MECHANISMS OF INHIBITION OF RETURN:

BRAIN, BEHAVIOR, AND COMPUTATIONAL MODELING

by

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This thesis is dedicated to my incredible life partner Joanna Michelle Nimeck Satel, whose love and support made this work possible.

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ABSTRACT

Inhibition of return (IOR) is a cognitive phenomenon whereby reaction times (RTs) are slower to cued relative to uncued targets at cue-target onset asynchronies (CTOAs) greater than approximately 300 ms. One important theory of IOR proposes that there are two mutually exclusive forms of IOR, with an attentional/perceptual form arising when the oculomotor system is actively suppressed, and a motoric form arising when it is engaged (Taylor & Klein, 2000). Other theories propose that IOR is the result of multiple, additive neural mechanisms (Abrams & Dobkin, 1994). Here, we have performed computational simulations and empirical investigations in an attempt to reconcile these two competing theories. Using a dynamic neural field (DNF) model of the intermediate layers of the superior colliculus (iSC), we have modeled both a sensory adaptation mechanism of IOR, and a motoric mechanism resulting from the aftereffects of saccadic eye movements. Simulating these mechanisms, we replicated behavior and neurophysiology in a number of variations on the traditional cue-target paradigm (Posner, 1980). Predictions driven by these simulations have led to the proposal of many behavioral and neuroimaging experiments which further examine the plausibility of a 2-mechanisms theory of IOR. Contrary to our original predictions, we demonstrated that saccades are biased away from cued targets in a paired target saccade averaging paradigm, even at short CTOAs. In paradigms thought to recruit both sensory and motoric mechanisms, we robustly demonstrated that there are at least two independent, additive mechanisms of IOR when tasks require saccadic responses to targets. When similar paradigms were tested with manual responses to targets, additivity effects did not hold, implying that the motoric mechanism of IOR does not transfer from the oculomotor to skeletomotor systems. Furthermore, across numerous experiments using event-related potential (ERP) techniques, we have demonstrated that P1 component reductions are neither necessary, nor sufficient, for the behavioral exhibition of IOR. We propose that a comprehensive framework for behavioral IOR must include (at least) four independent neural mechanisms, differentially active depending on circumstances, including sensory adaptation, saccadic aftereffects, local inhibition, and cortical habituation.

LIST OF ABBREVIATIONS USED

ACS	Attentional control setting
ANOVA	Analysis of variance
CTOA	Cue-target onset asynchrony
DNF	Dynamic neural field
EEG	Electroencephalography
EM	Eye movement
ERP	Event-related potential
FE	Foreperiod effect
FEF	Frontal eye fields
FIX	Fixation neurons
fMRI	Functional magnetic resonance imaging
IOR	Inhibition of return
ISI	Inter-stimulus interval
ISR	Inhibition of saccade return
M task	Motor task
PPC	Posterior parietal cortex
RT	Response (reaction) time
S1	First stimulus (cue)
S2	Second stimulus (target)
S task	Sensory task
SAC	Saccade neurons
SC	Superior colliculus
SCi (iSC)	Intermediate layers of the superior colliculus
SCs (sSC)	Superficial layers of the superior colliculus
SM task	Sensori-motor task
SOA	Stimulus onset asynchrony
SRT	Saccadic response (reaction) time
STD	Short-term depression

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I would like to express thanks to my committee and collaborators for all their help and advice, particularly Raymond M. Klein, Zhiguo Wang, and Matthew D. Hilchey.

CHAPTER 1 INTRODUCTION

This thesis comprises seven published manuscripts on which I am a co-author (Chapters 2-7). I contributed significantly to all aspects of these research projects, including experimental/simulation design, implementation, testing, data collection, data analysis, visualization, interpretation, manuscript writing, editing, and revising.

Cognitive science has always entailed the use of abstract models to provide theoretical frameworks that help guide our understanding of cognitive processes. The human brain is an extremely complex organ comprised of many interacting dynamical systems. To understand how these complex, dynamical systems work, research is required at a number of levels of analysis. At the lowest levels, it is important to understand how the biochemistry, molecular biology, and genetics of the brain put together such a complex organ. This work is often performed in laboratories that are isolated from higher levels of analysis. At the cellular and physiological levels, research is normally conducted on animals, raising questions about the applicability of this work to the understanding of human brains. At the highest levels of analysis, researchers are interested in how brain systems produce behavioral effects, but often without regard for the underlying neural dynamics. Given the complexity of the human brain, and the traditional isolation of researchers working at different levels of analysis, it is critical to tie together these different lines of research into cohesive theories explaining neurocognitive phenomena. Such theorizing allows for the synthesis of diverse, interdisciplinary, empirical results, as well as the generation of predictions that can be empirically tested.

Traditionally, cognitive scientists have relied on simple 'black box' verbal models to guide theory. Such models can provide theoretical frameworks that attempt to integrate and summarize the literature on a topic, often including abstract neuroanatomical details as well as making general predictions about behavior. However, because of their imprecision, it is difficult to make progress with verbal theories of cognitive processes. What is needed are mathematically/computationally explicit theories that can be explicitly

tested. There are many mathematically precise modeling approaches, but for those interested in how the organ of mind – the brain – implements cognitive processes, we prefer a model (see, for example, Trappenberg, Dorris, Munoz, & Klein, 2001) rooted in nervous system organization and functionality. We want to not only be able to predict behavior, but also to understand the underlying spatiotemporal neurodynamics that lead to behavior.

In the current work, we follow this general approach by using a mathematically explicit model of a neural structure (the intermediate layer of the superior colliculus; iSC) that is intimately involved in orienting behavior (particularly eye movement initiation) to simulate and make predictions about a cognitive phenomenon called inhibition of return (IOR). IOR is a robust behavioral phenomenon whereby reaction times (RTs) are impaired when targets requiring manual or saccadic responses are preceded by spatially overlapping cues and the time interval between stimulus onset (cue-target onset asynchrony; CTOA) exceeds approximately 300 ms (for reviews see Klein, 2000; 2004b). When the time interval between cue and target appearance is short, RTs are facilitated due to the remnants of cue-elicited activity and/or the lingering of attention at the cued location. After a few hundred milliseconds, these facilitatory processes have decayed and RTs are impeded in response to targets at locations that have previously been attended (see Figure 1.1 for a graphical illustration of the behavioral consequences of this phenomenon in a traditional Posner cue-target task; Posner, 1980). In the time since the term was coined (Posner, Rafal, Choate, & Vaughan, 1985), it has been determined that IOR is an important component of human orienting responses with a proposed role in

novelty seeking (Posner & Cohen, 1984) and the facilitation of foraging behavior (Klein, 1988). The past 30 years have seen a great deal of research devoted to the investigation of the temporal, spatial, and behavioral dynamics of IOR, as well as to applied and clinical research drawing on these results.

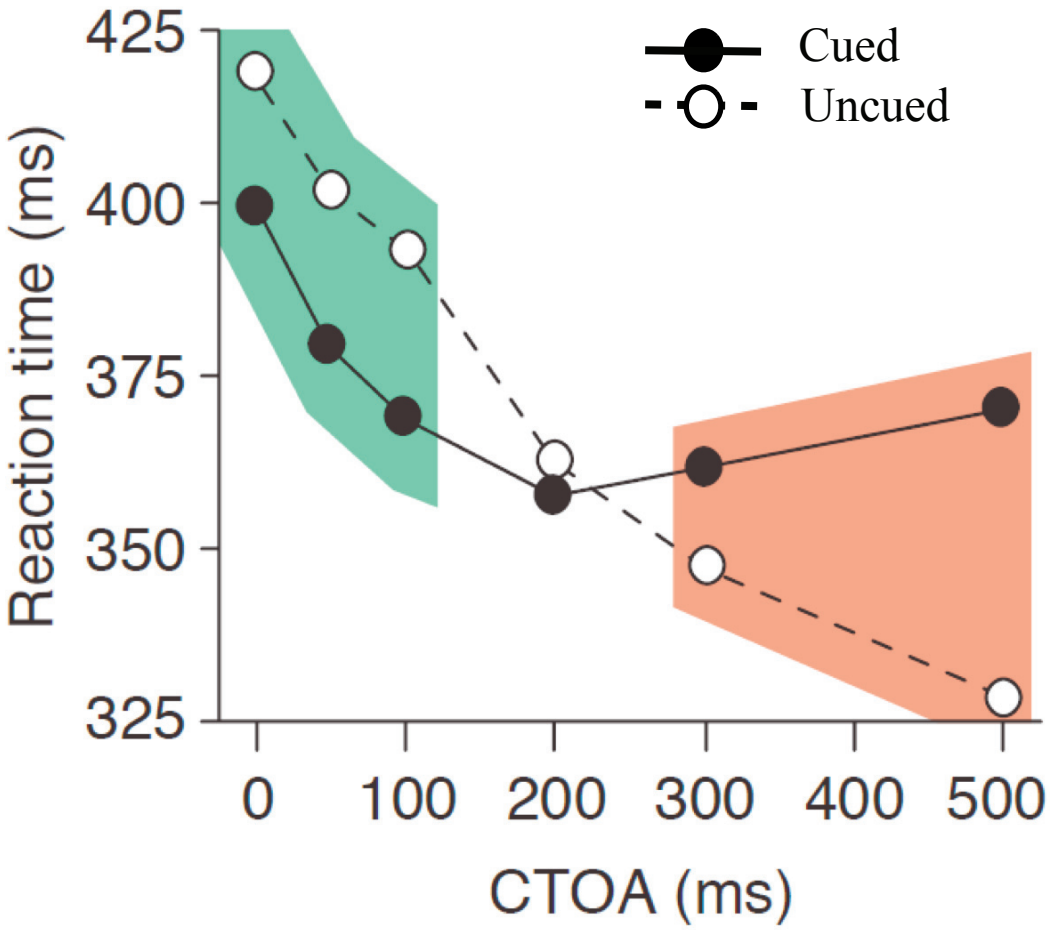

Figure 1.1: Human behavioral data illustrating behavioral facilitation and IOR effects on RT as a function of CTOA in a cue-target paradigm (from Klein, 2000). Cued trials are represented with black dots and a solid line, while uncued trials use white dots and a dashed line.

The majority of work investigating IOR has been conducted using the traditional Posner cueing paradigm, wherein subjects maintain central fixation throughout trials consisting of uninformative peripheral cues and make manual responses to subsequent

peripheral targets at either the same location as the cue or the opposite mirror location (see Figure 1.2 for illustration). IOR scores are calculated by subtracting mean RTs to previously cued targets (often referred to as the cued, same, or valid condition) from RTs in response to targets appearing at the mirror location of the cue (uncued, opposite, or invalid condition). A number of variations on this design have been developed to further investigate the boundary conditions under which IOR is generated (and can be measured), including the utilization of central endogenous stimuli and the incorporation of saccadic as well as manual responses to stimuli (e.g., Taylor & Klein, 2000).

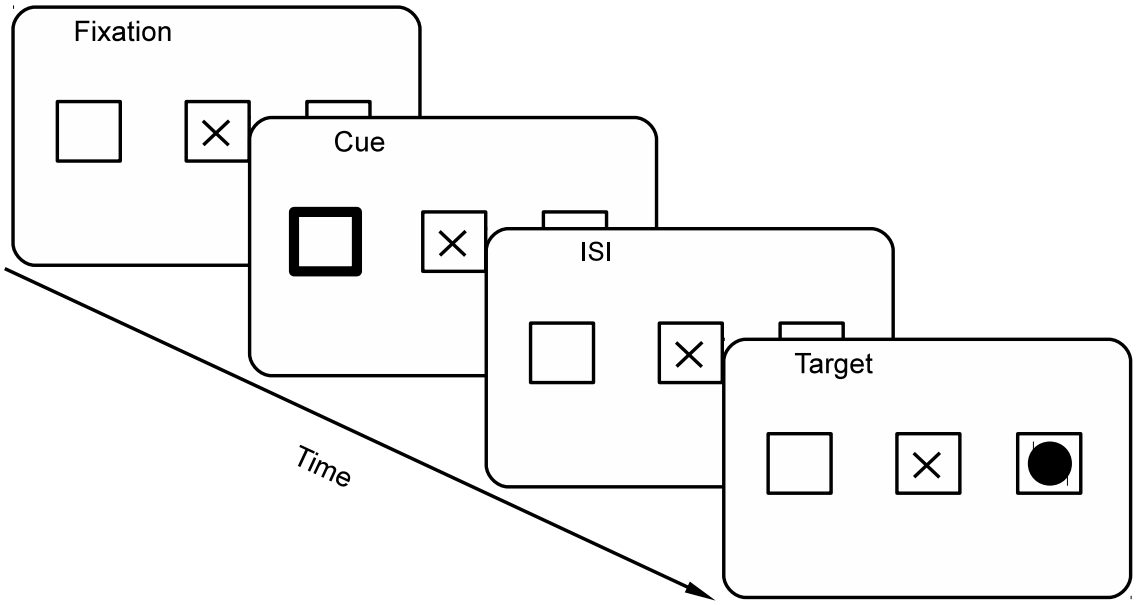

Figure 1.2: Posner cueing task design. After a pre-trial fixation period, an uninformative, irrelevant, peripheral cue is presented, then a target requiring a response is presented after an appropriate inter-stimulus interval (ISI).

More complex tasks, such as visual search, also require an inhibitory mechanism like IOR to discourage the re-inspection of highly salient non-target items (for a review see Wang & Klein, 2010). In the domains of computational vision and robotics, inhibitory

signals are often applied to previously inspected locations in order to discourage continual re-fixation of highly salient stimuli (e.g., Itti & Koch, 2001; Koch & Ullman, 1985).

The purpose of this dissertation is to present the early foundations of a comprehensive, neuroscientifically based, mathematically explicit, framework for IOR that can be used to stimulate further basic, applied, and clinical investigations of IOR and orienting behavior. Using computational modeling techniques, simulations are presented - founded on empirical results in the literature - which implement low level sensory and motor mechanisms of IOR that are generated when the oculomotor system is actively engaged (Chapters 2 & 3). Predictions generated from these simulations are then tested by running carefully designed human behavioral studies (Chapters 4, 5, & 6), providing further data that can be incorporated into IOR theory and modeling efforts driving further empirical research. To investigate the neural signature of IOR, related questions about IOR are also investigated using electroencephalographic (EEG) recordings and the analysis of target-elicited event-related potentials (ERPs) in various conditions (Chapters 7 & 8).

1.1 Behavioral investigations of IOR

Orienting refers to the preferential processing of neural inputs related to specific locations in the environment (Posner, 1980). Orienting can occur overtly, as when eye movements are directed toward objects that are observed visually, or covertly, as when attention is directed toward an object or location without fixating the target of attention. When orienting responses are driven by external sensations, such as visual or auditory events, it is referred to as exogenous attention or control. Orienting can also occur without

exogenous control, as in the directing of attention in response to internal commands from higher cognitive areas of the brain (i.e., localization responses to central arrows). These internal mechanisms are referred to as endogenous attention or control. Research has clearly demonstrated that exogenous and endogenous control mechanisms interact in a variety of ways to drive both overt and covert orienting.

Several lines of evidence have suggested that the oculomotor system, particularly the SC, is intimately involved with the generation and processing of IOR (e.g., Posner et al., 1985; Rafal, Calabresi, Brennan, & Sciolto, 1989; Sapir, Soroker, Berger, & Henik, 1999; Dorris, Taylor, Klein, & Munoz, 2002). Rafal et al. (1989) used a modified cueing task in which subjects either maintained fixation, prepared an eye movement, or saccaded to central arrow (endogenous) or peripheral (exogenous) cues. On trials with a subsequent central cue-back stimulus (returning the eyes to fixation or canceling prepared saccades), responses to probes at cued locations were slower than to uncued probes, demonstrating IOR behaviorally in all conditions *except* when the oculomotor system was inhibited and central arrow cues were used. Since peripheral onset cues are thought to activate the oculomotor system automatically, this was taken as evidence that the oculomotor system is directly involved in the dynamics of IOR. Observation of brain damaged patients also suggested that subcortical, but not cortical, systems are involved in the manifestation of IOR (e.g., Posner et al., 1985, Simion, Valenza, Umiltà, & Barba, 1995; Valenza, Simion, & Umiltà, 1994; Sapir et al., 1999; Sereno, Briand, Amador, & Szapiel, 2006). Sapir et al. (1999) examined IOR in a patient with a unilateral lesion of the right SC (which mediates leftward eye-movements), finding that IOR was absent in the temporal visual field of the

left eye and the nasal visual field of the right eye, but was still observed in the opposite direction (see also Sereno et al., 2006). Early developmental studies (Valenza et al., 1994; Simion et al., 1995) also demonstrated that oculomotor IOR is present in infants, whose subcortical (including the SC), but not cortical, neural machinery is developed. Based on these findings, several researchers have postulated that IOR is caused by activation of the neural machinery responsible for eye movements, particularly the SC.

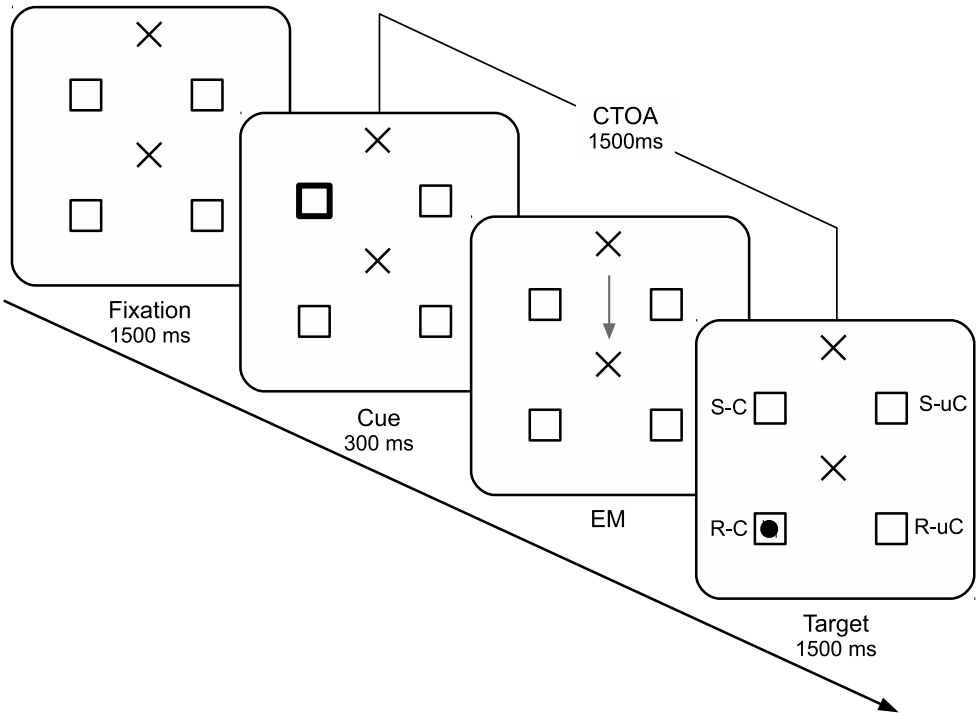

Figure 1.3: Task procedure. Participants fixate the upper fixation X until cue offset, then make an eye movement (EM; indicated by arrow) to the central fixation cross. In this illustration, the cue appears at the upper left box and the target appears in the lower left box. Consequently, the four possible target locations are labeled S-C (spatiotopic-cued), S-uC (spatiotopic-uncued), R-C (retinotopic-cued) and R-uC (retinotopic-uncued), respectively (from Satel, Wang, Hilchey, & Klein, 2012).

In terms of function, Posner and Cohen (1984) proposed that IOR is meant to encourage orienting toward novel items in the environment. Klein (1988) extended this

functional explanation of IOR to include a facilitatory role in visual search linked to IOR's tendency to reduce the likelihood of returning attention to previously inspected locations (for a review of related evidence, see Wang & Klein, 2010). In order for this role to be fulfilled, IOR must be represented in an environmentally-based, or spatiotopic, coordinate system. Since many areas of visual processing contain maps in retinotopic coordinates (i.e., always centered on the currently foveated location), it is important to investigate IOR when retinotopic and spatiotopic representations are dissociated by introducing an eye movement between cue and target appearance (see Figure 1.3 for an illustration of this task). Such studies have demonstrated that IOR is indeed represented in spatiotopic coordinates, with strong spatiotopic IOR exhibited and little or no retinotopic IOR observed when these coordinate systems are dissociated (e.g., Maylor & Hockey, 1985).

1.2 Neurophysiological investigations of IOR

Although there has been extensive discussion in the IOR literature regarding the stages of processing and neural generators of the phenomenon, there is general agreement that at least a subset of IOR-like phenomena are mediated at the level of the SC (Posner et al., 1985; Rafal et al., 1989; Sapir et al., 1999; Dorris et al., 2002; Fecteau & Munoz, 2005). The SC plays a crucial role in the initiation/generation of eye movements, receiving inputs from early sensory areas representing external stimuli as well as converging endogenous inputs from many other areas in the brain (see Figure 1.4 for a schematic illustration of the brain areas involved in oculomotor behavior). There is substantial evidence that IOR can have an effect on early input-based processing (e.g.,

Posner & Cohen, 1984; Reuter-Lorenz, Jha, & Rosenquist, 1996; Prime & Ward, 2006). Neurophysiological work has furthered this line of research by examining neural activity in the SC while monkeys perform behavioral tasks that generate IOR (for a review see Fecteau & Munoz, 2006; see Figure 1.5 for an illustrative summary of the monkey IOR data).

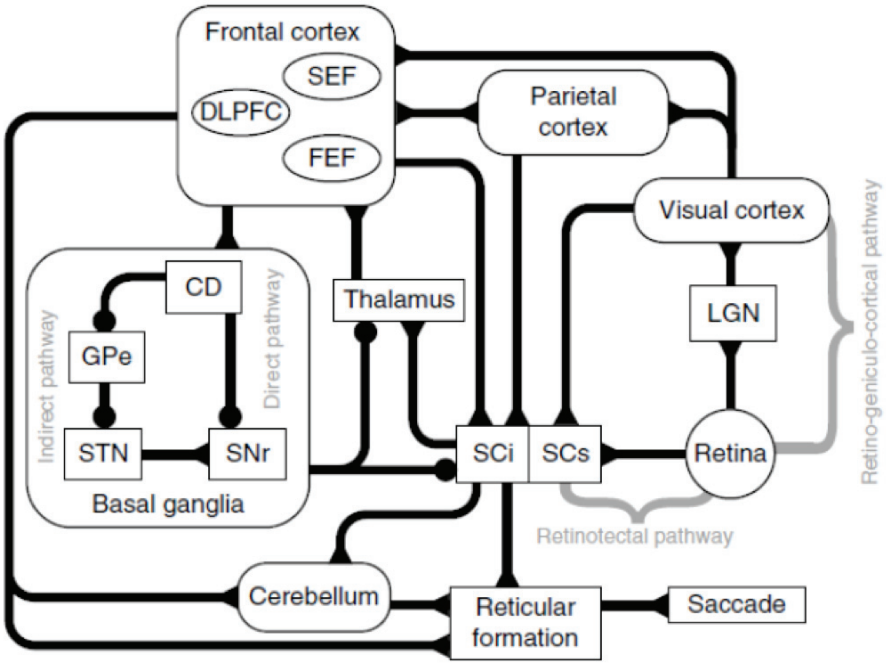

Figure 1.4: Schematic illustration of the brain areas involved in oculomotor behavior (from Munoz, Armstrong, & Coe, 2008). The intermediate layer of the superior colliculus (iSC) integrates many converging inputs from early sensory and higher cortical areas of the brain and is responsible for generating signals that initiate eye movements.

Superficial layers of the SC (sSC) receive input from early sensory areas, including the retina and primary visual cortex, as well as extrastriate areas (e.g., Lui, Gregory, Blanks, & Giolli, 1995; Rodieck & Watanabe, 1993). The superficial layers do not receive any feedback from the areas they project to, so they represent only early

sensory information that has not been contaminated by further processing in other regions (Clower, West, Lynch, & Strick, 2001). In contrast, the intermediate layers of the SC (iSC) receive, and integrate, input from a number of cortical regions that are involved in eye movements and covert attention, including prefrontal, parietal, and temporal areas (e.g., Sparks & Hartwich-Young, 1989; Lui et al., 1995; Clower et al., 2001). Contrasting neural activity in the different layers of the SC during performance of an IOR experiment can provide evidence about which stages of processing and areas of the brain are involved in the generation and manifestation of IOR (Fecteau & Munoz, 2005).

In the iSC, target-elicited neural activity is reduced for targets presented at previously cued, as compared to uncued, locations (Dorris et al., 2002; Fecteau & Munoz, 2005). This reduction in activity was significantly correlated with saccadic reaction times (SRTs) to cued targets, at least at CTOAs under 600 ms or so where IOR was observed behaviorally, further cementing the relationship of SC activity to behaviorally exhibited IOR (Fecteau & Munoz, 2005). It was also found that the residual neural activity following a cue is not directly suppressed in the iSC (Dorris et al., 2002). In fact, when electrical stimulation was delivered through the recording electrode to elicit a saccade, the latency of these electrically evoked saccades was faster for previously cued regions at a CTOA of 200 ms (Dorris et al., 2002). These findings suggested that IOR is not caused by active inhibition of recently stimulated iSC neurons, but rather by a reduction in the strength of subsequent input signals to these neurons, at least in monkeys, up to 200 ms after cue onset (Dorris et al., 2002; Fecteau & Munoz, 2005).

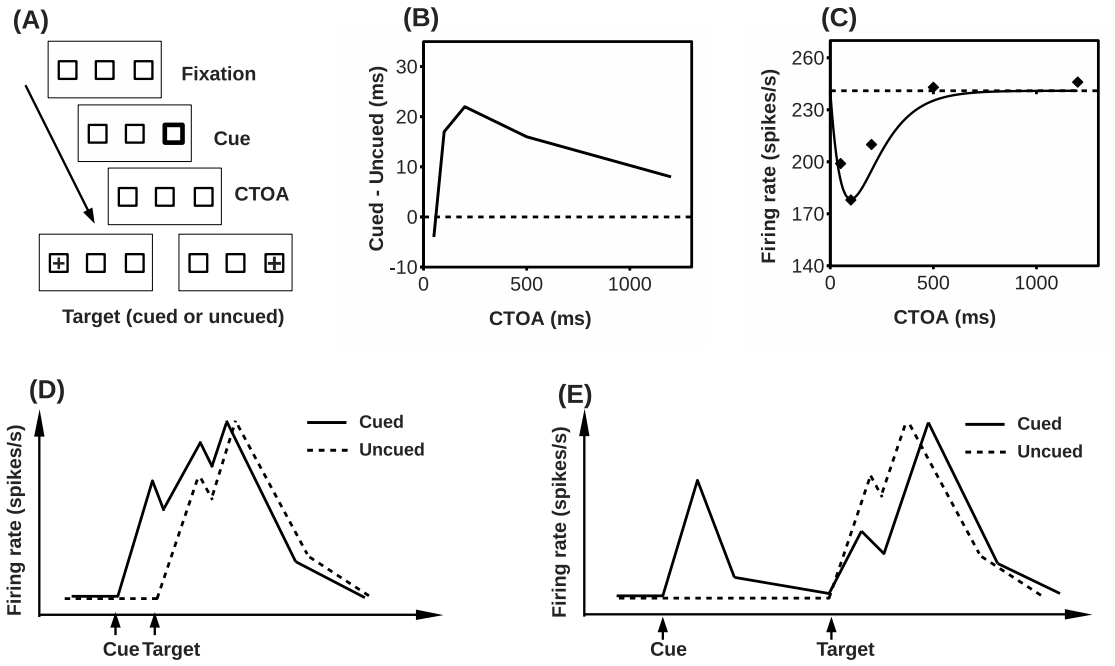

Figure 1.5: (A) The classic cue-target paradigm. (B) Typical behavioral findings in the cue-target paradigm (monkey behavioral data adapted from Fecteau & Munoz, 2005). Faster responses to cued targets are observed at short CTOAs (facilitation) and slower responses to cued targets are observed at long CTOAs (IOR). (C) Diamonds denote the response of visual neurons (in the sSC) to cued targets (adapted from Fecteau & Munoz, 2005). Dashed line denotes the average firing rate of visual neurons to uncued targets. An alpha function with parameters, A = -63, t_{max} = 100 ms, was used to fit these cell recordings. (D) and (E) Schematic cell activity during cued (solid) and uncued (dashed line) trials in the SCi. The last peak in both figures denotes a hypothetical endogenous "move" signal that projects to the SC once a visual target is detected.

Fecteau and Munoz (2005) further observed that the sSC also shows reduced responses to cued, as compared to uncued, targets when behavioral IOR is exhibited. This finding indicates that the reduction in visual input strength associated with cued targets is likely coming from very early stages of visual processing, since information represented in the superficial layers is purely sensory. Fecteau and Munoz (2005) postulated that IOR simply "reflects a habituated sensory response occurring in early sensory areas that is subsequently transmitted through the rest of the brain" (p. 1722). Dukewich (2009) has

furthered this line of thought by proposing a theory wherein IOR is simply the result of habituation-like processes in all areas of the brain, occurring anytime a pathway is repeatedly stimulated.

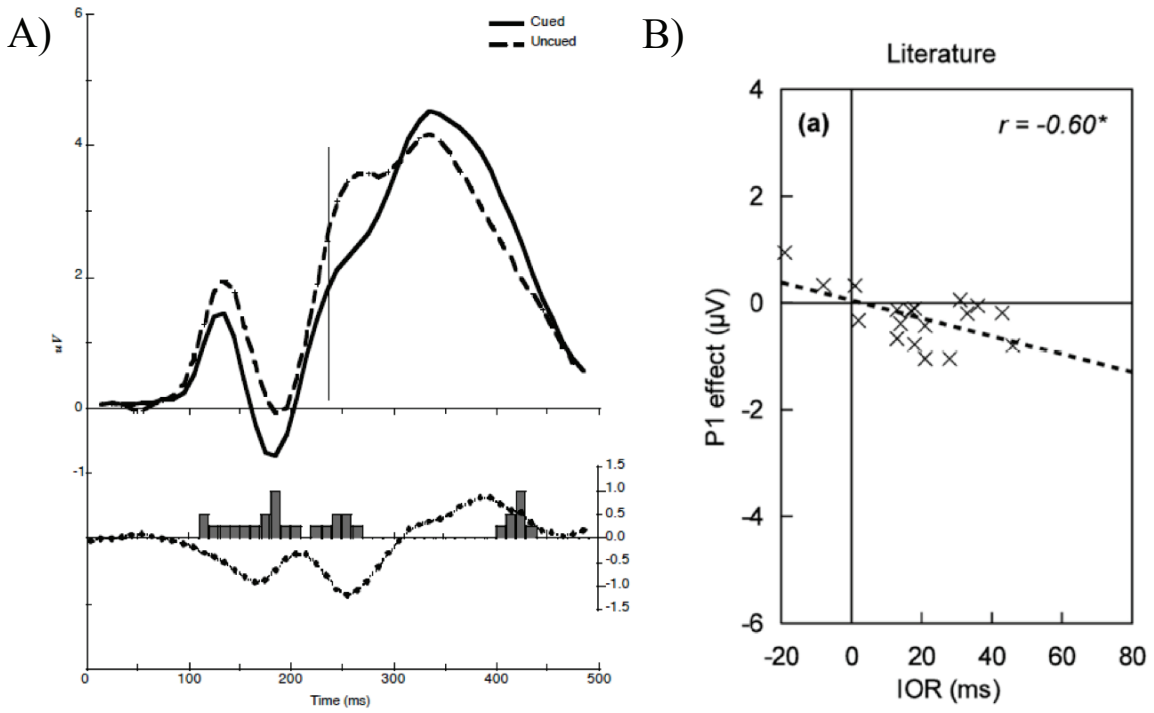

Figure 1.6: A) Data from the occipitally recorded ERP waveforms from six conditions of four studies (Hopfinger & Mangun, 1998; 2001; McDonald, Ward, & Kiehl, 1999; Prime & Ward, 2004). The upper plot shows the grand average target-elicited cued and uncued waveforms. The lower plot shows the difference waveform (cued minus uncued), and the histogram reveals where, based on t-tests with each study contributing one pair of values, the difference is significant. The heights of the bars represent p values of 0.01 (large), 0.025 (medium) and 0.05 (small) (from Klein, 2004). B) Scatterplot of IOR and P1 cueing effects (cued minus uncued), with r scores representing the correlation size and asterisks representing significance. X's represent the individual results of 19 published experiments from 9 published manuscripts (Hopfinger & Mangun, 1998; 2001; McDonald et al., 1999; Prime & Jolicoeur, 2009a; 2009b; Prime & Ward, 2004; 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005) in which a cue-ignored IOR paradigm was used (from Satel, Hilchey, Wang, Story, & Klein, 2013).

1.3 Neuroimaging investigations of IOR

EEG experiments have long been used to explore the dynamics of cognitive

phenomena. Although the spatial resolution of EEG is quite low, the high temporal resolution and non-invasiveness of this methodology provide an excellent opportunity to examine the temporal dynamics of brain processes while humans perform cognitive tasks. The traditional way of performing EEG experiments is to examine the event-related potentials (ERPs) time-locked to task events in different experimental conditions. That is, as events occur during an experimental protocol, associated changes in brain potentials are examined. Since these changes are often very small, it is normal to have many subjects perform the same task many times, and then to average brain potentials over the same events and different subjects.

In ERP studies of orienting and attention (for a review see, Luck, Woodman, & Vogel, 2000), it has been shown that attention can modulate early sensory processing (e.g., Mangun & Hillyard, 1991; Rugg, Milner, Lines, & Phalp, 1987). The P1 ERP component is the first positive deflection in an ERP waveform derived from electrodes over parieto-occipital sites (Clark & Hillyard, 1996), occurring around 100 ms after stimulus onset. Since visual information about the stimulus has only reached extrastriate cortex at this early time period, it is likely that any effects of attention on P1 components are related to early sensory/input processing (Luck et al., 2000). When contrasting the waveforms generated by attended and unattended stimuli, the P1 component shows clear enhancements in magnitude when attention has been directed to a stimulus. Such results have led researchers to propose that attention acts on early sensory/input processes as a type of sensory gain control mechanism (Luck et al., 2000).

In contrast to the P1 enhancements observed along with behavioral facilitation

when attention is directed to a stimulus, in designs where IOR is exhibited behaviorally, P1 amplitudes are normally reduced for cued targets (McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Satel, Hilchey, Wang, Story, & Klein, 2013; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005; Wascher & Tipper, 2004). Figure 1.6A presents a graphical depiction of mean ERP activity across several early studies (from Klein, 2004b) and Figure 1.6B illustrates the relationship between IOR scores and P1 modulations across all studies using an IOR design that have reported the data numerically (Hopfinger & Mangun, 1998; 2001; McDonald et al., 1999; Prime & Jolicoeur, 2009a; 2009b; Prime & Ward, 2004; 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005). These results show a clear pattern of P1 reductions for cued targets in a cue-target paradigm with manual responses. Note, however, that P1 cueing effects have also been observed without IOR (Doallo, Lorenzo-Lopez, Vizoso, Holguin, Amenedo, Bara, & Cadaveira, 2004; Hopfinger & Mangun, 1998), and IOR has also been observed without P1 cueing effects (Hopfinger & Mangun, 2001; McDonald et al., 1999; Prime & Ward, 2006; van der Lubbe et al., 2005), so the relationship between P1 modulation effects and IOR is still inconclusive. However, the consistent observation of P1 reductions associated with cued targets in these designs suggests that IOR may also act as a type of sensory gain control mechanism, reducing, rather than amplifying, the input signal of repeated exogenous stimuli.

Although not yet as thoroughly investigated, but deserving of further attention, there is some evidence that several other ERP components are modulated in association with IOR. Here, we will briefly discuss the potential relationship of two additional ERP

components to IOR, the Nd and N2pc components. The Nd, or negative difference, component, is a negative deflection in the ERP waveform in the time period between about 220 - 300 ms post-stimulus (Prime & Ward, 2006). In terms of IOR, many studies using a cue-target IOR paradigm have observed Nds for cued targets along with P1 reductions and behavioral IOR (McDonald et al., 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004, 2006; Satel et al., 2013; Wascher & Tipper, 2004), potentially reflecting an association with IOR (Satel, Wang, Hilchey, & Klein, 2013). Because the Nd component occurs later in time than the P1 component, its modulation by IOR suggests that IOR affects later cognitive processing, possibly in addition to providing a sensory gain control function, as implied the observed P1 modulation. Previous work using a go/no-go cueing paradigm (Prime & Ward, 2006) demonstrated that the Nd effect was absent on trials when no response was required, suggesting that the Nd effect may originate from motor programming.

Arising at around the same latency post-target as the Nd component, the N2pc component has also been implicated as potentially having a relationship with IOR (McDonald, Hickey, Green, & Whitman, 2009). The N2pc component is associated with focusing attention on targets in visual search paradigms, arising at parieto-occipital electrode sites that are contralateral to the locus of attention (Luck & Hillyard, 1994). Since the N2pc component is associated with the focusing of attention on a stimulus, its association with IOR would point to a later attentional component underlying the phenomenon. Further investigation of these ERP components could provide additional evidence about the distinction between early/input and late/output based inhibitory cueing

effects and should be pursued in conjunction with behavioral, neuroscientific, and computational techniques.

1.4 Computational investigations of IOR

As far as we know, there have been no previous computational modeling attempts specifically examining the spatiotemporal dynamics of IOR in a cue-target IOR paradigm. However, computational models of visual attention often include inhibitory, IOR-like mechanisms in their implementations. The biased competition hypothesis (Duncan & Humphreys, 1989; Desimone & Duncan, 1995) suggests that multiple, external stimuli activate neuronal populations that interact competitively. A winning stimulus, in terms of orienting toward it, is chosen based on competitive interaction between neurons representing bottom-up salience and top-down modulation. Many models of visual spatial attention have incorporated such winner-take-all mechanisms to examine attentional deployment and visual search mechanisms in competitive networks (for a review see Itti & Koch, 2001). These networks topographically represent bottom-up, exogenous information about external stimuli in saliency maps throughout the brain (Koch & Ullman, 1985; Itti, Koch, & Niebur, 1998; Itti & Koch, 2000). Some models also incorporate top-down, endogenous information, so that net salience is influenced by both exogenous and endogenous signals to varying degrees (Wolfe, 1994; Tsotsos, Culhane, Wai, Lai, Davis, & Nuflo, 1995; van de Laar, Heskes, & Gielen, 1997; Heinke & Humphreys, 2003; Navalpakkam & Itti, 2005; Chen & Zelinsky, 2006). Exogenous inputs to these saliency maps normally represent bottom-up signals from visual cortex, while endogenous inputs represent top-down modulatory signals from higher areas of cortex

(Itti & Koch, 2001; Shipp, 2004).

Researchers have developed models of such networks that are proposed to exist in areas such as the frontal and parietal eye fields (Thompson & Schall, 2000; Kusunoki, Gottlieb, & Goldberg, 2006), primary visual cortex (Li, 2002), posterior parietal cortex (Gottlieb, Kusunoki, & Goldberg, 1998; Deco, Pollatos, & Zihl, 2002), ventral pulvinar (Robinson & Petersen, 1992; Shipp, 2004), and the SC (Trappenberg et al., 2001; Godjin & Theeuwes, 2002). Each of these maps could represent information at different levels of processing, with varying degrees of contribution from exogenous and endogenous signals, reflecting their relative roles in perception and action (Itti & Koch, 2001).

A master saliency map is often proposed to competitively integrate exogenous and endogenous information received from other levels of processing (Itti & Koch, 2001; Shipp, 2004). The location of visual attentional deployment is then chosen based on activity in the master saliency map (Itti & Koch, 2001; Shipp, 2004). With this approach, stimuli that have previously captured attention will remain highly salient as long as they remain visible, so a mechanism must be introduced to reduce salience and discourage perseveration and re-inspections. As noted by Itti and Koch (2001), ”One efficient computational technique, which has received empirical support, consists of transiently inhibiting neurons in the saliency map at the currently attended location” (p. 199). This inhibition thus ”suppresses the last attended location from the saliency map, so that attention can focus onto the next most salient location” (p. 196). Most computational research modeling visual attention has followed this approach, using strong inhibitory signals in saliency maps to suppress previously attended locations (Itti & Koch, 2000;

Parkhurst, Law, & Niebur, 2002; Navalpakkam & Itti, 2005; Sun, Fisher, Wang, & Gomes, 2008). Although this approach captures the general behavioral dynamics of IOR, it assumes that IOR is the result of late attentional processing, is very strong, long lasting, and consistent across paradigms. While some of these conditions may be true (IOR is known to be long lasting), the neural implementation of the spatiotemporal dynamics of behaviorally exhibited IOR remain uncertain.

Desimone and Duncan (1995) proposed an alternative approach to the use of a master saliency map (where strong inhibition of previously attended stimuli represents IOR) based on the biased competition hypothesis. These authors propose that a master saliency map is not required to capture the properties of visual spatial attention (Desimone & Duncan, 1995; Deco et al., 2002). Implementation of this model uses a modular approach, with a location map in posterior parietal cortex (a type of saliency map) dynamically interacting with lower level feature maps and top-down influences (Deco et al., 2002). A selected stimulus location is a reflection of activity in multiple neural regions based on dynamic interactions between multiple areas (Deco et al., 2002), rather than just determined from a winning stimulus in a single network (Itti & Koch, 2001).

1.4.1 Dynamic neural field (DNF) modeling

The particular implementation used in our research group is that of a dynamic neural field (DNF) model of the iSC (see Figure 1.7 for a schematic illustration). DNF models are based on lateral inhibition type neural fields, as described in Wilson and Cowan (1973). The basic formulation of this model is equivalent to that used in recurrent

point-attractor neural networks that have been extensively studied in, for example, the investigation of associational memories (Cohen & Grossberg, 1983; Hopfield, 1982; Morita, 1993). Early work on neural fields provides a theoretical foundation for the investigation of competitive brain networks (Grossberg, 1973; Amari, 1977).

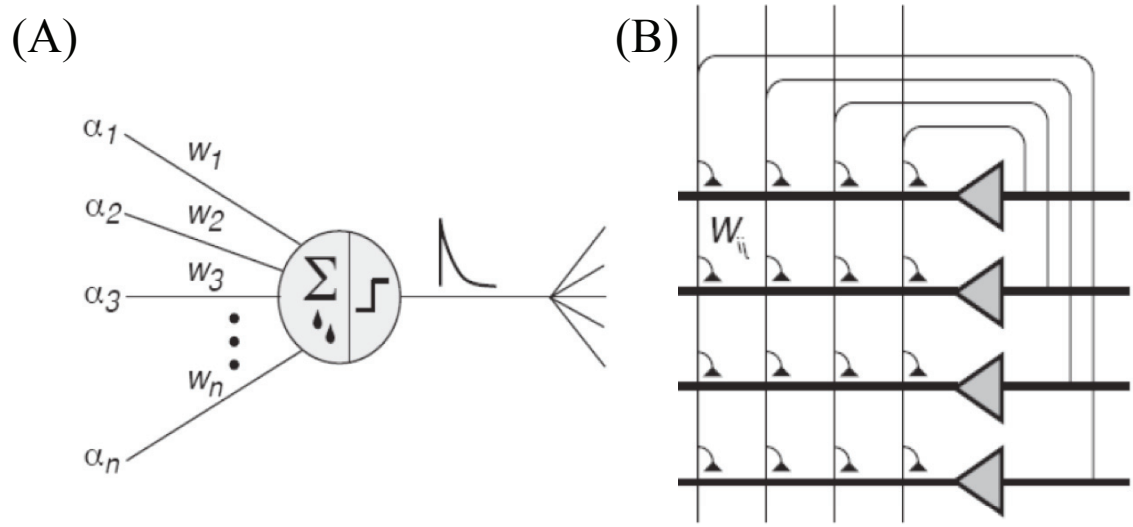

Figure 1.7: (A) Nodes in the network represent neurons in the iSC with retinotopically coded visuospatial response properties. These leaky nodes integrate the activity of all other sufficiently activated nodes (alpha$_n$) subject to the lateral interaction profile (w_n, the connection strength from one node to another). (B) All nodes are fully connected, receive external inputs, and send outputs to a saccade generator system (from Trappenberg, 2010).

Many researchers have used neural field models with competitive lateral interactions to simulate various brain areas. Examples of such work include models of the representation of visual stimuli in primary visual cortex (Janke, Erlhagen, Dinse, Akhavan, Giese, & Schoner, 1999; Suder, Worgotter, & Wennekers, 2000), path integration and cognitive mapping in hippocampal place cells (Samsonovich & McNaughton, 1997; Stringer, Trappenberg, Rolls & de Araujo, 2002), more general investigations regarding cortical information processing (Taylor & Alavi, 1995; Usher,

Stemmler, Koch, & Olami, 1996; Wu, Amari, & Nakahara, 2002), as well as attentional orienting phenomena (Satel, Trappenberg, & Klein, 2005; Standage, Trappenberg, & Klein, 2005; Trappenberg et al., 2001; Wilimzig, Schneider, & Schoner, 2006). The proliferation of these competitive networks in neural systems modeling and their effectiveness in reproducing experimental data suggests that they may be capturing fundamental properties of how the brain processes information (Desimone & Duncan, 1995; Trappenberg et al., 2001).

The iSC, in particular, has been extensively modeled with DNF modeling techniques (Kopecz, 1995; Kopecz & Schoner, 1995; Trappenberg et al., 2001; Satel et al., 2005; Wilimzig et al., 2006). As described above, this midbrain structure receives converging inputs from many other brain areas and is intimately involved with attentional orienting (Dorris, Pare, & Munoz, 1997; Munoz & Wurtz, 1995; Sparks & Hartwich-Young, 1989). The SC is a primary component of the oculomotor system and has been shown to be an active participant in the elicitation of IOR (Dorris et al., 2002; Fecteau & Munoz, 2005; Sapir et al., 1999). Kopecz (1995) and Kopecz and Schoner (1995) showed that such a DNF model could simulate the SRTs of various behavioral paradigms. Trappenberg et al. (2001) later advanced this model to more fully represent the iSC in a biologically plausible manner, and extended it to simulate additional behavioral effects. Our lab is currently furthering this line of research by advancing a DNF model of the iSC to account for IOR through the incorporation of additional, empirically motivated, mechanisms underlying IOR.

Model architecture: In the current work, we use a one-dimensional DNF model

with nodes that represent neurons in the iSC with retinotopically coded visuospatial response properties. Implementation of model dynamics is similar to that used in previous work (Trappenberg et al., 2001), with fully connected leaky nodes integrating the activity of all other sufficiently activated nodes, subject to the lateral interaction profile (the connection strength from one node to another). Gaussian-shaped external inputs to the network represent sensory signals from the external environment as well as endogenously-generated signals , and send outputs to a saccade generator system (from Trappenberg, 2010).

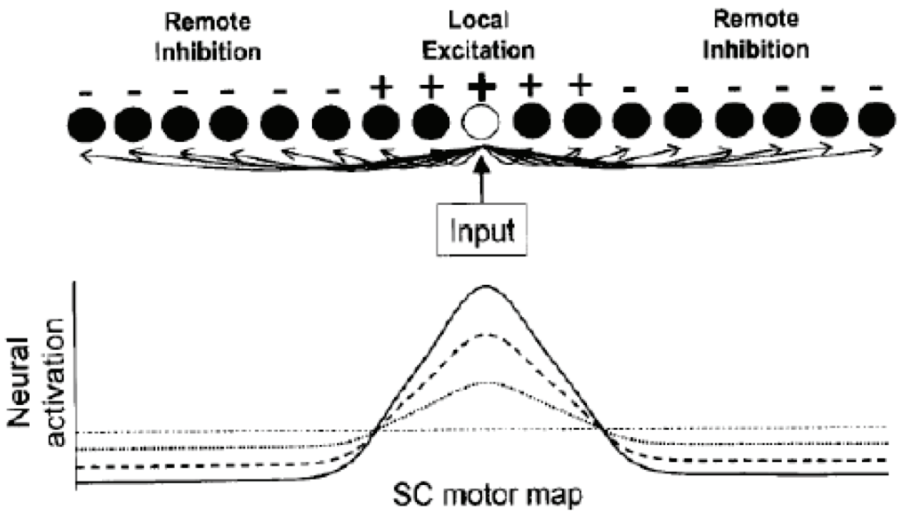

Figure 1.8: Lateral interaction profile (internal connectivity) of the collicular motor map (from Munoz & Fecteau, 2002). This interaction structure is characterized by short-distance excitation and long-distance inhibition, with a constant overall level of activity. Note that although our model uses a difference of Gaussians lateral interaction that aligns more closely with monkey neurophysiological evidence (see Trappenberg et al., 2001).

Nodes in the network are laterally connected such that proximal nodes have excitatory connections and distal nodes have inhibitory connections, in a Mexican-hat like configuration (Trappenberg et al., 2001). This interaction structure is captured by the

interaction matrix, *w*, that depends only on the spatial distance between nodes (see Figure 1.8 for a schematic illustration). Here, the interaction profile is approximated with two Gaussians, as defined in Equation 1.1, and is kept constant across all simulations. Although this lateral interaction was chosen to approximate cell recordings in the iSC of monkeys (see Trappenberg et al., 2001), it is not an exact fit to the neurophysiological data. All simulations used the following interaction matrix parameters: a = *72, b = 24, c = 6.4*, $\sigma_a = 0.6$, and $\sigma_b = 1.8$.

$$w_{ij} = a * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_a^2}\right) - b * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_b^2}\right) - c \qquad \text{Equation 1.1}$$

$$\tau \frac{du_i(t)}{dt} = -du_i(t) + \sum_j w_{ij} r_j(t) \Delta x + I_i(t) + u_0 \qquad \text{Equation 1.2}$$

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)} \qquad \text{Equation 1.3}$$

$$I_k = d * \exp\left(\frac{((k-i)\Delta x)^2}{2\sigma_d^2}\right) \qquad \text{Equation 1.4}$$

The dynamics of the internal state, $u_i(t)$, of node *i* is described in Equation 1.2, where $\tau = 10$ ms is a time constant, w_{ij} is the connection strength (weight) between node *i* and node *j*, $r_j(t)$ is the activity level (average firing rate) of node *j*, $I_i(t)$ represents the external input to node *i*, and $u_0 = 0$, is a constant resting level. The activity of node *i*, $r_i(t)$, as a function of its internal state, $u_i(t)$, is defined by a sigmoidal gain function (Equation 1.3), where $\beta = 0.07$ and $\theta = 0$ were used as parameters in all simulations to define the steepness and offset of the sigmoid.

The iSC is a neural structure where bottom-up (exogenous) inputs and top-down (endogenous) inputs are integrated (for a more detailed description of projections to and from the SC, see Fecteau & Munoz, 2006; Munoz & Fecteau, 2002). Our model of the iSC receives both exogenous (I_{exo}), and endogenous (I_{endo}) inputs. Both types of input signals take on a Gaussian spatial shape, centered at location *i*. Thus, input to other nodes (*k*) in the network depends on the distance between nodes *i* and *k,* as represented by Equation 1.4, where *d* represents the strength of the input, and σ_d represents the width of the input.

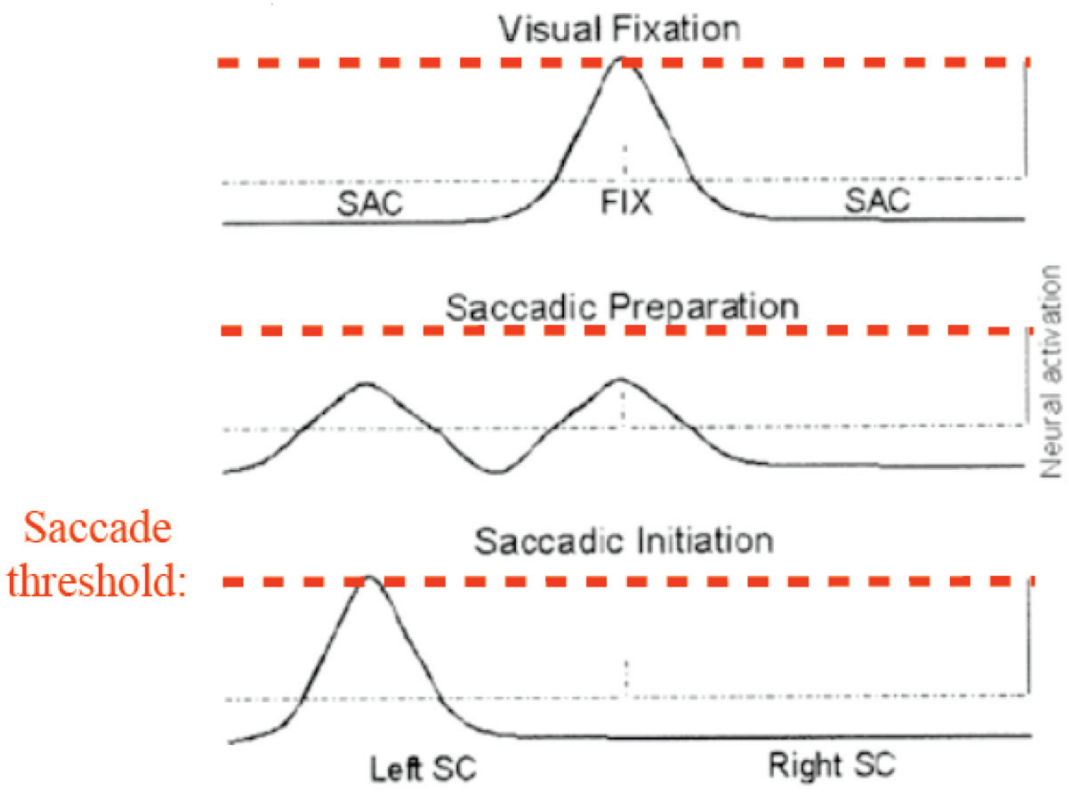

Figure 1.9: Schematic illustration of the activation required for a saccade to occur (modified from Munoz & Fecteau, 2002). During foveation of a visual stimulus, fixation neurons (FIX) at the rostral pole of the iSC fire tonically, inhibiting more caudal neurons (SAC). For a saccade to occur, external inputs must lead to enough activation for a neuron to cross threshold, generating an output signal that leads to the initiation of an eye movement to the location represented by the associated cell.

