ON THE INHIBITORY CONSEQUENCES OF VISUOSPATIAL ORIENTING: INHIBITION OF RETURN?

by

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ABSTRACT

Responding is typically slowest toward (an "output" bias) or about (an "input" bias) targets presented at the location indicated by a spatially uninformative visual transient (a "cue") when the onset asynchrony between the cue and target exceeds ~300 ms. The expression of this "inhibitory" (Posner, & Cohen, 1984) aftereffect depends crucially on the response characteristics of the task. If the task at any time requires reflexively-generated saccadic responses, manual and saccadic responses are slowest toward the cued location. If the task expressly forbids reflexively-generated saccadic responses and instead requires exclusively manual responses, responding is slowest about targets presented at the cued location (Taylor, & Klein, 2000). By mapping the time course of the "inhibitory" aftereffects of cueing, exploring their reference frames and manipulating response properties of the target, we identify (at least) three independent "inhibitory" cueing effects (ICEs). One form of ICE is "inhibition of return" (IOR; Posner, Rafal, Choate, & Vaughan, 1985). IOR is generated by cue-elicited activation in pathways responsible for reflexively-generated saccades (the superior colliculus) when the task involves reflexive, saccadic responses to transient onsets. Another form of ICE is "sensory adaptation" (Fecteau, & Munoz, 2006). Sensory adaptation is short-lasting and occurs most robustly when the oculomotor system responsible for reflexive saccades is in an active state and when two visual transients appear in close spatiotemporal proximity (as when the cue and target occupy the same location in space and when their onset asynchrony < 500 ms). A final form of long-lasting ICE is a bias against inputs at a cued object location when the oculomotor circuitry responsible for reflexively-generated saccades is tonically inhibited. In most cases this effect is a likely side-effect of cue-elicited response activation in the manual effector system (de Jong et al., 1994) and may be re-mapped into object-based coordinates by posterior parietal networks unique to the manual effector system. Unlike IOR, with which it is frequently confused, this form of ICE is expressed as an effect nearer the input end of the processing continuum.

LIST OF ABBREVIATIONS AND SYMBOLS USED

AIC	Akaike information criterion
ANOVA	Analysis of variance
ACS	Attentional control setting
CTOA	Cue-target onset asynchrony
DVA	Degrees of visual angle
FA	False alarm
FLSD	Fisher's least significant differences
ICE	"Inhibitory" cueing effect
IOR	Inhibition of return
LIP	Lateral intraparietal (area)
NCE	Negative compatibility effect
PSP	Progressive supranuclear palsy
RMANOVA	Repeated measures analysis of variance
RT	Reaction time
S-R	Stimulus-response (compatibility)
S1	First event/stimulus
S2	Second event/stimulus
SAT	Speed-accuracy trade-off
SC	Superior colliculus
SRT	Saccadic reaction time
T-R	Target-response (compatibility)
TOJ	Temporal order judgment
TTOA	Target-target onset asynchrony

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(~2010-2013) flavored much of the content in this dissertation although, I'm sure, there is plenty with which you might disagree. Finally – and I'm not sure if I should give thanks or apologies – but thanks to Greg MacLean and RJ Redden for allowing me to bend their ears whenever I encountered a theoretical/empirical problem, which was frequent, or flash of insight, which was less frequent. Greg, it was a pleasure to collaborate with you on your Masters' work and RJ, it is an ongoing pleasure to be involved in your research. Your research findings and successes have influenced the trajectory of my research program and, by extension, this dissertation.

CHAPTER 1:
INTRODUCTION

This dissertation is comprised of manuscripts [published (Chapters 2-6) or submitted (Chapter 7)]. Overlap across chapters is inevitable in order to make each manuscript self-contained. I am the lead investigator on all of the experimental research presented herein. My contributions include but are not limited to designing and programming experiments, conducting the research, analyzing and interpreting data, and writing. Although I contributed most significantly to all research reported herein, I note gratefully that my co-authors provided invaluable intellectual insight and guidance, which is reflected by and acknowledged in each chapter, as appropriate.

An “inhibitory” aftereffect of visuospatial orienting is elicited by administering variations of the Posner cueing paradigm (Posner, 1980), synonymously referred to as the spatial cueing or cue-target paradigm, the simple model task, and the cost-benefit paradigm (see Klein, 2000; 2005, for review). In the canonical demonstration of the effect, Posner and Cohen (1984) displayed three identical boxes along an imaginary horizontal axis. Observers were instructed to fixate a middle box that was flanked by two equi-eccentric peripheral boxes (one to the right and left of fixation). The middle box contained a to-be-detected onset target on 60% of the trials. On 20% of the trials, one of the peripheral boxes contained the onset target. Observers were instructed to depress a single key as soon as any target was detected. On the remaining 20% of the trials, there was no onset target (catch trials) which served to discourage anticipatory responding. On every trial, a task-irrelevant, spatially uninformative, and to-be-ignored brightening of one of the peripheral boxes [a cue, 150 milliseconds (ms) in duration] preceded the target. The high probability of a response-relevant stimulus at fixation, the instruction to hold gaze at fixation coupled with eye movement monitoring, and the triviality of the transient cues in peripheral vision might lead the uninitiated reader to predict that the cue would have minimal impact on human performance.

Against this expectation, when the cue-target onset asynchrony (CTOA) was short (0 - 100 ms) responses were fastest for targets appearing at distance-matched cued relative to uncued locations. This facilitatory effect of the cue is historically ascribed to exogenous (involuntary), covert spatial attentional orienting (Posner, Snyder, & Davidson, 1980) and, in accord with the premotor theory of attention (Craighero &

Rizzolatti, 1997), may be mediated by the primitive midbrain machinery responsible for reflexively-generated saccadic responding (Hoffman, & Subramaniam, 1995; Smith, Rorden, & Jackson, 2004; Smith, Schenk, & Rorden, 2012; see Smith & Schenk, 2012, for review; but see MacLean, Hilchey, & Klein, 2015) but also by complementary parietal networks (e.g., Posner, Cohen & Rafal, 1982; Posner, Walker, Friedrich, & Rafal, 1984; Posner, Inhoff, Friedrich, & Cohen, 1986; see Bisley & Goldberg, 2010, for review). Serendipitously, when the interval between the cue and the target exceeded ~300 ms, responses were slowest when the cue and target occupied the same location. This ipsilateral inhibition (Berlucchi, Tassinari, Marzi, & Di Stefano, 1989), alternatively inhibitory aftereffect (Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987) or inhibitory tagging (Klein, 1988), was referred to as simply inhibition in the flagship paper (Posner & Cohen, 1984) but was later famously coined inhibition of return (IOR) following a series of experiments demonstrating close relationships between cue-elicited oculomotor responding/priming and subsequent biases against orienting/responding to the location of previously programmed saccadic responses (Posner, Rafal, Choate, & Vaughan, 1985; see Klein & Hilchey, 2011, for review). The phenomenon has attracted considerable interest in the scientific community because the mechanisms underlying the inhibitory aftermath are presumed to operate in the service of novelty-seeking (Posner & Cohen, 1984) and/or foraging facilitation (Klein, 1988; see Wang, & Klein, 2010, for review). Accordingly, contemporary thinking dictates that phylogenically primitive substrates of the oculomotor response system have evolved to

maximize sampling of the visual environment (e.g., Sapir, Soroker, Berger, & Henik, 1999; Gabay, Leibovich, Ben-Simon, Henik, & Segev, 2013).

Whereas the ecological significance of the inhibitory aftermath is reasonably well-established (but see, e.g., Smith, & Henderson, 2009), what causes (Chica, Klein, Rafal, & Hopfinger, 2010; Smith, Jackson, & Rorden, 2009) and, once caused, what processes are affected by the inhibitory aftermath (cf Reuter-Lorenz, Jha, & Rosenquist, 1996; Fuentes, Vivas, & Humphreys, 1999; Kingstone & Pratt, 1999; Taylor & Klein, 2000; Fecteau, Au, Armstrong, & Munoz, 2004; Hilchey, Ivanoff, & Klein, 2012; Satel, Hilchey, Wang, Reiss, & Klein, 2014) remain bones of contention. Part of the controversy stems from the theoretically-suggestive nature of the phrase inhibition of return (IOR). This point was made clear by Juan Lupianez (2010, p. 19) in a topical review: "The problem is that not much research has been dedicated to understanding the mechanism that produces the IOR effect, as the mechanism suggested by the name of the effect (i.e., the inhibition of the return of attention to previously attended locations) has simply been taken for granted." Even still, of the – reasonably large corpus of – research that has been dedicated to uncovering "the mechanism" of "the IOR effect", there is scant agreement among studies. But in agreement with Lupianez, most likely subscribe to the circuitous notion that IOR implies a reluctance of attention – in whatever form it may take – to return to something previously attended which, I speculate further, inspires many contemporaries to call any cost in a (putatively) attentional paradigm IOR (e.g., Chica et al., 2014). If the prevailing wisdom then is that IOR is tantamount to attention in some shape or form and that there is but one IOR effect ("the IOR effect"),

much research has indeed been dedicated to characterizing IOR's, and by extension, attention's properties as either sensory (e.g., Reuter-Lorenz, Jha, Rosenquist, 1996; Fecteau & Munoz, 2006), motoric (e.g., Possamai, 1992; Taylor & Klein, 1994) or some combination of the two (Abrams & Dobkin, 1994a; Fuentes, Vivas & Humphreys, 1999; Taylor & Klein, 2000), with little consensus among researchers (e.g., Dukewich & Klein, 2015). Although the strategy of characterizing the IOR effect as either sensory, motoric or sensory and motoric in varying degrees would, at least at first blush, appear sensible enough, this brand of reductionism has not lent itself to a unified theory of IOR. The reason why, perhaps, is that embedded in the logic is the lofty assumption that there is but one IOR effect in the spatial cueing paradigm (e.g., McAuliffe, Pratt & O'Donnell, 2001). This latter assumption has been violated myriad times by the literature on IOR effects, a literature that has elegantly demonstrated in a variety of ways that inhibitory effects in variations of the spatial cueing paradigm are completely dissociable (e.g., Bourgeois, Chica, Migliaccio, Thiebaut de Schotten, & Bartolomeo, 2012; 2013; Satel, Story, Hilchey, Wang, & Klein, 2013; Zhang & Zhang, 2011; Chica, Taylor, Lupiañez, & Klein, 2010; Pratt & Neggers, 2008; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Hunt & Kingstone, 2003; Kingstone & Pratt, 1999; Taylor & Klein, 2000; Tassinari & Berlucchi, 1993). A very palpable consequence of the belief that there is but one IOR effect – despite the reality of multiple forms of cue-elicited inhibition – is a deeply fractured community of researchers who study the IOR effect. The reality of the situation is that the "IOR" community studies a diverse set of dissociable phenomena – united semantically or, perhaps functionally/ecologically, by the phrase IOR – but in

which all members lay claim to the phrase IOR for their effect and its unique theoretical baggage. Even among those who dissociate inhibitory aftereffects in the spatial cueing paradigm, there remains a proclivity to classify both/all inhibitory aftereffects as IOR (e.g., Taylor & Klein, 2000). In light of the flagrant "abuses" (Berlucchi, 2006) of the phrase IOR, the following essay attempts to restore meaning to IOR by couching it in the clear theoretical framework that was assigned to it by Posner et al. (1985), supplying an abbreviated history of research and ideas leading up to the name, describing – when relevant – how contemporary research comes to bear on these early ideas and finally, considering what is to be done with the phrase IOR.

