Correlates of Capture of Attention and Inhibition of Return across Stages of Visual Processing

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Abstract

■ How do visual signals evolve from early to late stages in sensory processing? We explored this question by examining two neural correlates of spatial attention. The capture of attention and inhibition of return refer to the initial advantage and subsequent disadvantage to respond to a visual target that follows an irrelevant visual cue at the same location. In the intermediate layers of the superior colliculus (a region that receives input from late stages in visual processing), both behavioral effects link to changes in the neural representation of the target: strong target-related activity correlates with the capture of attention and weak target-related activity correlates with inhibition of return. Contrasting these correlates with those obtained in the superficial layers (a functionally distinct region that receives input from early stages in visual processing), we show that the target-related activity of neurons in the intermediate layers was the best predictor of orienting behavior, although dramatic changes in the target-related response were observed in both subregions. We describe the important consequences of these findings for understanding the neural basis of the capture of attention and inhibition of return and interpreting changes in neural activity more generally. ■

INTRODUCTION

When exploring the neural basis of cognitive behavior, the first question asked is, ''Where does ability X originate in the brain''? Despite the simplicity of this question, history has demonstrated that it is not an easy one to answer. Take, as one example, visual spatial attention. Converging evidence from neuropsychological and functional imaging investigations in humans has shown that many brain areas participate in spatial attention tasks (e.g., Corbetta & Shulman, 2001; Posner & Petersen, 1990; Posner, Cohen, & Rafal, 1982; Mesulam, 1981, 1999). Monitoring the activity of single neurons in monkeys reveals that the neural correlates of spatial attention are represented as changes in the neural representation of the visual target (or object of attention; e.g., Bell, Fecteau, & Munoz, 2004; Fecteau, Au, Armstrong, & Munoz, 2004; Fecteau, Bell, & Munoz, 2004; Dorris, Klein, Everling, & Munoz, 2002; Constandtinidis & Steinmetz, 2001; Bichot & Schall, 1999, 2002; Gottleib, Kusunoki, & Goldberg, 1998; Robinson, Bowman, & Kertzman, 1995; Robinson & Kertzman, 1995; Schall, Hanes, Thompson, & King, 1995; Schall & Hanes, 1993; Goldberg & Wurtz, 1972) and these signals are expressed in remarkably similar ways across the cortical and subcortical areas (described in Fecteau, Bell, & Munoz, 2004; Schall, 2002, 2004). These observations have important ramifications—they suggest that the question of ''where'' spatial attention originates in the brain may be too simplistic. One alternative approach is to consider how these neural correlates of attention evolve across the network. That is, do these attentional processes influence sensory signals originating in early visual areas, which then are transmitted faithfully throughout the rest of the brain? Or are these signals modified through many possible intermediaries?

The superior colliculus is an ideal structure to adopt this levels-of-processing approach. Its intermediate layers receive visual input that has the potential of being processed through many intermediaries, including the prefrontal, parietal, and temporal cortices (e.g., Clower, West, Lynch, & Strick, 2001; Lui, Gregory, Blanks, & Giolli, 1995; Selemon & Goldman-Rakic, 1988; Stanton, Goldberg, & Bruce, 1988; Lynch, Graybiel, & Lobeck, 1985; Fries, 1984; Kuypers & Lawrence, 1967). By contrast, its superficial layers receive input from stations representing visual information very early in visual processing—from the retina, the primary visual cortex (V1), and low-level extra striate areas (e.g., Lui et al., 1995; Rodieck & Watanabe, 1993; Fries, 1984; Perry & Cowey, 1984)—and provide a clean index of early visual processing because this subregion is open-looped (i.e., the superficial layers do not receive feedback from the areas to which they project; Clower et al., 2001).

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Distinguishing between neurons residing in the superficial and intermediate layers is relatively straightforward on the basis of physiological and anatomical markers. Visuomotor neurons reside in the intermediate layers: These neurons produce a burst of neural activity when a visual object appears in their response field and a second burst of neural activity when a saccadic eye movement is generated to the same location (Figure 1). Visual neurons reside in the superficial layers: They produce a burst of neural activity when a visual object appears in their response field, no saccade-related activity, and must be encountered within the first 1000 μm or so after reaching the superior colliculus (Figure 1; Munoz & Wurtz, 1993; Mays & Sparks, 1980; Wurtz, Richmond, & Judge, 1980; Goldberg & Wurtz, 1972; Wurtz & Goldberg, 1971, 1972). Therefore, it is possible to explore how the neural representation of the visual target changes between early and late stages in processing by contrasting visual activity between visual neurons in the superficial layers and visuomotor neurons in the intermediate layers of the superior colliculus.

We used the cue–target task to elicit two behavioral indices of spatial attention. In this task, a flash of light in the peripheral visual field (the cue) is followed by a second visual stimulus (the target) that appears at the same or opposite location as the cue. Responding to the target probes the changing consequences of the salient cue on orienting attention towards a new object (i.e., the target). Manipulating the time between the cue and the target reveals two biases of spatial attention: the initial capture of attention to the locus of the cue when the time between the cue and target is short and inhibition of return (the preference of observers to explore new locations in the scene) when the time between the cue and target is longer (Figure 2; Posner & Cohen, 1984; Jonides, 1981; reviewed in Klein, 2000; Wright & Ward, 1998).

Opinions have been raised regarding where these biases in orienting spatial attention originate in visual

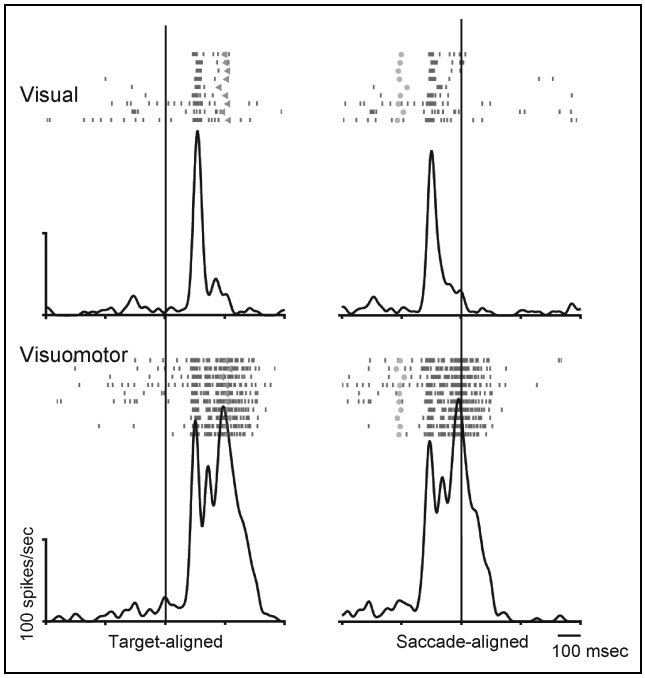

Figure 1. Classification of neurons. (A) Representative examples of visual and visuomotor neurons aligned on the target (left) and onset of saccade (right). Arrows on left rasters denote saccade onset. Circles on right rasters denote target appearance.

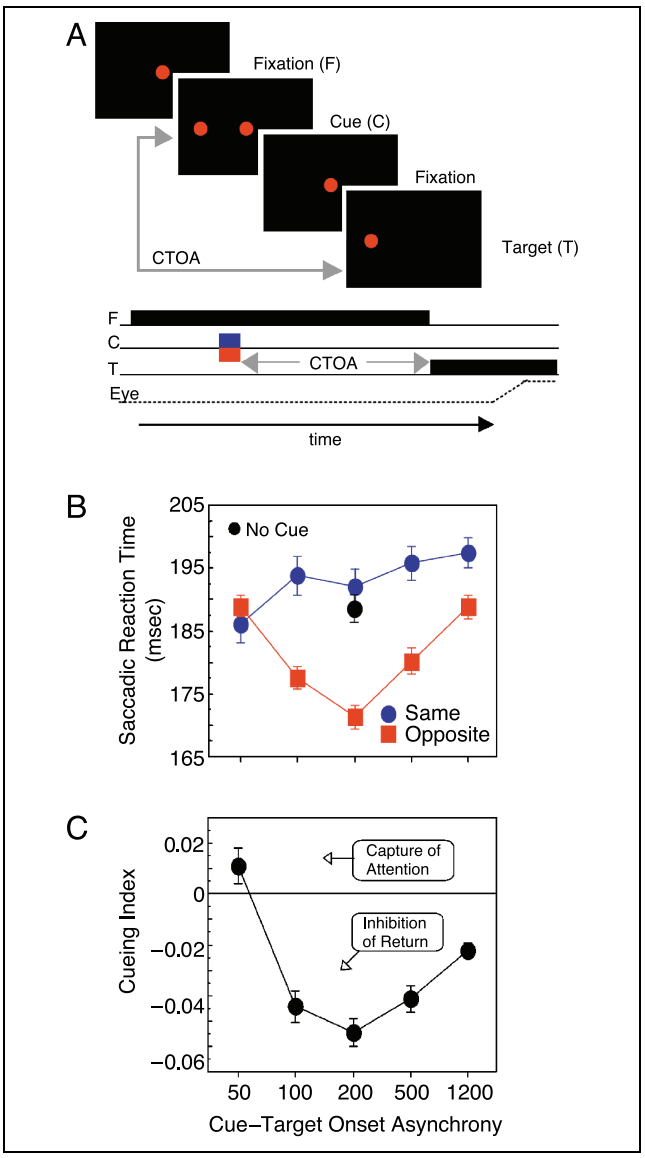

Figure 2. (A) Overview of the cue–target task (see text for description). (B) Mean correct saccadic reaction times when the cue and the target appeared at the same (blue) and opposite (red) locations and when no cue preceded the target (black). This represents the data from both monkeys across all sessions for which the neural activity is described. (C) The cueing index obtained from these data shows the difference between same and opposite conditions. Error bars represent ±1 standard error of the mean.

