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Brief article

Abrupt onsets and gaze direction cues trigger independent reflexive attentional effects

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Abstract

The present study investigated whether the detection of visual signals is influenced independently by two automatic visual orienting phenomena: orienting to a gazed-at location and inhibition of return (IOR) to the location of an abrupt onset. A schematic face served as both a nonpredictive gaze direction cue and an abrupt onset cue. Results indicated that target detection was facilitated at the gazed-at location, and that it was inhibited at the abrupt onset location. Importantly, these two different reflexive attention effects were triggered by the same event and exhibited overlapping time courses. Moreover, the IOR effect did not vary as a function of the facilitatory effect of gaze. These findings strongly suggest that reflexive attention to gaze direction and reflexive inhibition to an abrupt onset are independent processes, and that gaze direction does not produce IOR at the gazed-at location.

Keywords: Visual attention; Gaze direction; Reflexive orienting; Inhibition of return

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1. Introduction

Recent research has indicated that adults respond more quickly to targets appearing at a location that is gazed at by a centrally presented face than to targets appearing at a location that is not gazed at (e.g. Driver et al., 1999; Friesen & Kingstone, 1998; Hietanen, 1999; Langton & Bruce, 1999). This gaze direction effect exhibits some of the standard characteristics of reflexive covert attention (Cheal & Lyon, 1991; Jonides, 1981; Müller & Rabbitt, 1989). For example, it emerges rapidly, occurring as early as 100 milliseconds (ms) after the appearance of the gaze stimulus, and it occurs even though subjects are informed that gaze direction does not predict target location. However, there are some

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important differences between the reports of reflexive orienting to gaze direction and previous reports of reflexive covert orienting. First, nonpredictive gaze cues trigger reflexive orienting even though they appear at central fixation and direct attention to a peripheral location in much the same way that spatially predictive central arrow cues have been used traditionally to produce volitional orienting. In contrast, the type of cue used traditionally to produce reflexive orienting has been a nonpredictive cue appearing abruptly at some peripheral location where a target might subsequently appear (Posner, 1980; Posner, Cohen, & Rafal, 1982). Second, gaze-triggered reflexive orienting has a relatively long time course, much like orienting to spatially predictive cues such as central arrows, with the facilitation effect at the gazed-at location lasting for approximately 1 s. In contrast, facilitation effects at the location of an abrupt peripheral cue generally persist for 300 ms or less (Posner & Cohen, 1984). Third, traditional studies of reflexive orienting to peripheral cues report that when the cue-target stimulus onset asynchrony (SOA) is greater than 300 ms, response time (RT) is *longer* for a target appearing at the location of the cue than at a different (uncued) location. This delay in RT is generally attributed to attention being drawn to the location of the peripheral cue, and then removed; when a target later appears at the cued location, the return of attention to that location is inhibited. Accordingly this RT delay for targets that appear at a previously cued location has been called “inhibition of return” (IOR) (Posner, Rafal, Choate, & Vaughn, 1985). Importantly, no evidence of IOR has been observed in the nonpredictive gaze direction studies, i.e. RT has never been found to be longer for targets appearing at the cued (gazed-at) location compared to an uncued (not gazed-at) location.

There are three possible reasons why IOR has not been observed. The traditional explanation is that there is a single “spotlight” of attention that is responsible for both reflexive shifts of attention and the subsequent IOR effect (Posner et al., 1985). Within this framework, IOR and orienting to gaze direction should not be able to co-occur, because gaze-triggered facilitation persists for a relatively long time with its time course overlapping with the time course typically observed for IOR. The other two explanations both posit that orienting to gaze direction and IOR are separable phenomena that *can* co-occur. According to the second explanation, reflexive orienting to a gazed-at location does produce the standard IOR effect at the gazed-at location, but this effect is masked by the prolonged facilitation effect that is co-occurring at that same location. This account is consistent with the results of a recent study by Danziger and Kingstone (1999) demonstrating that attention to peripherally cued locations may mask the presence of IOR. The third explanation is that reflexive orienting to gaze direction simply does not produce IOR. If this were the case then IOR and gaze-triggered orienting should be able to occur at different locations at the same time.