On the origins of inhibition of return

Although inhibition or the phrase IOR in the context of spatial cueing evokes Posner's simple model task and the signature biphasic pattern of cueing on response latency that it yields, the tendency for delayed responding toward visual inputs at previously stimulated locations was discovered independently in the laboratories of Michael Posner (Cohen, 1981; Posner & Cohen, 1980), while studying the modes of spatial orienting, and Giovanni Berlucchi (Berlucchi et al., 1981), while studying interhemispheric transfer of visual inputs (see Berlucchi, 2006, for more detail). The first and second published papers concerned with the form of inhibition in which Berlucchi and Posner had common interest (Berlucchi, 2006) appeared in 1980 and 1982, respectively. The first findings appeared in a technical report (Posner & Cohen, 1980) from the Lake Wilderness (Seattle, Washington) conference on attention. The second series of findings were reported by the Philosophical Transactions of the Royal Society:

Biological Sciences (Posner, Cohen, & Rafal, 1982). These two launching documents represent the groundwork for a series of influential reports by Michael Posner, Elizabeth Maylor, Robert Hockey, Jonathan Vaughan, Robert Rafal, Yoav Cohen and Lisa Choate published in the early 1980s which, in my opinion, culminated in Posner, Rafal, Choate and Vaughan (1985), the report in which the phrase IOR was first used and its parameters first defined. These early reports helped clearly delineate what was and what was not intended by "inhibition". Thus, the nature of the "inhibition", as interpreted by these seminal documents, will be reviewed in the next sections.

1) Inhibition is not caused by voluntary, visuospatial orienting.

Posner and Cohen (1980) laid the empirical and theoretical foundation for Posner and Cohen (1984). Indeed, data and discussion from the "double cueing", the "four alternative", the "arrow" and, to a much lesser extent, the "dimming" experiments that impelled Posner and Cohen (1984)'s sensory account of inhibition can be found in their 1980 technical report. In the "arrows" experiment, Posner and Cohen (1980; 1984) demonstrated that inhibition is not a consequence of voluntary, covert attentional orienting, and its subsequent withdrawal from the location at which a foveal arrow reliably (80% valid) predicted a target. Moreover, strict independence between voluntary, or otherwise endogenous, visuospatial orienting and the inhibitory aftermath following a transient peripheral cue was noted by Berlucchi et al. (1981) during the earliest stages of research on ipsilateral inhibition: "[t]his effect cannot be explained in probabilistic terms, since a control experiment where the subject knew the position of the second stimulus gave the same result." The independence between voluntary, covert

orienting and the inhibitory aftermath of a peripheral cue was verified by Posner, Cohen, Choate, Hockey, and Maylor (1984) following trial sequence analyses in a paradigm in which a foveal arrow reliably predicted target location. Whenever the target location from trial n-1 repeated in trial n, responding was slowest (see also, e.g., Hale, 1967; Kirby, 1972; Kirby, 1976; Soetens, Boer, & Hueting, 1985; Soetens, 1998 for 'alternation biases' at relatively prolonged response-stimulus intervals; see Maylor & Hockey, 1987; Dodd & Pratt, 2007, for consideration of sequential dependencies in IOR research), regardless of arrow cue validity. The unmitigated persistence of inhibition at the location of voluntary attention has now been established beyond reasonable doubt (e.g., Berlucchi, Chelazzi, & Tassinari, 2000; Berger, Henik, & Rafal, 2005; see also, e.g., Maylor, & Hockey, 1985; Possimai, 1986; Ivanoff, & Klein, 2001; Rafal, Davies, & Lauder, 2006; Mele, Berlucchi, & Peru, 2012, for evidence of IOR at fixated locations).

2) Inhibition is not caused by attentional momentum or guessing strategies.

Recognizing that in many two-alternative forced choice (e.g., Kirby, 1972) reaction time tasks performance is superior when the response from the prior trial alternates as compared to repeats – sometimes referred to as a negative recency effect, an alternation advantage or a subjective expectancy – Posner and Cohen (1980; 1984) reasoned that the "inhibition" may have been been caused by little else than a guessing strategy. That is, shortly after the cue the observer simply anticipates a probe at the mirror opposite location. A second possibility was that the movement of attention away from the task-irrelevant peripheral cue back toward fixation may have aroused a tendency for visuospatial orienting to continue in the direction from which it came, an

idea that was later revived as attentional momentum or the opposite facilitation effect in spatial cueing paradigms (Pratt, Spalek, & Bradshaw, 1999; Spalek & Hammad, 2004; 2005; Spalek, 2007) and saccadic momentum in oculomotor visual search tasks (Smith, & Henderson, 2009; 2011a; 2011b). To resolve whether the location opposite the cue is facilitated or, conversely, whether the cued location is inhibited, two orthogonal placeholder boxes were added to the spatial cueing paradigm, one above and one below fixation (i.e., the "four alternative" experiment). As such, the to-be-detected target could appear randomly at either a cued location (10%), a mirror opposite location (10%), or a 90-degree location (10% each; 60% of the targets appeared at fixation). Simply, detection performance was worst at the cued location and approximately equivalent at all peripheral, uncued locations (see also, e.g., Machado, & Rafal, 2004; Sumner, 2006). Although these results have been challenged (e.g., Pratt, Spalek, & Bradshaw, 1999), it appears now – at best – that momentum is confined to a limited range of conditions (Snyder, Schmidt, & Kingstone, 2001) and is demonstrably independent of the more robustly occurring inhibition (Snyder, Schmidt, & Kingstone, 2009; MacInnes, Klein, Hilchey, & Hunt, 2013; Snyder, & Schmidt, 2014). The contribution of guessing strategies to the findings have been ruled out by dual-tasks requiring complex mental rotation and target detection (e.g., Fecteau, Au, Armstrong, & Munoz, 2004).

3) Inhibition may be caused by repetitive, spatially overlapping sensory stimulation.

To examine the potential contribution of sensory factors to cue-elicited inhibition, Posner and Cohen (1980; 1984) administered the "double cueing" paradigm,

the results from which proved enormously influential to the earliest theoretical consideration of inhibition. In this paradigm, the cue appeared randomly in either the left, right or both fields of vision simultaneously. Targets appeared randomly in either left or right peripheral vision and required a simple manual detection response. Short (80 ms) and long (500 ms) CTOAs were administered to evaluate the role of stimulation on early covert, exogenous visuospatial orienting and late inhibition. Comparisons between single cued and uncued trials replicated previous findings (i.e., cue-elicited facilitation followed by inhibition). Of greater theoretical relevance, there was minimal evidence for facilitation on double-cued as compared to uncued trials at 80 ms CTOAs, a finding that was consistent with Posner's belief that visuospatial orienting could not be split between non-contiguous visual areas. By contrast, inhibition was approximately equivalent on single and double cued trials. Collectively, these data stoked the hypotheses that facilitatory visuospatial orienting was independent of inhibition (i.e., Posner & Cohen, 1984) and that inhibition was, more or less, a form of sensory "habituation caused by the cue" (Posner & Cohen, 1980, p. 255; see also, e.g., Dukewich, 2009). These claims were bolstered by Posner, Cohen and Rafal (1982) upon demonstrating that the magnitude of exogenous facilitatory visuospatial orienting was reduced when a peripheral target reliably occurred (80%) at the location of a peripheral cue as compared to a condition in which a peripheral cue reliably predicted (80%) the appearance of a target at the mirror opposite location at 500 ms CTOAs. And, at least early on (see Posner, 1982), Posner believed that a demanding mental task interfered with exogenous facilitatory visuospatial orienting but not sensory inhibition (but see Posner, Cohen, Choate, Hockey

& Maylor, 1984, for alternative interpretation). Despite the apparent success of the sensory account of inhibition, subsequent research (Maylor, 1985; Tassinari & Berlucchi, 1993) – also using variations on the double cueing paradigm – challenged the ubiquity of it. At CTOAs > 500 ms, Maylor (1985) demonstrated little to no inhibition at the locations of prior visual stimulation, unless each location was successively, not simultaneously, stimulated (see also e.g., Snyder & Kingstone, 2000; 2001). This early empirical discrepancy between Maylor (1985) and Posner and Cohen (1980; 1984) compelled an alternative to the prevailing stimulus-based account and in part the inference that "inhibition occurs as a direct consequence of externally controlled orienting rather than as the inevitable result of sensory stimulation in the periphery." (Maylor, 1985, p. 198). These two conflicting schools of thought were eventually reconciled by Tassinari and Berlucchi (1993; but see Danziger & Kingstone, 1999) with their two-stage account of inhibition. In effect, Tassinari and Berlucchi confirmed the Posner and Cohen finding that responding is delayed at each of the simultaneously cued locations at short CTOAs (<500 ms) and also the Maylor (1985) finding of no or a substantially diminished effect of double cueing on response latencies at late CTOAs. More recent investigative work at short and late CTOAs in paradigms in which cues appear simultaneously and randomly at several of eight locations around the circumference of an imaginary circle off fixation strongly suggest that inhibition is most robust at the Euclidean midpoint of simultaneous visual stimuli with only minimal contribution from the local elements (Klein, Christie, & Morris, 2005; Langley et al.,

2011; Christie, Hilchey & Klein, 2013), a result that generalizes to exogenous facilitatory visuospatial orienting (Christie, Hilchey & Klein, 2015).