processing. The capture of attention is thought to originate early in sensory processing (Snowden, 2002; Egeth & Yantis, 1997; Steinmetz et al., 1994; Jonides, 1981), even before the cortical registration of visual input (e.g., Folk, Remington, & Wright, 1994). By contrast, mixed opinions have been raised for inhibition of return. Some researchers have proposed that inhibition of return is generated within the oculomotor system, therefore originating late in the sensory to motor processing stream (e.g., Taylor & Klein, 1998, 2000; Rafal, Calabresi, Brennan, & Sciolto, 1989). By contrast, other researchers have proposed that it originates early in sensory processing (Hopfinger, 2005; Prime & Ward, 2004; Reuter-Lorenz, Jha, & Rosenquist, 1996; Posner & Cohen, 1984). By probing changes in the target-related response at early and late stages of sensory processing, we were able to assess which of these claims are better supported by the data.

RESULTS

Quantifying the Capture of Attention and Inhibition of Return

Figure 2 illustrates the cue–target task that was used to elicit the capture of attention and inhibition of return (for full details, see Methods). In this task, a brief flash of light in the peripheral visual field (the cue) is followed by a second visual stimulus (the target) that appears at the same location as the cue or at the opposite location (Figure 2A). In this study, the cue was irrelevant to the monkeys' task, which was to initiate a saccade to the target's location. As evidenced in the mean correct reaction time data of the two monkeys who performed this task (Figure 2B), the influence of the cue changed depending on the time that elapsed between the onset of the cue and the target, as evidenced in the significant interaction between the variables *Cue–Target Relationship* (same side vs. opposite side) and *Cue–Target Onset Asynchrony* (*CTOA*; 50 msec, 100 msec, 200 msec, 500 msec, 1200 msec), $F(4,136) = 35.1$, $p < .05$.[1] At the 50-msec CTOA, the monkeys responded faster when the cue and the target appeared at the same location (blue below red). The same outcome has been observed in human observers and it has been interpreted as evidence of the capture of attention by the salient cue (e.g., Fecteau, Bell, Dorris, & Munoz, 2005; Posner & Cohen, 1984; Jonides, 1981). At the longer CTOAs, the monkeys responded more slowly when the cue and the target appeared at the same location (red below blue). This same-location disadvantage has been observed in human observers as well and it signifies the behavioral manifestation of inhibition of return (Posner, Rafal, Choate, & Vaughan, 1985; Posner & Cohen, 1984). The difference between the same and opposite cueing conditions represents these biases in orienting attention most clearly. This difference is shown as a normalized cueing index in Figure 2C [(opposite − same saccadic reaction time)/(opposite + same saccadic reaction time)].

Neuron Differences across Cue–Target Onset Asynchronies

The neurons in this study were divided into two classes on the basis of their distinct characteristics: Visual neurons reside in the superficial layers and visuomotor neurons reside in the intermediate layers of the colliculus (see Methods for details regarding cell classification). In the no-cue condition, visual neurons produce a single volley of activity shortly after the target appeared in their receptive field (Figure 1, top). Visuomotor neurons produce two volleys of activity in the same condition: The first signifies the registration of the visual target in the response field of the neuron and the second signifies the initiation of the saccade to the target's location (Figure 1, bottom). The same representative neurons shown in Figure 1 are shown in Figure 3 to reveal the changes in neural activity during trials when the cue appeared at the same location as the target or at the opposite location across the 50, 100, 200, and 500 msec CTOAs (top to bottom). These changes in the target-related response can be more easily visualized in Figure 4, which illustrates the target-related cueing index [(same − opposite spikes/sec)/(same + opposite spikes/sec)] averaged across every visual (Figure 4B) and visuomotor (Figure 4C) neuron sampled in this study. The behavioral data obtained from the same testing sessions are redrawn from Figure 2C for direct comparison (Figure 4A).

As reported previously (see Bell et al., 2004; Fecteau, Bell, Dorris, & Munoz, 2005; Fecteau, Bell, & Munoz, 2004; Dorris, Klein, et al., 2002), the target-related cueing index of visuomotor neurons changed depending on the amount of time that elapsed between the onset of the cue and the target, as evidenced in the main effect of CTOA, $F(4,80) = 3.9$, $p < .05$. This pattern of target-related activity was very similar to that obtained in behavior: The peak target-related response was stronger when the cue and the target appeared at the same location at the 50-msec CTOA, corresponding to a same-location advantage, or the capture of attention, in behavior and the peak target-related response was weaker at the longer CTOAs, corresponding to a same-location disadvantage, or inhibition of return, in behavior. The close relationship between target-related activity and saccadic reaction time was also evident in the strong, negative correlation between these measures on a trial-by-trial basis for each neuron (Figure 4C, right).[2]

By contrast, visual neurons produced a pattern of target-related activity that was unlike behavior (Figure 4B, left).[3] The target-related response was weak at the 50-msec CTOA when the cue and the target appeared at the same location, although a same-location

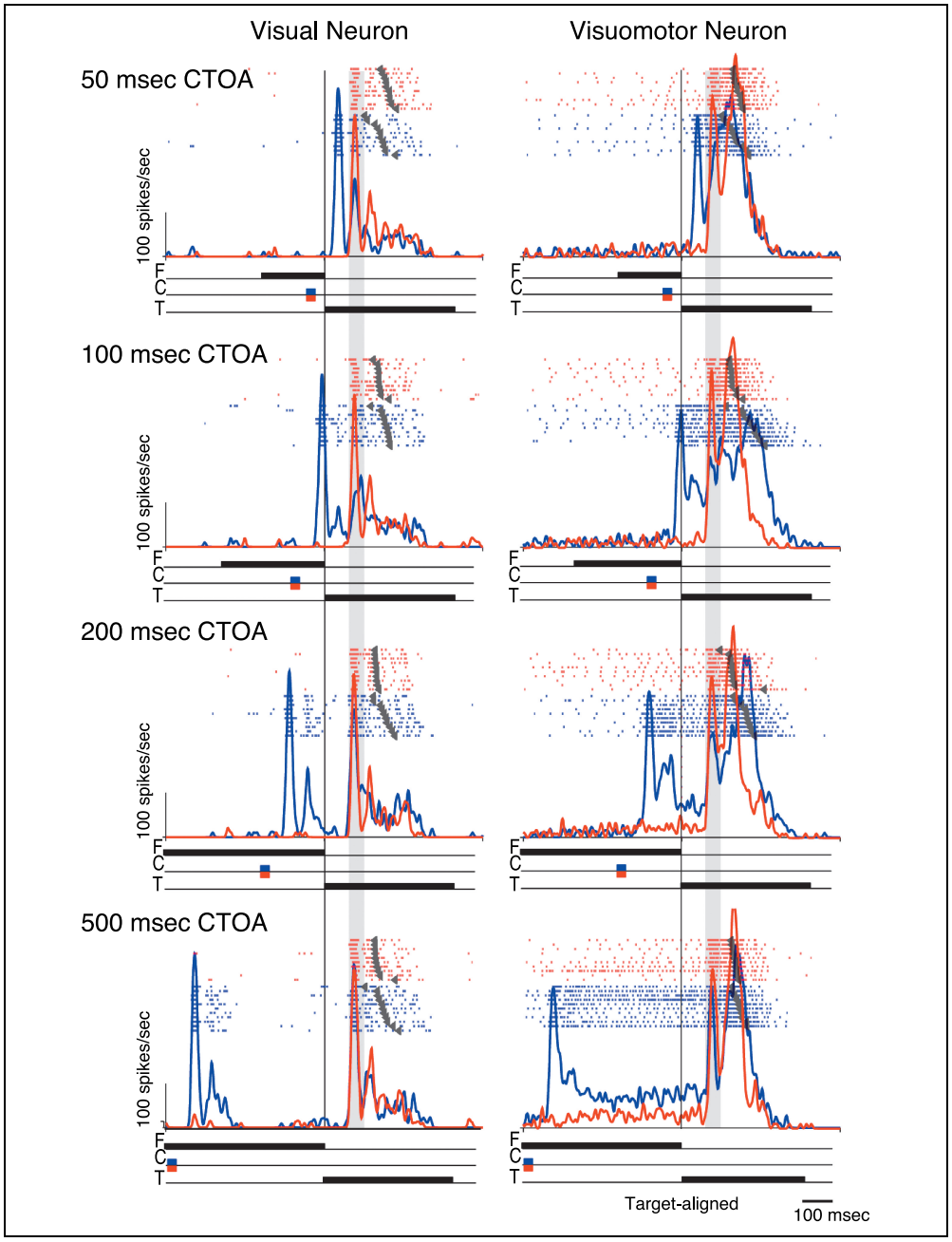

Figure 3. Changes in neural activity across 50, 100, 200, and 500 msec CTOAs (top to bottom) when the cue and the target appeared at the same location (blue) and at opposite locations (red) for the same representative visual (left) and visuomotor (right) neurons as shown in Figure 1. Gray bar represents target-related epoch. Small gray triangles on the rasters represent the onset of the saccade.

advantage was obtained in behavior, the effect was maximal at 100 msec, although the maximal inhibition of return effect was obtained at 200 msec, and it rebounded faster than the behavioral index of inhibition of return. These changes in the target-related cueing index produced a significant main effect of CTOA, $F(4,52) = 4.7$, $p < .05$, but they bore little relationship to behavior, as evidenced in the absence of strong correlations between target-related activity and saccadic reaction times on a trial-by-trial basis for the visual neurons in this sample (Figure 4B, right).

