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Attentional dynamics mediated by subcortical mechanisms

Shai Gabay · Marlene Behrmann

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Abstract Following a salient cue that attracts attention to a specific spatial location, perceptual processing of information at that location is facilitated if the interval between the cue and target is brief, or, is inhibited if the interval between the cue and target is long. The mechanisms mediating these attentional dynamics continue to be the subject on ongoing debate. On one classic account, facilitation and inhibition of return (IOR) are two ends of a continuum, generated by the same underlying mechanism. Other accounts have postulated that these two attentional processes emerge from independent systems. To address these alternatives, we report data from three experiments in which a cue and its ensuing target are presented to the same or different eyes at varying cue-target intervals. Whereas the onset of facilitation was apparent earlier when the cue and target shared the eye-of-origin, the onset of IOR was not affected by the eye to which the cue and target were presented. This finding implicates at least some, if not full, independence in the system(s) that give rise to attentional facilitation and IOR, and, moreover, suggests that facilitation may be more reliant on subcortical levels of the visual pathways than IOR.

Keywords Spatial attention · Exogenous orienting · Inhibition of return · Subcortical mechanisms · Monocular presentation

S. Gabay (✉) · M. Behrmann
Department of Psychology, Carnegie Mellon University, Baker Hall 342c, Pittsburgh, PA 15213, USA
e-mail: shaigaba@gmail.com

Introduction

Following a salient, exogenous cue that attracts attention to a specific location in the environment, perceptual processing of information appearing at that location is facilitated if the interval between the cue and target is brief, or, is inhibited if the interval between the cue and target is long. For example, under conditions of exogenous spatial cueing (Posner & Cohen, 1984), at short stimulus onset asynchronies (SOAs), reaction time (RT) for valid trials (i.e., target and cue presented at same spatial location) is faster than for invalid trials (i.e., target and cue presented at differing spatial locations)—an effect generally referred to as attentional facilitation. At longer SOAs, the converse is true: RT is slower for valid than for invalid trials. This latter effect, termed inhibition of return (IOR), has generated keen interest and considerable discussion about its underpinnings and its relationship to attentional facilitation. Furthermore, in addition to being a perceptual phenomenon, IOR also manifests as a 'motor' phenomenon (Taylor & Klein, 1998). For example, Sumner, Nachev, Vora, Husain and Kennard (2004) demonstrated a dissociation between perceptual and motor IOR in the eye-movement system, in that perceptual IOR but not motor IOR is observed in response to S-cone stimuli that are presumably not processed by the SC (but see Hall & Colby, 2014). Also, IOR has also been demonstrated in space-based (Sapir, Hayes, Henik, Danziger, & Rafal, 2004; van Koningsbruggen, Gabay, Sapir, Henik, & Rafal, 2010) and in object-based coordinates (Becker & Egeth, 2000; Tipper, Jordan, & Weaver, 1999) and not only in retinotopic coordinates, suggesting potentially that more complex computations might be involved. There is still ongoing debate regarding the psychological and neural mechanisms underlying these facilitatory and inhibitory attentional effects, and several contrasting proposals have been offered to

explain the mechanism/s underlying the reflexive attentional dynamics. The present study focuses on the attentional dynamics that are engaged when participants perform a visual detection task, and thus the focus is on the more perceptual aspects of enhancement and inhibitory mechanisms.

Attentional dynamics: unitary mechanism

The well-accepted reorienting theory offers an explanation in which both of these attentional dynamics, facilitation and IOR, emerge from a single underlying process (Klein, 2000). The theory is framed in terms of attention operating as a spotlight and postulates that the presence of the spotlight at a particular location facilitates foraging and other search behaviors. In a complementary fashion, so as to avoid returning to locations already visited, the visual spotlight's return to previously attended locations is inhibited, resulting in the observed IOR (Klein, 2000). On this account, the engagement of the spotlight of attention at a specific location produces facilitation, and, conversely, disengagement of the spotlight from an attended location generates an inhibitory tag for that location. Accordingly, both facilitation and IOR emerge from a single orienting process.

Unitary neural mechanism

Consistent with this unitary mechanism account, Sereno, Lehky, Patel and Peng (2010) have proposed that both facilitation and IOR result from an intrinsic and ubiquitous property of neural dynamics, namely repetition suppression. They suggest that the presentation of the initial cue activates visual neurons in the corresponding receptive field in the superior colliculus (SC). A subsequent activation of those same neurons in response to the presentation of the target within a short temporal interval then gives rise to the facilitation. In contrast, if a sufficiently long temporal interval ensues between the appearance of the cue and the target, those neurons initially responsive to the target become quiescent or refractory, and this results in IOR. Thus, both facilitation and IOR can be captured by a single adaptive, neurophysiologically plausible mechanism. Evidence compatible with the idea of a single subcortical mechanism supporting exogenous attention comes from the finding that both facilitation and IOR are observed in the archer fish; because this species does not have a fully developed cortex, and has only a retino-tectal (but not a geniculostriate) visual pathway, both attentional dynamics are likely mediated by a non-cortical system (Gabay, Leibovich, Ben-Simon, Henik, & Segev, 2013). Similar ideas (although not focused specifically on subcortical regions) have also been proposed by Dukewich and Boehnke (2008), who offer a habituation account of the attentional dynamics, and by Lupiáñez (2010), whose views of detection cost are compatible with the underlying computational forces that drive both early facilitation and later inhibition.

Attentional dynamics: dissociable mechanisms

In contrast with the views of attentional dynamics as emergent properties of the same underlying mechanism, in their seminal work, Posner and Cohen (1984) used a double cue experiment (the second cue was central) and demonstrated that there was no facilitation of response to the target but IOR was evident. Also, the size of the IOR effect in the double cue condition was not smaller than in the single cue condition. These authors concluded that inhibition does not arise from attentional orienting but from energy change present at the cued position. Since these early groundbreaking studies, additional compelling evidence has accumulated supporting the dissociation between facilitation and IOR. Several researchers have suggested that exogenous facilitation and IOR overlap in time (Chica & Lupiáñez, 2009; Lupiáñez & Weaver, 1998; Posner & Cohen, 1984; Tassinari, Aglioti, Chelazzi, Peru, & Berlucchi, 1994), and therefore are likely generated by different underlying mechanisms. Also, by using nonpredictive peripheral cues with nonpredictive central orienting cues (either arrows or gaze), Martín-Arévaloa, Kingstone and Lupiáñez (2013) have demonstrated recently a dissociation between the involuntary orienting of spatial attention and the IOR effect. These latter authors concluded that IOR operates independently of involuntary spatial orienting.