4) Inhibition is not caused by sensory habituation or cue-elicited manual response suppression.

Data from the four inaugural experiments on "inhibition" were the impetus for Posner and Cohen (1980; 1984)'s influential parallel process model which captures the notion that visuospatial attentional orienting and sensory habituation engage distinct neural pathways which, together, conspire to influence performance at previously stimulated locations (see Posner, 1982, for a more detailed history on the origins of this line of thinking). In the years that followed, the results of several innovative experiments shifted the emphasis away from a sensory habituation account for inhibition. One variation on the model task whose data led to a departure from the 1980's sensory habituation account was a paradigm in which responses were required to the cue (i.e., target-target paradigms). Posner and Cohen (1984) in the "Back-and-forth" experiment (see also Cohen, 1981) required subjects to make saccadic responses to small digits appearing abruptly in either the left or right visual field rather than the more conventional method of simply instructing observers to ignore the spatially uninformative cue. Subjects were required to read the digits aloud, an outcome that could not be achieved unless the digits were fixated. Six hundred ms after digit onset the middle box brightened for 200 ms, summoning eye gaze back to fixation. To-be-detected targets appeared randomly at the location of the prior cue, its mirror opposite location or at fixation at 1200 or 2050 ms target-target onset asynchronies (TTOAs). The critical

result was that there was ample evidence for inhibition at the location of the previously generated saccadic response (see also, e.g., Vaughan, 1984; Rafal, Calabresi, Brennan & Sciolto, 1989) and, perhaps, more relevantly, the effect seemed "at least as strong when subjects move their eyes as when the eyes remain fixed." (Posner & Cohen, 1984, p. 547; see also, e.g., Taylor & Klein, 2000). Evidently then, behaviorally relevant visual stimuli (i.e., cues that are also targets) are perfectly capable of generating inhibition, a possibility that was not addressed by Posner and Cohen (1980) which perhaps, in part, biased early interpretation in favor of sensory habituation. An alternative account, however, posits that inhibition may have occurred because the peripheral cue, which shares certain luminance and/or spatial properties with the target, may have primed a manual response whose activation would need to be suppressed, long-term, to reduce the likelihood of task-irrelevant responding to the cue (e.g., Harvey, 1980; see also, e.g., Burle, van den Wildenberg, & Ridderinkohf, 2005; Coward, Poliakoff, O'Boyle & Lowe, 2004, for response-activation suppression accounts). In a paradigm similar to the "four alternative paradigm" except in which simple manual detection response were required to the first and second visual stimulus (the target), Maylor and Hockey (1985) demonstrated first that inhibition remained robust and second, as noted previously, could not be accounted for by attentional momentum or visual masking. The first conclusion in many respects parallels that of Posner, Cohen, Choate, Hockey and Maylor (1984) in their analyses of whether a response target on a prior trial is at the same or opposite location of a current trial. These analyses indicated that the magnitude of the inhibition was comparable regardless of whether a manual response was made to the first signal.

Despite the apparent similarity between the inhibitory effects in cue-target and target-target paradigms ~ 1985, later demonstrations revealed, as anticipated by Tassinari et al. (1987), that there are often significant differences between the magnitudes of the inhibitory effects – especially in detection and discrimination tasks but less so in localization tasks – depending not only on whether a response is made to the cue (e.g., Welsh & Pratt 2006, for scholarly review and demonstrations), but also the type of response that the cue and/or target require (e.g., Maylor, 1985; Possamai, 1992; Tanaka & Shimojo, 1996; Taylor & Klein, 2000; Lupianez, Milliken, Solano, Weaver & Tipper, 2001; Taylor & Donnelly, 2002; Welsh & Pratt, 2006; Chica, Taylor, Lupianez & Klein, 2010; Gabay, Chica, Charras, Funes & Henik, 2012), the physical properties of the cue and target (e.g., Riggio, Patteri & Umilta, 2004; Hu, Samuel & Chan, 2011; Hu, Fan, Samuel & He, 2013) and the perceived spatial relevance of the cue and target locations (e.g., Klein, 2000; Snyder & Kingstone, 2001; Hilchey, Klein & Ivanoff, 2012; Satel, Hilchey, Wang, Story & Klein, 2012). Simply, much more than anticipated during the earliest research, the task-specific response requirements dramatically modulate the magnitudes and time courses of the observed inhibitory aftereffects (though see, e.g., Washcer, Schneider & Hoffman, 2015). The extent to which opposing facilitatory and inhibitory processes (and how many) are contributing to the net behavioral effects remains largely unknown and requires – and is receiving – much needed study. Note simply that behavioral inhibition is typically robust in target-target paradigms at later CTOAs – though its form may be altered (e.g., Chica et al., 2009) – unless manual discrimination responses are required to the cue and target on shared physical properties.

In this latter case, inhibition may be obscured by stimulus-response repetition heuristics (e.g., Taylor & Donnelly, 2002).

The inhibition – or at least the form of it leading up to the phrase IOR ~ 1984 – was not conceptualized as habituation of sensory or motoric activation induced by a behaviorally-irrelevant cue. As noted, however, an argument can be made that Posner and Cohen (1980) conceptualized the inhibition as sensory habituation. Nevertheless, the zeitgeist in 1984 and early 1985 was clearly toward a sensory account. Posner and Cohen (1984) noted that the inhibition "depends primarily or perhaps exclusively upon the sensory information" (p. 541) while Maylor (1985), though more adversarial, noted that "the inhibitory effect is characterized by an inability to respond as quickly (both manually and ocularly) to a target appearing in a recently stimulated location (either by a cue or by another target) as to one appearing in a different location" (p. 786). Clearly at this time, inhibition was related specifically to repeated stimulation along an input pathway, and its purpose was to bias processing against a prior sensory source; its origins were unrelated to habituation, granted little mention was made of what neuropsychological mechanism(s) might underly the effect.

Despite no clear mechanism for the inhibition, several additional results at this time were interpreted as evidence against a sensory habituation account which, at the time, weighed heavily on the collective minds of the research. For example, in the final Posner and Cohen (1984) "Right-angle experiment", in Vaughan (1984) – in an experiment demonstrating that inhibition from a saccadic eye movement to a peripheral visual stimulus radiates at least 1.5 degrees of visual angle out from the stimulus

location (see also Maylor & Hockey, 1985; see Wang, Hilchey, Cao & Wang, 2014, for more recent work on gradients of inhibition) – and in Maylor and Hockey (1985; see also Kruger & Hunt, 2013; Satel, Wang, Hilchey & Klein, 2012), an eye movement intervened between the first visual signal (cue or target) and eventual response probe (target) to dissociate the retinal from spatiotopic coordinates of the inhibitory aftermath of the cue. In simpler terms, an effort was made to determine whether cue-elicited inhibition – unlike cue-elicited facilitation (e.g., Posner & Cohen, 1984; Golomb, Chun & Mazer, 2008) – would persist at the environmental location of a cue despite an eye movement between cue and target that effectively displaced the retinal coordinate of the cue away from its absolute, environmental reference frame. At the time (e.g., Wurtz, Richmond, & Judge, 1980; Goldberg, & Wurtz, 1972; Rizzolatti et al., 1974; Cynader, & Berman, 1972) and to this day (e.g., Fecteau & Munoz, 2005; 2006; Boehnke et al., 2011), it was well-known that retinotopically-organized visual neurons residing in the superior colliculus – a midbrain saccade system – expressed short-term, attenuated responsivity upon repeat sensory stimulation. In effect, repeat sensory stimulation delays responses to targets appearing at previously stimulated locations (Satel, Wang, Trappenberg & Klein, 2011) and even by the mid- to late- 1980s, some researchers were still seriously considering the viability of this account at early CTOAs (e.g., Tassinari, Aglioti, Chelazzi, Marzi & Berlucchi, 1987; Berlucchi, Tassinari, Marzi & Di Stefano, 1989). Because, however, the inhibition was coded in an environmental, and perhaps to a much lesser extent retinotopic reference frame (e.g., Maylor & Hockey, 1985), and was by comparison long-lasting, sensory habituation was ruled out, at least at the lowest

level of sensory processing. Although the dynamic remapping of inhibition was crucial for dismissing sensory habituation, collectively the robust findings of exogenous visuospatial orienting from a peripheral cue at short cue-target onset asynchronies (but see Tassinari, Aglioti, Chelazzi, Peru & Berlucchi, 1994; Lupianez & Weaver, 1998), inhibitory gradients from peripheral cues (Vaughan, 1984; Maylor & Hockey, 1985), the prolonged time course of the inhibition (> 1.3 s; e.g., Maylor & Hockey; Posner & Cohen, 1984), the persistence of inhibition at foveated locations (Maylor & Hockey, 1985), the results from target-target paradigms (Posner et al., 1984; Posner & Cohen, 1984; Maylor & Hockey, 1985) and spatiotopic inhibition were considered while ruling out sensory habituation (but see, e.g., Dukewich, 2009).

After 1984, what is inhibition of return?

Unequivocally, the research at this time tended strongly toward a sensory cause and effect that operated independently of covert visuospatial orienting or, alternatively stated, the effect was "an inhibitory one that serves to reduce the efficiency of target detection" (Posner & Cohen, 1984, p 549). However, these initially strong sensory assertions about inhibition were upset and ultimately rebuffed by two research reports, one by Maylor (1985) and another by Posner, Rafal, Choate and Vaughan (1985), that fundamentally altered the theoretical course of inhibition in the spatial cueing paradigm. In effect, the conceptual and empirical chasm between the inhibitory accounts < 1985 and those in 1985 inspired decades of research in which a principal objective has centered on determining the contribution of sensorimotor processes to the inhibitory aftermath that follows from variations on the model task.