Once the input signals arrive at the SC, they are competitively integrated before sending an output signal to the brainstem saccade generator system. The current investigations use an output signal from the iSC to represent saccade generation. Neurophysiological microstimulation studies on non-human primates have revealed that neurons in the iSC are organized into a retinotopically coded motor map (e.g., Robinson, 1972). That is, all locations in the visual field are represented topographically in the SC. Thus, sufficient activation of a neuron in the iSC will produce a saccade to the represented location in the visual field. As in Trappenberg et al. (2001), SRTs were calculated as the difference between the time of external input onset and the time at which any node reaches 80% of its maximum firing rate. When a node reaches threshold, a saccade initiation signal is transmitted to the brainstem, which triggers a saccade to the associated retinotopic location (see Figure 1.9 for an illustration).

1.5 Theories of IOR

In perhaps the most exhaustive investigation of IOR to date, Taylor and Klein (2000) tested the Posner cueing paradigm with four mixed stimulus combinations (peripheral cue - peripheral target, peripheral cue - central target, central cue - peripheral target, and central cue - central target) in each of six different response combinations to the two stimuli (no response - manual, no response - saccade, manual - manual, manual - saccade, saccade - manual, and saccade - saccade), as illustrated in Figure 1.10. Based on their results, Taylor and Klein (2000) proposed that there are 2 mutually exclusive forms, or flavors, of IOR depending on whether or not the oculomotor system is actively suppressed. When eye movements are forbidden, an attentional/perceptual form of IOR

acts on input processes to degrade the quality of the target-elicited signals, but only when targets are peripheral onsets. When the oculomotor system is activated, either in response to the cue or target, then a motoric form of IOR acts on output processes, equally slowing any responses in the cued direction. Further support for this theory has been presented by Chica, Taylor, Lupianez, and Klein (2010) who administered a standard spatial cueing paradigm with a peripheral onset cue and a to-be-discriminated target. In different blocks, subjects were required to either remain fixated or make a saccade to the cues and return to fixation before target appearance. Results demonstrated that IOR expressed itself as a speed-accuracy tradeoff (SAT) when saccadic responses were made to cues (motoric - output-based - IOR), but not when the oculomotor system was suppressed (attentional/ perceptual - input-based - IOR).

It seems clear from these results that different forms of IOR arise depending on the activation state of the oculomotor system, though it is still possible that multiple neurophysiological mechanisms contribute to the different forms of IOR. Although their results have recently been contested (see Hilchey, Klein, & Ivanoff, 2012a), Abrams and Dobkin (1994a) presented evidence that there are at least two additive mechanisms underlying IOR. The present line of work will further investigate the viability of such a multiple mechanisms theory of IOR using a DNF model of the iSC to implement and test alternate theories of IOR in simulations of behavioral paradigms. Predictions generated by the model are then used to develop and test experimental designs using behavioral and ERP methodologies, further cementing our understanding of this important cognitive phenomenon.

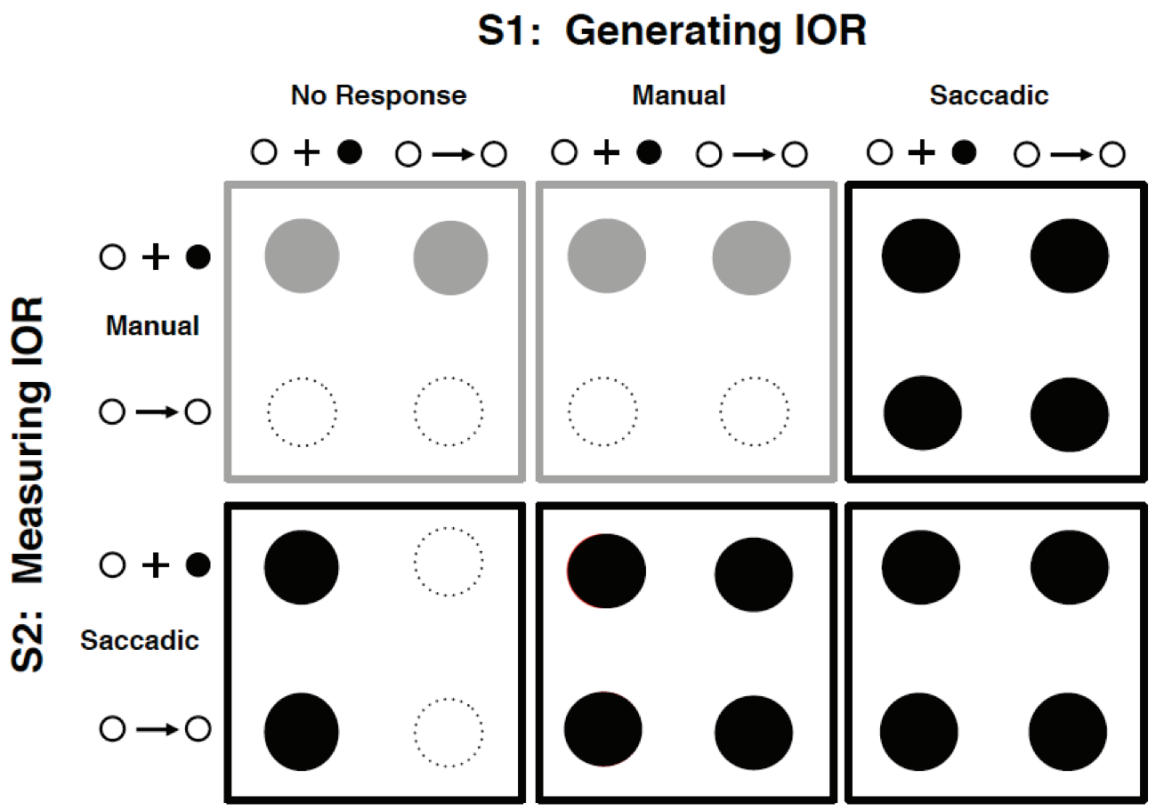

Figure 1.10: Each observer was tested in each of the six conditions on separate days. The sequence of events was identical in each of these conditions: a trial begins with the appearance of three rectangular landmarks, one at fixation and one left and right of fixation. The first event was a peripheral brightening (exogenous) or central directional arrow signal (endogenous) whose location and direction, respectively, did not correlate with a future second signal (S2), occurring 1 second after the first (S1). S2, used to measure the effect of S1, was a randomly-presented central arrow (endogenous) or peripheral onset disc (exogenous). The observer participated in each factorial combination of response type to S1 (no response, manual, or saccade) and S2 (manual or saccade) which, as noted, yielded six conditions. Note principally that it was not possible to measure the effect of S1 with a central arrow S2 when saccadic eye movements were not required (the two conditions highlighted in gray), whereas it was in all conditions where saccadic eye movements were required (conditions highlighted in black). Solid circles represent conditions in which significant IOR was observed behaviorally, while significant IOR was not observed in conditions with unfilled dotted circles (modified from Hilchey, Klein, & Satel, under review).

1.6 Chapter roadmap

This chapter has provided an overview of the essential background material on which the following chapters are founded, providing motivation and context for the

subsequently presented program of research.

Chapter 2 (Satel, Wang, Trappenberg, & Klein, 2011) presents the results of DNF simulations implementing a sensory mechanism of IOR that acts solely on repeatedly stimulated input pathways, reducing the strength of subsequent signals along the same input pathway. These simulations nicely reproduce the pattern of monkey behavioral and neurophysiological data when cues are ignored and targets are fixated (no response peripheral - saccade peripheral cell of Figure 1.10), but only at relatively short CTOAs (less than ~600 ms), and only for spatially overlapping repeated exogenous stimulation in retinotopic coordinate space.

Chapter 3 (Wang, Satel, Trappenberg, & Klein, 2011) presents further DNF simulation results implementing an independent motor mechanism of saccadic IOR that arises as a result of the aftereffects of an earlier eye movement. Since retinotopic space remaps after every eye movement (i.e., the center of a retinotopic map is always the currently foveated location), remaining activity after an eye movement will encourage forward saccades (saccades in the same direction as a previous saccade), at least until this activity decays. This motoric mechanism of IOR should arise after eye movements to cues in traditional cue-target designs, as well as in visual search paradigms. Simulations were able to reproduce the pattern of results in a number of such paradigms and predictions were made to drive further empirical research.

Chapter 4 (Wang, Satel, Hilchey, & Klein, 2012b) tests a behavioral prediction of our multiple mechanisms theory of IOR in a dual target paradigm used to induce averaging saccades at short CTOAs. Previous computational (Satel et al., 2011) and

empirical work (Watanabe, 2011) has demonstrated that, at a CTOA of 600 ms, when presented with a single cue and double targets, saccade landing sites are biased *away* from the cued location. This finding has been attributed to a spatial effect of IOR, suggesting that averaging saccades would be biased *toward* the cued location at CTOAs short enough to induce behavioral facilitation (Satel et al., 2011; see Chapter 2). Results of these experiments demonstrated that, contrary to our model predictions, averaging saccades were always biased away from cued locations.

Chapter 5 (Wang, Satel, & Klein, 2012a) tests another prediction of the 2-mechanisms (sensory and motor) theory of IOR, namely, that the sensory and motor mechanisms of IOR proposed in Satel et al. (2011; see Chapter 2) and Wang et al. (2011; see Chapter 3), respectively, should be additive in conditions that recruit both mechanisms. An experiment was carefully designed that had each participant perform three different tasks, separately, which recruited the sensory, motor, and both the sensory and motor mechanisms of IOR. Results robustly demonstrated that sensory and motor mechanisms of IOR were independent and additive, supporting a multiple mechanisms theory of IOR. However, it was found that the motor mechanism of IOR was much longer lasting than predicted, hinting at the existence of yet another motoric mechanism of IOR at long CTOAs.

Chapter 6 (Satel & Wang, 2012) continues the empirical research begun in Wang et al. (2012a; see Chapter 5) by expanding on the previous experimental design to replicate the additivity of sensory and motor mechanisms of IOR under various additional conditions. These results suggest that motoric and sensory forms of IOR may not be

mutually exclusive as previously suggested by Taylor and Klein (2000). Additivity continued to be observed in three additional experiments where potentially confounding experimental factors were eliminated from the previous design. However, when the same design was tested with manual responses to targets (instead of saccadic responses), equivalent IOR was observed in all three conditions and additivity was not observed. This result suggests that our identified motor mechanism of IOR does not carry over to the manual response system, but that there may be another manual inhibitory motor mechanism.

Chapter 7 (Satel et al., 2013) further investigates IOR behaviorally with manual responses to targets, while also recording EEG activity in order to analyze target-elicited differences in cued and uncued conditions, with and without eye movements to the cues. Previous ERP investigations of IOR have focused exclusively on ignored-cue paradigms with manual target responses. Since the state of the oculomotor system is critical to the form of IOR generated (e.g., Chica et al., 2010), we also incorporated eye monitoring technology to ensure suppression of the oculomotor system in one condition, and appropriate eye movement behavior in the other. Results demonstrated that the early sensory P1 ERP component was reduced in amplitude for cued targets in both eye movement conditions, but that these P1 modulations were only correlated with behavioral IOR scores when the oculomotor system was actively suppressed.

Chapter 8 (Satel, Wang, Hilchey, & Klein, 2012) presents the results of another ERP study of IOR using a paradigm that dissociates spatiotopic and retinotopic locations by including an eye movement between cue and target appearance. As observed

previously (Maylor & Hockey, 1985) - and as required by a novelty seeking or foraging facilitator functional hypothesis of IOR - IOR was observed in spatiotopic coordinates. However, target-elicited P1 reductions were only observed for retinotopically cued targets, and later Nd effects were only observed for spatiotopically cued targets. Along with the results of Satel et al. (2012; see Chapter 7), these results provide further evidence that P1 reductions are neither necessary nor sufficient to observe IOR behaviorally.

Chapter 9 (General discussion) integrates and summarizes the results of the previous chapters, focusing on potential limitations of this work and the proposal of further directions for research.

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CHAPTER 2 MODELING INHIBITION OF RETURN AS SHORT-TERM DEPRESSION OF EARLY SENSORY INPUT TO THE SUPERIOR COLLICULUS

Satel, J., Wang, Z., Trappenberg, T. P., and Klein, R. M. (2011). Modeling inhibition of return as short-term depression of early sensory input to the superior colliculus. Vision Research, 51(9): 987 - 996 (reformatted and reprinted with permission of the publisher, Elsevier).

2.1 Abstract

Inhibition of return (IOR) is an orienting phenomenon characterized by slower behavioral responses to spatially cued, relative to uncued targets, when the cue-target onset asynchronies (CTOAs) are long enough that cue-elicited attentional capture has dispersed. Here, we implement a short-term depression (STD) account of IOR within a neuroscientifically based dynamic neural field model (DNF) of the superior colliculus (SC). In addition to the prototypical findings in the cue-target paradigm (ie., the biphasic pattern of behavioral enhancement at short CTOAs and behavioral costs at long CTOAs), a variety of findings in the literature are generated with this model, including IOR in averaging saccades and the co-existence of IOR and endogenous orienting at the same location. Many findings that cannot be accommodated by this model could be accounted for by incorporating cortical contributions.

2.2 Introduction

Inhibition of return (IOR) is an orienting phenomenon characterized by slower behavioral responses to targets presented at spatially cued, relative to uncued locations, when the cue-target onset asynchrony (CTOA) is longer than approximately 200 ms (for a review, see Klein, 2000). This phenomenon was first discovered by Posner and Cohen (1984), with a model task (see Figure 2.1A for an illustration) in which non-predictive peripheral cues are followed by targets that require simple detection responses. Posner and Cohen (1984) showed that reaction times (RTs) to targets appearing at previously cued locations were faster than RTs to targets appearing at uncued locations, so long as the CTOA was short. However, when CTOA was extended, this early benefit evolved into

a behavioral cost, as exhibited by slower RTs for targets presented at cued locations than for targets at uncued locations (see Figure 2.1B for an illustration of these effects). This later effect has been termed IOR (Posner, Rafal, Choate, & Vaughan, 1985), and has since been demonstrated by many researchers using a number of experimental paradigms (for a discussion and testing of various IOR experimental paradigms, see Taylor & Klein, 1998; Taylor & Klein, 2000). Although the neural processes underlying IOR are still under investigation, previous behavioral (Rafal, Calabresi, Brennan, & Sciolto, 1989), lesion (Posner et al., 1985; Sapir, Soroker, Berger, & Henik, 1999; Sereno, Briand, Amador, & Szapiel, 2006), and developmental (Simion, Valenza, Umiltà, & Barba, 1994; Valenza, Simion, & Umiltà, 1995) studies have suggested that the oculomotor system, particularly the superior colliculus (SC), is intimately involved with the generation and processing of IOR. Neurophysiological work has further confirmed the involvement of the SC in IOR (for a review, see Fecteau & Munoz, 2006).

The superficial layer of the SC (sSC) receives input from the retina, primary visual cortex, and extra striate areas (Lui, Gregory, Blanks, & Giolli, 1995; Rodieck & Watanabe, 1993), and does not receive feedback from the areas it projects to. Thus, it represents only early sensory information that has not been contaminated by further processing in other regions (Clower, West, Lynch, & Strick, 2001). In contrast, the intermediate layer of the SC (iSC) receives and integrates sensory input as well as cortical inputs from the prefrontal, parietal, and temporal areas (Clower et al., 2001; Lui et al., 1995; Sparks & Hartwich-Young, 1989).

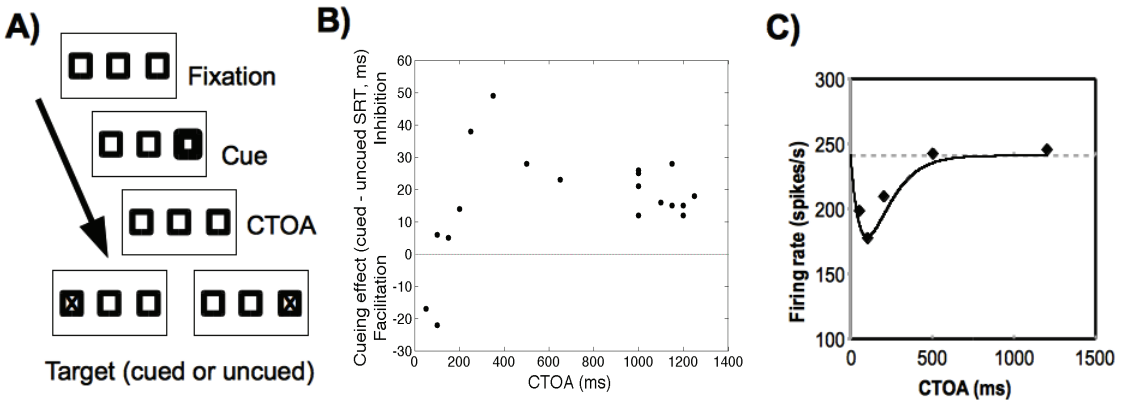

Figure 2.1: (A) Sequence of events in a typical trial using a traditional cue-target IOR paradigm. (B) Human and monkey behavioral data from studies using a cue-saccade paradigm demonstrating the time course of IOR. The cueing effect (cued – uncued SRT, ms) is shown as a function of CTOA, with facilitation seen at short CTOAs, and IOR at long CTOAs (adapted from Klein, 2000). (C) Illustration of the sensory STD thought to underlie the behavioral observation of IOR. The diamond data points denote the response of visual neurons (in the sSC) to cued targets, the dashed line denotes the average firing rate of visual neurons to uncued targets (adapted from Fecteau and Munoz, 2005). These single-unit recordings demonstrate that target elicited early sensory input strength is reduced following a previous stimulation. In our model, an alpha function is used to approximate this sensory STD process, as illustrated by the solid line.

Recent single-unit recording studies using the cue-target experimental paradigm have shown that target induced neural activity in the iSC is greatly reduced for previously cued, as compared to uncued targets (Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005). This reduction in activity is highly correlated with saccadic reaction times (SRTs) to targets, further cementing the relationship of iSC activity to behaviorally exhibited IOR (Fecteau & Munoz, 2005). More importantly, when electrical stimulation was delivered through the recording electrode to elicit a saccade, the latency of these electrically evoked saccades was actually faster for previously cued regions (Dorris et al., 2002), suggesting that neural activity was not directly suppressed in the iSC following a cue.

Thus, the IOR effect is not caused by active inhibition of recently stimulated iSC sites, but rather by a reduction in the strength of subsequent input signals to these neurons. This hypothesis was supported by Fecteau and Munoz (2005), who found reduced responses to cued targets in the sSC (see Figure 2.1C, diamonds). As mentioned earlier, this reduction of discharge in the sSC is purely sensory, hence, we label it short-term depression (STD) of sensory input. As shown in Figure 2.1C, this sensory STD can be modeled with an alpha function:

$$\alpha_{ctoa} = A \frac{t_{ctoa}}{t_{max}} e^{(1-\frac{t_{ctoa}}{t_{max}})}$$

with parameters $A = -63$ and $t_{MAX} = 100$ specifying the maximal discharge reduction and the time when this discharge reduction reaches its maximum. Also note that this STD function nicely correlates (negatively) with the behavioral IOR effects reported in previous studies (see Figure 2.1B). Based on this sensory STD in the sSC, Fecteau and Munoz (2005) postulated that IOR simply ”reflects a habituated sensory response occurring in early sensory areas that is subsequently transmitted through the rest of the brain” (p. 1722). Furthering this line of thought, Dukewich (2009) proposed a theory wherein IOR is simply the result of habituation-like mechanisms at multiple stages of processing, occurring anytime a pathway is repeatedly stimulated (see also Huber, 2008; Patel, Peng, & Sereno, 2010).

In sum, although IOR, as a behavioral effect, could have multiple underlying neural mechanisms, we believe a large set of IOR effects observed in the cue-target paradigm can be explained in the input domain, through STD of early sensory inputs. The primary purpose of this work is to implement and quantify this sensory STD hypothesis

of IOR by expanding an established DNF model of the iSC (Trappenberg, Dorris, Munoz, & Klein, 2001) to include STD of early sensory input strength. Furthermore, this work compares the results of simulations with established experimental results (Bell & Munoz, 2008; Fecteau & Munoz, 2005; Watanabe, 2001), and makes predictions that can be investigated empirically. Although we do not expect that this model will be able to account for all manifestations of IOR, we believe that much can be learned from the boundary conditions of its successes.

2.3 Dynamic neural field model of the SC

In the iSC, neurons are organized into a retinotopically coded motor map that specifies both the direction and the amplitude of saccades into the contralateral visual field. Converging inputs to this structure come from a multitude of cortical and subcortical regions which represent information related to both endogenous and exogenous control of attentional orienting (Klein, 2004a). When neural activity exceeds a predetermined threshold, an output signal is sent to the brainstem, generating a saccade. The interaction between neurons in the iSC is characterized by short-distance excitation and long-distance inhibition (for a review of related evidence, see Munoz & Fecteau, 2002). This lateral interaction can be easily captured through the use of dynamic neural field models (DNFs; Amari, 1977; Wilson & Cowan, 1973). Such models have been successfully used to model various eye movement related behaviors (Arai, Keller, & Edelman, 1994; Das, Keller, & Arai, 1996; Kopecz, 1995; Kopecz & Schöner, 1995; Trappenberg et al., 2001; Wilimzig, Schneider, & Schoener, 2006).

2.3.1 Model architecture

A one-dimensional DNF model that represents the iSC was used in the present simulations. Implementation of the model is similar to previous work (Trappenberg et al., 2001). We simplified the model by using only buildup neurons that are sufficient to describe the main dynamics leading to saccade initiation. The main enhancement we have made to the Trappenberg et al. (2001) model is the addition of a short-term plasticity mechanism to implement the STD hypothesized to underlie IOR (Dukewich, 2009; Fecteau & Munoz, 2005; Huber, 2008; Patel et al., 2010). Nodes in the network are laterally connected such that proximal nodes have excitatory connections and distal nodes have inhibitory connections, in a Mexican-hat like configuration (Trappenberg et al., 2001). In this model, $n = 1001$ nodes were used to represent 5 mm of each colliculus. Strong mutual inhibition was used to ensure that activity in the model will decay globally and reach an asymptotic inactive state.

The interaction structure within the iSC is captured by the interaction matrix, w, that depends only on the spatial distance between nodes (Trappenberg et al., 2001). This interaction profile is approximated with two Gaussians, as defined in Equation 1.1, and is kept constant across all simulations. Although this lateral interaction was chosen to approximate cell recordings in the iSC of monkeys (see Trappenberg et al., 2001), it is not an exact fit to the neurophysiological data. All simulations used the following interaction matrix parameters: $a = 72$, $b = 24$, $c = 6.4$, $\sigma_a = 0.6$, and $\sigma_b = 1.8$.

$$w_{ij} = a * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_a^2}\right) - b * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_b^2}\right) - c$$ Equation 1.1

$$\tau \frac{du_i(t)}{dt} = -du_i(t) + \sum_j w_{ij} r_j(t) \Delta x + I_i(t) + u_0$$ Equation 1.2

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)}$$ Equation 1.3

$$I_k = d * \exp\left(\frac{((k-i)\Delta x)^2}{2\sigma_d^2}\right)$$ Equation 1.4

The dynamics of the internal state, $u_i(t)$, of node *i* is described in Equation 1.2, where $\tau = 10$ ms is a time constant, w_{ij} is the connection strength (weight) between node *i* and node *j*, $r_j(t)$ is the activity level (average firing rate) of node *j*, $I_i(t)$ represents the external input to node *i,* and $u_0 = 0$, is a constant resting level. The activity of node *i*, $r_i(t)$, as a function of its internal state, $u_i(t)$, is defined by a sigmoidal gain function (Equation 1.3), where $\beta = 0.07$ and $\theta = 0$ were used as parameters in all simulations to define the steepness and offset of the sigmoid.

The iSC is a neural structure where bottom-up (exogenous) inputs and top-down (endogenous) inputs are integrated (for a description of projections to and from the SC, see Fecteau & Munoz, 2006; Munoz & Fecteau, 2002). Our model of the iSC receives both exogenous (I_{exo}), and endogenous (I_{endo}) inputs. Both types of input signals take on a Gaussian spatial shape, centered at location *i*. Thus, input to other nodes (*k*) in the network depends on the distance between nodes *i* and *k,* as represented by Equation 1.4, where *d* represents the strength of the input, and σ_d represents the width of the input.

2.3.2 Input and output parameters

Exogenous and endogenous input signals were modeled with a width of $\sigma_d = 0.7$, and fixation input signal width with $\sigma_d = 0.3$. A variable amplitude, d, was used, depending on the experimental task and types of input signals present (exogenous or endogenous), as described below. Fixation input was modeled as a sustained input signal, with a strength of $d = 5$, during times appropriate for the given experimental paradigm. Exogenous inputs were modeled with a transient dynamic, as in previous work, with a strength of d = *60* and an effective time constant of *teff = dt/10*, which decays the signal over time. A delay of 70 ms was added to the onset of all exogenous inputs, so that signals representing external visual stimuli appropriate to the simulated behavioral paradigm reach the network 70 ms after onset.

Endogenous move signal inputs, with an onset delay of 120 ms, were sustained until a reaction occurred. Reflecting the well-known foreperiod, or warning signal effect, reaction times vary with the interval between a warning cue and a target (Posner, Klein, Summers, & Buggie, 1973). Consequently, the strengths of endogenous input signals (the move signals) were modulated as a function of CTOA. The strength of these signals was always the same for both validly (same side) and invalidly (opposite side) cued targets, so they do not significantly affect the magnitude of the IOR effect, only the SRTs for different CTOAs. For this work, the strength of these signals have been chosen in order to fit monkey behavioral data (SRTs; Fecteau & Munoz, 2005) as accurately as possible. The foreperiod effect was simulated by using a linear equation ($y_1 = 7.3, m_1 = 0.3$) to increase the strength of the endogenous move signal as a function of CTOA, until *CTOA = 200*

ms, and a second linear equation ($y_2 = 14.5$, $m_2 = -0.0024$) to decrease the strength of this signal when CTOA was greater than 200 ms. Thus, due to temporal expectation effects, SRTs in all conditions are gradually increased as a function of CTOA until a CTOA of 200 ms, at which point they begin to decrease again. In simulations of the predictive cueing paradigm, an additional predictive, endogenous input, I_{pred}, was applied to the network after cue offset, with an initial strength of $d = 1$ and an effective time constant of $teff = dt/350$, which slowly increases the signal over time.

The strength of endogenous input signals varies as a function of SOA. All other input strengths (d), widths (σ_d), and rates of change ($teff$) were fixed according to the type of input signal (fixation, exogenous, endogenous, predictive, or double target). Fixation input, I_{fix}, was sustained over time when appropriate for the experimental paradigm being simulated, with a strength of $d = 5$, and a width of $\sigma_d = 0.3$. Exogenous input, I_{exo}, was transiently decayed over time ($teff_{exo} = -dt/10$), starting 70 ms after external stimuli appeared, with an initial strength of $d = 60$, and a width of $\sigma_d = 0.7$. The initial strength of exogenous inputs to locations which have been previously stimulated were decreased according to the STD function previously described. Endogenous move signal input, I_{endo}, was sustained over time starting 120 ms after external stimuli appeared, with a variable initial strength as a function of CTOA (as described above) between $d = 7.3$ and $d = 14.5$, and a width of $\sigma_d = 0.7$. Predictive input, I_{pred}, was transiently increased over time ($teff_{pred} = dt/350$), with an initial strength of $d = 1$, and a width of $\sigma_d = 0.7$. Simulations of the cue-double-target paradigm (described below), used a smaller exogenous input signal width ($\sigma_d = 0.45$) and a fixed endogenous move signal strength ($d = 10$). All other

parameters in the model were held constant.

As in Trappenberg et al. (2001), SRTs were calculated as the difference between the time of external input onset and the time at which any node reaches 80% of its maximum firing rate. When a node reaches threshold, a saccade initiation signal is transmitted to the brainstem, which triggers a saccade to the associated retinotopic location. An additional 20 ms efferent delay was added to simulated SRTs to approximate cell recording findings (Munoz & Wurtz, 1995; Robinson, 1972).

2.4 Simulations

DNF models of the the iSC have been successfully used to explain many orienting phenomena (Arai et al., 1994; Kopecz, 1995; Kopecz & Schöner, 1995; Meeter, Van der Stigchel, & Theeuwes, 2010; Trappenberg et al., 2001; Wilimzig et al., 2006). The simulations reported here expand previous work to examine the cue-target experimental paradigms used to empirically investigate IOR. The first set of simulations reproduce the classical findings in cue-target paradigms (ie., behavioral facilitation at short CTOAs and IOR at long CTOAs) with a simple sensory STD function and provide the foundation for the remaining simulations. Such sensory STD depends on the experimental setup and may interact with top-down, endogenous input from various cortical areas. By reproducing the findings of a cue-target experiment with predictive cues (Bell & Munoz, 2008), the second set of simulations, demonstrated that our model can represent the interaction between top-down and bottom-up inputs at the level the iSC. A third set of simulations explores saccadic averaging and IOR in a cue-target experimental paradigm with multiple simultaneous targets (Watanabe, 2001).

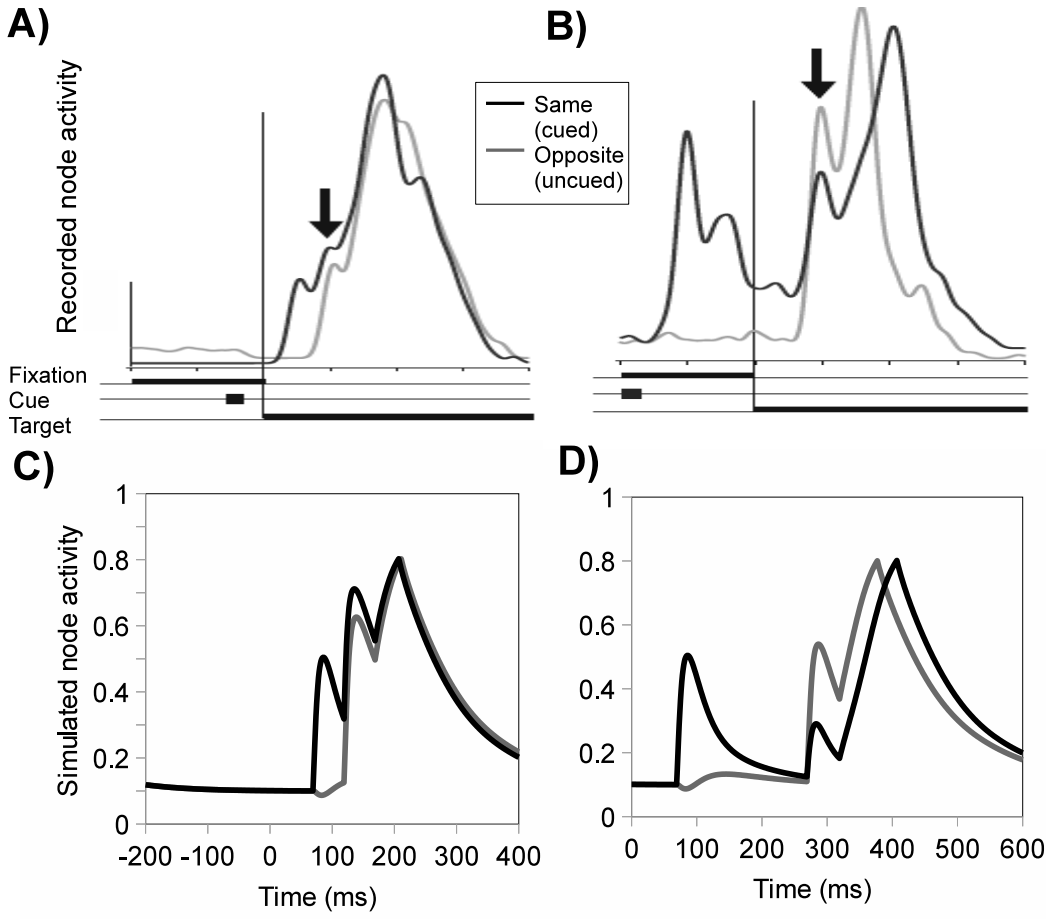

Figure 2.2: (A) Neurophysiological firing rates over time when CTOA = 50 ms. As indicated by the arrow, the strength of target elicited activity is reduced for cued, as compared to uncued, targets. Cued targets still hit threshold before uncued targets because the cue elicited activity has not yet dispersed, leading to faster SRTs for cued than for uncued targets (reprinted from Fecteau & Munoz, 2006). (B) Neurophysiological firing rates over time when CTOA = 200 ms. The cue elicited activity is transient and has nearly dispersed when targets appear. Due to sensory STD, target elicited exogenous inputs are reduced when cued, so the activity elicited by an uncued target hits threshold first, leading to faster SRTs for uncued targets (reprinted from Fecteau & Munoz, 2006). (C) and (D) Simulated node activity over time when CTOA = 50 ms and 200 ms.

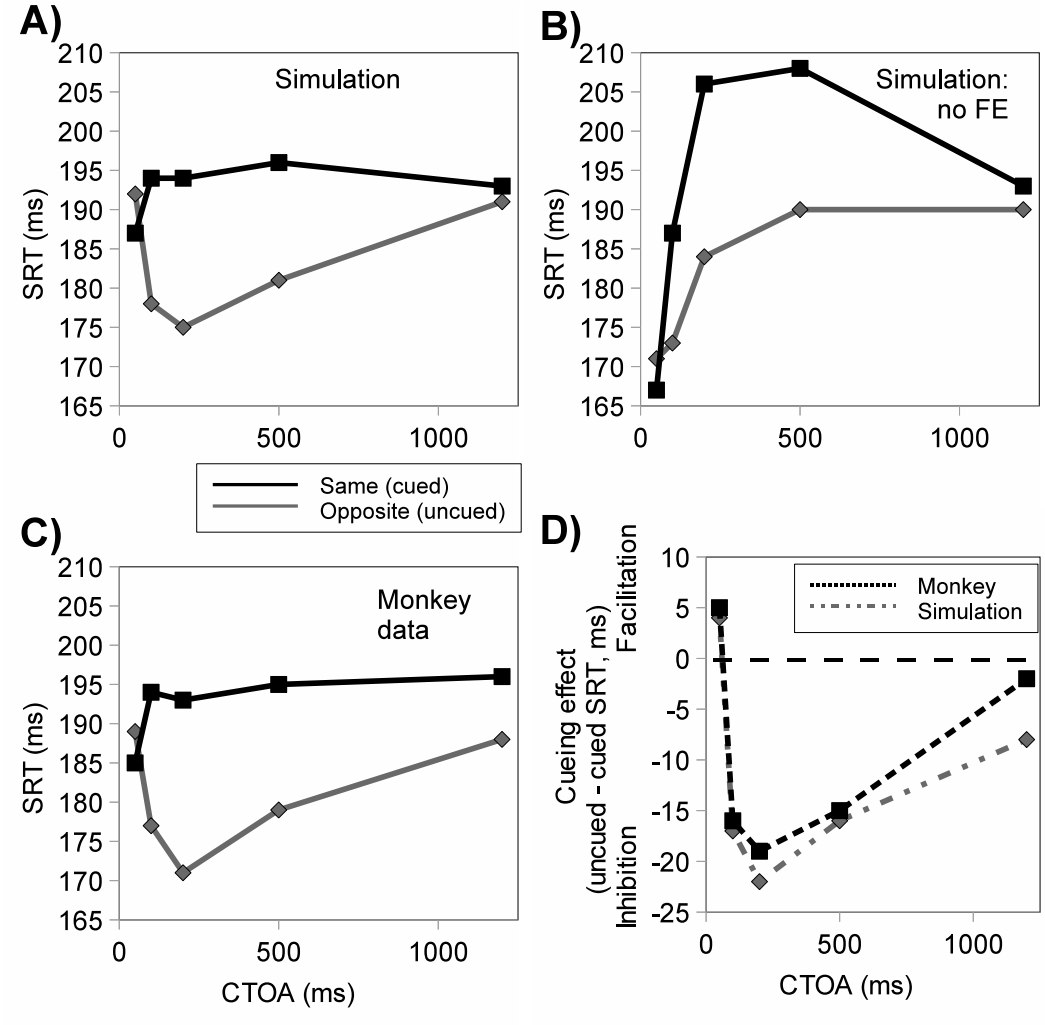

Figure 2.3: (A) Simulated SRTs for cued and uncued targets at various CTOAs, with the inclusion of a foreperiod effect (FE), which modulates endogenous move signal strength to account for temporal predictability. At CTOAs less than 200 ms, endogenous move signals are reduced (relative to simulations with no FE), slowing SRTs. At longer CTOAs, move signal strength is increased, leading to faster SRTs relative to simulations with no FE effect. (B) Simulated SRTs for cued and uncued targets at various CTOAs without temporal predictability. (C) Monkey SRTs for cued and uncued targets at various CTOAs (replotted from Fecteau and Munoz, 2005). (D) Simulated and monkey data (replotted from Fecteau and Munoz, 2005) illustrating cueing effects, which are calculated as uncued SRT - cued SRT, such that positive values indicate behavioral facilitation, and negative values indicate IOR.

2.4.1 Early benefits and subsequent costs following uninformative peripheral cues

When using the model cue-target IOR task, subjects often exhibit behavioral benefits (faster RTs) to cued targets at short CTOAs (Posner & Cohen, 1984). It has been proposed that attentional capture by a cue results in a brief period of enhanced processing in the vicinity of the cue, as if, during this period, the "effective contrast" (Reynolds & Chelazzi, 2004, p. 15) of stimuli presented there is increased. In our model, this phenomenon occurs, despite the reduced signal strength due to STD, as a result of the summation of cue and target-elicited neuronal activity (for similar explanations, see Bell, Fecteau, & Munoz, 2004; Dukewich, 2009). Such an activity summation process for a cued target is illustrated in the simulated node activity seen in Figure 2.2C, as well as the monkey neurophysiological data seen in Figure 2.2A (Fecteau & Munoz, 2006). Because neuronal activity elicited by exogenous inputs is transient, this behavioral benefit disappears when CTOA is increased.

When CTOA is larger than about 50 ms in monkeys, or 100 ms in humans, this behavioral benefit, as measured with SRTs, reverses into a behavioral cost (ie., IOR; Klein, 2004b). Note that we are exploring IOR using saccadic responses. It is well-known that the cross-over from benefits to costs at the cued location is quite a bit earlier when IOR is explored with saccades than when it is explored with manual responses (e.g. Briand, Larrison & Sereno, 2000; for a review, see Klein, 2004b). In our model, this later inhibitory effect is implemented in the input domain (ie., sensory STD). Once an exogenous input reaches the iSC, the amplitude, or strength, of subsequent exogenous inputs to the same iSC location is reduced for a specified period of time (see Figure

2.1C). This reduction of cued target related input strength has been demonstrated neurophysiologically in the monkey iSC (see Figure 2.2B; Dorris et al., 2002; Fecteau & Munoz, 2005), as well as the sSC (which receives only early sensory inputs; see Figure 2.1C; Fecteau & Munoz, 2005). Figure 2.2D demonstrates that the model closely reproduces neurophysiological results at a CTOA of 200 ms. Other CTOAs were also simulated and compared to behavioral data (see Figure 2.3), demonstrating that our model successfully reproduced monkey data at a number of CTOAs in a cue-target experimental paradigm.

2.4.2 IOR and predictive cueing

In a typical cue-target paradigm, the cue is uninformative. Early studies (e.g., Posner & Cohen, 1984) showed that the observed IOR effect disappears when the cues are predictive. A recent neurophysiological study (Bell & Munoz, 2008) sheds some light on this interesting observation. The experimental setup of this study was identical to the previously described cue-target paradigm, except that the target appeared at the cued location in 80% of the trials. Bell and Munoz (2008) found that when monkeys learned how to use the cue to predict target locations, behavioral IOR disappeared and facilitation was observed at long CTOAs (see Figure 2.4C). As shown in Figure 2.4A, this observation was accompanied by a pre-target buildup only for cued targets in cell recordings. This suggests that predictive, endogenous information reaches the iSC, bringing the neural activity of the expected iSC location closer to threshold before target appearance. Although the input strength for cued targets is reduced, due to sensory STD, the cued target cell still reached threshold first, leading to faster SRTs for cued targets at

relatively long CTOAs (e.g., 650 ms; see Figure 2.4A)

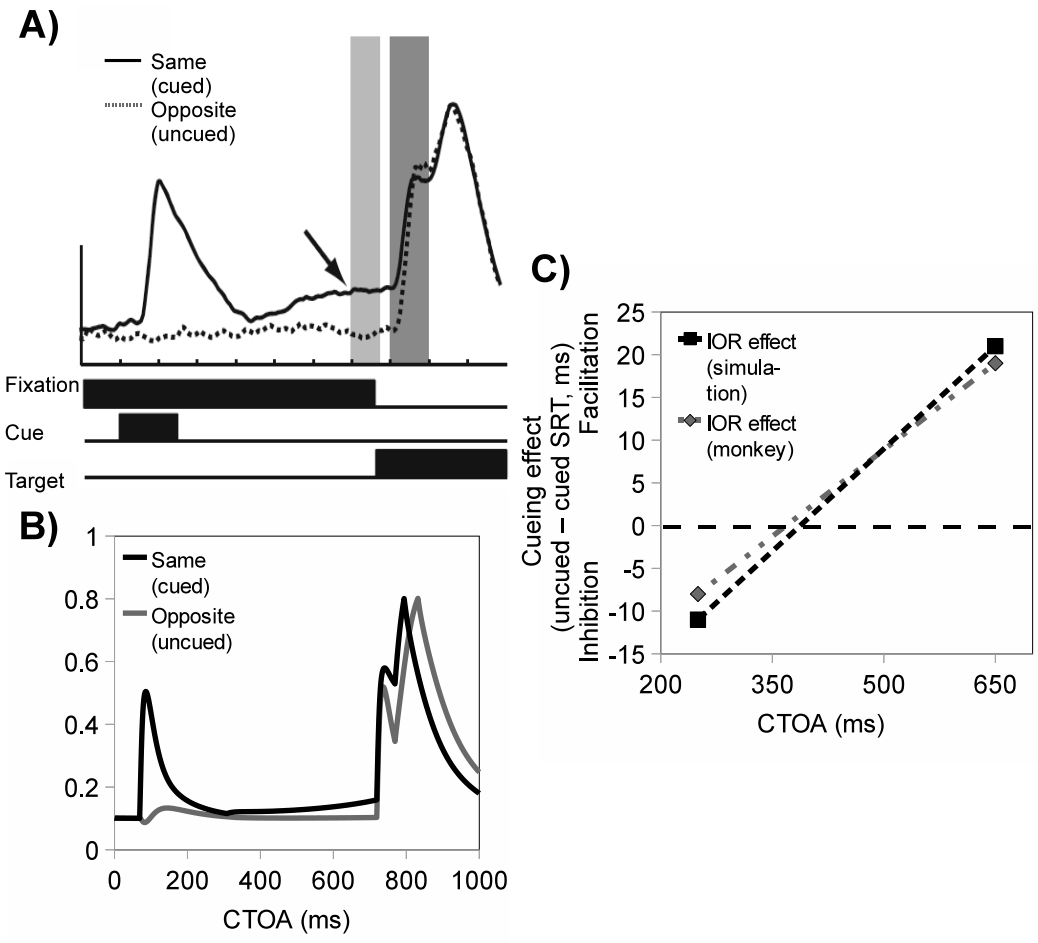

Figure 2.4: (A) Neurophysiological firing rates over time when CTOA = 650 ms in a predictive cueing paradigm (reprinted from Bell and Munoz (2008)). The arrow indicates that target cell activity is increased during the pre-target period (light gray bar time period), due to the predictive nature of the cue. The darker gray bar (post-target period) indicates the time period when target-elicited inputs arrive at the iSC, and demonstrates that cued inputs are reduced in strength as compared to uncued inputs. Even though cued target related inputs are reduced (STD), cued responses are still faster than uncued responses, due to the overwhelming strength of the top-down, predictive, cue elicited input, which builds up during the cue-target interval. (B) Corresponding simulation of node activity over time when CTOA = 650 ms. (C) Simulated and monkey data (replotted from Bell and Munoz, 2008) illustrating cueing effects (uncued SRT - cued SRT) in a predictive cueing paradigm.

To capture this finding in our simulations, shortly after cue onset a small, endogenous input that builds up slowly over time was transmitted to the cued iSC location (see Figure 2.4B). This implementation is also justified by other studies which have demonstrated that increases in target elicited activity during the pre-target period can be linked to top-down processes (Dorris & Munoz, 1998; Fecteau, Bell, & Munoz, 2004; Ignashchenkova, Dicke, Haarmeier, & Thier, 2004).

Simulated and monkey behavioral results (Bell & Munoz, 2008) are compared in Figure 2.4C, where IOR is still exhibited at a relatively short CTOA (250 ms), but is eliminated behaviorally at a longer CTOA (650 ms). This nicely demonstrates that there are experimental conditions for which the underlying mechanisms of IOR may be occurring, even though IOR is not exhibited behaviorally, due to the competition with top-down, endogenous inputs.

2.4.3 Saccadic averaging and IOR

When participants make a quick saccade to one of two stimuli that are presented simultaneously and in close spatial proximity, a first saccade is often directed to an intermediate location between these two stimuli. This phenomenon has been termed saccadic averaging (Ottes, Van Gisbergen, & Eggermont, 1984), and has been previously investigated with DNF modeling techniques (Wilimzig et al., 2006). Particularly in the presence of distractors that are nearby targets, saccadic curvature has also been observed either with saccades launched in the direction of the distractor and arriving at the target or with saccades arriving at the target on a curved path initially biased away from the distractor's location (e.g., Arai, McPeek, & Keller, 2004; Theeuwes, Kramer, Hahn, &

Irwin, 1998). Because our model of the iSC generates a saccade to the "winning" location when a threshold level of activation is exceeded, it cannot generate or predict curvature. Indeed, like Arai & Keller (2005), we believe that curvature is generated downstream from the iSC, perhaps at the level of the brainstem where signals from from the iSC and frontal eye fields converge.

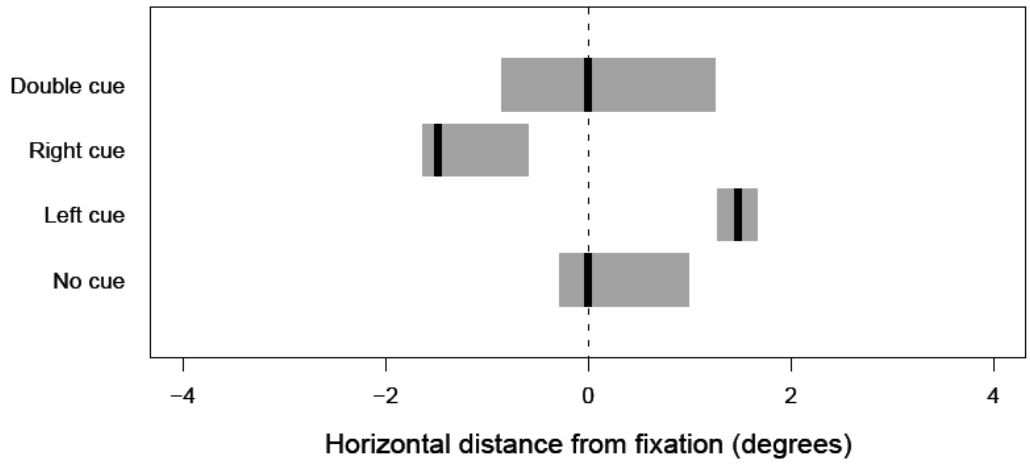

Figure 2.5: Saccadic averaging and IOR. When two targets are presented simultaneously, the majority of saccades land at the mid-point between both targets (no cue and double cue conditions). If one of the potential target locations is pre-cued (left and right cue conditions), saccades are biased toward the opposite location, due to the influence of IOR at the cued location. Gray bars denote the 95% confidence intervals of the mean landing positions in each condition calculated from the behavioral results reported in Watanabe (2001). Black lines denote the simulated mean landing position in each condition, obtained in our model without including any noise.

Watanabe (2001) further demonstrated that IOR interacts with saccadic averaging. When two identical visual targets were presented closely and simultaneously in the peripheral visual field, most saccades showed a tendency to land near the mid-point between the two targets (see Figure 2.5; gray bars show the 95% confidence intervals of

the mean landing positions observed in Watanabe, 2001), replicating previous work (Ottes et al., 1984). However, when a non-predictive visual cue was presented 600 ms before target appearance, saccades were biased away from the cued location (see Figure 2.5). This later finding was explained in terms of IOR biasing the average saccade away from previously cued (attended) locations (Watanabe, 2001). Note that behavioral results (Watanabe, 2001) tend to show a rightward bias in averaging saccades, although this bias is not incorporated into our model.

Saccadic averaging occurs due to the proximal locations of the two target stimuli (Watanabe, 2001; see also Chou, Sommer, & Schiller, 1999). In our model, when two closely located nodes are equally stimulated, due to the dynamic interaction of the iSC, neurons located around the middle of the two stimulated nodes will eventually become the most excited nodes, reaching the saccade initiation threshold first, and resulting in saccades landing in the middle of the two target locations. However, when one of the targets is cued, due to sensory STD, the actual visual input for the cued target will be reduced. That is, in our model, in the cue-double-target paradigm, two spatially proximal nodes will receive unbalanced inputs. Although these two inputs merge into one activity packet, the peak activity will be located closer to the nodes which received stronger input (ie., the uncued target). Note that saccadic averaging will not necessarily occur in all experimental paradigms. For example, if target locations are far enough apart, there will be a single winner, rather than an averaged landing point (for a more detailed investigation of saccadic averaging using DNF modeling, see Wilimzig et al., 2006). Some authors (e.g., Arai & Keller, 2005) have suggested that the iSC may not be

characterized by a Mexican-hat lateral interaction profile and that saccadic averaging behavior may be the result of downstream (brainstem) processes rather than merging bubbles in the iSC. However, these issues are still unresolved empirically, and we believe, as described above, that there is sufficient evidence for our theoretical interpretation.

Simulations were performed to demonstrate saccadic averaging (without cueing and with double cues), as well as how IOR interacts with saccade averaging (as in Watanabe, 2001; one of the targets was cued). In each simulation, transient exogenous inputs representing the appearance of cue and target stimuli were presented to the network with amplitudes of $d = 60$, widths of $\sigma_d = 0.45$ and sustained endogenous inputs which represent the move signal had amplitudes of $d = 10$. The distance between the two target locations was set to 5 degrees (as in Watanabe, 2001) and the CTOA was set to 600 ms since IOR was observed in both previous behavioral studies and our simulations with this CTOA (see Figures 2.1 and 2.3). The purpose of these simulations was not to precisely reproduce behavioral findings, but rather to demonstrate how IOR interacts with saccadic averaging (Watanabe, 2001). The parameters of our model, such as the noise level, could be modified to replicate the landing position distribution behavioral observations in Watanabe (2001).

The mean center of gravity of the saccade landing locations in all simulations are plotted in Figure 2.5 (solid black lines). It is clear from these results that saccades tend to land around the mid-point between two targets when no cues, or double cues are presented, reproducing previous behavioral results (as seen in Figure 2.5; simulation means are all within the 95% confidence intervals of behavioral results). When one of the

targets is cued, both real saccades, and those of our simulations (see Figure 2.5), tend to be biased away from the mid-line toward the uncued target, reflecting the effects of IOR on the cued location. These results clearly demonstrate that the interaction structure of the iSC is important to the behavioral exhibition of IOR, since saccadic averaging (which depends on the lateral interaction of the iSC) interacts with IOR, and can be explained with the sensory STD hypothesis of IOR.

2.5 Discussion

Simulations of a cue-target saccadic IOR paradigm revealed that typical behavioral benefits (faster SRTs) at short CTOAs and IOR (slower SRTs) at long CTOAs can be reproduced with a DNF model via a simple process of short-term plasticity of previously cued exogenous input signals. Based on previous work, the present exploration used a DNF model of the iSC (Trappenberg et al., 2001) along with the assumption of STD to simulate various findings concerning IOR. Neurophysiological studies have demonstrated that the SC, a key structure in the oculomotor system, is intimately involved with the generation of IOR. The sSC receives only early sensory inputs, while the iSC also receives inputs from multiple higher level brain constructs and is a structure which integrates bottom-up (exogenous) and top-down (endogenous) input signals.

These findings suggest that, with repeated stimulation, sensory input to the iSC will be reduced. To reflect this fact, our model reduces target-related exogenous input strengths when they are preceded by a cue at the same location. Thus, for a period of time following peripheral cues, subsequent exogenous inputs to cued locations are reduced in strength. With this simple manipulation, our model reproduces the prototypical

experimental effects in the Posner cueing paradigm. Capture of attention, as manifested by shortened SRTs to cued, relative to uncued targets, was observed at short CTOAs, and IOR, characterized by longer SRTs to cued than to uncued targets, was observed at longer CTOAs.