Dissociable neural mechanisms

As noted above, the psychological evidence pertaining to the relationship between facilitation and IOR does not offer a clear consensus of opinion. The inconsistency in the evidence is apparent in the findings from neural investigations, as well as being alluded to above to some extent. Many researchers have suggested that IOR is generated by the superior colliculus (SC) (Berger & Henik, 2000; Posner, Rafal, Choate, & Vaughan, 1985; Ro, Shelton, Lee, & Chang, 2004; Sapir, Soroker, Berger, & Henik, 1999)—a subcortical structure involved in the programming and execution of eye movements. For example, IOR has been demonstrated in newborns (Simion, Valenza, Umilta, & Dalla Barba, 1995), and in hemianopic patients (Danziger, Fendrich, & Rafal, 1997). Furthermore, Sapir et al. (1999) measured nasal/temporal asymmetries as a marker of retino-tectal mediation (Rafal, Calabresi, Brennan, & Sciolto, 1989) and demonstrated a larger IOR effect when stimuli were presented to the nasal hemiretina, indicating retino-tectal mediation. Other studies

have also implicated a retino-tectal mediation of facilitation (Rafal, Henik, & Smith, 1991) and reduced facilitation was observed for patients with collicular degeneration (Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988). It should be noted, however, that the evidence that IOR or facilitation are mediated by afferents through the subcortical retinotectal tracts does not necessarily imply that the further processing of the visual signal does not also involve processing at a cortical level.

Neuropsychological data can also shed light on the issue of the neural correlate of these processes. Whereas a collicular lesion can adversely affect IOR but does not impact facilitation (Sapir et al., 1999), a deficit in oculomotor ability can adversely impact facilitation but has no effect on IOR (Gabay, Henik, & Gradstein, 2010; Smith, Rorden, & Jackson, 2004). This double dissociation between the patterns of facilitation and IOR suggests that these dynamics arise from independent mechanisms. Another indication for the dissociation between facilitation and IOR comes from the independence of facilitatory and inhibitory tagging in split-brain patients (Tipper et al., 1997). In this study, object-based IOR was evident when a tagged object moved within the same visual field but object-based facilitation was revealed when the tagged object moved between visual fields. Thus, this study showed that both facilitatory and inhibitory tagging had persisting object-based effects that outlasted the duration of the cue. Perhaps more relevant, a facilitatory tag transferred subcortically between hemisphere, whereas interhemispheric transmission of IOR was transferred between the cortices of the hemispheres through the corpus callosum. Finally, recent findings reveal that only exogenous facilitation (but not IOR) depends on motor preparation (Smith, Schenk, & Rorden, 2012), further endorsing the claim of a dissociation between the attentional dynamics.

Although there is some agreement that the SC is involved in generating IOR, several studies have proposed that the SC may be necessary but not sufficient for the computations needed for IOR and that, consequently, higher brain regions are also implicated in generating IOR. Moreover, the claim is that any collicular involvement in IOR dynamic is merely a reflection of a neural signal that takes place upstream, likely in posterior parietal cortex (Dorris, Klein, Everling, & Munoz, 2002). Clear evidence for the additional involvement of cortical (and not solely subcortical) systems in IOR comes from the finding that damage to the right parietal cortex, which results in hemispatial neglect, impairs IOR, as measured in a manual response task for repeated right-sided targets (Bourgeois, Chica, Migliaccio, de Schotten, & Bartolomeo, 2012). In addition, transcranial magnetic stimulation (TMS) over the right intraparietal sulcus or right temporo-parietal junction impairs IOR (Bourgeois et al. 2012; Chica, Bartolomeo, & Valero-Cabré, 2011), revealing that cortical areas, and specifically right parietal regions, also play a functional role in the occurrence of IOR. Most imaging studies also suggest that attentional orienting involves the fronto-parietal cortical network (Kincade, Abrams, Astafiev, Shulman, & Corbetta, 2005; Peelen, Heslenfeld, & Theeuwes, 2004).

One possible account for the relative role of cortical versus subcortical regions proposes that different forms of attentional orienting, such as exogenous and endogenous orienting, engage different neural systems. For instance, it has been suggested that endogenous orienting might involve more cortical regions than exogenous attention and that exogenous attention also recruits subcortical processing (Lovejoy & Krauzlis, 2009; McAlonan, Cavanaugh, & Wurtz, 2008; Robinson & Kertzman, 1995; Zackon, Casson, Zafar, Stelmach, & Racette, 1999); but see also (Lovejoy & Krauzlis, 2009; McAlonan et al. 2008). In one influential model of attentional orienting (Corbetta and Shulman (2002), dorsal and ventral fronto-parietal networks are responsible for endogenous and exogenous orienting of attention, respectively, with some neural overlap between the networks. As evident from this brief review of the existing findings, the neural findings are rather inconsistent with debates regarding the engagement of purely cortical, purely subcortical or some union of these systems in orienting.

Our understanding of the psychological and neural mechanisms supporting attention dynamics is obviously complicated by the differing empirical findings, as outlined above. It is also the case that reaching definitive conclusions based on existing methods is subject to the limitations of the different methods commonly used to study the neural origin of behaviour. For instance, neuropsychological (lesion) and TMS studies are vulnerable to diaschisis, that is, a deficit in one brain region can remotely adversely influence the normal functioning of another distinct region which itself is not directly affected. Thus, it is possible that a deficit in cortical (parietal) regions can adversely impact the usual function of subcortical regions (SC), confounding the interpretation of the behavior as being of cortical or subcortical origin. Findings from functional magnetic resonance imaging (fMRI) are also potentially ambiguous as the cortical activity might reflect cascaded activations that are subcortical in origin but that, by virtue of subcortical regions being small and having low activation signal (LaBar, Gitelman, Mesulam, & Parrish, 2001), these subcortical regions are harder to detect.

In the current study, in order to examine the detailed profile of both facilitation and IOR, we avoid any manipulation that potentially disrupts the normal functioning of the neural system and also circumvents the difficulties associated with neuroimaging subcortical regions. Specifically, the approach we adopt is one in which, using a behavioral method and a Wheatstone stereoscope to ensure controlled presentation to a single eye or to two eyes, we probe the attentional dynamics that occur during

transmission of signals through the subcortical, monocular portion of the intact visual system, and contrast those with signals that arise at the binocular, cortical portions of the visual system.

Monocular segregation versus binocular contribution to attention

Visual input, once received by the retina, is propagated in an eye-specific fashion through the early stages of visual processing. This monocular segregation is evident through the lateral geniculate nucleus up through the input layers of the primary visual cortex (Horton, Dagi, McCrane, & de Monasterio, 1990; Menon, Ogawa, Strupp, & Ugurbil, 1997). In contrast, extrastriate visual areas are mostly binocular, and, consequently, their activation is not eye-dependent (Bi, Zhang, Tao, Harwerth, Smith, & Chino, 2011).