The phrase inhibition of return was introduced by Posner, Rafal, Choate and Vaughan (1985) in a series of experiments dedicated to uncovering the relationship between inhibition and the eye movement system in the simple model task. Prior to 1985, no study had succeeded in demonstrating any form of inhibition in the spatial cueing paradigm without spatially overlapping visual stimuli. Gradients of inhibition had been amply demonstrated by displacing the target off of the cued location at various eccentricities but all such data converged on the idea that the inhibitory aftermath was maximal when the cue and target occupied precisely the same location. No study at this point had detected any evidence of inhibition in the absence of spatially proximal visual signals. These apparent sensory constraints on the inhibition posed a formidable challenge to Posner and Cohen (1984)'s hypothesis that the mechanism(s) underlying the inhibitory effect operated effectively and generally to bias orienting against previously processed visual signals. If the inhibition were in effect only when multiple, abruptly occurring visual signals traversed the same input pathway, how could the mechanisms underlying the inhibition operate effectively in natural visual search during which repetitive, location-specific sensory stimulation is the exception rather than the rule? Such rigid sensory constraints lent themselves readily to the word (sensory) inhibition but certainly not generally to the presumed ecological significance of the mechanisms underlying it, mechanisms that putatively act to inhibit the return of orienting. In an even broader context, the phrase IOR and indeed its presumed ecological significance fundamentally belied the parallel process model advanced by Posner and Cohen (1984) for sensory inhibition. If sensory inhibition truly operated independent of visuospatial

orienting, the mechanisms underlying it would be of little ecological significance for orienting responses.

At the time of the Posner, Rafal, Choate and Vaughan (1985) report, several new findings – advanced principally by Elizabeth Maylor (1985) – were considered alongside prior findings. These (re-) considerations led to a dramatic re-conceptualization of the mechanical and theoretical properties of inhibition in the spatial cueing paradigm. By this time, Maylor (1985; Experiment 2) had already provided indirect evidence that the inhibition, contrary to Posner and Cohen (1980; 1984), was caused by visuospatial orienting and/or the oculomotor response system (Maylor, 1985, Experiment 1). In Maylor (1985)'s demonstration of a causal relationship between visuospatial orienting and subsequent inhibition, when spatial discontinuity was introduced to a secondary smooth-pursuit eye movement task at fixation at the time of cue presentation by abruptly changing the dimension (horizontal or vertical) on which the smooth pursuit occurred, there was neither evidence for visuospatial attentional facilitation nor inhibition. The "double cueing" experiment that motivated the sensory inhibition accounts in Posner and Cohen (1980; 1984) was also challenged by Maylor (1985; Experiment 3), as noted above. Recall that in this experiment, unlike in Posner and Cohen (1980; 1984), there was minimal evidence for inhibition when a target appeared at one of two previously stimulated locations; as such, it appeared as if inhibition, like externally elicited visuospatial orienting, could not be split between non-contiguous visual regions. Vaughan (1984) had already demonstrated that a saccade to a visual stimulus and back to fixation would delay subsequent saccades to the previously inspected location. Posner

and Cohen (1984) further demonstrated that saccades to visual stimuli followed by a return to fixation delayed manual detection responses for signals at the prior saccade landing site. Moreover, in the presence of intervening eye movements between the cue and target, the inhibition was robustly observed in a spatiotopic, not retinotopic reference frame (Posner & Cohen, 1984; Vaughan, 1984; Maylor & Hockey, 1985). Finally, Maylor (1985; Experiment 4) demonstrated that peripheral cueing at CTOAs > 500 ms had little to no effect on biasing perceptual judgment in a temporal order judgment task (i.e., in which the observed must determine which of two asynchronous sensory events appeared first); the foregoing result was particularly crucial for falsifying the reigning sensory accounts.

Encouraged by Maylor (1985)'s data, the ensuing belief that the inhibition could be "regarded as a consequence of externally controlled covert orienting to the cue" (p. 199), the implications of this belief for the ecological significance of the inhibition, and the circumstantial evidence hinting at a special role for eye movement systems in the effect, Posner, Rafal, Choate and Vaughan (1985) set out to explore the relationship between the oculomotor response system and inhibition, hereupon referred to as IOR.

1) Inhibition of return is closely related to the oculomotor response system.

First, Posner et al. (1985) tested a patient group with progressive supranuclear palsy (PSP), a degenerative neurological disease that initially affects the ability to make saccadic (especially vertical) eye movements, in addition to several clinical control groups comprising patients with Parkinson's diseases, frontal and parietal lesions, and a healthy control group (see also Posner, Cohen & Rafal, 1982). The most relevant result

upon administering the spatial cueing paradigm was that PSP patients failed to show inhibition for vertically presented stimuli whereas the inhibition remained intact for other patient and non-patient control groups. The importance of an intact SC for the generation of the inhibition, which was inferred from these findings, was later confirmed by Sapir, Soroker, Berger, and Henik (1999) and Sereno, Briand, Amador, and Szapiel (2006).

2) Inhibition of return is a response, not sensory, bias against a cued location.

Second, replicating Maylor (1985), Posner et al. (1985) administered a variation on the spatial cueing paradigm in which the effect was generated by a to-be-ignored and task-irrelevant peripheral onset stimulus (cue), but measured atypically by way of temporal order judgment to temporally asynchronous “targets" at both the cued and uncued locations at 1600 and 2100 ms CTOAs. Again, there was no evidence that the peripheral cue biased perceptual judgment against the cued location at either critical late CTOA for inhibition. However, in the same paradigm, when subjects were required to make a saccadic eye movement to whichever location felt “most comfortable”, there was a significant, albeit slight, bias against responding to the cued location.

3) Inhibition of return is caused by oculomotor responding.

Third, in a final critical experiment similar to the aforementioned “back-and-forth” experiment (Posner & Cohen, 1984), Posner et al. sought to determine whether spatially repetitive sensory stimulation was required to generate inhibition. In this paradigm, two equi-luminant digits – instead of one – appeared simultaneously left and right of a central fixation stimulus. An arrow at fixation commanded a saccadic eye

movement to the to-be-read digit upon which gaze was reoriented to fixation by the illumination of the central fixation stimulus. The interval between the onset of the saccade arrow and the to-be-detected luminance target, appearing at the location of the prior saccade (cued) or opposite to it (uncued), was randomly either 2600 or 3450 ms. Despite equivalent sensory stimulation at cued and uncued locations, responding was slowest at the location of the previous saccadic eye movement (see also, e.g., Posner et al., 1985; Rafal et al., 1989; Machado, & Rafal, 2004; Chica, Klein, Rafal, & Hopfinger, 2010; Satel, Hilchey, Wang, Reiss, & Klein, 2014).

Inhibition of return

Collectively, the experiments demonstrated a form of inhibition that does not depend on repeat sensory stimulation, does not degrade perceptual judgment but that rather depends on oculomotor response activation and biases saccadic and manual responses, or more generally orienting, against the location of the previous oculomotor orienting response. Implicit to this framework is a rejection of the Posner and Cohen (1984) parallel process model which treated sensory inhibition and visuospatial orienting separately. In precisely the way Posner et al. (1985) described, the mechanisms underlying the inhibition – which relate very closely to oculomotor response activation – could operate by "inhibit(ing) orienting towards visual locations which have been previously attended (inhibition of return)" (Posner et al., 1985, p. 211). Relieved from the constraints of sensory inhibition, a form of long-term inhibition (> 500 ms) caused by oculomotor response activation that biases all effector systems, or orienting generally, against previously targeted saccade locations could operate efficiently – and likely does

(e.g., MacInnes, Hunt, Hilchey, & Klein, 2014) – to discourage responding against already-charted space, the inhibition of return orienting responses.

Regarding the presumed ecological significance of IOR, there is no doubt mechanisms/transformations intercede between the initial oculomotor response activation and the eventual response bias to remap the effect dynamically into environmental coordinates (van Koningsbruggen, Gabay, Sapir, Henik & Rafal, 2010; Sapir, Hayes, Henik, & Danziger, 2004). Despite this, so long as the effect originates as an oculomotor response program and terminates as a general response bias against the location of the previously programmed eye movement, the effect may still warrant the label IOR. Finally, although "inhibition" likely occurs at various levels of the central nervous system (e.g., Dukewich, 2009) and although there are undoubtedly forms of inhibition that affect sensory information uptake at some level of processing (e.g., Posner & Cohen, 1984; Tassinari & Berlucchi, 1993; Reuter-Lorenz, Jha & Rosenquist, 1996; Lupianez et al., 1997; Taylor & Klein, 2000; Fecteau, Au, Armstrong & Munoz, 2004; Hunt & Kingstone, 2003; Fecteau & Munoz, 2005; 2006; Ivanoff & Klein, 2006; Prime & Ward, 2006; Muller & Kleinschmidt, 2007; Smith, Ball & Ellison, 2012; Satel et al., 2013; Chapter 1), sensory inhibition is not IOR (Posner et al., 1985), though proxies of it may be found in Posner and Cohen (1980; 1984). In veneration of Giovanni Berlucchi, who from very early on navigated the unsteady waters of IOR with aplomb (e.g., Tassinari & Berlucchi, 1995; Berlucchi, 2006), I recall the Shakespearian dictum that "a rose by any other name would smell just as sweet" and agree strongly that "by any other (more appropriate name), IOR-like effects would be equally or more

fascinating to study without the passive acceptance of dubious theoretical constraints" (Berlucchi, 2006; p. 1073). Indeed, the onus ought to lie on today's growing legion of researchers to demonstrate whether the many forms of "IOR" are indeed IOR. In the spirit of Berlucchi (2006), the following dissertation aims chiefly to dissociate IOR from the motley array of "inhibitory" cueing effects in variations of the model task.

CHAPTER 2:
RETURNING TO "INHIBITION OF RETURN" BY DISSOCIATING LONG-TERM OCULOMOTOR IOR FROM SHORT-TERM SENSORY ADAPTATION AND OTHER NONOCULOMOTOR "INHIBITORY" CUEING EFFECTS

The manuscript based on this study is presented below. Co-authors for this manuscript are Dr. Raymond M. Klein and Dr. Jason Satel, respectively.