Taken together then, although both visual and visuomotor neurons were influenced significantly by the appearance of the visual cue, only the activity of visuomotor neurons closely matched the changes in behavior.

Capture of Attention

A critical examination of Figure 4C reveals that the "stronger" target-related activity at the 50-msec CTOA did not reach statistical significance ($p > .1$). Does this mean that the simple story of relating stronger target-related activity to the capture of attention is invalid?

Before drawing this conclusion, it is important to consider that the behavioral manifestation of the capture of attention was not compelling either. This originated from averaging sessions that yielded a same-location

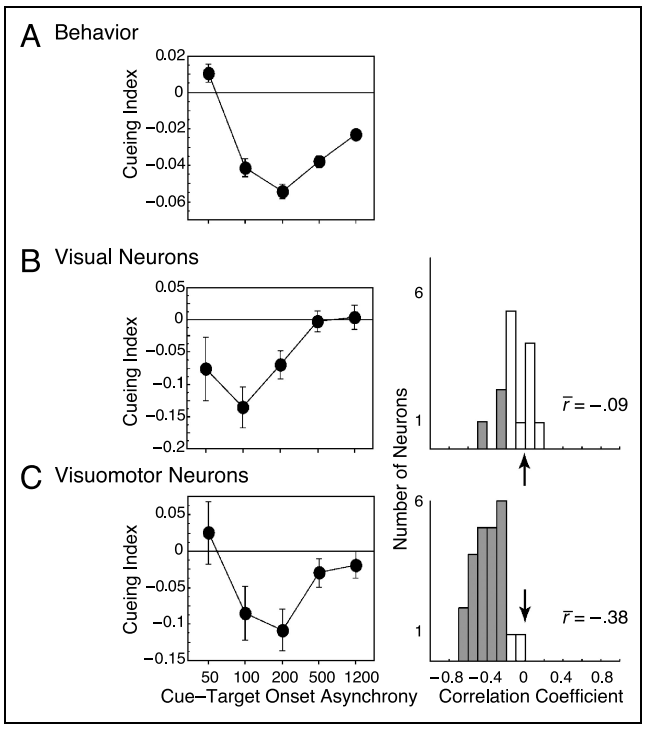

Figure 4. Left: Population averages for saccadic reaction time (A) and peak target-related activity for visual (B) and visuomotor neurons (C). Error bars represent ±1 standard error of the mean. Right: Histograms showing the trial-by-trial correlation between peak target-related activity and saccadic reaction time obtained for every visual (B) and visuomotor (C) neuron in the sample. Gray bars represent the neurons that produced a significant correlation ($p < .05$). Arrows highlight a correlation of 0.

advantage with those yielding a same-location disadvantage at the 50-msec CTOA. To determine whether a relationship between behavior and neural activity existed, we divided the recording sessions on the basis of whether or not the capture of attention was obtained in behavior (as described in the Methods section) and then assessed whether the target-related index differed on the basis of this criterion. For visuomotor neurons (Figure 5A), behavior predicted neural activity. The main effect of Capture (presence vs. absence of capture in behavior) was significant; $F(1,19) = 5.1$, $p < .05$, which reflected stronger target-related activity when a same-location advantage was obtained in behavior, $t(10)$ 4.6, $p < .05$, and weaker target-related activity when a same-location disadvantage was obtained in behavior, $t(9) = -4.8$, $p < .05$. The magnitude of the capture effect in behavior was positively correlated with the magnitude of the change in target-related activity for visuomotor neurons (Figure 5B; $r = .44$). Neither of these effects were observed for visual neurons ($F < 1$; $r = .04$).[4]

Showing that the capture of attention (a same location advantage) in behavior predicts a stronger target-related response does not explain why some sessions yielded the capture of attention and others did not. Exploring among some possible reasons for this difference revealed that it did not depend on which monkey performed the task, $F(1,33) < 1$, $p > .1$, or the amount of experience that each monkey had on this task;[5] instead, it appears to have depended on the region of visual space to which the neuron responded. As illustrated in Figure 5C, the capture of attention was less likely to be obtained when the visual stimuli appeared between 4° and 10° from fixation on the horizontal axis. Although there is nothing magical about these locations in the organization of the nervous system, there is when considering the experiences of these participants—this region marks where we put the visual stimuli when we initially trained these monkeys to perform this cueing task and to make saccadic eye movements more generally. In other words, the capture of attention was not obtained at locations where the monkeys were overtrained to respond to visual stimuli (see also, e.g., Munoz & Fecteau, 2004; Uka & DeAngelis, 2004; Green & Bavelier, 2003; Bichot et al., 1996 for additional evidence of the long-term consequences of training regimes).

Taken together then, the simple story that the capture of attention links to a stronger target-related response is very well founded on the basis of these data. Indeed, this relationship is quite strong—when a same-location advantage is obtained in behavior at the 50-msec CTOA, the target-related response is strong, whereas when a same-location disadvantage is obtained in behavior at the 50-msec CTOA, the target-related response is weak. Methodologically, the key difference between these sessions appears to be the locus of the visual cue and target—the capture of attention was not observed when the cue and the target appeared at regions of the visual field where the monkeys received the greatest amount of training. This striking relationship between target-related activity and behavior was observed only for visuomotor neurons.

Two Components of Inhibition of Return

Inhibition of return refers to an increase in saccadic reaction times when the cue and the target appear at the same location. This effect has been associated with weak target-related activity when the cue and the target appear at the same location (Fecteau, Bell, Dorris, et al., 2005; Bell et al., 2004; Dorris, Klein, et al., 2002). As evidenced in Figures 3 and 4, weak target-related activity was observed in both visual and visuomotor neurons, indicating that this effect originates in early visual areas and is transmitted throughout the brain. Conceptually, similar findings have been observed in V1 (Judge, Wurtz, & Richmond, 1980), the superficial layers of the superior colliculus (Robinson & Kertzman, 1995; Wurtz et al., 1980), LIP (Robinson et al., 1995), and 7a (Constandtinidis & Steinmetz, 2001; Steinmetz, Connor, Constantinidis, & McLaughlin, 1994) although these