It has been demonstrated that observers do not have explicit access to eye-of-origin information for a visual stimulus (Blake & Cormack, 1979; Schwarzkopf, Schindler, & Rees, 2010), and, as such, manipulating the cue and target's eye-of-origin, provides a useful tool for isolating monocular versus binocular neural channels. This approach has been used previously to address questions related to those we examine here. For example, using a binocular rivalry task, Kamphuisen, van Wezel, and van Ee (2007) have demonstrated that cueing attention to one of two binocularly presented stimuli prior to rivalrous viewing provided an advantage to the cued stimulus in subsequent binocular rivalry, regardless of the eye of presentation. This result implies that exogenously cuing a specific visual pattern is related to binocular visual areas (even though binocular rivalry has been demonstrated to implicate the lateral geniculate nucleus, see Haynes, Deichmann, & Rees, 2005; Wunderlich, Schneider, & Kastner, 2005). However, since this study examined only the enhancement of visual patterns by cueing, it is difficult to generalize this result to other types of exogenous cuing (e.g., spatial exogenous cuing). In another study, Self and Roelfsema (2010) examined the exogenous facilitation effect when both cue and target were presented to the same eye or different eyes. They demonstrated that when the cue and target were presented to the same eye, rapid facilitation was observed (at SOA of 100 ms). In contrast, when the cue and target were presented to different eyes, facilitation only emerged later at a SOA of 200 ms. This finding led the authors to conclude that facilitation occurs earlier at lower (subcortical) levels of the visual system. One cautionary note is that the authors used a difficult discrimination task and the attentional effects observed in detection and discrimination differ in their time course (IOR appears later in discrimination tasks) and susceptibility to other manipulations, such as temporal expectancy (Gabay, Chica, Charras, Funes, & Henik, 2012; Gabay & Henik, 2010; Lupianez, Milan, Tornay, Madrid, & Tudela, 1997). Also, Self and Roelfsema (2010) only examined facilitation but not IOR. If facilitation and IOR are products of a unitary mechanism, then a delay in facilitation should also delay the appearance of IOR. In contrast, if facilitation and IOR are generated by dissociable mechanisms, then the change in the onset of facilitation should not modulate the onset of IOR.

In the present study, we manipulated the eye to which the cue and target are presented to evaluate (1) whether both facilitation and IOR are subserved by the same mechanism and, if so, (2) whether both dynamics are generated at lower levels of the visual system. The task we adopt is a simple detection task in which IOR is known to onset earlier. If facilitation and IOR are generated by the successive activation of the same eye-specific monocular neurons in subcortical regions, we should observe a delay in onset of both facilitation and IOR when the cue and target are presented to different eyes compared to when they share eye-of-origin. Such a finding will provide support for the unitary mechanism account and will strengthen the claim that lower, monocular brain regions are involved in the dynamics of both attentional facilitation and suppression. If, however, we observe a delay in the onset of facilitation (as previously demonstrated by Self & Roelfsema, 2010) but not in the onset of IOR when the cue and target are presented to different eyes, this will implicate a dissociation between the two systems.

In addition to the shared versus different eye-of-origin for cue and target, we included a further control condition in which the cue and the target are presented to both eyes simultaneously: under this condition, facilitation at the short SOA and IOR at the long SOA is predicted, and this scenario mimics the standard procedure in which both eyes have access to the cue and the target. This condition should then reflect the results under standard presentation conditions: facilitation peaks when the interval between the cue and target is on the order of 100 ms (stimulus-onset-asynchrony; SOA) and IOR is evident at a SOA of roughly 225 ms (Posner & Cohen, 1984). In this study, we presented the exogenous cue for 100 ms and used a 100 ms cue-target onsets interval as our shortest SOA both because facilitation is present at this time and because masking might occur when cue and target overlap in time (Lupiáñez & Weaver, 1998).

Experiment 1

Method

Participants

Thirty participants (age range 18–32; 19 females and 11 males) volunteered to participate in exchange for payment. All had normal or corrected-to-normal vision and all signed informed consent to participate. The protocol was approved

by the Institutional Review Board of Carnegie Mellon University.

Apparatus and stimuli

Two computer monitors were positioned on a table in front of the participant, and each was placed 50 cm to the left or right side of the participant. The participant was positioned in a chin rest. Two mirrors, one at 45° and one at 135°, each reflecting one of the two monitors, were situated to the side of the participant's eyes. Two pieces of cardboard (dividers) were connected to the chin rest, blocking the participant's direct view of the monitors (see Fig. 1). The display consisted of a fixation plus (0.7°) at the center of the computer screen, and three square boxes (2° each side), one at the center of the screen, and two 9.4° from the center of the screen. All stimuli were white figures against a black background. Following the brightening of one of the two peripheral boxes, accomplished by enlarging the box's contour from 1 to 5 mm, an asterisk (1°) appeared in the center of one of the peripheral boxes. Participants responded to the target asterisk by pressing the space bar of a keyboard with their dominant hand. The use of two separate monitors, in contrast to the use of a single monitor divided into two halves (each half is presented to a different eye), allowed us to present the stimuli in a horizontal array as is commonly done in this task.

Procedure

Participants were tested in a dimly illuminated room. They were seated roughly 60 cm from the computer monitors (viewed through the two mirrors to induce the perception of a single monitor). All stimuli except for the cues and targets appeared on both monitors. A typical experimental trial is depicted in Fig. 2. Each trial began with the appearance of a fixation plus sign for 1,000 ms. Participants were instructed to maintain fixation throughout the experiment. At 1,000 ms after the disappearance of this fixation plus sign, there was a brightening of one of the two peripheral boxes (the cue) for 100 ms. The cue could appear on the left, right or both monitors (with equal probability and randomized). At 100 ms, 500 ms, or 1,000 ms after the onset of the cue, the target appeared on one of the peripheral boxes and remained in view until participants responded or for a total of 3,000 ms. The target could also appear on the left, right or both monitors (with equal probability). When the cue appeared on both monitors, the target also appeared on both monitors. When the cue appeared on one monitor, it did not predict which monitor would contain the target or the location of the target within the monitor (left or right square). Following target offset, there was a 1,000 ms inter-trial interval, in which only the three boxes were presented. Each experiment included 312 trials (16 trials for each cue-target eye congruency x SOA x cue validity), of which 24 were catch trials in which the target did not appear. Each experiment began with 16 practice trials. Participants were instructed to respond to the onset of the asterisk as quickly as possible by pressing the space bar on the keyboard and to withhold response if no asterisk appeared. It should be noted that although eye movements were not monitored, it is unlikely that a differential pattern of saccades would be evident for the different conditions. The different experimental conditions were presented randomly so participants could not adopt a specific strategy, or be affected differentially by the cues. Moreover, because the cues in the same or

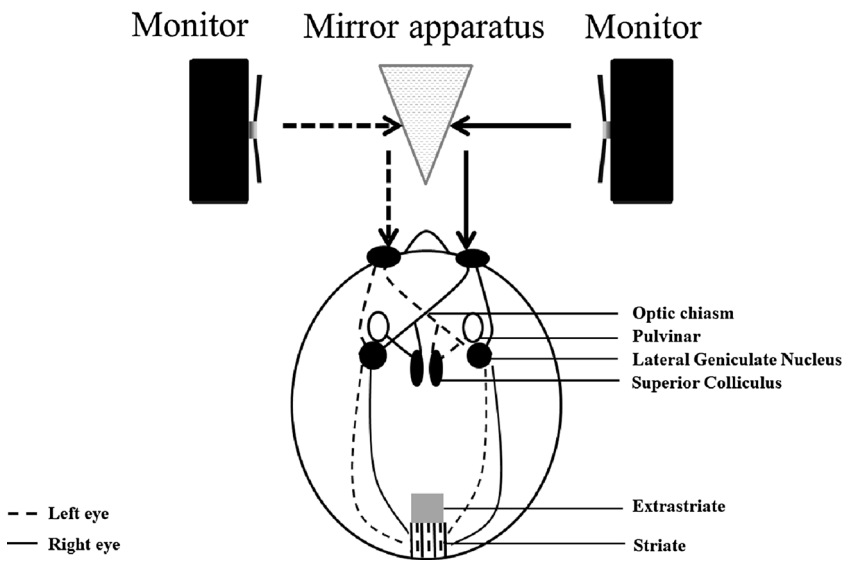

Fig. 1 Schematic illustration of the experimental apparatus and visual pathways from the eyes to the brain. Each monitor provided visual information to a different eye. From the eye, the visual information passes first through monocularly segregated subcortical regions (*dashed lines* left eye, *solid lines* right eye). This information is then projected to the lateral geniculate nucleus (LGN) and subsequently reaches striate and binocular extrastriate regions