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Abstract

We explored the nature and time course of effects generated by spatially uninformative peripheral cues by measuring these effects with localization responses to peripheral onsets or central arrow targets. In Experiment 1, participants made saccadic eye movements to equiprobable peripheral and central targets. At short cue-target onset asynchronies (CTOAs), responses to cued peripheral stimuli suffered from slowed responding attributable to *sensory adaptation* while responses to central targets were transiently facilitated, presumably due to cue-elicited *oculomotor activation*. At the longest CTOA, saccadic responses to central and peripheral targets were indistinguishably delayed, suggesting a common, output/decision effect (*inhibition of return*; IOR). In Experiment 2, we tested the hypothesis that the generation of this output effect is dependent on the activation state of the oculomotor system by forbidding eye movements and requiring keypress responses to frequent peripheral targets, while probing oculomotor behavior with saccades to infrequent central arrow targets. As predicted, saccades to central arrow targets showed neither the early facilitation nor later inhibitory effects that were robust in Experiment 1. At the long CTOA, manual responses to cued peripheral targets showed the typical delayed responses usually attributed to IOR. We recommend that this late *inhibitory cueing effect* (ICE) be distinguished from IOR because it lacks the cause (oculomotor activation) and effect (response bias) attributed to IOR when it was named by Posner, Rafal, Choate, and Vaughan (1985, Inhibition of return: neural basis and function. *Cognitive Neuropsychologia*, Vol. 2, pp. 211-228).

Introduction

The effect of increased response times (RTs) to previously stimulated (cued) locations relative to unstimulated (uncued) locations in vision was discovered independently in the laboratories of Giovanni Berlucchi (Berlucchi, Di Stefano, Marzi, Morelli, & Tassinari, 1981) and Michael Posner (Cohen, 1981; Posner, Cohen, & Rafal, 1982). This effect is most commonly studied using variations of the *spatial cueing paradigm* (Posner, & Cohen, 1984). In the model task, when the interval between a spatially uninformative cue in peripheral vision and a visual target calling for a simple detection response was short (0 - 100 ms), responses were faster for targets appearing at cued, relative to uncued, locations. When the interval exceeded 300 ms, responses were slower when a target occurred at cued, relative to uncued, locations. In the interest of remaining theoretically neutral, and because Posner and Cohen simply referred to this as "inhibition", we will refer to this finding as an “inhibitory” cueing effect (ICE; *cf* Hilchey, Satel, Ivanoff, & Klein, 2013).

Two different mechanisms were implied in the seminal studies

In a series of experiments, Posner and Cohen (1984) concluded that this ICE: 1) was *not* generated by voluntary attention, 2) existed at two locations simultaneously when both peripheral boxes were brightened simultaneously[1], and 3) mapped onto spatiotopic coordinates.

[1] A finding that was not replicated by Maylor (1985) and that has been called into question by the finding that ICEs often appear along the average vector or near the mid-point (or “center of gravity”) of simultaneously presented visual inputs (Klein, Christie, & Morris, 2005; Langley, Gayzur, Saville, Morlock, & Bagne, 2011; Christie, Hilchey, & Klein, 2013).

The initial series of findings led Posner and Cohen (1984) to conclude that the "effect is an inhibitory one that serves to reduce the efficiency of target detection (p. 549)" and that this inhibitory effect is caused by sensory stimulation, an input-based cause and an input-based effect. Despite this, because the ICE was long-lasting and, following an eye movement, was represented in an environmental rather than retinotopic reference frame, Posner and Cohen also reasoned that this ICE plays a "role in directing future covert and overt (p. 550)" orienting and that it "evolved to maximize sampling of the visual environment (p. 550)". These attributions are represented in the left-most column of Table 2.1.

Posner and colleagues (Posner, Rafal, Choate, & Vaughan, 1985) later demonstrated that a degenerative neurological condition (progressive nuclear palsy) impairing normal oculomotor function abolished ICEs. The importance of intact midbrain structures responsible for saccadic eye movements, specifically the superior colliculus (SC), was later confirmed by Sapir, Soroker, Berger, and Henik (1999) and Sereno, Briand, Amador, and Szapiel (2006a).

Having thus demonstrated a close relationship between this ICE and the eye movement system, Posner et al., (1985) proceeded to explore its cause and effect. They measured its effect (Experiment 2) by way of temporally asynchronous "targets" at both the cued and uncued locations. When observers were simply asked, at the end of the trial, which target had been presented first (a standard temporal order judgment; TOJ) there was no effect of cueing on perceptual arrival times [a finding confirmed by Maylor (1985) and others, see Klein, Schmidt & Müller, 1998]. When, in response to the same

sequence of events, observers were instructed to make an eye movement in whichever direction (to whichever peripheral target) felt most comfortable there was a significant bias for the eyes to move away from the previously stimulated location. Contrary to the "input effect" inference drawn by Posner and Cohen (1984) this pattern implies that the effect generated by the cue was output-related – a response bias. In their study of cause (Experiment 3) the observer fixated a central box while two equi-luminant digits appeared to the left and right of fixation. After one second elapsed, a left- or right-ward arrow appeared at fixation commanding the observer to make an eye movement to the left or right digit, respectively. When the observer's eye arrived at the digit, the observer reported the identity of the digit aloud. Directing the observer to return his/her gaze to fixation the central box was subsequently flashed briefly. The critical result was that simple detection RTs were slowed for targets presented at the previous saccade-target location relative to the opposite location, despite equivalent prior stimulation at both locations. This finding implies an effect that is caused, not by asymmetric stimulation, as had been inferred by Posner and Cohen (1984; see also Posner et al., 1982; Posner, Cohen, Choate, Hockey, & Maylor, 1984), but rather by prior activation of the oculomotor system.

Posner et al. (1985) named the ICE they had explored *inhibition of return* (IOR). The *theoretical* definition of IOR, which is illustrated in the right-most column of Table 2.1, encapsulates the inductions that: 1) *In cause,* IOR occurs in the aftermath of oculomotor activation (see also, e.g., Godijn & Theeuwes, 2002), and 2) *In effect*, IOR is a long-lasting response bias that affects overt and covert orienting. We believe that

although the ICE that was explored by Posner and Cohen (1984) might share the novelty-seeking function that Posner et al. (1985) attributed to IOR, if the two phenomena truly have different causes and effects on subsequent processing, then they should have different names (see Berlucchi, 2006, for similar advice and a scholarly discussion of this issue). As noted earlier, we will use the non-theoretically loaded term ICE when referring to the pattern of increased RTs to previously stimulated (cued), relative to unstimulated (uncued), locations, unless we are confident that what is observed is an example of IOR as defined theoretically by Posner et al. (1985).

More recent behavioral evidence for two types of late ICEs

In the years since IOR was first labelled, it has become reasonably well-established, theoretically and empirically, that there are (at least) two late ICEs with different causes and effects. One of the factors that determines the expression (or lack thereof) of an ICE is the extent to which the task activates the oculomotor system responsible for reflexively-generated saccades (Chica, Taylor, Lupianez, & Klein, 2010; Hunt & Kingstone, 2003; Kingstone & Pratt, 1999; Pratt & Neggers, 2008; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Taylor & Klein, 2000; Zhang & Zhang, 2011).

The clearest, and for our present purposes, most pertinent demonstration of the distinction between ICEs on the basis of the activation state of the oculomotor system was made by Taylor and Klein (2000). In their parametric investigation of ICEs, either a spatially uninformative, foveally presented left- or right- ward pointing arrow, or a transient luminance change in left or right peripheral vision randomly appeared. Remaining faithful to their nomenclature, we refer to this stimulus as the first signal

(S1). After S1, a brightening at fixation (cue-back stimulus) re-oriented attention to the center of the display. One second after the onset of S1, a second visual signal (S2) was presented. S2, like S1, was a randomly selected centrally presented arrow or peripheral stimulus. Participants were exposed to 6 different conditions (tested on different days) that were generated by the orthogonal combination of 3 responses that could be made to S1 (ignore, manual localization, or saccadic localization) and 2 responses that could be made to S2 (manual or saccadic localization).

The results from these conditions fall into two dramatically different patterns:

Output/decision-based ICEs (IOR)

If, at any point, the task required an oculomotor response (conditions 3, 4, 5 and 6, as listed above), ICEs were *observed*/*measured* in the form of slower responding to cued (relative to uncued) central *and* peripheral S2s. Importantly, the magnitudes of the ICEs were statistically indistinguishable whether *measured* by centrally- or peripherally-presented stimuli (see Figure 2.1). This last point is especially critical as it implies an effect that is wholly motoric, since – all else being equal – an input-based contribution would generate larger effects with peripheral than with central targets (see Hilchey, Klein, & Ivanoff, 2012). This pattern reinforces Posner et al.'s (1985) theoretical framework for IOR by demonstrating that cues in peripheral vision are not necessary to cause it (oculomotor responding is sufficient), and once it is caused the effect of IOR is a response bias that is unlikely to degrade the perception of visual signals at one second cue-target onset asynchronies (CTOAs). These findings are thus consistent with Posner et al.'s conceptualization of IOR and ought to be referred to by that name.

Input-based ICEs

In stark contrast to IOR/output-based ICEs, conditions in Taylor and Klein (2000) that never required an oculomotor response (conditions 1 and 2) showed no evidence of a motoric effect: only responses to peripheral S2s were delayed, implying an ICE purely along the input pathway. Because eye movements were prohibited throughout a trial in these two non-oculomotor conditions (no response-manual and manual-manual), we assume that they engaged suppression of the oculomotor system responsible for reflexive saccadic eye movements (Klein & Hilchey, 2011). The implications of the findings, in light of this assumption, are that: 1) when the reflexive oculomotor system is *suppressed*, ICEs are not caused by cue-elicited oculomotor activation, and 2) the nature of the effects on processing are closer to the *input* end of the processing continuum (*cf* Satel, Hilchey, Wang, Story, & Klein, 2013).

Neurophysiological evidence for ICEs and cue-elicited oculomotor response activation

Neurophysiological studies (Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005) have administered one cell (peripheral S1 and peripheral S2 of condition 4, no response-saccade paradigm) from Taylor and Klein's (2000) investigation to rhesus monkeys while recording extracellularly from visual and visuomotor neurons residing in the superficial and intermediate layers of the SC, respectively. Target-elicited activation of both visual and visuomotor neurons was reduced when the neuron was repeatedly stimulated by sensory input at short CTOAs (< 500 ms). Activity in visuomotor neurons correlated significantly with behavior (Fecteau & Munoz, 2005). The data from these studies demonstrated that two visual stimuli presented in close spatiotemporal proximity

(i.e., CTOAs < 500 ms) have an input-based or low-level visual processing effect (i.e., *sensory adaptation*) that translates into delayed oculomotor responding (i.e., an early input-based ICE).

