

## The Relationship Between Inhibition of Return and Saccade Trajectory Deviations

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After presentation of a peripheral cue, a subsequent saccade to the cued location is delayed (inhibition of return; IOR). Furthermore, saccades typically deviate away from the cued location. The present study examined the relationship between these inhibitory effects. IOR and saccade trajectory deviations were found after central (endogenous) and peripheral (exogenous) cuing of attention, and both effects were larger with an onset cue than with a color singleton cue. However, a dissociation in time course was found between IOR and saccade trajectory deviations. Saccade trajectory deviations occurred at short delays between the cue and the saccade, but IOR was found at longer delays. A model is proposed in which IOR is caused by inhibition applied to a precue-motor attentional map, whereas saccade trajectory deviations are caused by inhibition applied to the saccade map, in which the final stage of oculomotor programming takes place.

When observers view a visual scene, they typically make rapid eye movements (saccades) to stimuli in the visual scene that are of interest while avoiding saccades to irrelevant stimuli. Inhibitory mechanisms play an important role in the control of saccades. That is, to avoid executing a saccade to an irrelevant stimulus, a saccade to its location must be inhibited.

One well-documented effect in the literature that has been associated with inhibitory control is inhibition of return (IOR; Posner & Cohen, 1984). In a typical IOR study, a cue, such as a task-irrelevant luminance increment, is presented in the periphery and after a varying stimulus onset asynchrony (SOA), a target is presented at the cued or uncued location. IOR is measured as slower response times (manual or oculomotor) when the target is presented at the cued location than when it is presented at an uncued location. When examining IOR it is important to distinguish between its cause and its effect (see Taylor & Klein, 1998, 2000). The cause is associated with the processes occurring on presentation of the cue, whereas its effects are measured by responses to a target, presented at the cued location or at an uncued location. A great deal of research has shown that after IOR has been generated, it affects both manual keypress responses (e.g., Lupiáñez, Milán, Tornay, Madrid, & Tudela, 1997; Posner & Cohen, 1984; Pratt, Kingstone, & Khoe, 1997; Rafal, Calabresi, Brennan, & Sciolto, 1989) and oculomotor responses (e.g., Abrams & Dobkin, 1994; Godijn & Theeuwes, 2002a; Klein & MacInnes, 1999; Rafal, Egly, & Rhodes, 1994).

Although the cause of IOR is still under debate, there is converging evidence from behavioral and neuropsychological studies suggesting that the cause of IOR is related to eye movement programming. Rafal et al. (1989) were the first to provide clear

evidence for the critical role of the oculomotor system in the generation of IOR. In their study IOR was examined under conditions of peripheral (exogenous) and central (endogenous) cuing and with varying instructions associated with the cue. Peripheral cues were luminance increments of one of two peripheral boxes, and central cues were arrows, pointing to one of the two peripheral boxes. In the eyes-fixed condition participants kept their eyes fixated on the central location but shifted attention covertly to the cued location. In the saccade-execution condition participants executed a saccade to the cued location. Last, in the saccade-preparation condition they prepared a saccade to the cued location. On some trials the central location was subsequently cued through a luminance increment. Depending on the condition this brought attention back to the central location (eyes-fixed condition), brought the eyes back to the central location (saccade-execution condition), or canceled the saccade preparation to the cued location. Then, after a varying delay, a target was presented at the location of the first cue or at an uncued location. Participants were required to respond to the target with a manual detection response. The results revealed IOR at the cued location in all conditions in which a peripheral cue was used. That is, response times were longer when the target was presented at the cued location than when it was presented at the uncued location. With central cues, IOR was observed when a saccade had been prepared or executed to the cued location but not when covert attention had been directed to the cued location. Therefore, Rafal et al. concluded that IOR is activated by the oculomotor system.

The view that the cause of IOR lies in the oculomotor system is consistent with neuropsychological studies that have provided evidence that the superior colliculus (SC), a midbrain oculomotor structure, is somehow involved in the generation of IOR. A number of studies have shown that IOR is impaired in patients with progressive supranuclear palsy (PSP), a neurological disorder of midbrain structures including the SC (e.g., Posner, Rafal, Choate, & Vaughan, 1985; Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988; Sapir, Soroker, Berger, & Henik, 1999). Furthermore, single-cell recording studies have shown that neural responses in

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the SC are reduced when a target is presented at a cued location (e.g., Dorris, Klein, Everling, & Munoz, 2002; Dorris, Taylor, Klein, & Munoz, 1998).

Another inhibitory effect related to the oculomotor system is observed when examining saccade trajectories. Specifically, when a peripheral location is cued and participants are required to execute a saccade to a location elsewhere in the visual field, the saccade trajectory deviates away from the cued location (e.g., Doyle & Walker, 2001; Rizzolatti, Riggio, & Sheliga, 1994; Sheliga, Riggio, & Rizzolatti, 1994, 1995; Tipper, Howard, & Houghton, 2000; Tipper, Howard, & Paul, 2001; see also Godijn & Theeuwes, 2002a, 2002b). For example, in Doyle and Walker (2001), participants were required to execute a saccade in response to a central arrow or a peripheral target. On some trials a task-irrelevant peripheral stimulus was presented, which participants were required to ignore. The results revealed that the saccade trajectories deviated away from the location of the peripheral stimulus.

Saccade trajectory deviations do not only occur after the presentation of irrelevant peripheral stimuli but also when covert attention is shifted in response to central cues. In Sheliga et al. (1995, Experiment 3; see also Rizzolatti et al., 1994; Sheliga et al., 1994), a central arrow indicated the location of a peripheral target letter (a *T* or an inverted *T*). Participants had to covertly attend to the peripheral target letter without moving the eyes. If the target letter was a *T*, participants had to make a saccade to a square directly above the central fixation location; if it was an inverted *T*, participants had to make a saccade to a square directly below the

central fixation location. The results revealed that the eyes deviated away from the attended target letter. Sheliga et al. (1995) interpreted these results as evidence for the premotor theory of attention (e.g., Rizzolatti, Riggio, Dascola, & Umiltà, 1987; Rizzolatti et al., 1994), which assumes that shifts of attention are accomplished by saccade programming. According to Sheliga et al., when a saccade is to be executed to a different location than the attended location, the saccade program to the attended location must be inhibited, which is subsequently reflected in the deviation of the saccade away from the cued location.

Consistent with these results, we recently proposed a competitive integration model (Godijn & Theeuwes, 2002a, 2002b; see also Tipper et al., 2000, 2001), in which saccade trajectory deviations are achieved by inhibition applied within the oculomotor system. According to the competitive integration model, saccades are programmed in a common saccade map in which information from different sources (e.g., exogenous and endogenous) is integrated. Similar to previous models (e.g., Findlay & Walker, 1999; Kopecz, 1995; Trappenberg, Dorris, Munoz, & Klein, 2001), the competitive integration model assumes a lateral interaction structure in which activation at a specific location spreads to neighboring locations but inhibits distant locations. In addition to this lateral inhibition, another inhibitory mechanism is assumed that acts directly on the activation of a specific location (e.g., Tipper et al., 2000, 2001). This principle is illustrated in Figure 1, which represents the time course of activation in a task in which a saccade is executed to a specific saccade goal while the abrupt onset of an irrelevant distractor is ignored (Godijn & Theeuwes,

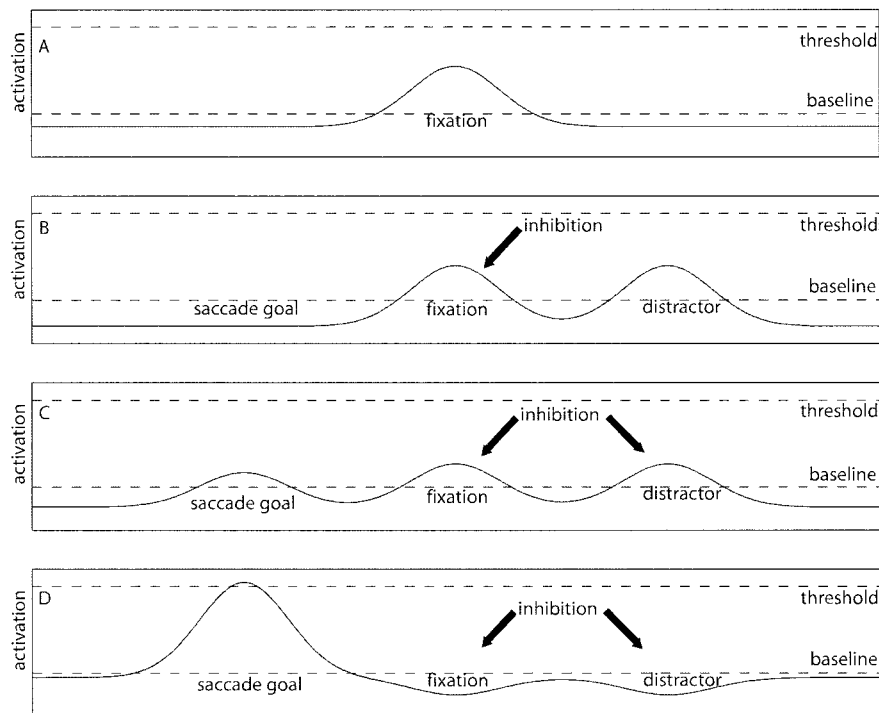


Figure 1. An illustration of the location-specific inhibition according to the competitive integration model (Godijn & Theeuwes, 2002b). In this illustration, a distractor is presented with an abrupt onset, but a saccade is required to a different location. Location-specific inhibition is applied to the fixation location and the distractor location when a saccade is required to a specific location. See the text for details.