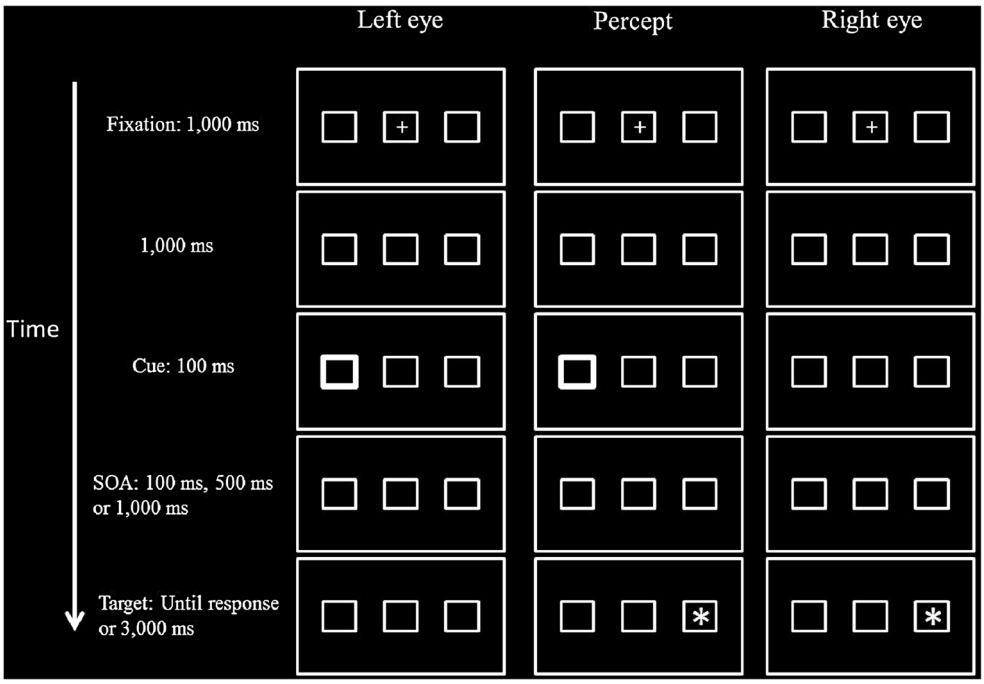

Fig. 2 A typical experimental invalid trial in which the cue is presented to the left eye (*left column*) and the target is presented to the right eye (*right column*). The *middle column* represents the participant's fused perception

different eye congruency conditions were visually identical, participants were unaware of the particular condition and unlikely to make different eye movements.

Results

Trials on which response time (RT) was longer than 2,000 ms or shorter than 100 ms were excluded from the analyses (1 %). Participants responded during catch trials on fewer than 1 % of the trials.

An analysis of variance (ANOVA) with cue-target eye congruency (Same, Different or Both eyes), SOA (100 ms, 500 ms or 1,000 ms), and validity (valid, invalid) as within-subjects factors was conducted with RT as the dependent variable. Figure 3 presents RTs as a function of cue-target congruency, SOA and validity. The main effect of SOA was significant, F (2, 58) = 56.04, MSE = 1,861, $P < 0.001$, indicating a decline in RT from the first to the second SOA, F (1, 29) = 95.76, MSE = 1,672, $P < 0.001$, but no difference in overall RT between the second and third SOAs, F (1, 29) < 1, NS. The significant main effect of cue-target eye congruency, F (2, 58) = 53.93, MSE = 936, $P < 0.001$, revealed faster RT when the cue and target were presented to both eyes compared to the two other conditions, F (1, 29) = 122.34, MSE = 789, $P < 0.001$, but no difference in RT when cue and target were presented to the same eye or to different eyes, F (1, 29) = 2.24, MSE = 1,081, $P = 0.1$.

The SOA x validity interaction was significant, F (2, 58) = 19.33, MSE = 1,500, $P < 0.001$, *indicating* a change from facilitation at the first SOA (valid faster than invalid), F (1, 29) = 14.23, MSE = 1,306, $P < 0.001$, to IOR (invalid faster than valid) at both the second SOA, F (1, 29) = 12.36, MSE = 2,422, $P < 0.01$, and the last SOA, F (1, 29) = 9.48, MSE = 2,113, $P < 0.01$.

The SOA x cue-target eye congruency interaction was also significant, F (4, 116) = 4.72, MSE = 671, $P < 0.01$, indicating a steeper linear trend in RT with increasing SOA when the cue and target were presented to both eyes compared to the two other conditions, F (1, 29) = 17.71, MSE = 624, $P < 0.001$, but no difference in RT when cue and target were presented to the same eye compared to when they were presented to different eyes, F (1, 29) = 1.81, MSE = 642, $P = 0.18$.

Most important for the current purpose, the three-way interaction between eye congruency x SOA x validity was significant,[1] F (4, 116) = 2.86, MSE = 734, $P < 0.05$. To analyze this interaction further, we examined the validity effect at every SOA separately for each cue-target eye congruency condition. When the cue and target were presented to both eyes, significant facilitation was present at the first SOA, and significant IOR was present at the two later SOAs, [F (1, 29) = 13.97, MSE = 814, $P < 0.001$; F (1, 29) = 15.13, MSE = 1,031, $P < 0.001$; F (1, 29) = 5.08, MSE = 1,306, $P < 0.05$, for the 100 ms, 500 ms and 1,000 ms SOAs, respectively]. When the cue and target were presented to the same eye, significant

[1] This interaction was also significant when examining only the 500 ms and 1,000 ms SOAs (excluding the 100 ms SOA) at all levels of eye congruency and validity [F (2, 58) = 3.15, MSE = 813, p= .05] and when excluding the both eye presentation condition from the analyses. It was also significant after employing the Greenhouse-Geisser correction for the sphericity assumption.