The goal of the present study was to determine which of these three accounts is valid. Our approach was straightforward. We would present a face abruptly at one of four possible locations. This abrupt onset event should produce IOR at that location. Importantly, the face that served as an abrupt onset cue would also serve as a gaze direction cue: if gaze was directed at one of the other locations, it should produce orienting to that location. This design allowed us to probe the location of the abrupt onset, the gazed-at location, and the uncued locations. Of particular interest was performance at an intermediate SOA of 555 ms, because previous studies indicate that this is an SOA at which both reflexive attention to gaze (e.g. Friesen & Kingstone, 1998) and IOR (Klein, 2000) could be expected to occur. If IOR and gaze-triggered orienting *cannot* occur together (the first account above), then at an intermediate SOA we should see either IOR at the location of the abrupt onset or facilitation at the gazed-at location – but not both. Alternatively, if the two effects *can* co-occur (accounts two and three), we should see both effects at the intermediate SOA. According to the second account gaze direction is producing (but masking) IOR at the gazed-at location. It is well established that the magnitude of IOR declines as it is distributed across an increasing number of locations (Abrams & Pratt, 1996; Danziger, Kingstone, & Snyder, 1998; Tipper, Weaver, & Watson, 1996). It follows then that the magnitude of IOR at the abrupt onset location should be smaller if IOR is also occurring at the gazed-at location compared to when the eyes are straight ahead. According to the third account, gaze direction does not produce IOR. Here, IOR at the abrupt onset location should co-occur with, but be unaffected by, gaze direction effects.

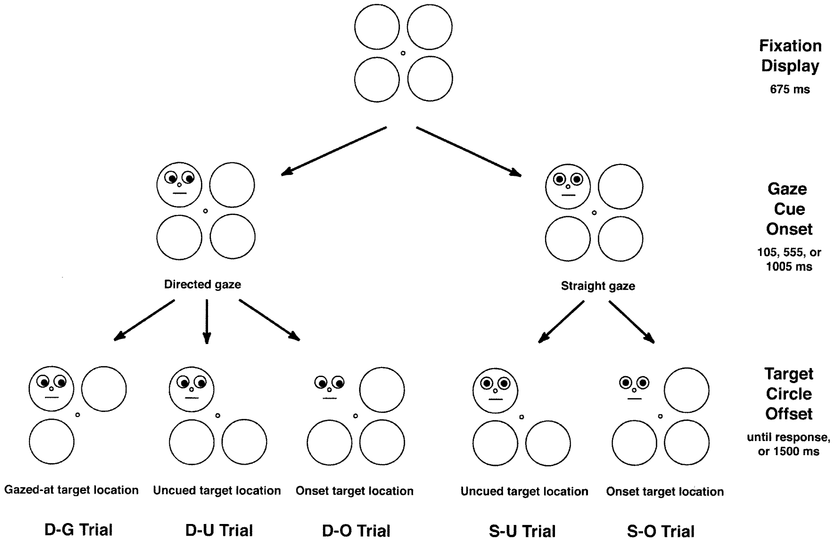

Fig. 1. Illustration of the trial sequence for each of the five possible cue-target conditions. Each trial began with four circles surrounding a central fixation dot. The cue was a gazing face, which appeared with equal probability in one of the four circles. The face's gaze was either *Directed* at one of the other three location circles, or *Straight* ahead. The target event was the offset of one of the four circles. Thus, a target occurred at either the *Gazed-at* location, at an *Uncued* location, or at the location of the *Onset* of the gazing face. Cue-to-target SOA, measured from the onset of the gazing face cue to the offset of a target circle, was randomly selected from durations of 105, 555, and 1005 ms. The intertrial interval was 675 ms. Stimuli were all colored black and presented on a white background. Target circles subtended 1.8°, mouth 0.7°, eye outlines 0.6°, pupils 0.3°, and nose and fixation point 0.1°. The distance from the center of each target circle to the fixation point was 1.1°. Each participant received 20 practice trials followed by 12 blocks of 54 test trials, for a total of 648 test trials. Catch trials, those without a target event, occurred on 11% of the trials.

