

# Distinct Cortical and Collicular Mechanisms of Inhibition of Return Revealed with S Cone Stimuli

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## Summary

Visual orienting of attention and gaze are widely considered to be mediated by shared neural pathways [1–5], with automatic phenomena such as inhibition of return (IOR)—the bias against returning to recently visited locations—being generated via the direct pathway from retina to superior colliculus (SC) [6–8]. Here, we show that IOR occurs without direct access to the SC, by using a technique that employs stimuli visible only to short-wave-sensitive (S) cones [9]. We found that these stimuli, to which the SC is blind [10–12], were quite capable of eliciting IOR, measured by traditional manual responses [6, 8, 13]. Critically, however, we found that S cone stimuli did *not* cause IOR when saccadic eye movement responses were required. This demonstrates that saccadic IOR is not the same as traditional IOR, providing support for two separate cortical and collicular mechanisms of IOR. These findings represent a clear dissociation between visual orienting of attention and gaze.

## Results

What we perceive is governed not only by where we look but also by where we attend. Given limited neural resources, efficient processing of the visual environment relies on our ability to direct gaze and attention to objects that are most likely to provide new information. Thus sudden visual events often capture our attention and elicit a fast eye movement (saccade) toward them [14]. Furthermore, a fundamental aspect of visual exploration seems to be that once a new object has been inspected, a bias often develops against returning attention or gaze to that location—so called inhibition of return (IOR) [13, 15, 16]. The mechanism underlying IOR is unknown and remains the subject of debate. A prominent theory [7, 8, 17] is that IOR is mediated by the retinotectal pathway to the superior colliculus (SC), a midbrain structure known to play a major role in initiating eye movements [18–20]. However, some aspects of IOR suggest a cortically mediated attentional mechanism [13, 21–23].

The evidence for collicular mediation of IOR comes from brain-damaged patients [15, 24, 25], electrophysiological recordings [26], and interactions with oculomotor

phenomena thought to arise in the SC [27, 28]. While these particular studies do not distinguish between retinal or cortical projections to the SC, the direct neural projection from the retina to the SC has been implicated by three converging lines of evidence: First, IOR was found to be larger following stimuli in the temporal hemifield compared to the nasal hemifield [6], in accordance with the naso-temporal asymmetry of projections in this retinotectal pathway ([29], but see [30]). Second, IOR has been demonstrated in newborns, in whom the SC pathway is far more developed than cortical pathways [31]. Third, IOR has been demonstrated in the hemianopic field of a patient with a primary visual cortex lesion [32].

Thus, it has become widely believed that “IOR is generated through retinotectal pathways to the superior colliculus” ([8], page 504), occurring when irrelevant peripheral stimuli produce reflexive oculomotor activation in the SC [6, 7, 33, 34]. Nevertheless, some properties of IOR suggest a cortical mechanism [13, 21–23]. For example, IOR has been found to operate partly in environmental coordinates such that if an eye movement is made, a location in space remains inhibited even though it now has new retinal coordinates [13]. Lesions of the parietal cortex disrupt such environmentally mapped IOR but not retinotopic IOR [8]. Attempts to reconcile cortical versus collicular theories of IOR argue that it is generated via the retinotectal pathway but is modulated through collicular-cortical interactions, perhaps via the pulvinar [7, 8, 16, 32]. This view forms part of the general notion that attention and eye movements share a common neural network involving in particular the SC, pulvinar, and cortical eye fields [1–5].

Here we employ a new method for testing whether an effect is mediated by the retinotectal pathway [9]. We exploit the fact that electrophysiological studies have reported that there are no projections to the SC from short-wave sensitive cones (S cones) [10–12]. Therefore, stimuli visible only to S cones should be invisible to the retinotectal pathway. If IOR is mediated via the retinotectal pathway, as commonly believed, then it should *not* be elicited by stimuli visible only to S cones. Neither would S cone IOR be expected as a result of projections from visual cortex to the SC because our S cone stimuli are invisible also to the magnocellular pathway, which feeds this projection [35]. Finally, if ocular and attentional control share neural resources and there is a single network that generates IOR, S cone stimuli should not be able to reveal a dissociation between IOR measured by manual or saccadic responses. We found, to the contrary, that IOR is elicited by S cone stimuli, and that these stimuli do reveal a dissociation between two types of IOR. Thus we conclude that reflexive oculomotor activation via the retinotectal pathway is not necessary for IOR generation, and we suggest that there are separate cortical and collicular origins of IOR.