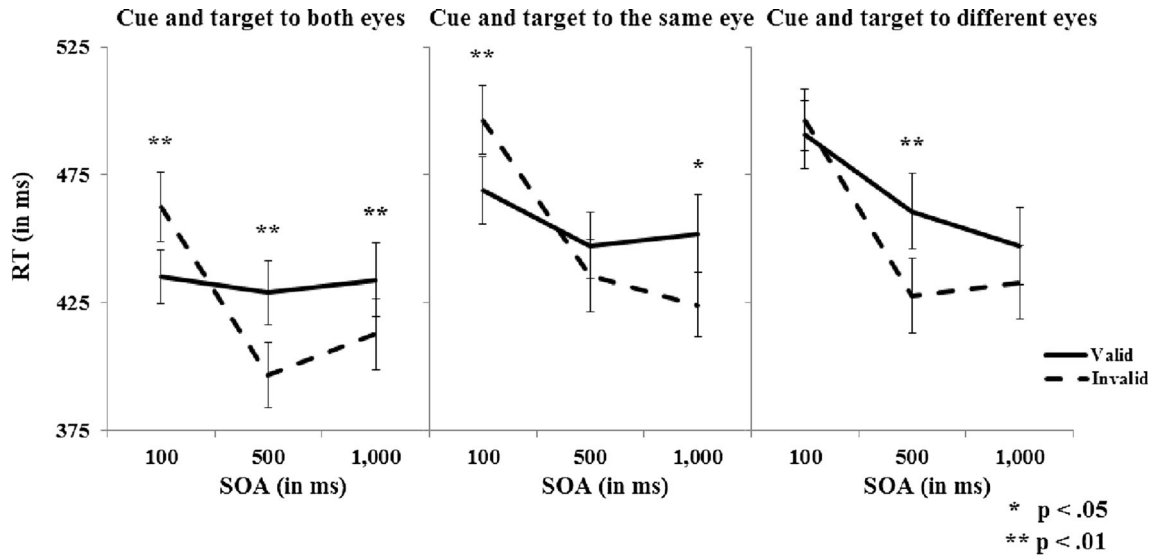

Fig. 3 Reaction time (RT) as a function of stimulus onset asynchronies (SOA), depicted for each cue-target eye congruency condition, with valid and invalid trials plotted separately

facilitation was present at the first SOA, no condition difference was evident at the second SOA, and significant IOR was present at the last SOA [F (1, 29) = 12.29, MSE = 937, P < 0.01; F (1, 29) = 1.48, MSE = 1,414, P = 0.23; F (1, 29) = 6.01, MSE = 1,954, P < 0.05, for the 100 ms, 500 ms and 1,000 ms SOAs, respectively). Finally, when the cue and target were presented to different eyes, no facilitation was present at the first SOA but there was IOR at the second and a trend for IOR at third SOA (F (1, 29) < 1, NS; F (1, 29) = 13.44, MSE = 1,237, P < 0.001; F (1, 29) = 2.25, MSE = 1,348, P = 0.14, for the 100 ms, 500 ms and 1,000 ms SOAs, respectively).

We also compared the validity effect between the same and different cue-target eye congruency conditions for every SOA. These comparisons yielded a significant difference for the first SOA (F (1, 29) = 5.3, MSE = 676, P < 0.05) in which the different cue-target eye congruency condition produced smaller facilitation than the same cue-target eye congruency condition. A significant comparison was also observed for the second SOA (F (1, 29) = 7.2, MSE =474, P < 0.05) and indicated a larger IOR effect for the different cue-target eye congruency condition than for the same cue-target eye congruency condition.

Discussion

This experiment examined whether exogenous attentional modulation, manifest as facilitation and/or IOR, occurs with respect to the eye to which the information is presented: here, the cue and ensuing target were presented to the same eye, to different eyes or to both eyes simultaneously. The standard findings were observed when both eyes simultaneously received information about the cue and the target, replicating the well-known early facilitation and later-appearing IOR. IOR was observed both when the cue and target were presented to the same eye as well as when they were presented to different eyes. As expected, facilitation was observed only when the cue and target were presented to the same eye. In contrast, and critically, IOR onset was not delayed when the cue and target were presented to different eyes compared to when the cue and target were presented to the same eye, demonstrating a dissociation between facilitation and IOR.

Closer examination of our results reveals one more intriguing aspect and this is the difference in the time course of IOR between the same and different cue-target eye congruency conditions. When the cue and target were presented to different eyes, IOR was apparent only at the 500 ms SOA (and perhaps present but reduced[2] at 1,000 ms SOA). When the cue and target were presented to the same eye, IOR was apparent only at the 1,000 ms SOA (the extended appearance of IOR at the same cue-target eye congruency condition will be addressed in the General discussion). A possible explanation for the earlier appearance of IOR in the different eyes condition is that, if, as suggested earlier, facilitation and IOR are dissociable processes, they might overlap in time. Thus, the absence (or reduction) of facilitation at the different cue-target eye congruency condition might enable IOR to be evident at earlier SOAs. In contrast, in the different cue-target eye congruency condition, facilitation might mask IOR, requiring a longer time interval for IOR to emerge. In order to examine the IOR effect,

[2] As also been demonstrated by the effect size for the IOR effect at the 1,000 ms SOA for the different eyes condition (η2p = .07) and the same eye condition (η2p = .17).

uncontaminated by overlap with facilitation, in the second experiment, we examined longer SOAs (at which point the effects of facilitation might have decayed, allowing a clearer view of IOR). To do so, we included 100 ms SOA to replicate the early facilitation but then selected more temporally extended SOAs than in Experiment 1.

Experiment 2

Method

Participants

Nineteen participants (age range 18–22; 12 females and 7 males) volunteered to participate in exchange for payment. All had normal or corrected-to-normal vision and all signed informed consent to participate. The protocol was approved by the Institutional Review Board of Carnegie Mellon University.

Apparatus and stimuli

The apparatus and stimuli were identical to the first experiment.

Procedure

The procedure was identical to the first experiment with one exception. The SOAs used were 100 ms, 800 ms, or 1,600 ms.

Results

Trials in which participants' response time (RT) was longer than 2,000 ms or shorter than 100 ms were excluded from the analyses (1 %). Participants responded during catch trials on fewer than 1 % of the trials.

An analysis of variance (ANOVA) with cue-target eye congruency (Same, Different or Both eyes), SOA (100 ms, 800 ms or 1,600 ms), and validity (valid, invalid) as within-subjects factors was conducted with RT as the dependent variable. Figure 4 presents RTs as a function of cue-target congruency, SOA and validity. The main effect of SOA was significant, $F(2, 36) = 7.79$, $MSE = 2{,}686$, $P < 0.01$, indicating no difference in overall RT between the first and second SOA, $F(1, 17) < 1$, NS, but a reduction in RT between the second and third SOAs, $F(1, 18) = 8.07$, $MSE = 2{,}391$, $P < 0.05$. The significant main effect of cue-target eye congruency, $F(2, 36) = 22.71$, $MSE = 1{,}675$, $P < 0.001$, revealed faster RT when the cue and target were presented to both eyes compared to the two other conditions, $F(1, 18) = 25.15$, $MSE = 2{,}970$, $P < 0.001$, but no difference in RT when cue and target were presented to the same eye compared to when they were presented to different eyes, $F(1, 18) < 1$, NS. A main effect of validity, $F(1, 18) = 22.71$, $MSE = 1{,}165$, $P < 0.001$, indicated faster RTs for invalid compared to valid trials. These effects largely replicate the findings from Experiment 1.