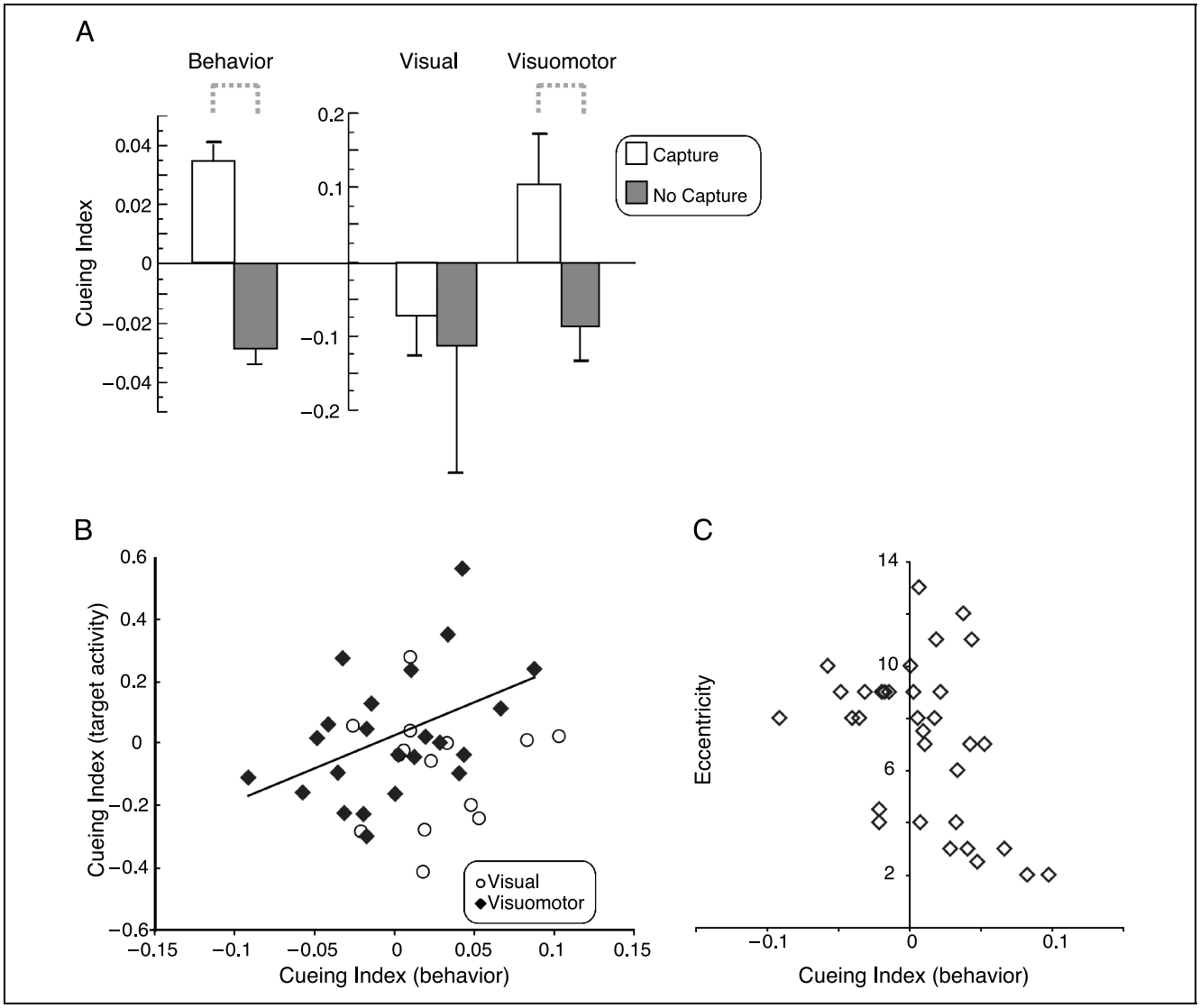

Figure 5. Correlates of capture at the 50-msec CTOA. (A) Sessions in which the capture of attention was (white) or was not (gray) obtained in behavior for visual and visuomotor neurons. (B) Scatterplot showing the relationship between the target-related and behavioral cueing indices for every visual and visuomotor neuron. (C) Scatterplot showing the relationship between eccentricity and behavioral cueing index. Error bars represent ±1 standard error of the mean. Dotted-bars index significant comparisons.

effects were not linked to inhibition of return in these studies.

However, it is noteworthy that the pattern of target-related activity was different between visual and visuomotor neurons. For visual neurons, the rebound occurred faster, and it did not correlate well with behavior on a trial-by-trial basis (Figure 4). This suggests that the change in the target-related response observed in visuomotor neurons is not a faithful rendering of what is present in early visual structures.

To explore what is responsible for this difference between visual and visuomotor neurons, we turned, again, to behavior (Figure 6). Rather than focusing on the cueing index, however, we focused on the mean correct saccadic reaction times from the interaction of the variables *Cue–Target Relationship* and *CTOA* (redrawn from Figure 2B). For comparative purposes, the no-cue data are illustrated in this figure as the single black dot. This plot reveals that the inhibition-of-return effect obtained in this study consists of two components, longer reaction times when the cue and the target appear at the same location (in blue) and shorter reaction times when the cue and the target appear at opposite locations (in red), $F(4,172) = 10.3, p < .05$ and $F(4,172) = 61.4, p < .05$, respectively. Importantly, the facilitated responding to the opposite location produced a distinct V-shaped pattern in these data.

Plotting neural activity in the same way (Figure 6) for visual and visuomotor neurons (keeping in mind that target-related activity and saccadic reaction time are inversely related) revealed a significant change in target-related activity when the cue and the target appeared

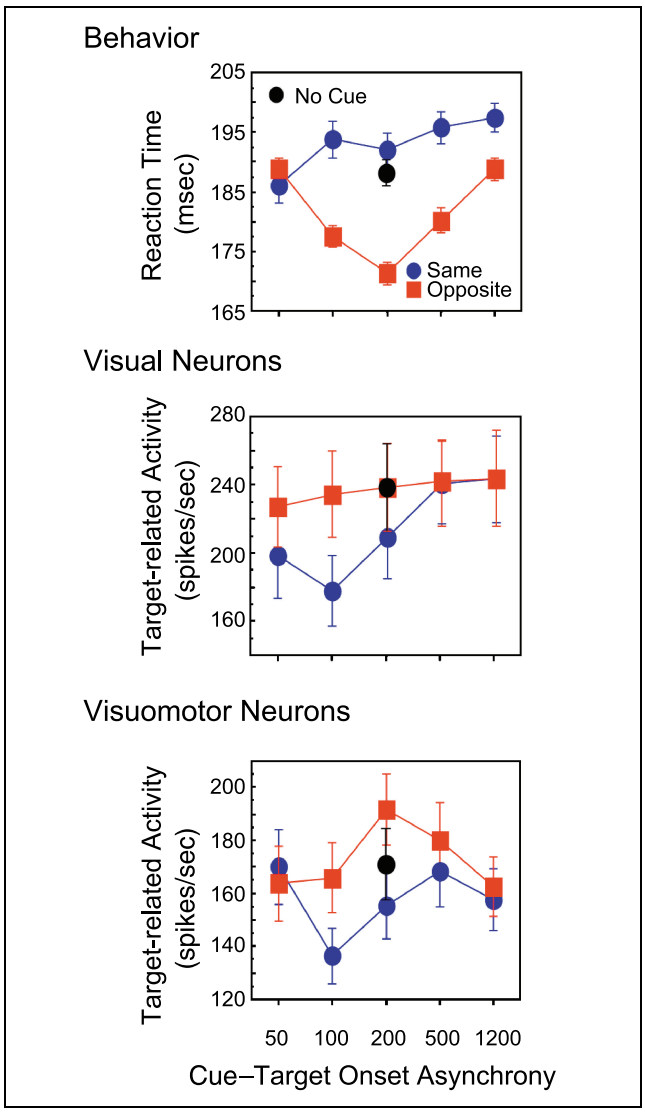

Figure 6. Correlates of inhibition of return. Population averages for mean correct saccadic reaction times and peak target-related activity for visual and visuomotor neurons when the cue and the target appear at the same (blue) and opposite (red) locations. Black circles represent population averages for no-cue trials. Error bars represent ±1 standard error of the mean.

at the same location for both visual and visuomotor neurons, as evidenced as a main effect of CTOA when only the same-location data were analyzed (Fs > 4.4, ps $< .05$). However, this change in neural activity did not match behavior perfectly for either visual or visuomotor neurons—reaction times remained relatively flat when the cue and the target appeared at the same location after the 50-msec CTOA, whereas neural activity rebounded across time. This lack of correspondence between target-related activity and behavior, particularly at the longer CTOAs, is described in further detail in the Discussion section. Regarding when the cue and the target appeared at opposite locations, only visuomotor neurons produced a similar V-shaped pattern, as evidenced as a main effect of CTOA when only the opposite side data were analyzed, $F(4,80) = 9.1$, $p < .05$ [visual neurons $F(4,52) < 1.4$, $p > .1$]. Finally, comparing the data obtained from the cueing conditions at the 200-msec CTOA with the no-cue condition, which shared the same timing as these cued trials, revealed a close relationship between behavior and neural activity for visuomotor neurons, but not for visual neurons. The trend towards a same-location disadvantage and a strong opposite-location advantage at the 200-msec CTOA, when compared to the no-cue condition in behavior [same location cue vs. no cue, $F(1,34) = 2.3$, $p < .14$; opposite location cue vs. no cue, $F(1,34) = 102.2$, $p < .05$], was accompanied by the same pattern for visuomotor neurons [same location cue vs. no cue, $F(1,20) = 3.55$, $p < .075$; opposite location cue vs. no cue, $F(1,20) = 10.18$, $p < .05$], but not visual neurons [same location cue vs. no cue, $F(1,13) = 4.6$, $p < .06$; opposite location cue vs. no cue, $F(1,13) < 1$]. Once again, a close relationship between behavior and the target-related activity exists for visuomotor neurons, but not for visual neurons.

Taken together, two distinct components contributed to the inhibition of return effect obtained in this study. Slower responding when the cue and the target appeared at the same location was, albeit imperfectly, associated with a weak target-related signal. This effect was observed in both visual and visuomotor neurons, indicating that it originates early in sensory processing and is transmitted throughout the brain. By contrast, faster responding when the cue and the target appeared at opposite locations was associated with a stronger target-related signal. This effect was observed only in visuomotor neurons, indicating that it originates later in sensory processing.