Dorris et al. (2002) further demonstrated that there is residual *oculomotor activation* in visuomotor neurons following a peripheral cue. When a brief train of microstimulation – sufficient to evoke an eye movement – was delivered to the intermediate layer of the SC shortly (200 ms) after a spatially-irrelevant peripheral cue, the latency of the electrically-evoked saccades was *faster* to previously stimulated, relative to unstimulated locations. Several conclusions follow from this pattern of results. Although the reduced responsivity following repeated stimulation (a neurophysiological ICE) could have been due to local inhibition of the originally stimulated region or sensory adaptation in the pathways leading to the SC, local inhibition was ruled out by the microstimulation results (Dorris et al., 2002).

These results, when considered collectively, suggest that the reduced target-elicited activity in visual and visuomotor neurons is, at least in part, a by-product of repeated visual stimulation (as noted by Fecteau & Munoz, 2005; 2006) and that, at least 200 ms post-cue, there is a response bias *toward*, not *against*, the cued location. It is findings like these – suggesting sensory adaptation as a mechanism of IOR (*cf* Boehnke, Berg, Marino, Baldi, Itti, & Munoz, 2011) – that have given impetus to two-component theories for oculomotor IOR (e.g., Sereno, Jeter, Pariyadath, & Briand, 2006b), multi-component theories for IOR (e.g., Dukewich, 2009) and the identification of one component of oculomotor IOR in computational models as a sensory adaptation process

that is still present at long CTOAs in humans (Satel, Wang, Trappenberg, & Klein, 2011; Wang, Satel, & Klein, 2012).

Oculomotor IOR is unlikely to be mediated by sensory adaptation

These neurophysiological data – which seem to identify "IOR" as a sensory effect – are in conflict with the above-mentioned behavioral data showing no evidence of a sensory component to IOR in humans (*cf*; Chica et al., 2010; Hilchey, Hashish, MacLean, Ivanoff, Satel, & Klein, in press; Hilchey et al., 2012; Klein & Hilchey, 2011; Satel et al., 2013; Taylor & Klein, 2000). At one end of the spectrum, Taylor and Klein asserted that what we are calling IOR comprises no input-based component whatsoever. At the opposite end of the spectrum, Fecteau and Munoz (2006) have asserted that IOR is primarily input-based.

The neurophysiological data, however, suggest that the input-based, sensory adaptation effect does not apply when the cue-target interval is as long as 1 second. That is, the association between "IOR" and low level visual processing in the SC is never found at long CTOAs (> 500 ms). Even though Dorris et al. (2002) tested for IOR at CTOAs of 200 and 1100 ms, they failed to observe any evidence of behavioral IOR at the longer interval. Fecteau and Munoz (2005) did observe a significant IOR effect at CTOAs of 500 and 1200 ms. Importantly, however, the visual neurons were no longer showing significant target-elicited reductions in neural firing at these longer CTOAs. We believe that the findings from the neurophysiological investigations have been overgeneralized: The sensory adaptation effect which had been identified as IOR are

actually short-lasting and not representative of the IOR effect as conceptualized and demonstrated by Posner et al. (1985).

The experiments

We conducted human behavioral experiments using a version of the spatial cueing paradigm (Posner, 1980) to resolve the apparent discrepancy between late oculomotor IOR, as described by Taylor and Klein (2000; see also Klein & Hilchey, 2011), and the early input-based sensory adaptation effects seen in monkey neurophysiological studies. Our method was to determine the time course of cueing effects generated by peripheral cues when these effects are measured by RTs to peripheral onset and central arrow targets. Ensuring that the oculomotor system would be in an active state (as in the monkey neurophysiological studies described above and in conditions 3-6 from Taylor & Klein, 2000), in Experiment 1, observers made saccadic responses to both types of target. In Experiment 2, we encouraged the active suppression of the oculomotor system responsible for reflexively-generated saccades by requiring only manual responses to peripheral targets while making these targets considerably more likely than the central arrow targets which, as in Experiment 1, required saccadic responses. In both experiments, to ensure the minimal contribution of attentional capture by the cue, a cue-back to fixation was presented immediately after the peripheral cue (100 ms before the earliest possible target). Based on studies that compared an early cue-back with no cue-back we were confident that its use would minimize the contribution of facilitatory processes at the cued location (Briand, Larrison, & Sereno, 2000; MacPherson, Klein & Moore, 2003).

Experiment 1

In our first experiment, ICEs will be generated by spatially non-predictive peripheral cues and measured by randomly intermixed and equiprobable peripheral onset and central arrow targets calling for localization responses. In contrast to most previous studies using these conditions, a range of CTOAs will be used to measure the time course of any facilitatory and inhibitory effects on saccadic RT initiated by the cue. The neurophysiological evidence on input-based cueing effects very clearly predicts that – *so long as the interval between the cue and target is relatively short (< ~ 500 ms) and so long as any facilitation at short cue-target intervals does not obscure the effect* – saccadic reaction times (SRTs) will be slowed to targets presented at previously cued locations.

By contrast, when the effect is measured by saccades in the direction of a central arrow stimulus (and thus there is no repeated sensory stimulation) and when the interval between the cue and target is short, the neurophysiological data predict *faster* saccadic eye movements in the direction of the previous cue. This prediction is based on the finding of residual neuronal activation following cue-onset and parallels the finding (Dorris et al., 2002) that saccades evoked by direct electrical stimulation are faster at the cued location. In both cases, the signal causing the eye movement bypasses the input pathway traversed by the cue.

At late cue-target intervals (> ~ 600 ms) and consistent with previous observations (Hilchey et al., 2012; Taylor & Klein, 2000) the inhibitory effect of the cue

will be similar when measured in response to central arrows or peripheral onsets (i.e., it will be output-based).

Methods

Subjects

Twenty undergraduate students (18 females and 2 males; 16 right-handed and 4 left-handed) from Dalhousie University participated in a 60-75 minute session for course credit or monetary compensation ($10-15 CDN). All observers were naive to the hypotheses of the experiment and reported normal or corrected-to-normal vision.

Apparatus and procedure

All observers were tested in a dimly-lit room and all stimuli were presented against a black background. Stimuli were presented on a 19" ViewSonic Optiquest Q95 CRT monitor (Q95-3) connected to an Intel Core Duo processor, at a viewing distance of 22.5". An Eyelink II head-mounted eye tracking system was used to monitor the eye movements of the observers. The Eyelink headset was connected to a host computer, operating on a Pentium Intel 4 processor, which projected online gaze coordinates to a second monitor that was concealed by a black curtain during experimentation. All observers were run through a 9-point calibration and validation procedure to ensure that the precision of the eye tracking was within half a degree of visual angle. The host computer provided accurate information about gaze position every 4 ms. The velocity threshold to detect a saccadic eye movement was set to 35°/s and the SRTs were computed by subtracting the time at which the eye movement exceeded the velocity

threshold from the time at which the imperative stimulus appeared onscreen. The accuracy of target-directed saccades was based on the landing position of the first saccade to exceed this threshold relative to the center of the target.

See Figure 2.2 for the sequence of events in each trial. At the beginning of every trial, three white-outlined placeholder boxes (each measuring 1.6° x 1.6° visual angle), separated center to center by 7.9°, were displayed along the horizontal meridian. When the observer's subjective experience was that of having successfully fixated the center of the central box, they were instructed to press the spacebar to initiate the events of the trial. The observer was instructed that if the trial didn't start (this is what would happen if the drift correction phase was not completed successfully) they should try again to fixate appropriately and press the spacebar again.

Following a successful drift correction, there was a 500 ms fixation interval, after which a to-be-ignored, uninformative, peripheral cue appeared onscreen for 50 ms. The cue, an asterisk with a diameter of 0.3° visual angle, appeared left or right of fixation. Observers were explicitly informed that this cue was "irrelevant", "meaningless" and "uninformative" to help ensure that the triviality of the cue was better appreciated. Immediately after the peripheral cue was extinguished, a cue-back appeared at fixation for 50 ms. The cue-back was a middle-box brightening where the middle placeholder outline tripled in size and served to summon any attention attracted by the peripheral onset back to fixation. The observers, unlike in many other investigations (e.g., Pratt & Fischer, 2002), were explicitly instructed that the cue-back served as signal to indicate that a response would be required to the next visual event. Following the cue-back, a

randomly selected 50, 150, 250, 350, or 950 ms interval elapsed. After this interval, a leftward or rightward pointing arrow (1° visual angle in width) target was presented in the central box or a peripheral target disk (0.3° visual angle in diameter) was presented in the cued or uncued peripheral box. Thus, the CTOAs were 150, 250, 350, 450 and 1050 ms. In the event of a central arrow, the observers were instructed to make a fast and accurate saccadic eye movement to the middle of the box to which the arrow was pointing. In the event of a peripheral onset – occurring in the middle of the placeholder box – observers were required to make an accurate and fast saccadic eye movement to the onset. Immediately following the initial saccadic eye movement or, alternatively, if a response was not made to the target within 2 seconds of its onset, all stimuli were "removed" from the display (*i.e.*, the display screen was black) and the next trial began one second later (*i.e.*, the fixation display re-appeared).

A minimum of 160 experimental trials were administered in each of four blocks of trials. To ensure that substantial saccadic eye movements were not made to the cue or at any time during the trial other than after presentation of the imperative signal, the trial abruptly terminated if the observer's gaze position deviated by more than 3° visual angle from the fixation stimulus and was followed by immediate visual feedback. The feedback indicated that an eye movement had been detected and that eye movements were strictly forbidden until the target was presented. Trials with such failures to maintain fixation were reshuffled into the list of trials.

Design

The design was completely within-subject, with the following factors randomly intermixed within each block of trials: CTOA (with 5 levels: 150, 250, 350, 450 and 1050 ms), Target Type (with two levels: peripheral and central) and Cueing (cued and uncued, referring to whether the target's location or direction, for peripheral and central targets, respectively, was the same as or different from the location of the cue). In each block of trials there were 8 trials for each of the 20 cells in this design.