The SOA x validity interaction was also significant, $F(2, 36) = 18.27$, $MSE = 1{,}914$, $P < 0.001$, indicating a change from facilitation at the first SOA, $F(1, 18) = 10.46$, $MSE = 1{,}135$, $P < 0.01$, to IOR at both the second SOA, $F(1, 18) = 26.11$, $MSE = 2{,}575$, $P < .001$, and at the last SOA, $F(1, 18) = 13.46$, $MSE = 1{,}282$, $P < .01$.

As in the first experiment, the three-way interaction between eye congruency x SOA x validity was significant,[3] $F(4, 72) = 2.72$, $MSE = 1{,}020$, $p < 0.05$. To analyze this interaction further, we examined the validity effect at every SOA separately for each cue-target eye congruency condition. When the cue and target were presented to both eyes, significant facilitation was clearly present at the first SOA, and significant IOR was clearly present at the two later SOAs, [$F(1, 18) = 4.41$, $MSE = 1{,}590$, $P < 0.05$; $F(1, 18) = 17.85$, $MSE = 1{,}344$, $P < 0.001$; $F(1, 18) = 36.89$, $MSE = 463$, $P < 0.001$, for the 100 ms, 800 ms and 1,600 ms SOAs, respectively].

When the cue and target were presented to the same eye, significant facilitation was present at the first SOA and significant IOR was present at the other two SOAs [$F(1, 18) = 9.45$, $MSE = 843$, $P < 0.01$; $F(1, 18) = 9.81$, $MSE = 3{,}717$, $P < 0.01$; $F(1, 18) = 4.61$, $MSE = 829$, $P < 0.05$, for the 100 ms, 800 ms and 1,600 ms SOAs, respectively). Finally, and telling in its specificity, when the cue and target were presented to different eyes, no facilitation was present at the first SOA but there was IOR at the second SOA [$F(1, 18) < 1$, NS; $F(1, 18) = 31.89$, $MSE = 334$, $P < 0.001$; $F(1, 18) < 1$, NS, for the 100 ms, 800 ms and 1,600 ms SOAs, respectively).

Discussion

The second experiment replicated the key result of the first experiment, indicating no facilitation when the cue and target were presented to different eyes. In contrast, at the 800 ms SOA, IOR was observed in all of the cue-target eye congruency conditions. Similar to the first

[3] This interaction was not significant when examining only the 800 ms and 1,600 ms SOAs (excluding the 100 ms SOA) at all levels of eye congruency and validity [$F(2, 36) = 1.11$, *N.S.*]. The interaction was marginally significant ($p = .06$) after employing the Greenhouse-Geisser correction for the sphericity assumption.

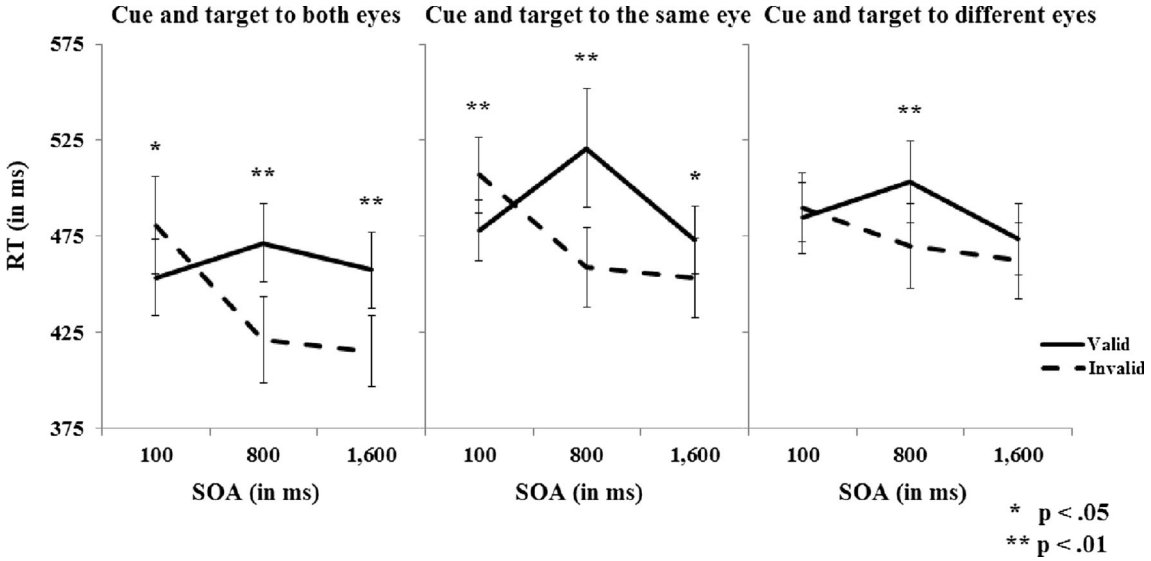

Fig. 4 RT as a function of SOA, depicted for each cue-target eye congruency condition, with valid and invalid trials plotted separately

experiment, IOR was not significant for the different cue-target eye congruency at the last SOA. We address the extended appearance of IOR in the same cue-target eye congruency condition further in the General discussion.

Thus far, we have demonstrated that IOR onset was not modulated by our eye-of-origin manipulation and that facilitation at 100 ms SOA is absent when the cue and target are presented to different eyes. However, we have not demonstrated that facilitation emerges later when the cue and target are presented to different eyes. As indicated in the Introduction, Self and Roelfsema (2010) reported that facilitation appears later (200 ms SOA) when the cue and target are presented to different eyes. To determine whether facilitation is delayed or entirely absent in the present task we have employed thus far, we replicate Experiment 1 but adopt a particular (and potentially revealing) set of SOAs so as to more closely track the rise and fall of the attentional dynamics.

Experiment 3

Method

Participants

Twenty participants (age range 18–22; 9 females and 11 males) volunteered to participate in exchange for course credits. All had normal or corrected-to-normal vision and all signed informed consent to participate. The protocol was approved by the Institutional Review Board of Carnegie Mellon University.

Apparatus and stimuli

The apparatus and stimuli were identical to the first experiment.

Procedure

The procedure was identical to the first experiment with two exceptions: (1) The cue duration was shortened to 75 ms and SOAs used were 75 ms, 150 ms, or 225 ms, and (2) there was no 'both eye presentation' condition as there was no need to replicate the standard finding yet again.

Results

Trials in which participants' response time (RT) was longer than 2,000 ms or shorter than 100 ms were excluded from the analyses (5 %). Participants responded during catch trials on fewer than 1 % of the trials.

An analysis of variance (ANOVA) with cue-target eye congruency (Same or Different eyes), SOA (75 ms, 150 ms or 225 ms), and validity (valid, invalid) as within-subjects factors was conducted with RT as the dependent variable. Figure 5 presents RTs as a function of cue-target congruency, SOA and validity. The main effect of SOA was significant, $F(2, 38) = 7.9$, $MSE = 1{,}743$, $P < 0.01$, indicating a reduction in RT between the first and second SOA, $F(1, 19) = 7.14$, $MSE = 2{,}138$, $P < 0.05$, and no difference in RT between the second and third SOAs, $F(1, 19) < 1$, NS. The significant main effect of validity, $F(1, 19) = 7.73$, $MSE = 7{,}369$, $P < 0.05$, revealed faster RTs for valid than for invalid trials.