2. Method

Eighteen naive undergraduates participated for course credit. As illustrated in Fig. 1, each trial began with four circles surrounding a central fixation point. Then, 675 ms later, a schematic face appeared abruptly in one of the four circles, with its eyes gazing either at one of the other three circles or straight ahead. At times of 105, 555, or 1005 ms after the onset of the face, the offset of one of the four circles served as the response target. The face, the fixation dot, and the three remaining circles stayed on the screen until a response was made or 1500 ms had elapsed, whichever came first. Participants were informed that the location of the face onset, and gaze direction, did not predict target location. They were instructed to maintain central fixation and to respond as quickly as possible to the target event by pressing the spacebar key.

Table 1
Mean RT, standard deviations, and errors rates[a]

Cue-target condition	105 ms SOA			555 ms SOA			1005 ms SOA		
	M	SD	%E	M	SD	%E	M	SD	%E
D-G: Directed gaze, Gazed-at target location	366.6	64	0.00	334.3	54	1.11	350.9	53	1.96
D-U: Directed gaze, Uncued target location	372.4	57	0.15	342.4	52	1.62	354.8	55	2.96
D-O: Directed gaze, Onset target location	379.6	56	0.32	403.4	61	1.68	411.7	66	1.66
S-U: Straight gaze, Uncued target location	367.4	62	0.31	342.7	52	2.19	357.2	54	1.85
S-O: Straight gaze, Onset target location	368.9	58	0.00	406.7	58	2.40	420.9	70	1.82

[a] Mean RT in milliseconds (ms), standard deviations (SD), and errors rates (%E) for each cue-target condition at each SOA interval. Error rates represent the percentage of target trials excluded from the analysis as anticipations (RTs of <100 ms) or timed-out trials (no response within 1500 ms).

3. Results

The false alarm rate on catch trials, which were excluded from the analysis, was 1.62%. For each participant, RTs greater or less than 2.0 standard deviations from their cell mean were excluded. These comprised 4.43% of the target trials. Anticipations (RTs of <100 ms) and timed-out trials (no response within 1500 ms) were classified as errors and were also excluded from the analysis. Error data are presented in Table 1. Anticipations accounted for 0.12% of the target trials, and timed-out trials accounted for 1.13%. Analyses revealed no effect of trial type on error rates (Fs < 1).

Mean RTs for correct responses are presented in Table 1 and illustrated in Fig. 2. An

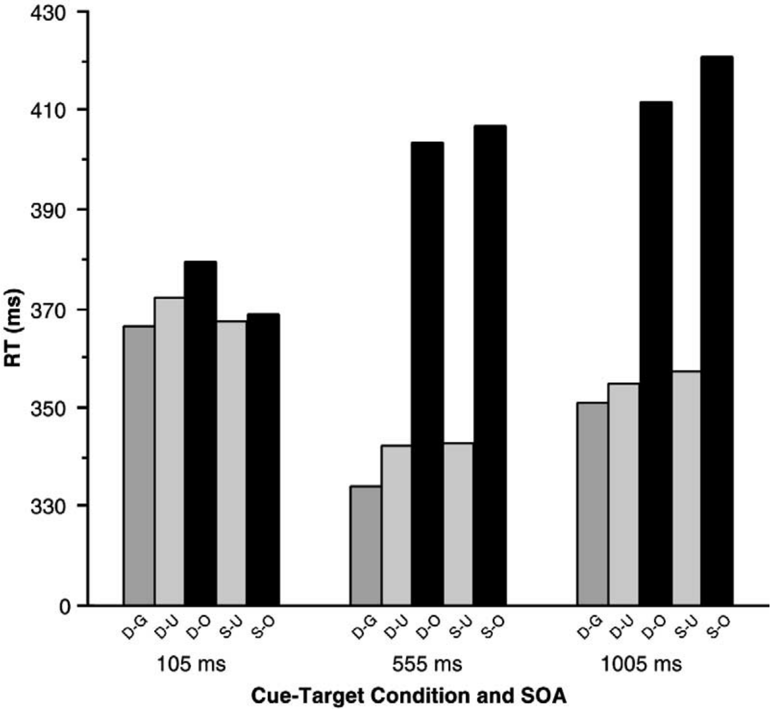

Fig. 2. Mean RT in milliseconds (ms) as a function of cue-target condition (D-G, Directed gaze, Gazed-at target location; D-U, Directed gaze, Uncued target location; D-O, Directed gaze, Onset target location; S-U, Straight gaze, Uncued target location; S-O, Straight gaze, Onset target location) and SOA. See Fig. 1 for an illustration of each of the five cue-target conditions. At 555 ms SOA, the *gaze effect* is D-G vs. D-U, and the *IOR effect* is: S-U vs. S-O.

omnibus ANOVA was conducted with SOA and cue-target condition as within-subject factors. Results revealed a significant main effect for SOA ($F(2, 17) = 4.16$, $P < 0.05$), a significant main effect for cue-target condition ($F(4, 17) = 67.02$, $P < 0.0001$), and a significant SOA × cue-target condition interaction ($F(8, 17) = 18.43$, $P < 0.0001$). Planned tests were used to answer the following questions.