To further test the robustness of this model, additional simulations were performed for a Posner cueing task with predictive cues (Bell & Munoz, 2008) and a saccadic averaging task (Watanabe, 2001). Results of both simulations fit nicely to the empirical data. The predictive cueing simulations demonstrated that competition with top-down, endogenous inputs can sometimes lead to the elimination of behaviorally exhibited IOR, even though the underlying exogenous input STD process is still occurring (as was reported by Robinson & Kertzman, 1995, in a monkey study using manual responses). The IOR effects seen in these simulations are driven by the sensory STD mechanism. A third set of simulations, using a double-target paradigm, demonstrated that the dynamics of the lateral interaction profile in the iSC is an important component of IOR. This neurocomputational approach provides an avenue to examine the degree to which different theories of IOR can, and cannot, be supported by existing evidence.

2.5.1 Mechanisms underlying behaviorally exhibited IOR

There has been extensive discussion regarding the underlying neurodynamics and the stages of cognitive processing involved in IOR, particularly regarding the relative contributions of early sensory and later attentional processes (Klein, 2000; Klein, 2004b). In most cases, different theories are exclusive, in the sense that each particular theory proposes a process that attempts to explain all of IOR, without allowing for the possibility

of other independent processes contributing to the phenomenon. A number of researchers have proposed that IOR is associated with relatively late attentional processes in neocortical areas of the brain (Godijn & Theeuwes, 2002; Ivanoff & Klein, 2001; Klein & Taylor, 1994; Rafal et al., 1989; Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987). These ideas seem to have been strongly influenced by early observations of behaviorally exhibited IOR in experimental paradigms that ensure little or no SC involvement (e.g., Tipper, Driver, & Weaver, 1991; Tipper, Weaver, & Watson, 1996). Other researchers have suggested that IOR is the result of early sensory processes, as demonstrated by decreased early sensory signals associated with validly cued IOR trials (Fecteau & Munoz, 2005; Hopfinger & Mangun, 2001; Ivanoff & Klein, 2006; Posner & Cohen, 1984; Prime & Ward, 2004; Reuter-Lorenz, Jha, & Rosenquist, 1996). Early behavioral studies also demonstrated that IOR did not follow voluntary shifts of attention without peripheral stimulation (Posner & Cohen, 1984; Rafal et al., 1989), suggesting that what generates IOR is peripheral stimulation, oculomotor activation, or both (Klein, 2004b). The involvement of early sensory processes in the generation of IOR was further supported by recent electroencephalographic (EEG) studies (for a review, see Prime & Ward, 2006). Several EEG studies have shown that behavioral IOR was accompanied by an amplitude reduction of the early visual P1 component. More importantly, neurophysiological investigations have found that both visual neurons located in the sSC (which only receives early sensory inputs), and visuomotor neurons in the iSC (which control the initiation of saccades), show reduced activation to cued targets in typical IOR tasks (Dorris et al., 2002; Fecteau & Munoz, 2005). It has further been shown that cells in

the iSC are not directly inhibited on validly cued trials, but receive reduced target-related inputs (Dorris et al., 2002; Fecteau & Munoz, 2005). The amount of this reduction in signal amplitude is a function of CTOA, as demonstrated through single unit recordings of the sSC and iSC (Fecteau & Munoz, 2005).

Based on this evidence, as well as other results in the literature (Hopfinger & Mangun, 1998; Posner & Cohen, 1984; Prime & Ward, 2004; Reuter-Lorenz et al., 1996), some researchers have proposed that IOR is related to habituation, or short-term plasticity, of early sensory inputs to the iSC (Bell, Corneil, Munoz, & Meredith, 2003; Fecteau & Munoz, 2005; Huber, 2008). An expansion of this idea, which has recently been put forward by (Dukewich, 2009), suggests that IOR can be explained in terms of habituation-like processes at multiple levels of processing, providing a theoretical framework that could perhaps explain all results in the literature. With these findings in mind, we kept our model of the iSC relatively simple, such that target-related exogenous visual inputs are reduced whenever the target has been previously cued. Although this may be a coarse approximation of the complex underlying neural processes of IOR, the model successfully reproduced the prototypical cueing effects, as well as the findings of a study investigating saccadic averaging of IOR (Watanabe, 2001), and a predictive cueing paradigm demonstrating the simultaneous presence of IOR and endogenous attention at the same location (Bell & Munoz, 2008).

2.5.2 Cortically-based IOR

A number of experiments have demonstrated IOR in cases with little or no SC involvement (Sumner, Nachev, Vora, Husain, & Kennard, 2004; Tipper et al., 1991;

Tipper et al., 1996), or with endogenous signals (e.g., Taylor & Klein, 2000). Results from these studies indicate that there may be additional, cortical processes contributing to IOR, in addition to sensory STD. A potential explanation of these results is that additional habituation-like processes could occur at, or be propagated to, multiple levels of processing (Dukewich, 2009; Fecteau & Munoz, 2005). For example, STD of inputs to saliency maps with environmental or object-based coordinates in posterior parietal cortex (PPC) may be able to explain certain experimental results (Tipper et al., 1991; Tipper et al., 1996) that the current implementation cannot. This proposal could be examined in future computational work by extending the current model to include more detailed, dynamic, endogenous input modulation. A modular implementation could also be developed with the inclusion of multiple dynamic networks representing different areas of the brain involved in orienting responses, including, for example, sSC, PPC, the frontal and supplementary eye fields, and prefrontal cortex.

2.5.3 Predictions

One value of an explicit theoretical model is that it can be used to generate new behavioral predictions; predictions not already tested in the model generation process. A further benefit of a neuroscientifically-founded model that generates behavior using a dynamic neural field, is that it can also generate predictions about neural behavior.

Prediction 1: *In a traditional cue-target experimental paradigm, early sensory target-related signals throughout the brain will be decreased in strength when the target location has been cued.*

If the STD hypothesis of IOR is correct, then spatially cueing a target will reduce

the strength of target-elicited signals (relative to uncued target stimuli) in various areas of the brain, including, for example, PPC, frontal eye fields, and striate cortex. Although evidence has shown that target-elicited signals to the iSC are reduced following cues at the same spatial location, it is still unclear where these input attenuations are occurring. It is likely that habituated retinotectal synapses are causing the input reduction, but it is also possible that the effect is occurring in the pathway from striate cortex, the frontal eye fields, or even posterior parietal cortex, or is simply the result of direct inhibition in one of these areas. Empirical investigations should test this prediction using neurophysiological techniques on monkeys, and brain imaging techniques on humans. Such empirical results could help to more accurately simulate the different inputs in our model.

Prediction 2: *In a cue-target experimental paradigm with multiple cues or distractors at the same location, target-elicited input strengths will be further reduced in strength due to summation or interaction of multiple STD processes at the same spatial location, leading to increased behavioral IOR.*

Empirical work should examine the degree of STD involved in different regions after multiple cues have been presented, since repeated stimulations are likely to further reduce the strength of exogenous signals. However, it is possible that an asymptote is reached and that subsequent stimulations do not have an additive effect, or that multiple stimulations interact in an unexpected way. Using manual responses, Dukewich and Boehnke (2008) tested this prediction with positive results. Further behavioral and neurophysiological investigations should be undertaken to elucidate this issue.

Prediction 3: *In a cue-target experimental paradigm with simultaneous distractors presented at different locations at the same time as target appearance, behavioral IOR will be increased.*

Since the total amount of activity in the iSC always remains constant, presenting additional distractor-elicited inputs to the network at the time of target onset will lead to a reduction of baseline neural activity at target locations. In our model, this will lead to slower SRTs due to the additional time required for target nodes to reach threshold, and a resulting increase in the amount of behaviorally exhibited IOR.

Prediction 4: *Varying the psychophysical properties of the target itself will lead to a similar effect, with brighter targets producing more behavioral IOR.*

Since STD is implemented in our model as a percentage reduction of target-elicited elicited input strength, based on the time since cue presentation, smaller target inputs will lead to less of an input reduction associated with STD, and less behaviorally exhibited IOR. Similarly, if the simulated target-elicited input strength is increased due to being larger or brighter empirically, this larger target-elicited input will lead to more behaviorally exhibited IOR.

Prediction 5: *In a cue-double-target experimental paradigm at short CTOAs, saccades will tend to be biased toward the cued location.*

The current simulations and the behavioral study of Watanabe (2001) demonstrated that saccades tend to be biased away from a cued location at a CTOA long enough to generate behavioral IOR. This is due to the interaction of IOR with the dynamic lateral interaction of the iSC which causes saccadic averaging in some

conditions. At shorter CTOAs known to cause behavioral facilitation, our model predicts that saccades will be biased toward the cued location, since the cue elicited exogenous input in the SC has not yet completely decayed, and consequently the cued target node will reach threshold and initiate a saccade before the uncued target node.

Prediction 6: *Behavioral IOR will not be observed in some experimental manipulations, even though the STD mechanism underlying IOR is still present, due to competition with top-down, endogenous signals.*

A particularly interesting simulation in the present paper, which examines the interaction of exogenous and endogenous signals, is the traditional Posner cueing task with predictive cues. In Bell and Munoz (2008), the Posner task with predictive cues was tested with monkeys. When monkeys learned how to use the predictive cueing information, they showed an IOR effect at short CTOAs, but not at longer CTOAs. Importantly, a slow buildup of target-related activity following the cue was observed at the longer CTOA. Because top-down inputs from higher level brain constructs are not well understood, this predictive cue was implemented in our model as a sustained endogenous input at the cued location. With this manipulation, the buildup of target-related activity was reproduced in our model and the IOR effect was observed at short CTOAs and disappeared at longer CTOAs. This finding nicely demonstrates that the competition between the bottom-up and top-down inputs at the level of the SC is an important factor that determines whether the IOR effect is observed behaviorally.

It should be mentioned that such top-down modulation of IOR has long been discussed and explored by IOR scholars. In a review of IOR (Klein, 2000), such top-

down modulation was referred to as an attentional control setting (ACS). The ACS theory states that the deployment of attention (both spatial and temporal) depends on the cognitive task requirements. In the Posner task with predictive cues, ACS would predict that more attentional resources would be placed at the cued location following a cue. Such endogenous input would summate with the target related input, bringing the cell activity to threshold more quickly. As demonstrated in Fecteau and Munoz (2005), target related exogenous inputs are reduced because the target location has been previously stimulated by the cue, due to STD. However, such STD reaches its maximum effect shortly after the onset of the cue and then decays over time. As a result, at short CTOAs, while the STD is still strong, endogenous inputs will not cancel all the effects caused by the STD. However, at longer CTOAs, when the STD process has further decayed, the behavioral observation of IOR disappears.

2.6 Conclusion

The current simulation results quantify the hypothesis that IOR is associated with habituation, or STD, of early, sensory, target related, exogenous, input signals that reach the iSC (Bell et al., 2003; Dorris et al., 2002; Fecteau & Munoz, 2005; Fecteau & Munoz, 2006). When considered along with the extensive neurophysiological data (Bell et al., 2003; Dorris et al., 2002; Fecteau & Munoz, 2005), as well as other results in the literature (Hopfinger & Mangun, 2001; Posner & Cohen, 1984; Prime & Ward, 2004; Reuter-Lorenz et al., 1996), these results strongly suggest that there is a process of short-term plasticity that occurs after presentation of exogenous stimuli, which contributes to behaviorally exhibited IOR. Furthermore, this process of early sensory habituation may

be dissociable from other potential sources of contribution to behaviorally exhibited IOR (Fecteau & Munoz, 2005).

Behavioral observation of IOR is likely the result of a combination of multiple, independent, dissociable processes. Habituation, or STD, of early sensory signals clearly contributes to this inhibitory phenomenon to a great degree, particularly when oculomotor neural machinery is activated. Further empirical investigations to examine the precise temporal dynamics of early sensory signal habituation should be pursued. It has also been proposed that additional inhibitory processes in cortical regions may contribute to IOR. One possible neural implementation of this theory is the involvement of habituation-like processes in other cortical areas related to attentional orienting. Neurophysiological and behavioral experiments should be designed to test this possibility. Future computational work will examine this issue in more depth through the modular incorporation of dynamic input modulation.

2.7 Acknowledgements

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2.8 References

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CHAPTER 3 AFTEREFFECTS OF SACCADES EXPLORED IN A DYNAMIC NEURAL FIELD MODEL OF THE SUPERIOR COLLICULUS

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3.1 Abstract

When viewing a scene or searching for a target, an observer usually makes a series of saccades that quickly shift the orientation of the eyes. The present study explored how one saccade affects subsequent saccades within a dynamic neural field model of the superior colliculus (SC). The SC contains a oculocentric motor map that encodes the vector of saccades and remaps to the new fixation location after each saccade. Our simulations demonstrated that the observation that saccades which reverse their vectors are slower to initiate than those which repeat vectors can be explained by the aforementioned remapping process and the internal dynamics of the SC. How this finding connects to the study of inhibition of return is discussed and suggestions for future studies are presented.

3.2 Introduction

When viewing a scene or searching for a target, an observer makes a series of rapid eye movements (saccades) interspersed by short intervals during which the eyes remain still. While awake, humans, on average, make 3-4 saccades per second. This unique eye movement behavior is ecologically important as it shifts the orientation of the eyes and brings visual information to the most sensitive part of the eye (i.e., fovea) for detailed processing. Previous behavioral, neurophysiological and computational efforts have greatly advanced our understanding of the underlying mechanisms of saccade generation. In the present study, we asked a slightly different theoretical question. Namely, how does one saccade affect the generation of subsequent saccades, or, similarly, what are the after effects of saccades? This question was explored within a dynamic

neural field (DNF) model of the superior colliculus (SC), which is a key component of the oculomotor system.

3.2.1 Saccades and the superior colliculus

The control of saccades involves a complex collection of brain areas, including the parietal and frontal cortices, basal ganglia, thalamus, SC, cerebellum, and brainstem reticular formation (Munoz & Fecteau, 2002). The SC is especially important in controlling eye movements, partly because it receives inputs from both the outside visual world and higher brain areas, making it a perfect candidate for studying how bottom-up (exogenous) and top-down (endogenous) inputs interact in saccade programming (Trappenberg, Dorris, Munoz, & Klein, 2001).

Single-unit recording studies have shown that the intermediate layer of each superior colliculus (SCi) contains a motor map that encodes the direction and amplitude of saccades into the contralateral visual field (e.g., Robinson, 1972). In this motor map, two types of neurons are known to play a critical role in the generation of saccades. Fixation neurons, located at the rostral pole of each colliculus, discharge tonically during active fixation and cease discharge shortly before saccade onset (Munoz & Wurtz, 1993). Buildup neurons, responsible for saccade preparation, are located more caudally and have a long-lead discharge increase before saccades (Munoz & Wurtz, 1995a; 1995b). These saccade-related SCi neurons are connected in such a manner that proximal neurons excite each other and distal neurons inhibit each other (for a review, see Munoz & Fecteau, 2002). This laterally connected motor map acts in a “winner-take-all” fashion, with competition between different inputs resulting in the initiation of a saccade to the

response field of a winning node. Before any input (exogenous or endogenous) reaches the SCi (i.e., the eyes are maintaining active fixation), fixation neurons at the rostral pole discharge tonically, whereas the caudal areas of both colliculi remain "silent". When any input arrives at the caudal area of the SCi, neuronal activity at the excited sites starts to increase and fixation neuron activity begins to decrease. When sufficient input has arrived, the excited caudal site (buildup neurons) will eventually dominate the map and shut down the rest of the map, including fixation neurons. When this activity crosses a particular activity threshold, a saccade is initiated through an output signal to the brainstem reticular formation (Munoz & Fecteau, 2002).

3.2.2 Computational explorations of saccade initiation

Several computational approaches have been used to explore various saccade-related behaviors (e.g., Findlay & Walker, 1999; Kopecz & Schöner, 1995). We believe the most fruitful theoretical approach connects neuronal and behavioral findings in a computationally explicit model implemented in terms of networks of artificial neurons. One such technique is the dynamic neural field (DNF) modeling approach (e.g., Amari, 1977; Wilson & Cowan, 1973). This model captures the lateral interaction in the SC and has been successfully used to explore various saccade-related behaviors in a variety of experimental paradigms (Arai, Keller, & Edelman, 1994; Das, Keller, & Arai, 1996; Kopecz, 1995; Kopecz & Schöner, 1995; Meeter, Van der Stigchel, & Theeuwes, 2010; Trappenberg et al., 2001; Wilimzig, Schneider, & Schoener, 2006). Using a behavioral distractor paradigm and monkey single-unit recording data, Trappenberg et al. (2001) parameterized the lateral interaction structure of the monkey SCi. With a Mexican-hat

shaped interaction kernel, the authors effectively reproduced not only cell recordings, but also behavioral performance data (e.g., saccadic reaction times, SRTs) in various experimental paradigms. We chose to use this model in the present study because: a) the lateral interaction kernel is constrained by neurophysiological data; b) it maintains a good balance between simplicity and theoretical explicitness; c) it is capable of reproducing and making predictions about both neuronal and behavioral data.

3.2.3 Why are return saccades slower to initiate than forward saccades

As mentioned earlier, the purpose of the present paper is to explore the after effects of saccades. More specifically, how does a given saccade affect the behavior of subsequent saccades? One such after effect that is frequently observed in the literature is that saccades which repeat previous vectors are faster to initiate than those which reverse vectors (Anderson, Yadav, & Carpenter, 2008; Dodd, Van der Stigchel, & Hollingworth, 2009; Hooge, Over, van Wezel, & Frens, 2005; Hooge & Frens, 2000; Klein & MacInnes, 1999; MacInnes & Klein, 2003; Smith & Henderson, 2009; for an exception, see Dorris, Taylor, Klein, & Munoz, 1999). For convenience, we will refer to saccades which repeat the vector of the immediately preceding saccade as "forward saccades" and those which reverse vector as "return saccades".

We propose an explicit theory that explains why forward saccades are faster to initiate than return saccades, simply on the basis of the "leftover" activity in the SCi associated with the immediately preceding saccade (as illustrated in Figure 3.1A). When the eyes are actively fixating a location in visual space (e.g., A in Figure 3.1A), fixation neurons at the rostral pole of the SCi (F') discharge tonically and take over the network.

To initiate a saccade to another spatial location (e.g., B in Figure 3.1A), inputs (which can be either exogenous or endogenous) arrive at neurons in the SCi representing this location (B' in Figure 3.1A). Shortly before a saccade to the new location (B) is initiated, the neuronal activity at B' in the SCi approaches, and eventually exceeds, the threshold for initiating a saccade. After the saccade is executed, neurons in the SCi are remapped to the new foveal location, which was the target of the saccade and is now represented by firing of fixation neurons at the rostral pole. Thus, the neurons in the SCi that originally drove the saccade (B' in Figure 3.1A), now represent a new spatial location (C in Figure 3.1A) which is, relative to the new fixation, in the same direction and of the same amplitude as the previous saccade. Although the discharge of fixation neurons at the rostral pole (F') starts to increase shortly before the saccade is completed, neuronal activity at B' does not die out immediately. In our model (see below), this leftover activity leads to asymmetric activation in the SCi and, as a result, saccades in the forward direction, particularly those with the same amplitude as the previous saccade, might be facilitated, while those directed back to the vicinity of the previous fixation location (reverse vector) might be impeded.

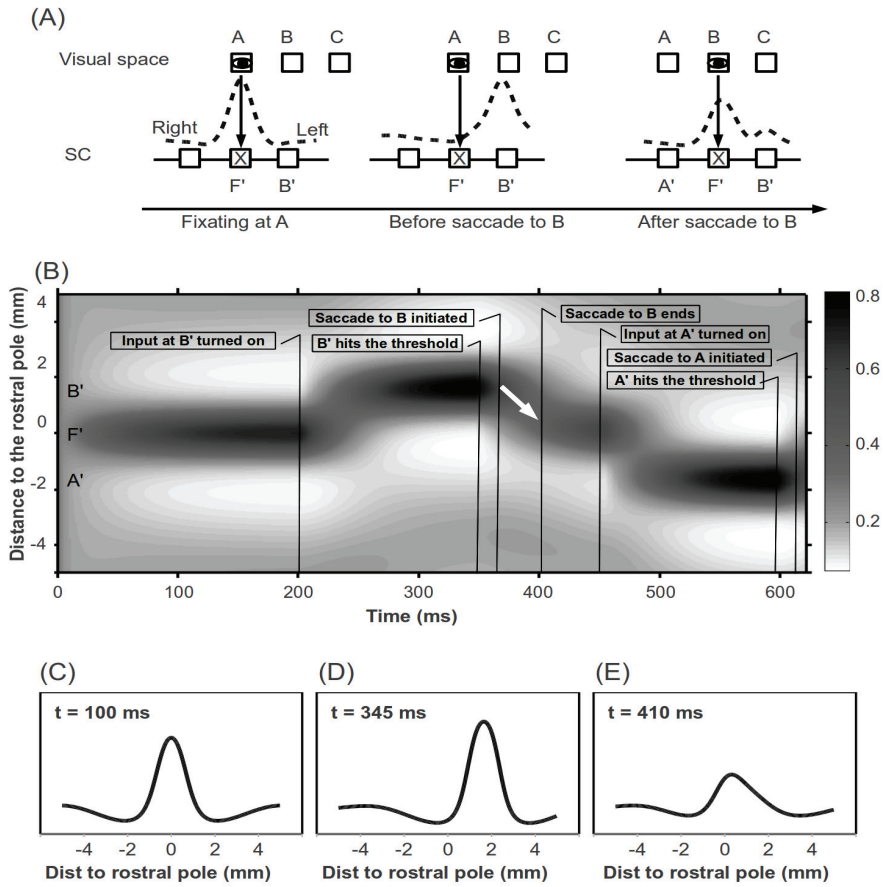

Figure 3.1: (A) Remapping of space in the SCi after a saccade results in asymmetric activation. Eyes mark the fixated spatial location. X's mark the rostral pole of the SCi. For convenience, the right colliculus is drawn on the left. (B) Illustration of a sample simulation trial. On the Y-axis, positive and negative values denote the right and left colliculi, respectively. The white arrow marks a hill of activity "moving" toward the rostral pole during the first saccade. (C), (D) and (E) Network activity during active fixation (t = 100 ms) shortly before a first saccade is initiated (t = 345 ms) and shortly after this saccade ends (t = 410 ms). The scales on the X axis denote distance from the rostral pole (mm). As clearly shown in (E), the SCi is asymmetrically activated shortly following a saccade, as illustrated in (A).

One may argue that the activity associated with a saccade decays so quickly that by the end of a saccade there should be little or no activity remaining at the activated SCi site. However, neurophysiological results (Munoz & Wurtz, 1995b) and our simulations demonstrate that this is not necessarily the case (see Figure 3.2A). The SCi is a "push-

pull" network; as pointed out by Munoz and Fecteau (2002), "the amount of activity expressed in the intermediate layers remains reasonably constant; with only the distribution of this activity changing. Therefore, if the activity of one node is strong, then the inhibition of distant nodes will be strong." (pp. 4-5). That is, the leftover activity associated with a saccade will lead to a peak and a trough. For saccades with repeat and reverse vectors, the inputs to the SCi will be at the peak and trough, respectively. This baseline difference will transfer into observable behavioral differences nonlinearly, if the time interval between saccades is short enough. The remainder of this paper will explore this theory computationally with simulations of various experimental paradigms.

Note that the present paper is not the first one to propose the above theory, similar idea has been expressed in Klein and MacInnes (1999): "Because oculomotor responses are likely initiated by a winner-take-all algorithm mediated by lateral inhibition (and implemented in the superior colliculus), any asymmetric preparation would result in inhibition of the least prepared saccades" (p. 351).

3.3 Model architecture

In our simulations, a 1-dimensional DNF model with parameters similar to previous work (Trappenberg et al., 2001) was used. In this model, $n=1001$ nodes were used to represent 5 mm of each colliculus, with nodes laterally connected in a manner such that proximal nodes excite each other while distal nodes inhibit each other. The connection strength, or weight (w_{ij}), between two nodes i and j was set with two Gaussians (Equation 3.1). The following parameters for internal connectivity were used in all simulations: $a = 72,\ b = 24,\ c = 6.4$, $\sigma_a = 0.6,\ \sigma_b = 1.8$. Although this lateral

interaction was chosen to approximate cell recordings in the SCi of monkeys in Trappenberg et al. (2001), it is not an exact fit to the physiology data.

$$w_{ij} = a * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_a^2}\right) - b * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_b^2}\right) - c \qquad \text{Equation 3.1}$$

$$\tau \frac{du_i(t)}{dt} = -du_i(t) + \sum_j w_{ij} r_j(t) \Delta x + I_i(t) + u_0 \qquad \text{Equation 3.2}$$

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)} \qquad \text{Equation 3.3}$$

$$I_k = d * \exp\left(\frac{((k-i)\Delta x)^2}{2\sigma_d^2}\right) \qquad \text{Equation 3.4}$$

The dynamics of the internal state $u_i(t)$ of node i is described in Equation 3.2, where $\tau = 10$ *ms*, is a time constant defining the rate of relaxation, w_{ij} is the connection strength (weight) between node i and node j, $r_j(t)$ is the activity level (average firing rate) of node j, $I_i(t)$ represents the external input to node i, and $u_0 = 0$ is a constant resting level. The activity of node i, $r_i(t)$, as a function of its internal state $u_i(t)$, is defined by a sigmoidal gain function (Equation 3.3), where $\beta = 0.07$ and $\theta = 0$ were used in our simulations.

The activity of buildup neurons in response to a visual stimulus is characterized by two peaks. The first peak represents the incoming visual input, which decays exponentially, and the second peak represents a sustained "move signal", presumably from higher cortical areas. These two distinct inputs were labeled exogenous and endogenous inputs in previous studies (e.g., Kopecz, 1995; Kopecz & Schöner, 1995; Trappenberg et al., 2001). This distinction was ignored in the present exploration because our theory is about the dynamics within the SC and our simulations do not depend on the

sources of inputs to the SC. Besides, in the case of free viewing or searching of a static scene, saccades are normally controlled voluntarily (i.e., initiated by endogenous inputs to the SC). Thus, sustained (endogenous-like) inputs are used in all of our simulations. These inputs are assumed to have a Gaussian spatial shape, centered at location *i*. As a consequence, the input to other nodes (*k*) in the network depends on the distance between *i* and *k*, as represented by Equation 3.4. Whenever the activity of a node reaches a threshold of 80% of its maximum firing rate, a saccade to its response field is initiated after a 20 ms efferent delay. In our simulations, the input strength (*d*) and the input width (σ_d) were varied between different experimental tasks.

The activity of fixations neurons is characterized by tonic discharge during active fixation, a pause during saccades, and reactivation shortly before a saccade ends (Munoz & Wurtz, 1993). An assumption made in our simulations was that the reactivation of fixation neurons is crucial for the maintenance of fixation at the saccade target location. Thus, in our simulations, input is fed to the fixation neurons whenever a buildup neuron in the caudal area reaches the saccade initiation threshold.

3.4 Simulations

3.4.1 Residual activity after saccades

One critical aspect of our theory is that it depends on how much activity remains at a SCi site when a saccade to its response field ends. As can be seen in Figure 3.1E, this residual activity and its decay rate determines how long the "asymmetric activation" in the SCi lasts. To determine the sensitivity of this residual activity to the amplitude of saccades, we simulated saccades with various amplitudes and recorded the activity level

of the associated nodes at the end of these saccades. Constant inputs to fixation neurons at the rostral pole ($a = 6, \sigma_d = 0.6$) and buildup neurons at various caudal sites ($d = 12, \sigma_d = 0.6$) were used. Saccade duration as a function of saccade amplitude was estimated with the following equation, *duration = 1.8*amplitude + 17* (Chu & Kaneko, 1995).

Our simulation predictions are presented along with neurophysiological data (Munoz & Wurtz, 1995b, Figure 9B) in Figure 3.2A. Normalized discharge level (activity remaining) at the end of a saccade was plotted against the amplitude of the saccade. As can be seen in this figure, more activity remains in the SCi following small, than following large, saccades. This is the result of two factors. First, node activity decays exponentially once external input to the network ceases. Because the duration of small saccades is shorter than large saccades, by the end of a saccade more activity will have decayed for large than for small saccades. Second, and more importantly, due to long-distance inhibition, the leftover activity associated with a large saccade is quickly inhibited by the fixation neuron activity at the rostral pole. However, due to short-distance excitation, the leftover activity associated with small saccades collaborates and merges with the fixation neuron activity and even drags the fixation activity toward itself (see Figure 3.1E). Consequently, the leftover activity has a larger and longer lasting effect on behavior following small saccades than following large saccades. That is, the mechanism we are proposing is relatively confined to smaller saccades. One might wonder how often this mechanism applies to real-world saccadic explorations of the environment. Given the fact that the amplitudes of normal saccades are Poisson, or exponentially, distributed (with means around 6° visual angle or less (e.g., Carpenter, 1988; Wartburg et al., 2007),

this mechanism will influence the initiation time of the majority of the saccades we make.

3.4.2 Simulation of behavioral findings

We further explored our theory by comparing our simulation results to behavioral findings. Our simulations were relatively straight forward; a sample trial in which a first saccade is followed by a return saccade is illustrated in Figure 3.1B. At the beginning of each trial, an input was given to the rostral pole fixation neurons (F' in Figure 3.1A) to maintain active fixation. Two hundred milliseconds later, an input was fed to a caudal site (B') to initiate a first saccade. At the same time, the input to fixation neurons was turned off. When activity at the excited caudal site (B') crossed the saccade initiation threshold, input to this site was turned off and input to fixation neurons (F') was switched back on, so that fixation would start at the end of a saccade. Under optimal conditions, this input change, together with the lateral interaction in the SCi, will result in what looks like a "hill of activity" moving toward the rostral pole (marked with a white arrow in Figure 3.1B; see the Appendix for a brief exploration of this phenomenon). After a 20 ms efferent delay, a saccade was initiated to the response field of the SC site which reached threshold (B' in Figure 3.1A) and its duration was estimated with the following equation: $duration = 2.2*amplitude + 21$ (Carpenter, 1988). Then, after various time intervals (0-100 ms), another input was fed to the symmetrically opposite site in the SCi (A' in Figure 3.1A) to initiate a return saccade. 20 ms after the activity at this site (A') reached threshold, a second saccade was initiated to the response field of this site (A in Figure 3.1A). The amplitude and direction of the first and second saccades, as well as the latency of the second saccade, were recorded for each trial for further analysis. Our simulations of

three behavioral experiments (Hooge & Frens, 2000, Experiment 2a; Klein & MacInnes, 1999, Experiment 1; Smith & Henderson, 2009, Experiment 1) are summarized below. In these simulations, inputs for fixation and buildup neurons were fixed at $d = 6$, $\sigma_d = 0.6$ and $d = 10.5$, $\sigma_d = 0.6$, respectively.

Hooge and Frens (2000): In Hooge and Frens (2000, Experiment 2a), participants were asked to saccade between three loci as quickly as possible (as illustrated in Figure 3.2B). They found a latency cost for saccades that reversed vectors, as compared to those that repeated vectors. This cost was attributed to "inhibition of saccade return". In our simulations of this behavioral experiment, two consecutive saccades with randomly selected directions (left or right) were simulated in each trial. For both directions, the saccade amplitude was fixed at 7.5°, as in Hooge and Frens (2000, Experiment 2a). The time interval between the termination of the first saccade and the onset of the second saccade, which was randomly selected to repeat vectors (forward saccade) or return to the initial fixation (return saccade), was randomized between 0 and 100 ms. The latency of the second saccade (fixation duration before the onset of the second saccade), as a function of its relative direction to the first saccade ("Return" or "Forward"), is plotted in Figure 3.2B along with the behavioral results from Hooge and Frens (2000, Experiment 2a).

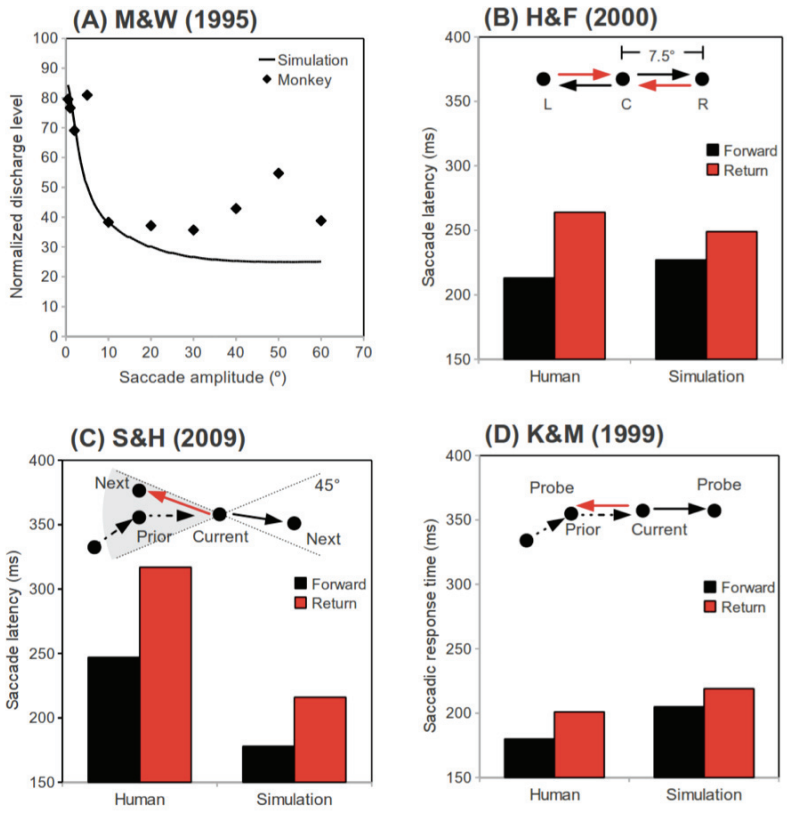

Figure 3.2: (A) Normalized discharge level at the end of saccades as a function of saccade amplitude. Monkey data is adapted from Munoz and Wurtz (1995a). (B) Behavioral data of a single participant from Hooge and Frens (2000, Experiment 2a) and associated simulation results. Saccades L→C and R→C are "Return" saccades, and saccades C→R and C→L are "Forward" saccades. (C) Behavioral data from Smith and Henderson (2009, Experiment 1) and associated simulation results. Only saccades with roughly the same amplitude as their preceding saccades are plotted. Saccades landing in a 45° binned region centered in the direction from the "Current" to the "Prior" fixation are "Return" saccades, while saccades landing in a 45° binned region centered in the direction from the "Prior" to the "Current" fixation are "Forward" saccades. (D) Behavioral data from Klein and MacInnes (1999, Experiment 1) and associated simulation results. While participants were searching for a camouflaged target, saccades were visually directed to the "Prior" fixation location ("Return" saccades), or a location on a circle defined by the "Current" and the "Prior" fixations, but at 180° (angular distance) from the "Prior" fixation ("Forward" saccades).

Smith and Henderson (2009). In Smith and Henderson (2009, Experiment 1), participants freely inspected photographic scenes while their eye movements were monitored. The freely made saccades were later analyzed to reveal how a saccade was

affected by its immediately preceding saccade. It was found that saccades that went back to the vicinity of their preceding fixation locations took longer to initiate than those which roughly repeated the vector of their preceding saccades, a finding that was attributed to "saccadic momentum." For simplicity, we compared our simulation results to only two data points from Smith and Henderson (2009, Figure 3.4): the 0° ("Return" saccades) and 180° ("Forward" saccades) data bins with 0° amplitude differences (see Figure 3.2C). Although saccade amplitudes were not reported in Smith and Henderson (2009), we used their reported image size (25.7° × 19.4°) to estimate the amplitudes based on the findings of von Wartburg et al. (2007). These amplitudes were characterized by an exponential distribution with a mean of 5.5°. Small amplitude (< 1°) and very large amplitude (> 30°) saccades were excluded from our simulations, because they were excluded from analysis in Smith and Henderson (2009), or were not made by their participants. In our simulations, the direction (left or right) and amplitude of the two consecutive saccades in each trial were randomized. Because we wanted to compare return and forward saccades with comparable amplitudes, only trials in which the two saccades had an amplitude difference of less than 1° were included in our analysis. The time interval between the end of the first saccade and the input onset of the second saccade was randomized between 0 and 100 ms. The simulation results, along with the behavioral data from Smith and Henderson (2009), are presented in Figure 3.2C.

Klein and MacInnes (1999): In Klein and MacInnes (1999), participant's eye movements were monitored on line while they searched for a camouflaged target. After a few saccades, a probe was presented at the immediately preceding fixation location, or at

one of 5 equi-eccentric novel locations, and a saccadic response was required (Experiment 1). Klein & MacInnes (1999) reported that saccades to probes at a previously fixated location took longer to initiate than saccade to probes at equieccentric locations that had not been fixated, a difference that they attributed to inhibition of return. Here, we have only simulated saccades to probes (exogenous) that landed at the last fixation location, or an equi-eccentric new location lying in the same direction of the last saccade (denoting the 180 ° condition by Klein and MacInnes, 1999). This restriction is necessary because our model is, so far, one-dimensional. A 2-dimensional version would be required to explore vector differences between successive saccades other than 0° and 180°. Partly because the search task was very difficult, saccade amplitudes were small in this experiment; re-analysis of Klein and MacInnes' (1999) raw data files revealed that the average amplitude for the last saccade before the probe was 2.8°. Thus, as in our simulations of Smith and Henderson (2009), the amplitudes were randomly drawn from an exponential distribution, with a mean of 2.8° in the present simulation. One consequence of the probe method used by Klein and MacInnes (1999) is that successive saccades in each trial of our simulations will necessarily have the same amplitude. In Klein and MacInnes (1999), the probe was presented about 20 ms after the last saccade, and neuroscientific data (e.g., Dorris, Pare, & Munoz, 1997) suggests that it would take about 70 ms for this visual input to reach the SCi. Thus, the time interval between the end of the first saccade and the input onset of the second saccade was fixed at 90 ms in our simulations. The simulation results along with the behavioral data from Klein and MacInnes (1999) are presented in Figure 3.2D.

Summary of behavioral simulation results: Our behavioral simulation results are compared to behavioral data in Figures 3.2B-D. As clearly shown in these figures, our simulations successfully reproduced the pattern of behavioral findings in Hooge and Frens (200), Smith and Henderson (2009) and Klein and MacInnes (1999). One might wonder how do the model accounts for the differences between behavioral findings and the simulation results. First and foremost, the purpose of these simulations is to demonstrate a theory (or a principle) other than to fit behavioral data. The present exploration did not fiddle with model parameters, even the input signal strength. Second, the SCi receives input from the retina, the primary visual cortex, and other cortical areas (e.g., FEF, LIP). Because the mechanism explored here is about the internal dynamics of the SC, the difference between these input sources was ignored in the simulations. Third, a 1-dimensional is used in the simulations; some variations in behavioral data which is collected in 2-dimentional space (e.g., Smith and Henderson, 2009) can not be captured by the model.

Only two critical parameters were varied in our simulation of behavioral experiments, namely, the amplitudes of the two consecutive saccades, and the input delay for the second saccade. The three behavioral studies were chosen to demonstrate how varying one, or both, of these parameters will produce virtually the same pattern of results. In our simulations of Hooge and Frens (2000), the amplitudes of the two saccades were fixed across trials, while the input delay for the second saccade was varied across trials. In the simulations of Klein and MacInnes (1999), the amplitude of the two saccades varied across trials, while the input delay for the second saccade was held relatively

constant. In the simulations of Smith and Henderson (2009), both parameters were varied. Despite these variations, the pattern of results observed in our simulations was very consistent, suggesting the findings in our simulations are robust. We did not vary the input strength in our simulations because our theory is about the internal dynamics of the SC and adding noise to the inputs will not change the pattern of results in our simulations. It is worth noting that with one relatively low-level mechanism we have simulated behavioral effects that have, in the literature, been attributed to three different underlying mechanisms: inhibition of saccade return, saccadic momentum and inhibition of return.

3.4.3 Spatio-temporal characteristics

In the previous section, we demonstrated that saccades which go back to their immediately preceding fixation locations are slower to initiate than those which repeat their vectors. How long will this behavioral effect last? What will happen if the two consecutive saccades differ in size? Because the leftover activity in the SCi following a saccade decays relatively quickly, it is reasonable to predict that the behavioral effect (i.e., return saccades being slower to initiate) will not last very long. In our simulations, the time interval between the end of the first saccade and the input onset of the second saccade (see Figure 3.1B) was varied between 20, 70 and 170 ms. For convenience, we will refer to this time interval as the "input delay of the second saccade." The amplitude of the first saccade was varied between 2°, 5°, 10°, 20° and 30°, and the amplitude of the second saccade was varied between 2°, 3°, 5°, 7°, 10°, 15°, 20°, 25° and 30°. As in previous simulations, inputs for fixation ($d = 6$, $\sigma_d = 0.6$) and buildup ($d = 10.5$, $\sigma_d = 0.6$) neurons were fixed in all trials. Our simulation findings are presented in Figure 3.3.

Several interesting findings are revealed in Figure 3.3. First, following small-sized saccades, small return saccades are slower to initiate, as compared to small forward saccades. However, large return saccades are faster to initiate as compared to large forward saccades. Second, following large saccades, the opposite pattern of results was obtained. That is, small return saccades are faster to initiate as compared to small forward saccades; large return saccades are slower to initiate as compared to large forward saccades. Third, these effects decay quickly as a function of the input delay of the second saccade. Further simulations showed that, regardless of the size of the first saccade, there is virtually no SRT difference between forward and return saccades when the input delay of the second saccade exceeds 270 ms. Note that some of these effects critically depend on the lateral interaction kernel in our DNF model. For example, the leftover activity associated with large saccades competes with the building up of activity at the rostral pole. Because our lateral interaction kernel has a Mexican hat shape, at the rostral pole, nodes closer to the leftover activity will get stronger inhibition, as compared to these which are further away. As a result, small return saccades are faster to initiate than small forward saccades. Note that the results in Figure 3.3 are derived from simulations that only consider the spatio-temporal dynamics within the SCi. The inclusion of other brain systems involved in saccade initiation might interact with these predictions.

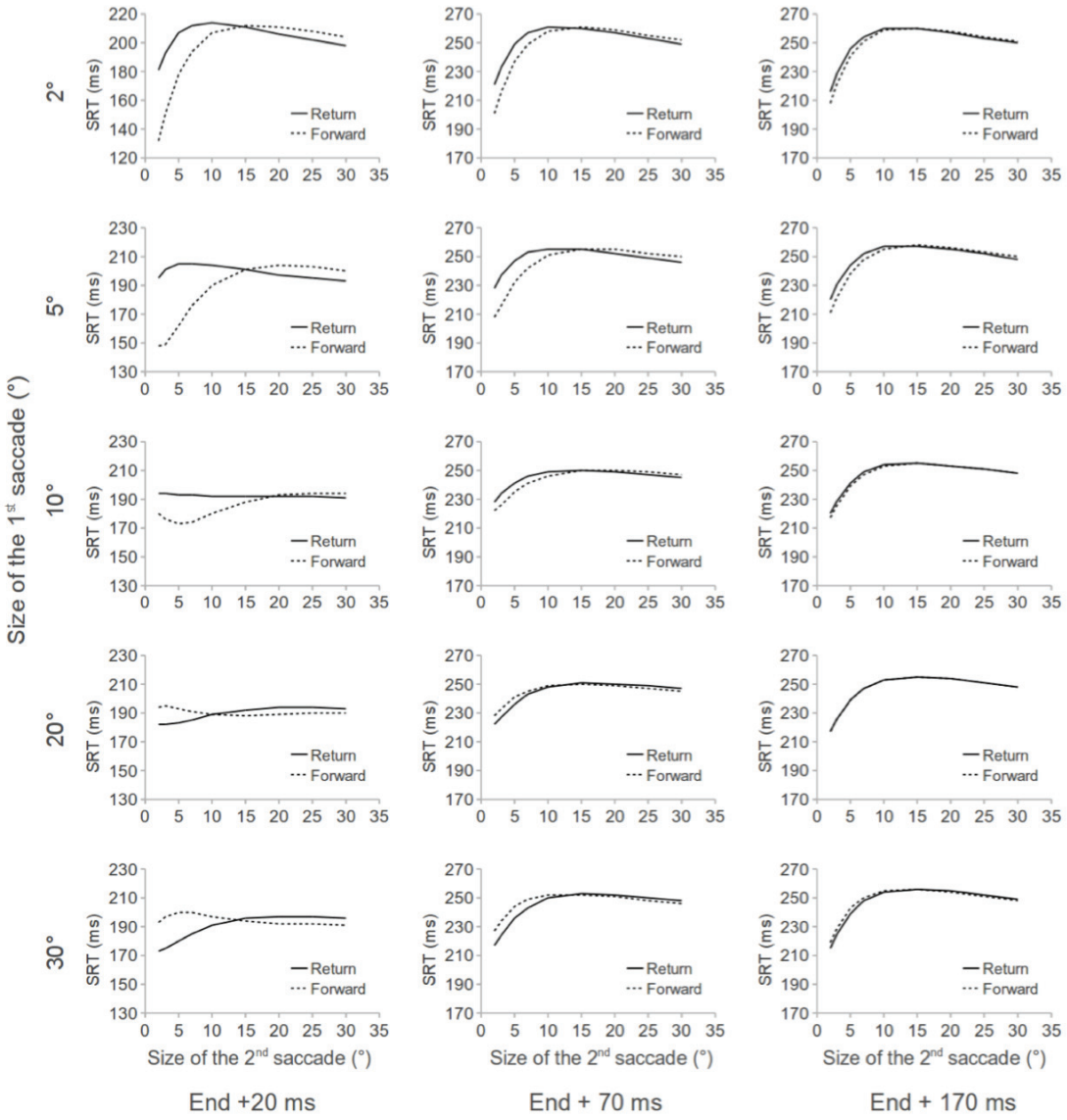

Figure 3.3: Parametric testing of the aftereffects of a saccade in our DNF model. In each panel saccadic RT is plotted as a function of the amplitude of the second saccade. Timing of the input to the network for the 2nd saccade is represented in the columns and amplitude of the preceding saccade is represented in the rows.

3.5 General discussion

3.5.1 Why are return saccades slower to initiate?

Although researchers have frequently reported the behavioral effect that return saccades are slower to initiate than forward saccades, there is no consensus regarding the

mechanism underlying this effect. Some researchers believe it is IOR (Dodd et al., 2009; Klein & MacInnes, 1999; MacInnes & Klein, 2003), while others have attributed the effect to inhibition of saccade return (Hooge & Frens, 2000), or saccadic momentum (Smith & Henderson, 2009).

IOR is a term loosely used by scholars in the field (for a discussion, see Berlucchi, 2006); sometimes it refers to an attention mechanism, sometimes it refers to a behavioral effect, i.e., slower return to previously "attended" locations. When IOR is referred to as a mechanism, it denotes that, as originally proposed by Posner and Cohen (1984), attention is "inhibited" from (overtly or covertly) returning to a previously attended location. Inhibition of saccade return (ISR) is a term used to describe a phenomenon, i.e., slowed saccades to the immediate preceding fixation. ISR is about multiple consecutive saccades, it differs from IOR in a sense that IOR is usually explored in the cue-target paradigm (Hooge & Frens, 2000). Similar to IOR, ISR is local; it "inhibits" the immediate preceding fixation location, it is not caused by a reversal of saccade direction (Hooge & Frens, 2000, Experiment 2b). Saccadic momentum, on the other hand, states that the increase of fixation duration before return saccades is caused by reversing direction (Smith & Henderson, 2009).

Our simulations demonstrate that the delay experienced by return saccades, as compared to forward saccades, could simply be a consequence of the passive remapping of space in the SCi and the lateral interaction within the SCi. This theory does not agree with IOR and ISR which assume a local inhibition. As shown in Figure 3.1A, due to the lateral interaction in the SC, following a saccade, the excited colliculus has residual

activation, while the other colliculus has a decrease of activity (inhibition). Both inhibition and excitation contribute to the exhibited behavioral effect. In fact, Anderson et al. (2008) has demonstrated that the SRT difference between forward and return saccades is contributed by both forward saccades being faster and return saccades being slower. Furthermore, our theory does not agree with "saccadic momentum" as a general phenomenon. As demonstrated in Figure 3.3, our model predicts that the size of a saccade matters. Following small saccades, large reversal saccades are faster to initiate than large forward saccades. Following large saccades, small reversal saccades are faster to initiate than forward saccades, too. Further behavioral testing is needed to clear up this issue.

To sum up, the execution of a saccade will lead to asymmetric activation in the SC and causes saccades which reverse vectors slower to initiate than those which repeat vector. This only holds true for relatively small saccades.

3.5.2 IOR in saccade-saccade paradigms

In the IOR literature, a large set of experimental paradigms have been recruited to explore how previous orienting behavior affects subsequent deployment of attention. One such experimental paradigm is the saccade-saccade paradigm. In a saccade-saccade paradigm, participants are required to make a saccadic response to the cue, then saccade back to the central fixation position, followed by a final saccadic response to the target. The cues and targets can be exogenous (ie., brightening of a peripheral box) or endogenous (ie., an arrow in the central box pointing to one of the peripheral boxes). The findings in this paradigm are similar to that in the cue-target paradigm, with slower (saccadic) responses to cued targets, as compared to uncued targets (but see Dorris et al.,

1999, for an exception with highly practiced monkeys). This effect was believed to be caused by the response to the cue (see Taylor & Klein, 1998; Taylor & Klein, 2000). However, previous researchers have overlooked the fact that following the saccade back to central fixation, saccades to the uncued location are forward saccades while those to the cued location are return saccades. The observed "IOR" effect in this case then, is likely caused (or contaminated) by the saccade back to the central box, rather than the saccade to the cued box. This is especially true when both cues and targets are endogenous stimuli (see Taylor & Klein, 2000, for an example). The mechanism explored here is relatively short-lived (see Figure 3.3). In a saccade-saccade paradigm, this mechanism will make no (or little) contribution to the IOR effect when saccades to the final target are preceded by a long fixation.

Contrary to the common IOR findings (i.e., slower responses to cued relative to uncued targets) one frequently cited study which investigated the saccade-saccade task with monkeys, reported faster SRTs to cued targets (Dorris et al., 1999). This finding does not challenge our theory because the time interval between the end of the saccade back to the central box and the input onset for the saccade to the target was longer than 300 ms, so the mechanism discussed here will have little, or no, behavioral effect on the saccade to the target. The observed effect in that study is likely caused by the same mechanism underlying the IOR effect in traditional cue-target paradigms (i.e., STD elicited by the cue and cue-back sensory input signals). Note that the authors used exogenous stimuli for the cues and targets, and the saccade back from the cued location to fixation was also guided by an exogenous stimulus. Due to this exogenous "cue back", the upstream pathway

responsible for the uncued peripheral box was actually "cued". Thus, while participants were fixating the central box, awaiting the appearance of the target, the cued box still has an old sensory STD process and the uncued box has a new sensory STD process. As a result, responses to cued targets were faster, and the difference between cued and uncued targets were relatively unaffected by cue-target SOAs (see Dorris et al., 1999, Figure 3.3A). With a similar experimental task, the opposite pattern of results (i.e., significant "IOR" effect of21 ms), was reported in Taylor and Klein (2000) with human participants. A closer look at their experimental setup reveals that only 500 ms was allocated for participants to saccade back to the central fixation box. While the authors did not report how fast these saccades were, similar experiments in our lab suggest that, on average, these saccades will take about 330 ms to complete. That is, the time interval between the end of the saccade back and the onset of the target should be relatively short in a large portion of trials in Taylor and Klein (2000). As a result, the mechanism discussed in the present paper should have contributed to their observed "IOR" effect.

3.5.3 IOR in visual search

In contrast to visual search theorists who have claimed that there is no need for a "memory mechanism" which discourages return of attention to previously inspected locations (or items) (e.g., Horowitz & Wolfe, 1998), many researchers believe that IOR is one such mechanism (e.g., Klein, 1988; Koch & Ullman, 1985). The most direct evidence comes from the findings of a "probe-following-search" paradigm during which participant's eye movements are monitored (for a review, see Wang & Klein, 2010). The experimental paradigm used in this line of research is described in our simulations of

Klein and MacInnes (1999). We believe the behavioral findings in this line of research (i.e., forward saccades in response to probes being faster than return saccades) may have been caused, at least in part, by the mechanism explored in the present study.

A search task usually involves multiple saccades. For convenience, based on their ordinal positions relative to the current fixation, previous fixations have been labeled as 1-back (the immediately preceding fixation), 2-back (the fixation prior to the last one), …, n-back, in previous studies. One critical characteristic of the mechanism explored here is that it tends to be short-lived, at least on a behavioral scale, raising the question of whether it explains slower return to fixations beyond the 1-back fixation (as in Dodd et al., 2009; Klein & MacInnes, 1999). One of the consequences of the mechanism presented here is that it encourages saccades to repeat direction (e.g., Hooge et al., 2005; Klein & MacInnes, 1999). As a result, a saccade to the 2-back fixation location will necessarily be larger than the most recent saccade and this difference will, on average, increase for further back saccades. However, saccades made in search tend to be small in size, so a 2-back (or 3-back) saccade will be still in range where a return saccade is slower than a forward saccade (see Figure 3.4). Furthermore, our theory predicts that the number of intervening saccades does not matter too much, saccades to the target location will be slower than those which repeat the vector of their immediately preceding saccade, so long as the target location is in the vicinity of the immediately preceding fixation location and the time interval is sufficiently short.

3.5.4 Another IOR mechanism?

IOR was originally explored in the cue-target paradigm and was characterized by

slower responses (manual or saccadic) to previously cued than to uncued targets. Recent physiological (e.g., Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005), behavioral (e.g., Dukewich & Boehnke, 2008), computational (Satel, Wang, Trappenberg, & Klein, 2011), and theoretical (Dukewich, 2009) developments suggest that IOR in the cue-target paradigm may be largely due to a reduction of target-elicited sensory input, namely, short-term depression (STD) of sensory inputs. This "sensory STD" mechanism of IOR affects the strength of inputs motor programming maps (e.g., in the SC). The sensory STD mechanism, by its nature, is retinotopic. However, depending on the experimental setup, on a behavioral level, this mechanism may appear to be spatiotopic (e.g., Maylor & Hockey, 1985), retinotopic (e.g., Souto & Kerzel, 2009), or both (Mathôt & Theeuwes, 2010).