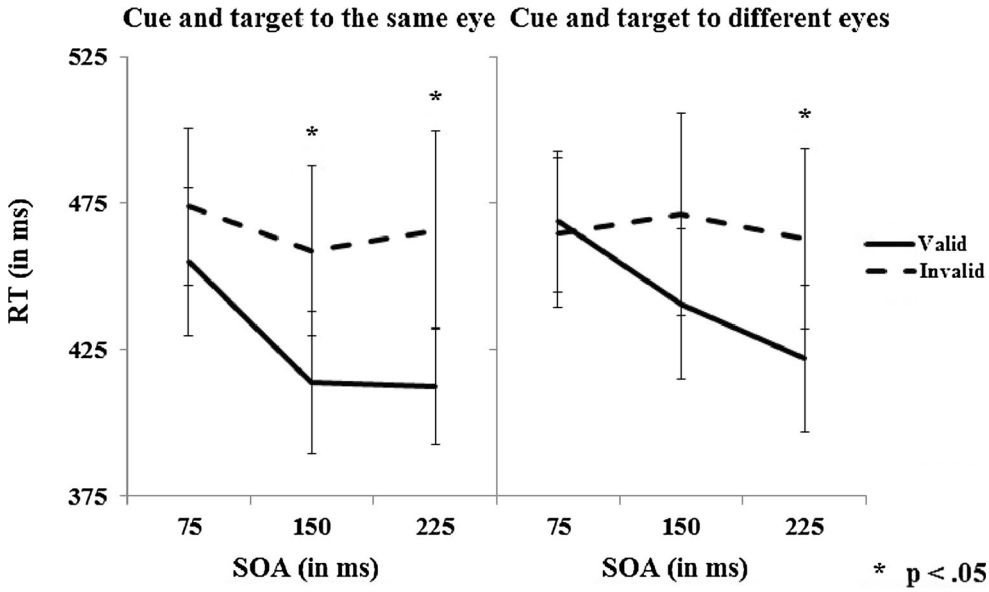

Fig. 5 RT as a function of SOA, depicted for each cue-target eye congruency condition, with valid and invalid trials plotted separately

The SOA x validity interaction was significant, F (2, 38) = 4.44, MSE = 1,942, $P < 0.05$, indicating a gradual development of facilitation from a non-significant validity effect at the first SOA, F (1, 19) < 1, NS, to a significant validity effect at the second, F (1, 19) = 5.74, MSE = 4,977, $P < 0.05$, and the third SOA, F (1, 19) = 9.76, MSE = 4,558, $p < 0.01$.

Most importantly, the cue-target eye congruency x validity interaction was also significant, F (1, 19) = 6.01, MSE = 668, $P < 0.05$, indicating a smaller validity effect when the cue and target are presented to different eyes compared to when they are presented to the same eye.

Although the SOA x validity x cue-target eye congruency interaction was not significant, F (2, 38) < 1, NS, we wished to examine the time course of facilitation. In order to do so, we examined the validity effect at every SOA separately for each cue-target eye congruency condition. When the cue and target were presented to the same eye, facilitation was marginally significant at the first SOA, and significant at the two later SOAs, [F (1, 19) = 3.16, MSE = 1,122, $P = 0.09$; F (1, 19) = 7.86, MSE = 2,553, $P < 0.05$; F (1, 19) = 6.47, MSE = 4,400, $P < 0.05$, for the 75 ms, 150 ms and 225 ms SOAs, respectively].

When the cue and target were presented to different eyes, significant facilitation was only present at the last SOA [F (1, 19) < 1, NS; F (1, 19) = 2.56, NS; F (1, 19) = 7.15, MSE = 2,348, $p < 0.05$, for the 75 ms, 150 ms and 225 ms SOAs, respectively].

To examine whether there were any differences in the magnitude of facilitation at its maximal magnitude, we directly compared the facilitation effect at the last SOA between the two cue-target eye congruency conditions. Facilitation in the two conditions was similar in size [F (1, 19) < 1, NS].

Discussion

The third experiment revealed that, although emerging later, a small amount of facilitation does exist even when cue and target are presented to different eyes. In accordance with Self and Roelfsema (2010), facilitation was delayed and reduced when cue and target were presented to different eyes relative to when the cue and target are presented to the same eye.

General discussion

In three experiments, we have demonstrated a dissociation between the characteristics of facilitation and those of IOR as a function of cue and target eye-of-origin. When the cue and target were presented to different eyes, the onset of facilitation was delayed. In contrast, the onset of IOR was not modulated.

Our study is not the first to examine the nature of the relationship between mechanisms supporting IOR and the mechanisms supporting facilitation. While our results are consistent with some existing results, some effects are at odds with the extant literature, and indeed, many published results are contradictory in and of themselves as evident from our introductory overview. Our approach has the potential to shed light on the issues under discussion and our experimental design overcomes several possible limitations that exist in commonly used methods to study the neural origin of behavior. For instance, in contrast to studies exploiting TMS and investigations of the effect of a brain lesion on performance, our method examined the involvement of subcortical regions without any disruption of the brain's normal functioning. In addition, in comparison to more demanding discrimination tasks that have been used previously, we examined the influence of cue and target eye of origin in a simple detection task. This procedure

might allow for more subtle and sensitive probing of the attentional dynamics than is possible with more complex tasks.

A unitary mechanism, in which both facilitation and IOR are successive phenomena generated by the same mechanism, would predict that a delay in the emergence of facilitation will also result in a delay in the manifestation of IOR. In our experiments, we demonstrate that this is not the case and, although facilitation is delayed when cue and target are presented to different eyes, IOR onset was not. We consider the differences in the temporal profile of these attentional dynamics to indicate that they are unlikely to arise from a common underlying mechanism. If IOR reflects the habituation of a unitary process (following facilitation) this habituation should be related (both in time and magnitude) to the facilitation preceding it. Because we have demonstrated that this is not the case, our study implicates some dissociation in the computations that mediate facilitation and those that mediate IOR.

To reveal the microgenesis of the IOR, Fig. 6 shows the aggregated results across the multiple SOAs from the two first experiments. As can be seen from the figure, IOR reached its peak at roughly 800 ms SOA in all experimental conditions, indicating that the time course of IOR was similar in all conditions. The fact that IOR was not delayed at the different cue-target eye congruency conditions, suggest that it is mediated by cortical regions. The smaller magnitude of IOR in the different cue-target eye congruency condition also suggests that, in addition to cortical involvement, subcortical regions might also play a role in its expression. But perhaps more revealing is the dissociation between IOR and facilitation. Facilitation onset was influenced by the eye-of-origin manipulation while IOR was not.

It should be noted that a dependency might exist in measuring facilitation and IOR. It has been suggested that the two might overlap in time (Chica & Lupiáñez, 2009; Lupiáñez & Weaver, 1998; Posner & Cohen, 1984; Tassinari et al., 1994), which might influence the ability to measure their time course independently. However, one might predict that a delay in facilitation, as demonstrated in the different eye condition, should, in turn, delay IOR (as a result of masking). The fact that we observe a similar onset of IOR in both the same eye and different eye conditions suggests that IOR onset is either similar in the two conditions or actually appears earlier in the different eyes condition. Both cases suggest that IOR and facilitation have different neural substrates and that facilitation might be more related to lower regions of the visual pathway.