2002b; see also Theeuwes, Kramer, Hahn, & Irwin, 1998; Theeuwes, Kramer, Hahn, Irwin, & Zelinsky, 1999). Figure 1A shows that before the decision is made to execute a saccade, there is strong activation at the fixation location, resulting in lateral inhibition at all peripheral locations.<sup>1</sup> Figure 1B demonstrates that if a saccade is required to a specific saccade goal location, location-specific inhibition is applied to the fixation location to release peripheral locations from the lateral inhibition caused by activation at the fixation location. Furthermore, if an onset distractor is presented, saccade-related activation at the distractor location reaches the saccade map. To program a saccade to the saccade goal and to avoid a saccade to the distractor location, location-specific inhibition is also applied to the distractor location; this can be seen in Figure 1C. Last, as shown in Figure 1D, because of the location-specific inhibition at the fixation location and the distractor location, a subbaseline level of activation is reached at these locations (see also Tipper et al., 2000, 2001).

After a threshold is reached at a location represented in the saccade map, a saccade is executed. The eyes do not simply move directly toward the location at which the threshold is reached; instead they move in the direction of the mean vector of activation. Therefore, if inhibition at a certain location causes a subbaseline level of activation at that location, the mean vector of activity is shifted away from the inhibited location, resulting in a saccade trajectory that deviates away from this location.<sup>2</sup>

The idea that saccade trajectory deviations are caused by location-specific inhibition applied to a saccade map is consistent with recent studies in which regions of the SC in monkeys were inactivated through injection with muscimol. In Aizawa and Wurtz (1998; see also Hanes & Wurtz, 2001; Quaia, Aizawa, Optican, & Wurtz, 1998), monkeys were trained to execute saccades to a peripheral target. After local inactivation through muscimol injections, the eyes typically did not reach the target when it was presented in the region of the visual field represented by the inactivated SC region. However, when the target was presented at a location outside this region, the eyes did reach the target, but saccade trajectories deviated away from the inactivated region.

In general, saccade trajectory deviations and IOR are observed in different measures of oculomotor behavior (i.e., trajectories vs. latencies). However, there are a number of striking similarities. First, both effects appear to reflect the inhibition of orienting to cued locations. That is, it is assumed that IOR and saccade trajectory deviations are caused by inhibition applied to a location at which attention had been directed or toward which a saccade had been programmed. Second, the SC is somehow involved in IOR (e.g., Posner et al., 1985; Rafal et al., 1988; Sapir et al., 1999; see also Klein, 2000), and it is involved in saccade trajectory deviations (e.g., Aizawa & Wurtz, 1998; Hanes & Wurtz, 2001; McPeck, Han, & Keller, 2003; Quaia et al., 1998). Because the SC is an area within the oculomotor system, its role in IOR and saccade trajectory deviations is consistent with the presumed role of the oculomotor system in IOR (e.g., Rafal et al., 1989) and saccade trajectory deviations (e.g., Rizzolatti et al., 1994).

Given the similarities between IOR and saccade trajectory deviations, we proposed that both effects may be related to a common inhibitory mechanism (Godijn & Theeuwes, 2002a). According to this working hypothesis, which we called the *oculomotor suppression hypothesis*, IOR and saccade trajectory deviations are the result of inhibition applied to a to-be-ignored location that has

been activated in the saccade map. As a consequence, according to the oculomotor suppression hypothesis, any condition resulting in saccade trajectory deviations should also result in IOR, and vice versa. Alternatively, the relationship between IOR and saccade trajectory deviations may be more complex, and the mechanisms that cause IOR may be different from the mechanisms that cause saccade trajectory deviations.

Despite the similarities between IOR and saccade trajectory deviations, no study has yet examined their relationship. However, Howard, Lupianez, and Tipper (1999) examined IOR and saccade trajectory deviations in a selective reaching task. Participants were required to ignore a red distractor key and to reach for a green target key, which was presented either 200 ms or 600 ms after the distractor key. The results revealed IOR at the distractor locations at both SOAs, but only at the 600-ms SOA were there trajectory deviations of the hand relative to the position of the distractor. However, instead of finding trajectory deviations away from the distractor location, Howard et al. found trajectory deviations toward the location of the distractor. Such deviations toward the distractor have also been found in the oculomotor domain (e.g., Godijn & Theeuwes, 2002a, 2002b; McPeck et al., 2003), and it is typically assumed that they reflect oculomotor activity at the distractor location. Thus, either the oculomotor activity at the distractor location is not inhibited or inhibition is not strong enough to fully suppress the activity at that location (e.g., Howard et al., 1999).

The goal of the present study was to examine the relationship between IOR and saccade trajectory deviations. In Experiment 1, we examined the time course of IOR and saccade trajectory deviations after peripheral (exogenous) cuing. In Experiment 2, we examined whether a saliency manipulation has a similar effect on IOR and saccade trajectory deviations. Finally, in Experiment 3, IOR and saccade trajectory deviations were examined under conditions of peripheral and central cuing.

## Experiment 1

The goal of Experiment 1 was to examine the time course of IOR and saccade trajectory deviations after peripheral cuing. A task-irrelevant onset cue was presented at one of four squares positioned on the corners of an imaginary square centered around a central fixation point. After a variable SOA, a central arrow (saccade cue) was presented, indicating the saccade goal.

<sup>1</sup> In Figure 1, the lateral inhibition is illustrated as a constant inhibition for simplicity's sake. It has been suggested (e.g., Trappenberg et al., 2001) that around the peak of activation, there is strong inhibition that is reduced as a function of distance from the peak (in the shape of a Mexican hat). However, single-cell recording studies have shown that lateral inhibition is widespread through the (SC) saccade map (e.g., Munoz & Istvan, 1998). Further research is required to determine the distribution of lateral inhibition throughout the saccade map in more detail.

<sup>2</sup> Although it is assumed that the eyes start moving in the direction of the mean vector of activity, during the saccade, online corrections of this initial deviation occur such that the eyes curve back toward the target. It is typically assumed that these corrections are achieved through a feedback loop involving a desired eye-displacement signal or a desired eye-position signal (e.g., Moschovakis, 1996; Nichols & Sparks, 1995). However, the present study is only concerned with the deviation of the saccade trajectory, not with the online correction of the deviation.

## Method

**Participants.** Fourteen students were paid for their participation and reported having normal or corrected-to-normal vision. They were not familiar with the purpose of the experiment.

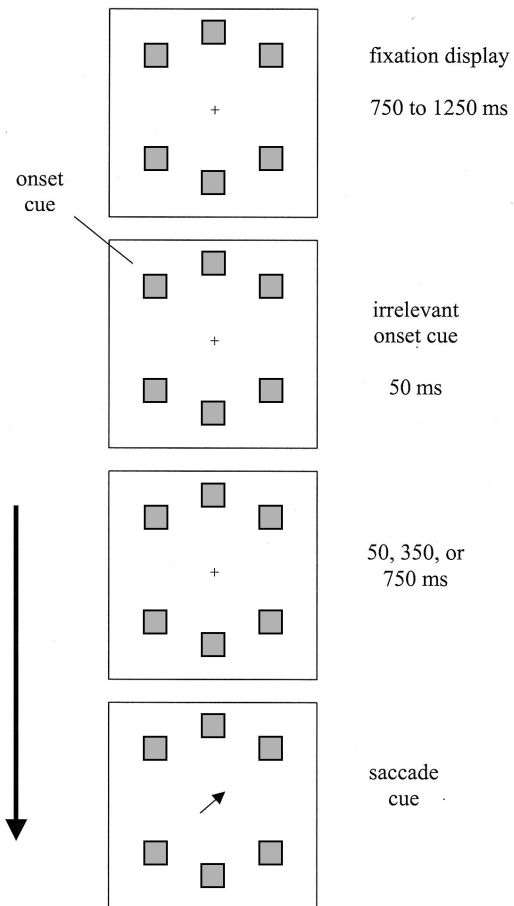
**Apparatus.** A Pentium II computer with a 21-in. (53.34-cm) color monitor controlled the timing of the events and generated stimuli. Eye movements were recorded by means of an EyeLink tracker (SR Research Ltd., Mississauga, Ontario, Canada) with a 250-Hz temporal resolution and a 0.2° spatial resolution. The EyeLink tracker uses an infrared, video-based tracking technology to compute the pupil center of both eyes. An eye movement was considered a saccade when the velocity exceeded 35°/s or the acceleration exceeded 9,500°/s<sup>2</sup>. When participants were fixating the central fixation point at the start of each trial, they pressed a key, which caused a recalibration of the participants' gaze point on the central fixation point. After this, the trial started. Each participant was tested in a dimly lit room. Participants held their head on a chin-rest, located 75 cm away from the monitor.

**Stimuli, procedure, and design.** At the start of each trial, participants viewed displays containing six gray squares (1.0° of visual angle; luminance = 4.6 cd/m<sup>2</sup>) around a central fixation cross at an eccentricity of 8.0°. Two squares were located directly above and below the central fixation cross, and the four other squares were located at the corner positions of an imaginary square around the fixation cross. We refer to these locations as *left top*, *center top*, *right top*, *left bottom*, *center bottom*, and *right bottom*. After 1,250–1,750 ms, an onset cue appeared (51.3 cd/m<sup>2</sup>) at the left-top, right-top, left-bottom, or right-bottom location. After 50 ms, the luminance of the onset cue was reset. Another 50, 350, or 750 ms later, a central arrow appeared, pointing to one of the six squares. Therefore, the SOA between onset cue and central arrow was 100, 400, or 800 ms. See Figure 2 for an illustration of the display sequence. Participants were instructed to ignore the onset cue and to execute a saccade to the square indicated by the central arrow. Saccade latencies were defined as the time from the presentation of the onset cue until the initiation of the saccade. After a practice block of 20 trials, participants performed eight blocks of 144 trials. Conditions were randomized within blocks.

## Results

**Discarded data.** Trials on which saccade latency was shorter than 100 ms (0.2%) or longer than 600 ms (0.1%) were discarded from analyses. An additional 10.8% of the trials were discarded because the eyes were not within 2.0° of the fixation cross when the central arrow was presented or because the saccade target was not fixated within a margin of 4.0°.

