et al., 2007). Trajectories of saccades are often found to deviate away from a previously attended location, or from the location of a peripheral distractor onset. Saccade trajectories have also been shown to deviate away from attended locations during inhibition of return (IOR), where slower reaction times are recorded when responses are made to cued rather than uncued peripheral locations (Godijn & Theeuwes, 2004; Theeuwes

& Van der Stigchel, 2009). Saccade deviations are attributed to inhibition in the spatial maps that encode potential saccade targets, such as that thought to be formed by neurons in the superior colliculus (SC) (Aizawa & Wurtz, 1998; McPeek, 2006; McPeek, Han, & Keller, 2003). Evidence for the role of inhibition in these maps was found by directly recording from cells in the SC - positive curvature was found following microstimulation of SC cells, with increased activation for distractor locations correlating with increased curvature (McPeek, Han, & Keller, 2003; McPeek, 2006). The inhibition of the distractor-related activity may depend on inhibitory projections from the substantia nigra rather than on top-down signals from cortical oculomotor centres (White, Theeuwes, & Munoz, 2011, 2012). Similar to the motor inhibition account of the NCE, the modulation of trajectory deviation has been found to depend on the time course, with deviation towards occurring for short latency responses and deviation away occurring at longer latencies (McSorley et al., 2006).

Because of the strong similarities between the assumed underlying mechanisms of the NCE and saccade trajectory deviations, the two phenomena have been studied in conjunction, by adopting a paradigm in which saccadic response times and trajectories were measured under highly similar conditions (Hermens et al., 2010). The expectation in this study was that saccades would deviate in the direction of primes during PCE reactions, but away from the direction of primes with NCEs that allow inhibition to be applied to that location in the motor map. In contrast to this prediction, strong trajectory deviations away from the prime directions were found during PCE reactions with non-significant deviation under NCE conditions (Hermens et al., 2010). This suggested a general dissociation between the activation and inhibition processes that lead to the PCE/NCE effects, and the inhibitory spatial processes behind saccade curvature, but it was unclear what could have led to this dissociation, because the PCE and NCE effects in the study were generated by varying several aspects of the stimulus timing. For this reason, a number of possible factors were

identified, including the durations of the prime, the mask and the delays, which may all have contributed to the dissociation of the two effects (NCE and saccade trajectories).

To establish the source of the dissociation between saccadic response times and saccade trajectories, the present study keeps all the stimulus timings constant across two conditions (short-ISI and long-ISI), and only manipulates the interval between the mask and target designed to vary the overall-ISI between prime offset and target onset. This interval between mask and target will be referred to as the 'pre-target-delay' to distinguish it from the overall-ISI. As in Hermens et al. (2010), saccadic response times and curvature are measured, by the inclusion of prime-and-target congruent and incongruent trials as well as oblique 'probe' trials aimed to measure trajectory curvature.

By keeping all stimulus factors constant except for the pre-target-delay, we can directly test the influence of the overall-ISI as a critical factor in the PCE and NCE. The overall-ISI has previously been shown to be an important factor in response times (e.g. Boy & Sumner, 2010), but its influence on saccade trajectories is unclear. It is predicted that short-ISI conditions will produce PCEs while long-ISI conditions will show NCEs in saccade latency. A dissociation between response time effects and curvature effects is expected only if different underlying activation/inhibition processes cause the two effects. After pilot testing[1], we found a stimulus combination in which a variation of just the pre-target-delay led to a PCE in one condition and an NCE in another condition. With this combination, we tested a total of 31 participants to examine whether a similar pattern on saccade trajectories could be found.

[1] 12 participants were tested in an initial pilot with 100ms masks and a 50ms pre-target delay for the long-ISI condition (no delay for short-ISI). Both short and long-ISI conditions produced NCE effects, suggesting that the overall-ISI was too long. For the experiment in the current paper, we reduced the mask duration to 40ms in order to get PCE and NCE effects in short and long-ISI conditions respectively.

Methods

2.1.1. Participants

Thirty-one participants (12 male), aged between 19-40 years took part in the experiment. These included students at Royal Holloway University of London and some of the authors. Participants demonstrated having understood the instructions and all reported normal or corrected-to-normal vision. All participated voluntarily without receiving reimbursement. The experimental procedures were approved by the local ethics committee.

2.1.2. Apparatus

An Eyelink II system (SR Research Osgood, ON, Canada) tracked and recorded binocular eye movements at a sampling rate of 500 Hz (pupil-only mode) using two cameras mounted on chin and forehead rests. The system was positioned 57cm from the 21inch CRT screen on which all stimuli were presented (100 Hz refresh rate). An Intel Pentium 4 computer controlled the eye tracking system. This was connected to a second AMD Athlon 2400+ computer which controlled the stimulus screen and computed the data received from the eye tracker.

2.1.3. Stimuli and Design

The stimulus sequence in all trials consisted of a central fixation point, a prime, a mask, and a target, all of which were displayed in white on a black background (see Figure 1 below).