The effect explored in the present study, i.e., saccades which reverse vectors are slower to initiate than those which repeat vectors, is phenomenologically similar to IOR "effects". However, the underlying mechanism of this effect is quite different. This mechanism is a "motor" mechanism implemented in the SCi; it has little, if nothing, to do with the sensory input itself. On a behavioral level, this mechanism operates on a "spatiotopic" coordinate.

The critical question is can we call this mechanism an "IOR" mechanism? We believe so. First, this mechanism is about orienting and its behavioral consequence is similar to the IOR "effect" observed in the cue-target paradigm. Second, this mechanism biases orienting away from previously fixated locations; this is exactly the function as IOR most nowadays scholars agree upon.

3.5.5 Limitations of the present study

As mentioned earlier, the mechanism proposed here will have little or no effect if a return saccade is preceded by a long fixation (>300 ms). However, this prediction is challenged by several studies. In MacInnes and Klein (2003), a "probe-following-search" task (Klein & MacInnnes, 1999) was tested and participants were instructed to stop searching when they found something "interesting". Probes were delivered 500 ms later.Because the mechanism proposed here is short-lived it does not predict the results of this study: slower responses to probes presented at the immediately preceding fixation locations, as compared to those at locations straight ahead (47 ms). Similarly, Rafal, Egly and Rhodes (1994) explored IOR in saccade-saccade paradigms and an IOR-like effect (27 ms) was observed when the pre-target fixation duration was 500 or 750 ms (Experiment 2). Our model does not predict this pattern either.

It is worth a note that our model is ONLY about the SC, it is possible that the slower initiation of return saccades is also contributed by other cortical maps, especially the FEF which plays a critical role in voluntary control of eye movements (Munoz & Schall, 2003). Besides, previous study showed that visual stimulation temporarily releases inhibitory inputs from substantia nigra, pars reticulata (SNr) to the SC (e.g., Jiang, Stein, & McHaffie, 2003). Following visually guided saccades, the residual activity at the excited SCi sites, and thus the asymmetric activation in the SCi, will last longer than would have been predicted by our model.

Furthermore, we want to mention some technical limitations of the present study. First, a 1-dimensional model was used in the present exploration. Although this one

dimensionality does not undermine the theory we are proposing, we are unable to systematically explore how a saccade affects the latency of subsequent saccades with varied directional deviations without extending the model into two dimensions. Second, in our model, periodic boundary conditions were used to minimize boundary effects. Thus, in the model representation, the caudal area of the two colliculi is connected. This might have caused an underestimation of the residual activity associated with large saccades (see Figure 3.2A).

3.6 Conclusion

In present paper, we have demonstrated that the internal dynamics of the SC can explain why saccades that reverse vectors often have longer latencies than those which repeat vectors. In addition to emphasizing how this finding relates to the IOR literature, we would like to end this paper with a few research proposals.

First, our simulations produce (at least) two novel predictions: a) following small saccades, large return saccades are faster to initiate than large forward saccades; b) following large saccades, small return saccades are faster to initiate than small forward saccades (see Figure 3.3). These effects depend critically on the Mexican-hat shaped lateral interaction kernel used in our DNF model. This lateral interaction kernel is backed by previous single-unit recording studies (e.g., Trappenberg et al., 2001). With these two predictions in mind, it is also possible to validate this lateral interaction kernel with behavioral experiments.

Second, the mechanism discussed here is about the internal dynamics of the SC and will be put into play whenever a saccade is made. So, this mechanism will affect the

behavioral observations of any IOR experiment which involves multiple saccades. In a cue-saccade paradigm in which participants maintain fixation until a target appears in the periphery, the IOR effect is largely caused by sensory STD at the cued retinotopic location reducing the target input to the SC. If a saccadic response is also required to the cue, as in a saccade-saccade paradigm with exogenous cues and targets, the mechanism we are proposing will come into play and increase the observed "IOR" effect. However, this additional effect will appear only if: a) the saccade back to the central fixation is not guided by a visual onset at the central fixation, because such a stimulus will cause STD at the uncued location; b) the time interval between the end of the saccade back to the central fixation and the onset of the target is relatively short.

Third, in saliency-based computational models of orienting (e.g., Itti & Koch, 2001; Koch & Ullman, 1985), IOR is regarded as a low-level mechanism that could overcome the salience of a "winning" item once it has been inspected. In the case of overt orienting, the performance of such models would be significantly improved if the mechanism described here is considered.

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3.9 Appendix: Moving hill or jumping hill?

Early observations in cats showed a "hill" of activity moving toward the rostral pole of the SC during saccades (e.g., Munoz, Pelisson, & Guitton, 1991). However, this phenomenon was not consistently observed in the primate SC (e.g., Anderson, Keller, Gandhi, & Das, 1998; Choi & Guitton, 2009; Munoz & Wurtz, 1995a; Soetedjo, Kaneko, & Fuchs, 2002) and the ecological significance of this "moving hill" is controversial. Our simulation results suggest that the "moving hill" does not encode the trajectory of

saccades (Munoz et al., 1991), nor does it encode the distance between the current gaze position and the target location during multi-step gaze shifts (Bergeron, Matsuo, & Guitton, 2003); it is a byproduct of the input changes and the lateral interaction in the SC (see Figure 3.1B). From a computational perspective, whether a "moving hill" appears during a saccade depends on the lateral interaction kernel, the amplitude of the saccade and the width of the input signals. Figure 3.4A and B illustrates the network activity before, during and after a 10° and 25° saccade. A moving hill was obvious during the 10° saccade while the activity during the 25° is more like a "jumping hill".

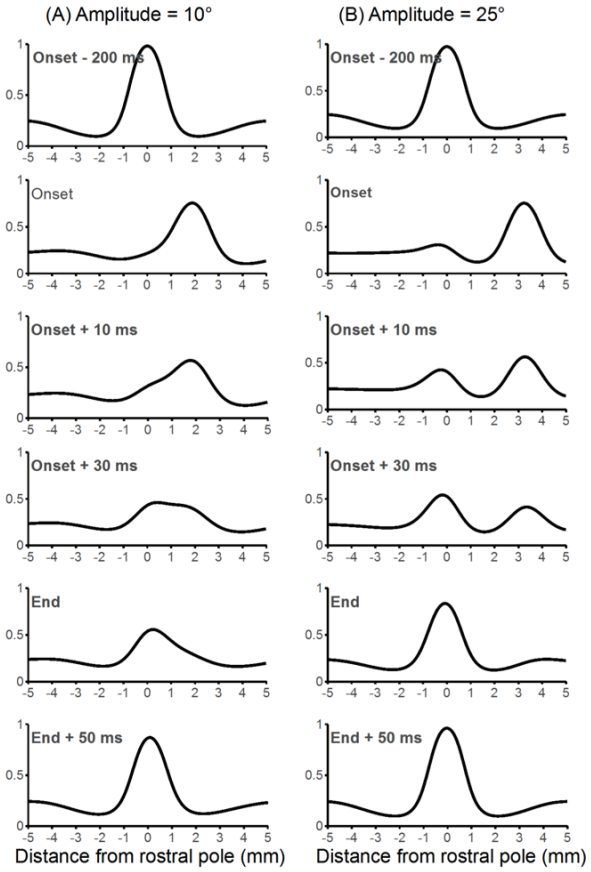

Figure 3.4: Moving hill and jumping hill during a saccade. In these simulations, input for fixation and buildup neurons were set to d = 10, σ_d = 0.7 and d = 12, σ_d = 0.7, respectively. "Onset" means the time when a saccade starts and "End" means the time when a saccade ends.

CHAPTER 4 AVERAGING SACCADES ARE REPELLED BY PRIOR UNINFORMATIVE CUES AT BOTH SHORT AND LONG INTERVALS

Wang, Z., Satel, J., Hilchey, M. D., and Klein, R. M. (2012). Averaging saccades are repelled by prior uninformative cues at both short and long intervals. Visual Cognition, 20(7): 825-847 (reformatted and reprinted with permission of the publisher, Taylor & Francis).

4.1 Abstract

When two spatially proximal stimuli are presented simultaneously, a first saccade is often directed to an intermediate location between the stimuli (averaging saccade). In an earlier study, Watanabe (2001) showed that, at a long cue-target onset asynchrony (CTOA; 600 ms), uninformative cues not only slowed saccadic response times (SRTs) to targets presented at the cued location in single target trials (inhibition of return, IOR), but also biased averaging saccades away from the cue in double target trials. The present study replicated Watanabe's (2001) experimental task with a short CTOA (50 ms), as well as with mixed short (50 ms) and long (600 ms) CTOAs. In all conditions on double target trials, uninformative cues robustly biased averaging saccades away from cued locations. Although SRTs on single target trials were delayed at previously cued locations at both CTOAs when they were mixed, this delay was not observed in the blocked, short CTOA condition. We suggest that top-down factors, such as expectation and attentional control settings, may have asymmetric effects on the temporal and spatial dynamics of oculomotor processing.

4.2 Introduction

Efficient visual orienting is needed for an organism to adapt effectively to the environment. In the laboratory, exogenous control over visual orienting is usually explored with a simple *spatial cueing paradigm* in which a target that requires an orienting response is preceded by an uninformative onset cue (Posner, 1980). When the cue-target onset asynchrony (CTOA) is short, responses to targets that appear at the cued locations are facilitated. This early facilitatory effect is generally attributed to attention

being captured by the cue. When the CTOA exceeds 200 ms, however, an opposite behavioral effect emerges, that is, responses to cued locations are slower than responses to uncued locations (Posner & Cohen, 1984). This latter effect was named "inhibition of return" (IOR), a term implying that attention is "inhibited" from returning to previously attended locations (Posner, Rafal, Choate, & Vaughan, 1985; see Klein, 2000, for a review). Both attentional capture and IOR are important orienting mechanisms. While attentional capture prioritizes processing of salient objects in the environment, IOR encourages orienting toward novelty (Posner & Cohen, 1984) and thus facilitates visual foraging (Itti & Koch, 2001; Klein, 1988; Koch & Ullman, 1985). In the literature, these two opposing orienting mechanisms are typically revealed by their temporal effects (i.e., orienting responses are either sped up or slowed down). How they affect the spatial metrics of overt orienting responses (e.g., saccadic eye movements) has not been thoroughly explored in the literature (but see Theeuwes & Godijn, 2004; Watanabe, 2001).

4.2.1 IOR and saccade averaging

When two proximal visual stimuli are presented simultaneously, the initial saccade often lands at an intermediate location between the stimuli (Becker & Jürgens, 1979; Chou, Sommer, & Schiller, 1999; Coren & Hoenig, 1972; Findlay, 1982; Ottes, van Gisbergen, & Eggermont, 1984; Walker, Deubel, Schneider, & Findlay, 1997; Watanabe, 2001; for a review, see van der Stigchel & Nijboer, 2011); a phenomenon that has been referred to as "saccadic averaging" (Ottes, van Gisbergen, & Eggermont, 1984), or "the global effect" (Findlay, 1982). Watanabe (2001) explored how the aftermath of an

exogenous shift of attention interacts with averaging saccades. Two identical and proximal visual targets were presented simultaneously in the peripheral visual field and participants were instructed to initiate a saccade to one of the targets. The results replicated previous work (e.g., Chou, Sommer, & Schiller, 1999; Ottes et al., 1984), with many saccades landing near the mid-point between the two targets (averaging saccades). However, when a cue was presented at one of the possible target locations before such a paired target (CTOA: 600 ms), averaging saccades were biased away from the cue. To determine the extent to which these non-predictive visual cues might induce a negative cueing effect consistent with the operational definition of IOR, Watanabe (2001) also tested conditions for which a single target was presented. As expected, saccadic reaction time (SRT) to cued targets was, relative to uncued targets, delayed. Because Watanabe's cues were successful at eliciting a negative cueing effect in a standard spatial cueing condition, it was inferred that the mechanisms underlying this result were: 1) likely related to IOR, 2) in effect on the double target trials, and 3) affected the oculomotor system by biasing responses against previously processed spatial events. In a second experiment, a similar pattern of results (i.e., slower responses to cued targets and averaging saccades deviating away from the cued location) was observed when one of the targets served as saccade target while the other served as a distractor (see also Theeuwes & Godijn, 2004). These findings suggest that covert exogenous shifts of attention have spatial, as well as temporal, effects.

4.2.2 Neural field modeling of averaging saccades and IOR

Although widespread agreement about the neural implementation of averaging

saccades is lacking (see Glimcher & Sparks, 1993; van Opstal & van Gisbergen, 1990), recent computational work (e.g., Satel, Wang, Trappenberg, & Klein, 2011; Wilimzig, Schneider, & Schoener, 2006) endorses the idea that averaging saccades can be programmed at the level of the superior colliculus (SC). The SC contains a winner-take-all motor map that encodes the direction and amplitude of saccades (van Gisbergen, van Opstal, & Tax, 1987; Robinson, 1972). Neurons in this map are laterally connected in a manner such that proximal neurons excite each other, while distal neurons inhibit each other (for a summary of related evidence, see Marino, Trappenberg, Dorris, & Munoz, 2011; Munoz & Fecteau, 2002). When balanced inputs are given to two spatially proximal locations, due to short-distance collaboration, the input-elicited activity will merge together, peaking in between these two locations, leading to averaging saccades (Glimcher & Sparks, 1993).

Previous single-unit recording studies have shown that, in a spatial cueing paradigm, target-related SC activity is reduced if the target appears at the same location as the cue (e.g., Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005) at relatively short CTOAs. Similar reductions in neural activation in the extrastriate cortex in response to cued targets has been observed in human ERP (see Prime & Ward, 2006, for a review) and fMRI (e.g., Anderson & Rees, 2011) studies at longer CTOAs. Previously, these findings led us to model IOR as short-term depression (STD) of signal strength in the early visual pathway (Satel et al., 2011; see also Ibáñez-Gijón & Jacobs, 2012). This STD process attenuates target-related visual input, leading to longer response times to cued than to uncued targets (IOR). However, when the CTOA is very short, the

cue-evoked activity has not yet abated. Consequently, the cued target-elicited activation (even though attenuated by STD) is still able to cross the saccade threshold faster than the uncued target-elicited activation since the cued target's baseline remains elevated before target appearance as compared to the uncued target's resting baseline. This residual activation from the cue – when greater than the effect of STD – can lead to a faster crossing of the saccade threshold (facilitation). Similar explanations of the early facilitation and later IOR effects observed in spatial cueing paradigms have been proposed by Bell, Fecteau, and Munoz (2004) and Dukewich (2009).

As demonstrated in Satel et al. (2011), the STD theory of IOR can be used to simulate Watanabe's (2001) findings on deviation in averaging saccades at long CTOAs. When one of the targets is cued, as in Watanabe (2001), the peak of the merged activity drifts away from the cued location, through lateral network interaction, leading to averaging saccade deviation away from the cue (Satel et al., 2011) because the visual input of the cued target is attenuated. In addition to successfully simulating Watanabe (2001), Satel et al. (2011) suggested that "At shorter CTOAs known to cause behavioral facilitation, our model predicts that saccades will be biased toward the cued location, since the cue elicited exogenous input in the SC has not yet completely decayed, and consequently the cued target node will reach threshold and initiate a saccade before the uncued target node" (p. 994).

4.2.3 Purpose of the present study

The CTOA tested in Watanabe (2001) was sufficiently long (600 ms) to observe behavioral evidence consistent with the operational definition of IOR. However, it is not

clear whether attentional capture is also accompanied by deviation in averaging saccades when a very short CTOA is tested. The temporal benefit immediately following exogenous shifts of attention seems to suggest that averaging saccades should deviate toward the previously attended location. Since the onset cue is thought to reflexively capture attention, it would be logical, and adaptive, for saccades to deviate toward a location where attention is directed. Based on the these arguments and their simulation results at a long CTOA, Satel et al. (2011) predicted that saccades would deviate *toward* cued locations in a double target paradigm when the interval between cue and target is very short. In the present study, we set out to explore this possibility empirically by replicating the experimental task of Watanabe (2001, Experiment 1) with a short CTOA (50 ms).

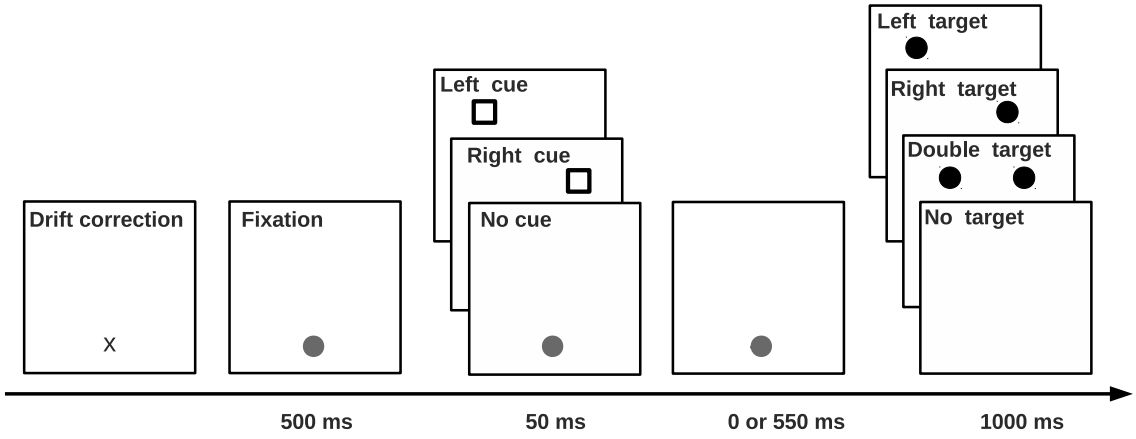

Figure 4.1: Sequence of events in a trial, see text for details. In Experiment 2, following the cue, a fixation point was presented for 550 ms in long CTOA conditions.

4.3 Experiment 4.1: Short CTOA only

As in Watanabe (2001, Experiment 1), the present experiment introduces an exogenous cueing (Posner, 1980) stimulus to a double target paradigm (Chou et al., 1999;

Findlay, 1982). Unlike Watanabe (2001), which tested a long CTOA (600 ms) and the effects of paired cues on saccadic responses, we tested a short CTOA (50 ms) to reveal how an uninformative cue affects averaging saccades at a short CTOA.

4.3.1 Methods

Participants: The 14 participants (9 female, 5 male) were university students who reported normal or corrected to normal visual acuity and who participated in exchange for extra course points. Two participants were dropped from the analysis because they did not finish the experimental task.

Stimuli and apparatus: The fixation point was a gray disk (d = 0.43°) at the center of the screen, the cue was a bright open square (1.0° × 1.0°) with a border thickness of 1 pixel, and the target was a bright disk (d = 0.57°). Two locations, 10° from fixation in the upper visual field, were chosen to present the cue and target(s). The horizontal distance between these two peripheral locations was 5.2°, hence, relative to fixation, the angular separation between them was 30°.

All visual stimuli were presented on a 17 inch, SVGA, Viewsonic computer monitor with screen resolution set to 1024 × 768 pixels. The visible area of the monitor measured 36.2° (width) × 27° (height) at a viewing distance of 57 cm. An EyeLink® video-based eye-tracking system was used to monitor participants' direction of gaze every 4 ms with a spatial resolution of 0.1° or better.

Design and procedure: The experimental task is illustrated in Figure 4.1. Drift correction was performed at the beginning of each trial; the drift correction target was a gray cross (×) measuring 0.5° × 0.5°. A fixation point was then presented for 500 ms. On

cued trials, a cue was then presented at either the left or right target location for 50 ms, after which the target(s) was presented immediately (except on catch trials) with the fixation point removed at the same time. That is, there was no temporal gap between fixation offset and target onset. The target(s) stayed on screen for 1000 ms, and the participant was instructed to maintain fixation if no target was presented, to quickly move their eyes to look at the target when only one target was presented, or to look at their preferred target if two targets were presented simultaneously.

Participants were first exposed to 12 practice trials, then they were tested with three blocks of 204 trials. The cues had equal probability of not being presented, or being presented at the left or the right target location. To discourage anticipatory responses, the target was not presented on 25% of the trials (catch trials). In the remaining 75% of the trials, the target was equally likely to appear at the left, right, or both target locations. To discourage saccadic responses to the cue, trials on which the SRTs were faster than 120 ms were considered as error trials and recycled. Trials in which saccades were initiated when no target was presented were also recycled.

4.3.2 Results

Trials on which gaze deviated more then 1° from the fixation point before target onset (7.99%), when a saccade was made in the downward direction (0.02%), or when the SRT was slower than 550 ms (2.17%) were discarded. Trials on which the first saccade had a horizontal amplitude greater than 5.4° (0.64%), or had a vertical amplitude of less than 5° (6.44%) or greater than 12° (4.18%), were also discarded. 82.37% of the trials remained after this data cleansing procedure.

Exp	CTOA (ms)	Left target			Right target			Double target		
		Left-cue	No-cue	Right-cue	Left-cue	No-cue	Right-cue	Left-cue	No-cue	Right-cue
Exp. 1	50	285	251	284	277	249	277	272	254	270
Exp. 2	50	322	273	315	305	268	327	298	269	314
Exp. 2	600	271	259	249	240	254	269	249	256	257

Table 4.1: Mean SRT (ms) of each condition in Experiments 4.1 and 4.2.

SRTs: Mean SRTs for single-target and double-target trials are presented in Table 4.1. Single-target trials were first analyzed to see whether facilitation or IOR was observed behaviorally. There were three types of single-target trials: 1) no cue, 2) target and cue presented at the same location (same-side), and 3) target presented on the opposite side of the cue (opposite-side). A repeated measures ANOVA on the SRTs revealed a significant difference between no-cue (250 ms), same-side (281 ms) and opposite-side (281 ms) trials [$F(2, 22) = 8.85$, $p < 0.01$, $\eta_G^2 = 0.07$]. It should be noted that the effect size measure reported in the present paper is generalized eta squared (Bakeman, 2005). Planned comparisons showed longer SRTs for same-side [$t(11) = 4.49$, $p < 0.001$] and opposite-side [$t(11) = 4.20$, $p < 0.001$] trials than for no-cue trials. However, no facilitation or IOR effect was observed [$t(11) = 0.02$, n.s.] (same-side SRTs were equal to opposite-side SRTs). Analysis of double-target trials revealed longer SRTs when either the left or right cue was presented (271 ms) than when no cue was presented (254 ms) [$F(1, 11) = 17.58$, $p < 0.01$, $\eta_G^2 = 0.02$].

Exp	CTOA (ms)	Left target			Right target			Double target		
		Left-cue	No-cue	Right-cue	Left-cue	No-cue	Right-cue	Left-cue	No-cue	Right-cue
Exp. 1	50	-2.13	-2.14	-2.10	2.57	2.70	2.66	1.30	0.81	-0.28
Exp. 2	50	-2.34	-2.32	-2.24	2.35	2.44	2.40	0.56	0.15	-0.67
Exp. 2	600	-2.29	-2.34	-2.33	2.43	2.45	2.36	0.59	0.20	-0.33

Table 4.2: Mean horizontal amplitude (°) of each condition in Experiments 4.1 and 4.2. Positive and negative values denote right and left to the fixation, respectively.

Saccade landing positions (Horizontal amplitudes): The landing position and horizontal amplitude distribution of the first saccade following the presentation of the target(s) in all conditions are presented in Figure 4.2; the mean horizontal amplitude of each condition is presented in Table 4.2. As clearly shown in the first column of Figure 4.2, saccades tended to land in between the two targets in double-target conditions, replicating previous observations of saccade averaging (e.g., Chou et al., 1999; Ottes et al., 1984). For the mean horizontal amplitudes, ANOVAs were performed separately for single- and double-target conditions. For single-target conditions, only the main effect of target location (left vs. right) reached significance [$F(1, 11) = 1222$, $p < 0.001$, $\eta_G^2 = 0.97$]. The non-significant main effect of cue location (no-cue, left-cue, or right-cue) [$F(2,22) = 2.73$, n.s., $\eta_G^2 = 0.00$] and the non-significant interaction between target location and cue location [$F(2,22) = 1.78$, n.s., $\eta_G^2 = 0.00$] suggest that the mean landing position in single-target conditions was largely unaffected by the cue. For double-target conditions, the main effect of cue location reached significance [$F(2, 22) = 12.87$, $p < 0.001$, $\eta_G^2 = 0.19$]. As is clear from Figure 4.2, while averaging was evident in double-target conditions, both left and right cues biased the mean landing position of saccades away from the cued locations (see also Table 4.2).

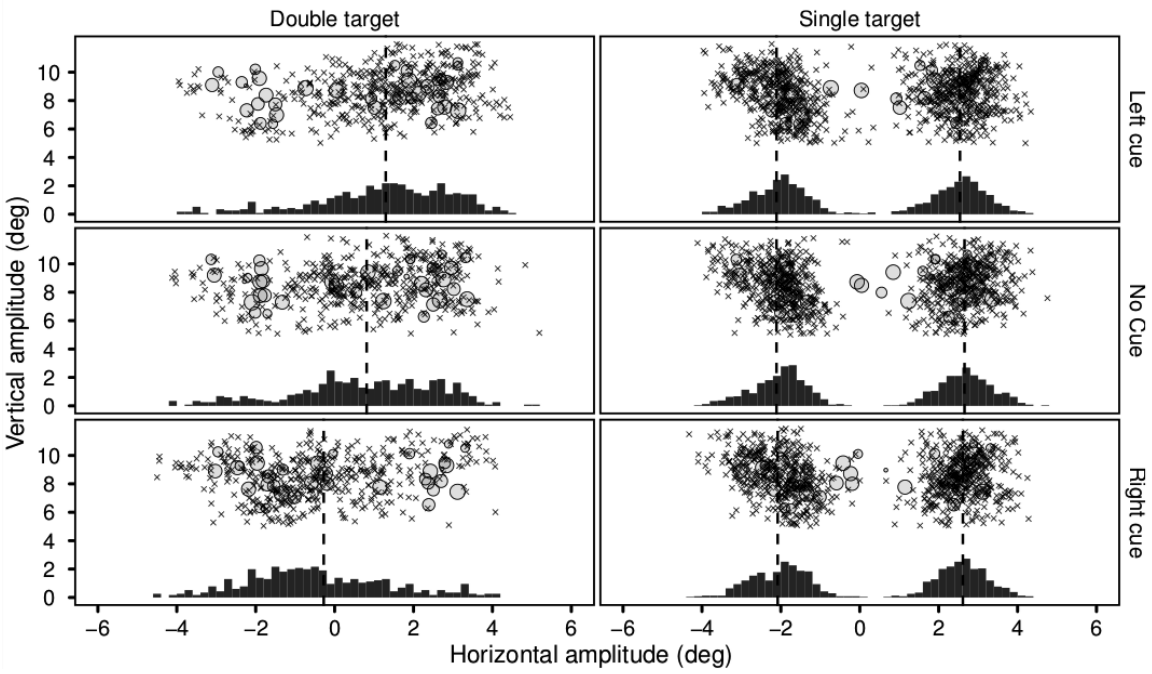

Figure 4.2: Landing positions of the saccades made in Experiment 4.1. Black crosses denote the landing positions in space. The center-of-gravity of each participant is represented by gray disks of which the size represents the sample size. Dashed black lines denote the mean horizontal amplitude for each condition. The histograms are used to represent the distribution of the horizontal amplitudes of saccades. For clearer representation of the data, the y-axis of the histogram (density) was amplified by a factor of 8.

4.3.3 Discussion

The purpose of Experiment 4.1 was to replicate Watanabe (2001, Experiment 1) with a short CTOA at which a behavioral facilitation effect (rather than IOR) is frequently observed (e.g., Briand, Larrison, & Sereno, 2000; Khatoon, Briand, & Sereno, 2002). This early facilitation effect has been attributed to attention being captured by the cue (Posner, 1980; Posner & Cohen, 1984). If exogenous shifts of attention affect averaging saccades, one would expect averaging saccades to deviate toward a cue that has captured attention at a short CTOA. This hypothesis was not supported by Experiment 4.1, which

showed that averaging saccades deviated away from, rather than toward, the cued location, even at a short CTOA of 50 ms (see Table 4.1 and Figure 4.2). However, since no behavioral facilitation effect was observed in Experiment 4.1, one cannot yet preclude a possible relationship between exogenous shifts of attention and averaging saccades. Scholars in the field have long proposed that capture of attention and IOR may be two parallel processes (Klein, 2000; Posner & Cohen, 1984). It is possible that the 0.79° deviation was caused by IOR, while the behavioral manifestation of IOR in SRTs was masked by cue-elicited attentional capture.

4.4 Experiment 4.2: Mixed short and long CTOAs

Averaging saccades deviated away from the cue (in double-target trials) by 0.79° in our Experiment 4.1 (short CTOA). This amount of deviation was considerably less than that reported in Watanabe's (2001) long CTOA experiment (approximately 1.5°; data extracted from his figures) . Together with the fact that no IOR effect (0 ms) was observed in our short CTOA experiment (Experiment 4.1) and robust IOR (24 ms; calculated from Watanabe, 2001, Figure 4.2) was observed at a long CTOA (600 ms) in Watanabe (2001), both the behavioral effect of IOR and the deviation in averaging saccades seem to increase with CTOA. It should be noted that a 100 ms temporal gap between fixation offset and target onset was used in Watanabe (2001). This temporal gap was impossible, practically, for our 50 ms CTOA in Experiment 4.1. Thus, we cannot make strong inferences about the relationship between IOR and deviation in averaging saccades across CTOAs by comparing our results to those reported in Watanabe (2001). In order to more accurately assess IOR and deviation in averaging saccades across

CTOAs, we ran a second experiment in which short and long CTOAs were intermixed and in which there was no temporal gap between the fixation stimulus offset and target onset. This design allows for direct comparisons between results at the short and long CTOAs. Given that IOR and deviation in averaging saccades were observed in Watanabe (2001), we expected to observe evidence for behavioral IOR in the single-target conditions and saccade deviation away from the cued location in double-target conditions at the long CTOA, while at the short CTOA, we expected to reproduce the pattern of results from Experiment 4.1, that is, very weak, or no, evidence for temporal IOR despite saccade deviation away from the cue. Furthermore, if the deviation in averaging saccades increases with CTOA, then stronger deviations in averaging saccades away from cued locations would be expected in the long CTOA condition.

4.4.1 Methods

Eighteen university students (11 female, 7 male) participated in Experiment 4.2 to get extra course points. One participant was dropped from analysis because she finished less than 50% of the trials. The stimuli and task were the same as Experiment 4.1 except that, in addition to the short CTOA (50 ms), a long CTOA (600 ms) was also tested, in a mixed condition experimental design (see Figure 4.1). Note that there was no temporal gap between fixation offset and target onset; fixation remained on until target presentation. Furthermore, the number of trials in which no target or cue was presented was reduced to 1/23; thus, the total number of catch (no target) trials decreased to 21.7% (5/23). All participants were tested with three blocks of 207 trials preceded by 23 practice trials.

4.4.2 Results

As in Experiment 4.1, trials in which the eyes deviated more then 1° from the fixation point before target onset (12.0%), or when a saccade was made in the wrong direction (0.5%), or when saccade latency was slower than 550 ms (2.4%) were discarded. Trials in which the first saccade had a horizontal amplitude greater than 5.4° (0.8%), or had a vertical amplitude of less than 5° (5.3%) or greater than 12° (2.9%) were also discarded. After this data cleansing procedure, 77.7% of the total trials remained.

SRTs: The mean SRTs for each condition are presented in Table 4.1. An ANOVA of the SRTs from the single-target conditions revealed a main effect of CTOA (short vs. long) [$F(1, 16) = 148.5$, $p < 0.001$, $\eta_G^2 = 0.33$]. As shown in Table 4.1, there was a general trend for faster SRTs at the long CTOA, likely caused by temporal expectation of the target (Kingstone, 1992). The main effect of cueing (no-cue, same-side, or opposite-side) [$F(2, 32) = 32.51$, $p < 0.001$, $\eta_G^2 = 0.16$] and the 2-way interaction [$F(2, 32) = 28.98$, $p < 0.001$, $\eta_G^2 = 0.11$] were also significant. The interaction between cueing and CTOA occurred because at the short CTOA there was about a 46 ms delay in SRT on trials with a cue (cued and uncued combined) compared to trials with no cue; a delay that was entirely eliminated in the long CTOA condition (see Table 4.1). To examine IOR, an ANOVA omitting no-cue conditions was performed. These results showed a main effect of cueing (same-side vs. opposite-side) [$F(1, 16) = 38.74$, $p < 0.001$, $\eta_G^2 = 0.10$] that did not interact with CTOA [$F(1, 16) = 1.09$, n.s., $\eta_G^2 = 0.00$]. These effects suggest that, though numerically larger in the long (25 ms) than in the short (15 ms) CTOA condition, the IOR effects (the SRT difference between same-side and opposite-side conditions) were

relatively unaffected by CTOA.

An ANOVA of the SRTs of double-target trials, with the variables cue location (no-cue, left cue, or right cue) and CTOA, revealed significant main effects for cue location [$F(1, 16) = 11.87$, $p < 0.01$, $\eta_G^2 = 0.07$] and CTOA [$F(1, 16) = 30.93$, $p < 0.001$, $\eta_G^2 = 0.17$]. An interaction between cue location and CTOA was also observed [$F(1, 16) = 14.01$, $p < 0.01$, $\eta_G^2 = 0.08$]. As shown in Table 4.1, the 2-way interaction occurred because trials containing a cue produced longer SRTs in the short CTOA conditions, while SRTs in the long CTOA conditions were relatively unaffected by the presence of the cue.

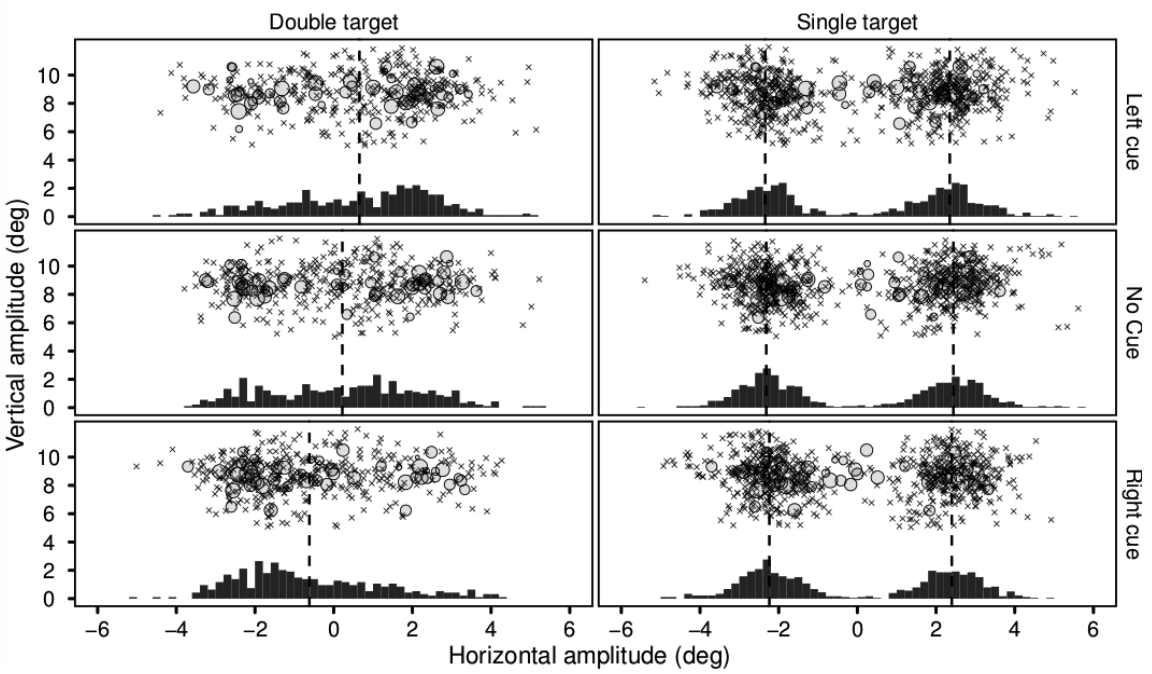

Figure 4.3: Landing positions of saccades made in the short CTOA (50 ms) of Experiment 4.2. Same notations as in Figure 4.2.

Saccade landing positions (Horizontal amplitudes): Saccade landing positions and the distribution of the horizontal amplitudes for each condition are presented in Figures 4.3 and 4.4; the mean horizontal amplitude of each condition is presented in Table 4.2. As in Experiment 4.1, separate ANOVAs were performed for single- and double-target conditions. For single-target conditions, the analysis revealed a main effect of target (left-target vs. right-target) [$F(1, 16) = 26.90$, $p < 0.001$, $\eta_G^2 = 0.95$]. The interaction between cue location (left-cue, right-cue, or no-cue) and target location [$F(2, 32) = 1.97$, n.s., $\eta_G^2 = 0.00$] did not reach significance, suggesting that, as in Experiment 4.1, saccade landing positions in single-target conditions were largely unaffected by the cue. Interestingly, a significant interaction was observed between cue location and CTOA [$F(2, 32) = 3.82$, $p < 0.05$, $\eta_G^2 = 0.00$]. As shown in Table 4.2, as compared to conditions where the cue appeared at the same location as the target, when the cues appeared at the opposite side of the target, there seemed to be a trend for saccades to deviate toward the cued location at the short CTOA and to deviate away from the cued location at the long CTOA. Further analysis, however, failed to reveal this 2-way interaction for both the left target [$F(2, 32) = 2.42$, n.s., $\eta_G^2 = 0.00$] and right target [$F(2, 32) = 1.76$, n.s., $\eta_G^2 = 0.00$] conditions. When the no cue conditions were dropped from the ANOVAs, this interaction reached significance only for left target conditions [$F(1, 16) = 4.90$, $p < 0.05$, $\eta_G^2 = 0.00$]. This effect, though extremely small (see Table 4.2), warrants further scrutiny in future studies.

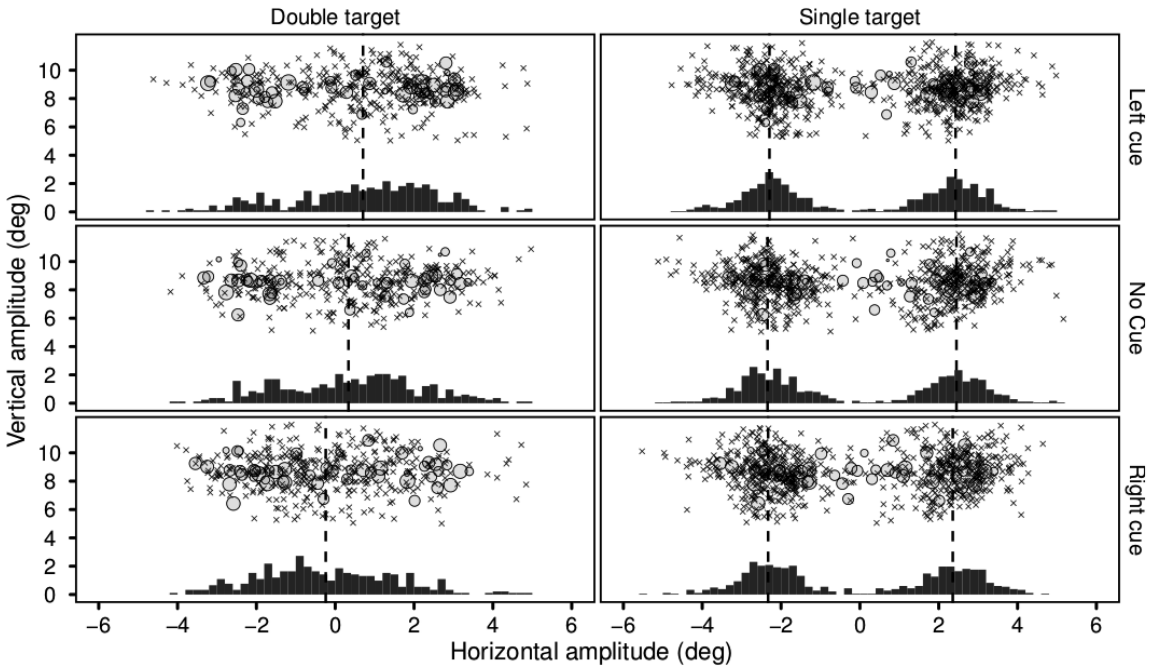

Figure 4.4: Landing positions of saccades made in the long CTOA (600 ms) of Experiment 4.2. Same notations as in Figure 4.2.

For double-target trials, analysis revealed significant main effects for cue location [$F(2, 32)= 34.8$, $p < 0.001$, $\eta_G^2 = 0.14$] and CTOA [$F(1, 16) = 9.11$, $p < 0.01$, $\eta_G^2 = 0.00$]. As shown in Figure 4.3, Figure 4.4, and Table 4.1, the main effect of cue location occurred because the mean landing position of saccades was biased away from the cued location at both CTOAs. The non-significant interaction between cue location and CTOA [$F(2, 32) = 1.11$, n.s., $\eta_G^2 = 0.00$] suggests that the direction of deviation was consistent across the CTOAs. As manifested by the main effect of CTOA and data presented in Table 4.1, relatively stronger deviation was observed for the short than for the long CTOA.

4.4.3 Discussion

The primary purpose of Experiment 4.2 was to explore how IOR and deviation in

averaging saccades vary with CTOA. As shown in Table 4.1, IOR and averaging saccades deviating away from the cued location were observed at both CTOAs in Experiment 4.2. However, in Experiment 4.1, deviation away from the cue was observed in the absence of any behavioral evidence of IOR. This pattern of results seems to suggest a dissociation between behavioral IOR and deviation in averaging saccades. That is, when a short CTOA was tested, averaging saccades deviated away from the cued location irrespective of the observation of behavioral IOR.

The only difference between Experiment 4.1 and 4.2 was that short CTOA trials were intermixed with long CTOA trials in Experiment 4.2, whereas only the short CTOA was tested in Experiment 4.1. As shown in Table 4.1, SRTs in the short CTOA condition of Experiment 4.2 were numerically longer than those in Experiment 4.1. ANOVAs on the SRTs from the short CTOA conditions, with experiment (Experiment 4.1 vs. Experiment 4.2) as a factor, revealed a marginally significant main effect of experiment for single-target conditions [$F(1, 27) = 3.83$, $p = 0.06$, $\eta_G^2 = 0.11$], but not for double-target conditions [$F(1, 27) = 2.70$, n.s., $\eta_G^2 = 0.08$]. These data provide some evidence that the single CTOA in Experiment 4.1 may have evoked a strong non-spatial target expectancy that eliminated behavioral IOR or that observers attempted to take advantage of the temporal relationship between the cue and target in Experiment 4.1, the aftermath of which was a bias to attend or remain attending at the cued location. We will evaluate both of these hypotheses in turn in the General Discussion section.

4.5 General discussion

Whether exogenous cueing affects the metrics of overt spatial orienting responses

has long been explored in the literature. Watanabe (2001) found that at a 600 ms CTOA, when a cueing paradigm was combined with a double-target paradigm, IOR was accompanied by averaging saccades deviating away from the cued location. Furthering this line of research, the present study replicated Watanabe's (2001) experimental task with a very short CTOA to explore whether early facilitation is also accompanied by deviations in averaging saccades, as predicted by Satel et al. (2011). To our surprise, no behavioral evidence for a facilitation effect was observed when a 50 ms CTOA was tested alone (Experiment 4.1), and yet the saccade landing site consistently deviated away from the cued location on double target trials. When the 50 ms CTOA was mixed with a 600 ms CTOA (Experiment 4.2), IOR was observed at both CTOAs and averaging saccades deviated away from cued locations in comparable magnitudes in both experiments.

In the next section, using a computationally explicit neural field model in which the SC is the site of convergence of bottom-up and top-down signals that generate oculomotor behavior (Trappenberg, Dorris, Munoz, & Klein, 2001), we will first examine the possibility that non-spatial target expectancy eliminated IOR, but not averaging saccade deviation, in Experiment 4.1. Whereas the model succeeds in simulating many aspects of our results from both experiments, without ad hoc (if not implausible) assumptions the model cannot simulate the entire pattern of results. Hence, in the final section of this general discussion, we also present an alternate theoretical account for the present data set by relying on abstract cognitive principles. This cognitive account successfully predicts the present data set but is lacking in mathematical explicitness.

4.5.1 Explaining cue-induced effects on saccadic behavior with a DNF model

We have suggested that a non-spatial target expectancy could eliminate IOR (SRTs in the single target condition) while preserving effects on landing position (in the double target condition). Because the model used by Satel et al. (2011) has a top-down component related to spatial and temporal expectancy of the saccade target, this model can be used to determine if this target expectancy explanation for our behavioral findings is computationally feasible.

$$w_{ij} = a * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_a^2}\right) - b * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_b^2}\right) - c \qquad \text{Equation 4.1}$$

$$\tau \frac{du_i(t)}{dt} = -du_i(t) + \sum_j w_{ij} r_j(t) \Delta x + I_i(t) + u_0 \qquad \text{Equation 4.2}$$

$$I_k = d * \exp\left(\frac{((k-i)\Delta x)^2}{2\sigma_d^2}\right) \qquad \text{Equation 4.3}$$

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)} \qquad \text{Equation 4.4}$$

Model architecture and parameters: The structure of the neural field model used in the present study has been described in previous work by our group (Satel et al., 2011; Wang, Satel, Trappenberg, & Klein, 2011). Here, a slice of SC tissue encoding 10° (amplitude) saccades of various directions was represented with $n = 1001$ (population) nodes. The lateral connection strength (w_{ij}) between two nodes i and j, depending on their physical distance in the SC, was defined by Eq. 4.1, with parameters $a = 72$, $b = 24$, $c = 6.5$, $\sigma_a = 0.6$ mm, and $\sigma_b = 1.8$ mm. These parameters were estimated by Trappenberg et

al. (2001) based on cell recordings in the monkey SC. The dynamics of the internal state of a node i, $u_i(t)$, is described in Eq. 4.2, where w_{ij} is the strength of connection from node j to node i, $r_j(t)$ is the firing rate of node j, $I_i^{exo,endo}(t)$ is the external input to node i, $u_0 = 10$ regulates the resting level, and $\tau = 20\ ms$ is a time constant. Two different sources of external inputs were considered in this model (Kopecz & Schöner, 1995; Trappenberg et al., 2001). The exogenous (exo) input represents the visual cortex and the endogenous (endo) input represents higher cortical areas where intentional deployment strategies (e.g., motor preparation to "saccade upward") likely arise. The exogenous input was assumed to arrive at the SC 70 ms after stimulus onset and, for imperative visual stimuli, the endogenous input (a hypothetical "go" signal) arrived at the SC 50 ms later. Both exogenous and endogenous inputs were assumed to have a Gaussian spatial shape (Eq. 4.3). Based on data obtained with cell recordings in the monkey SC (Marino et al., 2011), the input width was set to $\sigma_d = 0.6\ mm$ for both exogenous and endogenous inputs. Following the STD theory of IOR, the exogenous input of an imperative visual stimulus (target) is reduced if it appears at a retinal location previously occupied by a visual cue (Satel et al., 2011). Based on the STD parameters used by Satel et al. (2011), in our simulations this reduction was set to 32.5% for a CTOA of 50 ms. A sigmoid gain function (Eq. 4.4) was used to relate the firing rate of a node i, $r_i(t)$, to its internal state, $u_i(t)$, where $\beta = 0.07$. Once the activity of an SC node reaches 80% of its maximum firing rate, a saccade is triggered to the response field of that node, with an additional 25 ms efferent delay.

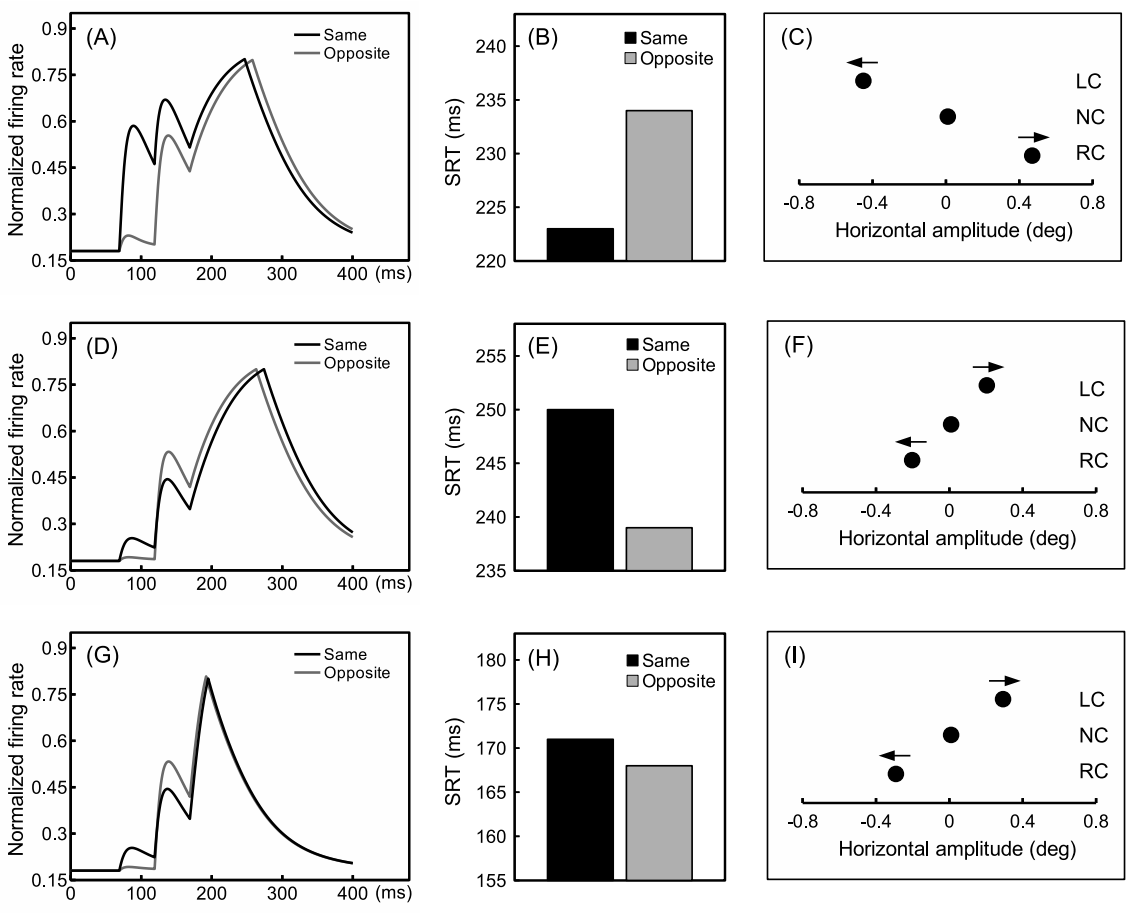

Figure 4.5: Results of Simulations 1-3 (rows 1-3, respectively). Column 1 illustrates simulated node activity in single-target trials where the target was either cued (same) or uncued (opposite); corresponding SRTs are presented in column 2. Column 3 summarizes the horizontal amplitudes of averaging saccades when a left-cue (LC), right-cue (RC), or no-cue (NC) was presented in double-target trials.

Simulation results: Three simulations were performed, with the main results presented in Figure 4.5 (A-C for Simulation 1; D-F for Simulation 2; and G-I for Simulation 3). Simulation 1 was performed to illustrate Satel et al.'s (2011) prediction that at a short CTOA averaging saccades could deviate toward cued locations. Simulations 2 and 3 were performed to reproduce the findings of the short CTOA conditions of Experiment 4.2 and Experiment 4.1, respectively. The first column in Figure 4.5 shows the simulated node activity in single target trials with the target either appearing at the

cued (same) or uncued (opposite) locations. When the target appeared at the cued location, the three peaks were evoked by the exogenous cue input, the exogenous target input, and the endogenous target input, respectively. When the target appeared at the uncued location, only target related activation was present in the simulated node activity. Simulated SRTs and deviations in saccade landing position are presented in columns 2 and 3 of Figure 4.5.

According to the STD theory proposed by Satel et al. (2011), the facilitation effect immediately following exogenous cueing is caused by residual cue-related activation. If the cue-related activation is strong enough to overcome the reduction in exogenous target input caused by STD, faster responses to cued versus uncued targets will be observed. If the cue related activation is very weak, slower responses to cued targets will be obtained. Our failure to observe a behavioral facilitation effect in the present experiments might have occurred because the cue-elicited activation was not strong enough to overcome the STD-elicited target input reduction. To illustrate this possibility, the exogenous input strength of the cue was set to $e = 80$ for Simulation 1, and $e = 20$ for Simulations 2 and 3. It should be noted that cue-related activation can be modulated by factors other than the exogenous cue input, such as the informativeness of the cue (Bell & Munoz, 2008). For simplicity, we chose to use exogenous cue input strength to manipulate cue-related activation in our simulations. The exogenous input for the targets was fixed at $e = 70$ in all simulations. As is clear from Figure 4.5D, due to STD, cued targets (black traces) evoked weaker activation than uncued targets (gray traces). As a result, the node activity took longer to cross threshold, leading to longer SRTs to cued than to uncued targets

(IOR, see Figure 4.5E). At the same time, the landing position of averaging saccades deviated away from cued locations. When the cue-related activation was strong (Simulation 1), cued targets crossed threshold earlier than uncued targets (Figure 4.5A), leading to faster SRTs to cued than to uncued targets (Figure 4.5B) and deviation toward cued locations (Figure 4.5C).