Results

Trials were initially excluded due to a failure to maintain fixation prior to target presentation (3.1%), anticipatory saccades with a latency less than 100 ms (0.5%), slow saccades with a latency greater than 1 second (0.6%), and a failure of the saccade to land within 3 degrees visual angle of the target (3.5%). On the remaining trials, we discovered that saccadic eye movements to central arrow targets were less precise[2] than those to peripheral targets [$F(1, 19) = 30.54$, $p < 0.01$]. Because there is a gradient of oculomotor IOR around a cued location (Dorris, Taylor, Klein, & Munoz, 1999; Hooge & Frens, 2000; Vaughan, 1984), the further the landing position of a saccade is from the cued location, the less inhibition one ought to observe in SRT. To minimize this confound, we also excluded any trial for which the landing position of saccades did not fall within 1° visual angle of the target location. After this final exclusion, there were 7184 "accurate" saccadic responses (on average over 350 per participant).

[2] Scatterplots (available by request from the first author) of the landing positions reveal that saccades to the two types of target were slightly hypometric (ranging from 2% to 7%) and that increased variable error was the primary cause of the lower accuracy of saccades made to arrow stimuli, though in the 1050 ms CTOA conditions, there was a tendency for slightly greater undershooting with saccades to central arrows.

SRTs for each level of each factor can be found in Table 2.2. The SRTs for accurate saccades were submitted to a 5 (CTOA: 150, 250, 350, 450 and 1050 ms) x 2 (Cueing: cued or uncued) x 2 (Target Type: central arrow or peripheral onset) repeated measures ANOVA. There was a main effect of Cueing [$F(1, 19) = 9.92$, $p < 0.01$], where SRTs were, overall, slower to the cued location as compared to the uncued location. There was a main effect of Target Type [$F(1, 19) = 21.43$, $p < 0.01$], where central arrow targets showed longer SRTs as compared to peripheral onset targets. There was a main effect of CTOA [$F(4, 76) = 35.99$, $p < 0.01$], where SRTs were slowest at the shortest CTOA and roughly equivalent at all CTOAs beyond 150 ms. The interaction between Cueing and Target Type was significant [$F(1, 19) = 37.06$, $p < 0.01$], indicating that the difference between cued and uncued SRTs was greater for peripheral targets as compared to central targets. The interaction between Target Type and CTOA was also significant [$F(4, 76) = 4.86$, $p < 0.01$]. This interaction comes about because SRTs to peripheral targets benefited more from the increase in CTOA beyond 150 ms than did SRTs to central targets. Critically, there was a three-way interaction among factors CTOA, Target Type and Cueing [$F(4, 76) = 3.57$, $p < 0.05$]. This three way interaction can be visualized most easily by representing Cueing as a difference score (uncued target SRT minus cued target SRT) and by showing how this difference score for the two target types is is affected by CTOA (see Figure 2.3).

To aide in interpretation of Figure 2.3, we note the following: 1) Saccadic responses to cued peripheral onset targets are invariably slowed relative to when they are uncued, 2) there is a substantial facilitatory cueing effect for the central arrows at the

short CTOA (~ 20 ms) that rapidly decays to non-significance before the later appearance of an ICE, and 3) the difference between the ICEs for central arrows (15 ms) and peripheral onsets (22 ms) at the longest CTOA is not significant (neither using the FLSDs, as before, nor using a pairwise t-test [$t(19) = 1.51$, $p > 0.10$]). Specifically, the present pattern of results can be described as follows: when measured with a peripheral target, there is evidence of an "inhibitory" effect of the cue at every CTOA, but when measured by a central arrow there is a "facilitatory" cueing effect at the early CTOA that transitions to an ICE by the longest CTOA where it is statistically indistinguishable from the ICE measured by a peripheral target.

Discussion

The present design has yielded several useful findings for arbitrating between a sensory adaptation or input-based account of IOR and the oculomotor or output-based account of IOR. First, when the interval between an uninformative, to-be-ignored, peripheral onset cue and an imperative saccade target was short (< 450 ms), saccadic responses to peripheral onset targets suffered from prolonged ICEs while saccadic responses to central arrow targets showed relatively short-lived facilitatory cueing effects. Together, these findings suggest that *when the target occurs at the same location as the cue*, it is likely that sensory adaptation and residual cue-elicited oculomotor facilitation are occurring in parallel, with the former effect larger than the latter so that the net effect (with peripheral targets) is inhibitory. Second, consistent with data from previous work, saccadic responses at the longest CTOA show statistically indistinguishable ICEs whether measured by a central arrow or peripheral onset. As

argued previously, this equivalence implies an effect generated by the cue that is, at this point in time, completely located in the output pathway, consistent with Posner et al.'s (1985) theoretical attributions for IOR.

It is possible to capture the nature of these effects with three mechanisms. Two of these mechanisms, *sensory adaptation* and *oculomotor facilitation,* are firmly rooted in monkey neurophysiology and, as noted, were relied on to predict the observed pattern of results at the short CTOAs. A third mechanism, *IOR* (in the sense proposed by Posner et al., 1985)*,* is invoked to explain the data at the longest CTOA.

Experiment 2

As noted in the Introduction and as illustrated by Figure 2.1, the behavioral expression of late ICEs changes dramatically depending on whether or not the task entails oculomotor responding. Simply, in the presence of oculomotor responding, late ICEs are expressed in behavior as output-based (i.e., IOR). By contrast, if oculomotor responding is forbidden, late ICEs are expressed as input-based. Whereas Taylor and Klein (2000) assumed that which of these two forms was generated depended on the general activation state of the oculomotor system, this idea was refined by Klein and Hilchey (2011) to help explain, among other findings, the results when anti-saccades are used to generate or measure the inhibitory aftereffects of orienting (Fecteau, Au, Armstrong, & Munoz, 2004; Rafal, Egly, & Rhodes, 1994). According to Klein and Hilchey, it is the activation state of the oculomotor system specifically responsible for reflexive saccades that determines which form of late ICE will be generated. From a logical perspective, the reflexive oculomotor system ought to be tonically suppressed in

tasks for which reflexive (prosaccadic) oculomotor responding is forbidden to peripheral stimuli. Such suppression (of visuomotor neurons residing in the intermediate layers of the SC) has been demonstrated in neurophysiological investigations when the task is such that oculomotor responding is prohibited to peripheral onset events (e.g., Everling, Dorris, Klein, & Munoz, 1999; Ignaschenkova, Dicke, Haarmeier, & Their, 2004).

In Experiment 2, this proposal is evaluated by measuring the activation state of the oculomotor system using eye movement responses to central arrows in a situation in which only manual, and not oculomotor, responses are required to peripheral targets. We hypothesized that to the extent that the reflexive oculomotor system would be suppressed under these conditions, we would observe an ICE with manual responses to peripheral targets at a long CTOA in the absence of oculomotor facilitation at the early CTOA and in the absence of IOR at the long CTOA. In other words, the ICE generated in the context of reflexive oculomotor suppression would be input-based.

To encourage suppression of the reflexive oculomotor system and test these predictions, we made three major methodological changes to Experiment 1. First, and critically, keypress localization responses were required to peripheral targets, while saccadic eye movements to *all* peripheral events (cues and targets) were strictly prohibited. If, at any point in time (prior to a central arrow target, calling for a saccadic response), an eye movement was detected during the task, the trial abruptly ended and the observer received visual feedback warning them not to shift their eyes from fixation. Second, the probability of arrow targets, still requiring saccadic responses, was reduced to 20%, primarily to ensure (much like Klein, 1980; Klein & Pontefract, 1994) that we

were only occasionally probing the state of activation of the oculomotor system, and that the participant would be highly prepared for the primary task (to make manual responses to peripheral events). Third, to allow for the reduced probability of central arrow targets, while generating sufficient data from such trials, we removed the intermediate (250, 350 and 450 ms) CTOAs, choosing to focus on the most analytic shortest (150 ms) and longest (1050 ms) CTOAs.

Method

Subjects

Twenty undergraduate students (15 females and 5 males; 19 right-handed and 1 left-handed) from Dalhousie University participated in a 60-75 minute session for course credit or monetary compensation ($10-15 CDN). All observers were naive to the hypotheses of the experiment, reported normal or corrected-to-normal vision, and had not participated in Experiment 1.

Apparatus, procedure, and design

Experiment 2 was identical to Experiment 1 with the following exceptions: 1) The 250, 350 and 450 ms CTOAs were removed, 2) the ratio of peripheral to central arrow targets was 4:1 and the observers were explicitly told about this, and 3) keypress, not oculomotor, responses were required to peripheral S2s (targets). If, at any point in time, an eye movement was detected when not permitted, the trial abruptly terminated and the observer was given immediate visual feedback: “Eye movement detected. Do not move your eyes unless instructed to by a central arrow”. Trials were recycled if eye

movements were made before the target but not otherwise. There were a minimum of 160 trials in each of 5 blocks of experimental trials.

The design was completely within-subject with the factors CTOA (150, 1050), Target/Response Type (peripheral/manual, central/saccade), and Cueing (cued, uncued). Because of the probability asymmetry, 32 trials in a block entailed central targets while 128 trials entailed peripheral targets.

Results

Two participants were excluded because they did not complete the experiment. Trials were initially excluded from the RT analyses due to a failure to maintain fixation prior to target presentation (15.1%) and responses by the incorrect effector system (3.8% eye movements to peripheral targets). A further 1.4% were excluded because of misses, anticipations, slow responses, or manual response errors. As in Experiment 1, and on trials requiring an oculomotor response to central arrow targets, all trials on which the eye movement did not land within 1° visual angle were excluded as “inaccurate”. After these exclusions, there were 1366 “accurate” saccades available for analysis of SRTs and 10749 "correct" trials available for analysis of manual RTs.

RTs/SRTs for each level of factors Cueing (cued or uncued), CTOA (150 or 1050), and Target/Response Type (peripheral/manual, central/saccade) can be found in Table 2.3. All correct RTs/SRTs were submitted to a 2 (Cueing) x 2 (Target Type) x 2 (CTOA) repeated measures ANOVA. There were significant main effects of Cueing [$F(1, 17) = 8.26$, $p < 0.05$] and CTOA [$F(1, 17) = 150.53$, $p < 0.01$] and a significant interaction between Target/Response Type and CTOA [$F(1, 17) = 57.73$, $p <$

0.01]. This interaction comes about because, as in Experiment 1, RTs to peripheral targets benefited more from the increase in CTOA than did SRTs to central targets (102 ms versus 43 ms, respectively). Critically, the data were qualified by a significant three-way interaction [$F(1, 17) = 4.89$, $p < 0.05$].