## Experiments 1 and 2: S Cone Stimuli Elicit IOR

We adapted the standard IOR paradigm of Posner and Cohen [13]. Participants simply made a speeded manual

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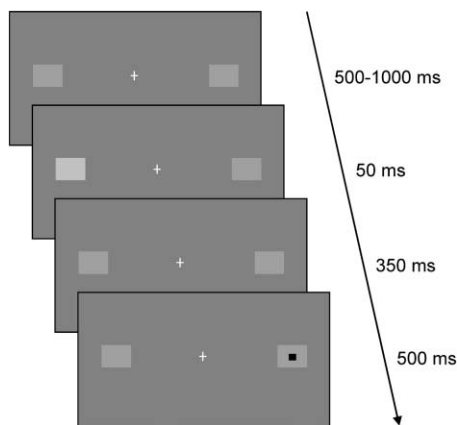


Figure 1. Schematic Diagram of One Trial

Subjects pressed a single button (Experiments 1 and 2) or moved their eyes (Experiments 3 and 4) as fast as they could in response to a small, dark gray target that could appear  $8^\circ$  to the right or left of fixation with equal probability. To avoid anticipatory responses in Experiments 1 and 2, targets did not appear on a third of the trials (catch trials). Solid gray “guide boxes” were present throughout each trial to mark the locations where a target could appear. These rectangles displayed “luminance noise” by changing luminance every 50 ms to a value drawn randomly from between preset limits ( $24\text{--}26\text{ cdm}^{-2}$  in Experiment 1;  $24.6\text{--}25.4\text{ cdm}^{-2}$  in Experiments 2–4). On every trial, either the left or right guide box made a large step in either luminance or color for 50 ms. This was the uninformative “cue”, and the cue-target interval was 400 ms in Experiments 1–3 and 500 ms in Experiment 4. The luminance cue was a brightening of the box to  $30\text{ cdm}^{-2}$ . The color cue was precalibrated for each subject so that only S cones could detect its difference from the flickering gray (see Supplemental Data, available with this article online, for more details).

response to targets that could appear  $8^\circ$  to the left or right of fixation. Uninformative peripheral “cues” occurred 400 ms before the targets (see Figure 1). Whereas 50% of the cues were brightness changes, as is traditional in IOR experiments, the other 50% were color changes visible only to S cones. The exact color change needed differs across people and across retinal locations, so this was individually calibrated for each subject using the “transient tritanopia” procedure [36]. Luminance noise was used for ensuring that only chromatic pathways could detect the S cone signal [37], and this noise was perceived by the subjects as flicker around the two areas of possible target presentation. Subjects were instructed to fixate on the central cross and to ignore all other stimuli except the targets. Previous findings have confirmed that S cone stimuli calibrated in this way are invisible to the retinotectal pathway [9].

IOR is defined as the extent to which reaction times to targets following ipsilateral cues are slower than to targets following contralateral cues, indexing a bias against the cued location. Figure 2 (left panel) shows that S cone cues produced IOR (13 ms,  $t = 2.23$ ,  $df = 7$ ,  $p < 0.05$ ) comparable to that produced by the luminance cues (9 ms,  $t = 4.74$ ,  $df = 7$ ,  $p < 0.01$ ). This would *not* be expected if signals in the retinotectal pathway were necessary to elicit IOR.

In Experiment 2, we tested whether S cone IOR is a robust phenomenon and whether it is affected by luminance noise magnitude. Luminance noise is necessary