DISCUSSION

A central question in cognitive neuroscience is to understand where cognitive phenomena originate in the brain. Here, we used a levels-of-processing approach to explore this question for the capture of attention and inhibition of return. Previous studies have demonstrated that both of these biases in attention yield observable changes in the neural representation of the target in the intermediate layers of the superior colliculus (Bell et al., 2004; Fecteau, Bell, et al., 2004; Dorris, Klein, et al., 2002). Nevertheless, it was impossible to know from these studies whether this change in the target-related response originated early in visual processing and was faithfully transmitted throughout the rest of the brain or whether it was modified through many possible intermediaries. Here, we compared the activity of neurons in the superficial and intermediate layers of the superior colliculus to provide an answer to this question. These regions receive input from different regions of the brain (as described in the Introduction) and contain neurons

with unique qualities: Visual neurons reside in the superficial layers and visuomotor neurons reside in the intermediate layers (Wurtz et al., 1980; Goldberg & Wurtz, 1972; Wurtz & Goldberg, 1971, 1972). Here, we have shown that both classes of neurons were modified by the appearance of the cue. Even so, only the target-related activity of visuomotor neurons consistently matched orienting behavior.

Capture of Attention Originates "Late" in Visual Processing

For visuomotor neurons, the capture of attention was associated with stronger target-related activity when the cue and the target appeared at the same location (Figure 5; see also Bell et al., 2004; Fecteau, Bell, et al., 2004). Here, we have shown that this relationship is compelling: Experimental sessions that yielded a same-side advantage at the 50-msec CTOA also yielded a stronger target-related response, whereas sessions yielding a same-location disadvantage at the 50-msec CTOA yielded a weaker target-related response.

This close relationship between target-related activity and orienting behavior was limited to visuomotor neurons. Visual neurons did not produce this relationship; instead, a weak target-related response was obtained uniformly at the 50-msec CTOA, indicating that whatever is responsible for the capture of attention, it originates later in visual processing.

Of the possible methodological reasons for this difference that we could assess, the capture of attention was observed less often at loci with which the monkeys had a great deal of experience. This suggests, perhaps, that excessive practice eventually eliminates the capture of attention and that this occurs, at least initially, in a spatially specific manner. Other consequences of practice have been observed in this task as well. In monkey observers, practice weakens and can eliminate inhibition of return at longer CTOAs (cf. Dorris, Klein, et al., 2002; Dorris, Taylor, Klein, & Munoz, 1999) and the crossover from the capture of attention to inhibition of return is shifted to earlier CTOAs when comparing the data of monkey (crossover at 80 msec) and human (crossover at 200 msec) observers (Fecteau, Bell, Dorris, et al., 2005). Future studies should explore the reasons why this change occurs.

Inhibition of Return Originates at "Early" and "Late" Stages of Visual Processing

Under the conditions of this study, the inhibition of return effect comprised two components—slower responding when the cue and the target appear at the same location and faster responding when the cue and the target appear at opposite locations. A benefit for the opposite location has been observed, albeit inconsistently, in human investigations as well (Machado & Rafal, 2004; Snyder, Kingstone, & Schmidt, 2001; Pratt, Spalek, & Bradshaw, 1999; Posner & Cohen, 1984). Here we have shown that both of these effects have distinct neural correlates. The same-location disadvantage corresponds to weak target-related activity when the cue and the target appeared at the same location. This effect was observed in visual and visuomotor neurons, indicating that the mechanism responsible occurs very early in visual processing. Indeed, a conceptually similar reduction of the incoming visual signal has been observed in V1 (Judge et al., 1980), indicating that the source of this effect originates earlier than this structure, perhaps at the level of the retina (Judge et al., 1980). By contrast, facilitation to the opposite location was associated with an enhancement of target-related activity. This effect was observed only for visuomotor neurons and therefore originates later in visual processing. Changes in the peak target-related response cannot account for all aspects of the data, however. At longer CTOAs (500 and 1200 msec), the correspondence between target-related activity and behavior grows weaker, as evidenced in both the trial-by-trial correlations (see footnote 3) and the rebound of target-related activity despite relatively flat reaction times. Currently, we are exploring what is responsible for both the enhancement effect when the cue and the target appear at opposite locations and the weaker correspondence between neural activity and behavior at the longer CTOAs.

One important issue to keep in mind is that, on the basis of neurophysiological evidence, inhibition of return does not appear to originate from one source, but from several. For instance, Bichot and Schall (2002) reported that inhibition of return was associated with a delay of the neural selection of the target from the distractor in the frontal eye fields when using a visual search task. In search, the neural correlate of target selection is not observed in the first peak of activity registering the presence of a visual object in the neuron's response field, but in the later evolution of the sensory/cognitive response. Placing this target selection activity within the early/late dichotomy used here, the neural correlate of target selection is observed in the intermediate layers of the superior colliculus, much like in the frontal eye fields, but it is not observed in the superficial layers (McPeek & Keller, 2002). Thus, the distinct components of the inhibition of return effect can be manifest neuronally in several different ways: early in sensory processing (here), late in sensory processing (here, Bichot & Schall, 2002), and even within different neural epochs (cf., here vs. Bichot & Schall, 2002). This indicates that inhibition of return does not originate from one single neural process nor does it appear to be a single phenomenon, instead, many neural processes can lead to this slowing of response time. How this relates to the many flavors of inhibition of return observed across different tasks (e.g., Lupianez, Milan, Tornay, Madrid, & Tudela, 1997; Tipper, Weaver,

Jerreat, & Burak, 1994; Klein, 1988; Maylor & Hockey, 1985) remains to be established.

It was brought to our attention that our investigations (here, Bell et al., 2004; Fecteau, Bell, et al., 2004; Dorris, Klein, et al., 2002), and all studies showing that inhibition of return has a strong sensory component (Hopfinger, 2005; Prime & Ward, 2004; Reuter-Lorenz et al., 1996; Posner & Cohen, 1984), do not reveal the cause of inhibition of return, but reflect the consequences of it. This is an important point because it is possible to imagine that the oculomotor network generates inhibition of return by feeding back to early sensory areas and suppressing the incoming target-related response. According to this view then, the evidence for the oculomotor and sensory bases of inhibition of return simply reflects the cause and the consequence of inhibition of return, respectively.

Although we do not dismiss the thoughtful reasoning behind this point, there are several reasons why we believe, for the cue–target task used in this study, that inhibition of return reflects a habituated sensory response occurring in early sensory areas that is subsequently transmitted through the rest of the brain, rather than reflecting an active suppression mechanism of the oculomotor system. For one, the evidence used as support of the oculomotor basis of inhibition of return can be interpreted as evidence of sensory processing as well. (a) Temporal–nasal asymmetries have been observed in inhibition of return. This has been interpreted as evidence that the superior colliculus is involved in generating inhibition of return because the projection from the retina to the superior colliculus has a strong asymmetry (Rafal, Calabresi, et al., 1989). This line of evidence has been met with skepticism because the very strong asymmetry found in rodents and cats is less strong in primates; however, there is an additional problem with this interpretation. Even if an asymmetry in the retinal projection to the superficial layers of the superior colliculus exists and promotes the behavioral effect observed in humans, we cannot forget that this is a sensory input that is registered in a sensory structure (the superficial layers of the colliculus) before it is sent by way of the pulvinar (and possibly additional cortical stations) to the intermediate layers of the superior colliculus. That is, this asymmetry reflects a sensory, not an oculomotor, bias. (b) Although lesions to the superior colliculus eliminate inhibition of return (Sapir, Soroker, Berger, & Henik, 1999), this dependence could originate from the sensory or oculomotor regions of the superior colliculus.

Second, the proposal that active suppression of the incoming target-related response is responsible for weak target-related response cannot account for all of the data obtained in this study. (a) In general, feedback signals do not influence the initial sensory peak, but influence later sensory/cognitive epochs (Lamme & Roelfsema, 2000). In this study, it was the initial registration of the target that was correlated with changes in behavior, not later epochs. (b) The reduction of the target-related response was observed in the superficial and intermediate layers of the colliculus. As mentioned in the Introduction, the superficial layer is an open-looped system (Clower et al., 2001), and therefore, cannot receive direct inhibitory signals from oculomotor structures.

Third, the strongest evidence in support of the oculomotor generation of inhibition of return is that planning a saccade, in the absence of a peripheral visual cue, produces inhibition of return (Rafal, Calabresi, et al., 1989). Although we do not wish to extend our neurophysiological evidence to these findings, there is existing behavioral evidence indicating that a pure oculomotor view is not perfectly validated. (a) Similar conditions can produce inhibition of return, even when no saccadic plan is required of the participants. For instance, Taylor and Klein (2002) obtained a significant inhibition-of-return effect following the presentation of a central arrow, although their task was to ignore the arrow and to initiate a manual response to a peripherally appearing target (pp. 1644–1645). (b) Antisaccade versions of the cue–target task (more accurately, the target–target task), which pit sensory input against oculomotor planning, uniformly show that inhibition of return follows the visual stimulus, not the saccade (Fecteau, Au, et al., 2004; Rafal, Egly, & Rhodes, 1994). Although this may seem to be consistent with the idea that reduced sensory processing is the consequence, as opposed to the cause, of inhibition of return, consider, however, that this inhibited sensory response should be at the same location as end point of the preceding saccadic eye movement, not the locus of the preceding visual stimulus. Simply put, this outcome is opposite to what would be expected if inhibition of return was generated through oculomotor planning.