3.1. Did participants shift attention to a gazed-at location?

We conducted a separate ANOVA for directed gaze trials in which targets occurred either at gazed-at locations (D-G) or at uncued locations (D-U) crossed with SOA. Results revealed a significant main effect for SOA ($F(2, 17) = 27.24$, $P < 0.0001$), with RT longest at the shortest SOA, reflecting a standard cue-target foreperiod effect. There was also a significant main effect for cue-target condition ($F(1, 17) = 5.02$, $P < 0.05$), reflecting RT facilitation for gazed-at target locations. The SOA × cue-target condition interaction was not significant ($F(2, 17) = 0.49$, $P > 0.60$), indicating that the magnitude of gaze direction's facilitatory effect was constant across the three SOA durations. Importantly, D-G

RT was never longer than D-U RT; thus, there was no evidence of IOR at the *gazed-at* location. A planned contrast between these two conditions at our critical 555 ms SOA confirmed a significant facilitation effect of gaze direction ($t(17) = 2.70$, $P < 0.02$).

3.2. Did the abrupt face onset produce IOR?

To test for the IOR effect apart from any gaze direction effects, we conducted an ANOVA on the two straight gaze conditions (S-O and S-U) crossed with SOA. There was a main effect for SOA ($F(2, 17) = 4.99$, $P < 0.02$), with RT increasing as SOA increased. There was also a main effect for cue-target condition ($F(1, 17) = 71.31$, $P < 0.0001$), reflecting the IOR effect (S-O RT > S-U RT). Finally, there was a significant SOA × cue-target condition interaction ($F(2, 17) = 18.89$, $P < 0.0001$), reflecting that IOR occurred at the two longer SOAs, but not at the short 105 ms SOA where neither facilitation nor inhibition was seen.[1] A planned comparison of S-O vs. S-U RT at the critical 555 ms SOA revealed a significant IOR effect ($t(17) = 7.72$, $P < 0.0001$). This IOR effect is clearly shown in Fig. 2.[2]

To simplify presentation of results, we will focus on the 555 ms SOA when answering our remaining questions.

3.3. Did IOR co-occur with orienting to a gazed-at location?

Our analysis has already shown that the abrupt onset of a face gazing at a different location will trigger a shift of attention to the gazed-at location at 555 ms SOA. Does this same face also produce IOR at the onset location at the 555 ms SOA? To examine this issue we compared directed gaze trials with the target occurring at the onset location (D-O) vs. directed gaze trials with the target occurring at an uncued location (D-U). This test revealed that participants were slower to respond on D-O trials than on D-U trials ($t(17) = 12.75$, $P < 0.0001$). Thus, the same directed gaze stimulus that caused a reflexive shift of attention to a gazed-at location also produced IOR at its onset location.[3]

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[1] Although IOR often follows an initial stage of facilitation, the fact that we saw neither facilitation nor inhibition for the abrupt onset cue at the short 105 ms SOA is not problematic since it is now well established that IOR can occur in the absence of prior facilitation, especially when offsets are used as targets (Samuel & Weiner, 2001).

[2] Since schematic faces may engage configural processing, one might wonder whether the lack of facilitation produced by the abrupt onset cue at the short 105 ms SOA and the large RT delays seen at the two longer SOAs occurred because it is generally more difficult to detect the offset of a circle when that circle constitutes the circumference of a face compared to when that circle stands alone. To test this possibility, we ran eight new subjects with an abrupt onset cue that was either face features (with straight gaze) appearing in the middle of a circle or five dots arranged as a cross and appearing in the middle of a circle (cues counterbalanced across two sets of 324 trials). Again there was no facilitation or inhibition at the 105 ms SOA, and RTs for abrupt onset locations were significantly longer than RTs for uncued locations at both the 555 ms and the 1005 ms SOA (costs of 52 and 53 ms, respectively). Most importantly, the pattern of results was precisely the same whether the abrupt onset cue was a face or a cross; type of cue had no significant main or interactive effect (all Fs < 1). Furthermore, RT costs in the new control experiment did not differ significantly from RT costs in the main experiment for S-U vs. S-O trials ($P > 0.59$). In other words, the RT delays seen at the longer SOAs in Fig. 2 are not specific to the abrupt onset being a face but instead reflect more general IOR effects.