In our simulations, endogenous target input was further dissected into target expectancy and a hypothetical "go" signal. The "go" signal reflected the instruction to make a saccade to the target (in single-target trials) or to one of the two targets (in double-target trials); it was thus spatially specific. The strength of this signal was fixed at $e = 3$ in all simulations. Target expectancy is manifested by the well-known foreperiod or warning signal effect (see Niemi & Näätänen, 1981, for a review). This endogenous signal was input to both target locations in double-target trials, and the single target location in single-target trials, 120 ms after target(s) onset[1], at the same time as the endogenous "go" signal. Because the temporal uncertainty regarding target appearance was weaker in Experiment 4.2 than in Experiment 4.1, target expectancy was assumed to be weaker in Simulation 2 ($e = 12$) than in Simulation 3 ($e = 29$). In simulated node activities, this difference was reflected by the speed of rising of the third peak in Figures 4.5D and 4.5G. Even though STD reduced target related activity in both Simulations 2 and 3, when target

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[1] One may suggest that target expectancy should be input to the network before target onset. Such an implementation is unjustified for the following reasons. First of all, if target expectancy reaches the SC long before target onset, in a spatial cueing task, one would expect a slow buildup of neuronal activity following cue onset when the CTOA is relatively long. However, this is not the case (for recordings, see Fecteau & Munoz, 2005; Dorris et al. 2002). Second, it has been shown that fixation offset can serve as a warning signal and raise the activation level in the SC (Dorris, Pare, & Munoz, 1997), starting from about 150 ms after fixation offset (Marino, Trappenberg, Dorris, & Munoz, 2011). However, because no temporal gap was included in the present behavioral experiments, fixation offset could not possibly be used as a warning signal by the participant. Thus, in the present experiments, it is most like that target expectancy boosted the top-down decision about where the eyes should go, rather than the baseline activation level in the SC.

expectancy was very strong in Simulation 3, network activity quickly crossed the saccade threshold. As a result, the SRT difference between cued and uncued targets (IOR) was diluted (Figure 4.5H). Nevertheless, the landing position of averaging saccades still deviated away from cued locations (Figure 4.5I), reproducing the pattern of results of Experiment 4.1.

Limitations of our modeling work: With careful selection of parameters, our neural field model can generate a pattern of results with saccade landing position deviating toward the cued location in double-target trials when behavioral facilitation (11 ms) is observed on single-target trials (see first row of Figure 4.5, Simulation 1), as suggested by Satel et al. (2011). Our failure to confirm this prediction in our behavioral experiments may be due to weak cue-related activation (see Figures 4.5D & 4.5G). As shown in Figure 4.5, our model can successfully simulate the short CTOA conditions of our experiments. However, when the same implementation was extended to the 600 ms CTOA condition of Experiment 4.2, we were unable to reproduce the full pattern of SRTs observed behaviorally. Using the parameters of Simulation 2, our model was able to generate IOR (12 ms) and saccade deviation away from cued locations (0.25°) at a 600 ms CTOA, replicating the pattern of simulation results reported in Satel et al. (2011) and approximating the pattern observed in our Experiment 4.2's long CTOA condition. However, the simulated SRTs for a 600 ms CTOA were not faster than those for a 50 ms CTOA, which conflicts with the findings of our Experiment 4.2. Factors that may affect SRTs at relatively long CTOAs should be explored in future computational studies of IOR. We decided at this point that further manipulation of parameters across CTOAs to

try and match behavior perfectly was unwarranted without further empirical data. More importantly, here we are implementing a 2-dimensional phenomenon with a 1-dimensional model, in which the fixation activity at the rostral pole of the SC cannot be considered. Future modeling work incorporating the use of a 2-dimensional model is strongly encouraged.

4.5.2 Alternate theoretical interpretation of behavioral results

Although the neural field model we used implements a top-down component representing cognitive factors, such as expectation, little neurophysiology is known about this top-down component. Here, we want to emphasize that there are successful cognitively-based models that, though lacking mathematical explicitness, can account for the present data set. As we see it, the most apparent and parsimonious cognitive model makes four assumptions: 1) Attentional facilitation and IOR act in parallel when the interval between a cue and a target is brief (cf. Posner & Cohen, 1984) and there is no luminance change at fixation to rapidly disengage attention from the cued location (Briand, Larrison, & Sereno, 2000), or incentive to voluntarily disengage attention (Klein, 2000); 2) If, especially at relatively short CTOAs (Dorris et al., 2002; Fecteau & Munoz, 2005), IOR is truly reflected by a low-level sensory adaptation effect that cascades from the superficial to the intermediate layers of the SC (Fecteau & Munoz, 2005), then the magnitude of IOR is assumed to be relatively unaffected by task demands, whereas the magnitude of attentional facilitation can vary with task demands (e.g., Klein, 2000; Klein, 2004); 3) SRTs are reduced at long CTOAs in mixed CTOA designs owing to increased certainty that a target will occur after the first CTOA elapses (Correa, Lupiáñez, Milliken,

& Tudela, 2004; Gabay & Henik, 2010; Snyder & Kingston, 2001); and 4) A failure to voluntarily disengage attention from, or endogenously sustain attention at, the cued location has no apparent effect on the landing sites of saccades that are exogenously generated by way of two spatially proximal onset stimuli.

The first and second assumptions account for statistically significant IOR at the short CTOA in Experiment 4.2, and the absence of such an effect in Experiment 4.1. In this case, and to reiterate, we assume that the magnitude of the STD effect is the same in the 50 ms CTOA conditions of both experiments, whereas the magnitude of attentional facilitation is free to vary between experiments. One potential reason for increased attentional facilitation in a blocked CTOA design is that the cue onset reliably predicts when the target will occur, excepting the rare catch trial. Thus, in Experiment 4.1, the observer might – at least on some proportion of the trials – sustain attention at the cued location in an effort to gain temporal certainty about target onset time or, alternatively, fail to voluntarily disengage exogenously captured attention from an uninformative cue. By contrast, in the mixed CTOA design, the cue loses a substantial amount of its temporal predictability. If, in the mixed CTOA design, attention were more rapidly disengaged from the cued location, owing to its comparably diminished temporal uninformativeness, similar magnitudes of IOR would be obtained at the short and long CTOAs.

The third assumption reflects the ubiquitous finding that when a short and somewhat longer foreperiod are randomly intermixed, reaction times are significantly reduced at the longer foreperiod (Kingstone, 1992); consequently, it accounts for this finding in our Experiment 4.2. The fourth assumption maintains that there is no effect of

either delayed disengagement or endogenously sustained attention at a cued location. This assumption is supported by the observation that there were no statistically significant differences in the magnitude of the landing site deviation on double target trials in both experiments, despite evidence that the IOR effect on SRTs was different between experiments.

4.6 Conclusion

On a behavioral level, our findings seem to contradict the claim that exogenous cueing affects averaging saccades since averaging saccades consistently deviated away from the cued location, in similar magnitudes, in the absence (Experiment 4.1) or presence of IOR (Experiment 4.2). The same pattern of results was produced in our simulations when we assumed that Experiment 4.1 fostered a strong target expectancy. When this top-down target expectancy was reduced by mixing short and long CTOAs in Experiment 4.2, the close relationship between IOR and deviation in averaging saccades re-emerged (see also Simulation 2). This computational explanation, although notably successful when predicting behavior at short CTOAs, fails at longer CTOAs.

We can also account for the observed pattern of results at the short CTOAs by assuming that the utility of the cue is increased in Experiment 4.1 as compared to Experiment 4.2 which, in effect, increases the amount of attentional facilitation competing with IOR/STD for behavioral expression. One must also assume, however, that the amount of attentional facilitation has no apparent effect on the magnitude of the saccade landing site deviation when paired targets automatically elicit an averaging saccade. So long as this latter assumption is preserved, this cognitive model accounts

neatly for the observed data. This theory, although notably successful when predicting the range of oculomotor behavior shown here, must await advances in neurophysiological research on cueing before making the idea computationally explicit in a neurophysiologically-based neural field model.

In both models, it is possible to maintain a reliable effect of IOR on the metrics of exogenously generated saccadic eye movements despite differences between experiments on the magnitude of the IOR effect on SRT, by assuming that some form of attention (whether it be non-spatial temporal orienting or spatial orienting to a temporally informative cue) has an asymmetric modulatory effect on the temporal and spatial dynamics of oculomotor processing (Theeuwes & Godijn, 2004; Smith & Henderson, 2011). In this vein, the modulatory attentional effect has a more pronounced effect on the temporal dynamics of oculomotor processing to a single exogenous target than on the spatial dynamics of oculomotor processing to paired exogenous targets. Although we cannot be sure precisely how these effects are occurring on a biomechanical level from behavioral observation alone, the models and data presented here support the proposition that exogenous cueing has a general effect of biasing an averaging saccade against a cued location, a finding that supports that claim that IOR can function effectively and generally as a novelty seeking mechanism in visual foraging.

4.6 Acknowledgements

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CHAPTER 5 SENSORY AND MOTOR MECHANISMS OF OCULOMOTOR INHIBITION OF RETURN

Wang, Z., Satel, J., and Klein, R. M. (2012). Sensory and motor mechanisms of oculomotor inhibition of return. Experimental Brain Research, 218(3): 441-453 (reformatted and reprinted with permission of the publisher, Springer).

5.1 Abstract

We propose two explicit mechanisms contributing to oculomotor inhibition of return (IOR): sensory and motor. Sensory mechanism: Repeated visual stimulation results in a reduction of visual input to the superior colliculus (SC); consequently, saccades to targets which appear at previously stimulated retinotopic locations will have longer latencies than those which appear at unstimulated locations. Motor mechanism: The execution of a saccade results in asymmetric activation in the SC; as a result, saccades which reverse vectors will have longer latencies than those which repeat vectors. In the IOR literature, these two mechanisms correspond to IOR effects observed following covert exogenous orienting and overt endogenous orienting, respectively. We predict that these two independent mechanisms will have additive effects; a prediction that is confirmed in a behavioral experiment. We then discuss how our theory and findings relate to the oculomotor IOR literature.

5.2 Introduction

Inhibition of return (IOR), first discovered by Posner and Cohen (1984) using a cue-target paradigm, is a behavioral effect originally characterized by slower response times (RTs) to cued than to uncued targets (for a review, see Klein, 2000). In the model (cue-target) task pioneered by Posner and Cohen (1984; see Figure 5.1A for an illustration), an uninformative peripheral cue is ignored by the participant. Subsequently, various investigators required responses (manual and/or saccadic) to both the cue and target (for a review, see Taylor & Klein, 1998). In these target-target experiments, the cue and the target can control orienting exogenously (as when the target is a peripheral visual

onset) or endogenously (as when the target is an arrow at fixation). Considering both the cue-target and target-target paradigms, and the use of exogenous and endogenous cues and targets, Taylor and Klein (1998) noted that there were 24 possible combinations (2 types of the cue [exogenous or endogenous] × 3 response possibilities to the cue [no response, manual, or saccadic response] × 2 types of target [exogenous or endogenous] × 2 response possibilities to the target [manual or saccadic response]), all of which were tested by Taylor and Klein (2000; see Figure 5.9 for a summary of their findings). Regardless of the underlying neural mechanisms elicited by these variations of experimental tasks, the behavioral effects (slower responses to cued targets) observed have been labeled as IOR by scholars. Partially because scholars have extended the functional significance of IOR to the area of visual search (Klein, 1988; Klein & Macinnes, 1999; Snyder & Kingstone, 2000; Tipper, Weaver, Jerreat, & Burak, 1994), slower return (overly or covertly) to previously inspected locations during search is also labeled as IOR (for a review, see Wang & Klein, 2010). Because IOR is a term used loosely by scholars (see Berlucchi, 2006, for a discussion), in the present paper we regard IOR as a behavioral effect in the context of orienting, both covert and overt. But, despite the singular name, we will propose and then demonstrate two distinct and physiologically plausible neural mechanisms underlying the behavioral manifestation of IOR when measured with eye movement responses.

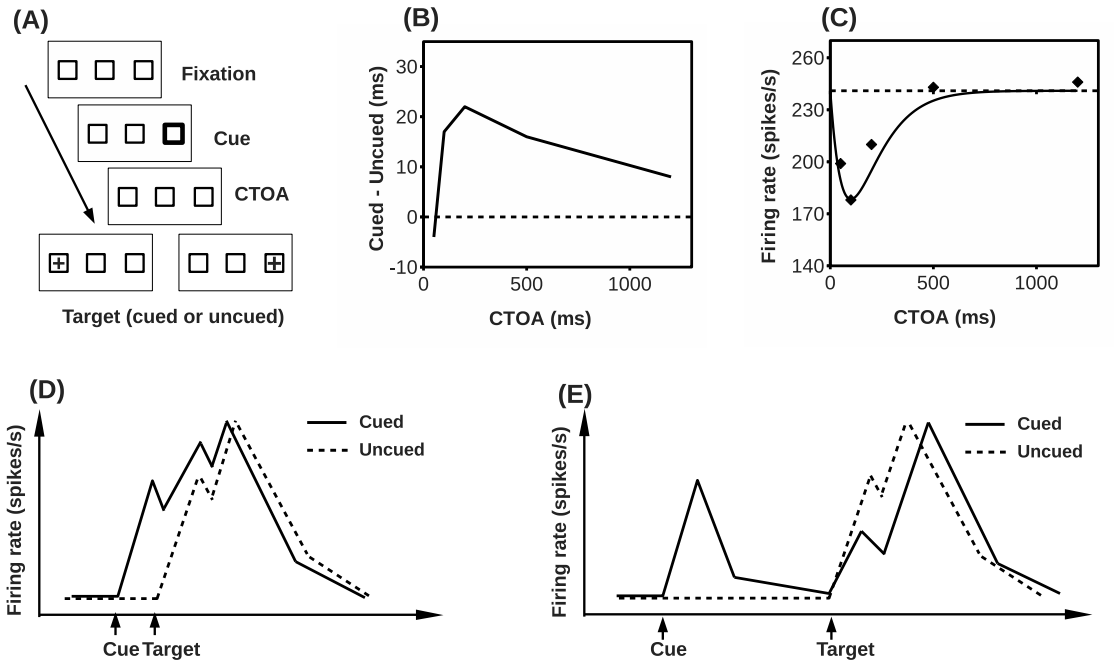

Figure 5.1: (A) The classic cue-target paradigm. (B) Typical behavioral findings in the cue-target paradigm (monkey behavioral data adapted from Fecteau & Munoz, 2005). Faster responses to cued targets are observed at short CTOAs (facilitation) and slower responses to cued targets are observed at long CTOAs (IOR). (C) Diamonds denote the response of visual neurons to cued targets (adapted from Fecteau & Munoz, 2005). Dashed line denotes the average firing rate of visual neurons to uncued targets. An alpha function with parameters, A = -63, t_{max} = 100 ms, was used to fit these cell recordings. (D) and (E) Schematic cell activity during cued (solid) and uncued (dashed line) trials in the SCi. The last peak in both figures denotes a hypothetical endogenous "move" signal that projects to the SC once a visual target is detected.

Unlike theorists who strive to explain all the findings in the IOR literature, we have confined our theory to only a subset of the experimental paradigms explored by Taylor and Klein (2000). In contrast to manual responses, the neural implementation of saccadic responses is relatively well-known and neural models of the oculomotor system, notably the superior colliculus (SC), are widely available. With the aim of developing a computationally explicit theory of IOR, we have so far confined our theory of IOR to experimental paradigms that involve only saccadic responses to the targets, or to both

cues and targets. Furthermore, our theory is closely tied to neurophysiological studies of the monkey oculomotor system, leading to our use of the term "oculomotor IOR" in the present paper (for a recent review of "oculomotor IOR", see Klein & Hilchey, 2011).

5.3 Sensory and motor mechanisms: A theory

Based on existing behavioral and neurophysiological evidence, we propose that there are two physiologically plausible mechanisms of IOR: sensory and motor. Furthermore, these two mechanisms has been explored computationally with dynamic neural field (DNF) models (Wilson & Cowan, 1973; Amari, 1977) of the SC (Satel, Wang, Trappenberg, & Klein, 2011; Wang, Satel, Trappenberg, & Klein, 2011), providing precise theoretical descriptions from which explicit predictions follow that can drive further empirical research, both behavioral and neurobiological.

5.3.1 Sensory mechanism (Satel et al., 2011)

There are several lines of evidence supporting the idea that, as originally proposed by Posner and Cohen (1984), an IOR effect can be caused by peripheral visual stimulation. The most compelling evidence comes from single-unit recordings of SC neurons while monkeys are performing the classic Posner cueing task (see Fecteau & Munoz, 2006, for a review). Dorris, Klein, Everling, and Munoz (2002) showed that the activity of neurons in the intermediate layers of the SC (SCi) was reduced for cued, as compared to uncued targets. This reduction of neuronal activity was highly correlated with IOR, as measured with saccadic response times (SRTs) to cued and uncued targets. Importantly, when electrical stimulation was delivered through the recording electrode to elicit a saccade, shorter latency was observed for saccades to previously stimulated (cued)

regions. These findings suggest that this form of IOR is not characterized by active local inhibition of previously stimulated (cued) SCi neurons, but rather by a reduction of the strength of visual input to these neurons, also referred to as sensory short-term depression (STD). Confirming this hypothesis, Fecteau and Munoz (2005) observed a reduction of neuronal activity of cells in both the superficial (SCs) and intermediate layers of the SC. Unlike the SCi, which receives inputs from both the visual cortex as well as from higher cortical areas, the SCs only receives inputs from early visual pathways (Fecteau & Munoz, 2005). These findings are consistent with a form of IOR that simply "reflects a habituated sensory response occurring in early sensory areas that is subsequently transmitted through the rest of the brain" (Fecteau & Munoz, 2005, p 1772).[2] Furthering this line of thought, Dukewich (2009) proposed that IOR was the result of habituation-like processes that can take place at any level of processing and in different brain areas (for a discussion of similar ideas, see Huber, 2008; Patel, Peng, & Sereno, 2010).

To sum up, the sensory mechanism of IOR states that, in a typical "cue-target" paradigm (see Figure 5.1A), when targets appear at previously cued locations, the target-elicited visual ***input to the SC*** will be **reduced** for some period of time**,** because the location was previously stimulated. As a result, responses made to cued targets in the cue-target IOR paradigm are slower than those made to uncued targets (see Figure 5.1B). The time course of this input reduction as a function of the cue-target onset asynchrony

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[2] Human electrophysiological (EEG) studies also showed that target-elicited early visual ERP components (P1 and N1) are reduced for cued targets (see Prime & Ward, 2006, for a review). Reduced BOLD signal to cued targets has also been observed in human visual cortex (e.g., Anderson & Rees, 2011).

(CTOA) can be expressed as an alpha function[3]:

$$\alpha_{ctoa} = A \frac{t_{ctoa}}{t_{max}} e^{(1-\frac{t_{ctoa}}{t_{max}})}$$

where the parameters A and t_{max} specify the maximal input reduction and the time at which this input reduction reaches its maximum, respectively. Although this function has only two parameters, computational simulations (Satel et al., 2011) have demonstrated that it can be used to nicely fit cell-recordings (see Figure 5.1C), as well as behavioral results (see Figure 5.1B).

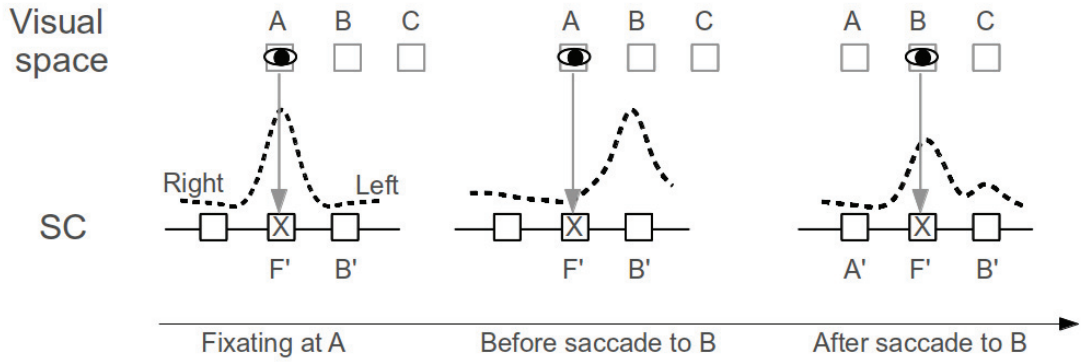

Figure 5.2: Remapping results in asymmetric activation in the SCi. Eyes mark the fixated spatial location. X's mark the rostral pole of the SCi. For convenience, the right SC is plotted on the left. Reprinted from Wang et al. (2011), with permission. Dashed lines represent hypothetical activation profiles of the motor map in the SCi.

The underlying neuronal mechanisms of the early facilitation and later IOR effects observed in the cue-target paradigm are illustrated in Figure 5.1D and E (for

[3] One could always use two differential equations to approximate this input reduction, but we chose to use the alpha function for simplicity.

similar illustrations, see Bell, Fecteau, & Munoz, 2004; Dukewich, 2009). At short CTOAs, although the visual input for cued targets is reduced (see the slope of the second peak in Figure 5.1D), because the visual response to the cue has not yet died out, faster SRTs are observed. At longer CTOAs (Figure 5.1E), because input for cued targets is reduced, the time for the corresponding SCi neurons to reach the saccade initiation threshold is prolonged, resulting in slower SRTs.

5.3.2 Motor mechanism (Wang et al., 2011)

Saccade-related neurons in the SCi include fixation and buildup neurons. Fixation neurons, responsible for active fixation, are located at the rostral pole of each colliculus (Munoz & Wurtz, 1993). Buildup neurons, responsible for saccade initiation, are located more caudally (Munoz & Wurtz, 1995a; Munoz & Wurtz, 1995b). These saccade-related SCi neurons are organized into a "winner-take-all" motor map that encodes the direction and amplitude of saccades into the contralateral visual field. More importantly, this map is oculocentric, and remaps to the visual space after each saccade. This passive remapping process was described and modeled by Wang et al. (2011) and is illustrated in Figure 5.2. When the eyes are actively fixating a location in visual space (e.g., A in Figure 5.2), fixation neurons at the rostral pole of the SCi (F') discharge tonically and take over the network. To initiate a saccade to another spatial location (e.g., B in Figure 5.2), inputs (which can be either exogenous or endogenous) arrive at neurons in the SCi representing this location (B' in Figure 5.2). Shortly before a saccade to the new location (B) is initiated, the neuronal activity at B' in the SCi approaches, and eventually exceeds, the threshold for initiating a saccade. After the saccade is executed, neurons in the SCi are

remapped to the new foveal location, which was the target of the saccade and is now represented by firing of fixation neurons at the rostral pole. Thus, the neurons in the SCi that originally drove the saccade (B' in Figure 5.2) now represent a new spatial location (C in Figure 5.2) which is, relative to the new fixation, in the same direction and of the same amplitude as the previous saccade. Although the discharge of fixation neurons at the rostral pole (F') starts to increase shortly before the saccade is completed, neuronal activity at B' does not die out immediately. In our model of the remapping process (see Wang et al., 2011), this leftover activity leads to asymmetric activation in the SCi and, as a result, saccades in the forward direction are facilitated, while those directed back to the vicinity of the previous fixation location (reverse vector), due to lateral inhibition, are impeded (for similar ideas, see Klein & Macinnes, 1999; Smith & Henderson, 2009; Smith & Henderson, 2011a).

This remapping process defines the "motor" mechanism of oculomotor IOR.[4] That is, following a saccade, due to **asymmetric** *activation* in the SCi, saccades that repeat vectors will be initiated faster than those that reverse vectors. We propose that this remapping process is the neural mechanism (Wang et al., 2011) that underlies what has been called "inhibition of saccade return" (Hooge & Frens, 2000; Hooge, Over, van

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[4] One might suggest that this mechanism should not be regarded as a contributor to IOR because the forward facilitation it generates (saccade momentum) causes a disadvantage for return saccades. Although such a suggestion has merit, so too does our maintenance of the IOR terminology. Our previous modeling papers (Satel et al., 2011; Wang et al., 2011) have addressed two extreme situations where the behavioral effects had been, by convention in the field, labeled as IOR: a) covert orienting where IOR is elicited by uninformative peripheral cues while the participant maintains fixation until target presentation; b) overt orienting where IOR is used to label longer latencies for saccades returning to previously fixated locations (e.g., Klein & MacInnes, 1999). We prefer to continue to use "IOR" to label both cases because we are following the "convention" in the field where IOR is used to loosely refer to a set of processes that facilitate orienting to novelty. The importance of our contribution rests not on terminology but on the computationally explicit nature of the sensory and motor mechanisms we have proposed to implement these processes.

Wezel, & Frens, 2005), "saccadic momentum" (Smith & Henderson, 2009; Smith & Henderson, 2011a; Smith & Henderson, 2011b) and some forms of IOR (e.g., Klein & MacInnes, 1999; Taylor & Klein, 2000).

One critical aspect of this motor mechanism is that it is short-lived: the **activity** caused by the execution of saccades **decays rapidly**, although the precise spatiotemporal dynamic of the asymmetric activation in the SCi depends on the size (amplitude) of the saccades (*cf.* Wang et al., 2011). Following small saccades, the residual activity will not only merge with the activity at the rostral pole, it will even drag the activity at the rostral pole toward itself, leading to a longer lasting asymmetric state. However, following large sized saccades, the residual activity will decay more quickly.

5.3.3 Computational modeling

As mentioned above, we have implemented the aforementioned sensory (Satel et al., 2011) and motor (Wang et al., 2011) mechanisms of IOR using a computational model of the SC.

The interaction between the SCi neurons is characterized by short-distance excitation and long-distance inhibition (for a review of related evidence, see Munoz & Fecteau, 2002). Such dynamic lateral interaction has a Mexican hat shape (Trappenberg, Dorris, Munoz, & Klein, 2001; Dorris, Olivier, & Munoz, 2007) and can be captured using DNF models (Wilson & Cowan, 1973; Amari, 1977). Trappenberg et al. (2001) demonstrated that by tuning such a model to closely reflect cell activity patterns in the SCi of monkeys, this model could reasonably reproduce a wide variety of behavioral data in both humans and monkeys. Following this approach, we have extended this model to

explore the sensory and motor mechanisms of IOR. In Satel et al. (2011), we explored the sensory mechanism of IOR in the DNF model. In addition to the classic physiological (for a review, see Fecteau & Munoz, 2006) and behavioral effects of IOR, a range of behavioral phenomena were reproduced with our DNF model, including how IOR affects averaging saccades (Watanabe, 2001) and how predictive information affects the behavioral manifestation of IOR (Bell & Munoz, 2008). In Wang et al. (2011), we explored the motor mechanism of IOR in the DNF model and simulated various behavioral findings that have been attributed to inhibition of saccade return (Hooge & Frens, 2000; Hooge et al., 2005), saccadic momentum (Smith & Henderson, 2009; Smith & Henderson, 2011a; Smith & Henderson, 2011b) and IOR in oculomotor search (Klein & Macinnes, 1999; Macinnes & Klein, 2003).

5.3.4 Reference frame

Previous studies, **on a behavioral level**, have suggested spatiotopic (e.g., Maylor & Hockey, 1985), retinotopic (Mathôt & Theeuwes, 2011; Pertzov, Zohary, & Avidan, 2011) and even object-based (e.g., Tipper et al., 1994) coding for IOR. So what are the coordinates of the two IOR mechanisms presented here? The motor mechanism is about the internal dynamics of the SC, and on a behavioral level, it appears to be spatiotopic. The sensory mechanism is about STD in the early visual pathway elicited by visual stimulation which, by its nature, operates in retinotopic coordinates.

5.3.5 Predictions

Because our theory of IOR is confined to the oculomotor system, manual responses to cues and targets are not considered here. With this simplification, our theory

can generate clear predictions for three different IOR tasks (which are illustrated in Figure 5.3) from the literature. For convenience, we label these three tasks as S (sensory), M (motor), and SM (sensory-motor) tasks. In the S task, peripheral visual stimuli are presented as cues and targets while participants are required to maintain fixation until target onset. The IOR effect in the S task is purely sensory because only the sensory mechanism (repeated stimulation) is evoked in this task. This is the traditional Posner cue-target paradigm. In the M task, participants are required to saccade in response to an endogenous cue, saccade back to central fixation, then saccade to an endogenous target. The IOR effect in the M task is purely motor because no peripheral stimulation is involved. In the SM task, as in the S task, both the cues and the targets are peripheral stimuli. However, this task differs from the S task in that participants are required to saccade to the peripheral cue and saccade back to central fixation before the final saccade to the peripheral target. Consequently, the IOR effect in the SM task involves both the sensory and motor mechanisms.

Our predictions regarding these three IOR experimental paradigms are as follows:

Prediction 1: Due to the temporal characteristics of the sensory mechanism, the IOR effect in the S task will change with the time interval between the cue and the target (CTOA), and the time course should roughly mimic the alpha function (see Figure 5.4).

Prediction 2: According to the motor mechanism, the IOR effect in the M task is caused by facilitation of the repeated vector of the saccade back to central fixation. Therefore, in contrast to our prediction for the S task, the IOR effect in the M task should change with the time interval between the saccade back to fixation and the target (SOA2),

rather than the CTOA. Based on single-unit recording data from monkeys, our model predicts that this effect decays relatively quickly (see Figure 5.5).

Prediction 3: The IOR effect in the SM task should be larger than that in either the S or M tasks (so long as SOA2 is relatively short) and, to the extent that additive factors logic (Sternberg, 1967) applies, should be roughly the sum of the IOR effects in the S and M tasks.

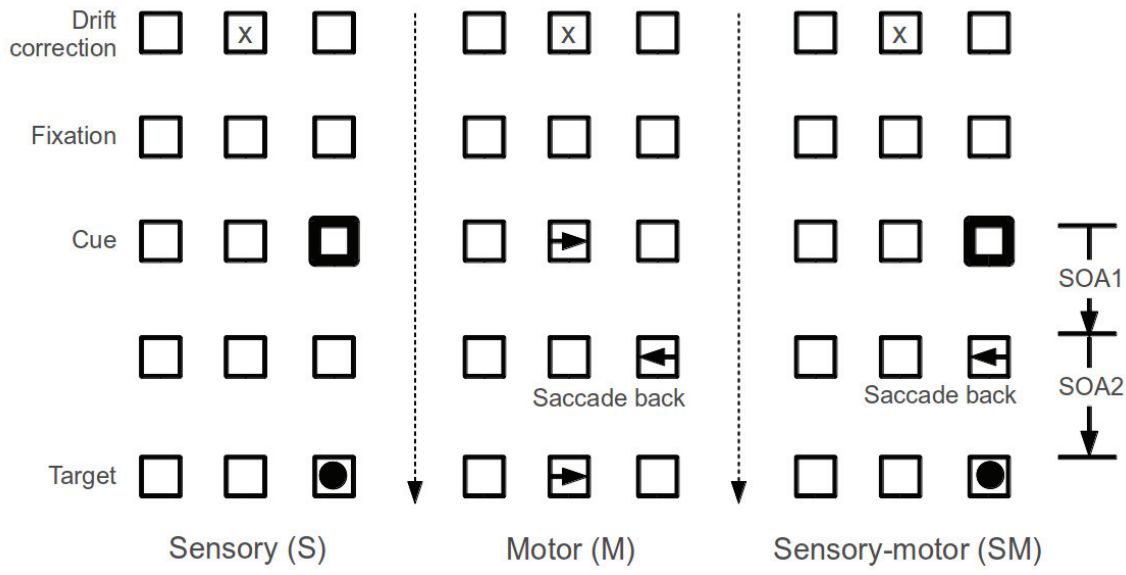

Figure 5.3: Experimental design used to test the predictions of our 2-mechanism theory of IOR. In the S and SM tasks, both the cue and the target are peripheral visual onsets. In the M task, the cue and the target are arrows presented at fixation. The S task only involves a saccade to the target, while saccades to the cue are also required in the M and SM tasks. See text for details.

5.4 Testing the predictions using the IOR literature

Previous researchers have done extensive work related to the three predictions presented in the previous section, so we now present a graphical review of related

literature to identify theoretically important issues for further experiments.

Prediction 1, which is about the sensory mechanism, is well supported by the existing literature (Dorris, Klein, Everling, & Munoz, 1999; Dorris et al. 2002; Samuel & Kat, 2003; Bell et al., 2004; Fecteau, Bell, & Munoz, 2004; Fecteau & Munoz, 2005). A graphical review of this literature appears in Figure 5.4, along with the results of our DNF simulations. As can be seen in this figure, our simulations fit monkey behavioral data nicely. Thus, Prediction 1, which requires the variation of CTOAs was not tested in the present study.

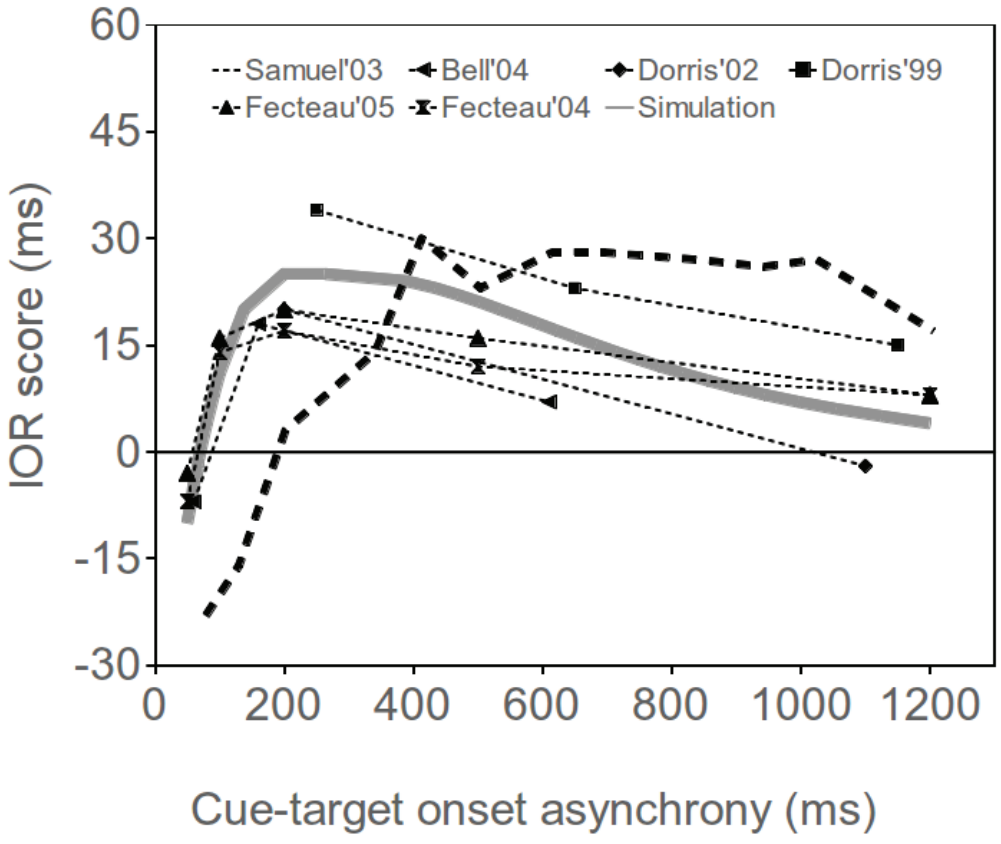

Figure 5.4: Monkey and human behavioral findings in cue-saccade (S) tasks, along with simulation results. Data points from the same study are connected by thin dashed lines. Samuel and Kat (2003) did a graphical review of human behavioral findings in the cue-target paradigm. Data from their review (their Figure 1, bottom panel) is represented with a thick dashed line.

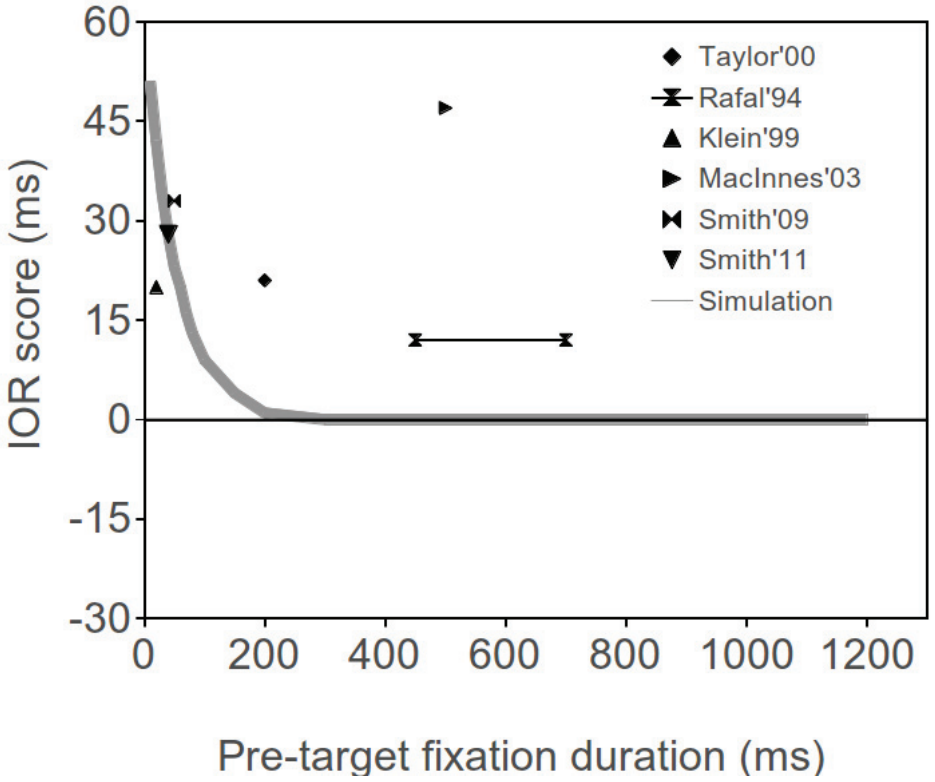

Figure 5.5: Graphical review of human studies that involved only the motor mechanism and accompanying simulations. Data points from the same study are connected by dashed lines. The two data points from Rafal, Egly, and Rhodes (1994) are connected by solid line to denote that the authors randomly varied the pre-target fixation duration between about 450-700 ms. The IOR score for Smith and Henderson (2009) was calculated from the fixation durations for visual onsets presented at the immediately preceding fixation or a location 180° (angular distance) from the immediately preceding fixation (see their Figure 3). The pre-target fixation durations for Smith and Henderson (2009) was about 40 ms (Smith, personal communication, 2011 February). This duration was not recorded in Taylor and Klein (2000; Taylor, personal communication, 2010 November), so the value used in this plot is our best estimate.

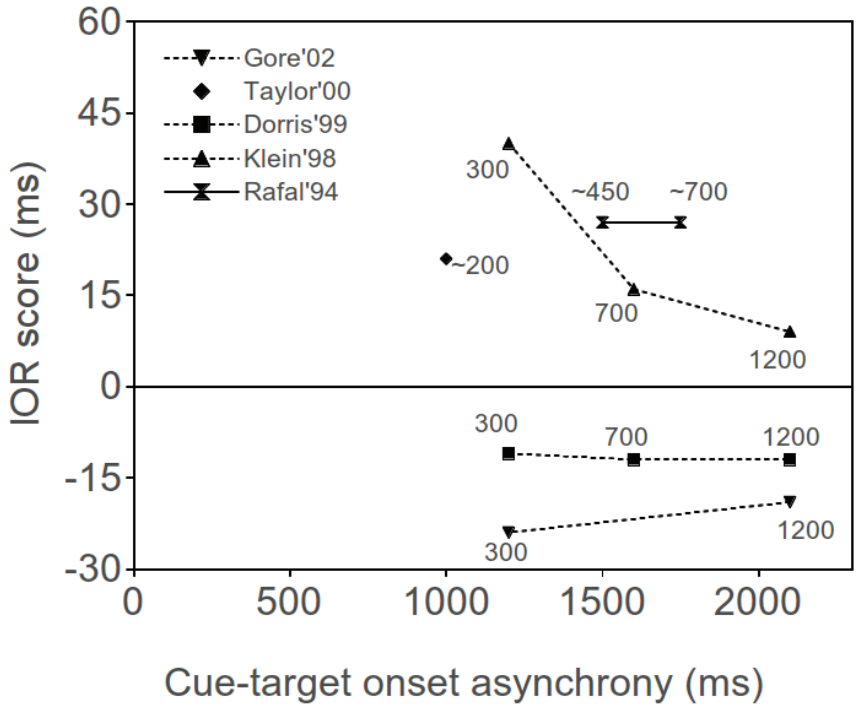

Figure 5.6: Summary of behavioral studies (with monkey and human subjects) that recruited both the sensory and motor mechanisms. Values in the figure are pre-target fixation durations (which affect the manifestation of the motor mechanism) for each data point. As before, data points from the same study are connected by dashed lines. Note that the pre-target fixation duration in Taylor and Klein (2000) and Rafal et al. (1994) were not reported by the authors, so the values in this figure are our best estimates.

Prediction 2 is about the motor mechanism. To our knowledge, this prediction has not been tested with monkeys. However, quite a few human studies have explored this mechanism (Rafal, Egly, & Rhodes, 1994; Klein & Macinnes, 1999; Taylor & Klein, 2000; Macinnes & Klein, 2003; Smith & Henderson, 2009; Smith & Henderson, 2011a), although not with the same rationale and purpose as we have presented here. A graphical review of this line of research and accompanying simulations are presented in Figure 5.5. As shown in this Figure, our simulations predict that the IOR effect caused by the motor mechanism should decay quickly and exponentially. However, in the literature, motoric

IOR effects have been reported at relatively long pre-target fixation durations (Rafal et al., 1994; Macinnes & Klein, 2003). Possible reasons for this discrepancy will be addressed in the General discussion. One of the purposes of the present study is to explore how the motor mechanism changes with pre-target fixation durations (SOA2 in Figure 5.3) in humans.

A graphical review of behavioral studies (Rafal et al., 1994; Klein, Munoz, Dorris, & Taylor, 1998; Dorris et al., 1999; Gore, Dorris, & Munoz, 2002) that involved both the sensory and motor mechanisms is presented in Figure 5.6. The most prominent finding illustrated in this figure is that, instead of IOR, which is regularly observed in studies with human participants, monkeys produced significant facilitation effects in this condition at long CTOAs (Dorris et al., 1999; Gore et al., 2002). This finding is predicted neither by the sensory nor by the motor mechanism. One unique property of these two studies is that the pre-target saccade is guided by a visual stimulus (the "cue-back") at the original central fixation point. This "cue-back" is presented at the same retinotopic location as the subsequent target in "uncued" trials. That is, neurons representing the retinotopic location of "uncued" targets were actually cued by the "cue-back" at central fixation. The sensory mechanism predicts that this stimulation will lead to attenuation of target-related input, and that this input attenuation decays with time. Thus, the input attenuation for "uncued" targets (caused by the "cue-back") should be larger than the input attenuation for "cued" targets (caused by the "cue"), at the time of target appearance. As a result, SRTs to cued targets are faster than those to uncued targets. Simulations of Dorris et al. (1999) which support this explanation are presented in Figure 5.7. However, this explanation cannot

account for the human behavioral findings (Rafal et al., 1994; Klein et al., 1998), unless we assume that the motor mechanism in humans decays much more slower than in monkeys, or that a cortical mechanism in humans extends the effect of the motor mechanism (see General discussion).

The most interesting prediction of our theory is that the sensory and motor mechanisms will have additive behavioral effects because they are operating at different (input and output, respectively) stages of processing (Sternberg, 1967). We are aware of only one study that included versions of each of the three tasks (i.e., S, M and SM) necessary to test this additivity prediction. Among the 24 conditions tested by Taylor and Klein (2000), the three that correspond to these conditions revealed IOR effects in the "S", "M" and "SM" tasks of 11 ms, 21 ms and 21 ms, respectively (see Figure 5.9). These numbers do not agree with our additivity prediction. Note, however, that the methodological confound in Dorris et al. (1999) and Gore et al. (2002) was also present in Taylor and Klein (2000). Namely, after the saccade to a cue, a "cue-back" was used to return gaze to the central, straight-ahead position. Thus, the pre-target visual stimulation at the original central fixation point contaminated the SRTs to uncued targets (which is the baseline used to calculate IOR scores) and therefore may have led to an underestimation of the IOR effect in the "SM" task.

5.5 Testing the predictions experimentally

The primary purpose of the experiment described in this section is to test the additivity prediction while removing the untoward methodological feature of a central "cue-back". Our review of studies that recruited the motor mechanism showed that,

contrary to our model, the motor mechanism in human subjects could be relatively long lasting. A second purpose of the present experiment is to clarify this issue by manipulating the pre-target fixation duration (SOA2 in Figure 5.3).

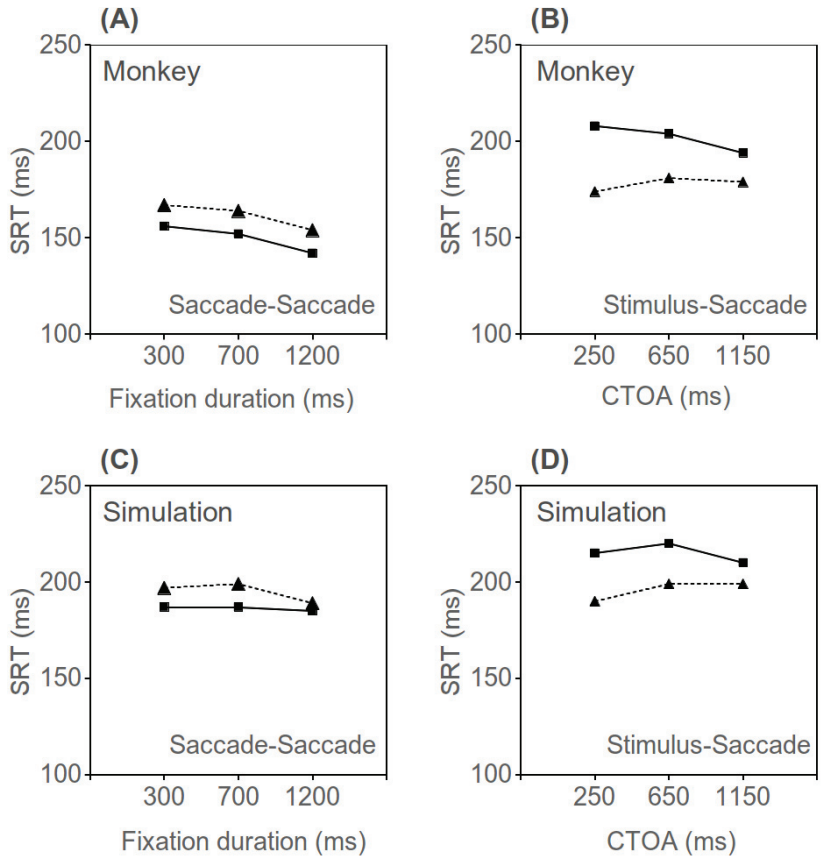

Figure 5.7: Findings of Dorris et al. (1999) and corresponding simulation results. The "stimulus- saccade" task used in this study was the same as the "S" task, monkeys maintained fixation until a target was presented on the screen. The "saccade-saccade" task was the same as the "SM" task except the saccade back from the cued peripheral location was guide by a visual onset at the original central fixation. The top row presents the behavioral findings of Dorris et al. (1999), the bottom row presents the corresponding simulation results. Triangles connected by dashed lines denote SRTs for "uncued" targets while squares connected by solid lines denote SRTs for "cued" targets.

5.5.1 Methods

Participants: Thirteen undergraduate students (4 males) participated for extra course points and reported normal or corrected to normal visual acuity. Seven were assigned to the short SOA2 condition and the rest were assigned to the long SOA2 condition.

Stimuli and apparatus: Gray boxes that subtended 1.8° visual angle were used as placeholders and the thickness of their borders measured 1 pixel. Three boxes were placed horizontally in the center of the display. The distance between the centers of two adjacent boxes was set to 9°. The peripheral cue was implemented as a brightening of one of the peripheral placeholder boxes. The peripheral target was a bright, filled circle, which subtended 1°. The central cue, central target and the peripheral cue-back signal were arrows pointing to the left or right, which measured 0.8° (width) x 0.2° (height).

Visual stimuli were presented on a 17 inch, SVGA, Viewsonic computer monitor with screen resolution set to 1024 × 768 pixels. The visible area of the monitor measured 25° (width) × 19.4° (height) at a viewing distance of 57 cm. The experiment was programmed in Python. An EyeLink® video-based eye-tracking system was used to monitor participants' direction of gaze every 4 ms with a resolution of 0.1° or better.

Design and procedure: The three tasks we used are illustrated in Figure 5.3. In the S task, participants were required to fixate the central box until a peripheral target (bright filled circle) was detected. Peripheral cues were to be ignored. In both the M and SM tasks, saccadic responses were required to both the cue, the cue-back, and the target. These tasks differ in that the cue and target for the M task was a foveally presented arrow

while for the S task they were changes (brightening of a peripheral box or appearance of a filled circle in a peripheral box, respectively) in the periphery. Although the CTOA for these tasks was set to 1500 ms, the time of the cue-back was varied (between subjects) such that the SOA2 was either short (500 ms) or long (1000 ms).

On half of the trials, the target appeared at the cued peripheral box (S and SM task) or pointed in the same direction as the cue (M task). On the other half of the trials, the target appeared at the uncued peripheral box (S and SM task) or pointed in the opposite direction as the cue (M task). For convenience, we will refer to these two types of trials as "cued" and "uncued" trials regardless of the differences between tasks. The target SRT difference between cued and uncued trials in each task was regarded as a measure of IOR. These manipulations yielded a mixed design with SOA2 (short or long) as a between-subject factor and type of task (S, M or SM) and trial type (cued or uncued) as within-subject factors. The three task types were presented in separate blocks of 72 trials (36 cued and 36 uncued) each, and the order of task presentation (S, M, or SM) was varied across participants. Each task was preceded by a 24-trial practice block.

The sequences of events in the S, M and SM tasks are illustrated in Figure 5.3. Drift correction was performed at the beginning of each trial, followed by a fixation display (three gray boxes) which was presented for 500 ms. Participants were required to fixate the central box during this fixation period, then the cue was presented for 300 ms. In the S task, participants ignored the peripheral cue and maintained fixation; in the M task, participants saccaded to the peripheral box pointed to by an arrow presented in the central box; in the SM task, participants saccaded to the peripherally cued box. In the M

and SM tasks, 1000 ms (short SOA2 condition) or 500 ms (long SOA2 condition) after the appearance of the cue, an arrow appeared in the cued box for 300 ms and participants saccaded back to the central box. 1500 ms after the appearance of the cue, in the S and SM task, a bright filled circle (target) appeared in either the cued or uncued box; in the M task, an arrow pointed to one of the peripheral boxes which appeared in the central box.

During the experiment, incorrect eye movements resulted in trials being aborted. Trials were aborted if a saccade was detected during the fixation period, or if a saccade was not made to the cued peripheral box, and then back to fixation, within 500 ms each (in the M and SM tasks). Saccadic responses within 1000 ms were required to the target and failure to respond to the target also resulted in trials being aborted. All aborted trials were recycled, such that every participant performed the same number of trials correctly.

Note that three saccades (to the cued box, back to central fixation and to the target) were required for both the M and SM tasks. In the M task, these saccades were initiated by endogenous stimuli (arrows) presented at current fixation, no peripheral stimulation thus no sensory mechanism was involved in the M task. In the SM task, cues and targets were peripheral stimuli and saccades to these stimuli were required, thus both the sensory and motor mechanisms were invoked in this task.

	S task			M task			SM task		
	Cued	Uncued	IOR	Cued	Uncued	IOR	Cued	Uncued	IOR
SOA2: 500 ms	261	224	37	317	290	27	264	187	77
SOA2: 1000 ms	310	257	53	314	269	45	294	211	83

Table 5.1: Mean SRTs (ms) to cued and uncued targets in the long and short SOA2 conditions of each task.

5.5.2 Results and discussion

Trials in which SRT to the cue, the cue-back signal or the target was less than 100 ms or more than 500 ms were also excluded (7.75%). The participant was required to make a saccade in response to the cue or the cue-back signal; the mean SRTs to the cue and the cue-back signal were 263 ms and 266 ms for the M task and 185 ms and 285 ms for the SM task, respectively.[5] The target SRTs for each condition are summarized in Table 5.1. An ANOVA of the SRTs, with the variables SOA2 (short or long), task (S, M or SM) and trial type (cued or uncued), revealed significant main effects for task [$F(2, 22) = 21.78$, $p < 0.001$], reflecting the difference in SRT to peripheral versus central targets, and trial type [$F(1,11) = 111.20$, $p < 0.001$], reflecting an overall IOR effect. Significant interactions were observed between task and SOA2 [$F(2, 22) = 4.55$, $p < 0.05$], and task and trial type [$F(2, 22) = 11.46$, $p < 0.001$]. The former 2-way interaction occurred because faster target SRTs were observed for the long SOA2 condition of the M task (see Table 5.1); the latter 2-way interaction suggests that the IOR effects differed in the S, M and SM tasks; however, the 3-way interaction was not significant [$F(2, 22) = 0.22$, $p > 0.81$], suggesting that these task differences in IOR were unaffected by SOA2. The mean SRTs for the S, M and SM tasks are presented in Figure 5.8A. Planned comparisons revealed that the IOR effects (Figure 5.8B) for the S task (44 ms) and the M task (35 ms) did not differ [$t(12) = 0.90$, $p = 0.39$], while the IOR effect for the SM task (80 ms) was larger than those for both the S [$t(12) = 3.64$, $p < 0.01$] and M [$t(12) = 4.94$, $p < 0.001$]

[5] Readers may be surprised that the saccade back to the central fixation was no slower than the one to the peripheral box (in response to the cue) in the M task. Note that unlike saccades to the targets of which the direction was unpredictable, saccades made in response to the cue-back signal were always directed to the central box.

tasks. Importantly, the sum of the IOR effects for the S and M task did not differ from the SM task [$t(12) = 0.08$, $p > 0.93$].