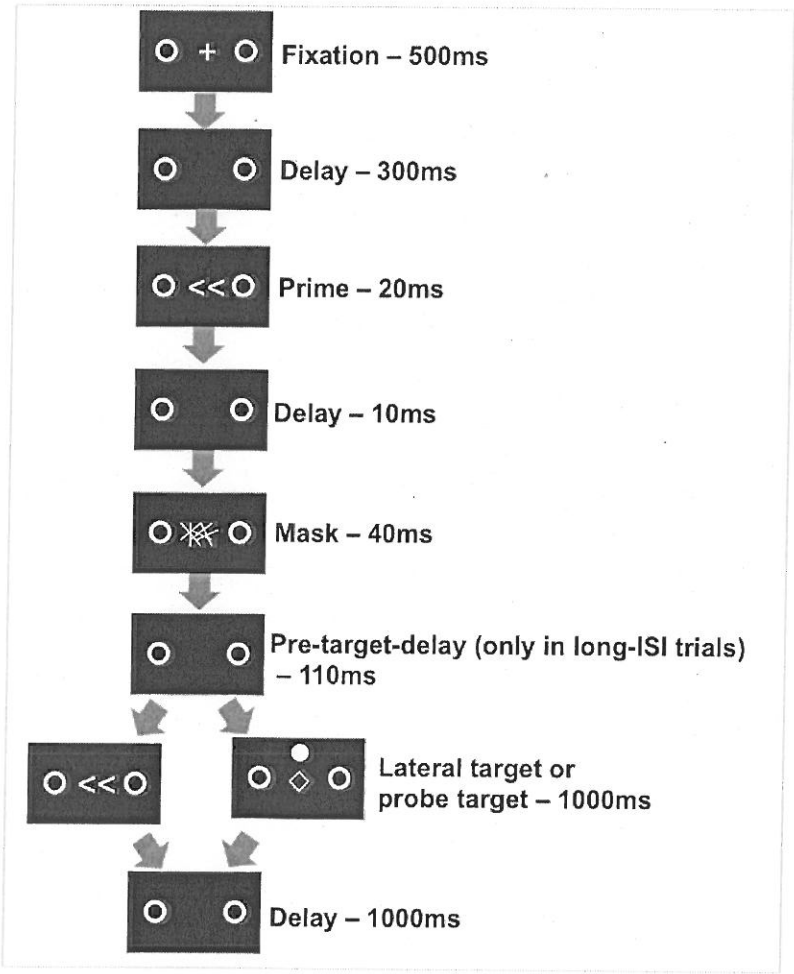

Figure 1. *Illustration of stimulus sequence and stimulus timings across conditions. A centrally presented fixation symbol was replaced by the prime for 20ms (2 retraces of the CRT screen), a delay of 10ms, a mask for 40ms, and a pre-target delay of 0ms or 110ms until the target was presented. Participants were asked to look at the placeholder indicated by the target or to the dot presented above fixation (if present).*

Circular placeholders with a diameter of 0.6° were permanently present on right and left sides of the screen at 7° from the centre, from fixation to target offset. The fixation symbol measured 1° horizontally and vertically in the form of a '+' sign. Primes were composed of

two rightward ('>>') or two leftward ('<<') arrows horizontally measuring 0.5° with a separation of 0.7°. The mask was kept constant across all trials and formed a patch measuring 1.8° vertically and 2° horizontally consisting of 30 randomly oriented intersecting lines. Targets in horizontal saccade trials were identical in structure to the prime arrows. Vertical saccade trials for measuring curvature (or 'probe' trials) used a central diamond probe measuring 1° horizontally and vertically in place of the arrow targets, and occurred with the simultaneous onset of a filled white circular stimulus 0.5° in diameter and placed 8° vertically upward from the centre of the screen to which saccades were made.

Participants each completed two different types of trial blocks, namely short-ISI and long-ISI trials. Each block consisted of 84 trials in total. Within each block, 48 trials were horizontal trials (rightward or leftward targets). These consisted of either a congruent prime and target, or a prime and target pointing in opposite directions, each with an equal chance of occurring (24 of each). These congruent trials and incongruent trials were further divided into an equal number of rightward and leftward responses (12 of each). The remaining 36 trials in each block were the vertical trials. These used either a rightward/leftward arrow as the prime, or an 'X' symbol (as a baseline for the trajectory measure) and each contributed to an equal number of vertical 'probe' trials (12 of each). Trials with the 'X' symbol (which itself measured 0.6°) were used to obtain a baseline measure of saccade curvature for each participant, from which changes in saccade deviations on arrow primed vertical trials could subsequently be determined. The presentation order of all trials in each block was random.

There were four blocks in total for each participant (two short-ISI and two long-ISI). In order to compensate for practice and fatigue effects, blocks were presented in A-B-B-A format. In addition, the first block to be presented was counter-balanced across participants.