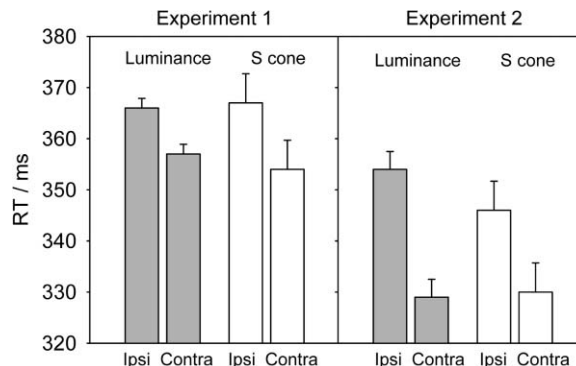


Figure 2. Traditional IOR

Mean manual RT for targets following luminance and S cone “cues” in Experiments 1 and 2. For both types of cue, RT was slower following cues on the same side as the target (ipsilateral) than following cues on the opposite side (contralateral). This difference is the measure of IOR (error bars are standard errors of this difference within subject). In Experiment 1, there were no effects in the error rates (mean 4.0%). In the errors in Experiment 2, there was no IOR effect for luminance cues (4.5% and 5.3% errors for ipsi- and contralateral targets), but there was for S cone cues (5.3% and 2.9%,  $p = 0.05$ ).

not only because equiluminance calibrations carry some small degree of possible error ( $0.2\text{ cdm}^{-2}$  in our case) but also because many nonchromatic cells will detect “equiluminant” color changes owing to local differences in the ratio of cone types in their receptive fields. Because S cones make little if any contribution to nonchromatic pathways [38–41], the amount of luminance noise needed to mask this signal is small. Experiment 1 used a conservatively large range of luminance noise ( $24\text{--}26\text{ cdm}^{-2}$ ), and such flicker may make the cues less salient or may itself capture attention, activate oculomotor plans, and produce competing IOR. This may be why the measured IOR effect in Experiment 1 was on the small side of previously reported effects ( $\sim 10\text{--}30\text{ ms}$ ) [8, 13, 15]. In Experiment 2, we reduced the range of luminance noise range to  $24.6\text{--}25.4\text{ cdm}^{-2}$ . Subjectively, the flicker was still perceptible, but it was much less distracting than in Experiment 1. All other aspects of the procedure were identical to Experiment 1 (see Figure 1). A new group of participants were used, and as before, the S cone stimuli were individually calibrated for each participant.

Figure 2 (right panel) shows that response time (RT) to targets following ipsilateral cues was slower than to targets following contralateral cues. In other words, there was an IOR effect, and this was true for both S cone cues (16 ms,  $t = 2.80$ ,  $df = 7$ ,  $p = 0.01$ ) and luminance cues (25 ms,  $t = 7.01$ ,  $df = 7$ ,  $p < 0.01$ ). This confirms the results of Experiment 1, showing, in a different group of subjects, that signals in the retinotectal pathway are not necessary for eliciting IOR. Furthermore, as predicted, the IOR effects in Experiment 2 were larger than in Experiment 1. This increase was large for the luminance cues (16 ms,  $t = 3.96$ ,  $df = 14$ ,  $p < 0.01$ ) but small for the S cone cues ( $p > 0.1$ ), suggesting that the main factor was cue salience (luminance flicker primarily reducing the salience of lumi-

nance stimuli) rather than competing oculomotor or attentional effects (which should be elicited by the flicker regardless of the cue type on that trial). It is important to note that we do not make strong conclusions based on the exact relative size of the S cone and luminance IOR effects because there is no secure way of equating such stimuli for salience (for example, multiples of threshold are not appropriate for stimuli that are not close to threshold because the responses of luminance and color pathways are nonlinear in different ways). Thus, the key results of Experiments 1 and 2 are simply that IOR was consistently elicited in response to S cone stimuli.

Note that S cone stimuli in luminance noise are also invisible to the magnocellular pathway from retina to cortex because it is not color opponent and receives little if any input from S cones [38–42]. Thus, the results of Experiments 1 and 2 demonstrate that IOR does not require signals in either the direct retinotectal pathway or the magnocellular pathway, which feeds the projection from visual cortex to the SC [35].

#### Experiments 3 and 4: S Cone Stimuli Do Not Elicit Saccadic IOR

Having shown that IOR generation does not require direct collicular activation, we tested in Experiment 3 the possibility that there is more than one way in which IOR can be elicited. If attention and eye movement control shared neural resources, and IOR is generated by a single mechanism, the same kinds of stimuli should be able to generate IOR regardless of whether it is measured with manual responses or saccades (eye movements). On the other hand, if there are separate cortical and collicular origins of IOR, S cone stimuli could reveal a dissociation between them. Saccadic IOR [7, 21, 34] may primarily tap the collicular origin, and thus, we might predict that it would not be produced by S cone stimuli, even though we have found that these stimuli do produce traditional manual IOR.