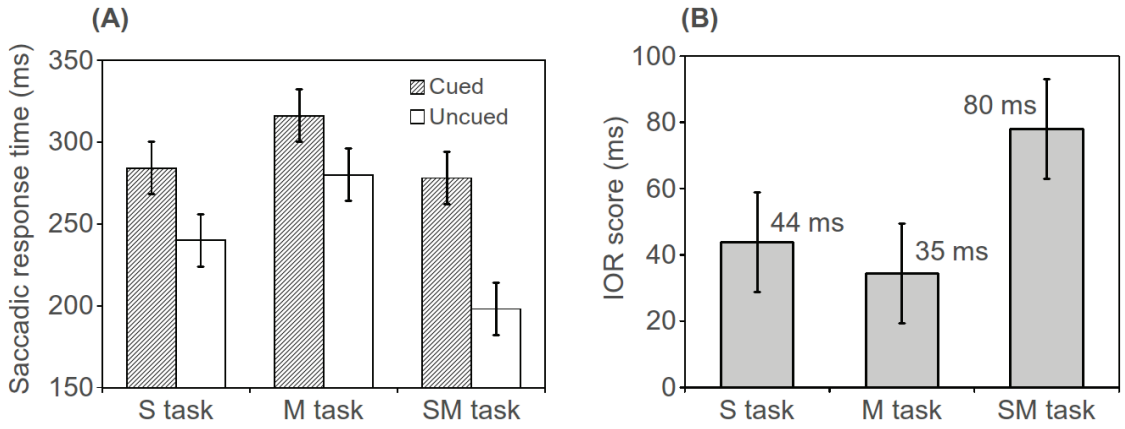

Figure 5.8: (A) Mean SRTs to cued and uncued targets in each task, averaged over short and long SOA2 conditions. (B) Comparison of the IOR scores for the S, M and SM task. Error bars in both figures denote 95% confidence intervals (Masson & Loftus, 2003).

As can be seen in Table 5.1, and as illustrated in Figure 5.8, the IOR effect for the SM task is, in accordance with Prediction 3, approximately the sum of those for the S and M tasks. It appears that the sensory and motor mechanisms outlined above can have additive effects on the behavioral IOR score in a saccade-saccade paradigm with peripheral cues and targets. However, contrary to our prediction 2, the IOR scores in the M and SM tasks (both of which should include the motor mechanism) did not vary with SOA2. Further discussion of this issue is presented below.

5.6 General discussion

5.6.1 Relation of our theory to similar proposals from the IOR literature

The idea that both input and/or output processes may contribute to behavioral IOR

effects is not new. When considering earlier proposals it is important to distinguish (see Taylor & Klein, 1998) whether it is the cause of IOR or its effect on behavior that is presumed to be sensory and/or motor. Abrams and Dobkin (1994) proposed that, once caused, IOR has two component effects: visual and motor. They drew this conclusion from two conditions of an experiment in which IOR was caused by an ignored peripheral cue and then was measured by a saccade made to the cued or opposite location. The key factor was the nature of the imperative stimulus that guided the execution of this saccade. In the "central target" condition, this stimulus was a foveally presented arrow pointing to the cued or opposite location. In the other "peripheral target" condition, this stimulus was the appearance of a peripheral target inside the box that marked the cued or opposite location. Abrams and Dobkin (1994) reasoned that if one effect of IOR was motoric, this component should be present so long as a response was made toward or away from the cued location and therefore this component should be operating whether the target was a stimulus in the periphery or an arrow at fixation. They also reasoned that if another effect of IOR operated on visual inputs, then this effect could only be measured by responses to peripherally presented targets. Abrams and Dobkin (1994) found a small, but significant, amount of IOR in the central target condition (~10 ms) and significantly more IOR in the peripheral target condition (25 ms). From this pattern they inferred that the IOR effect measured by saccadic responses to peripheral targets was the sum of a motor component (10 ms) and a perceptual component (15 ms). Although their logic was sound, their results have not been replicated and their methods have been shown to be flawed (Hilchey, Klein, & Ivanoff, 2012a).

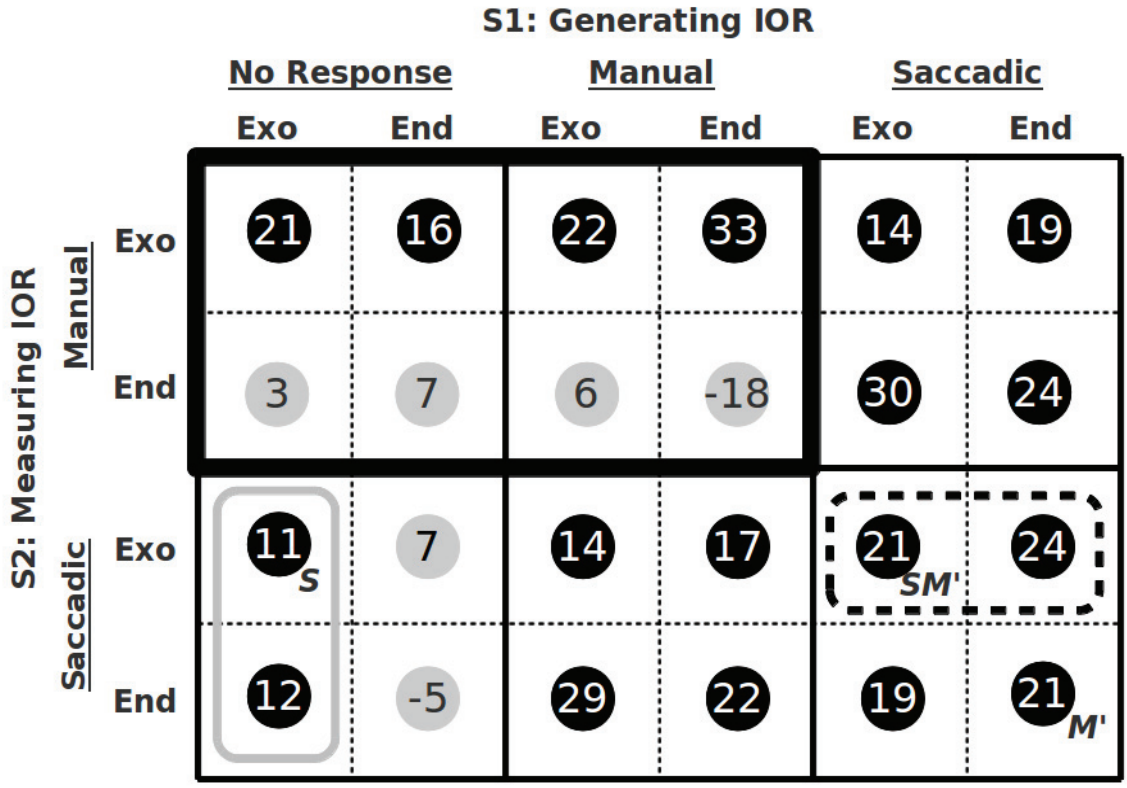

Figure 5.9: The methods and findings from Taylor and Klein (2000). Numbers in the cells are IOR scores observed for each condition; black disks denote IORs reached significance while those in gray denote non-significant IORs were observed. Conditions labeled with S, M' and SM' are those most pertinent to the additivity prediction. Conditions enclosed in the gray box are those from which, in their experiments, Abrams and Dobkin (1994) put forward the proposal that when measured with eye movements, IOR had two components: perceptual and motoric. Hilchey et al. (2012a) demonstrated that these two conditions were entirely motoric. Conditions enclosed by the dashed box are those in which the problematic cue-back to fixation (see text for explanation) was used to shift gaze back to center after the initial eye movement made to the cue (S1). IORs enclosed in the bold black rectangle are perceptual/attentional flavored while the remaining are motoric flavored (cf. Taylor & Klein, 2000).

Among the 24 conditions Taylor and Klein (2000) used to explore the causes and effects of IOR, two (see the data marked by the gray box in Figure 5.9) were essentially the same as those used by Abrams and Dobkin (1994). Suggesting the existence of only a

motor component when the task calls for saccadic responses, Taylor and Klein (2000) found no difference in RT between these conditions. Hilchey et al. (2012a) recently highlighted the fact that the peripheral and central target conditions used by Abrams and Dobkin (1994) were run in separate blocks. When they replicated the conditions used by Abrams and Dobkin (1994), with the exception that they randomly intermixed the central and peripheral targets, they found (replicating Taylor & Klein, 2000) identical amounts of IOR in the two target conditions. They pointed out that blocking the two target types permitted the participants to adopt different attentional control settings in the two blocks and the setting with central targets could have allowed the participants to effectively filter out the uninformative peripheral cues. Randomly mixing the target types is necessary to rule out such a possibility.

In contrast to the components view of Abrams and Dobkin (1994), Taylor and Klein (2000) proposed that there are two different flavors of IOR's effects: perceptual/ attentional and motor. This proposal was based on the pattern of IOR effects illustrated in Figure 5.9. In one subset of conditions, IOR was measurable when the target (S2) was presented in the periphery, but not when the target was an arrow at fixation. These conditions are enclosed in the bold rectangle in Figure 5.9, and they are characterized by the absence of eye movements to both the cue and the target. Because, in these conditions, the IOR could only be measured by a peripheral stimulus, it was assumed to be an effect that was operating either on the input pathway or on the allocation of attention. In the remaining conditions, whenever IOR was observed in response to a peripheral target it was also observed to an arrow at fixation. Moreover, collapsing across

all of these conditions, there was no more IOR with peripheral targets than with central ones, suggesting an IOR effect that was solely about delayed motor responses in the direction of the cue. In all of these conditions, the oculomotor system must have been activated either because a saccade was required to the cue (S1) or the target (S2).

Conceptually, our 2-mechanism theory is quite different from these previous "component" and "flavor" proposals. It may seem similar to the Abrams and Dobkin (1994) proposal, in that both are restricted to oculomotor IOR, but differs in several ways. Perhaps most importantly, our proposal links cause and effect. That is, in our framework, a peripheral stimulus causes suppression of subsequent peripheral signals,[6] and a saccade is followed by a period during which a subsequent saccade with approximately the same vector will be facilitated (and therefore be faster than a return saccade). In contrast, Abrams and Dobkin's (1994) proposal was about the effects upon oculomotor behavior of IOR when caused by an ignored peripheral cue. It is clearly different from Taylor and Klein's (2000) 2 flavors proposal. In their proposal, which incorporates manual and oculomotor methods for measuring IOR, depending on the involvement of the oculomotor system in the overall task, IOR is either motoric or perceptual. In contrast, our theory is specifically about IOR measured with oculomotor behavior and we allow for these two effects to be simultaneously present.

While we are discussing Taylor and Klein (2000) it is, perhaps, worth repeating the criticism we have made of their cue-back procedure. Whenever an eye movement was

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[6] Although the conceptual underpinning seems quite different (rooted as it is in the notion of cue-target integration) Lupianez's proposal of a cue-induced “onset detection cost” (Lupiáñez, 2010; see also Hu, Samuel, & Chan (2011) shares quite a few properties with this suppression effect that is fundamental to our sensory mechanism.

made to their cues (S1; see the dashed box in Figure 5.9), the eyes were signaled back to the original fixation by a stimulus presented there. This stimulus (being in the periphery at the time of its presentation) will, according to our theory, invoke the sensory mechanism of IOR. Because this mechanism is coded retinotopically, after the eyes have returned to the original fixation, targets that are presented at the originally uncued location will be suffering from retinotopic IOR generated by the cue-back. Because only peripheral targets will suffer from this unintentional IOR effect, according to our theory, the IOR scores within the dashed box of Figure 5.9 that are measured by exogenous targets (row 3, column 5-6) underestimate the true amounts of IOR that would be measured if the return cue, like the one we used in the present experiment, did not stimulate the periphery.

In contrast to all other theories of IOR of which we are aware, a major strength of our theory is that it is clearly defined and computationally explicit. These properties are responsible for our ability to generate the precise predictions that we have tested using the literature and the experiment reported here.

5.6.2 Long-lasting motoric IOR?

Based on simulations in a DNF model of the SC, we proposed (Wang et al., 2011) that the motor mechanism is short-lived, because it is based on rapidly decaying residual activity associated with a saccade (see Figure 5.2). This characteristic of the motor mechanism is critical for our successful simulations of the "saccade-saccade" experiment reported in Dorris et al. (1999). Because the motor mechanism is short-lived, it should have dispersed by the time of target presentation in the "saccade-saccade" experiment of Dorris et al. (1999), leaving only the sensory mechanism of IOR to affect the SRTs. Klein

and colleagues (1998) tested human subjects with exactly the same experimental tasks used in Dorris et al. (1999). Unlike monkeys, who produce facilitation in the "saccade-saccade" task, human subjects produce IOR (see Figure 5.6). One way to reconcile this species difference is to assume that the motor mechanism in humans lasts longer than in monkeys so that, by the time of target presentation, it competes with the sensory mechanism (STD) at the uncued location resulting from the cue-back at fixation.

Our manipulation of SOA2 in the M and SM tasks was designed to directly test whether the time course prediction of our motor mechanism also holds true in humans. Disconfirming our prediction, the length of SOA2 did not affect the IOR scores in either the M or SM tasks. There are a few possible explanations for the failure of this prediction with human subjects. It might be, for example, that cell activity decays more slowly in the human SC than in the monkey SC. This is unlikely given the fact that the SC is a fairly primitive subcortical brain structure. Alternatively, it is possible that the human brain has developed a cognitive strategy to take advantage of this mechanism by preparing the SC sites encoding saccades in the forward direction. One consequence of our motor mechanism is that saccades which repeat their vectors are associated with behavioral benefits relative to those which reverse vectors, and such a tendency would discourage immediate re-inspections and encourage saccades in the forward direction. This explanation is in line with the observation that a large portion of saccades are directed in the forward direction during normal search of visual scenes (Macinnes & Klein 2003; Hooge et al., 2005; Smith & Henderson, 2011a). Finally, and related to the last suggestion, perhaps cortical modules outside the SC, such as the frontal eye fields (FEF)

or posterior parietal cortex (PPC), through their projections to the SC, provide the mechanism for this temporal extension of behaviorally exhibited IOR. Further empirical exploration in this direction is encouraged.

5.6.3 Boundary conditions of our theory

As mentioned at the beginning of this paper, the purpose of the present paper is to develop a computationally explicit theory that is capable of explaining a subset of behavioral findings that have been labeled IOR. It is important to acknowledge that the computational theory (model) presented here cannot explain all the findings in the IOR literature.

The sensory and motor mechanisms explored in the present paper can explain the findings of a subset of experimental tasks tested in Taylor and Klein (2000). For the tasks presented in Figure 5.9 (*cf.* Taylor & Klein, 2000), the sensory mechanism contributes to IORs observed in tasks that used peripheral onset cues and peripheral onset targets (where column 1, 3 and 5 and row 1 and 3 meets), the motor mechanism contributes to IORs observed in the tasks that required saccadic responses to both cues and targets (all saccadic-saccadic cells). The IOR effects in other tasks in Figure 5.9 cannot be explained by the present theory.

In tasks with rich visual elements (e.g., visual search), the motor mechanism can explain slower return to immediately preceding fixations (e.g., Klein & Macinnes, 1999). Because the motor mechanism in our SC-only model is short-lived, whether the motor mechanism can explain slower return to fixations beyond the immediately preceding ones (Klein & Macinnes ,1999; Dodd, van der Stigchel, & Hollingworth, 2009) remains to be

explored. In search tasks where maintenance of fixation is required, slower response to probes presented at search item occupied locations has also been labeled IOR (e.g., Klein, 1988). This type of IOR cannot be handled by the present theory. Furthermore, our theory does not explain spatiotopic (e.g., Maylor & Hockey, 1985) or object-based coding (e.g., Tipper et al., 1994) of IOR.

Before closing this paper, we would like to mention that when Posner and Cohen (1984) referred to "an inhibitory effect" of the cue and Posner et al. (1985) named it IOR, they were hypothesizing a "cognitive" inhibition without necessarily implying a specific neural implementation or even inhibition in the neuroscientific sense of this term (*cf.* Klein & Taylor, 1994; Posner, personal communication with R. M. Klein). While our modeling effort as reported here does not include any explicit simulation of neural inhibition, it is possible that some form of neural inhibition will be needed to explain all the findings in the IOR literature.

5.7 Conclusion

Based on the findings of our simulations and behavioral experiment, we have come to the following conclusions. First, the sensory and motor mechanisms have additive behavioral effects, supporting our prediction about multiple dissociable neural mechanisms contributing to behavioral oculomotor IOR. Second, as evidenced by previous human studies and the present investigation, the motor mechanism has relatively long-lasting behavioral effects, suggesting that IOR in humans recruits greater cortical involvement than in monkeys. Limitations of the model demonstrate that a comprehensive theory of IOR will require the incorporation of additional mechanisms

and/or cortical dynamics. Next steps for the field should be to examine the neural dynamics of cortical contributions to IOR and their connections to the current SC-centric model. Further empirical study is encouraged to further elucidate the spatiotemporal dynamics of the neural mechanisms underlying the behavioral exhibition of IOR.

5.7 Acknowledgements

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CHAPTER 6 INVESTIGATING A TWO-CAUSES THEORY OF INHIBITION OF RETURN

Satel, J. and Wang, Z. (2012). Investigating a two-causes theory of inhibition of return. Experimental Brain Research, 223(4): 469-478 (reformatted and reprinted with permission of the publisher, Springer).

6.1 Abstract

It has recently been demonstrated that there are independent sensory and motor mechanisms underlying inhibition of return (IOR) when measured with oculomotor responses (Wang, Satel, & Klein, 2012a). However, these results are seemingly in conflict with previous empirical results which led to the proposal that there are two mutually exclusive flavors of IOR (Taylor & Klein, 2000). The observed differences in empirical results across these studies and the theoretical frameworks that were proposed based on the results are likely due to differences in the experimental designs. The current experiments establish that the existence of additive sensory and motor contributions to IOR do not depend on target type, repeated spatiotopic stimulation, attentional control settings, or a temporal gap between fixation offset and cue onset, when measured with saccadic responses. Furthermore, our experiments show that the motor mechanism proposed by Wang et al. (2012a) is likely restricted to the oculomotor system, since the additivity effect does not carry over into the manual response modality.

6.2 Introduction

The ability to orient efficiently to visual events is critical for an individual to quickly adapt to the ever changing environment. In the laboratory, visual orienting is frequently explored with a cueing paradigm in which targets are preceded by uninformative visual cues (Posner, 1980). Both facilitatory and inhibitory behavioral effects can be observed in such experimental tasks. With a short cue-target onset asynchrony (CTOA), responses are faster to targets presented at cued locations (facilitation), while a long CTOA leads to slower responses to targets appearing at cued

locations (Posner & Cohen, 1984). The later effect has been named inhibition of return (IOR; Posner, Rafal, Choate, & Vaughan, 1985; for a review, see Klein, 2000).

6.2.1 Sensory and motor mechanisms of IOR

In the past quarter century, a large literature has been devoted to the discussion of whether IOR is an attentional/visual effect, or a motoric effect (for a review, see Taylor & Klein, 1998), as well as whether there are multiple additive underlying mechanisms (e.g., Abrams & Dobkin, 1994; Wang et al., 2012a), or mutually exclusive forms of IOR (e.g., Taylor & Klein, 2000).

In a traditional IOR task, the first signal (S1: cue) elicits IOR, while the second signal (S2: target) measures IOR. S1 and S2 stimuli can be exogenous (peripheral visual onsets) or endogenous (e.g., arrows presented at fixation), and can require saccadic or manual responses. A thorough test of 24 S1 and S2 stimulus/response combinations (see Figure 6.1) led Taylor and Klein (2000) to conclude that there are two forms of IOR: visual and motor. “One form of inhibition specifically impaired responses made to peripheral visual stimuli but only when eye movements were not made... The other form of inhibition impaired responding to both central and peripheral S2s but only when eye movements were made (to S1, S2, or both)” (pp. 1651-1652).

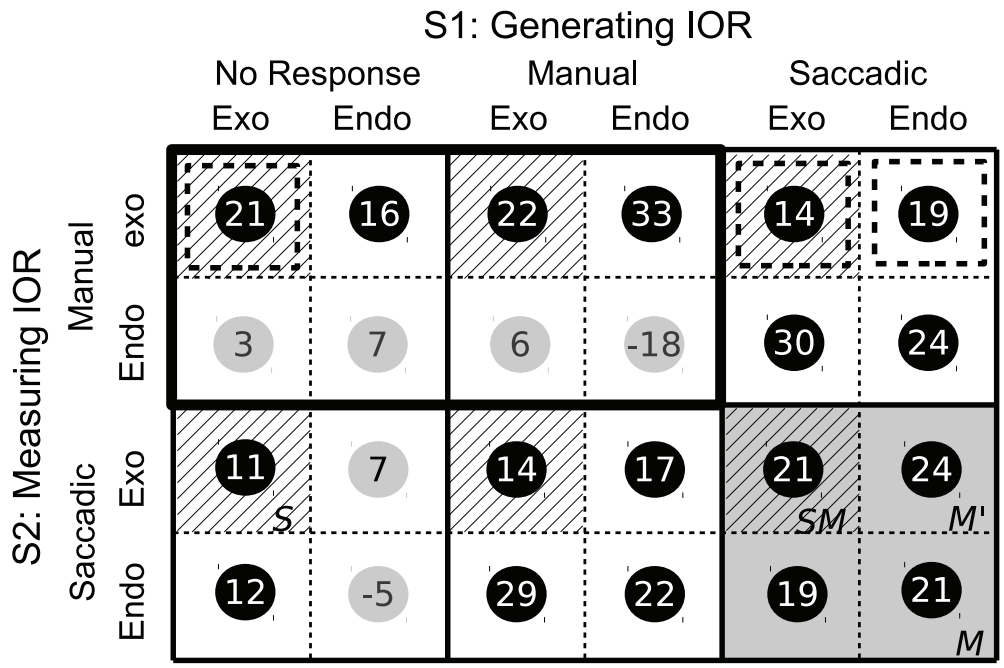

Figure 6.1: Summary of the findings of Taylor and Klein (2000) and a comparison of the sensory and motor mechanisms theory (Wang et al., 2012a) and the visual and motor flavors theory (Taylor & Klein, 2000). Exo: peripheral visual onset; Endo: arrow presented at fixation. Numbers are IOR scores observed in each condition in Taylor and Klein (2000); black disks denote significant, while gray disks denote non-significant IOR scores. According to two-flavors theory, IOR scores within the bold square are attentional/perceptual flavored, while the other IOR scores are motor flavored (see Taylor & Klein, 2000). Hatched cells denote experimental tasks which evoke the sensory mechanism of IOR, while shaded (gray) cells denote those which evoke the motor mechanism of IOR (Wang et al., 2012a). Cells marked with S, M, and SM are tasks tested in Wang et al. (2012a). The M task tested in Wang et al. (2012a) was replaced with the M' task in Experiment 6.1. The tasks tested in Experiment 2 are marked with dashed squares. Experiments 6.3 and 6.4 tested the SM and M' tasks in a paradigm with mixed cue types and exogenous targets.

In a concerted effort to further develop an explicit causal theory of IOR, a computational approach has been adopted in recent work by our group (Satel, Wang, & Klein, 2011; Wang, Satel, & Klein, 2011; Wang et al., 2012a). Satel et al. (2011) proposed that the IOR effect observed in a Posner cueing paradigm, in which fixation is maintained

until target presentation, is caused by short-term depression (STD) of early visual signals initiated by the cue. Due to STD, responses to targets presented at the cued location are prolonged because the target-elicited visual input is weakened (for neurophysiological evidence, see Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005; for human imaging evidence, see Prime & Ward, 2006; Anderson & Rees, 2011). Wang et al. (2011), on the other hand, explored how a first saccade affects the latency of a subsequent saccade, with simulations reproducing previous observations that saccades returning to the immediately preceding fixation location have longer latencies than those which continue in the same direction (e.g., Klein & MacInnes, 1999; Hooge & Frens, 2000). This computational modeling effort led us to propose two separate mechanisms for IOR in the oculomotor system, which we refer to here as sensory and motor mechanisms of IOR. The sensory mechanism represents the observation that a peripheral visual onset attenuates subsequent peripheral onsets at the same spatial location during the input stage of processing. The motor mechanism represents the observation that the execution of a saccade affects the latency of a subsequent saccade. These two mechanisms correspond to IOR effects that are generated by covert exogenous orienting and overt endogenous orienting, respectively.

The 24 experimental task combinations tested by Taylor and Klein (2000), as well as their findings, are summarized in Figure 6.1. According to Taylor and Klein (2000), IOR effects in tasks highlighted by the bold rectangle are visual flavored (suppressed oculomotor system) while IOR effects in all other tasks, where eye movements were made to either the cue, the target or both, are motor flavored (active oculomotor system).

According to Wang et al. (2012a), hatched cells in Figure 6.1 represent experimental tasks that evoke the sensory mechanism, because both the cue and the target are exogenous visual onsets. Shaded (gray) cells represent experimental tasks that evoke the motor mechanism, because the final saccade in response to a target is preceded by a saccade back to the original central fixation location (see also Figure 6.2).

One important property of Wang et al.'s (2012a) two causes theory of IOR is that because the sensory and motor mechanisms affect two different stages of processing (i.e., input and output), tasks in which the two mechanisms overlap will produce larger oculomotor IOR effects. In Wang et al. (2012a), this prediction was verified in an experiment that tested three of the tasks examined in Taylor and Klein (2000). These tasks are labeled as S (sensory), M (motor), and SM (sensori-motor) tasks to reflect the fact that the IOR effects observed in these tasks are contributed to by the sensory, motor, or both the sensory and motor mechanisms of IOR (see Figure 6.2A for an illustration of the tasks). In the S task, peripheral visual stimuli were presented as cues and targets, and participants were required to maintain fixation until target onset. Because there is no saccade before target onset, the IOR effect in the S task recruits only the sensory mechanism (repeated stimulation). In the M task, participants make an eye movement in response to a central endogenous cue (central arrow), return their eyes to central fixation, then move in response to a central endogenous target (central arrow). According to Wang et al. (2012a), the IOR effect in the M task is the result of the aftereffects of saccades back to the central fixation box[7]. In the SM task, as in the S task, both the cues and the targets

[7] Although in the M task the arrows (cue, cue back, and target) were all presented at fixation (repeated stimulation) and should thus also have evoked the sensory mechanism, this repeated stimulation is equated across cued and uncued trials and so does not contribute to the final IOR score.

are peripheral stimuli. However, this task differs from the S task in that participants are required to make an eye movement to the peripheral cue and back to central fixation before the final saccade to the peripheral target (as in the M task). Consequently, the IOR effect in the SM task involves both the sensory and motor mechanisms. Wang et al. (2012a) observed IOR scores for the S, M, and SM tasks of 44 ms, 35 ms, and 80 ms, respectively (see Table 6.1). Importantly, the IOR effect for the SM task was statistically equivalent to the sum of those for the S and M tasks, suggesting that the sensory and motor mechanisms have additive behavioral effects.

6.2.2 Methodological limitations of Wang et al. (2012a)

The results reported in Wang et al. (2012a) support the idea that there are two separate mechanisms (sensory and motor) underlying IOR - when revealed with saccadic responses - and that they have additive behavioral effects. Apart from computational explicitness, the additivity claim is the most prominent difference between Wang et al.'s (2012a) sensory/motor theory of IOR and Taylor and Klein's (2000) two flavors theory. However, some design issues in the methodology used by Wang et al. (2012a) may have confounded the theoretical interpretation of these results. These potential confounds include repeated spatiotopic stimulations in the SM task, different target types (endogenous) in the M task, a temporal gap inadvertently introduced by turning off the drift correction target (see Figure 6.2), and differences in attentional control settings (ACSs) across tasks due to the use of a blocked design.

First, in the SM task, an arrow was presented at the peripherally cued location to inform the participant to move their eyes back to the original, central, fixation location

(the cue back signal) while the participant was fixating the peripherally cued location (see Figure 6.2A). Since this cue back arrow occupied the same spatiotopic location as the peripheral cue, cued boxes were spatiotopically stimulated twice, first by the peripheral cue, then by the cue back signal. Since it is clear from previous studies that IOR can be coded in spatiotopic coordinates (Hilchey, Klein, Satel, & Wang, 2012b; Maylor & Hockey, 1984; Mathot & Theeuwes, 2011; Pertzov, Zohary, & Avidan, 2011; Posner & Cohen, 1984; Satel, Wang, Hilchey, & Klein, 2012), it is possible that multiple spatiotopic stimulations in the SM task led to a stronger IOR effect in this condition (e.g., Dukewich & Boehnke, 2008). To eliminate this potential confound, the current experiments used an auditory cue back signal (Experiments 6.1 and 6.2), or no cue back signal (Experiments 6.3 and 6.4), rather than a visual cue back signal in the cued peripheral box (as in Wang et al., 2012a).

Second, Wang et al. (2012a) used a central endogenous target in the M task, but peripheral exogenous targets in the S and SM tasks. This design led to overall slower responses in the M task than in the other tasks (see Table 6.1). It is unclear whether this delay in saccadic response time (SRT) has affected the pattern of results. Thus, in the current experiments, exogenous peripheral targets were used in all tasks (see Figure 6.2B). We will refer to the new motor task using peripheral targets and auditory cue back signals as the M' task.

Third, Wang et. al (2012a) blocked the three conditions, using endogenous cues (central arrows) in the M task, and exogenous cues (peripheral flashes) in the S and SM tasks. Because processing of central endogenous cues requires focused central attention,

while the detection of peripheral exogenous cues requires attention to be spread more diffusely, top-down ACSs may have come into play to affect the pattern of results. To address this issue, Experiments 6.3 and 6.4 mixed the M' and SM tasks together to test whether the sensory and motor mechanisms, which are fairly low-level mechanisms, are resilient to the influence of top-down ACSs.

Finally, a temporal gap between fixation offset and cue onset was inadvertently included in the original experimental design. This temporal gap was the result of a self-paced drift correction process. At the beginning of every trial, subjects were required to fixate a central cross and press the space bar in order to initiate trials. Successful drift correction was signaled by the disappearance of the drift correction target (a cross in the central box, see Figure 6.2). Since temporal gaps are known to interact with IOR (e.g., Abrams & Dobkin, 1994; Hunt & Kingstone, 2003), in Experiment 6.4, the drift-correction target was left on the screen for the entire trial duration.

6.2.3 IOR with manual responses

Furthermore, Wang et al. (2012a) suggested that their proposed motor mechanism of IOR is restricted to the oculomotor system - though other findings suggest that oculomotor activity may carry over to the skeletomotor system (e.g., Nagy, Kruse, Rottmann, Dannenberg, & Hoffman, 2006; Werner, Dannenberg, & Hoffman, 1997). Taylor and Klein (2000) proposed that tasks requiring manual responses to targets would recruit either the attention/perception form of IOR if the oculomotor system was suppressed, or the motor form of IOR if eye movements were made to cues. To explore these possibilities, we also replicated the design of Experiment 6.1 with manual rather

than saccadic responses to targets (Experiment 6.2).

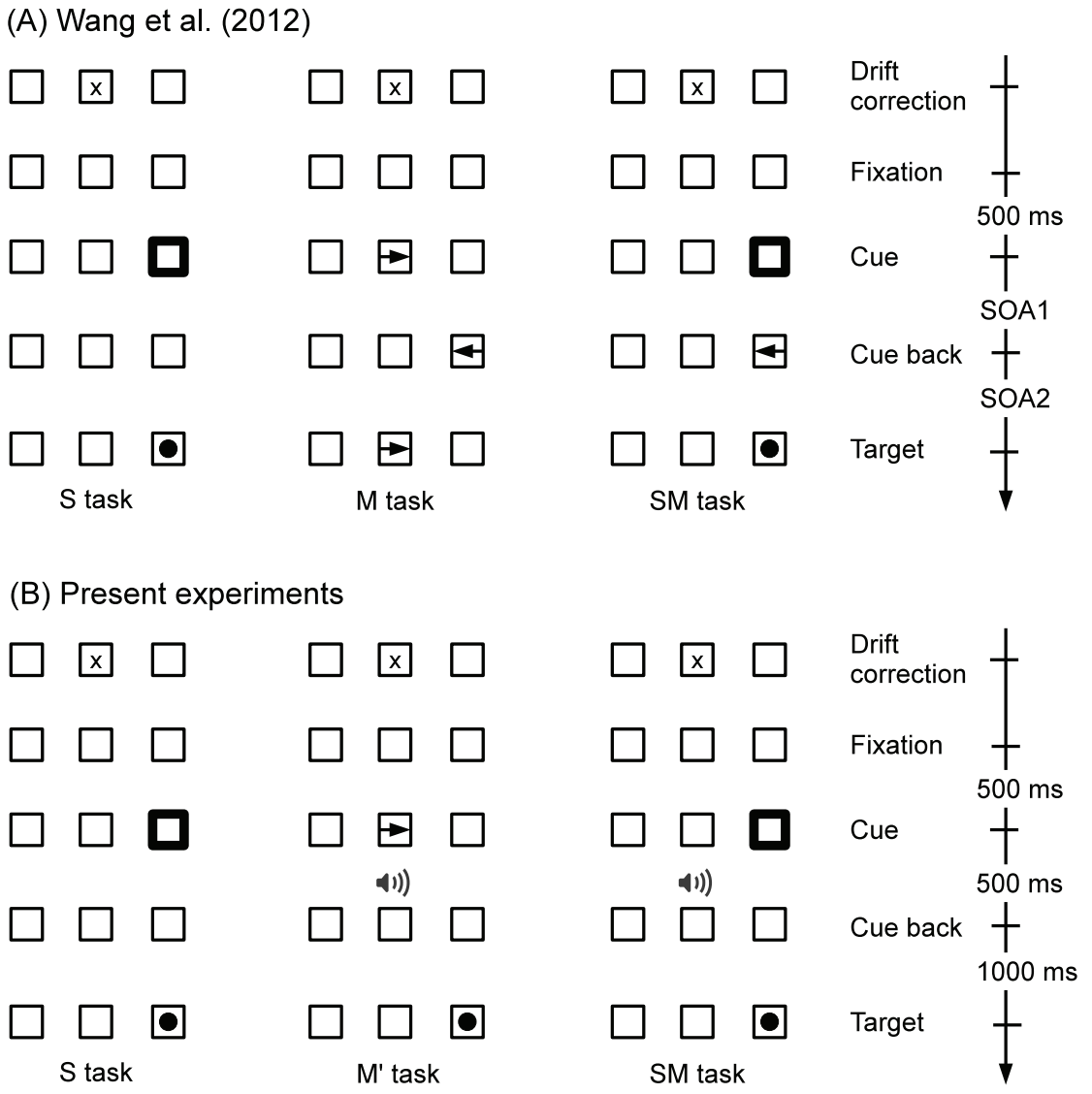

Figure 6.2 : (A) The S, M, and SM tasks tested in Wang et al. (2012a). (B) The S, M', and SM tasks tested in Experiments 6.1 *and* 6.2. *Experiment* 6.1 *required saccadic responses to targets, while Experiment* 6.2 *required manual responses to targets. In Experiments* 6.3 *and* 6.4, *only the M' and SM tasks were run, in a mixed-cue design with the auditory cue back removed. Furthermore, in Experiment* 6.4, *the drift correction target (a gray cross) was presented in the central box throughout trials.*

6.2.4 Summary of the present experiments

The primary purpose of the present experiments was to replicate Wang et al. (2012a) while eliminating potential confounds of the original experimental design. All experiments presented here used exogenous peripheral targets in all conditions. Experiments 6.1 and 6.2 also incorporated an auditory, rather than visual, cue back signal. Experiment 6.2 required manual responses to targets, while all other experiments required oculomotor responses. Experiments 6.3 and 6.4 mixed peripheral and central cue types and did not use any cue back signal. Experiment 6.4 further eliminated the temporal gap introduced by drift corrections in previous designs. The results of these experiments continue to support the existence of two independent mechanisms underlying the behavioral exhibition of IOR with oculomotor responses to targets, but not with manual responses.

6.3 Experiment 6.1: Exogenous targets and auditory cue back signal

Using exogenous targets and auditory cue back signals, Experiment 6.1 replicated Wang et al. (2012a) while eliminating the repeated spatiotopic stimulation and target type confounds.

6.3.1 Methods

Participants: All participants of the present study were university students who reported normal or corrected-to-normal vision. Twelve university students (7 female, 5 male) participated in this experiment for extra course points.

Stimuli and apparatus: Visual stimuli were presented on a 17 inch, SVGA, Viewsonic computer monitor with screen resolution set to 1024 × 768 pixels. The visible

area of the monitor measured 36.2° (width) × 27° (height) at a viewing distance of 57 cm. The experiment was programmed with Python. An EyeLink® video-based eye-tracking system was used to monitor participants' direction of gaze every 4 ms with a resolution of 0.1° or better.

Three gray boxes were placed horizontally in the center of the display, which subtended 1.8° of visual angle, with the thickness of their borders measuring 1 pixel. The center of adjacent boxes were separated by 8.2°. Peripheral cues were implemented as a brightening (and thickening) of one of the peripheral placeholder boxes. Peripheral targets were bright, filled circles subtending 1°. The central cues were arrows pointing to the left or right, measuring 0.8° (width) × 0.2° (height). Unlike Wang et al. (2012a), which used an arrow at the peripherally cued location as a cue back signal, the cue back signal in the present experiment was a tone presented 500 ms after cue onset.

Design and procedure: The three tasks used were similar to those used in Wang et al. (2012a), see Figure 6.2B. In the S task, participants were required to fixate the central box until a peripheral target (bright filled circle) was detected. Peripheral cues were uninformative and ignored. In both the M' and SM tasks, saccadic responses were required to both the cue, the cue back signal, and the target. Cues used for the M' task were foveally presented arrows, while the S and SM tasks used peripheral onset cues.

On half of the trials, the target appeared at the cued peripheral box, and on the other half of trials the target appeared at the uncued peripheral box. For convenience, we will refer to these two types of trials as cued and uncued trials, regardless of the differences between tasks. The SRT difference between cued and uncued trials in each

task was regarded as a measure of IOR. This experiment used a within-subjects design of two factors: task (S, M', or SM) and cueing (cued or uncued). The three tasks were presented in separate blocks of 72 trials (36 cued and 36 uncued) each, and the order of task presentation was balanced across participants. Each task was preceded by a 24-trial practice block.

Drift correction was performed at the beginning of each trial, followed by a fixation display (three empty gray boxes) that was presented for 500 ms. Participants were required to fixate the central box during this fixation period, then the cue was presented for 300 ms. In the S task, participants ignored the peripheral cue and maintained fixation; in the M' task, participants made an eye movement to the peripheral box pointed to by an arrow presented in the central box; in the SM task, participants made eye movements to the peripherally cued box. In the M' and SM tasks, the auditory cue back signal was presented 500 ms after the appearance of the cue and lasted for 300 ms. In response to this cue back signal, the participant made eye movements back to the central box. 700 ms later, the target appeared in either the cued or uncued box.

During the experiment, incorrect eye movements resulted in trials being aborted. Trials were aborted if a saccade (> 2°) was detected during the fixation period, or if a saccade landed more than 2° away from the cued peripheral box or the central box (following the cue back signal) in the M' and SM tasks. Saccadic responses within 1000 ms were required to the target, with failures to respond to the target or saccades landing more than 2° away from the center of the target box resulting in trials being aborted. All aborted trials were recycled, such that every participant performed the same number of

trials correctly.

6.3.2 Results and discussion

For correctly completed trials, those with SRTs faster than 80 ms or slower than 400 ms were excluded from analysis (5.17%). The mean SRTs for each condition are presented in Table 6.1. An ANOVA of the SRTs, with variables task (S, M', or SM) and cueing (cued vs. uncued), revealed significant main effects for task [$F(2, 11) = 7.86$, $p < 0.01$, $\eta_G^2 = 0.18$] and cueing [$F(1, 11) = 46.52$, $p < 0.001$, $\eta_G^2 = 0.13$]. Note that here we report generalized eta squared (Olejnik & Algina, 2003) as a measure of effect size. The main effect of cueing suggests that IOR was observed in Experiment 6.1. Importantly, as revealed by the significant interaction between cueing and task [$F(2, 22) = 7.82$, $p < 0.01$, $\eta_G^2 = 0.02$], the magnitude of IOR differed across the S, M', and SM tasks (see Table 6.1 and Figure 6.3). Planned comparisons revealed stronger IOR for the SM task than for the S [$t(11) = 4.26$, $p < 0.001$] and M' [$t(11) = 3.50$, $p < 0.01$] tasks. The IOR scores did not differ between the S and M' tasks [$t(11) = 0.43$, n.s.], and IOR for the SM task was not significantly different from the sum of those for the S and M' tasks [$t(11) = 0.37$, n.s.].

After eliminating two potential methodological confounds of Wang et al. (2012a), Experiment 6.1 successfully reproduced the pattern of results reported in Wang et al. (2012a). The IOR effects for the S, M', and SM tasks in the present experiment were 20 ms, 23 ms, and 40 ms, respectively. As in Wang et al. (2012a), the pattern of results observed in the present experiment was very strong, with 10 out of the 12 participants producing numerically larger IOR scores for the SM than for the S and M' tasks. These results support the claim that there are at least two mechanisms (sensory and motor)

contributing to the behavioral effects of IOR when eye movements are required to targets, and that these two mechanisms have additive effects.

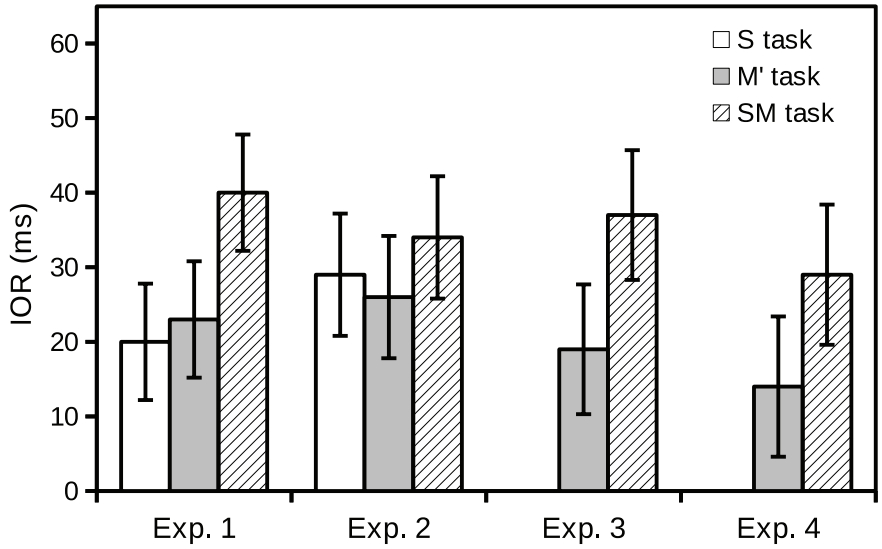

Figure 6.3: Comparison of IOR scores observed in the different tasks of the current experiments. Error bars denote 95% within-subject confidence intervals, based on the error term of task (Masson & Loftus, 2003).

6.4 Experiment 6.2: Manual responses to targets

According to Wang et al. (2012a), the motor mechanism is mediated by connections within the oculomotor-generating machinery of the superior colliculus (SC) and is likely restricted to the oculomotor system. However, a close examination of the findings of Taylor and Klein (2000) reveals that the pattern of results in their saccade-manual conditions (the four upper-right cells in Figure 6.1) is similar to that observed in the saccade-saccade conditions (the four lower-right cells in Figure 6.1). That is, when manual responses to targets are preceded by saccades to the cue and then back to the

central fixation, IOR was also observed regardless of the nature (exogenous or endogenous) of the cue and target stimuli. These observations suggest that the motor mechanism of IOR proposed in Wang et al. (2012a) may carry over to the skeletomotor system. This possibility was explored in Experiment 6.2, in which manual rather than saccadic target responses were required (cells with dashed squares in Figure 6.1). For convenience, we will still refer to these tasks as the S, M', and SM tasks.

Robust IOR effects were expected in the S and SM tasks because they invoke the sensory mechanism of IOR (hatched cells). Although not predicted by Wang et al.'s (2012a) sensory/motor theory, we also expected to observe IOR in the M' task because this task has been investigated previously (e.g., Taylor & Klein, 2000). More importantly, if this IOR effect is simply caused by the motor mechanism carrying over to the skeletomotor system, it should be additive with the sensory mechanism and stronger IOR should be observed in the SM than in the S and M' tasks, as observed in Experiment 6.1 and Wang et al. (2012a).

6.4.1 Methods

Participants: Twenty-four university students (16 female, 8 male) participated in this experiment for extra course credit.

Stimuli, apparatus, design, and procedure: The stimuli, devices, and task procedure used in this experiment were the same as used in Experiment 6.1, except that the targets required manual localization responses. Participants pressed the “z” or “/” key with the index fingers of the left and right hands, respectively, for targets appearing in the left or right peripheral boxes.

6.4.2 Results and discussion

Trials with RTs faster than 200 ms or slower than 600 ms were excluded from analysis (3.69%). The mean RTs for each condition are presented in Table 6.1. An ANOVA of the RTs, with the variables task (S, M', or SM) and cueing (cued vs. uncued), revealed a marginally significant main effect for task [$F(2, 46) = 2.86$, $p = 0.07$, $\eta_G^2 = 0.01$] and a significant main effect of cueing [$F(1, 23) = 52.92$, $p < 0.001$, $\eta_G^2 = 0.08$]. The main effect of cueing suggests that IOR (i.e., slower responses to cued than to uncued targets) was observed in Experiment 6.2. However, as revealed by the non-significant interaction between cueing and task [$F(2, 46) = 1.18$, $p = 0.32$, $\eta_G^2 = 0.00$], the magnitude of IOR did not differ across the S, M', and SM tasks in Experiment 6.2 (see Figure 6.3). The IOR scores were 29 ms, 26 ms, and 35 ms for the S, M', and SM tasks, respectively.

Since the motor mechanism of IOR is thought to only affect saccadic responses to targets, the sensory/motor theory (Wang et al., 2012a) predicts no IOR in the M' task with manual responses. However, replicating Taylor and Klein (2000), robust IOR was observed in the M' task. Note that Experiment 6.1 and Wang et al. (2012a) demonstrated that the motor mechanism is additive with the sensory mechanism when oculomotor responses are required to targets. If the IOR effect observed in the M' task was caused by the same motor mechanism as that recruited with saccadic responses, additivity should also be observed in Experiment 6.2. Our failure to observe additivity in Experiment 6.2 is in agreement with Wang et al.'s (2012a) claim that the motor mechanism previously identified in computational simulations (Wang et al., 2011) is restricted to the oculomotor

system[8].

	S task			M/M' task			SM task		
	Cued	Uncued	IOR	Cued	Uncued	IOR	Cued	Uncued	IOR
Wang et al. (2012a)	284	240	44	315	280	35	278	198	80
Experiment 6.1	233	213	20	198	175	23	214	174	40
Experiment 6.2	350	321	29	361	335	26	365	330	34
Experiment 6.3	-	-	-	230	211	19	244	209	35
Experiment 6.4	-	-	-	190	176	14	202	173	29

Table 6.1: Mean target RTs (ms) and IOR scores for each condition in Wang et al. (2012a) and the present experiments.

6.5 Experiments 6.3 and 6.4: Blocked versus mixed designs

As discussed previously, when the M' and SM tasks are blocked, at the time of cue presentation, the central cue (arrow presented at fixation) in the M' task may have elicited focused central attention, while cues being in the periphery in the SM task may have encouraged a more diffuse deployment of attention. It is possible that this difference in ACSs has led to larger IOR scores in the SM than in the M' task. To investigate this possibility, Experiments 6.3 and 6.4 mixed central and peripheral cues within the same block so as to encourage participants to adopt the same ACS for both the M' and SM tasks.

Also note that in Experiments 6.1, 6.2, and 6.3, and in Wang et al. (2012a), the

[8] An ANOVA was performed on the IOR scores, with the variables experiment (Experiment 6.1 vs. Experiment 6.2) and task (S, M', or SM). The main effect of experiment did not reach significance [$F(1, 34) = 0.1$, $p = 0.75$, $\mu_G^2 = 0.00$], suggesting that the overall magnitude of IOR effects did not differ across the two experiments. The main effect of task did reach significance [$F(2, 68) = 4.46$, $p < 0.05$, $\mu_G^2 = 0.05$] but the interaction between task and experiment did not reach significance [$F(2, 68) = 1.25$, $p = 0.29$, $\mu_G^2 = 0.02$], suggesting that the overall pattern of results did not differ across the two experiments. However, separate ANOVAs performed for each experiment revealed a large effect of task for Experiment 6.1 [$F(2, 22) = 7.82$, $p < 0.01$, $\mu_G^2 = 0.22$] and a small effect for Experiment 6.2 [$F(2, 46) = 1.18$, $p = 0.32$, $\mu_G^2 = 0.02$], suggesting that, although we cannot assert so statistically, the difference in the pattern of results between Experiment 6.1 and Experiment 6.2 is likely a qualitative one.

drift correction target was removed 500 ms before cue onset. It has been documented that such a temporal gap between fixation offset and stimulus onset may affect SRTs (Saslow, 1967) and IOR scores (e.g., Abrams & Dobkin, 1994; Hunt & Kingstone 2003). To rule out this potential confound, in Experiment 6.4, a dark gray cross in the central box was used as the drift correction target and was presented throughout a trial. At the same time, the cue back signal was completely eliminated from these two experiments and participants were simply required to quickly move their eyes back to the central box once their eyes reached the cued peripheral box.

6.5.1 Methods

Participants: Experiment 6.3 had 8 participants (7 female, 1 male) and Experiment 6.4 had 9 participants (7 female, 2 male).

Stimuli, device and procedure: The stimuli and device used in Experiments 6.3 and 6.4 were the same as Experiment 6.1 and 6.2, with the following modifications[9]. The S task was not tested in Experiments 6.3 and 6.4 and the M' and SM tasks were mixed rather than blocked. In Experiment 6.4, the drift correction target was drawn in dark gray

[9] Due to a programing lapse, in Experiments 6.3 and 6.4, the cue duration (and the overall CTOA) were extended on trials in which eye movements in response to cues occurred within 300 ms (29.2% in Experiment 6.3 and 47.5% in Experiment 6.4). As a result, in Experiment 6.3, the mean cue duration was 477 ms (mean CTOA = 1625 ms) and 316 ms (mean CTOA = 1513 ms) for exogenous and endogenous cue trials, respectively. In Experiment 6.4, the mean cue duration was 446 ms (mean CTOA = 1648 ms) and and 339 ms (mean CTOA = 1542 ms) for exogenous and endogenous cue trials, respectively. We still report these two experiments because: a) as will be shown in the Results sections, the SM task produced larger IOR effects despite the fact that the CTOA was longer in this task. The two causes theory of IOR predicts that the contribution of both the sensory and motor mechanisms to behavioral IOR decays with time. Consequently, the IOR effects in the SM tasks were slightly underestimated in Experiment 6.3 and 6.4. Nonetheless, IOR effects in the SM tasks were still larger than those for the M' tasks in both experiments. b) The pattern of results was essentially the same (i.e., stronger IOR for the SM task) in Experiment 6.3 when only trials not affected by the programming lapse were analyzed. IOR scores for the M' and SM task were 19 ms and 44 ms in Experiment 6.3 [$t(7) = 3.45$, $p < 0.05$]. In Experiment 6.4, only four participants produced enough normal trials (at least 6 trials per experimental cell) for such an analysis. The IOR effect for the SM task (60 ms) was still larger than that for the M' task (31 ms) [$t(4) = 2.95$, $p = 0.06$] under these conditions.

and was presented in the central box throughout a trial. Both tasks were tested for 80 trials (40 cued and 40 uncued), in both experiments. Participants were exposed to a 24-trial practice at the beginning of each experimental session.

6.5.2 Results: Experiment 6.3

Based on inspection of the SRT distribution, trials with SRTs slower than 400 ms or faster than 100 ms were excluded from analysis (1.6%). The mean SRTs for each condition of the M' and SM tasks are presented in Table 6.1. An ANOVA of the SRTs, with the variables task (M' or SM) and cueing (cued or uncued) revealed a significant main effect of cueing [$F(1, 7) = 57.75$, $p < 0.001$, $\eta_G^2 = 0.54$] that interacted with task [$F(1, 7) = 11.89$, $p < 0.05$, $\eta_G^2 = 0.10$], suggesting that IOR was observed and that the IOR effect in the SM task (35 ms) was larger than that in the M' task (19 ms). These results suggest that the sensory and motor mechanisms identified by Wang et al. (2012a) are resilient to top-down ACSs.

6.5.3 Results: Experiment 6.4

All trials with SRTs slower than 400 ms or faster than 80 ms were excluded from analysis (3.1%). The mean SRTs for each condition are presented in Table 6.1. An ANOVA of the SRTs revealed a significant main effect of cueing [$F(1, 8) = 13.24$, $p < 0.01$, $\eta_G^2 = 0.10$] that interacted with task [$F(1, 8) = 7.63$, $p < 0.05$, $\eta_G^2 = 0.01$]. These results confirmed the findings of Experiment 6.3, with larger IOR observed in the SM task (29 ms) than in the M' task (14 ms).