2.1.4. Procedure

Participants were seated in the dimly lit experiment room with their head resting on the chin and headrest onto which the eye-tracker cameras were attached. An initial saccadic calibration process was performed before the start of the trials where participants were instructed to fixate a circular stimulus as it appeared at 9 points on the screen (3x3 grid presented in a random sequence). This calibration process was repeated if necessary until adequate alignment with eye positions was obtained. A standard Eyelink drift correction procedure was also performed to compensate for any post-calibration head movements every 21 trials within a block.

Participants were asked to respond quickly and accurately throughout the experiment avoiding head movements and blinking within trials when possible. It was made clear that responses should only to be executed to the target stimulus that followed the initial 'flashed' figures (prime and mask). Following a block of 10 practice trials, participants performed the four experimental blocks. The timing of the stimuli (described in detail above) was as follows. All trials began with a 500ms central fixation point followed by a 300ms blank screen (except for placeholders). A 20ms prime, a 10ms blank screen (placeholders), and a 40ms mask were then presented. The mask was then followed by a short delay (0ms) or a long delay (110ms, see Figure 1). Targets appeared and remained on screen for 1000ms as responses were made, and a 1000ms entirely blank screen followed before fixation for the next trial.

2.1.5. Data Analysis

Saccadic response times were obtained from the horizontal saccade trials, whereas saccade curvature was measured on the vertical saccade probe trials. To extract saccades from the continuously sampled signal, the Eyelink's inbuilt algorithm was used, with a

combined 22 deg/s velocity and 8000 deg/s^2 acceleration criterion. Exclusion criteria removed trials in which a blink occurred during the saccade and trials in which the initial saccade following target onset was of insufficient amplitude (<3.8°) or was in the wrong direction. Trials in which the saccade was initiated less than 100ms after target onset or more than 2.5 times the standard deviation after the mean reaction time were also excluded, along with probe trials with a peak deviation of over 50% of the saccade amplitude (Hermens et al., 2010). Only data from the right eye were analysed.

To compute saccade curvature in probe trials, the peak trajectory deviation (pixels) from the straight line connecting the start and end points of the saccade was divided by the total saccade amplitude (pixels). The change in curvature was then calculated by subtracting the mean baseline curvature obtained from 'X' primed probe trials from the mean curvature in arrow cued probe trials for each participant, resulting in a measure representing deviations as a proportion (dimensionless) of saccade amplitude relative to baseline.

Results

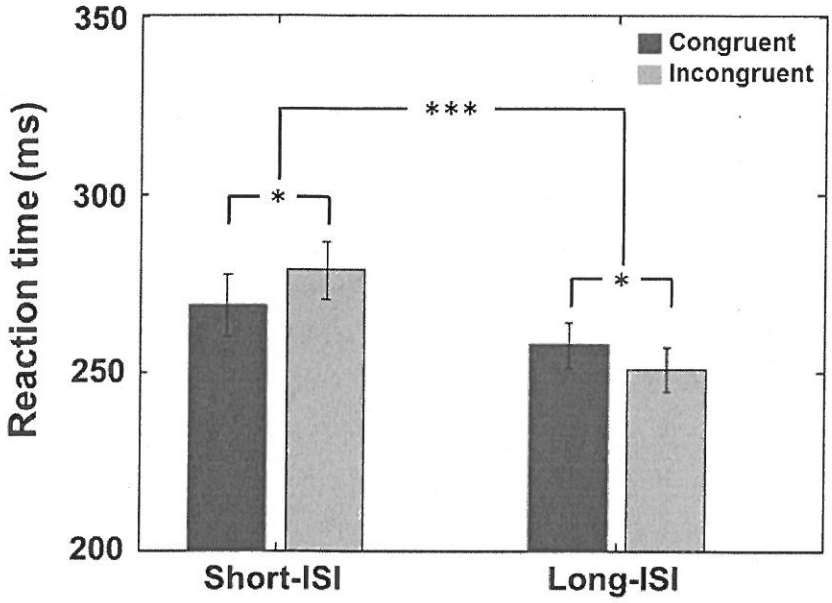

Figure 2. *Mean saccade response times (ms) from 31 participants for short and long ISI blocks with congruent and incongruent trials (*p < 0.05 (one-tailed), ***p < 0.001). Error bars show the standard error of the mean across participants.*

Mean reaction times for horizontal saccade trials under both ISI conditions are shown in Figure 2. A two-factor repeated measures ANOVA showed a main effect of ISI, with the short ISI leading to longer latencies, ($F(1,30) = 21.39$, $p < 0.001$). No main effect of congruency was found, ($F(1,30) = 0.18$, $p > 0.05$), but a significant interaction between ISI and congruency was found, ($F(1,30) = 27.53$, $p < 0.001$). Paired samples t-tests showed the predicted and well-established effects of PCE and NCE. There were significantly faster response times for congruent than incongruent trials (PCE) in the short-ISI condition, ($t(30) = -2.34$, $p < 0.05$, one-tailed). Conversely, the long-ISI condition exhibited faster response times for incongruent trials than congruent trials (NCE), ($t(30) = -1.84$, $p < 0.05$, one-tailed). This means a PCE was found in the short-ISI condition while an NCE was found in the long-

ISI condition, purely as a result of a 110ms pre-target-delay following the mask in the latter condition.