All aspects of Experiment 3 were identical to Experiment 2 (see Figure 1) except that participants responded by making saccades (eye movements) toward the targets rather than pressing a button. A new group of participants were used, and the S cone stimuli were individually calibrated for them. Figure 3 (left panel) shows the results of Experiment 3. There was a standard IOR effect for the luminance cues (18 ms,  $t = 4.5$ ,  $df = 7$ ,  $p < 0.01$ ) such that RT to targets following ipsilateral cues was slower than to targets following contralateral cues. However, for S cone cues, there was no hint of IOR (–3 ms). The only methodological difference from Experiment 2, besides the saccadic versus manual distinction, was that the saccadic responses were directional toward the target, whereas the manual response was a single central button pressed regardless of target location. However, this cannot account for the dissociation between Experiments 2 and 3 because it would predict a dissociation in the opposite direction. IOR is more—not less—likely with directional responses because if the participant must withhold responses to the cue, the pattern of direct response inhibition is different for ipsilateral and contralateral cues, whereas for a single response it is the same.

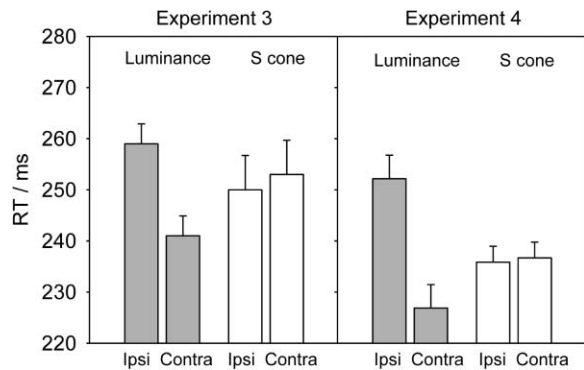


Figure 3. Saccadic IOR

Mean saccadic latency in Experiments 3 and 4, showing there was IOR for luminance cues but not for S cone cues. In Experiment 3, error rates followed latency, with an IOR effect for luminance cues (6.4% and 3.3% for ipsi- and contralateral targets,  $p < 0.05$ ) but no IOR effect for S cone cues (3.7% and 3.6%). In Experiment 4, there were no effects in the errors (2.4% and 2.1% for ipsi- and contralateral targets with luminance cues, 1.7% and 1.5% for S cone cues).

In Experiment 4, we repeated Experiment 3 with the time between cue and target increased by 100 ms, and we used a new group of participants. This was to rule out the possibility that S cone IOR did not have time to develop in Experiment 3—the saccadic latencies were shorter than the manual RTs of Experiments 1 and 2, and signals in the S cone pathway are known to be delayed relative to luminance signals by up to about 30 ms [43, 44]. Again, there was a standard IOR effect for the luminance cues (25 ms,  $t = 5.5$ ,  $df = 7$ ,  $p < 0.001$ ) but no IOR (–1 ms) for S cone cues (see right panel of Figure 3). The results of Experiments 3 and 4 are thus consistent with the idea that saccadic IOR is mediated by signals in the retinotectal pathway, and the dissociation with Experiments 1 and 2 indicates that there must be two origins of IOR.

#### Discussion

Our findings lead us to draw two important inferences. First, current theories of collicular IOR generation are inadequate, as we have shown that IOR is reliably elicited by color changes visible only to short-wave-sensitive cones (S cones), which are invisible both to the direct pathway from retina to SC and to the corticotectal projection derived from the magnocellular pathway. The “oculomotor-priming” theory suggests that IOR is produced directly from the colliculus by a corollary discharge that occurs whenever the oculomotor system is activated [6, 17, 45]. The “oculomotor-suppression” account proposes that IOR arises from inhibition applied to activity in SC build-up cells to avoid reflexively making an unwanted saccade to a new stimulus [7, 33, 34]. These theories differ in the second stage of IOR production (automatic corollary discharge or voluntary inhibition), but they agree that the first stage of IOR generation in response to uninformative peripheral stimuli is the initiation of a reflexive saccade plan to such stimuli in the SC. Our results indicate that peripheral stimuli can elicit IOR without reflexively initiating saccades via the

retinotectal pathway, or indeed via the corticotectal projection derived from the magnocellular pathway.