Taken together, although it is important to keep in mind that the causes and the consequences of inhibition of return may not be the same thing, the evidence, nevertheless, suggests that inhibition of return does not originate from one neural mechanism (as indicated through neurophysiological studies to date), and the generation of inhibition of return does appear to have some dependence on sensory processing.

Importance of Comparing Neural Activity Directly to Behavior

Finally, our study has important ramifications for thinking, more generally, of how to interpret changes in neural activity. In cognitive neurophysiology, the goal is to understand how cognitive behaviors are represented in the brain; therefore, it seems natural that researchers exploring this relationship should directly show how changes in neural activity correspond to changes in cognitive behavior. Many laboratories routinely show these relationships (e.g., Ignashchenkova, Dicke,

Haarmeier, & Their, 2004; Bichot & Schall, 1999, 2002; Dorris & Munoz, 1998; Shadlen & Newsome, 1996; Schall & Hanes, 1993; Britten, Shadlen, Newsome, & Movshon, 1992); however, many laboratories do not. In this study, we have shown that conducting detailed analyses of behavior can explain some of the variability present in the neural data (see, e.g., Figure 5). Moreover, we have shown that it is possible to obtain striking changes in neural activity even though these changes do not share any immediate relationship to behavior (see Figures 4, 5, and 6). These observations have an important consequence; they suggest that, through mapping this relationship between brain and behavior, we can better interpret what is being monitored at the end of an electrode.

METHODS

Two male rhesus monkeys (weighing between 7 and 10 kg) participated in this study. The techniques used to collect behavioral data and to obtain physiological recordings have been described previously (Munoz & Istvan, 1998) and were approved by the Queen's University Animal Care committee.

Behavioral Task

Each trial began with the monkeys maintaining gaze upon a central fixation marker for 500–1000 msec. Then a visual stimulus appeared briefly (30 msec) to the left or to the right field. The fixation marker remained in view until the target appeared, which occurred at one of five lags after the initial appearance of the cue (50, 100, 200, 500, or 1200 msec). The monkeys received a liquid reward for initiating a saccade to the target's location within 500 msec of its appearance. The visual stimuli consisted of red light-emitting diodes (0.03 cd/m^2) that were rear-projected onto a tangent screen in front of the participant. One of the cue–target locations was positioned to elicit the optimal response from the neuron being monitored and the other cue–target location appeared at its mirror position (across the vertical and horizontal axes). CTOA and the location of the target relative to the cue (*Cue–Target Relationship*; same vs. opposite) were equally probable and randomly selected during the testing session. In addition, a neutral condition was interleaved with the cued trials. The timing of these no-cue trials was identical to the 200-msec CTOA, except no cue was presented. The data obtained from these no-cue trials were used to help classify the neuron.[6] In each recording session, the experimenter attempted to obtain a minimum of 10 correct trials for each condition, yielding a total of 220 trials: 200 cued trials originating from the combination of *Cue–Target Relationship* (same vs. opposite), *CTOA* (50, 100, 200, 500, vs. 1200 msec), and *Target location* (in response field vs. out of response field); and 20 no-cue trials (10 in and 10 out of response field). Although full counts of trials were achieved in most sessions, they were not achieved in all sessions because the isolation of the neuron was lost or because the monkey was satiated.

Saccadic reaction time was used as the behavioral estimate of spatial attention because it encourages the immediate applicability of our findings to previous human studies, as many have used reaction time as the primary dependent measure (e.g., Maylor, 1985; Posner & Cohen, 1984; Jonides, 1981).

Behavioral and Neural Analyses

Sixty neurons met the criteria for inclusion in this study: at least 4 observations were obtained per condition (i.e., in the factorial breakdown of the experimental design; 10 or more observations were more common) and the average peak target-related burst (maximum activity occurring 70 through 120 msec, target-aligned raster) exceeded 70 Hz in the no-cue condition. The action potentials on each trial were convolved using a Gaussian kernel ($\sigma = 10$).

We used several criteria to divide the neurons into visual (superficial layer neurons) and visuomotor (intermediate layer neurons) groups (see Figure 1A for representative examples). All neurons elicited a burst of neural activity in association with the appearance of cue (70–120 msec cue-aligned) at the 500- and 1200-msec CTOAs, indicating the presence of a pure visual response. We used the micrometer depth measures from the microdrive to help guide the classification of superficial versus intermediate neurons. However, there are significant limitations to using this measure only: (1) we cannot assume identical compression of tissue in every experimental session (requiring similar descension rates or similar waiting period between reaching the superior colliculus and lowering electrode) and (2) the dorsal surface of the superior colliculus cannot always be reliably determined after repeated penetrations. Therefore, visual and visuomotor neurons were distinguished on the basis of two further characteristics. First, the differences between the peak target-related and saccade-related (maximum activity occurring −20 through 10 msec, saccade aligned raster) activities were used to distinguish these classes. Visual neurons had stronger target-related than saccade-related activities. Visuomotor neurons had stronger saccade-related than target-related activities. Second, peak neural activities in the saccade-related epoch were compared when the cue appeared at the same and opposite location as the target. The location of the target relative to the cue does not influence the saccadic burst (Fecteau, Bell, & Munoz, 2004), but has a large influence on target-related activity (Bell et al., 2004; Fecteau, Bell, & Munoz, 2004; Dorris, Klein, et al., 2002; Robinson & Kertzman, 1995; Robinson et al., 1995; Steinmetz et al., 1994). Therefore,

visual neurons yield significant differences between these conditions at this saccade-related epoch, whereas visuomotor neurons do not. On the basis of these criteria, 22 neurons fell into the visual category, 35 neurons fell into the visuomotor category, and 3 neurons could not be classified and were therefore removed from the analysis. For about half of the neurons in this sample, a delayed saccade task confirmed that these, albeit idiosyncratic, criteria successfully distinguished visual and visuomotor neurons. The experimenter visually confirmed the classification of every neuron in the sample.

In addition, the tonic activity of neurons was assessed because this activity can be quite different for visual and visuomotor neurons: Visual neurons have little tonic activity, whereas some visuomotor neurons have a great deal of tonic activity. The ratio of the pretarget response (average activity 45–65 msec, cue and target in response field, target aligned, CTOAs of 200 and 500 msec) to the peak target-related response (no-cue trials and 1200 msec CTOA opposite condition) was generated for each neuron. For neurons with low tonic activity, pretarget activity was less than 10% of the peak target-related response. For neurons with moderate tonic activity, the value of this ratio fell between 10% and 30%. Finally, for neurons with high tonic activity, the value of this ratio was >30%. Visual neurons fell in the low ($n = 8$) and moderate categories ($n = 13$). Only one visual neuron was placed in the high tonic category. Visuomotor neurons were members of all three categories (low $n = 7$, moderate $n = 14$, high $n = 14$). We wanted to equate the neurons on the basis of this tonicity measure (1) to ensure that the inclusion of high tonic visuomotor neurons was not the sole source of differences between visual and visuomotor neurons and (2) because several analyses in this article could not be accomplished on neurons with high tonic activity. Therefore, neurons from the high tonic condition were excluded from further analyses, leaving 21 visual and 21 visuomotor neurons. Finally, to ensure that the visual neurons resided only in the superficial layers, we included only those neurons that were encountered within the first 1050 μm after reaching the superior colliculus. This criterion removed an additional 7 neurons from the analysis, resulting in 14 visual and 21 visuomotor neurons.

The behavioral data included in this study were obtained from the same sessions as the neural data (35 sessions in total). Only the data from correctly performed trials were included in these analyses, which consisted of a single saccade that was initiated to the target's location within 125–300 msec of the target's appearance. These cutoffs removed anticipatory responses (<70 msec; <0.4% of the data), very short latency saccades (70–124.9 msec; <4% of the data), and atypically long responses (>300 msec; 1% of the data).

The average peak target-related responses were obtained for each neuron in each condition and were entered into mixed-design analysis of variance (ANOVA), including the between-subjects factor of *Class* (visual versus visuomotor) and the within-subject variables of *Cue–Target Relationship* (same location vs. opposite location), and *CTOA* (50, 100, 200, 500 vs. 1200 msec). For the neural analyses, only the trials when the target appeared within the response field of the neuron were analyzed because only these trials yield a target-related response. In this initial analysis, the variable *Class* produced a main effect, $F(1,33) = 6.9$, $p < .05$, it interacted with *CTOA*, $F(4,132) = 3.2$, $p < .05$, and with *Cue–Target Relationship* and *CTOA*, $F(4,132) = 2.4$, $p < .06$. Because of these interactions, we describe the outcomes of repeated-measures ANOVAs (*Cue–Target Relationship* and *CTOA*) for the visual and visuomotor neurons in the Results section separately. The mean correct saccadic reaction time data (from every experimental session included in the neural analyses) were entered into repeated-measures ANOVA involving the factors *Cue–Target Relationship* and *CTOA*. For the behavioral analyses, the factor *Class* was not included and the data were collapsed with respect to the absolute location of the target (in or out of the response field) because these variables were not of theoretical interest to this article.