**IOR.** To examine IOR, we compared latencies of saccades to the cued location with saccade latencies to uncued locations (the top and bottom locations were excluded because the cue location was always at one of the four corner locations and never the top or bottom location). An analysis of variance (ANOVA) was conducted on saccade latencies with stimulus onset asynchrony (100 ms, 400 ms, 800 ms) and saccade location (cued vs. uncued) as factors. The effect of saccade location did not reach significance,  $F(1, 12) = 3.66, p > .05$ , but there was a Saccade Location  $\times$  SOA interaction,  $F(1, 12) = 8.43, p < .01$ . A main effect of SOA was also found,  $F(1, 12) = 48.35, p < .001$ . Planned comparisons revealed no difference in saccade latency between the cued and uncued location at a 100-ms SOA,  $t(12) < 1$  ( $M_s = 274$  ms at the cued location and 276 ms at the uncued location), but at SOAs of 400 and 800 ms, saccade latencies were longer at the cued location than at the uncued location,  $t(12) = 2.19, p < .05$ , and  $t(12) = 3.98, p < .01$ , respectively, revealing IOR at these longer SOAs.



**Figure 2.** An illustration of the display sequence in Experiment 1. Participants were required to ignore an irrelevant onset cue and to execute a saccade in the direction of a central arrow (saccade cue), presented 100, 400, or 800 ms after the onset cue.

At the 400-ms SOA, mean saccade latency was 258 ms at the cued location and 252 ms at the uncued locations. At the 800-ms SOA, the mean saccade latencies were 256 and 247 ms, respectively. Figure 3 shows saccade latencies for all locations at each SOA. Figure 4 shows the IOR effect as a function of SOA.

**Saccade trajectory deviations.** For all of the samples between initiation and termination of the saccade, the angular deviation was calculated relative to a straight line from the start point of the saccade to the saccade target.<sup>3</sup> The angular deviation was then averaged across the whole saccade. This analysis was conducted separately for the left and right eyes and then averaged across both

<sup>3</sup> There are several alternative ways in which to examine saccade trajectories. The majority of previous studies have examined the deviation of the trajectory relative to a straight line from the start point of the saccade to the endpoint. Note that this examines curvature (the degree to which a saccade is curved) and not deviation from the saccade target. We examined the deviation relative to the saccade target because we are concerned with inhibition, which we assume causes the eyes to deviate away from the saccade target. We calculated the angular deviation (the vector deviation) because it is assumed that saccades are programmed in terms of vectors.

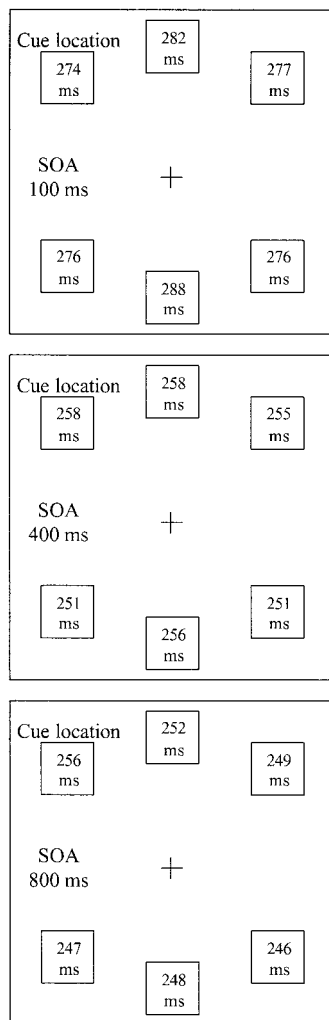


Figure 3. Saccade latencies as a function of saccade goal and stimulus onset asynchrony (SOA) in Experiment 1. All cue locations are reflected to the left-top location (reflections about a vertical and/or horizontal meridian), and saccade latencies are averaged across cue location.

eyes. Positive and negative deviations refer to deviations to the right and left, respectively. To examine the effect of cue location on saccade trajectories, trials with a cue in the left visual field (closest to the saccade goal) were compared with trials with a cue in the right visual field (closest to the saccade goal). For vertical saccade goals, this implied an angular deviation between cue location and saccade goal of  $45^\circ$ . For oblique saccades, similar analyses were conducted. On these trials, the cue locations on either side of the saccade goal were at an angular deviation of  $90^\circ$ . Table 1 shows the mean angular deviation of saccade trajectories as a function of SOA and cue side, and the effect of cue side (cue on the left vs. cue on the right) on saccade trajectories is shown in Figure 4 (averaged across saccade direction).

An ANOVA with saccade direction (vertical or oblique), SOA, and cue side as factors revealed a main effect of cue side,  $F(1, 12) = 89.17$ ,  $p < .001$ , indicating saccade trajectory deviations away from the cued location. There was no main effect of SOA,

$F(2, 24) < 1$ , and the effect of saccade direction failed to reach significance,  $F(1, 12) = 4.32$ ,  $p > .05$ . There was an SOA  $\times$  Cue Side interaction,  $F(1, 12) = 76.44$ ,  $p < .001$ ; saccade trajectory deviations decreased as a function of SOA. There was also a Saccade Direction  $\times$  Cue Side interaction,  $F(1, 12) = 26.93$ ,  $p < .001$ , which indicated larger saccade trajectory deviations away from the cued location for vertical saccades than for oblique saccades. Finally, a three-way Saccade Direction  $\times$  SOA  $\times$  Cue Side interaction was found,  $F(2, 24) = 8.81$ ,  $p < .001$ .

### Discussion

The results of Experiment 1 revealed that peripheral onset cues generated both saccade trajectory deviations and IOR.<sup>4</sup> However, the time course of these effects showed a different pattern. IOR was found at SOAs of 400 and 800 ms but not at an SOA of 100 ms, whereas saccade trajectory deviations were found at the 100-ms SOA but were significantly decreased at the 400- and 800-ms SOAs (see Figure 4). This dissociation in time course is inconsistent with the oculomotor suppression hypothesis, which assumes that IOR and saccade trajectory deviations are caused by inhibition applied to a common saccade map. According to this hypothesis, whenever IOR is found in a specific condition, saccade trajectories should also be found.

Now that the time course of both effects had been established, in Experiments 2 and 3 we further examined the relationship between IOR and saccade trajectories by looking at whether they are both affected in the same way by a saliency manipulation (Experiment 2) and whether they also occur after central cuing of attention in addition to peripheral cuing (Experiment 3).

### Experiment 2

If IOR and saccade trajectory deviations are strongly related, it may be expected that they are both affected by specific manipulations in a similar manner. The goal of Experiment 2 was to examine whether IOR and saccade trajectory deviations are affected by a saliency manipulation (a salient onset cue vs. a less salient color singleton cue) in a similar manner. A second goal of Experiment 2 was to test whether the pattern of results of Experiment 1 could also be observed when participants executed saccades between the presentation of the cue and the presentation of the central arrow.

Participants were required to execute a vertical saccade in response to a peripheral cue, which was either an onset (luminance increment) or a color singleton. A cue was presented at one of four squares positioned on the corners of an imaginary square centered around a central fixation point, similar to the setup in Experiment 1. A cue presented at one of the top squares indicated that an upward saccade was required, and a cue presented at one of the bottom squares indicated that a downward saccade was required. After this initial saccade, participants were required to execute a

<sup>4</sup> Although the IOR effects of 6 and 9 ms are rather small, they are reliable differences. Previous studies examining IOR for saccades in response to central cues after peripheral cuing have also found relatively small IOR effects ( $\sim 10$  ms; e.g., Abrams & Dobkin, 1994; Taylor & Klein, 2000).

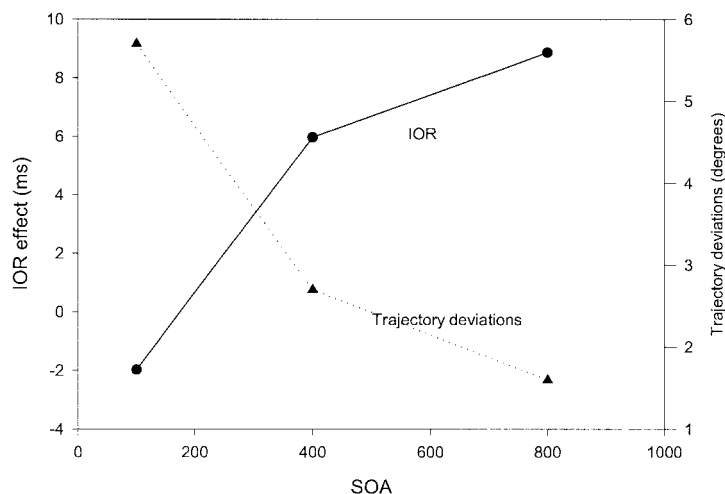


Figure 4. Inhibition of return (IOR) and saccade trajectory deviations as a function of stimulus onset asynchrony (SOA) in Experiment 1. Shown are the IOR effect (mean latency of saccades to the cued location – mean latency of saccades to the uncued corner locations) and the effect of cue side on the saccade trajectory deviations averaged across saccade direction (vertical and oblique saccades).

saccade back to the central fixation point, where an arrow cue would be presented, which indicated the final saccade goal.

Method

Participants. Nine students who were not familiar with the purpose of the experiment participated in Experiment 2.