[3] This was also the case for the 1005 ms SOA trials as shown by a similar t test comparing D-O and D-U trials at this SOA ($t(17) = 11.89$, $P < 0.0001$).

3.4. Did gaze direction affect the magnitude of IOR?

To investigate whether IOR was modulated by gaze direction, we conducted an ANOVA on 555 ms trials with target location (uncued, onset) and gaze (directed, straight) as within-subjects factors. Our primary interest here was to determine whether the interaction was significant – that is, does the magnitude of the IOR effect for the abrupt onset of straight-ahead gaze (S-O minus S-U) differ significantly from the magnitude of the IOR effect for the abrupt onset of directed gaze (D-O minus D-U)? The answer was “no”: the target × gaze interaction was not significant ($F(1, 17) = 0.13$, $P > 0.70$).[4]

4. Discussion

Both reflexive orienting to gaze direction and IOR were observed. The nonpredictive gaze direction of a schematic face produced shorter RTs (and no IOR) for targets at a gazed-at location, and IOR appeared at the location of the abrupt face onset. The key and new finding is that at the 555 ms SOA, reflexive attention to gaze *and* reflexive IOR occurred at the same SOA and at different locations *in response to exactly the same stimulus*. When gaze was directed at one of the three other locations and a target occurred at one of these locations, RT was shorter if the target occurred at the gazed-at location than if it occurred at a location that was not gazed-at (the gaze direction effect). And when gaze was directed at one of the three other locations and a target occurred at the location of the abrupt onset, RT was much longer (the IOR effect). Most importantly, the magnitude of the IOR effect at the abrupt onset location was not affected by whether gaze was directing participants’ attention to another possible target location. Together, these results indicate that automatic orienting to gaze direction and IOR are separate effects that can co-occur. Moreover, consistent with the third account outlined in Section 1, IOR did not appear to occur at the gazed-at location.

There are several important issues relating to our findings that warrant further discussion. First, one might question whether the RT delay we observed at the onset location reflects low-level perceptual masking of the target. We have already ruled out the possibility that configural processing triggered by the presentation of a schematic face was responsible for the RT delay (see footnote 2). If, instead, low-level perceptual masking were causing the RT delay, this would predict that the RT delay should be greatest at the shortest SOA (Breitmeyer, 1984). This prediction is not, however, borne out by the data. There was no RT delay at the cue onset location at the short 105 ms SOA. Rather, RT delays occurred only at the longer cue-target intervals – when IOR to an abrupt onset event is known to affect performance.

Second, our position is that the orienting triggered by nonpredictive gaze direction is reflexive because it occurs in response to spatially nonpredictive gaze cues at short SOAs. Nevertheless, one might question whether the gaze effect we observed at 555 ms SOA represents reflexive orienting. Perhaps the advantage for a gazed-at target location at this

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[4] The same ANOVA conducted on 1005 ms SOA trials also failed to show a significant target × gaze interaction ($F(1, 17) = 0.24$, $P > 0.60$). This provides further confirmation that gaze direction effects and the IOR effect are independent.

longer SOA results from participants shifting covert attention there volitionally or moving their own eyes to the gazed-at location. Our data fail to support these types of explanation because they each predict that the facilitatory effect of gaze should grow significantly with SOA (Jonides, 1981; Shepherd, Findlay, & Hockey, 1986). Instead, we found that facilitation at the gazed-at location was present at the shortest SOA and remained steady across all SOAs. (Indeed, if anything the gaze effect was declining at the longest SOA.)