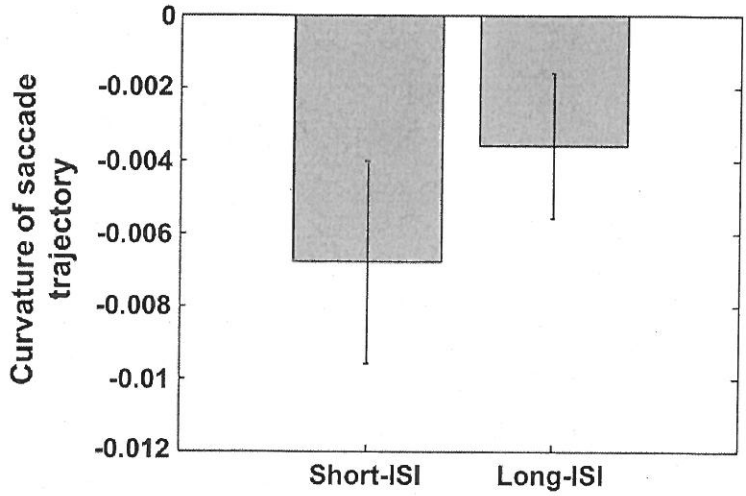

Figure 3. *Saccade trajectory deviations (as a proportion of saccade amplitude) following directional primes (relative to neutral baseline) in short and long ISI conditions. Negative curvature indicates deviation away from the direction of the prime. Error bars show the standard error of the mean.*

Mean curvature results for saccade trajectory are shown in Figure 3. The reaction times of vertical saccades made on probe trials (used to calculate trajectory deviations) were slower for the short-ISI (mean: 232.99ms) than the long-ISI condition (mean: 217.49ms), ($t(30) = 2.73$, $p < 0.05$, two-tailed), which should be taken into account when interpreting saccade curvature. As shown in Figure 3, deviations away from prime directions were found in both short-ISI and long-ISI conditions. Curvature away from the prime in the short-ISI condition was significantly different to 0, ($t(30) = -2.44$, $p < 0.05$, two-tailed). Curvature in the long-ISI condition showed a trend towards being different from 0, ($t(30) = -1.79$, $p = 0.08$, two-tailed). There was no difference in curvature between the two conditions, ($t(30) = -$

1.02, $p > 0.05$, two-tailed). This suggests that the introduction of a delay in the long-ISI condition had no significant effect on saccade curvature, despite the effect of saccade reaction time. Furthermore, although primes in the short-ISI condition facilitated congruent responses (producing a PCE), saccade trajectories by contrast showed a significant curvature *away* from the direction of the prime.

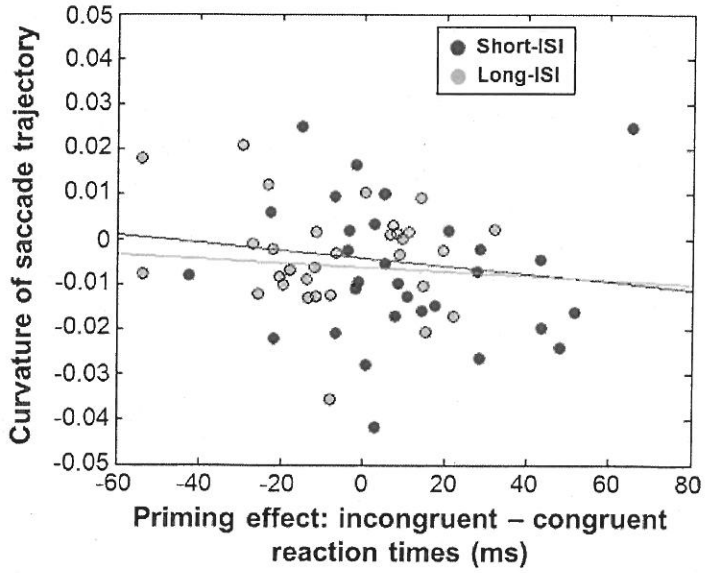

Figure 4. *Scatterplot of saccade trajectory deviations (peak deviation as a proportion of saccade amplitude) relative to baseline against priming effects (difference between reaction times for responses incongruent and congruent with the prime). Positive values on the y-axis indicate curvature towards, and negative values away from, the prime direction. Positive values on the x-axis indicate a PCE RT facilitation effect and negative values an NCE RT inhibitory effect. Each participant has two data points: one for each condition (short-ISI and long-ISI).*

To examine the relation between response time effects and saccade curvature, these two measures are plotted against each other for each participant in Figure 4. As suggested by

an absence of a clear trend and analysis of correlations, no significant association was found between saccade curvature and priming effect in the long-ISI condition ($r = -0.16$, $p = 0.39$), the short-ISI condition, ($r = -0.07$, $p = 0.7$) or both ISI conditions together ($r = -0.14$, $p = 0.28$). Therefore, reaction times (and the PCE/NCE) had no clear association with trajectory deviations.