Stimuli, procedure, and design. At the start of each trial, participants viewed displays containing six gray squares (1.0° of visual angle; luminance of 12.9 cd/m<sup>2</sup>) around a central fixation cross at an eccentricity of 8.0°. The positions of these squares were the same as in Experiment 1. After 750–1,250 ms, a cue appeared at the left-top, right-top, left-bottom, or right-bottom location. On onset cue trials, the gray square at the cue location turned white (51.3 cd/m<sup>2</sup>) and the other squares remained gray. On color cue trials, the square at the cue location turned red (11.5 cd/m<sup>2</sup>) and the other squares turned green (12.4 cd/m<sup>2</sup>). Participants were instructed to execute a saccade to the center-top square if the cue was at the left-top or right-top location and to execute a saccade to the center-bottom location if

the cue was at the left-bottom or right-bottom location. They were instructed to execute a saccade back to the central fixation cross directly after the first saccade. After 800 ms, the color of all circles was reset to gray. Another 800–1,350 ms later, a central arrow appeared, pointing to one of the six squares. Participants were required to execute a final saccade to the square indicated by the central arrow. After a practice block of 60 trials (30 with an onset cue and 30 with a color cue), participants performed eight blocks of 96 trials. Half of the participants started with a block with onset cues, and half started with a block with color cues. At the start of each block, the cue type was altered from onset to color, or vice versa. The location of the cue and the final saccade goal was randomized within blocks.

Results

Discarded data. Trials on which the latency of the final saccade was shorter than 100 ms (0.2%) or longer than 600 ms (0.4%) were discarded from analyses. An additional 21.3% of the trials were discarded because the eyes were not within 2° of the fixation cross when the peripheral cue or the central arrow was presented or because either the first or final saccade target was not fixated within a margin of 4°.

IOR. First, we examined latencies of the final saccade to the location of the onset cue and to the location of the color singleton cue. Note that the initial saccade never went to this location (the initial saccade always went to the center-top or center-bottom location). To examine IOR at the cued location, we compared latencies of the final saccade to the cued location with latencies of the final saccade to the uncued locations, to which the first saccade was never directed (thus, the vertical saccades were excluded from this analysis and were analyzed separately).

An ANOVA was conducted on the latencies of the third saccade, with cue type (onset vs. color singleton) and saccade goal (cued vs. uncued) as factors. There was a main effect of saccade goal,  $F(5, 40) = 14.22, p < .01$ , indicating longer saccade latencies to the cued location (302 ms and 292 ms with onset and color singleton cues, respectively) than to the uncued locations (288 ms

Table 1  
Mean Saccade Trajectory Deviations as a Function of SOA (Between Cue and Saccade Target) and Cue Side in Experiment 1

Saccade type	SOA (ms)	Cue side		Effect size (left–right)
		Left	Right	
Vertical <sup>a</sup>	100	5.94°	–3.51°	9.44°*
	400	3.34°	–0.54°	3.87°*
	800	2.51°	0.26°	2.25°*
Oblique <sup>b</sup>	100	2.55°	–2.00°	4.55°*
	400	0.90°	–0.34°	1.24°
	800	0.92°	–0.01°	0.93°

Note. SOA = stimulus onset asynchrony.  
<sup>a</sup> Cues at 45° angular distance from saccade goal. <sup>b</sup> Cues at 90° angular distance from saccade goal.  
 \*  $p < .05$ .

and 287 ms with onset and color singleton cues, respectively). Furthermore, there was a Saccade Goal  $\times$  Cue Type interaction,  $F(5, 40) = 13.47, p < .01$ . Specifically, the effect of saccade goal was larger with an onset cue than with a color singleton cue, reflecting greater IOR with onset cues (14 ms) than with color singleton cues (5 ms). Planned comparisons revealed a significant IOR effect for onset cues,  $t(8) = 4.12, p < .01$ , as well as for color singleton cues,  $t(8) = 2.40, p < .05$ . Figure 5 shows the latencies of the final saccade separately for the onset cue condition and the color singleton cue condition.

We also analyzed the latencies of the final saccade to the center-top and center-bottom locations. These were the locations to

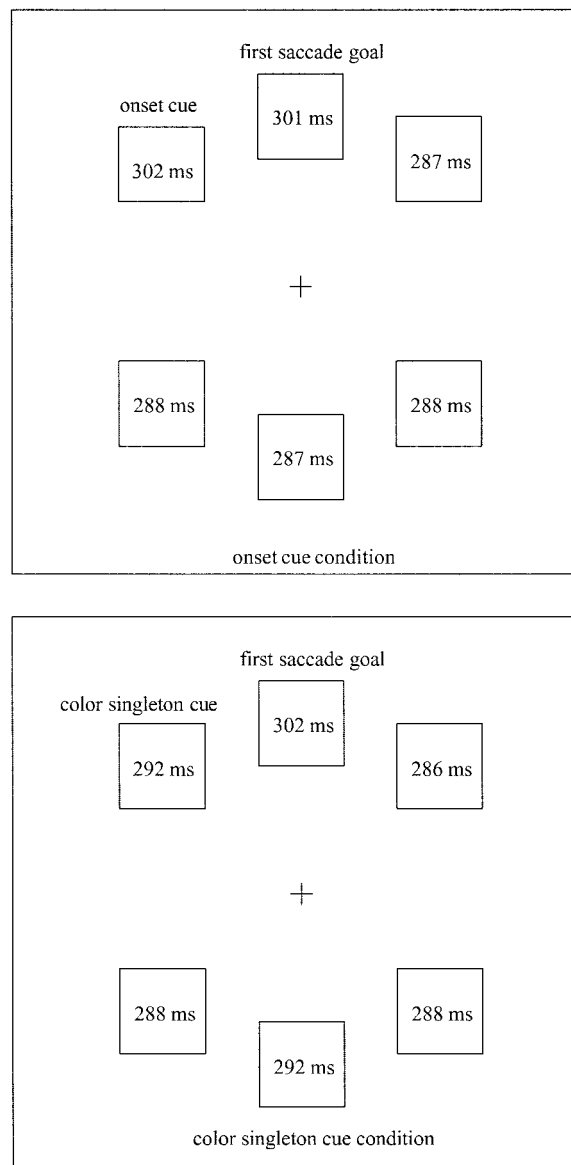


Figure 5. The latency of the final saccade (the third saccade executed in response to the central arrow) as a function of the final saccade goal and cue type in Experiment 2. All cue locations are reflected to the left-top location (reflections about a vertical and/or horizontal meridian), and saccade latencies are averaged across cue location.

which the first saccade was made. A planned comparison revealed IOR at the first saccade goal in the onset cue condition,  $t(8) = 4.38, p < .005$ , and in the color singleton cue condition,  $t(8) = 2.62, p < .03$ , as reflected by longer latencies of the final saccade to the first saccade goal than to the location opposite the first saccade goal.

*Saccade trajectory deviations.* To examine saccade trajectory deviations relative to the cued location across saccades, we examined trajectory deviations relative to the cued location for all vertical saccades. Because the first saccade was always directed to the central location next to the cue location ( $45^\circ$  angular deviation), the trajectory of the final saccade was examined in those conditions in which the final saccade was also directed to the location adjacent to the cued location. The trajectory deviations were calculated in the same way as in Experiment 1 (see Table 2 for the mean saccade trajectory deviations). The saccade trajectories of the first saccade (in response to the peripheral cue) are illustrated in Figure 6.

An ANOVA was conducted on the mean angular deviation of saccade trajectories, with saccade (first, second, or third saccade), cue type (onset or color singleton), and cue side (cue on the left or right relative to the saccade target) as factors. There was a main effect of cue side,  $F(1, 8) = 35.09, p < .001$ , indicating a deviation in saccade trajectories away from the cued location. Furthermore, a Cue Side  $\times$  Cue Type interaction,  $F(1, 8) = 13.10, p < .01$ , indicated larger deviations away from the cued location with onset cues than with color singleton cues. There was also a Cue Side  $\times$  Saccade interaction,  $F(2, 16) = 11.70, p < .01$ ; saccade trajectory deviations were reduced across saccades. A three-way interaction between Saccade  $\times$  Cue Type  $\times$  Cue Side interaction was also found,  $F(2, 16) = 7.19, p < .01$ . Planned comparisons revealed significant trajectory deviations away from the cued location for the first saccade both with onset cues,  $t(8) = 5.34, p < .001$ , and color singleton cues,  $t(8) = 3.77, p < .01$ . For the second saccade, significant trajectory deviations were found with onset cues,  $t(8) = 3.69, p < .01$ , but not with color singleton cues,  $t(8) = 1.01, p > .30$ . For the third saccade, no significant trajectory deviations were found with onset cues,  $t(8) = 2.20, p > .05$ , or with color singleton cues,  $t(8) = 1.71, p > .10$ .

*Relationship between saccade latencies and saccade trajectory deviations.* To examine the relationship between saccade latencies and saccade trajectory deviations at the level of individual trials, we calculated the correlation between saccade latency of the final saccade (which revealed IOR) and saccade trajectory deviations (for the first vertical saccade, which revealed saccade trajectory deviations) for trials in which the first saccade target was next to the cue location ( $45^\circ$  angular distance) and in which the final saccade target was at the cued location. For each participant, the correlation was calculated separately for each of the four cue locations and for both cue types (color singleton and onset). We then performed Wilcoxon signed ranks tests to determine whether the correlation differed significantly from zero. For none of the Cue Location  $\times$  Cue Type combinations did the correlation differ significantly from zero (all  $ps > .10$ ). To increase the power of the test, the correlations were averaged across cue location for both cue types. The correlation did not differ significantly from zero for onset cues (Wilcoxon's  $W = 16, p > .40$ ) or for color singleton cues, although the correlation did reveal a trend toward a signifi-

Table 2  
*Mean Saccade Trajectory Deviations as a Function of Saccade (First, Second, or Final), Cue Side (Left or Right), and Cue Type (Onset or Color Singleton) in Experiment 2*

Saccade and cue type	Cue side		Effect size (left-right)
	Left	Right	
First			
Onset	5.52°	-3.96°	9.48°*
Color	3.36°	-1.70°	5.06°*
Second			
Onset	2.18°	-0.30°	2.48°*
Color	1.04°	0.68°	0.36°
Final			
Onset	1.91°	0.04°	-1.87°
Color	1.50°	0.07°	1.43°

*Note.* Positive and negative values indicate deviations to the right and left, respectively, relative to a straight line from the start of the saccade to the saccade target.