6.5.4 Discussion

Experiment 6.3 explored whether ACSs elicited by blocking the M' and SM tasks,

as in the previous experiments and Wang et al. (2012a), affects the pattern of observed results (i.e., stronger IOR for the SM task). Experiment 6.4 further probed whether this pattern of results was caused by the untoward introduction of a temporal gap. The pattern of results was very consistent, with stronger IOR observed for the SM than for the M' task in both experiments. Further analysis with experiment (Experiment 6.3 vs. Experiment 6.4) as a factor revealed that, although the IOR scores were numerically larger in Experiment 6.3, this difference did not reach significance [$F(1, 15) = 0.76$, n.s., $\eta_G^2 = 0.00$]. It is worth noting that the pattern of results in both Experiments 6.3 and 6.4 was highly consistent across participants, with all participants in Experiment 6.3, and 7 out of 9 participants in Experiment 6.4 producing larger IOR scores in the SM task. Taken together, this pattern of results suggests that the observation of additivity of mechanisms seen in Experiment 6.1 and Wang et al. (2012a) were not caused by ACSs or the temporal gap following the drift correction display.

6.6 General discussion

As demonstrated by Wang et al. (2012a), there seems to be two additive, dissociable mechanisms underlying IOR when measured with oculomotor responses. The current experiments have further demonstrated that the observation of these independent mechanisms does not depend on the precise experimental design used in Wang et al. (2012a). We have identified potential confounds in the original design and have run experiments that attempt to overcome these potential limitations, demonstrating that the robustness of independent sensory and motor mechanisms underlying IOR does not depend on repeated spatiotopic stimulation, target type, blocking of tasks, or temporal

gap. We have also demonstrated that when the same design is performed with manual responses, additive, dissociable mechanisms of IOR are *not* observed.

6.6.1 Differences between the present experiments and Taylor and Klein (2000)

In the spirit of Taylor and Klein (2000), our experimental design (see also Wang et al., 2012a) dissociates sensory and motor mechanisms of IOR by manipulating the initial *cause* of IOR (central or peripheral cues) and the degree of *oculomotor activation* (whether or not an eye movement is required to cues). Our experiments with saccadic responses produced a stronger IOR effect for the SM than for the M/M' tasks, while Taylor and Klein (2000) reported equivalent IOR effects for their versions of these tasks. Why do our results differ so dramatically from those in Taylor and Klein (2000)?

As has been discussed in Wang et al. (2012a), Taylor and Klein (2000) presented an exogenous cue back stimulus at the original fixation point while participants fixated the cued location. Such cue back signals stimulate cells representing the retinotopic location that is subsequently occupied by uncued targets, thereby potentially causing an underestimation of the contribution of the sensory mechanism of IOR in conditions where cues are foveated. Further work parametrically investigating the effects of cue back signals in various paradigms are encouraged to further delineate the conditions under which these signals interact with mechanisms of IOR.

Furthermore, in Taylor and Klein (2000), cue and target types (endogenous and exogenous) were mixed in order to ensure comparable ACSs across tasks. Since task relevant events could occur at any location when endogenous (central) and exogenous (peripheral) cues and targets were mixed, a diffuse ACS was likely established that

encompassed both central and peripheral locations. In contrast, a blocked condition design was used in Wang et al. (2012a) and Experiments 6.1 and 6.2 here. Since there were differences in the possible locations of task relevant events in each block, the intensity of attention to the periphery was likely different across tasks. Previous work has shown that ACSs established under different task demands can reduce or eliminate observed IOR (e.g., Hilchey, Klein, & Ivanoff, 2012a; Wang & Klein, 2012). It is currently unknown, however, whether ACSs (and other top-down factors) directly affect the underlying IOR mechanisms (e.g., STD) or indirectly affect the behavioral manifestation of these mechanisms. In Experiments 6.3 and 6.4, we mixed cue types together and continued to observe stronger IOR for the SM task. However, note that only exogenous targets were used in the present experiment. So whether Taylor and Klein's (2000) mixed design was responsible for their failure to observe stronger IOR in the SM task remains unclear. In any event, the present experiments suggest that Wang et al.'s (2012a) observation of stronger IOR for the SM task is fairly robust.

6.6.2 IOR in the M' task with manual responses

As mentioned previously, Taylor and Klein (2000) showed that when saccades were made to the cue and then back to the central fixation, IOR could be revealed by manual responses regardless of the nature of the target (exogenous or endogenous). This pattern of results seems to suggest that IOR effects in Taylor and Klein's (2000) saccade-manual conditions (the four upper-right cells in Figure 6.1) are caused, or at least contributed to, by a “motor” mechanism. The robust IOR effect observed in the M' task of Experiment 6.2 is in agreement with this supposition. However, the exact neural substrate

of this “motor” mechanism is still unclear. It is possible that there exists a general habituation-like mechanism in motor systems where responses toward spatial locations are always slowed by previous responses to the same location (Dukewich, 2009), even across different response modalities. Note that, in Taylor and Klein (2000), IOR effects were also observed in all manual-saccade conditions (the four lower-middle cells in Figure 6.1), regardless of the nature of the cues and targets. Further behavioral and neuroimaging studies are required to reveal the mechanism(s) and neural substrate(s) underlying IOR effects observed in the M' task with manual responses.

6.7 Conclusion

The experiments presented here have demonstrated that the observation of sensory and motor mechanisms of IOR, when revealed with eye movements to targets, does not depend on target type, repeated spatiotopic stimulation, blocking of cue types, or a temporal gap. Furthermore, a potentially different ‘motor mechanism’ that is not additive with the sensory mechanism was revealed when IOR was measured with manual rather than saccadic responses. Additional behavioral and neuroscientific experiments are encouraged to further examine the boundary conditions under which sensory (input-driven) and motor (output-driven) mechanisms contribute to IOR.

6.8 Acknowledgements

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6.9 References

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CHAPTER 7 THE EFFECTS OF IGNORED VERSUS FOVEATED CUES UPON INHIBITION OF RETURN: AN EVENT-RELATED POTENTIAL STUDY

Satel, J., Hilchey, M. D., Wang, Z., Story, R., and Klein, R. M. (2013). The effects of ignored versus foveated cues upon spatial orienting: An event-related potential study. Attention, Perception, & Psychophysics, 75(1): 29-40 (reformatted and reprinted with permission of the publisher, Springer).

7.1 Abstract

Taylor and Klein (2000) discovered two mutually exclusive "flavors" of inhibition of return (IOR): when the oculomotor system is "actively suppressed", IOR affects input processes (perception/attention flavor); whereas, when the oculomotor system is "engaged", IOR affects output processes (motor flavor). Studies of brain activity with ignored cues have typically reported that IOR reduces an early sensory event-related potential (ERP) component (*i.e.*, the P1 ERP component) of the brain's response to the target. Since eye movements are discouraged in these experiments, the P1 reduction might be a reflection of the perception/attention flavor of IOR. If, instead of ignoring the cue, participants make a prosaccade to the cue (and then return to fixation) before responding to the target, then the motor flavor of IOR should be generated. We compared these two conditions while monitoring eye position and recording ERPs to the targets. If the P1 modulation is related to the perceptual/attentional flavor of IOR, we hypothesized that it might be absent when the motoric flavor of IOR is generated by a prosaccade to the cue. Our results demonstrated that target-related P1 reductions and behavioral IOR were similar, and significant, in both conditions. However, P1 modulations were significantly correlated with behavioral IOR only when the oculomotor system was actively suppressed, suggesting that P1 modulations may only affect behaviorally exhibited IOR when the attentional/perceptual flavor of IOR is recruited.

7.2 Introduction

To efficiently navigate the visual environment, humans have ostensibly adapted a mechanism that biases against previously processed space (Posner, Rafal, Choate, &

Vaughan, 1985; Klein & McInnes, 1999). A great deal of laboratory research has demonstrated that keypress and saccadic eye movement response times (RTs) are slower to targets presented in previously processed relative to unprocessed space when the interval between the target and a prior inputs exceeds approximately 300 ms (for a review, see Klein, 2000). These effects are often interpreted to reflect a bias against previously processed inputs. Although there may be multiple neural mechanisms underlying this effect (e.g., Kingstone & Pratt, 1999; Taylor & Klein, 2000; Hunt & Kingstone, 2003; Sumner et al., 2004; Zhang & Zhang, 2011), inhibition of return (IOR; Posner, Rafal, Choate, & Vaughan, 1985) is commonly used when referring to them.

7.2.1 Attentional/perceptual vs. motoric flavored IOR

The cornerstone finding that is most commonly attributed to IOR (Posner et al., 1985) was first discovered by Posner and Cohen (1984) in a spatial cueing paradigm in which the participant was instructed to ignore a spatially uninformative visual onset stimulus (the cue) and to respond to another onset (the target) that appeared sometime later. The imperative response stimulus (the target) appeared randomly at either the cued or uncued location. In this seminal study, observers depressed a single key to acknowledge the appearance of the target. Critically, Posner and Cohen (1984) also manipulated the time between the cue and target onset, often referred to as the cue-target onset asynchrony (CTOA). Replicating previous findings (*cf*. Posner, 1980), RTs were faster to targets at the cued relative to the uncued location if the CTOA was shorter than 200 ms. The surprising, and critical, finding was that when the CTOA exceeded 300 ms, RTs were slower to targets appearing at the cued, as compared to the uncued, location.

Using variations on this model task, Taylor and Klein (2000) parametrically manipulated the physical nature of the first and second signal (S1 and S2, respectively) in a dedicated effort to uncover the cause(s) and effect(s) of IOR (see Figure 7.1). S1 and S2 comprised centrally presented arrows (pointing left or right) and peripherally presented luminance increases (occurring left or right of fixation). These signals were randomly intermixed *in* each block (see below). The response modes for S1 and S2 were manipulated *between* blocks. To generate the effect, S1 required either: 1) no response, 2) a saccadic response, or 3) a manual response. To measure the effect, S2 required either: 1) a saccadic response, or 2) a manual response. These manipulations yielded six unique response combinations of S1 and S2 (no response - manual, manual - manual, saccade - manual, no response - saccade, manual - saccade, and saccade - saccade) that were run, as noted above, in separate blocks. The CTOA was held constant at one second and feedback was provided if a saccadic eye movement was made to a non-eye movement signal.

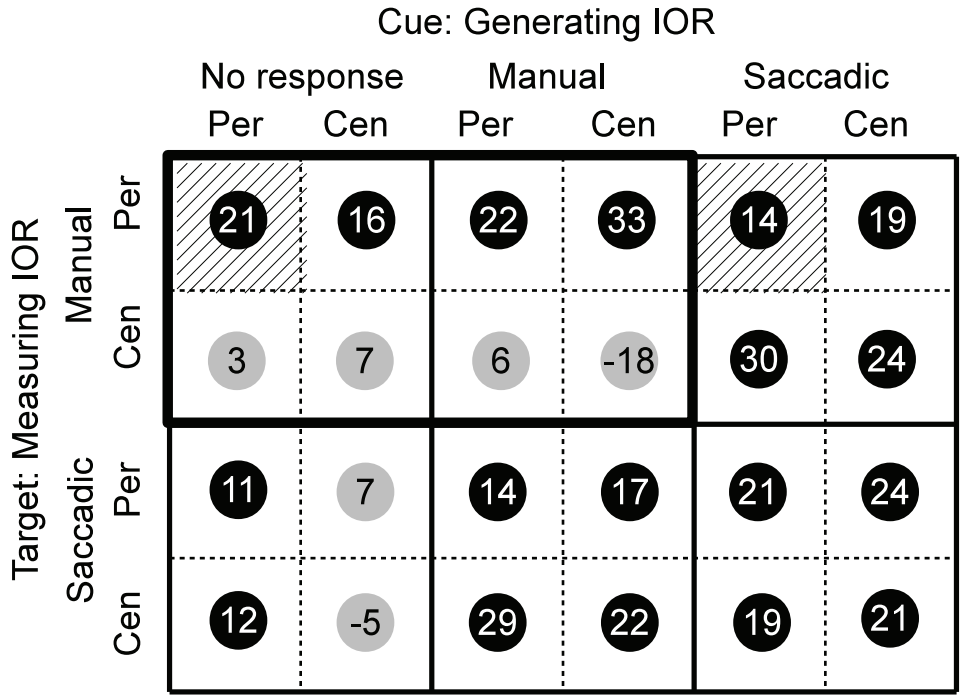

Figure 7.1: Each observer was tested in each of the six conditions on separate days. The sequence of events was identical in each of these conditions: a trial begins with the appearance of three rectangular landmarks, one at fixation and one left and right of fixation. The first event was a peripheral box brightening (exogenous) or central directional arrow signal (endogenous) whose location and direction, respectively, did not correlate with a future second signal (S2), occurring 1 second after the first (S1). S2, used to measure the effect of S1, was a randomly-presented central arrow (endogenous) or peripheral onset disc (exogenous). A cue-back to fixation (a center-box brightening) invariably followed, 500 ms after the cue onset. The observer participated in each factorial combination of response type to S1 (no response, manual, or saccade) and S2 (manual or saccade) which, as noted, yielded six conditions. Note principally that it was not possible to measure the effect of S1 with a central arrow S2 when saccadic eye movements were not required (the two conditions highlighted by the emboldened rectangle), whereas it was in all conditions where saccadic eye movements were required. The two hatched cells are those being compared in the current experiment (no response peripheral S1 - manual response peripheral S2 versus saccadic response peripheral S1 - manual response peripheral S2).

Considering all stimulus-response combinations described above, Taylor and Klein (2000) determined that there are two mutually exclusive effects of IOR. One effect of IOR, seen in conditions where a saccadic response was required to either S1, S2, or

both, exhibited a primarily – if not entirely – motoric behavioral expression. Whether the cueing effect was measured by a peripheral onset or central arrow S2, and whether the response to the target was executed by a saccade or keypress, the magnitude of the IOR considering all possible pairs of conditions was no larger with peripheral than central S2s [in fact, numerically, it was larger with central (22 ms) than peripheral S2s (17 ms)]. It therefore appeared as if there was no effect of spatially overlapping signals on IOR and it thus seemed as if IOR was affecting only the response (priority, or output/decision processes) and not the quality of the signal (salience, or input processes). Another effect of IOR, measured in conditions where a saccadic eye movement response was strictly forbidden, exhibited a primarily – if not entirely – attentional/perceptual behavioral expression. In these cases, IOR was only observed with peripheral onset S2s. If there were a motoric component, one would – at bare minimum – expect IOR when measured by responses to a central arrow S2.

According to Klein and Hilchey's (2011) theoretical framework for IOR effects – which builds off of Taylor and Klein (2000) – when oculomotor responses are not permitted to task-relevant space, the reflexive oculomotor system is actively suppressed. In consequence, ancillary attentional/perceptual pathways are recruited to meet the task-demands. By contrast, when oculomotor activation is necessary to meet task-demands, the effect of IOR is primarily motoric.

The validity of this oculomotor theory of IOR was recently evaluated by Chica, Taylor, Lupianez, and Klein (2010). Chica et al. (2010) administered a relatively standard spatial cueing paradigm wherein a peripheral onset cue preceded a to-be-discriminated

target. In the key conditions, the imperative response signal was a manual discrimination response to a red- or green-colored circle and Chica et al. (2010) manipulated, in separate blocks, whether or not a saccadic response was required to the peripheral onset cue. If, following Taylor and Klein (2000), the effect of IOR were on the quality of perceptual processing or the rate of information accrual when eye movements are *not* required, one would expect RTs to be slower at the cued as compared to the uncued location, while accuracy would be either reduced or equivalent at the cued as compared to the uncued location. If the effect of IOR was to bias against responding to previously cued locations and *not* on the accrual of perceptual information when saccadic eye movements were required, then responses would be slower to targets occurring at the cued location while accuracy would be greater (a speed-accuracy tradeoff would characterize the performance difference between cued and uncued targets). As such, a slower response to targets at the cued location would allow more perceptual processing to occur which would, in effect, increase accuracy at the cued as compared to the uncued location. As would be predicted from Taylor and Klein's (2000) theoretical framework, this study demonstrated that IOR expressed itself as a speed-accuracy tradeoff when saccadic responses were made to S1 (cues), but not when the oculomotor system was actively suppressed. Inspired by these findings, a primary objective of the present study was to pit these attentional (no speed-accuracy tradeoff observed) and oculomotor (speed-accuracy tradeoff observed) cells against each other while recording EEG to reveal to what extent attentional processes were modulating sensory inputs.

7.2.2 Neural signature of IOR

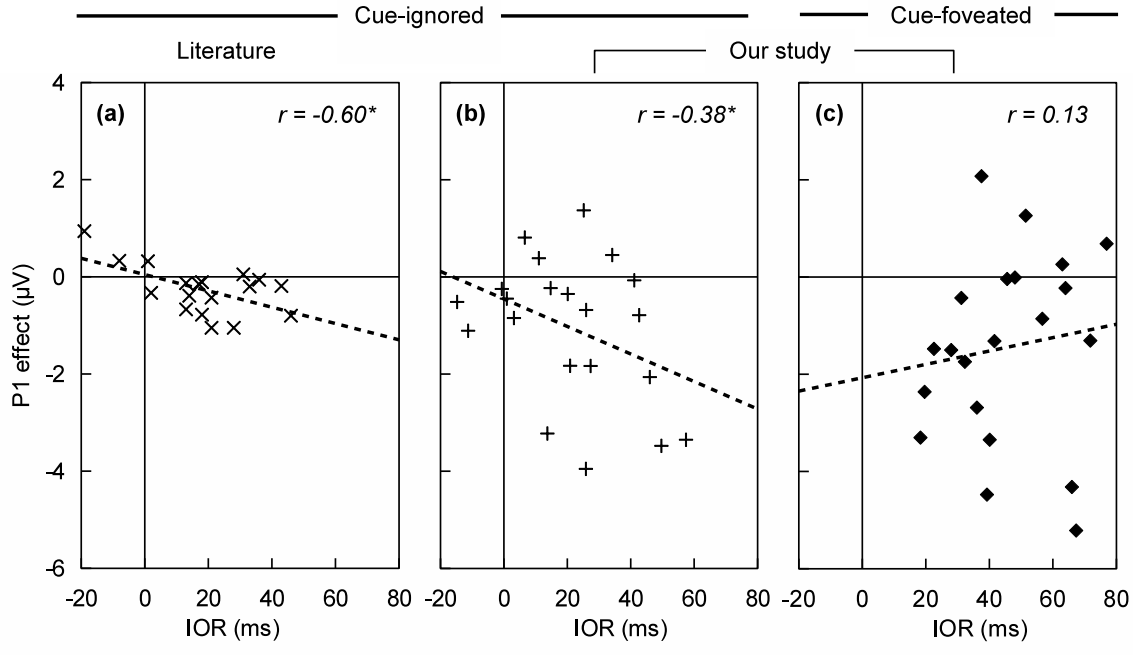

Figure 7.2: Comparison of IOR and P1 cueing effects (cued minus uncued), with r scores representing the correlation size and asterisks representing significance [For r = -0.60, $^{*}p < 0.05$ (two-tailed); for r = -0.38, $^{*}p < 0.05$ (one-tailed), $p < 0.10$ (two-tailed); see text for more details]. (A) X's represent the mean results of 19 published experiments from 9 published manuscripts (Hopfinger & Mangun, 1998; Hopfinger & Mangun, 2001; McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009a; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005) in which a cue-ignored IOR paradigm was used. (B) Data points from the current experiment's cue-ignored condition are represented with +'s. (C) Data points from the current experiment's cue-foveated condition are represented with diamonds. Although these previous studies have verbally discouraged eye movements and have attempted to remove all trials on which eye movements were made, this does not guarantee that there were not trials with eye movements and, even more importantly, it does not guarantee that the reflexive machinery in the oculomotor system was actively suppressed. In the current investigation, we explicitly monitored eye position and provided online (trial-by-trial) feedback about incorrect eye movements or deviations from fixation, so we can unequivocally evaluate an eye movement was made and can therefore ensure that oculomotor responses are not contributing to the data pattern.

ERP investigations of orienting and attention have been particularly fruitful in examining the time course of attentional effects on different stages of processing (for a

review see, Luck, Woodman, & Vogel, 2000). Research has demonstrated that sensory input processing, as revealed with the P1 ERP component (*i.e.*, the first stimulus-induced positive peak in the EEG waveform), which arises from areas such as extrastriate cortex (e.g., Clark & Hillyard, 1996), can be modulated by attention (e.g., Mangun & Hillyard, 1988; Rugg, Milner, Lines, & Phalp, 1987). These studies have determined that the target-elicited early sensory P1 component is significantly enhanced when the target stimulus is attended, relative to when the same stimuli are not attended. In such paradigms, pre-trial, task-induced, attentional control settings lead to enhanced activity within the latency range of the P1 component measured at electrodes over parieto-occipital cortex.

Other studies have recorded EEG while participants performed a spatial cueing task with CTOAs in the range that normally leads to behavioral IOR. In all these studies, subjects have been instructed to maintain fixation throughout trials. To the extent that the observers obeyed this request, these investigations were likely measuring the perceptual/ attentional aftereffect of spatial cueing. In addition to observing IOR behaviorally, many of these studies have also observed significant P1 reductions (P1 cueing effects) for cued targets (McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005; Wascher & Tipper, 2004). However, as illustrated in Figure 7.2, not all experiments that have observed P1 cueing effects have also observed IOR (Doallo, Lorenzo-Lopez, Vizoso, Holguin, Amenedo, Bara, & Cadaveira, 2004; Hopfinger & Mangun, 1998), and not all experiments that have observed IOR have also observed P1 cueing effects (Hopfinger & Mangun, 2001; McDonald et al., 1999; Prime & Ward, 2006; van der

Lubbe et al., 2005), so the relationship between IOR and P1 cueing effects has not yet been conclusively determined.

To the extent that explicitly discouraging saccadic eye movements actively suppresses the oculomotor system and thus leads to a perceptual/attentional form of IOR, we expect to see target-elicited P1 reductions on the cued side as compared to the uncued side. Furthermore, we expect a between-subject relationship between IOR and these target-elicited P1 reductions at long CTOAs akin to that which has been shown on a trial-by-trial basis in single unit activity studies at short CTOAs (Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005). These studies have uncovered a correlation between the neural activity of visuo-motor neurons in the intermediate layer of the superior colliculus and RTs in spatial cueing paradigms wherein monkeys exhibit behavioral IOR. Similarly, a significant correlation is obtained when looking at the mean experimental IOR scores and P1 cueing effects in the ERP literature (see Figure 7.2A).

As described above, a number of previous studies have examined IOR behaviorally along with ERP analyses. Given these results, we performed a correlation analysis on the mean experiment-by-experiment IOR scores and cue-driven P1 modulation effects from all studies which have reported these numbers (19 published experiments from 9 published manuscripts; Hopfinger & Mangun, 1998; Hopfinger & Mangun, 2001; McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009a; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005). As illustrated in Figure 7.2A, previous work has presented a clear pattern of larger (more negative) P1 cueing effects along with increased

IOR scores ($r = -0.60$, $p < 0.05$; two-tailed).

In addition to determining whether the data pattern that has been reported in the literature will be obtained in a condition for which the reflexive oculomotor machinery is truly "turned off", we intend to use ERPs to provide converging evidence for the hypothesis that when the oculomotor system is in an activated state, the nature of the inhibitory aftereffect will be qualitatively different from that when the oculomotor system is actively suppressed. To reiterate, according to Taylor and Klein (2000) and Klein and Hilchey (2011), the oculomotor system is hypothesized to be in an activated state when pro-saccadic responses are permitted/made to spatial inputs. Thus, by requiring observers to make a pro-saccade to the first signal and a manual response to the second signal we should, in theory, be measuring a primarily motoric effect. If this claim is valid, we might expect to see either: 1) no target-elicited P1 reductions at the cued relative to the uncued location, or 2) if there are target-elicited P1 reductions at the cued location, these reductions should bare no relationship to IOR. In two separate blocks of trials, participants either ignored the cue (peripheral onset) or made saccadic eye movements to foveate the cue. EEG data was recorded and gaze position was monitored online with trial-by-trial feedback provided to participants when their oculomotor behavior did not conform to the task requirements.

7.3 Methods

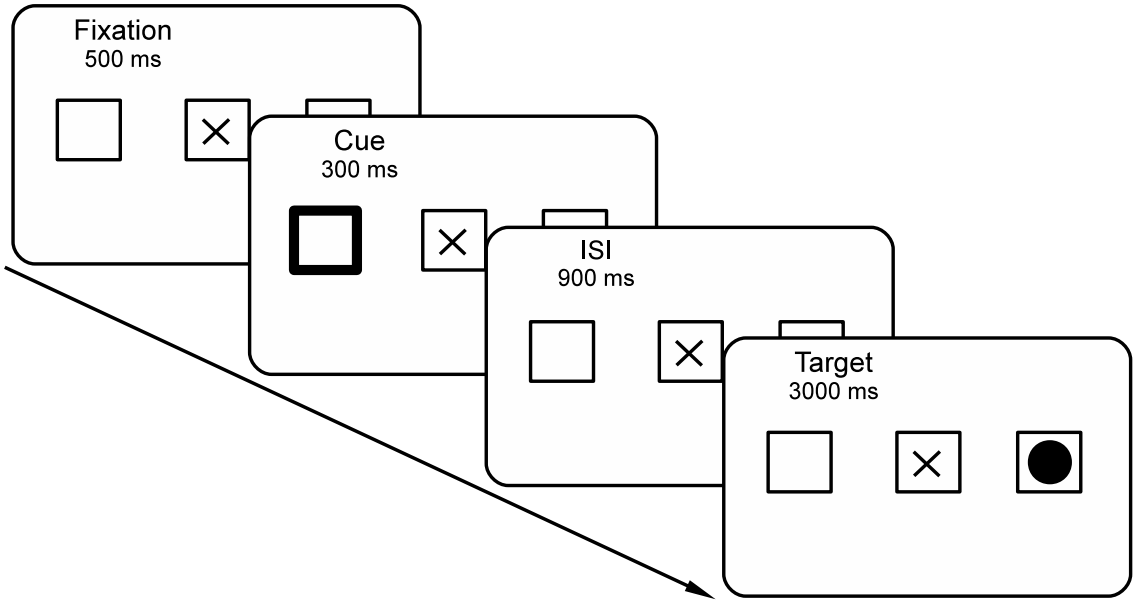

Figure 7.3: Task procedure. The cue-ignored condition required maintenance of central fixation throughout trials. In the cue-foveated condition, saccades were required to the cued location, then back to central fixation. Figure is for illustrative purposes only, stimuli are not drawn to scale (see text for details).

7.3.1 Participants

Twenty-four students took part in this experiment in exchange for extra course credit. They were recruited from the participant pool at Dalhousie University. All participants reported normal, or corrected-to-normal vision. Two participants were excluded from analysis due to technical problems during EEG acquisition and one other participant was excluded due to an excessive number of anticipatory responses. Consequently, the analyses presented here are based on the remaining twenty-one participants (13 females, 8 males; 20.6 years old on average).

7.3.2 Apparatus and stimuli

Presentation of stimuli, timing and behavioral data collection was controlled by a personal computer running Python scripts. Stimuli were presented on a 19" Asus LCD monitor and responses were collected with a Microsoft keyboard. All stimuli were presented in white on a black background. Three boxes (4.5°×4.5° visual angle) were used as placeholders, a fixation cross (0.8°×0.8°) was presented inside the central box, and the distance between the centers of adjacent boxes was 8.7°. Cues appeared as a thickening of one of the peripheral boxes, and targets were bright disks with d = 2.4°. Participants were tested in a dark, electromagnetically shielded room, with their head resting on a chin rest which maintained the visual distance at about 57 cm.

Gaze position was constantly monitored during the experiment using a desktop mounted eye tracking system (EyeLink® 1000) sampling at 250 Hz. EEG data was recorded continuously at 256 Hz with a BioSemi Active-Two amplifier system that used 64 Ag-AgCl electrodes mounted in an elastic cap according to the international 10-20 system. Electrodes were also placed at the outer canthi of the eyes as well as above and below the left eye, and on the mastoids. Two additional electrodes served as recording reference and ground.

7.3.3 Design and procedure

The experimental procedure, as illustrated in Figure 7.3, was similar to the design used by Chica et al. (2010). A self-paced drift correction was performed at the beginning of each trial, after which participants maintained fixation for 500 ms before a cue (non-predictive of target location) appeared at one of the peripheral boxes for a duration of 300

ms. After a 900 ms inter-stimulus interval, the target appeared in one of the peripheral boxes. The target was presented for 3000 ms or until the participant issued a localization response by pressing either the ‘z’ or ‘/’ key for left or right targets, respectively. After an inter-trial interval of 1000-1500 ms, another trial began.

The experiment consisted of two blocked conditions, the order of which was counter-balanced across participants. In one condition, participants made eye movements to the cued box and then back to central fixation before target appearance (cue-foveated), while in the other condition participants maintained central fixation throughout the trials (cue-ignored). Each participant was tested for 200 trials per condition. Before beginning the experiment, subjects were provided with enough practice trials to feel comfortable performing the task.

In the cue-ignored condition, a trial was aborted if the participants' eyes deviated more than 3° from central fixation. In the cue-foveated condition, a trial was additionally aborted if the participant failed to make an eye movement to the cued box and return to central fixation within 600 ms. An error message stating “Invalid eye movement. Press space to continue.” was presented whenever a trial was aborted[10].

7.3.4 Electroencephalographic and behavioral analysis

EEG data was filtered with a highpass filter of 0.1 Hz and a lowpass filter of 30 Hz. Bad electrodes were identified through visual inspection and interpolated using an automatic interpolation technique. Data was then re-referenced to the average of all electrodes, and segmented into epochs beginning 100 ms before, and ending 400 ms after,

[10] Although every aborted trial was put back into the queue to be rerun later and the remaining "upcoming" trials were randomized, the length of each block was still limited to 200 trials to ensure that sessions would end in a timely fashion.

target appearance. After performing a 100 ms baseline correction, trials with excessive artifacts (+/- 75 microvolts) were excluded from analysis. Trials with incorrect behavioral responses or any incorrect eye movements (based on data from the Eyelink 1000 system) were also excluded from further analysis. The P1 ERP component was quantified by measuring each subject's mean EEG amplitude at parieto-occipital electrodes (PO7/8) over a 20 ms window centered around the peak of the P1 component in the grand average waveform for contralateral (130-150 ms) and ipsilateral (140-160 ms) electrodes.

7.4 Results

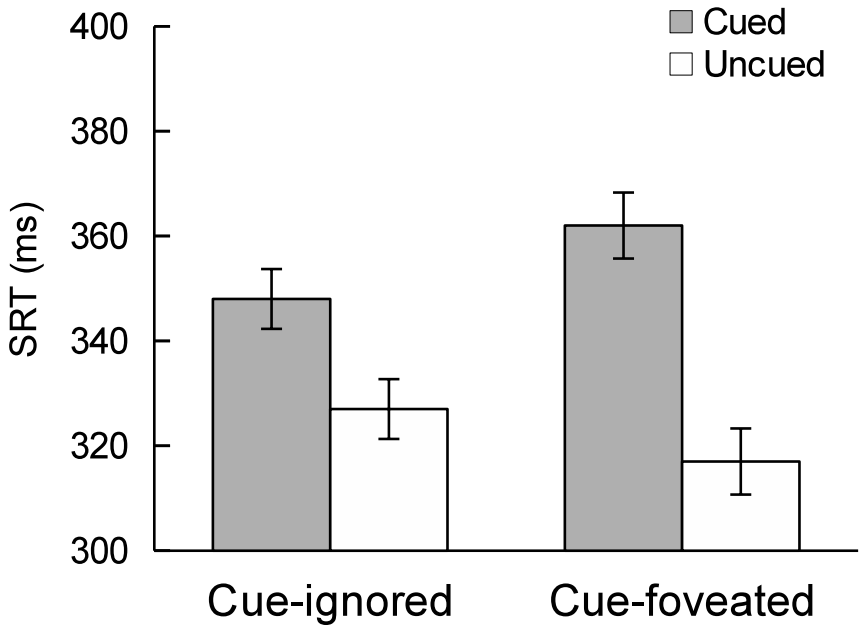

Figure 7.4: Mean reaction time plotted as a function of condition and cueing. Error bars are 95% within-subject confidence intervals based on the error term of the effect of cue condition in each of the conditions.

7.4.1 Behavioral performance

Trials with incorrect responses to targets were excluded from analysis (1.06%). Careful examination of the overall RT distribution of correct responses led to the removal

of another 3.84% of trials in which the RTs were faster than 200 ms or slower than 600 ms (*i.e.*, trials with RTs that were too fast or slow to be considered representative). Trials rejected from the ERP analysis (e.g., trials with excessive artifacts, or faulty electrode activity) were also excluded from all behavioral analyses (11.14%). It should be noted that inclusion of trials with EEG artifacts in behavioral analyses did not change the pattern of the results. The remaining correct RTs were subjected to an ANOVA with the factors condition (cue-ignored or cue-foveated) and cueing (cued or uncued). This analysis revealed a main effect of cueing [$F(1, 20) = 113.6$, $p < 0.001$, $\eta_G^2 = 0.12$; generalized eta squared (Bakeman, 2005)], where observers were slower to respond to cued relative to uncued targets (behavioral IOR). The main effect of condition did not approach significance [$F(1, 20) < 1$, n.s., $\eta_G^2 = 0.00$], suggesting that there was no overall RT difference between cue-ignored and cue-foveated conditions. As can be seen in Table 7.1, behavioral IOR effects were observed in both the cue-ignored (attentional/perceptual) and cue-foveated (motoric) conditions. However, the interaction between cueing and condition also reached significance [$F(1, 20) = 21.6$, $p < 0.001$, $\eta_G^2 = 0.02$], suggesting that stronger IOR was observed when the cue was foveated (46 ms) [$t(20) = 11.82$, $p < 0.001$] than when it was ignored (21 ms) [$t(20) = 4.85$, $p < 0.001$].

7.4.2 Event-related potentials (ERPs)

The primary purpose of the present study was to explore how uninformative cues modulated target-elicited P1s when the oculomotor system was theoretically in either an active or suppressed state (Taylor & Klein, 2000) and to explore how any cue modulated target-elicited P1 activation related to behavioral IOR effects. As can be seen in Figure

7.4, cued targets elicited smaller P1 components than uncued targets in both conditions. This cue-modulated P1 effect (reduced amplitudes for cued targets) was accompanied by a behavioral effect (increased RTs for cued targets; IOR; see Table 7.1).

Condition	RT (ms)			P1 cueing effect (μV)		IOR-P1 cueing effect correlation	
	Cued	Uncued	IOR	Ipsi	Contra	Ipsi	Contra
Cue-ignored	348	327	21**	-1.05**	0.13	-0.38*	-0.13
Cue-foveated	362	317	45**	-1.44**	-0.66	0.13	-0.13

*Table 7.1: Summary of behavioral and ERP data. P1 cueing effects and IOR scores are calculated as cued minus uncued P1s and RTs, respectively [**$p < 0.01$ (one-tailed); *$p < 0.05$ (one-tailed), $p < 0.10$ (two-tailed)].*

For statistical analyses, the P1 ERP component was quantified as the average activity over a 20 ms time window centered around the P1 peaks in the grand average waveforms of parieto-occipital electrodes (PO7/8). As illustrated in the topographical activity maps presented in the Appendix (Figure 7.6), neural activity was maximal over ipsilateral parieto-occipital sites during the period of ERP analysis. For easy comparison to behavioral IOR effects (RT differences between cued and uncued targets), we calculated the mean amplitude difference between cued and uncued target-elicited P1 ERP components (cued minus uncued), which we refer to as the P1 cueing effect. Table 7.1 summarizes the P1 cueing effects numerically.

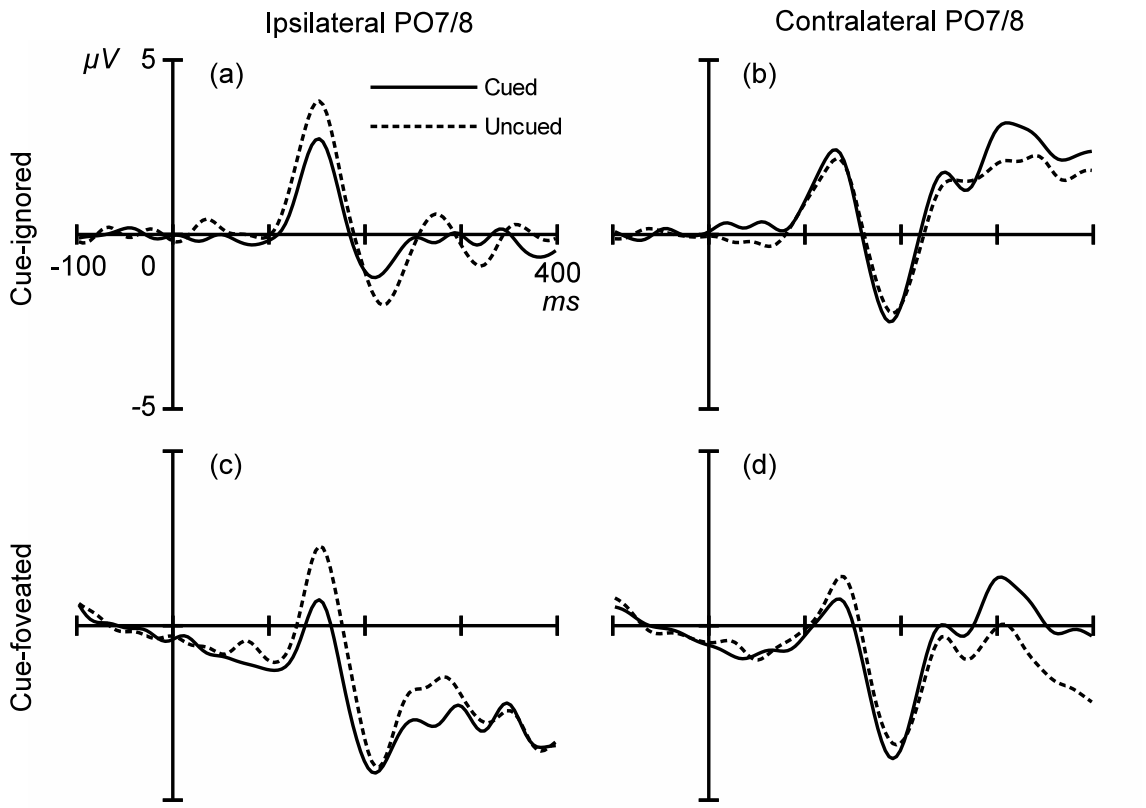

Figure 7.5: Target-elicited ERP waveforms at ipsilateral and contralateral parieto-occipital electrodes (PO7/8) for the cue-ignored and cue-foveated conditions.

ANOVAs with factors condition (cue-ignored vs. cue-foveated) and cueing (cued vs. uncued) were performed on P1 components separately for ipsilateral and contralateral electrodes (PO7/PO8). For ipsilateral electrodes, a significant main effect of condition was observed [$F(1, 20) = 14.15$, $p < 0.01$, $\eta_G^2 = 0.20$], with larger P1s in the cue-ignored (3.09 μV) than in the cue-foveated condition (1.26 μV). This main effect was probably caused by an overall negative drift following the eye movements made in the cue-foveated condition. It should be noted that this negative drift was similar for cued and uncued targets. A significant main effect of cueing was also observed [$F(1, 20) = 16.98$, $p < 0.001$, $\eta_G^2 = 0.10$], with overall smaller P1s observed for cued (1.55 μV) than for

uncued (2.80 μV) targets. The interaction between condition and cueing did not reach significance [$F(1, 20) = 0.78$, n.s., $\eta_G^2 = 0.00$], suggesting that the P1 cueing effect was similar across conditions. Planned comparisons revealed significant P1 reductions for cued targets in both the cue-ignored (1.05 μV) [$t(20) = 3.24$, $p < 0.01$)], and cue-foveated (1.45 μV)[$t(20) = 3.41$, $p < 0.01$] conditions. For contralateral electrodes, only a main effect of condition was observed [$F(1, 20) = 5.94$, $p < 0.05$, $\eta_G^2 = 0.04$]. This main effect also reflected the negative shift observed in the cue-foveated condition (see Figure 7.4).

7.4.3 Relation between P1 modulations and the cueing effect

As demonstrated in previous studies (e.g., McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005; Wascher & Tipper, 2004) and the present experiment, behavioral IOR effects are normally accompanied by a reduction of the early sensory P1 component. Although precise localization of early sensory attentional modulation is still inconclusive, Figure 7.6 demonstrates that the P1 cueing effects seen here are maximal at ipsilateral parieto-occipital sites, as observed previously (e.g., Chica & Lupianez, 2009). To provide a more rigorous test of the relationship between IOR (behavioral cueing effects) and P1 modulation effects, we calculated the subject-by-subject correlation between IOR and P1 cueing effects for both the cue-foveated (motoric flavor) and cue-ignored (attentional/perceptual flavor) conditions. In the cue-foveated condition, IOR did not correlate with ipsilateral P1 cueing effects ($r = 0.13$, n.s.; see Figure 7.2C). However, in the cue-ignored condition, a clear negative correlation was observed between IOR and ipsilateral P1 cueing effects [$r = - 0.38$, $p < .05$ (one-tailed), p

< 0.10 (two-tailed); see Figure 7.2B]. As can be seen in Figure 7.2B, the association observed in the cue-ignored condition (relative to uncued targets, larger reductions in P1 amplitude are associated with larger RT increases for cued targets) does not seem to be driven by outliers.

7.5 Discussion

Previous EEG studies have demonstrated that P1 component amplitudes are reduced on cued trials in spatial cueing paradigms when IOR is expressed behaviorally (McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005; Wascher & Tipper, 2004). The present study is the first ERP study of IOR that: 1) uses eye tracking equipment to ensure that observers were conforming to task demands, and 2) compares a variation on the spatial cueing paradigm in which the oculomotor system has been hypothesized to be engaged (pro-saccadic response to S1, keypress response to S2) against a version in which the oculomotor system has been hypothesized to be actively suppressed. With regard to the activation state of the oculomotor system in the no response to S1 - manual response to S2 condition, since subjects made very few errors we have little reason to believe that the state of the oculomotor system was anything other than actively suppressed. Following the theoretical framework proposed in the introduction, this allows for comparisons between P1 activation levels and spatial cueing when the oculomotor system is inferred to be "actively suppressed" (no response to S1-manual response to S2) or "engaged" (saccadic response to S1 - manual response to S2).

Replicating previous findings, IOR was found to be accompanied by a P1 cueing

effect. In addition, the present experiment produced several important observations. First, significant P1 cueing effects were observed whether the cues were ignored (suppressed oculomotor system) or foveated (active oculomotor system). Second, a significant correlation between the P1 reduction and IOR was only observed in the cue-ignored condition (where fixation was maintained throughout a trial). When saccades were made to the cue, there was no correlation between P1 cueing effects and IOR scores. Third, stronger IOR was observed when the oculomotor system was actively involved in the cue-foveated condition. The theoretical and empirical implications of these findings are considered below.

7.5.1 Why did foveated S1s lead to stronger IOR?

Since the two conditions of this experiment were blocked, differences in IOR scores across conditions are likely to be explained by different attentional control settings induced by different task demands in the two conditions (*cf.* Hilchey, Klein, & Ivanoff, 2012a; Wang & Klein, 2012). In the cue-ignored condition, subjects have little reason to attend the periphery at the time of cue presentation, since no response is required to the cue, and cues are uninformative with regard to target location. However, in the cue-foveated condition, saccadic responses are required to the cues, so the peripheral cue locations must be attended at the time of cue presentation. Thus, the cue-ignored condition should result in a much tighter attentional control setting focused on central fixation, whereas the cue-foveated condition should result in a more diffuse attentional control setting that encompasses the peripheral cue locations. This could result in a weaker intensity attentional beam at peripheral cue locations in the cue-ignored condition,

relative to the cue-foveated condition. Since previous work has demonstrated that IOR can be reduced or eliminated for cues presented outside of a spatial attentional control setting, the larger IOR score seen in the cue-foveated (46 ms) as compared to the cue-ignored (21 ms) condition is not surprising[11].

However, this finding is seemingly in contradiction to Taylor and Klein (2000), who found mathematically larger IOR effects with manual responses following ignored (21 ms) than foveated (14 ms) cues (a difference that was not subjected to a statistical test by Taylor & Klein, 2000). How has this occurred? There are two obvious methodological differences between Taylor and Klein (2000) and the present investigation: 1) Taylor and Klein (2000) adopted the convention of delivering a cue-back to fixation which, in theory, helped to ensure that any attention that may have been drawn to the first, spatially irrelevant peripheral onset signal was returned to fixation (a neutral baseline; see Klein 2004), and 2) in Taylor and Klein (2000), centrally presented arrow signals were randomly intermixed with peripheral onset signals whereas we only presented peripheral onset signals. Since both of these experimental manipulations are known to have strong effects on IOR scores (Wang, Satel, & Klein, 2012a; Prime and Jolicoeur (2009b); Wang & Klein, 2012; Hilchey et al., 2012a), it is likely that the differences in behavioral results arise from these differences in experimental design.

As discussed in Wang et al. (2012a), the presence of a visual cue-back could be contaminating the IOR scores in conditions when the cue is foveated. Since the central fixation location is in the same retinotopic representation as the uncued target location at

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[11] For demonstrations showing that making the fixation location more relevant can reduce/eliminate the magnitude of IOR, see Wang and Klein (2012) and Hilchey, Klein, and Ivanoff (2012).

the time of cue-back onset, an inhibitory cueing effect could be operating on the uncued target location. Thus, when comparing cued to uncued RTs, we may actually be comparing locations that have each been subjected to inhibitory mechanisms (*cf.* Satel, Wang, Trappenberg, & Klein, 2011; Wang, Satel, Trappenberg, & Klein, 2011; Wang et al., 2012a). This could explain the difference across studies in the cue-foveated condition IOR scores, with much less IOR observed in Taylor and Klein (2000; 14 ms) than here (46 ms) because of a competing inhibitory mechanism operating at the uncued location.

In the present investigation, it is worth reiterating that there were never task-relevant events at fixations (*i.e.*, there were neither central arrows nor cue-backs to fixation). By contrast, as aforementioned, imperative signals occurred at fixation in both conditions (saccadic response to S1 - manual response to S2) in Taylor and Klein (2000). As stated above, we believe that it is possible that the absence of relevant inputs occurring at fixation will allow for greater processing of the peripheral S1 events in our experiment as compared to Taylor and Klein (2000) because there was no task demand (*i.e.*, relevant fixation events or the requirement to remain fixated) that strongly encouraged increased processing at the fixation stimulus (at the cost of peripheral processing; see Wang et al., 2011; Hilchey et al., 2012a).

7.5.2 IOR and P1 modulation as a function of the state of the oculomotor system

Previous findings from a wide range of studies have indirectly support the relationship between P1 cueing effects and the behavioral expression of IOR. On the one hand, that there are many differences between these studies suggests caution when interpreting these results as whole (see Figure 7.2A). On the other hand, this

methodological eclecticism, together with so many observations, suggests that the significant correlation observed here is probably closely related to what they have in common: cued versus uncued targets with CTOAs long enough for IOR to be present. The correlation between IOR and P1 cueing effects for these previous experiments, none of which required eye movements, is significant ($r = -0.60$, $p < 0.05$). Thus, at least in cases when the oculomotor system is not required to be actively engaged to meet task requirements, it seems that there is a legitimate association between P1 cueing effects and IOR.

To further investigate the relationship between IOR and P1 modulations, we looked at the subject-by-subject correlations between these measurements in the current experiment. Because Taylor and Klein (2000) claimed that the effect of IOR is primarily attentional/perceptual when the oculomotor system is "actively suppressed", we expected a correlation between IOR and P1 modulation in the no response to S1 - manual response to S2 condition. By contrast, because Taylor and Klein (2000) claim that the effect of IOR is primarily (if not entirely) motoric when the oculomotor system is "engaged", we expected that there would be no P1 modulations in the saccadic response to S1 - manual response to S2 condition.

This inference was recently supported by an investigation highlighted in the introduction (Chica et al., 2010) and on which the present investigation was largely based. Chica et al. (2010) demonstrated that in the aftermath of a saccadic eye movement to a region in the visual field, and a subsequent return eye movement to fixation, observers were *slower* but more *accurate* to identify a target occurring at previously foveated as

compared to unfoveated regions. By contrast, when observers withheld oculomotor responses to spatially-irrelevant peripheral onset cues and subsequently identified a target at the cued or uncued region, RTs were *slower* for the cued region and response *accuracy* was either equivalent between the cued and uncued region or perhaps slightly worse at the cued region.

Taken together, the foregoing results suggest that in the presence of saccadic eye movements or, possibly, when eye movements are permitted (whether or not they are actually executed), the effect of IOR is akin to the "criterion shift" account (Klein & Taylor, 1994) that was expounded on by Ivanoff, Klein, and Lupianez (2002). In broad strokes, this theory posits that IOR affects decision-making such that increased evidence is required before response-execution (*i.e.*, there is an increased response criterion for the cued relative to uncued location) while the accrual of information at cued and uncued locations remains equivalent. In stark contrast, the effect of IOR when eye movements are strictly forbidden (the oculomotor system is "inactive" if not actively inhibited) is in better agreement with Posner and Cohen's (1984) "inhibited attention" hypothesis. As conceptualized by Ivanoff, Klein, and Lupianez (2002), this theory posits that there are slower RTs to targets at the cued as compared to the uncued location because of a delay in the activation of the task-relevant stimulus-response code (*cf.* Hilchey, Ivanoff, Taylor, & Klein, 2011); this effect *could* arise simply because the quality of visual information processing at the cued location is degraded to some extent (*i.e.*, a perceptual/attentional account).

Converging on the claim that the effect of IOR is primarily attentional/perceptual

when the oculomotor system is "actively suppressed", P1 cueing effects were correlated with IOR in the no response to S1 - manual response to S2 condition. On the other hand, there was no apparent relationship between IOR and P1 modulations when the oculomotor system was "engaged". This is a striking result[12]. The most immediate implication is that whereas IOR is commonly associated with P1 reductions in non-eye movement spatial cueing studies, this pattern – whether or not it is indexing the existence of an attentional effect of IOR in the brain – only expresses itself behaviorally when the oculomotor system is "actively suppressed". Thus, following an act of overt orienting toward a peripheral stimulus (and return to the original fixation) there may be a reduction in input pathways but, this effect does not appear to significantly influence manual RTs. Future work is encouraged to further investigate how this comes about.

7.6 Conclusion

This experiment examined IOR when generated by ignored or foveated cues, while simultaneously recording EEG and eye position and providing feedback to participants about any incorrect oculomotor behavior. Our motivation was two-fold: 1) we wanted to ensure that previous EEG work on IOR was not compromised by the presence of unintended saccadic eye movements to cues or targets, and 2) we wanted to evaluate the claim from Taylor and Klein (2000) that the activation state of the oculomotor system would determine the effect of IOR (*i.e.*, motoric or attentional/perceptual). When the oculomotor system was "actively suppressed", we observed IOR, P1 cueing effects, and a

[12] Although the correlation for our cue-ignored condition was only significant with a one-tailed test,when compared against each other (for details on this analytical technique see Steiger, 1980), the correlation seen in previous work (r = -0.60) was not significantly different from that obtained in our cue-ignored condition (r = -0.38). Furthermore, the correlation obtained in our cue-foveated condition (r = 0.13) *was* significantly different from that in the cue-ignored condition (Z = -3.04). This provides converging evidence that two different mechanisms are likely operating in the two conditions.

statistically significant brain-behavior relationship between these dependent variables. When the oculomotor system was “engaged”, we observed statistically equivalent P1 cueing effects compared to those obtained when the oculomotor system was “actively suppressed”, behavioral IOR that was a statistically greater than when the oculomotor system was “actively suppressed”, and no relationship between P1 cueing effects and IOR. On the balance, our data are consistent with the idea that P1 cueing effects in IOR paradigms result from the successive presentation of peripheral stimuli and that this P1 cueing effect is closely associated with RT when the oculomotor system is suppressed. By contrast, there is no apparent association between P1 cueing effects and IOR when the oculomotor system is activated due to requiring saccades to cues.

7.7 Acknowledgements

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7.9 Appendix

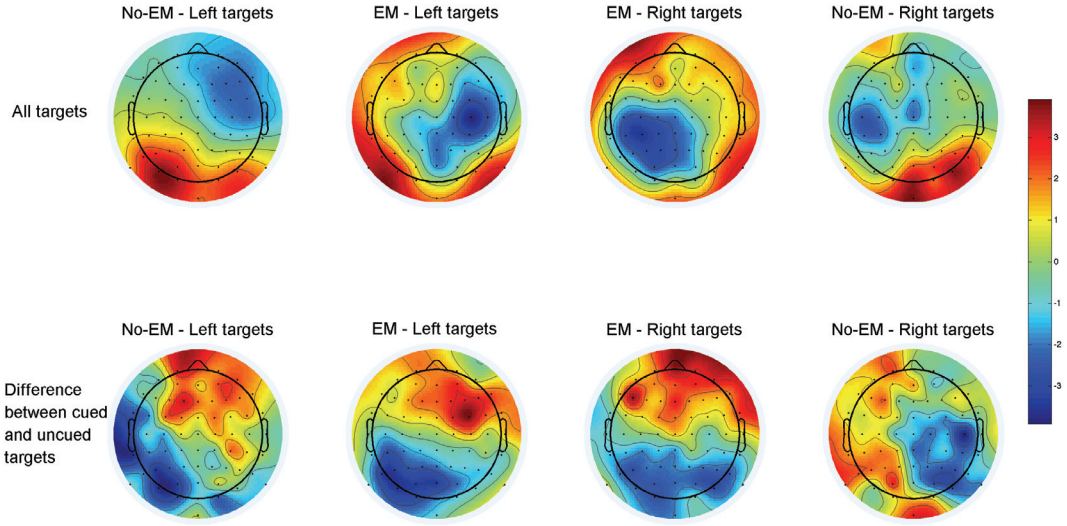

Figure 7.6: Topographical activity plots averaged over the time period used for ipsilateral P1 analyses (140-160 ms post-cue). Top row: mean of cued and uncued activity for left and right targets in both conditions. Bottom row: difference between cued and uncued activity for left and right targets in both conditions.