4. General Discussion

The results of our experiment showed that when overall-ISIs are reduced by only varying the pre-target delay, saccadic latencies show an NCE for a long pre-target delay and a PCE for a short pre-target delay. These results are consistent with earlier studies that have shown that variations of the pre-target delay can reverse performance benefits for congruent trials into benefits for incongruent trials (Boy & Sumner, 2010). In contrast, saccade trajectories did not differ across these conditions, with deviation away from the prime location appearing to be independent of the effect on saccadic reaction times. Our data are clear in showing that there is no pattern of curvature towards the prime direction during facilitatory PCE priming, and curvature away during inhibitory priming. No common inhibition across the two effects appears to be present.

The present results extend findings that show performance benefits for responding to a target that is incongruent with an earlier prime (NCEs) (Eimer, 1999; Eimer & Schlaghecken, 1998; Klapp & Hinkley, 2002; Schlaghecken & Eimer, 2002). This occurs when the interval between prime and target (overall-ISI) is large, usually as a result of a longer mask. We replicate previously reported findings showing that a longer pre-target delay after the mask (long-ISI conditions) can turn PCE responses into NCE responses (e.g. Boy & Sumner, 2010). Our findings support the notion that the overall length of time between prime

offset and target onset is a critical factor in determining whether a PCE or NCE is exhibited, independent of prime and mask durations (Sumner, 2007).

The timing manipulations that created the PCE and NCE effects in the present study, fell within the relevant guidelines proposed by Sumner (2007), at 0-60ms for PCEs and 100-200ms for NCEs. These seem relevant to saccades as well as manual responses according to the present results. The inhibition response underlying NCEs seems to be a reflexive mechanism self-generated by the motor system following activity from the prime (Eimer & Schlaghecken, 1998; Eimer & Schlaghecken, 2003). There is evidence to suggest that the external mask stimulus can trigger inhibition (Boy et al., 2008; Jaśkowski, 2008), but in the case of the present experiment, the self-generated inhibition hypothesis seems better suited to explain the data, especially given some of our pilot results in which a different mask duration (see footnote on page 7) had little effect on saccade latency effects when overall-ISI was kept the same. If the external mask was responsible for triggering inhibitory mechanisms, significantly different inhibitory effects might have been expected when using longer mask durations (i.e., a more salient mask), which was not the case. In contrast, self-generated inhibition would not predict differences with varying mask durations when the overall-ISI is maintained.

Previous studies have provided evidence for a triphasic pattern of priming activity as responses are increasingly delayed. The interval durations for which NCEs are found, are followed by another facilitation phase producing positive priming at an overall-ISI of approximately 500ms (Sumner & Brandwood, 2008). These complex oscillation patterns in priming seem to be an inherent characteristic of the motor system itself rather than a reaction to a mask stimulus. Sumner (2008) found that mask-induced priming as assumed by an object-updating model (Jaśkowski & Przekoracka-Krawczyk, 2005; Lleras & Enns, 2004; Lleras & Enns, 2006) can contribute to NCEs under certain task-relevant masking conditions,

but that inhibition is primarily responsible for NCEs beyond these conditions. Computational modelling of inhibitory processes also provides a simple and plausible account that explains a large range of masking data (Bowman, Schlaghecken, & Eimer, 2006).

Saccade trajectories showed a consistent trend away from directions of primes, in agreement with our earlier study (cf. Hermens et al., 2010). Also in agreement with Godijn & Theeuwes (2004), our results show that effects of symbolic cues on curvature are independent of their effects on response times. We show that this dissociation holds true even when all stimuli in the visual sequence are held constant and only a pre-target delay is manipulated. These results suggest that saccade deviations may result from inhibitory activity in the saccade spatial maps (White, Theeuwes, & Munoz, 2011, 2012) while reaction times appear to involve separate processes (Casteau & Vitu, 2012; Findlay & Walker, 1999; Godijn & Theeuwes, 2004).

Dissociated effects of primes on saccadic latencies and saccade trajectories were found in both the present study and that by Hermens et al. (2010), but these are not the first times that dissociations between saccade latency and spatial modulation effects have been reported. White et al. (2012) report that suppression at the location of a distractor can delay saccades without affecting trajectories. In other studies, the offset of central fixation before saccades reduces latency, while visual onsets, remote from the saccade goal, increase latency, but these manipulations have no, or small effects on landing position (Casteau & Vitu, 2012; Walker, Deubel, Schneider, & Findlay, 1997). Moreover, dissociations between IOR and saccade trajectories have been reported (Godijn & Theeuwes, 2004), and dissociations have been found for effects of moving stimuli on response times, error rates and saccade trajectory deviations (Hermens & Walker, 2012).