\*  $p < .05$ .

cant correlation between saccade latencies and saccade trajectory deviations for color singleton cues (Wilcoxon's  $W = 8$ ,  $p > .05$ ). With onset cues, the mean correlation was  $-.04$ , and for color singleton cues, the mean correlation was  $.14$  (with positive correlations reflecting greater trajectory deviations away from the cued location as a function of saccade latency).

### Discussion

The results of Experiment 2 showed that both onset and color singleton cues resulted in IOR and saccade trajectory deviations away from the cued location. Moreover, both IOR and saccade trajectory deviations were larger with an onset cue than with a color singleton cue. Thus, both effects are affected by the saliency of the cue in a similar manner, consistent with the view that IOR and saccade trajectory deviations are related. However, similar to Experiment 1, the time course of the two effects was different. Saccade trajectory deviations were rapidly reduced across saccades, and the third saccades did not reveal saccade trajectory deviations relative to the position of the cue. In contrast, IOR was found for the third saccades. Thus, after execution of two saccades (to the top or bottom location and back to the central location), IOR was found for the subsequent saccade, but saccade trajectory deviations were not.

In addition to finding a dissociation in time course between IOR and saccade trajectory deviations, we also found that saccade latencies to the cued location and saccade trajectory deviations were uncorrelated. However, because of the relatively large variance in both saccade latencies and saccade trajectories and the small size of the IOR and trajectory deviation effects, it is hard to interpret this result, particularly because there was a nonsignificant trend toward greater saccade trajectory deviations away from color singleton cues as saccade latency to the cued location increased.

The finding of IOR with color singleton cues may seem inconsistent with a number of previous studies in which color singleton cues did not elicit IOR (e.g., Gibson & Amelio, 2000; Pratt & McAuliffe, 2002; but see Theeuwes & Godijn, 2002). However,

there are at least two major differences between these previous studies and the present experiment. First, in the present experiment, participants executed saccades, whereas in previous studies, participants responded with manual keypresses. Second, in previous studies, the cue was always task irrelevant. In contrast, in the present study, the color singleton cue was task relevant because it indicated the saccade goal. Therefore, participants endogenously allocated attention to the cued location before executing the saccade. These differences between the present experiment and previous experiments may have been responsible for the different pattern of results.

### Experiment 3

The goal of Experiment 3 was to examine IOR and saccade trajectory deviations after peripheral and central cuing of attention. Previous research has shown that saccade trajectories deviate away from peripherally and centrally cued locations (e.g., Rizzolatti et al., 1994; Sheliga et al., 1994, 1995). However, IOR has only been found after peripheral cuing, not after central cuing of attention (e.g., Posner & Cohen, 1984; Rafal et al., 1989). Because the methods of previous IOR studies were very different from studies

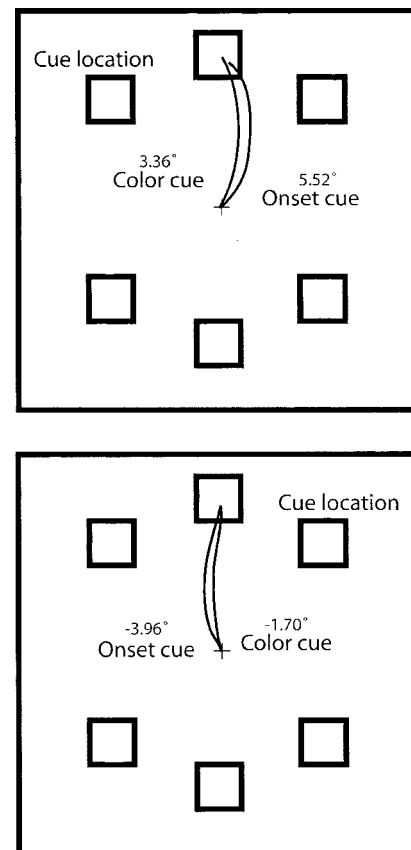


Figure 6. An illustration of the saccade trajectory deviations of the first saccade in Experiment 2. The cue was presented to the left (top panel) or right (bottom panel) of the saccade goal. The saccade trajectory deviations away from the cued location were larger with an onset cue than with a color cue.

in which saccade trajectory deviations were examined, the present experiment examined both effects with the same setup.

Typically, it is assumed that IOR is revealed after attention has been removed from the cued location (e.g., Posner & Cohen, 1984). Therefore, to examine IOR after central cuing of attention, attention must be directed to a location (without moving the eyes) in response to a symbolic cue (e.g., a central arrow), and then a stimulus must be presented, indicating that attention must be removed from the cued location. Thus, in Experiment 3, we examined saccade latencies (IOR) and saccade trajectory deviations after peripheral and central cuing of attention in two conditions. In the first condition, the location to which the eyes were required to move was indicated by the value of a digit presented at the attentionally cued location (peripheral attention condition). In the second condition, attention was removed from the attentionally cued location, back to the central fixation location, where a digit subsequently appeared, indicating the location to which the eyes were required to move (removed attention condition). Because attention had been removed from the attentionally cued location in the removed attention condition, this condition was similar to typical IOR studies.

## Method

*Participants.* Eight students who were not familiar with the purpose of the experiment participated in Experiment 3.

*Stimuli, procedure, and design.* At the start of each trial, participants viewed displays containing four squares ( $2.6^\circ$  of visual angle) with a gray (luminance =  $4.6 \text{ cd/m}^2$ ) outline positioned on an imaginary square around a central fixation cross at an eccentricity of  $8^\circ$ . After 800 ms, a cue was presented for 400 ms (the attention cue), indicating the location at which on half of the trials a digit appeared (the saccade cue digit) that informed the participant of the saccade goal. In the peripheral cue condition, the attention cue was a luminance ( $51.3 \text{ cd/m}^2$ ) increment of the outline of one of the squares. In the central cue condition, the attention cue was a central arrow, pointing to one of the squares. Participants were instructed to attend to the (attentionally) cued location without moving the eyes. Another 100 ms after offset of the attention cue, digits appeared in all four squares. On half of the trials (the peripheral attention trials), the digit in the attentionally cued location was the saccade cue, which was 1, 2, 3, or 4 with equal probability. Digits in the uncued locations were randomly taken without replacement from the same set of digits, with exclusion of the saccade cue.

Participants were required to execute a saccade to the location indicated by the saccade cue. If the saccade cue was a 1, the saccade goal was the left-top location; if it was a 2, the saccade goal was the right-top location; if it was a 3, the saccade goal was the right-bottom location; and if it was a 4, the saccade goal was the left-bottom location. On the other half of the trials (the removed attention trials), the digit in the attentionally cued location was a 0. On these trials, participants were required to remain fixated on the central location. Another 600 ms after presentation of the 0 digit, each digit changed into an 8, and at the same time a white ( $51.3 \text{ cd/m}^2$ ) square was presented around the central fixation location. Then, 400 ms later, the saccade cue appeared in the central square, indicating the saccade goal (1, 2, 3, or 4), and participants were required to execute a saccade depending on the identity of the saccade cue. Eye movements were monitored to ensure compliance with the instruction to keep the eyes at the center prior to the presentation of the saccade cue. Half of the participants first performed a block of 384 trials with peripheral cues, followed by a block of 384 trials with central cues. For the other half of the participants, this order was reversed. There were 120 practice trials before the first block and 40 practice trials before the second block. Conditions were randomized within blocks. See Figure 7 for examples of the display sequence.

## Results

*Discarded data.* Trials on which saccade latency was shorter than 120 ms (2.1%) or longer than 900 ms (3.3%) were discarded from analyses. An additional 18.3% of the trials were discarded because the eyes were not within  $2^\circ$  of the fixation cross when the saccade cue was presented or because the saccade target was not fixated within a margin of  $4^\circ$ .

*IOR.* Figure 8 shows the saccade latencies as a function of saccade goal and cue type, both for the peripheral attention condition (Figure 8A) and the removed attention condition (Figure 8B). An ANOVA was conducted on saccade latency, with trial type (peripheral attention or removed attention), saccade goal (cued or uncued location), and cue type (peripheral or central) as factors. A main effect of saccade goal was found,  $F(1, 7) = 22.79$ ,  $p < .001$ . As can be seen in Figure 8, saccade latencies were longer when the cued location was the saccade goal ( $M = 537 \text{ ms}$ ) than when one of the uncued locations was the saccade goal ( $M = 510 \text{ ms}$ ). There was also a main effect of trial type,  $F(1, 7) = 45.45$ ,  $p < .001$ . Saccade latencies were shorter in the removed attention condition ( $M = 450 \text{ ms}$ ) than in the peripheral attention condition ( $M = 596 \text{ ms}$ ). There was no main effect of cue type,  $F(1, 7) < 1$ . The Cue Type  $\times$  Saccade Goal interaction failed to reach significance,  $F(1, 7) = 3.09$ ,  $p > .10$ , but there was a three-way Cue Type  $\times$  Saccade Goal  $\times$  Trial Type interaction,  $F(1, 7) = 7.39$ ,  $p < .03$ . This three-way interaction indicates that the IOR effect (cued vs. uncued) was larger with central cues in the peripheral attention condition but larger with peripheral cues in the removed attention condition. To further examine this three-way effect, separate ANOVAs were conducted for both trial types. The difference in IOR between peripheral and central cues did not reach significance in the peripheral attention condition,  $F(1, 7) = 3.72$ ,  $p > .05$ , or in the removed attention condition,  $F(1, 7) = 1.41$ ,  $p > .25$ .