Finally, one could ask whether the co-occurrence of gaze-triggered orienting and IOR on directed gaze trials might have been produced by participants shifting attention to the gazed-at location on some trials and inhibiting the onset location on other trials. Such a "mixture" hypothesis predicts that for onset location targets, the RT variance should be greater in the directed gaze (D-O) condition than in the straight gaze (S-O) condition. We examined the untrimmed data for a difference in variability between these two conditions at the critical 555 ms SOA, and found that the average standard deviation was *identical* in the two conditions (120 ms).[5] We also looked at individual participants' RT distributions. Bimodality is predicted in the directed gaze condition, but none was found in either condition. Finally, we examined whether gaze-triggered orienting (and not IOR) occurred for some participants, and IOR (and not gaze-triggered orienting) occurred for other participants at the 555 ms SOA. We found no such negative correlation (Pearson's $r = -0.06$). Thus, our data favor the conclusion that reflexive orienting to gaze direction and IOR are separable effects that can co-occur in response to the same stimulus.

To our knowledge, our finding that a reflexive shift of spatial attention to one location and reflexive spatial IOR at another location can co-occur has never been reported. Danziger and Kingstone (1999) demonstrated recently that *volitional* orienting away from an abrupt onset location can co-occur with reflexive IOR. Similarly, Tipper, Jordan, and Weaver (1999) have shown that reflexive facilitation and IOR can co-occur, but only when the stimulus items are inter-connected and are therefore represented in an object-centered reference frame. In contrast, when stimulus items are not connected, items are coded in a scene-based frame of reference, and only IOR is observed. This suggested to Tipper et al. that reflexive IOR and facilitation cannot co-exist in a scene-based frame of reference. However, our data show that this conclusion does not extend to when the abrupt onset is a biologically relevant stimulus such as a gazing face. In the present study the stimulus items were not inter-connected, and were therefore represented in a scene-based frame of reference. Nevertheless, the abrupt onset of a face triggered both IOR at the onset location and facilitation at the gazed-at location.

The fact that attentional orienting and IOR can co-occur when a biologically relevant directional cue is involved supports the notion that IOR to abrupt onsets and orienting to gaze direction are subserved by different brain mechanisms. There is a wealth of evidence indicating that the subcortical superior colliculus is critical to the occurrence of IOR to abrupt onsets (e.g. Rafal, Calabresi, Brennan, & Sciolto, 1989; Rafal, Henik, & Smith, 1991; Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988). In contrast, recent research suggests that gaze direction engages a reflexive attentional system that is preferentially biased toward cortical mechanisms. For example, Kingstone and colleagues showed that

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[5] Examination of the average standard deviations from the trimmed data also revealed that RTs on D-O trials (mean SD = 83 ms) were not more variable than RTs on S-O trials (mean SD = 85 ms) at 555 ms SOA.

with split-brain patients, reflexive attention to gaze direction occurred only in the hemisphere specialized for face processing (Kingstone, Friesen, and Gazzaniga, 2000) whereas reflexive orienting to stimuli that were not biologically relevant produced effects in both hemispheres (Ristic, Friesen, & Kingstone, in press) (stimuli that were not biologically relevant were used only in the Ristic et al. study). Functional neuroimaging studies have also found cortical activation in response to gaze direction in temporal areas analogous to the monkey superior temporal sulcus, as well as in parietal cortex (Hoffman & Haxby, 2000; Wicker, Michel, Henaff, & Decety, 1998). Further support for cortical involvement comes from nonhuman primate studies indicating that there are cells in inferior temporal cortex that respond selectively to faces and eyes (for a review, see Desimone, 1991) and cells in the superior temporal sulcus that respond selectively to particular gaze directions (e.g. Perrett et al., 1985).

In sum, our study suggests that the mechanisms supporting reflexive attention to gaze direction are independent from, and can co-occur with, the mechanisms supporting IOR. Moreover, our results indicate that gaze does not trigger IOR at the gazed-at location. These data agree with a growing literature that converges on the notion that cortical brain mechanisms are critical for orienting to gaze direction and that subcortical mechanisms play a crucial role in the inhibition of attentional return to locations that have been stimulated by an abrupt visual onset. Importantly, they also reinforce the general conclusion (Friesen & Kingstone, 1998; Kingstone et al., 2000; Ristic et al., in press) that orienting to biologically relevant stimuli represents a form of attentional processing that is unique, and qualitatively distinct from reflexive and volitional forms of orienting studied with nonbiologically-irrelevant stimuli in the past (cf. Posner, 1980).

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