Models that propose separate programming of spatial and temporal aspects of saccade generation in two separate processing streams (e.g. Findlay and Walker, 1999) are consistent

with the current data, but there is uncertainty about the neural basis for these streams. In terms of saccade latency, a human lesion study has implicated a role for the human supplementary eye fields (SEF) in the NCE (Sumner et al., 2007). In this study, two cases of focal lesions of the SEF, and supplementary motor areas (SMA) revealed a reversal of typical masked prime effects (Sumner et al. 2007), showing PCEs under conditions that reliably produced NCEs in control participants. This suggests that the latency effects we find in our study may be mediated by the SEF.

When examining the spatial modulation of saccades, it has often been reported that trajectory effects reflect competitive interactions between separate populations of neurons in the SC. Neurons in the deeper layers of the SC are regarded as forming a two-dimensional representation of saccade goals for the control of saccade amplitude and direction (Munoz et al., 2000). The deviation of saccades, either towards, or away, from a competing location has been attributed to the level of distractor-related activity, or inhibition, at the time the saccade is triggered (McPeek et al., 2003). In addition, the substantia nigra pars reticulata (SNr) exerts inhibitory modulation over the SC, and may have an important role in saccade suppression (Hikosaka & Wurtz, 1985; Hikosaka et al., 2000). Many suggest that top-down inhibition at distractor locations is responsible for saccade deviation away from distractors (Van der Stigchel et al., 2007; Walker et al., 2006). More recently however, extracellular recordings in the monkey SC suggest no inhibition of distractor-related activity within the time frame one would expect during saccade deviation away from distractors (White et al., 2012). The frontal eye fields (FEF) are another potentially good candidate for top-down inhibition of distractor locations (Walker et al., 2006) and FEF cells show reduced activity at the distractor location when saccades curve away from a distractor and increased activity when curving towards a distractor (McPeek, 2006).

The evidence on the neural basis of saccade latency and curvature effects is far from conclusive. The most surprising aspect of the current findings is that during traditional PCE reaction times, saccade trajectories significantly curved away from the direction of the prime, even though responses congruent with the prime were facilitated in terms of latency. Current models of saccade programming cannot easily account for this dissociation. Even given the proposed separate streams for the programming of temporal and spatial aspects of saccade generation (Findlay & Walker, 1999), it remains unclear how opposing temporal latency and spatial curvature effects can arise from the same spatial prime. Inhibitory modulation still seems to offer the most adequate explanation for saccade curvature away from distractors, but future work must explore the neural basis of such inhibition in saccade trajectory programming. In the case of the present data, it seems plausible that the latency-curvature effect dissociation is being caused by dissociated inhibitory modulation in the SEF and areas such as the FEF/SC in response to the spatial prime. Although current evidence offers no conclusive suggestion about the relative functions of these latter areas or their communication in influencing saccade curvature, it seems likely that activity in the SEF may be primarily responsible for the saccade latency effects while inhibitory modulation in the FEF/SC is responsible for the curvature effects.

The present results support the motor inhibition account of NCE reaction times, but suggest that inhibitory mechanisms underlying saccade curvature are part of another independent process. Our results support the idea that the overall-ISI from prime offset to target onset is a more important factor distinguishing between a PCE and NCE than the individual durations of the prime and mask. We contribute to previous findings by showing that a pre-target-delay can introduce NCE saccade latencies without leading to any corresponding reversal in saccade curvature effects. A single spatial prime is therefore

capable of creating a cascade of dissociated inhibitory patterns in maps across the oculomotor system.