*Saccade trajectory deviations.* Table 3 shows the mean angular deviation of saccade trajectories as a function of trial type (peripheral attention or removed attention), cue type (peripheral or central), and cue side (left or right). An ANOVA revealed a main effect of cue side,  $F(1, 7) = 20.35$ ,  $p < .01$ , indicating saccade trajectory deviations away from the cued location. There was no main effect of cue type,  $F(1, 7) < 1$ , but there was an effect of trial type,  $F(1, 7) = 22.51$ ,  $p < .01$ . There was also a Trial Type  $\times$  Cue Side interaction,  $F(1, 7) = 26.83$ ,  $p < .001$ , which indicated that the effect of cue side was larger on peripheral attention trials than on removed attention trials. There were no further two-way interactions ( $F_s < 1$ ), but a three-way Trial Type  $\times$  Cue Type  $\times$  Cue Side interaction was found,  $F(1, 7) = 7.20$ ,  $p < .04$ . Separate ANOVAs were conducted for peripheral attention trials and removed attention trials to further examine the effects of cue side and cue type in these conditions. On peripheral attention trials, an effect of cue side was found,  $F(1, 7) = 35.37$ ,  $p < .001$ , indicating saccade trajectory deviations away from the cued location. There was no effect of cue type,  $F(1, 7) < 1$ , and the interaction between Cue Type  $\times$  Cue Side interaction failed to reach significance,  $F(1, 7) = 3.30$ ,  $p > .10$ . However, there was a trend toward larger saccade trajectory deviations away from the cued location with central cues than with peripheral cues. In the removed attention condition, there was no effect of cue side,  $F(1, 7) < 1$ , or cue type,  $F(1, 7) < 1$ , and there was no Cue Side  $\times$  Cue Type interaction,  $F(1, 7) = 1.35$ ,  $p > .30$ . Thus, in the removed attention condition,

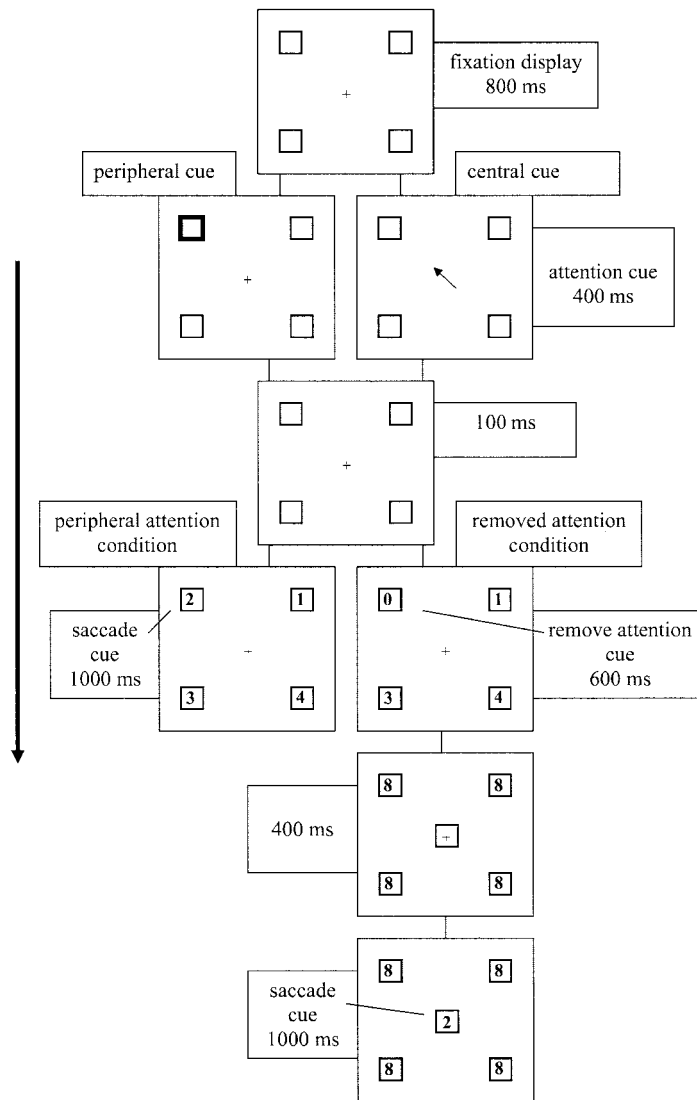


Figure 7. Examples of the display sequence of Experiment 3.

there were no trajectory deviations relative to the attentionally cued location.

*Discussion*

The results of Experiment 3 showed that IOR and saccade trajectory deviations occurred after peripheral and central cues. Thus, similar to Experiments 1 and 2, IOR and saccade trajectory deviations occurred in the same conditions. However, as in the previous experiments, IOR was present at long cue–target delays (removed attention condition), but saccade trajectory deviations were present only at relatively short cue–target delays (peripheral attention condition). Specifically, when the target digit was presented after attention had been removed back to fixation, IOR was found but no saccade trajectory deviations were found. IOR was not only found on removed attention trials but also on peripheral attention trials. This suggests that IOR can occur even when

attention has not been removed from the cued location. This finding is discussed further in the General Discussion.

The finding of IOR with central cues seems inconsistent with previous studies in which no IOR was found after central cuing of attention (e.g., Posner & Cohen, 1984; Rafal et al., 1989). However, there are many differences between the present experiment and previous studies examining IOR after central cuing, and these may have been responsible for the different results. For example, in the present experiment, not only was attention directed at the cued location, there was also processing of information at the cued location. In contrast, in previous studies examining IOR after central cuing (Posner & Cohen, 1984; Rafal et al., 1989), when attention was removed from the cued location, no stimulus had been presented there. A second major difference between the present experiment and previous studies is that participants in the present experiment were set to execute saccades. That is, although

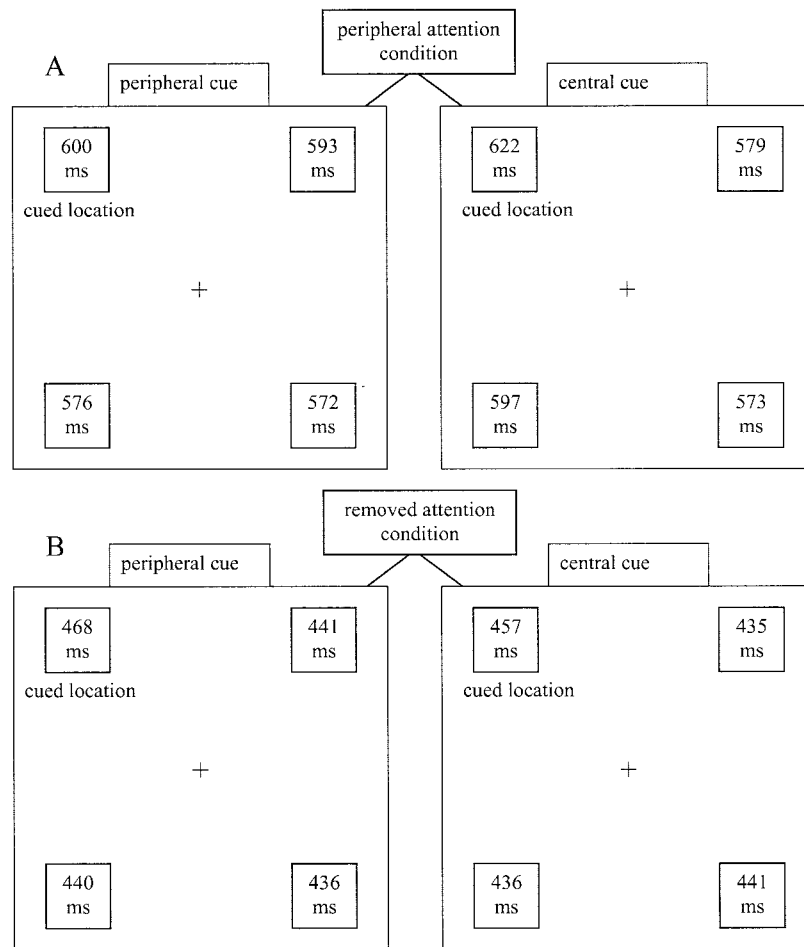


Figure 8. Mean saccade latencies of Experiment 3 as a function of cue type (peripheral or central cue) and saccade goal. All cued locations are reflected to the left-top location (reflections about a vertical and/or horizontal meridian), and saccade latencies are averaged across cue location. A: Mean saccade latencies in the peripheral attention condition. B: Mean saccade latencies in the removed attention condition.

the cue was uninformative with regard to the saccade goal, participants were required to execute a saccade on each trial. Because IOR may be related to oculomotor programming, the degree to which participants are set to execute saccades may be an important factor in the generation of IOR.

Table 3  
Mean Saccade Trajectory Deviations as a Function of Cue Type (Peripheral or Central) and Cue Side for the Peripheral Attention and Removed Attention Conditions in Experiment 3

Condition	Cue type	Cue side		Effect size (left-right)
		Left	Right	
Peripheral attention	Peripheral	2.99°	-2.40°	5.39°*
	Central	3.46°	-3.19°	6.65°*
Removed attention	Peripheral	-0.14°	-0.96°	0.82°
	Central	-0.87°	-0.81°	-0.06°

\*  $p < .05$ .

## General Discussion

The present study examined the relationship between IOR and saccade trajectory deviations. The results indicated that IOR and saccade trajectory deviations occurred in the same conditions. Furthermore, both IOR and saccade trajectory deviations were larger with an onset cue than with a color singleton cue, suggesting that the effects are stronger when the saliency of the cue is increased. However, a clear dissociation was found in the time course of the effects. That is, saccade trajectory deviations were largest when the saccade was executed shortly after the presentation of the cue, whereas IOR was found at much longer delays between cue and saccade.

### Relationship Between IOR and Saccade Trajectory Deviations

According to the oculomotor suppression hypothesis, which we previously suggested (Godijn & Theeuwes, 2002a), IOR and saccade trajectory deviations are caused by location-specific inhibi-

tion applied to a spatial map in which saccade programming occurs (the saccade map). This hypothesis is consistent with the finding of the present study that IOR and saccade trajectory deviations occurred in the same conditions and were affected in a similar way by a saliency manipulation. However, the oculomotor suppression hypothesis is inconsistent with the dissociation in time course between IOR and saccade trajectory deviations. That is, if both effects are caused by inhibition within the saccade map, it may be assumed that the time course of both effects should be the same. For example, in the present study, when the delay between the cue and the saccade was relatively long, there was IOR but no saccade trajectory deviations. Thus, the inhibition reflected in the saccade latencies (IOR) was not reflected in the saccade trajectories. Before rejecting the hypothesis that IOR and saccade trajectory deviations are caused by inhibition applied to a single saccade map, below we consider ways in which this hypothesis might be able to account for the time-course dissociation.