CHAPTER 8 EXAMINING THE DISSOCIATION OF RETINOTOPIC AND SPATIOTOPIC INHIBITION OF RETURN

Satel, J., Wang, Z., Hilchey, M. D., and Klein, R. M. (2012). Examining the dissociation of retinotopic and spatiotopic inhibition of return with event-related potentials. Neuroscience Letters, 524(1): 40-44 (reformatted and reprinted with permission of the publisher, Elsevier).

8.1 Abstract

Inhibition of return (IOR) is thought to reflect a mechanism that biases orienting which, under some circumstances, reduces perceptual processing at previously processed locations. Studies using event-related potentials (ERPs) have generally revealed that IOR is accompanied by an amplitude reduction of early sensory ERP components (P1). While behavioral studies suggest that IOR may be represented in both spatiotopic and retinotopic coordinates, all previous ERP studies have used the prototypical spatial cueing paradigm and have thus confounded retinotopic and spatiotopic reference frames. Because of this confound it is unknown whether the P1 reduction that has been associated with IOR will be observed in retinotopic or spatiotopic coordinates when these are dissociated. The current experiment investigated whether the P1 component would be modulated by IOR when the retinotopic and spatiotopic reference frames were dissociated by an eye movement between cue and target onset. Strong spatiotopic IOR was found to be accompanied by a negative difference (Nd) in the 200-300 ms time window, while a P1 reduction was absent, suggesting that P1 reductions do not provide an accurate reflection of IOR.

8.2 Introduction

In a spatial cueing paradigm, when the interval between an uninformative peripheral onset cue and peripheral target [cue-target onset asynchrony (CTOA)] exceeds approximately 300 ms, inhibition of return (IOR) reveals itself behaviorally as slowed response times (RTs) to cued, as compared to uncued targets (Klein, 2000; Posner & Cohen, 1984). In effect, this observation has been characterized to represent a reluctance

to reorient to previously inspected locations (Posner & Cohen, 1984). This theoretical attribution has inspired the proposal that IOR serves to bias search against previously processed regions in the visual field (Klein, 1988; Posner, Rafal, Choate, & Vaughan, 1985) and has led to dedicated scientific explorations of this possibility (Wang & Klein, 2010).

Humans normally make 2-3 rapid eye movements (saccades) per second, leading to a constantly changing retinal image of the environment. In order to accomplish the aforementioned functional significance, IOR must be coded in spatiotopic (environmental), rather than retinotopic (retinal) coordinates (Maylor & Hockey, 1985; Posner & Cohen, 1984; Posner et al., 1985). To dissociate spatiotopic from retinotopic reference frames, it is necessary to introduce an eye movement (EM) between cue and target onset. This approach has demonstrated that IOR can be observed in spatiotopic coordinates as soon as an EM shifts the retinotopic to a spatiotopic reference frame (Hilchey, Klein, Satel, & Wang, 2012b; Pertzov, Zohary, & Avidan, 2010).

Because of the proposed relationship between spatial orienting and IOR, we turned to the literature measuring event-related potentials (ERPs) in spatial cueing paradigms, where neurophysiological markers of attention have been identified. For example, when attention is endogenously directed toward a particular region in the visual display, behavioral facilitation is accompanied by P1 enhancements (Luck, Woodman, & Vogel, 2000). At longer CTOAs, using paradigms known to produce IOR, the early P1 ERP component is normally reduced in magnitude for cued as compared to uncued target locations (McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009; Prime & Ward,

2004; Prime & Ward, 2006; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005; Wascher & Tipper, 2004). These findings have given rise to the idea that differences in P1 activation reflect the modulation of early sensory processes by mechanisms related to attentional orienting and IOR (Prime & Ward, 2006).

Since maps in the early visual cortices are retinotopically organized, the P1 cueing effect that is measured in traditional spatial cueing paradigms seems to imply a retinotopic coding of IOR which, on the surface, seems difficult to reconcile with the well-known observation that IOR exists in spatiotopic coordinates. However, no previous ERP study of IOR has dissociated retinotopic and spatiotopic reference frames. Here, we sought to determine whether a P1 cueing effect – in the presence of behaviorally observable spatiotopic IOR – would be observed when the spatiotopic reference frame was not confounded by the retinotopic reference frame. Such an effect, if observed, would be consistent with the idea that IOR reflects an attentional modulation of sensory processing at previously processed locations. To evaluate the prospective relationship between perceptual processing and IOR, EEG activity was recorded while participants performed a version of the classic spatial cueing paradigm where an EM intervenes between cue and target onset (Maylor & Hockey, 1985) and where, thus, retinotopic and spatiotopic reference frames are unconfounded.

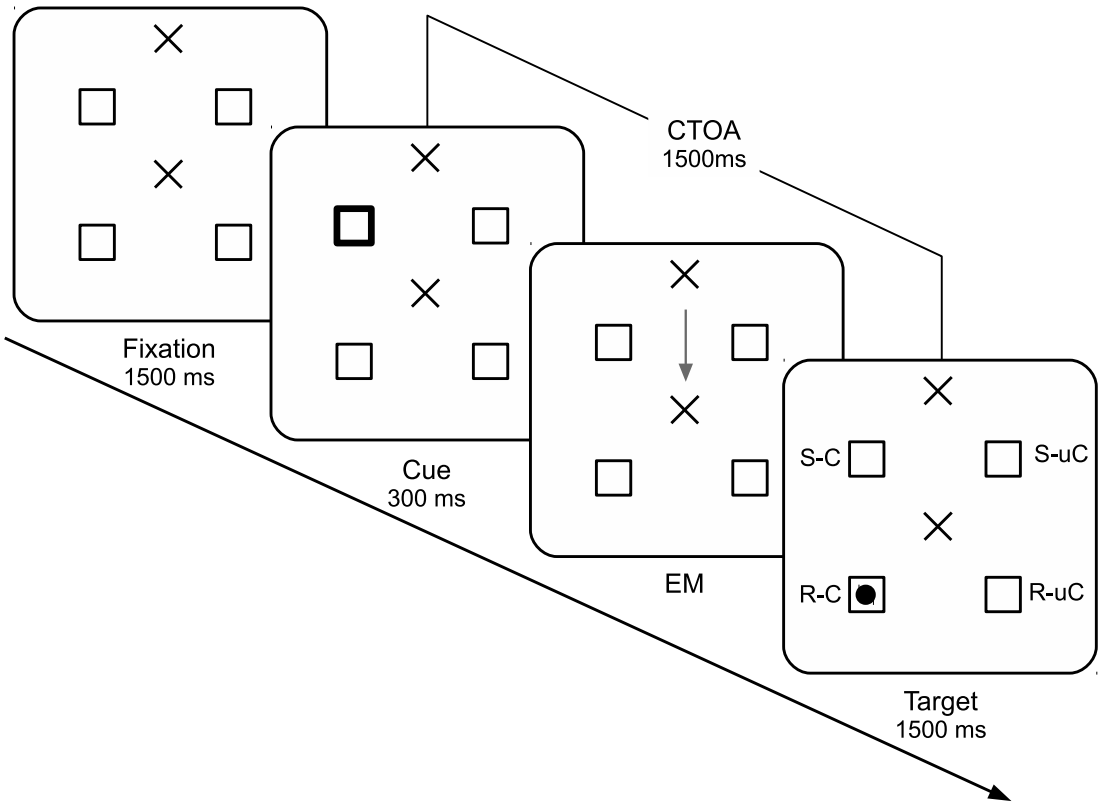

Figure 8.1: Task procedure. Participants fixated the upper fixation cross until cue offset, then made an eye movement (EM; indicated by a gray arrow) to the central fixation cross. In this illustration, the cue appears at the upper left box and the target appears in the lower left box. Consequently, the four possible target locations are labeled S-C (spatiotopic-cued), S-uC (spatiotopic-), R-C (retinotopic-cued) and R-uC (retinotopic-uncued), respectively.

8.3 Methods

Twenty-four university students (17 female; average age 22.2) took part in this experiment in exchange for course credit. They were recruited from the participant pool at Dalhousie University and all reported normal, or corrected-to-normal, visual acuity. Presentation of stimuli, timing operations, and behavioral data collection was controlled by an AMD Athlon 64 personal computer running Python scripts. Stimuli were presented on a 19-inch Asus LCD monitor and responses were collected with a Microsoft keyboard.

All participants were tested in a dimly lit, electromagnetically shielded room, with a chin rest used to maintain a viewing distance of about 57 cm. All stimuli were presented in white on a black background. The initial display consisted of four gray landmark boxes (1.5° × 1.5°, visual angle) forming the corners of a box 6° above/below and left/right of a central fixation cross, as well as another fixation cross presented 12° above center (see Figure 8.1). Cues appeared as a brightening (and thickening) of one of the upper landmark boxes, while targets were bright disks (d = 0.75°) inside one of the landmark boxes. EEG was recorded continuously at 256 Hz with a BioSemi Active-Two amplifier system with 64 Ag-AgCl electrodes mounted in an elastic cap according to the international 10-20 system along with electrodes placed on the mastoids.

The experimental procedure, as illustrated in Figure 8.1, was similar to Experiment 3 of Maylor and Hockey (1985). The participant maintained fixation at the upper fixation cross for 1500 ms before a cue appeared at one of the upper landmark boxes for 300 ms. Participants moved their eyes to central fixation as soon as they detected the offset of the cue. 1500 ms after the onset of the cue, the target appeared in one of the four landmark positions for 1500 ms, or until the participant responded to the target by pressing the space bar. After an inter-trial interval of 750-1500 ms, another trial began. All participants were tested for a total of 384 trials after performing practice trials until participants felt comfortable performing the task.

Offline, data was digitally filtered with a highpass filter of 0.1 Hz and a lowpass filter of 30 Hz. Bad electrodes were identified through visual inspection and interpolated from surrounding electrodes. Data was then re-referenced to the mean of the mastoid

electrodes, and segmented into epochs starting 100 ms before target onset and ending 400 ms after target appearance. After performing a 100 ms baseline correction, trials with excessive artifacts (+/- 75 microvolts) were excluded from analysis. Trials with incorrect behavioral responses or any incorrect EMs (as detected by the eye monitoring system) were also excluded from further analysis. The P1 and N1 ERP components were quantified by measuring each subject's mean EEG amplitude at parieto-occipital electrodes (PO7/8) over a 40 ms time window centered around the peak of the components in the ipsilateral (P1: 120-160 ms; N1: 180-220 ms) and contralateral (P1: 110-150 ms; N1: 160-200 ms) grand average waveforms. The Nd component was quantified as mean amplitude at PO7/8 between 220-300 ms post-target.

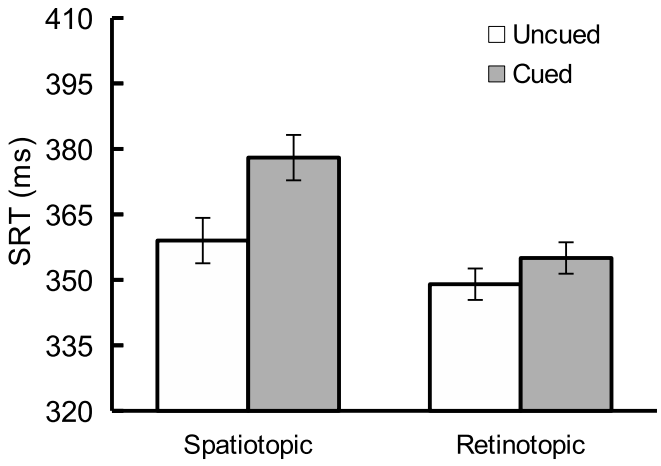

Figure 8.2: Mean reaction time plotted as a function of coordinate frame and cue condition. In each data figure, error bars are 95% within-subject confidence intervals based on the error term of the effect of cue condition in each of the coordinate conditions.

8.4 Results

Trials on which participants missed the target were discarded (1.69%). Trials with RTs faster than 200 ms (4.38 %) or slower than 800 ms (2.79%) were also excluded from

analysis. Trials rejected for behavioral reasons were also excluded from subsequent ERP data analysis. Trials with artifacts in the EEG data were also excluded from ERP data analysis (8.29%). The mean RT and the mean amplitude of the P1, N1, and Nd ERP components for each condition are presented in Table 8.1. IOR effects were calculated as the difference between RTs to cued and uncued targets (cued RT - uncued RT). A similar difference score was calculated for the ERP components, which we refer to as P1, N1, and Nd cueing effects.

	Spatiotopic			Retinotopic		
	Cued	Uncued	Cueing effect	Cued	Uncued	Cueing effect
RT (ms)	378.0	358.6	19.4**	355.1	348.9	6.2*
P1 (μV)						
Ipsi	2.65	2.75	0.10	1.80	2.37	-0.57&
Contra	1.79	1.82	-0.03	0.48	0.39	0.10
N1 (μV)						
Ipsi	0.98	1.45	-0.47	0.33	0.88	-0.55&
Contra	0.13	0.56	-0.43	-1.91	-1.93	0.02
Nd (μV)						
Ipsi	2.01	2.80	-0.79&	3.16	3.63	-0.47
Contra	2.86	4.13	-1.27**	3.17	3.35	-0.18

*Table 8.1: Mean RTs and ERP amplitudes for cued and uncued target in each condition (**p < 0.001, *p < 0.05, & p < 0.14). Cueing effects are calculated as cued minus uncued RTs and ERPs.*

An ANOVA of the RTs (see Figure 8.2), with the variables Coordinates (retinotopic vs. spatiotopic) and Cueing (cued vs. uncued), revealed a significant main effect for both Coordinates [$F(1, 23) = 11.72$, $p < 0.01$] and Cueing [$F(1, 23) = 44.40$, $p < 0.001$]. The former main effect is a result of faster RTs for retinotopic than spatiotopic

trials. Whereas the latter main effect suggests an overall IOR effect, a significant interaction between Coordinates and Cueing was also observed [$F(1, 23) = 8.10$, $p < 0.01$], signifying stronger IOR in the spatiotopic (19.4 ms) than in the retinotopic (6.2 ms) condition. Planned comparisons revealed that IOR effects were significant for both spatiotopic [$F(1, 23) = 30.59$, $p < 0.001$] and retinotopic [$F(1, 23) = 6.65$, $p < 0.05$] Coordinates. These observations are in agreement with those reported by Maylor and Hockey (1985), whose spatiotopic and retinotopic IOR effects were about 38 ms and 7 ms, respectively.

ANOVAs were performed on mean P1 amplitudes separately for ipsilateral and contralateral electrodes (PO7/8), with variables Coordinates (retinotopic vs. spatiotopic) and Cueing (cued vs. uncued). For ipsilateral electrodes, there was no main effect of Coordinates [$F(1, 23) = 2.91$, $p = 0.10$] or Cueing [$F(1, 23) = 1.75$, $p = 0.20$], and there was no interaction between Cueing and Coordinates [$F(1, 23) = 1.04$, $p = 0.32$]. Planned comparisons were performed for the both coordinate conditions because the retinotopic condition produced a numerically large P1 cueing effect not observed for spatiotopic targets and the literature strongly suggests the existence of P1 cueing effects. This analysis revealed no main effect of Cueing for either the spatiotopic [P1 cueing effect = -0.10 μV ; $F(1, 23) = 0.11$, $p = 0.74$] or the retinotopic [P1 cueing effect = -0.57 μV; $F(1, 23) = 2.22$, $p = 0.15$] condition. For contralateral electrodes, the ANOVA revealed a main effect of Coordinates [$F(1, 23) = 16.89$, $p < 0.001$], reflecting larger overall P1s for targets at spatiotopic locations, but neither the main effect of Cueing [$F(1, 23) = 0.03$, $p = 0.86$], nor the interaction between Cueing and Coordinates [$F(1, 23) = 0.06$, $p = 0.81$]

were significant. Planned comparisons revealed no main effect of cueing for either the spatiotopic [P1 cueing effect = -0.03 μV; $F(1, 23) = 0.01$, $p = 0.90$] or the retinotopic [P1 cueing effect = 0.10 μV; $F(1, 23) = 0.06$, $p = 0.81$] condition.

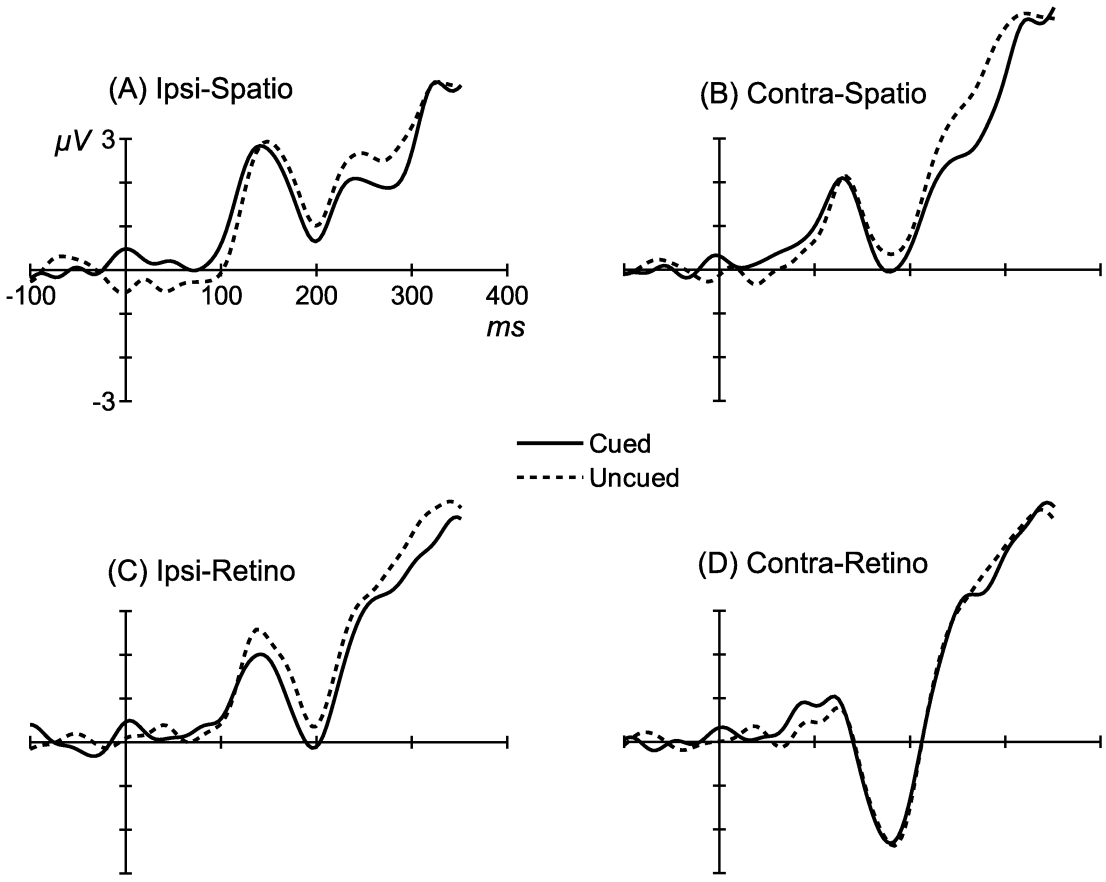

Figure 8.3: Target-elicited ERP waveforms and at ipsilateral (A & C) and contralateral (B & D) parieto-occipital electrodes (PO7/8) for retinotopic (C & D) and spatiotopic (A & B) conditions.

Mean N1 amplitudes were subjected to the same statistical tests as the P1s. For ipsilateral electrodes, there was a significant main effect of Cueing [$F(1, 23) = 4.60$, $p < 0.05$], due to more negative N1s for uncued than cued targets, while the main effect of Coordinates [$F(1, 23) = 2.03$, $p = 0.17$] and the 2-way interaction [$F(1, 23) = 0.03$, $p =$

0.88] did not reach significance. Planned comparisons revealed that the main effect of Cueing was not significant for either the spatiotopic [N1 cueing effect = -0.47 μV; $F(1, 23) = 1.67$, $p = 0.21$] or the retinotopic [N1 cueing effect = -0.55 μV; $F(1, 23) = 2.46$, $p = 0.13$] conditions. For contralateral electrodes, there was a significant main effect of Coordinates [$F(1, 23) = 19.01$, $p < 0.001$], due to larger overall N1s in the retinotopic condition. The main effect of Cueing [$F(1, 23) = 0.74$, $p = 0.40$] and the 2-way interaction [$F(1, 23) = 0.69$, $p = 0.42$] did not reach significance. Planned comparisons failed to reveal a significant main effect of Cueing for either the spatiotopic [N1 cueing effect = -0.43 μV; $F(1, 23) = 1.59$, $p = 0.22$] or retinotopic [N1 cueing effect = 0.02 μV; $F(1, 23) = 0.00$, $p = 0.96$] conditions.

ANOVAs were also performed on mean Nd amplitudes with factors Coordinates and Cueing. For ipsilateral electrodes, a main effect was observed for Coordinates [$F(1, 23) = 4.50$, $p < 0.05$], reflecting more positive amplitudes for the retinotopic condition. A main effect was also observed for Cueing [$F(1, 23) = 8.00$ $p < 0.01$], reflecting more negative amplitudes for the cued condition, but the interaction was not significant [$F(1, 23) = 0.29$, $p = 0.60$]. Planned comparisons revealed a marginally significant main effect of Cueing for the spatiotopic condition [Nd cueing effect = -0.79 μV; $F(1, 23) = 3.79$, $p = 0.06$], that was not significant in the retinotopic condition [Nd cueing effect = -0.47 μV; $F(1, 23) = 2.08$, $p = 0.16$]. For contralateral electrodes, the ANOVA revealed a main effect of Cueing [$F(1, 23) = 12.07$, $p < 0.01$], reflecting more negative amplitudes for the cued conditions, and an interaction between Cueing and Coordinates [$F(1, 23) = 4.45$ $p < 0.05$], demonstrating that the Nd cueing effect was larger for the spatiotopic condition. The main

effect of Coordinates did not reach significance [$F(1, 23) = 0.22$, $p = 0.65$]. Planned comparisons revealed a main effect of Cueing for the spatiotopic [$F(1, 23) = 15.22$, $p < 0.001$], but not the retinotopic condition [$F(1, 23) = 0.31$, $p = 0.58$].

8.5 Discussion

Historically, several imaging techniques, including extracellular recording, EEG, and fMRI, have been used to explore the neural signature of IOR in paradigms where retinotopic and spatiotopic representations of space are perfectly confounded. For instance, Fecteau and Munoz (2005) relied on extracellular recording techniques to monitor neuronal activation in the superior colliculus (SC) while monkeys performed a spatial cueing task. A significant reduction of neuronal activity was observed for cued, relative to uncued targets. Similarly, a recent fMRI study using a similar spatial cueing paradigm revealed a reduction of activation in early visual cortices (Anderson & Rees, 2011). Together with the EEG findings, the neuronal activation patterns converge on the idea that IOR is accompanied by reduced activation levels in retinotopically-organized brain regions. Although interesting, until now it has remained unclear whether the same neural signature would be observed for unconfounded spatiotopic IOR.

For IOR to function as a foraging facilitator in the real world, it is imperative that the inhibitory cueing effect exist in a spatiotopic reference frame (Klein, 1988). Here, a strong behavioral spatiotopic IOR effect was observed along with a much weaker retinotopic IOR effect, replicating previous observations (Maylor & Hockey, 1985). Although it might be suggested that the small retinotopic IOR score is due to a gradient of IOR centered on the spatiotopically cued location, the similarity of our retinotopic IOR

score to that of Maylor and Hockey (1985) strongly suggests otherwise[13]. It could similarly be suggested that the pattern of P1 cueing effects are the result of a gradient centered on the retinotopically cued location. However, these comparisons are not possible here since all retinotopic targets are presented in the lower visual field and all spatiotopic targets are presented in the upper visual field and it is unclear what effect this elevation confound will have on the target-elicited ERPs.

From the differences in ERP patterns observed when frame of reference is unconfounded (see Figure 8.3), it is clear that distinct neural signatures exist for retinotopic and spatiotopic IOR. Though only marginally significant for ipsilateral electrodes [$t(23) = 1.49$, $p = 0.075$; 1-tailed], P1 cueing effects that have been previously shown to accompany behavioral IOR were only observed in the retinotopic condition. These findings suggest that the target-elicited P1/N1 cueing effects that are commonly observed at long CTOAs might be more closely linked to a negative aftermath of retinotopically-organized attentional facilitation (Golomb, Chun, & Mazer, 2008; Posner & Cohen, 1984) than to spatiotopically-organized IOR (Klein & Hilchey, 2011; Taylor & Klein, 2000). On the other hand, robust posterior Nd cueing effects for both ipsilateral [$t(23) = 1.95$, $p < 0.05$; 1-tailed] and contralateral [$t(23) = 3.90$, $p < 0.001$; 1-tailed] electrodes were only observed in the spatiotopic condition, suggesting that the Nd component may be a neural signature of spatiotopic attentional maps. As we have already

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[13] RTs are longest at the spatiotopically cued location (378 ms) and decrease with increasing distance from this location, by 21 ms for targets 12° away and by a further 8 ms for targets diagonally opposite the cued location (17° away). However, a similar amount of retinotopic IOR was observed (Maylor & Hockey, 1985) while using locations that were further apart than those used in the present experiment, so it is still unclear whether the retinotopic IOR is the result of a separate neural mechanism from spatiotopic IOR or an inhibitory gradient. Given that no gradient in RT has been reported in previous IOR studies that dissociated retinotopic and spatiotopic frames of references, further empirical study is needed to clear up this issue.

noted, no previous ERP studies have dissociated spatiotopic and retinotopic frames of reference. Here, when coordinates were unconfounded, an Nd cueing effect was observed along with spatiotopic, but not retinotopic IOR. This finding suggests that P1/N1 reductions may reflect perceptual/attentional modulation of early retinotopic coding, while Nd reductions may reflect later spatiotopic (priority map) coding of IOR. Note that although the Nd cueing effect has been reported in previous ERP studies of IOR (McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009; Prime & Ward, 2004; Prime & Ward, 2006), the meaning and origin of this effect is unclear (Prime & Ward, 2006). Previous work using a go/no-go cueing paradigm (Prime & Ward, 2006) has demonstrated that the Nd effect was absent on trials when no response was required, suggesting that the Nd effect may originate from motor programming. Together with our behavioral observation of strong spatiotopic IOR, it seems likely that: 1) the Nd cueing effect is a neural signature of the spatiotopic representation of IOR, and 2) the spatiotopic/ retinotopic representation of the outside world is activated only when needed (Golomb et al., 2008).

Some authors have inferred a relationship between P1/N1 cueing effects and IOR, mainly because rarely does one effect materialize without the other. However, all previous studies have used spatial cueing paradigms where retinotopic and spatiotopic reference frames are confounded. It is worth reinforcing that the traditional conceptualization of IOR (Posner et al., 1985) unambiguously acknowledges that IOR must exist in an environment-based reference frame. Because there is no clear relationship between P1/N1 activation levels and classically-defined IOR, it is difficult to determine what bearing, if

any, the target-elicited P1/N1 cueing effects observed in previous studies have on IOR. We therefore strongly encourage follow-up work attempting to evaluate: 1) rudimentary relationships between ERPs and spatial cueing in separate reference frames, and 2) more challengingly, any relationship that might exist between retinotopic and spatiotopic representations of negative cueing effects (operationally-defined IOR).

8.6 Conclusion

Behaviorally, the current study has replicated the finding (Maylor & Hockey, 1985; Posner & Cohen, 1984) that when an EM is interposed between an uninformative peripheral cue and a target calling for a manual response, IOR associates more strongly with the spatiotopic than with the retinotopic location of the cue. Importantly, by recording EEG during this experiment, we have demonstrated that behavioral IOR at the spatiotopic location is not associated with a reduction in early ERP components. Furthermore, a significant reduction in the posterior Nd component, possibly related to spatiotopic priority output maps, is only observed at spatiotopically cued locations. These findings challenge common notions that target-elicited P1/N1 reductions in retinotopically-organized brain regions index IOR, and that the behavioral effect of IOR is related to input processing. Future work should examine whether the P1/N1 cueing effects observed in previous studies are related to a functionally distinct attentional/ perceptual network of IOR, possibly coming into play when EMs are forbidden (Taylor & Klein, 2000), or whether the P1/N1 reduction is unrelated to behavioral IOR and is simply a consequence of repeated stimulation along retinotopic pathways in the absence of attentional facilitation.

8.7 Acknowledgements

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8.8 References

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CHAPTER 9 DISCUSSION

In this thesis, I have used a relatively simple (parsimonious) and computationally explicit neural field model to provide a framework for implementing sensory and motor mechanisms of IOR. This simple implementation led to quantitative and qualitative predictions about behavioral IOR and the neural signals that might accompany it. These were tested and usually confirmed. Nevertheless, several findings from my studies and in the literature cannot be accommodated by the simple model. These "failures", or "limitations", of the model are as important as the successes because they point to the need for additional complexity (mechanisms). This final chapter will attempt to summarize and integrate the results presented in this dissertation, highlighting limitations of the current work and proposing ideas for future simulations and experiments.

9.1 Forms or mechanisms?

Seemingly in contrast to a 2-forms theory of IOR (Taylor & Klein, 2000), Chapters 2 and 3 presented simulation results (Satel, Wang, Trappenberg, & Klein, 2011; Wang, Satel, Trappenberg, & Klein, 2011) that illustrate the viability of a 2-mechanisms theory of IOR (Wang, Satel, & Klein, 2012a). Recall that the crucial difference between these theories lies in whether sensory and motor contributions to IOR are additive and coexist, or are mutually exclusive. Taylor and Klein's (2000) 2-forms theory of IOR postulates that IOR exhibits itself in *either* a motoric/output-based form, *or* a sensory/ input-based form, depending on the activation state of the oculomotor system. Wang et al.'s (2012a) 2-mechanisms theory of IOR proposes that - when the oculomotor system is activated - IOR results from both motoric and sensory mechanisms of IOR that can contribute to behavior simultaneously.

Chapter 2 explored a sensory mechanism of IOR that results in attenuated inputs - and behavioral IOR - when exogenous stimuli are repeatedly presented in the same spatial location. This sensory mechanism - which we also refer to as sensory adaptation, short-term depression (STD), or input attenuation - builds up quickly after a first stimulus onset and decays by about 600 ms post-cue in the superficial layers of the superior colliculus (sSC; Fecteau & Munoz, 2005). We propose that this sensory mechanism is a cause of behavioral IOR in conditions with repeated peripheral exogenous cues and targets, whether responses are made with saccades or button presses. Chapter 3 proposed a motor mechanism of IOR that arises after saccades have been executed, resulting in faster saccades when they continue in the same direction. This motor mechanism is the result of residual activity in the intermediate layers of the SC (iSC) after an eye movement, with maximal activity immediately after a saccade, lasting up to a few hundred milliseconds depending on the amplitude of the first saccade. We propose that this motor mechanism is a cause of behavioral IOR in conditions where saccadic responses are preceded by earlier saccadic responses, at short intervals.

Various simulation results were presented in Chapters 2-5 exploring the behavioral and neurophysiological consequences of sensory and motor mechanisms of IOR when implementing representative empirical paradigms. Chapters 2 and 3 presented simulations of the sensory and motor mechanisms, demonstrating the viability of such mechanisms as contributing factors to IOR, under certain conditions. Chapter 4 expanded on the saccade averaging simulations presented in Chapter 2 and presented two behavioral experiments looking at the influence of early facilitation (rather than IOR) on

landing sites. In Chapter 5, the 2-mechanisms theory of IOR was presented, proposing that the sensory and motor mechanisms of IOR would be additive under conditions where they are both generated (Wang et al., 2012a). If the sensory and motor mechanisms of IOR are independent, dissociable, contributing factors to IOR, then experimental conditions which recruit both mechanisms should show stronger IOR. This prediction was borne out in a series of behavioral experiments presented in Chapters 5 and 6, further demonstrating the robustness of a 2-mechanisms theory of IOR (Satel & Wang, 2012; Wang et al., 2012a). All the evidence presented here supports a 2-mechanisms theory of IOR when measured with saccadic responses.

However, mostly based on the evidence obtained in their parametric investigation of IOR, Taylor and Klein (2000) proposed that the critical factor determining whether IOR affects input or output processes is the activation state of the oculomotor system: When the reflexive oculomotor system is engaged, IOR only affects output processes - hence Taylor and Klein (2000) calling this the motoric form of IOR. When the oculomotor system is actively inhibited (Klein & Hilchey, 2011), IOR only affects input processes, generating attentional/perceptual IOR. It should be made clear that the 2-mechanisms theory of IOR proposes that both independent mechanisms contribute to IOR additively *only* when eye movements are made to *both* cues and targets *and* there is repeated peripheral stimulation. This theory does not directly speak to IOR with inhibited oculomotor systems (such as when measured with solely manual responses), although the simple principle is that the sensory mechanism will always be generated when there is repeated sensory stimulation and the motor mechanism will always be generated when

multiple eye movements are made. Thus, the critical difference between the 2-forms and 2-mechanisms theories is that 2-mechanisms theory (Wang et al., 2012a) proposes that there are both sensory and motoric contributions to IOR, even when the oculomotor system is activated, while 2-forms theory (Taylor & Klein, 2000) proposes there is no sensory contribution when the oculomotor system is engaged.

So what is the crucial difference underlying the interpretations arrived at when looking at the results of our series of behavioral experiments and those of Taylor and Klein (2000)? There are a number of differences in the experimental designs that must be considered. First of all, participants in the Taylor and Klein (2000) experiments were highly practiced since they returned on multiple days to participate in all conditions of the parametric investigation. While this approach allowed a within subject comparison of all conditions, it may have inadvertently had an effect on IOR scores, since practice is known to reduce or modulate IOR. As discussed extensively in Chapter 5, another major design difference across experiments is in the use of cue back stimuli by Taylor and Klein (2000), but not by us. However, when considering all the evidence, it seems unlikely that cue back stimuli alone could be responsible for the differences in experimental results. It is still unclear exactly how cognitive processes associated with cue back stimuli interact with mechanisms of IOR and so we encourage additional modeling and empirical work in this area. The only other major difference across experimental designs is in the adoption of blocked versus mixed experimental conditions.

9.2 Top-down attentional control settings (ACSs) and response mappings

Taylor and Klein (2000) always mixed exogenous (peripheral) and endogenous

(central) cues and targets, equalizing attentional control settings (ACSs) across experimental conditions. So, on any given trial, cues and targets could appear as peripheral exogenous onsets or central endogenous arrows. This procedure ensures that participants are utilizing the same task strategy - adopting the same ACSs - across the different cue and target stimulus conditions. If this were not the case, it would be possible that participants maintained broad attentional beams that encompass peripheral locations only during time periods when such stimuli can appear, and maintain much tighter attentional focus on fixation when only central stimuli can appear. Previous work has demonstrated that such strategies can modulate IOR (for demonstrations that centrally focused ACSs can reduce or eliminate IOR, see Wang & Klein, 2012; Hilchey, Klein, & Ivanoff, 2012), so it is clear that blocking conditions introduces a confound between conditions that can be avoided by mixing conditions.

Since our studies used a blocked experimental design while Taylor and Klein (2000) mixed trial types, we attempted to overcome this problem by using mixed cue types in Experiments 5.3 and 5.4, forcing a broad attentional beam and allowing the generation of IOR during the time of cue appearance in conditions when eye movements are forbidden to the cue. Additivity of the proposed sensory and motor mechanisms of IOR continued to be observed, suggesting that the additivity of these mechanisms is resilient to top-down ACSs. However, we only mixed cue types in these experiments, while Taylor and Klein (2000) mixed both cue and target types in each condition. Given that recent work in our lab has demonstrated a relationship between response mappings and IOR scores (Hilchey, Satel, Ivanoff, & Klein, in press), and differential accuracy of

endogenously versus exogenously directed saccades (Hilchey, Klein, & Satel, under review b), the degree to which such processes are contributing to our observation of multiple additive mechanisms remains unclear. Further work systematically investigating these possibilities under various experimental conditions are strongly encouraged to clear up these remaining issues. For now, it seems likely that there are multiple mechanisms of IOR, differentially recruited depending on the activation state of the oculomotor system, as well as other factors, though strong conclusions and a comprehensive framework must await the accumulation of further data.

9.3 Timecourse of IOR mechanisms

Since IOR has been observed without repeated peripheral stimulation, another point of contention is whether or not IOR generated by central (endogenous) versus peripheral (exogenous) cues results from the same underlying processes. Similarly, is IOR measured in response to peripheral targets the same as IOR measured in response to central targets? A growing body of research suggests that - when peripheral cues are ignored and IOR is measured with saccadic responses to targets at long cue target onset asynchronies (CTOAs; >~ 1 second) - IOR is equivalent, and likely the result of the same mechanism whether measured with central or peripheral targets (Hilchey et al., under review b; Hilchey, Ivanoff, & Klein, 2012a; Taylor & Klein, 2000). Note that we did not test such a hypothesis in the experiments presented here, since we were focused on testing conditions that would provide insight on the additivity of our proposed sensory and motor mechanisms. In fact, the 2-mechanisms theory, as presented thus far, cannot explain IOR in an ignored peripheral cue - central target paradigm at any CTOA (or, similarly, in an

ignored central cue - peripheral target condition). Under such conditions, the sensory mechanism would not be active since there is not repeated sensory stimulation at the same spatial location, and the motor mechanism would not be active since a saccade was not made to the cue. We thus propose that there are (at least) two additional neural mechanisms that contribute to behavioral IOR scores under various conditions.

9.3.1 Local inhibition in the iSC

In part to overcome the limitations of existing theories, Hilchey et al. (under review b) have proposed the existence of a local inhibitory input to the iSC following cue-elicited peripheral stimulation. Such a possibility was formerly considered impossible, mostly due to neurophysiological microstimulation evidence demonstrating that there was no such signal present in the iSC at 200 ms post-cue (Dorris, Klein, Everling, & Munoz, 2002). However, recent behavioral evidence suggests that such a signal may not arise until at least 500 ms post-cue (Hilchey et al., under review b). We would thus be very interested to see single cell recording studies that more fully investigate such a possibility at longer CTOAs when monkeys do show IOR.

We have performed a number of simulations (Satel, Story, Hilchey, & Klein, in preparation c) investigating the viability of such a possibility and now believe that such a third inhibitory mechanism exists, at least when the oculomotor system is engaged - a direct inhibitory signal arising sometime between 500 and 1000 ms post-cue. Such direct local inhibition in the iSC can accommodate IOR generated/measured in response to endogenous stimuli and the equivalence of such IOR to that generated/measured in response to exogenous stimuli at long CTOAs. Further studies are necessary in order to

fully delineate the dynamics of, and the conditions under which, such local inhibition could be present.

9.3.2 Cortical habituation

Though unlikely to occur very often in the real world, the vast majority of research investigating IOR has focused on the cue-target paradigm using a condition where the oculomotor system is actively inhibited throughout trials. This allows for a reductionist investigation of the dynamics of associated processes in as simple a task as possible. Since saccades cannot be made to targets in this paradigm, manual button presses (often with detection, localization, or discrimination tasks) are normally required in response to target stimuli. In this paradigm, the sensory mechanism of IOR should still be elicited when repeated peripheral stimuli are presented, though it is likely to have decayed by the time of target appearance at such long CTOAs. The motor mechanism of IOR is never elicited here, since eye movements are never made to the cues (or targets). As demonstrated in our Experiment 6.2, when measured with manual responses and an inhibited oculomotor system, IOR is equivalent whether generated by exogenous or endogenous cues. This pattern clearly cannot be explained by any combination of our sensory or motor mechanisms of IOR.

Furthermore, the existence of 2 forms of IOR depending on the activation state of the oculomotor system (Chica, Taylor, Lupianez, & Klein, 2010; Taylor & Klein, 2000) and the results of neuroimaging studies showing reductions of early sensory activity at long CTOAs (Prime & Ward, 2006) seem to preclude the same mechanism of direct collicular local inhibition being responsible for IOR at long CTOAs across oculomotor

activation states. That is, in all conditions when the oculomotor system is actively inhibited, an input-based form of IOR is found, but when engaged, an output-based form of IOR is normally observed. Of course, the proposed sensory mechanism of IOR could potentially have a long tail that produces a small amount of IOR for longer periods of time in humans, and both the sensory and motor mechanisms could have a longer timecourse in humans than in monkeys, but these possibilities seem unlikely. A more likely possibility is that a more general form of cortical habituation results in attenuated signals at longer time periods (Dukewich, 2009), though this mechanism may not have strong effects on behavior when the oculomotor system is engaged (e.g., Satel, Hilchey, Wang, Story, & Klein, 2013). Although there is still limited neuroscientific evidence for such a possibility, it is clear that some form of input-based inhibitory mechanism arises at long CTOAs when the oculomotor system is actively inhibited.

9.4 Neural foundations of IOR

A great deal of neuroscientific research has shown evidence for IOR as a sensory phenomenon. Monkey single cell recordings (Dorris et al., 2002; Fecteau & Munoz, 2005) have revealed cued target input attenuation relative to uncued targets, though such evidence is limited to relatively short CTOAs since monkeys do not show IOR at longer intervals. It is likely that such input attenuation reflects the 2-mechanism theory's sensory mechanism of IOR, since these studies used repeated peripheral stimulation at short intervals (relative to those normally tested with humans). Human functional magnetic resonance imaging (fMRI; Anderson & Rees, 2011) and electroencephalographic (EEG; for reviews, see Klein, 2004b; Prime & Ward, 2006; Satel et al., 2013) studies have

further shown reduced target activity for cued, relative to uncued, targets, when peripheral exogenous stimuli are used in cue-target paradigms with ignored cues and manual responses. Since the sensory mechanism of IOR is likely to have decayed by the time of target appearance in these studies, we believe the cue-induced, input-based, neural reductions observed in neuroimaging studies are related to longer term processes of cortical habituation, as discussed above.

9.4.1 ERP studies of IOR

Continuing to support an input-based account of IOR in these conditions (ignored cue - manual target), numerous event-related potential (ERP) studies have observed reductions in the amplitude of the early sensory P1 component for cued targets (McDonald, Ward, & Kiehl, 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004; Prime & Ward, 2006; Satel et al., 2013; Tian & Yao, 2008; van der Lubbe, Vogel, & Postma, 2005; Wascher & Tipper, 2004). These results support the idea that IOR acts on input processes by reducing the strength of repeatedly sensory inputs. However, P1 reductions have also been observed without behavioral IOR (Doallo, Lorenzo-Lopez, Vizoso, Holguin, Amenedo, Bara, & Cadaveira, 2004; Hopfinger & Mangun, 1998), and IOR has also been observed without P1 reductions (Hopfinger & Mangun, 2001; McDonald et al., 1999; Prime & Ward, 2006; van der Lubbe et al., 2005). Many of these studies have also observed later Nd reductions for cued targets (McDonald et al., 1999; Prime & Jolicoeur, 2009b; Prime & Ward, 2004, 2006; Satel, Wang, Hilchey, & Klein, 2012; Wascher & Tipper, 2004). Though the source and function of this component are less clear, its possible role in motor programming and later onset time, hint at a relationship to an

output-based form of IOR. The vast majority of these studies have used exogenous peripheral cues and targets, with participants ignoring cues and responding to targets with manual button presses, probably leading to actively inhibited oculomotor systems.

In Chapter 7, we demonstrated that P1 reductions are found for cued targets regardless of whether the cue is ignored or foveated (Satel et al., 2013). This is an unexpected result because, according to the 2-forms theory of IOR (Taylor & Klein, 2000), the motoric form of IOR should not affect input processes. However, cue-generated modulations of target-elicited inputs suggest an input based component of IOR in both conditions. Although this result seems to refute the 2-forms theory of IOR, it must be stated that P1 reductions were only correlated with behavioral IOR scores when the cues were ignored. It thus seems likely that P1 reductions are not a necessary and sufficient condition for the observation of IOR, at least when the oculomotor system is not actively inhibited.

Recent work in our lab using peripheral targets requiring manual responses and mixed central and peripheral cues has further demonstrated that, when cues are foveated, P1 reductions are observed only for peripherally cued targets - though IOR is observed in both conditions - (Satel, Reiss, Wang, Hilchey, & Klein, in preparation b). When cues are ignored, P1 reductions are observed for both cue types - though IOR is only observed for peripherally cued targets (Satel, Hilchey, Ivanoff, & Klein, in preparation a; for an analysis of the behavioral results of these experiments see Hilchey et al., in press). These results further support the idea that P1 reductions are not directly related to IOR, at least when the oculomotor system is activated or when endogenous stimuli are used.

9.4.2 Neural representation of IOR

In order for IOR to fulfill its proposed role as a novelty seeker (Posner, Rafal, Choate, & Vaughan, 1985) or foraging facilitator (Klein, 1988), IOR needs to operate on space in spatiotopic, or environmentally-based coordinates. In order to measure IOR in spatiotopic reference frames in a cue-target task, it is necessary to introduce an eye movement between cue and target onset. This approach has been used in a number of studies that have demonstrated the existence of behavioral IOR in spatiotopic coordinates (Hilchey, Klein, Satel, & Wang, 2012b; Maylor & Hockey, 1985; Pertzov, Zohary, & Avidan, 2010; Posner & Cohen, 1984; Satel et al., 2012). In Chapter 8, we further demonstrated that spatiotopically cued targets showed a large amount of behavioral IOR, but no P1 reductions (no sensory/input cueing effect). Spatiotopically cued targets did show a Nd modulations, potentially reflecting a motor/output cueing effect (Satel et al., 2012). Retinotopically cued targets showed a marginal P1 effect (small sensory/input cueing effect and very little IOR), and no Nd effect (no motor/output cueing effect). This result provides still further evidence that P1 reductions are neither necessary nor sufficient for the observation of IOR behaviorally, encouraging further work using cue-target designs optimized to investigate later ERP components.

9.5 Toward a comprehensive theoretical framework for IOR

We propose that there are (at least) 4 independent inhibitory mechanisms contributing to behavioral IOR in traditional cue-target paradigms in addition to a facilitatory mechanism that decays quickly after cue onset (see Figure 9.1 for a schematic illustration). Together, these 5 cueing mechanisms can accommodate the vast majority of

experimental results in the IOR literature and provide an early framework for a comprehensive theory of IOR.

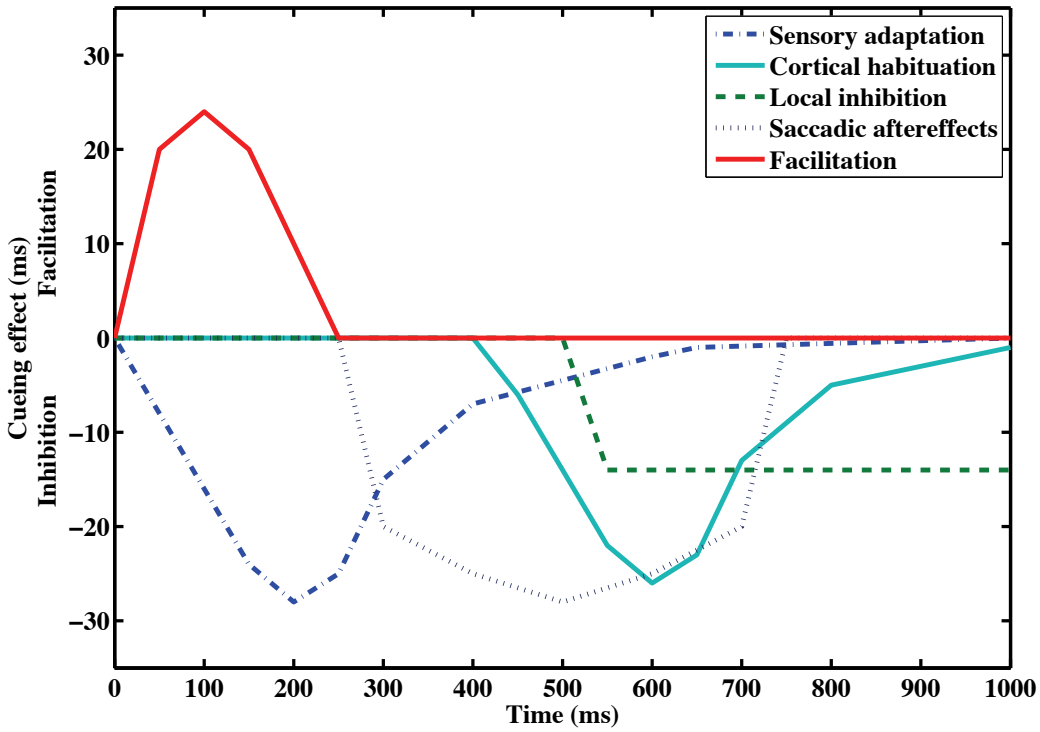

Figure 9.1: Schematic illustration of the time course of different cueing mechanisms. Time begins with the onset of the cue (at time = 0 ms). Facilitation occurs immediately after cue onset and summates with sensory adaptation when present. Cortical habituation is thought to occur sometime later and for now is assumed to have a similar time course to sensory adaptation. Local inhibition begins by 600 ms after the cue and represents 'true IOR', in terms of it's foraging facilitator function. Saccadic aftereffects could also serve such a function, though such a mechanism would only occur after eye movements and decay quickly (here, an eye movement is assumed to have occurred around 300 ms post-cue).

9.5.1 Sensory adaptation

The first inhibitory cueing mechanism generated after stimulus onset is sensory adaptation/input attenuation/STD, also referred to as the sensory mechanism of IOR and described extensively in Chapters 2, 4, and 5. The strongest evidence for the existence of

this mechanism is the monkey single-cell recording data (Dorris et al., 2002; Fecteau & Munoz, 2005), which clearly show a reduction in the target-elicited input signal activity when previously cued, at least up until about 600 ms post-cue. Further evidence for sensory adaptation has been provided by behavioral studies demonstrating that IOR increases in conjunction with the number of cue repetitions (Dukewich & Boehnke, 2008). However, it now seems unlikely that this mechanism is still active at the CTOAs normally investigated in human behavioral IOR studies. Nonetheless, we believe that this input-based inhibitory mechanism will be present - and summate with - other cueing mechanisms whenever there is repeated peripheral stimulation and CTOAs are less than about 600 ms.

9.5.2 Cortical habituation

If sensory adaptation is no longer present at CTOAs greater than 600 ms, then it cannot be a contributor to behavioral IOR measured at these intervals, and it is unlikely to be responsible for input-based reductions in neural activity observed in such experiments (e.g., Anderson & Rees, 2011; Prime & Ward, 2006). Furthermore, the most commonly investigated condition involves an inhibited oculomotor system with manual target responses - a condition which has been demonstrated repeatedly to be associated with input-based inhibitory mechanisms (Chica et al., 2010; Taylor & Klein, 2000). Thus, there must be another input-based mechanism of IOR that lasts somewhat longer than sensory adaptation. Further work is required to disentangle this mechanism from sensory adaptation and other output-based inhibitory cueing mechanisms, but for now we will assume that this mechanism represents a later mechanism of cortical habituation along the

input pathway - downstream from the retinotectal pathway thought to be involved in sensory adaptation.

9.5.3 Local inhibition

A number of studies have demonstrated that IOR can be measured even without repeated peripheral stimulation or eye movements to the cues (e.g., Hilchey et al., under review b; Hilchey, Satel, Ivanoff, & Klein, in press; Taylor & Klein, 2000). It has also been shown that at CTOAs longer than 1000 ms, IOR generated by a peripheral onset is equivalent whether measured with a central or peripheral target (Hilchey et al., under review b). Along with the consensus that there is an output-based mechanism of IOR when the oculomotor system is active from SAT studies, all this evidence points to the existence of a direct, local, inhibitory cueing mechanism that likely arises about 600 ms post-stimulus in the iSC. This output-based mechanism of IOR is likely generated in all conditions with eye movements, and fulfills the traditional functional hypothesis for IOR as a novelty seeker or foraging facilitator.

9.5.4 Saccadic aftereffects

Finally, as described in Chapters 3 and 4, a motoric inhibitory cueing mechanism will arise following any saccade. This mechanism of IOR is expected to last about 300 ms after a saccade, due to activity remaining in the iSC after a first saccade. The degree of inhibition and temporal dynamics of this effect are dependent upon the amplitude and direction of eye movements. This motor mechanism of IOR could also play a short-term role in novelty seeking behavior, perhaps in conjunction with the proposed mechanism of local inhibition.

9.6 Conclusion

In sum, we propose that there are multiple, independent, dissociable neural mechanisms contributing to the behavioral observation of IOR, with their differential levels of activation determined by experimental conditions - including stimulus and response properties, oculomotor activation state, ACSs, and stimulus-response mappings. Input attenuation is clearly a contributing factor under some conditions, whether it is short-term retinotectal adaptation, or longer-term cortical habituation. However, exactly how input based mechanisms of IOR affect behavior when the oculomotor system is activated is still unclear. The residual activity of saccades clearly affects the latency of subsequent saccades, but this process decays relatively quickly. For IOR to exist in spatiotopic coordinates and be present an equivalent form when using endogenous stimuli, there is likely a mechanism of long-term, local inhibition, but an input-based inhibitory mechanism seems to dominate when the oculomotor system is actively suppressed. Further work is strongly encouraged using mathematical modeling and interdisciplinary empirical techniques to generate a comprehensive framework for IOR and to further elucidate the boundary conditions under which different mechanisms contribute to IOR.

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