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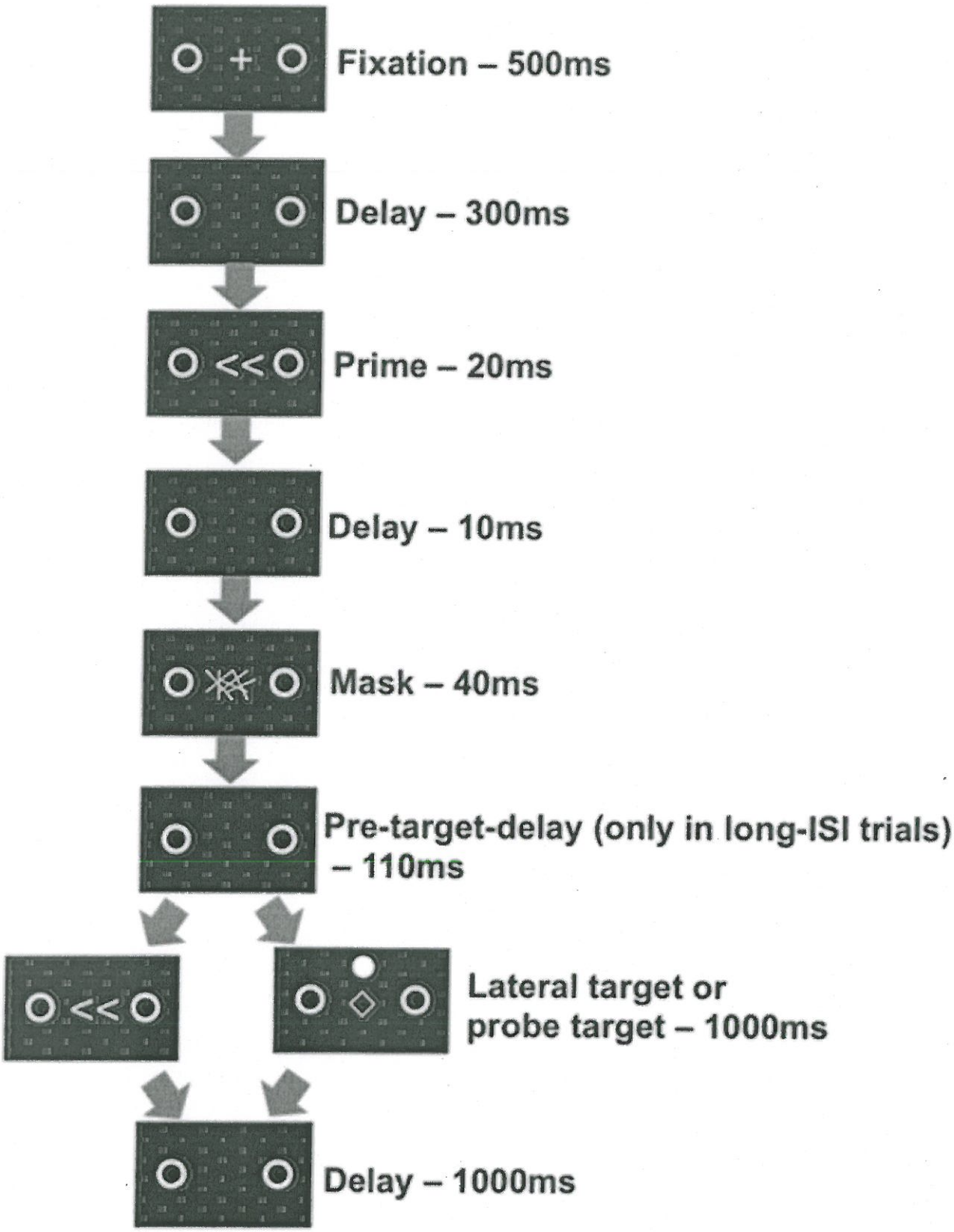

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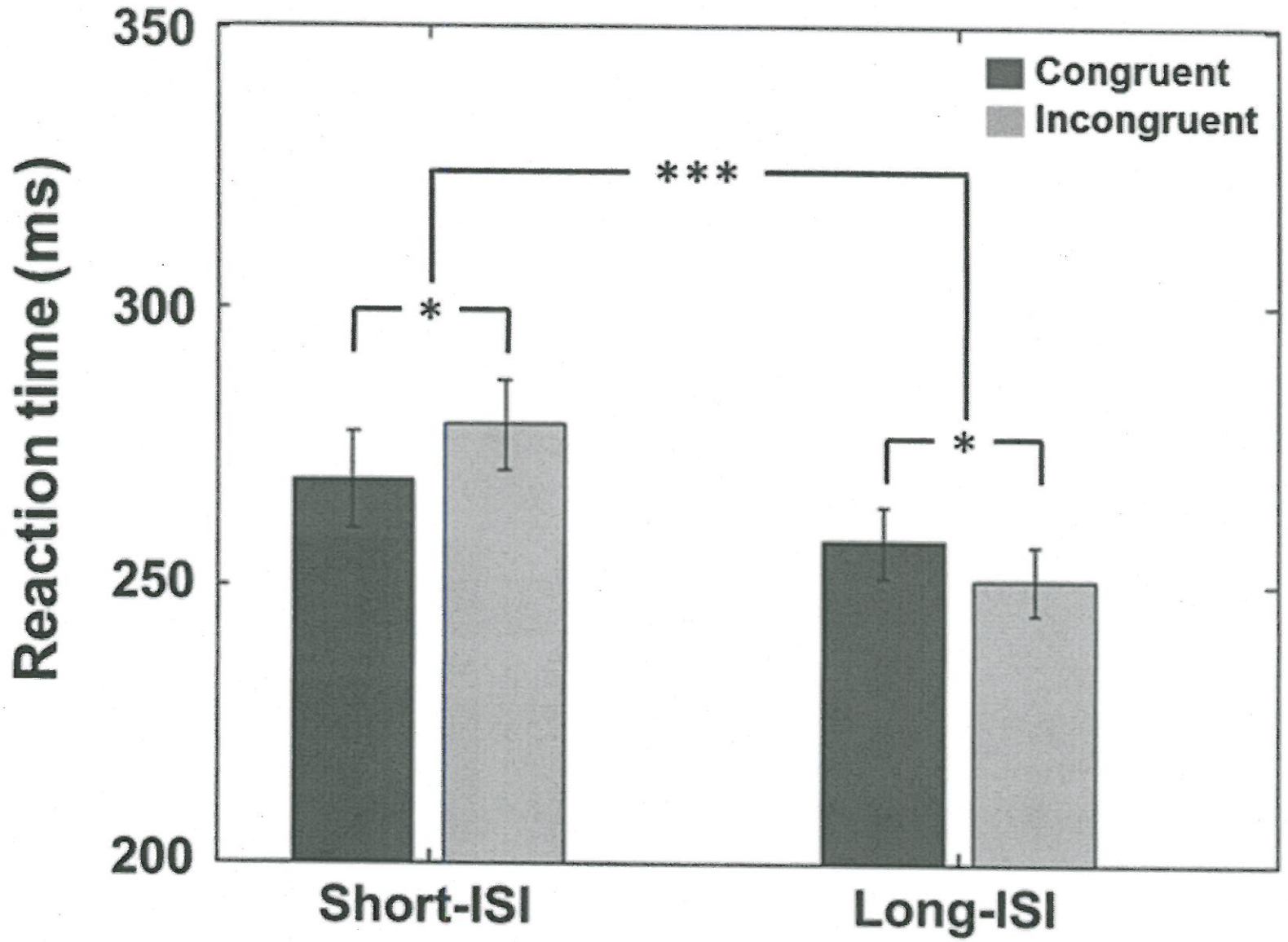