One reason for caution in interpreting the dissociation in time course between IOR and saccade trajectory deviations is that it may be assumed that saccade trajectory deviations depend on the distribution of activity throughout the saccade map, whereas IOR (saccade latencies to cued vs. uncued locations) depends on activation levels at specific locations (i.e., specific cued and uncued locations) within the saccade map. For example, if the inhibition around the cued location is initially quite broad and becomes more focused around the cued location as a function of time, saccade trajectory deviations might be reduced as a function of time (because the mean vector of activity might be affected more by broad inhibition than by localized inhibition), whereas IOR is not reduced (because the strength of the inhibition at the cued location need not be reduced as broad inhibition changes into localized inhibition at the cued location). Although this idea can account for the time-course dissociation of the present study, it is quite speculative, and it is unclear why the inhibition should initially be broad and then later more localized around the cued location. Furthermore, a recent single-cell recording study by Dorris et al. (2002) has provided evidence that IOR is not the result of inhibition applied to the saccade map (in the SC) but is likely caused by inhibition preceding the saccade map. If it is assumed that saccade trajectory deviations are caused by inhibition in the (SC) saccade map, this suggests that IOR and saccade trajectory deviations are not caused by inhibition applied to the same system. This idea is further elaborated in the following sections.

A more plausible explanation for the dissociation in time course between IOR and saccade trajectory deviations is that they are caused by inhibition applied to separate systems. It is likely that these systems are strongly related, because IOR and saccade trajectory deviations are also strongly related. That is, they occur in the same conditions and they are affected in the same way by a saliency manipulation. We propose that saccade trajectory deviations are caused by inhibition applied to the saccade map, in which the final stage of oculomotor programming takes place, whereas IOR is caused by inhibition applied to a preoculomotor system that provides input to the saccade map. In the next sections, the evidence for this proposal is discussed and a framework is presented for understanding the relationship between IOR and saccade trajectory deviations.

### *Saccade Trajectory Deviations: Inhibition in the Saccade Map*

According to the competitive integration model (e.g., Godijn & Theeuwes, 2002a, 2002b), when a saccade is triggered, the eyes start moving in the direction of the mean vector of activity within the saccade map. When a specific location is inhibited, this results in a subbaseline level of activation at this location (see Figure 1). This subbaseline level of activity is reflected in a saccade trajectory deviation away from the inhibited location. Evidence for this idea has been provided by Aizawa and Wurtz (1998; see also Quail et al., 1998), who found similar saccade trajectory deviations after local inactivation of a region of the SC. Furthermore, McPeck et al. (2003) showed that activity of SC neurons that were coding locations other than the saccade goal was reflected in the saccade trajectory deviation. Specifically, after stimulation of neurons (below threshold for eliciting a saccade) coding a nonsaccade goal, the trajectory deviated toward the location represented by the stimulated region. Although these saccade trajectory deviations were opposite those reported in the present study, the results of McPeck et al. show that neural activity of locations other than the saccade goal are reflected in trajectory deviations. Moreover, in a recent study in which participants performed a visual search task (Theeuwes & Godijn, in press), we found both types of saccade trajectory deviations. Fast saccades to the target deviated toward the distractor, whereas slow saccades deviated away from the distractor. This finding reflects the time course of activation at the distractor location. First the distractor elicits activity in the SC saccade map (causing fast saccades to curve toward the distractor), which is subsequently inhibited, resulting in a deviation of the saccade trajectory away from the distractor location.

In the present study, we only found saccade trajectory deviations away from cued locations, which indicates that at the time of saccade execution, the activation at the cued location had already been inhibited. Saccade trajectory deviations occurred away from centrally and peripherally cued locations and away from both onset cues and color singleton cues. This suggests that these cues elicited activity in the saccade map, which was subsequently inhibited. According to Tipper et al. (2000, 2001), the strength of inhibition depends on the level of activation of the to-be-inhibited location. Following this logic, in Experiment 2, the salient onset cue elicited more activation than the less salient color singleton cue, resulting in stronger inhibition and therefore larger trajectory deviations with an onset cue than with a color singleton cue.

### *IOR: Inhibition in a Preoculomotor Attentional Map*

If saccade trajectory deviations are caused by inhibition applied to the SC saccade map, the inhibition resulting in IOR is likely caused by inhibition within a different system. That is, because saccade trajectory deviations are rapidly reduced as a function of time, the inhibition in the saccade map is presumably quickly diminished. Therefore, this cannot be the cause of the longer lasting IOR effect. One piece of evidence suggesting that the inhibition resulting in IOR occurs within a system preceding the saccade programming within the SC saccade map was provided by Dorris et al. (2002). In this study, neural activity of SC cells was recorded while monkeys performed an IOR task that required a saccade to a peripheral target. During the interval between cue and

target, neural activity at the cued location was higher than at the uncued location. Thus, there was no evidence for active inhibition of neural activity during the cue–target interval within the SC. However, when the target appeared, the target-related burst of activity of SC cells was greater when the target was presented at an uncued location than at a cued location. Therefore, the inhibition resulting in IOR was not caused by active inhibition of SC cells but by reduced input into the SC.

In accordance with the results of Dorris et al. (2002), we propose that IOR is caused by inhibition within a system that provides input to the saccade map. It is likely that this preculomotor system is related to attentional processing. There is substantial evidence that attention shifts to the saccade goal prior to saccade execution (e.g., Deubel & Schneider, 1996; Godijn & Pratt, 2002; Hoffman & Subramaniam, 1995; Irwin & Gordon, 1998; Kowler, Anderson, Doshier, & Blaser, 1995). According to one view of the relationship between attention and saccades, attentional shifts are responsible for selecting the saccade goal (e.g., Deubel & Schneider, 1996; Godijn & Theeuwes, 2003; Kowler et al., 1995). An even stronger relationship was proposed by Rizzolatti and colleagues (e.g., Rizzolatti et al., 1987, 1994). According to their premotor theory, attention shifts are accomplished by programming a movement. Rizzolatti et al. (1994) argued that in the brain, there is no single superordinate attentional system that is independent of motor programming. That is, the activation of neural structures, related to attention, appears to be dependent on the required motor action. For example, the posterior parietal cortex, which is assumed to be related to attentional selection (e.g., Chelazzi & Corbetta, 2000; Colby & Goldberg, 1999; LaBerge, 1995), consists of several areas with neural properties related to a specific motor domain. In another example, the lateral intraparietal area (LIP) is involved in selection for saccades, whereas the anterior intraparietal area is involved for selection for grasping (e.g., Murata, Gallese, Luppino, Kaseda, & Sakata, 2000).

The idea that IOR occurs within a system related to attention that provides input to the SC saccade map is consistent with previous studies on IOR. Although the oculomotor system appears to play an important role in IOR (e.g., Rafal et al., 1989; Taylor & Klein, 1998, 2000), there is also a great deal of evidence that attention is inhibited from returning to previously cued locations (e.g., Handy, Jha, & Mangun, 1999; Lupiáñez et al., 1997; McDonald, Ward, & Kiehl, 1999; Pratt et al., 1997). If it is assumed that attention and motor programming are achieved within a network, it may also be assumed that inhibition applied to a preculomotor attentional map is passed on throughout the network, subsequently affecting other motor responses in addition to saccades. This idea is consistent with Briand, Larrison, and Sereno (2000), who showed that IOR occurs at shorter cue–target delays for saccades than for manual responses.

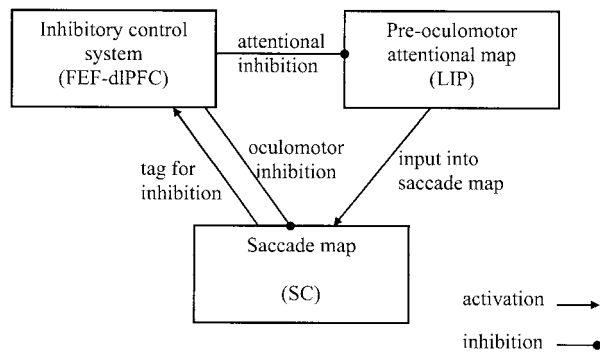
### *A Framework for Understanding Inhibition of Saccades*

In the previous sections, we argued that saccade trajectory deviations and IOR are caused by inhibition applied to different systems—respectively, the saccade map, in which the final stage of saccade programming takes place, and a preculomotor attentional map. In this section, we address the issue of the relationship between inhibition in these two systems.

Although IOR and saccade trajectory deviations may be caused by inhibition within separate systems, the results of the present study suggest that both effects occur under the same conditions. Both effects were found with onset cues and color singleton cues and after both peripheral and central cuing of attention. Both IOR and saccade trajectory deviations were larger with an onset cue than with a color singleton cue. Furthermore, previous research has shown that the SC saccade map is involved in both saccade trajectory deviations and IOR. In fact, a necessary condition for IOR and saccade trajectory deviations may be the activation of a specific location in the saccade map. First, it is assumed (e.g., Godijn & Theeuwes, 2002a, 2002b; see also Tipper et al., 2000, 2001) that saccade trajectory deviations occur when a location is activated in the saccade map at the moment that a saccade is required to a different location. To overcome the interference by the nonsaccade goal, this location is inhibited in the saccade map, causing saccade trajectory deviations away from the inhibited location. Second, previous research on IOR has shown that an intact SC is required for IOR to occur (e.g., Posner et al., 1985; Rafal et al., 1988; Sapir et al., 1999). Furthermore, previous research has suggested that saccade programming (but not necessarily saccade execution) is critical for IOR to occur (e.g., Rafal et al., 1989). Therefore, Danziger, Fenrich, and Rafal (1997; see also Klein, 2000) suggested that the SC is responsible for generating a tag, which is required for the inhibitory signal. According to Danziger et al., this tag is transmitted to cortical areas, where the inhibitory signal is subsequently applied.