A cueing index was used to show the difference between same and opposite locations directly for both behavior and target-related activity. For behavior, this index followed the equation [(opposite − same saccadic reaction time)/(opposite + same saccadic reaction time)] so that positive values indexed shorter latency responses when the cue and the target appeared at the same location. For neural activity, this index followed the equation [(same − opposite spikes/sec)/(same + opposite spikes/sec)] so that positive values indexed stronger target-related activity when the cue and target appeared at the same location.

Trial-by-trial correlation analyses compared the saccadic reaction time and peak target-related activity on each trial for every neuron (i.e., session). Same and opposite cueing conditions (target in response field only) and all CTOAs were included in this correlation analysis and the significance of the r value for each neuron was determined with a t test.

At the 50-msec CTOA, sessions yielding the capture of attention in behavior were separated from sessions not yielding the capture of attention in behavior. This division was not based on a statistical difference between same and opposite cueing conditions for each session; instead, it was based upon whether the behavioral cueing index fell above or below zero. On the basis of this criterion, 23 sessions yielded a same-side advantage (11 visuomotor and 12 visual neurons) and 12 sessions did not (10 visuomotor and 2 visual). A mixed-design ANOVA was used to assess whether there was a difference between visual and visuomotor neu-

rons, using the factors *Class* (visual vs. visuomotor) and *Capture* (present vs. absent). A between-subject ANOVA, conducted separately for visuomotor and visual neurons, was used to assess whether the presence or absence of Capture resulted in different patterns of target-related activity and, when a difference was obtained, *t* tests were used to determine whether the target-related index in each bin was significantly different from zero (e.g., was the stronger target-related cueing index when the capture of attention was obtained for visuomotor neurons significant?). Finally, because we divided the sessions on the basis of whether the cueing index fell above or below zero, a correlation analysis was to determine whether the relationship between the magnitude of the behavioral cueing index and target-related cueing indices for visual and visuomotor neurons separately.

For the analyses of inhibition of return, the behavioral (the mean correct saccadic reaction times) and neural (mean peak target-related responses) data were analyzed separately for same location and opposite location cueing conditions across *CTOA*.

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Notes

1. The main effects of Cue Location, $F(1,34) = 52.3$, $p < .05$, and CTOA, $F(4,136) = 25.1$, $p < .05$ were also significant in this analysis.
2. This correlation consists of all cued trials (same and opposite) at all CTOAs, when the target appeared within the response field of the neuron. Importantly, similar negative correlations between target-related activity and saccadic reaction times were obtained when each CTOA was tested alone (average correlation 50 msec = −.39, 100 msec = −.45, 200 msec = −.52, 500 msec = −.33, 1200 msec = −.21).
3. As described in the Methods section, a mixed-design ANOVA revealed that the factor Class, which directly compares visual and visuomotor neurons, was involved in higher-order interactions with Cue–target relationship and CTOA.
4. An analysis comparing involving the factors Class (visual vs. visuomotor) and Capture (present vs. absent) resulted in a marginal two-way interaction involving these factors, $F(3,31) = 2.4$, $p < .1$. This interaction reflected that the presence of the capture of attention in behavior at the 50-msec CTOA resulted in strong target-related activity for visuomotor neurons and weak target-related activity for visual neurons, $F(1,21) = 4.1$, $p < .06$. No difference between visual and visuomotor neurons was obtained during the sessions when the capture of attention was not obtained in behavior, $F(1,10) < 1$. Only two visual neurons contributed to this latter comparison, making this analysis unreliable.
5. Although this was true for the present dataset, all evidence of a same-location advantage was eliminated eventually from one monkey after further extensive training.
6. It was brought to our attention that the no-cue condition was an insufficient control condition because we should have matched the timing of events for all CTOAs, not just the 200-msec CTOA. This is an important point because changing the fixation duration also changes the speed with which monkeys respond (Pare & Munoz, 1996). However, if the fixation duration produced a systematic change in saccadic reaction times, then both same and opposite cueing conditions would be influenced in the same manner (i.e., it would produce a main effect, not interact with same and opposite cueing conditions). Therefore, despite the flaw in the design of the no-cue trials, it does not change the conclusions of this article.

REFERENCES

Bell, A. H., Fecteau, J. H., & Munoz, D. P. (2004). Using auditory and visual stimuli to investigate the behavioral and neuronal consequences of reflexive covert orienting. *Journal of Neurophysiology, 91,* 2172–2184.

Bichot, N. P., & Schall, J. D. (1999). Effects of similarity and history on neural mechanisms of visual selection. *Nature Neuroscience, 2,* 549–554.

Bichot, N. P., Schall, J. D., & Thompson, K. G. (1996). Visual feature selectivity in frontal eye fields induced by experience in mature macaques. *Nature, 381,* 697–699.

Bichot, N. P., & Schall, J. D. (2002). Priming in macaque frontal cortex during popout visual search: Feature-based facilitation and location-based inhibition of return. *Journal of Neuroscience, 22,* 4675–4685.

Britten, K. H., Shadlen, M. N., Newsome, W. T., & Movshon, J. A. (1992). The analysis of visual motion: A comparison of neuronal and psychophysical performance. *Journal of Neuroscience, 12,* 4745–4765.

Clower, D. M., West, R. A., Lynch, J. C., & Strick, P. L. (2001). The inferior parietal lobule is the target of output from the superior colliculus, hippocampus, and cerebellum. *Journal of Neuroscience, 21,* 6283–6291.

Constandtinidis, C., & Steinmetz, M. A. (2001). Neuronal responses in area 7a to multiple stimulus displays: II. Responses are suppressed at the cued location. *Cerebral Cortex, 11,* 592–597.

Corbetta, M., & Shulman, G. L. (2001). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3,* 201–215.

Dorris, M. C., Klein, R. M., Everling, S., & Munoz, D. P. (2002). Contribution of the primate superior colliculus to inhibition of return. *Journal of Cognitive Neuroscience, 14,* 1256–1263.

Dorris, M. C., & Munoz, D. P. (1998). Saccadic probability influences motor preparation signals and time to saccadic initiation. *Journal of Neuroscience, 18,* 7015–7026.

Dorris, M. C., Taylor, T. L., Klein, R. M., & Munoz, D. P. (1999). Influence of previous visual stimulus or saccade on saccadic reaction times in monkey. *Journal of Neurophysiology, 81,* 2429–2436.

Egeth, H. E., & Yantis, S. (1997). Visual attention: Control, representation, and time course. *Annual Review of Psychology, 48,* 269–297.

Fecteau, J. H., Au, C., Armstrong, I. T., & Munoz, D. P. (2004). Sensory biases produce alternation advantage found in