Second, S cone stimuli reveal an important dissociation between two types of IOR. When saccadic responses were used instead of the traditional manual responses, S cone stimuli no longer elicited IOR, demonstrating that saccadic and manual IOR do not share a single neural mechanism. This dissociation also confirms that signals from our S cone stimuli were indeed transmitted by a different pathway from the luminance stimuli, which is useful because anatomical or electrophysiological studies that have not found S cone projections to the SC cannot prove they do not exist. The fact that S cone stimuli do not produce a saccade distractor effect [9] also confirms that they are transmitted by a different pathway, and all evidence points to this pathway projecting to layers 3B and 4A of the primary visual cortex via small and large bistratified ganglion cells in the retina and koniocellular layers of the lateral geniculate nucleus (LGN) [46, 47]. It would be extraordinary if there were a separate retinotectal S cone pathway that could affect manual responses but not saccades. The dissociation also rules out the possible objection that IOR was elicited by S cone stimuli only because we sensitized participants to these colors during the calibration procedure [48]. Because the calibration procedure was identical in all experiments, this cannot explain the presence of IOR in some conditions and not others.

The question of what neural pathways cause IOR is separate from what neural processes are inhibited by IOR [22]. However, combining our findings—of two distinct causes of IOR—with those reporting a dissociation between oculomotor and attentional effects of IOR [21, 49] suggests the following model. One mechanism generates IOR in response to oculomotor initiation via the retinotectal pathway, while another generates IOR following attentional capture by signals in cortical pathways. The former retinotectal mechanism causes saccadic IOR, but both retinotectal and cortical mechanisms contribute to traditional IOR, measured with a single manual response. This would explain why for S cone stimuli, which activate only the cortical mechanism, IOR was present for manual responses but absent for saccadic responses. Furthermore, if both mechanisms normally contribute to traditional IOR, this model also explains why the size of S cone IOR was toward the small end of the range of previously measured effects.

The findings presented here also show that IOR is elicited without signals in the magnocellular pathway. This is surprising given that this pathway from retina to visual cortex is considered to be largely responsible for the detection of sudden visual changes and, thus, thought to mediate attentional orienting [50]. It is also the pathway that supplies the main cortical input to the SC [35]. So, given that IOR does not require the retinotectal pathway, the magnocellular pathway would be the likeliest candidate, but our S cone stimuli were invisible to both these pathways. The S cone pathway, which is thought primarily to subservise color vision [51], might be considered unsuited to mediate fundamental attentional processes because of its slower transmission speed [44]. Thus, even given that IOR is cortically generated, it is actually counter intuitive that IOR should

be elicited by S cone stimuli. Our results therefore suggest that IOR generation is not restricted to one or two most-suited pathways, but rather that many pathways may contribute to attentional orienting (see also [9, 52]).

While our findings demonstrate that traditional IOR occurs without retinotectal or magnocellular input to the SC, they do not exclude the possibility of any collicular involvement in “cortical” IOR. Most cortical systems are likely to be modulated by cortico-subcortical loops. But if the SC is crucially involved in all IOR production, it becomes difficult to explain our clear dissociation between saccadic and manual IOR. Likewise, while the results of Experiments 3 and 4 are most easily explained by collicular mediation of saccadic IOR, they do not rule out cortico-collicular mediation via structures such as the frontal or parietal eye fields [7, 8, 16, 32], which may also be blind to S cone stimuli. However, what remains clear is that our dissociation of saccadic and manual IOR shows that there is a fundamental distinction between the oculomotor IOR mechanism, which was not stimulated by S cone stimuli, and the attentional IOR that was elicited by S cone stimuli.

#### Supplemental Data

Supplemental Data including Experimental Procedures are available at <http://www.current-biology.com/cgi/content/full/14/24/2259/DC1/>.

#### Acknowledgments

We would like to thank Sarah Castor-Perry for help with Experiment 4 and Geraint Rees, Andy Parton, Adrian Burgess, Martin Batty and Kwok Sze-Chai for comments. The research was supported by the Hammersmith Hospitals Trust Research Committee, the University of London Central Research Fund and the Wellcome Trust.

Received: October 1, 2004

Revised: October 21, 2004

Accepted: October 21, 2004

Published: December 29, 2004

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