When these behavioral and neural findings of IOR and saccade trajectory deviations are integrated, a network of three separate systems appears necessary to accommodate both inhibitory effects. The first system is the (SC) saccade map, in which the final stage of saccade programming takes place. This saccade map is also responsible for activating the tag required for inhibition to occur. The second system is an inhibitory control system, which receives the inhibitory tag from the SC and applies the inhibition. Although the brain areas that apply the inhibitory signals have not yet been determined, we speculate that this inhibitory control system may include either or both the dorsolateral prefrontal cortex (dlPFC) and the frontal eye fields (FEFs). It is typically assumed that these areas are involved in inhibition of saccades. Evidence supporting this idea comes from lesion studies that have shown that lesions in the FEFs (e.g., Guitton, Buchtel, & Douglas, 1985; Rafal, Machado, Ro, & Ingle, 2000) or the dlPFC (e.g., Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991; Walker, Husain, Hodgson, Harrison, & Kennard, 1998) result in disinhibition of saccades. For simplicity's sake, we assume a single inhibitory control system, although it is possible that a network exists of separate but related inhibitory control systems, one for IOR and one for saccade trajectory deviations. The third system is a preculomotor attentional map (presumably LIP), which provides input to the saccade map. This framework is illustrated in Figure 9.

When an irrelevant stimulus is presented in the periphery, it typically captures attention in an exogenous way (see, e.g., Theeuwes, 1991). This so-called “capture of attention” implies that there is exogenous activation within the preculomotor attentional map (i.e., LIP). This in turn generates oculomotor activation within the saccade map (i.e., SC) corresponding to the location in space where the stimulus was presented. However, as long as no eye movement has to be made and the observer remains fixated, this



*Figure 9.* A framework for understanding the relationship between inhibition of return (IOR) and saccade trajectory deviations. The framework consists of a network of three subsystems that are involved in inhibitory control of saccades: a preoculomotor attentional map, the saccade map, and an inhibitory control system. When a cue is presented in a typical cue-target saccade task, activation flows through the preoculomotor attentional map to the saccade map. This saccade map is responsible for the tag that is required for inhibitory control. On the basis of the activation within the saccade map, the tag is passed on to the inhibitory control system. After receiving the tag from the saccade map, the inhibitory control system inhibits activation at the cued location in the preoculomotor attentional map. When a saccade is required to a location other than the cued (tagged) location, the inhibitory control system inhibits activation at the cued location in the saccade map. If a saccade is required to the cued location, the inhibition in the preoculomotor attentional map at the cued location results in a reduced input at that location into the saccade map, delaying the execution of the saccade (IOR). We assume that the eyes move in the direction of the mean vector of activity in the saccade map. Therefore, if a saccade is required to a different location than the cued location, the inhibition applied to the cued location results in a subbaseline level of activity, causing the eyes to deviate away from the cued location. Although this framework is primarily a functional framework, we speculate that the inhibitory control system is represented by the frontal eye fields (FEFs) and/or the dorsolateral prefrontal cortex (dlPFC), that the preoculomotor attentional map is represented by the lateral intraparietal area (LIP), and that the saccade map is represented by the superior colliculus (SC). Note that this is a simplified framework in which the focus is on understanding IOR and saccade trajectory deviations. Therefore, other subsystems that may be involved in eye-movement control have been omitted.

oculomotor activation within the saccade map has no consequences other than the generation of an inhibitory tag corresponding to the location of activation within the saccade map. This inhibitory tag is delivered to the inhibitory control system. By inhibiting the preoculomotor attentional map, the inhibitory control system inhibits activation at the location at which the initial stimulus was presented. This mechanism of inhibitory control is what is typically referred to as IOR: After attention is reflexively shifted to the location of the initially presented stimulus, there is delayed responding to stimuli subsequently displayed at that location. This interpretation fits with the presumed role of IOR as an inhibitory mechanism that would encourage the sampling of new information in the visual scene (e.g., Posner & Cohen, 1984). Note that as long as no saccade has to be made, the activation generated by the peripheral stimulus in the saccade map is not inhibited by the inhibitory control system. This is consistent with the results of Dorris et al. (2002), who found no evidence for inhibition in the

SC saccade map during the interval between the presentation of a peripheral stimulus and the saccade target.

The situation becomes somewhat different when observers have to make an eye movement to a different location than the location at which the (irrelevant) stimulus was presented. As noted, the peripheral stimulus generates activation in the saccade map. When observers are required to make a saccade to a different location, the activation at the location of the irrelevant stimulus is suppressed in the saccade map, so a saccade can be programmed to the goal location without interference (i.e., lateral inhibition) from the activation at the location of the irrelevant stimulus. Therefore, when a saccade is required to a particular location in space, the inhibitory control system inhibits the irrelevant activation in the saccade map (see Figure 1). This location-specific inhibition resolves the conflict when two distant locations are strongly activated and biases saccade programming toward the desired location. Because the eyes move in the direction of the mean vector of activity, the inhibition in the saccade map results in trajectory deviations. Whether the saccade trajectory deviates toward or away from the initial stimulus location depends on the degree of inhibition that has been applied at the moment the saccade is executed. When the activation at the location of the stimulus has not yet been completely inhibited (above baseline level of activation), the eyes deviate toward the location of the stimulus. In contrast, when the inhibition is complete and the irrelevant stimulus location becomes subbaseline, the saccade trajectory deviates away from the location of the stimulus.

Within the proposed framework, the time-course dissociation between saccade trajectory deviations (which are rapidly reduced) and IOR (with is longer lasting) may be explained. First, we further assume that the strength of the inhibitory tag is reduced as a function of time. This has important consequences for the time course of saccade trajectory deviations. Because inhibition is only applied to the saccade map when a saccade is required to a location different from the initial stimulus location, the strength of the inhibition applied to the saccade map is reduced as the delay between the irrelevant stimulus and saccade execution is increased. Therefore, the trajectory deviations are also reduced as the delay between the irrelevant stimulus and saccade execution is increased. In contrast, the size of the IOR effect is not rapidly reduced as a function of time, because the inhibition applied to the preoculomotor attentional map (which we assume causes IOR) is applied shortly after the onset of the irrelevant stimulus. That is, inhibition is applied to the preoculomotor attentional map at a time when the inhibitory tag is still strong.

Second, the results of the present study suggest that the inhibition within the oculomotor system decays more rapidly than the inhibition within the preoculomotor attentional map. For example, in Experiment 2, three vertical saccades were executed, and saccade trajectory deviations away from the cued location were reduced across saccades and were even absent for the third saccade, for which IOR still occurred. This suggests that after inhibition is applied to the oculomotor system and the preoculomotor system, the inhibition rapidly decays in the oculomotor system but is maintained in the preoculomotor system. The idea that inhibition is maintained in the preoculomotor attentional system is consistent with the finding that IOR occurs for multiple sequentially cued locations (e.g., Snyder & Kingstone, 2001; Tipper, Weaver, &

Watson, 1996; but see Abrams & Pratt, 1996; Pratt & Abrams, 1995).

Not only is the time at which inhibition is applied to the precue motor attentional map and the saccade map different, the reason for the inhibition also differs. Inhibition to the saccade map occurs to overcome the interference from irrelevant locations on the saccade to a goal location. Inhibition applied to the precue motor attentional map is not necessarily related to the execution of saccades. That is, even when a saccade is not (yet) required, inhibition is applied to the tagged location in the precue motor attentional map. According to the traditional view of IOR, the inhibition is applied to prevent attention from returning to previously attended locations (e.g., Posner & Cohen, 1984). However, in Experiment 3, we found that IOR even occurred in a condition in which attention was directed at the cued location to determine the saccade goal. Finding IOR in this condition may be the consequence of participants attending to the cued location covertly, without expecting to respond to that location. That is, the digit presented at the cued location must be identified, but the probability of executing a saccade to the cued location is as high as the probability of executing a saccade to any of the uncued locations. Thus, attending covertly to the cue, without the intention to respond to that location (or to prepare a response to that location), may involve inhibition of the cued location. Because attention and saccades are so strongly related in people's everyday lives (people usually look at the focus of their attention), inhibition applied to the precue motor attentional map may be needed to process a specific stimulus with covert attention without preparing a saccade in its direction. According to this logic, inhibition is applied to the precue motor attentional map when a specific stimulus that is attended or has been attended is deemed irrelevant for the oculomotor system. Although these ideas seem consistent with the findings of the present study and with previous studies on IOR, they are rather speculative, and further research is required to acquire a full understanding of the cause and function of IOR.

In the present study, IOR and saccade trajectory deviations occurred after both peripheral and central cuing of attention. According to our proposed framework, this indicates that both peripheral and central cuing of attention cause activation within the SC saccade map. Although there is substantial evidence that peripheral cues do indeed activate the saccade map (e.g., Dorris et al., 2002; Everling, Dorris, Klein, & Munoz, 1999; Trappenberg et al., 2001), there is less consensus on whether central cuing of attention activates the saccade map (e.g., Klein, 1980; Klein & Pontefract, 1994; Rizzolatti et al., 1987, 1994). However, in the present study, the saccade map may have been activated not by the endogenous allocation of attention in response to the central cue but by the presentation of a task-relevant digit at the attended location. Therefore, further research is required to determine whether the endogenous allocation of attention, without the presentation of a task-relevant stimulus at the attended location, would lead to results similar to those in Experiment 3 of the present study.

The framework presented is intended to improve understanding of inhibition of saccades. It is clearly a simplified model, because the oculomotor network consists of many subsystems, each with their own specific role. However, our framework focuses on those aspects that are deemed crucial for the understanding of inhibition of saccades. It predicts that any condition that results in IOR also

results in saccade trajectory deviations but with a different time course.

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