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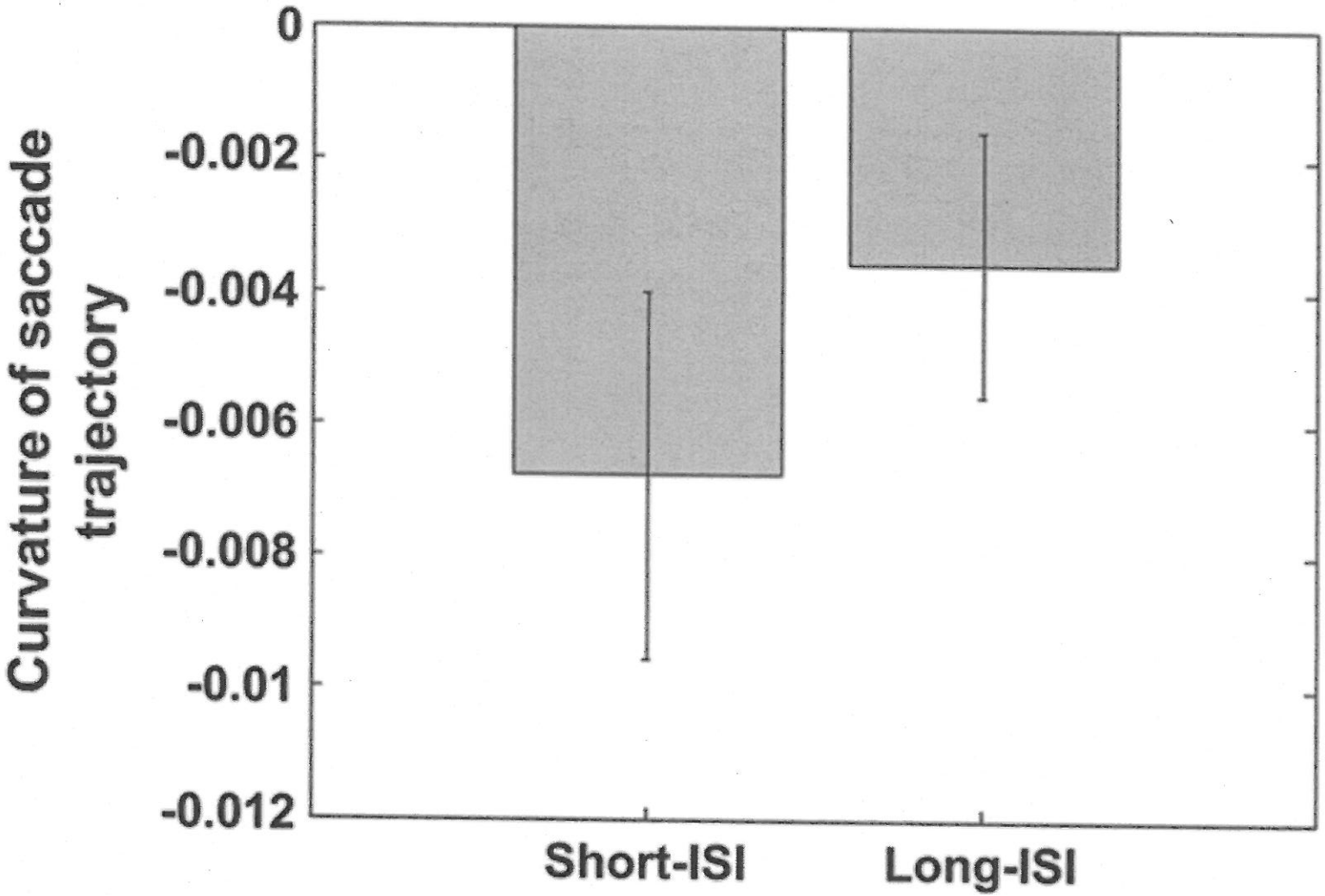

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