sequential saccadic eye movement tasks. *Experimental Brain Research, 159,* 84–91.
Fecteau, J. H., Bell, A. H., Dorris, M. C., & Munoz, D. P. (2005). Neurophysiological correlates of the reflexive orienting of spatial attention. In L. Itti, G. Rees, & J. Tsotsos (Eds.), *Encyclopedia on the neurobiology of attention* (pp. 389–394).
Fecteau, J. H., Bell, A. H., & Munoz, D. P. (2004). Neural correlates of the automatic and goal-driven biases in orienting spatial attention. *Journal of Neurophysiology, 92,* 1728–1737.
Folk, C. L., Remington, R. W., & Wright, J. H. (1994). The structure of attentional control: Contingent attentional capture by apparent motion, abrupt onset, and color. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 317–329.
Fries, W. (1984). Cortical projections to the superior colliculus in the macaque monkey: A retrograde study using horseradish peroxidase. *Journal of Comparative Neurology, 230,* 55–76.
Goldberg, M. E., & Wurtz, R. H. (1972). Activity of superior colliculus in behaving monkey. II. Effect of attention on neuronal responses. *Journal of Neurophysiology, 35,* 560–574.
Gottleib, J. P., Kusunoki, M., & Goldberg, M. E. (1998). The representation of visual salience in monkey parietal cortex. *Nature, 391,* 481–484.
Green, C. S., & Bavelier, D. (2003). Action video game modifies visual selective attention. *Nature, 423,* 534–537.
Hopfinger, J. (2005). Electrophysiology of reflexive attention. In L. Itti, G. Rees, & J. Tsotsos (Eds.), *Encyclopedia on the neurobiology of attention* (pp. 219–235).
Ignashchenkova A., Dicke, P. W., Haarmeier, T., & Their, P. (2004). Neuron-specific contribution of the superior colliculus to overt and covert shifts of attention. *Nature Neuroscience, 7,* 56–64.
Jonides, J. (1981). Voluntary vs. Automatic control over the mind's eye's movement. In J. B. Long & A. D. Baddeley (Eds.), *Attention and performance IX* (pp. 187–203). Hillsdale, NJ: Erlbaum.
Judge, S. J., Wurtz, R. H., & Richmond, B. J. (1980). Vision during saccadic eye movements: I. Visual interactions in striate cortex. *Journal of Neurophysiology, 43,* 1133–1155.
Klein, R. (1988). Inhibitory tagging system facilitates visual search. *Nature, 334,* 430–431.
Klein, R. (2000). Inhibition of return. *Trends in Cognitive Sciences, 4,* 138–147.
Kuypers, H. G., & Lawrence, D. G. (1967). Cortical projections to the red nucleus and the brain stem in the Rhesus monkey. *Brain Research, 4,* 151–188.
Lamme, V. A. F., & Roelfsema, P. R. (2000). The distinct modes of vision offered by feedforward and recurrent processing. *Trends in Neurosciences, 23,* 571–579.
Lui, F., Gregory, K. M., Blanks, R. H., & Giolli, R. A. (1995). Projections from visual areas of the cerebral cortex to pretectal nuclear complex, terminal accessory optic nuclei, and superior colliculus in the macaque monkey. *Journal of Comparative Neurology, 363,* 439–460.
Lupianez, J., Milan, E. G., Tornay, F. J., Madrid, E., & Tudela, P. (1997). Does IOR occur in discrimination tasks? Yes, it does, but later. *Perception & Psychophysics, 59,* 1241–1254.
Lynch, J. C., Graybiel, A. M., & Lobeck, L. J. (1985). The differential projection of two cytoarchitectonic subregions of the inferior parietal lobule of macaque upon the deep layers of the superior colliculus. *Journal of Comparative Neurology, 235,* 241–254.
Machado, L., & Rafal, R. (2004). Inhibition of return generated by voluntary saccades is independent of attentional momentum. *Quarterly Journal of Experimental Psychology A, 57,* 789–796.
Maylor, E. A. (1985). Facilitatory and inhibitory components of orienting in visual space. In M. I. Posner & O. S. M. Marin (Eds.), *Attention and performance XI* (pp. 189–204). Hillsdale, NJ: Erlbaum.
Maylor, E. A., & Hockey, G. R. J. (1985). Inhibitory component of externally controlled covert orienting in visual space. *Journal of Experimental Psychology: Human Perception and Performance, 11,* 777–787.
Mays, L. E., & Sparks, D. L. (1980). Dissociation of visual and saccade-related responses in superior colliculus neurons. *Journal of Neurophysiology, 43,* 207–232.
McPeek, R. M., & Keller, E. L. (2002). Saccade target selection in the superior colliculus during a visual search task. *Journal of Neurophysiology, 88,* 2019–2034.
Mesulam, M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology, 10,* 309–325.
Mesulam, M. M. (1999). Spatial attention and neglect: Parietal, frontal and cingulate contributions to the mental representation and attentional targeting of salient extrapersonal events. *Philosophical Transactions of the Royal Society of London, Series B. Biological Sciences, 354,* 1325–1346.
Munoz, D. P., & Fecteau, J. H. (2004). Reconciling the differences in previous trial effects between humans and monkeys. *Canadian Society of Brain, Behavior, and Cognitive Science.*
Munoz, D. P., & Istvan, P. J. (1998). Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *Journal of Neurophysiology, 79,* 1193–1209.
Munoz, D. P., & Wurtz, R. H. (1993). Fixation cells in monkey superior colliculus I. Characteristics of cell discharge. *Journal of Neurophysiology, 70,* 559–575.
Pare, M., & Munoz, D. P. (1996). Saccadic reaction time in the monkey: Advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *Journal of Neurophysiology, 76,* 3666–3681.
Perry, V. H., & Cowey, A. (1984). Retinal ganglion cells that project to the superior colliculus and pretectum in the macaque monkey. *Neuroscience, 12,* 1125–1137.
Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. In H. Bouma & D. G. Bouwhuis (Eds.), *Attention and performance X* (pp. 531–556). Hillsdale, NJ: Erlbaum.
Posner, M. I., Cohen, Y., & Rafal, R. D. (1982). Neural systems control of spatial orienting. *Philosophical Transactions of the Royal Society of London, Series B. Biological Sciences, 298,* 187–198.
Posner, M. I., & Petersen, S. E. (1990). The attentional system of the human brain. *Annual Review of Neuroscience, 13,* 25–42.
Posner, M. I., Rafal, R. D., Choate, L. S., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. *Cognitive Neuropsychology, 2,* 211–228.
Pratt, J., Spalek, T. M., & Bradshaw, F. (1999). The time to detect targets and inhibited and non-inhibited locations: Preliminary evidence for attentional momentum. *Journal of Experimental Psychology: Human Perception and Performance, 25,* 730–746.
Prime, D. J., & Ward, L. M. (2004). Inhibition of return from stimulus to response. *Psychological Science, 15,* 272–276.

Rafal, R., Egly, R., & Rhodes, D. (1994). Effects of inhibition of return on voluntary and visually guided saccades. *Canadian Journal of Experimental Psychology, 48,* 284–230.

Rafal, R. D., Calabresi, P. A., Brennan, C. W., & Sciolto, T. K. (1989). Saccade preparation inhibits reorienting to recently attended locations. *Journal of Experimental Psychology: Human Perception and Performance, 15,* 673–685.

Reuter-Lorenz, P. A., Jha, A. P., & Rosenquist, J. N. (1996). What is inhibited in inhibition of return. *Journal of Experimental Psychology: Human Perception and Performance, 22,* 367–378.

Robinson, D. L., Bowman, E. M., & Kertzman, C. (1995). Covert orienting of attention in macaques: II. Contributions of the parietal cortex. *Journal of Neurophysiology, 74,* 698–712.

Robinson, D. L., & Kertzman, C. (1995). Covert orienting of attention in macaques: III. Contributions of the superior colliculus. *Journal of Neurophysiology, 74,* 713–721.

Rodieck, R. W., & Watanabe, M. (1993). Survey of the morphology of macaque retinal ganglion cells that project to the pretectum, superior colliculus, and parvicellular laminae of the lateral geniculate nucleus. *Journal of Comparative Neurology, 338,* 289–303.

Sapir, A., Soroker, N., Berger, A., & Henik, A. (1999). Inhibition of return in spatial attention: Direct evidence for collicular generation. *Nature Neuroscience, 2,* 1053–1054.

Schall, J. D. (2002). The neural selection and control of saccades by the frontal eye field. *Philosophical Transactions of the Royal Society of London, Series B. Biological Sciences, 357,* 1073–1082.

Schall, J. D. (2004). On building a bridge between brain and behavior. *Annual Review of Psychology, 55,* 23–50.

Schall, J. D., & Hanes, D. P. (1993). Neural basis of saccade target selection in frontal eye field during visual search. *Nature, 366,* 467–469.

Schall, J. D., Hanes, D. P., Thompson, K. G., & King, D. J. (1995). Saccade target selection in frontal eye field of macaque: I. Visual and premovement activation. *Journal of Neuroscience, 15,* 6905–6918.

Selemon, L. D., & Goldman-Rakic, P. S. (1988). Common cortical and subcortical targets of the dorsolateral prefrontal and posterior parietal cortices in the rhesus monkey: Evidence for a distributed neural network subserving spatially guided behavior. *Journal of Neuroscience, 8,* 4049–4068.

Shadlen, M. N., & Newsome, W. T. (1996). Motion perception: Seeing and deciding. *Proceedings of the National Academy of Sciences, 93,* 628–633.

Snowden, R. J. (2002). Visual attention to color: Parvocellular guidance of attentional resources? *Psychological Science, 13,* 180–184.

Snyder, J. J., Kingstone, A., & Schmidt, W. C. (2001). Attentional momentum does not underlie the inhibition of return effect. *Journal of Experimental Psychology: Human Perception and Performance, 27,* 1420–1432.

Stanton, G. B., Goldberg, M. E., & Bruce, C. L. (1988). Frontal eye field efferents in the macaque monkey: II. Topography of terminal fields in midbrain and pons. *Journal of Comparative Neurology, 22,* 493–506.

Steinmetz, M. A., Connor, C. E., Constantinidis, C., & McLaughlin, J. R. (1994). Covert attention suppresses neuronal responses in area 7a of the posterior parietal cortex. *Journal of Neurophysiology, 72,* 1020–1023.

Taylor, T. L., & Klein, R. M. (2000). Visual and motor effects in inhibition of return. *Journal of Experimental Psychology: Human Perception and Performance, 26,* 1639–1656.

Taylor, T. L., & Klein, R. M. (1998). On the causes and effects of inhibition of return. *Psychonomic Bulletin and Review, 5,* 625–643.

Tipper, S. P., Weaver, B., Jerreat, L. M., & Burak, A. L. (1994). Object-based and environmental-based inhibition of return of visual attention. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 478–499.

Uka, T., & DeAngelis, G. C. (2004). Contribution of area MT to stereoscopic depth perception: Choice-related response modulations reflect task strategy. *Neuron, 22,* 297–310.

Wright, R. D., & Ward, L. (1998). The control of visual attention. In R. D. Wright (Ed.), *Visual attention* (pp. 132–186). London: Oxford University Press.

Wurtz, R. H., & Goldberg, M. I. (1971). Superior colliculus cell responses related to eye movements in awake monkey. *Science, 171,* 82–84.

Wurtz, R. H., & Goldberg, M. I. (1972). Activity of superior colliculus in behaving monkey: 3. Cells discharging before eye movements. *Journal of Neurophysiology, 35,* 575–586.

Wurtz, R. H., Richmond, B. J., & Judge, S. J. (1980). Vision during saccadic eye movements: III. Visual interactions in the monkey superior colliculus. *Journal of Neurophysiology, 43,* 1168–1181.