This dissociation of the profiles of facilitation and IOR, reflecting the separability of the underlying mechanisms, is compatible with findings reporting the selective impairment in facilitation, but not in IOR, in patients suffering from a movement deficit, limited to one eye (Gabay et al., 2010). In such patients, testing with an eye-patch manipulation revealed that facilitation was adversely impacted only when the stimuli

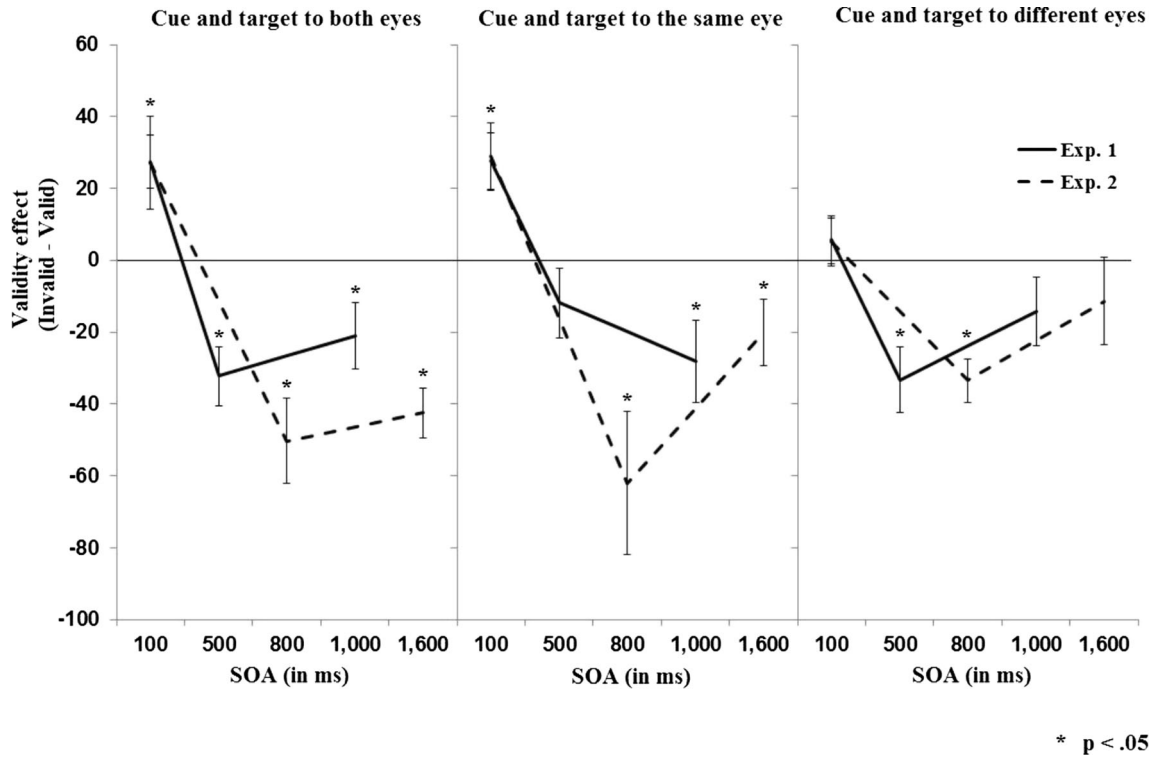

Fig. 6 Data collectively assembled from all three experiments to reflect the time course of the attentional dynamics. The validity effect, as a function of SOA is depicted for each cue-target eye congruency condition, with the data from each of the three experiments plotted separately

were presented to the affected visual field in the affected eye, whereas a normal pattern of attentional orienting was observed in the unaffected eye. Along with the dissociation between facilitation and IOR (see also Sapir et al., 1999), this finding also suggests that facilitation, in contrast to IOR, is more reliant on monocular mechanisms. Self and Roelfsema (2010) proposed that a possible source for monocular facilitation might actually be a result of inhibitory interactions between lateral geniculate nucleus (LGN) neurons that are partially mediated through the thalamic reticular nucleus that provides inhibition to the LGN relay cells (Crick, 1984; Singer, 1977). It is possible that facilitation is mediated by the LGN yet IOR is mediated by an interaction between SC and posterior parietal cortex (Dorris et al., 2002).

The finding that facilitation in our study is modulated by whether the cue and target are presented to the same eye is compatible with the claim that facilitation may result from simple neuronal re-activation at the lower levels of the visual system (Sereno et al., 2010). Thus, when the cue and target are presented to the same eye, they activate predominantly the same neural pathway in the monocular, lower levels of the visual system, and processing of the target is enhanced. This neural repetition might take longer when the cue and target are presented to different eyes since it will require some feedback from cortical regions. Although consistent with Sereno et al. (2010) in terms of the neural repetition explanation, our account differs in that our results indicate that exogenous facilitation is dissociable from IOR whereas Sereno et al. (2010) postulate an association between them.

Our findings also indicate that subcortical mechanisms alone may not suffice for IOR. The presence of IOR when cue and target were presented to different eyes suggests an involvement of higher cortical regions in the generation of IOR (Bourgeois et al., 2012; Chica et al., 2011; Sapir et al., 2004; van Koningsbruggen et al., 2010) and implies that the generation of IOR may not be dependent solely on subcortical regions. The finding that IOR onset was not delayed (and was actually observed earlier) when the cue and target were presented to different eyes is consistent with SC activation being dependent on higher brain regions in order to produce IOR (Dorris et al., 2002). We also found a modulation of the IOR time course between the same and different eye congruency conditions. First, IOR was observed earlier in the different eye congruency condition and we have postulated that this may occur because there was less early facilitation that masks the appearance of IOR at SOAs of around 500 ms. Secondly, although there was a trend for IOR to emerge in all SOAs longer than 500 ms, IOR was statistically significant at both the long SOAs examined (1,000 and 1,600 ms) for the same but not for the different eye congruency condition. This might result from the combined activation of IOR by collicular and cortical regions for the same eye congruency condition, but only cortical involvement in IOR for the different eye congruency condition. Since the visual system has many feedback and feedforward connections, a joint activation of both cortical and subcortical regions might resonate throughout the system longer, resulting in the prolonged effect even at extended SOAs. Examining nasal/temporal asymmetries as a marker of retino-tectal mediation, combined with the present experimental method might be informative regarding the exact visual pathway mediating facilitation and IOR and should be examined in future research.

In conclusion, the present findings demonstrate that facilitation and IOR attentional processes are modulated differently in the context of the visual system. Facilitation appears to be more reliant on lower levels of the visual system where the visual pathways are still segregated monocularly whereas IOR appears to be subserved by higher cortical visual regions. Whether two entirely independent mechanisms are required to produce the differential attentional dynamics or whether a single mechanism with some partial independence suffices, remains to be determined. Critically, the central finding from these experiments is that facilitation and IOR are not simply two ends of the same continuum.

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