The three-way interaction can be visualized most easily by representing Cueing as a difference score (uncued target RT/SRT - cued target RT/SRT) and by plotting the Cueing effects as a function of CTOA and Target/Response Type (see Figure 2.4). As can be seen in Figure 2.4, cueing effects are statistically non-discriminable from 0 with one notable exception: peripheral targets at 1050 ms CTOAs. This was confirmed by planned comparisons (t-tests) against 0 which reveal that neither the peripherally-measured Cueing effect at the 150 ms CTOA [$t(17) = 0.68$, $p > 0.10$], nor the centrally-measured Cueing effect at the 150 ms CTOA [$t(17) = 1.45$, $p > 0.10$], nor the arrow measured Cueing effect at the 1050 ms CTOA [$t(17) = 0.76$, $p > 0.10$] were significant. The peripherally-measured 20 ms Cueing effect at the 1050 ms CTOA was significant [$t(17) = 4.00$, $p < 0.01$].

Discussion

As predicted, the pattern of results were markedly different from what was observed in Experiment 1[3] when, with the same stimulus presentation procedures, eye movements were required to peripheral onset S2s. First and foremost, we obtained an ICE at the long CTOA when it was measured with manual responses to peripheral

[3] This is clear visually from Figures 2.3 and 2.4. We confirmed it statistically by subjecting the data from the common SOAs in Experiments 1 and 2 to a between-experiment ANOVA in which the 4-way interaction (Experiment x SOA x Cueing x Target Type) was significant [$F(1,36) = 14.81$, $p < 0.01$].

targets. Second, as measured in response to the central arrow targets, the cue elicited neither the early oculomotor facilitation nor the later ICE. The early absence of oculomotor facilitation in response to central arrow targets is consistent with our goal of ensuring that the oculomotor system responsible for reflexive saccadic eye movements was in a suppressed state in this experiment. That the ICE measured with manual responses to peripheral targets was not accompanied by a significant ICE in the saccadic responses to central arrow targets supports the proposal (Klein & Hilchey, 2011) that peripheral cues presented in the context of reflexive oculomotor system suppression generated an ICE that was input-based. The entire logic of the experiment strongly implies that this ICE is not caused by oculomotor activation. And the aforementioned absence of early oculomotor facilitation converges with this conclusion.

Somewhat surprisingly, there was no evidence of delayed keypress responding to peripheral targets at the short CTOA. This result, which contrasts with what was found with saccadic responses to the same targets in Experiment 1, might be explained by assuming that sensory adaptation has a minimal effect along the pathways leading to keypress responses (see, e.g., Wurtz & Mohler, 1974, for evidence that visual neurons at the level of the superficial layers of the SC might be more closely related to oculomotor than to keypress responding). Alternatively, perhaps sensory adaptation was present but obscured by some other effect, unique to manual responses, such as cue-elicited automatic manual response activation. Research exploring this possibility is underway in our laboratory.

The entire logic of the experiment strongly implies that the ICE we have observed here is not caused by oculomotor activation. And the aforementioned absence of early oculomotor facilitation converges with this conclusion. Nevertheless, some readers may question whether some aspect of the dual task (rather than active suppression of midbrain structures responsible for reflexive saccades, as we have proposed) may have abolished sensory adaptation, oculomotor facilitation and IOR. Against this possibility, and in line with our thinking here, note that Taylor and Klein (2000)'s saccade-manual and manual-saccade conditions were dual tasks and yet, in both cases, there was clear evidence for output-based ICEs (IOR). On the other hand, there are single task studies with manual responses which suggest that ICEs might be reduced, if not eliminated, for low probability responses (Ivanoff & Klein, 2004; Lupianez, Ruz, Funes, & Milliken, 2007). And, arguing against the possibility that these studies compromise our thinking, in a dual-task study, Klein and Pontefract (1994, Experiment 2) found that low probability, endogenously generated saccades were sensitive to local inhibitory mechanisms associated with endogenous covert spatial orienting. Research in which the relative probabilities of central and peripheral targets – and the responses assigned to them – were manipulated, might resolve this question. An alternative approach might derive from converging evidence (some of which is described below) in support of the mechanisms we have postulated to explain our findings.

General discussion

In two experiments, a spatially uninformative peripheral cue was followed by either a peripheral onset or central arrow target at variable CTOAs. In Experiment 1, in

which both target types called for a saccadic localization responses, the immediate effects of a peripheral cue were *oculomotor facilitation* (as inferred from faster saccades in response to arrow targets calling for responses toward the initial cue) and *sensory adaptation* (as inferred from slower saccades in response to peripheral targets that repeated the input pathway stimulated by the cue). The sensory adaptation effect was initially large enough to obscure the effect of oculomotor facilitation when the target was an onset in the periphery and it outlasted oculomotor facilitation. One second after the onset of the cue, all oculomotor responding in the direction of the cue was slowed. Because this inhibitory effect was statistically indistinguishable whether the target repeated (peripheral) or did not repeat (central) the location of the cue, we infer an output effect: *oculomotor IOR* or, simply, *IOR*.

In Experiment 2, we prohibited reflexively-generated saccadic eye movements to all visual signals appearing in peripheral vision (requiring, instead, manual responses to peripheral targets, while reducing the probability of arrow targets which called for an endogenously generated directional saccade). In this experiment, all traces of oculomotor facilitation (at the short CTOA) and oculomotor IOR (at the long CTOA) were abolished, as was sensory adaptation at the earliest CTOA. That saccades in response to central arrows showed no evidence of inhibition from the peripheral cues, while an inhibitory effect was observed when peripheral targets called for a manual response implies an ICE closer to the input end of the processing continuum.

Oculomotor facilitation

Neurophysiological investigations of exogenous spatial cueing in monkeys have established that there is transient, cue-elicited residual activity in visuomotor neurons residing in the intermediate layers of the SC (Dorris et al., 2002; Fecteau & Munoz, 2005). We reasoned – to the extent that common mechanisms underlie oculomotor facilitation in humans and monkeys – that the behavioral expression of this effect could be visualized most easily by commanding saccadic eye movements to cued or uncued visual regions by way of foveally-presented, left- or right- ward pointing arrows. As expected, the peripheral cues clearly facilitated oculomotor responding at the shortest CTOA. Although the behavioral expression of oculomotor facilitation was not as long-lasting as the neurophysiological data suggested it might be, these findings – when considered in conjunction with the neurophysiological data from this paradigm – support the notion that residual activity of visuomotor neurons in the SC is likely, at least in part, responsible for oculomotor facilitation in humans.

When reflexive oculomotor responses were actively discouraged by feedback, oculomotor facilitation was abolished (i.e., Experiment 2). In our estimation, this result follows logically from the idea that the reflexive oculomotor system would have been tonically suppressed in tasks forbidding reflexive (pro-saccadic) oculomotor responding (Klein & Hilchey, 2011). Indeed, converging evidence comes from neurophysiological studies of the anti-saccade task (e.g., Fecteau et al., 2004), which demonstrate either substantially diminished (Everling, et al., 1999), or no (Ignaschenkova et al., 2004) response related target-elicited activation at the level of the visuomotor and motor cells.

Sensory adaptation

When the CTOA is short (< 500 ms), visual neurons residing in the superficial layers of the SC respond less vigorously to saccade targets occurring at cued as compared to uncued locations (e.g., Boehnke et al., 2011; Cynader & Berman, 1972; Dorris et al., 2002; Fecteau & Munoz, 2005; Wurtz, Richmond, & Judge, 1980). This mechanism has been modeled faithfully in neurophysiologically-plausible dynamic neural field models of the SC (Trappenberg, Dorris, Munoz, & Klein, 2001; Wang, Satel, Trappenberg, & Klein, 2011) as short-term sensory depression or adaptation (Satel et al., 2011; Wang et al., 2012). In effect, the attenuation in target-elicited visual activity at the cued location allows visual inputs at the uncued location to reach saccade threshold more rapidly. Conforming to predictions derived from the monkey neurophysiology and, by extension, the dynamic neural field model of the SC, oculomotor responses were delayed at CTOAs < 500 ms in Experiment 1 when the input pathway was repeatedly stimulated (see also, e.g., Dukewich & Boehnke, 2008; Dukewich, 2009). There was no behavioral evidence for sensory adaptation at the early CTOA in Experiment 2. At this juncture, we cannot confidently endorse an explanation for this finding (see discussion of Experiment 2).

Finally, it bears reminding that to account for IOR effects in spatial cueing paradigms requiring saccadic responses (Wang et al., 2012; Satel & Wang, 2012) short-term sensory adaptation (an early input-based ICE) has been assumed to have a duration substantially greater than that observed in the monkey neurophysiology. However tempting it might be to accept the idea that the IOR observed for peripheral onset targets when previously cued is merely a prolonged instance of sensory adaptation, there are a

variety of findings in the literature which are incompatible with this proposal. Consider first that IOR has no apparent effect on perceptual arrival times in illusory line motion and TOJ tasks (see Klein et al., 1998; Schmidt, 1996). Second, when an eye movement intervenes between a cue and target, IOR is observed in spatiotopic rather than retinotopic coordinates (Maylor & Hockey, 1985; Posner & Cohen, 1984; Satel, Wang, Hilchey, & Klein, 2012). Sensory adaptation at the level of the retinotopically-organized SC cannot explain spatiotopic IOR. Third, oculomotor IOR is robustly observed in an environmental reference frame in natural, overt search tasks in which there are never repeatedly presented sensory inputs (for a review, see Klein & Hilchey, 2011). We therefore reject the "prolonged sensory adaptation" proposal and advance instead the idea that IOR is a late-appearing, long-lasting (> one second), output-based effect occurring in the aftermath of oculomotor response activation that serves principally to bias responding away from previously targeted locations, as originally conceptualized by Posner et al. (1985).

Oculomotor IOR (or simply, IOR)

Experiment 1 demonstrated that when oculomotor responses were required to visual signals appearing in peripheral vision, oculomotor responses were delayed at the late CTOA by roughly the same amount whether in response to a saccade target at, or an arrow commanding a saccade to, the cued location. A precursor to the effect was transient oculomotor facilitation at the cued location when un-confounded by sensory adaptation (as seen with central targets). Thus, in cause and effect, this oculomotor ICE conforms to the theoretical framework of IOR advanced by Posner et al. (1985).

Moreover, the findings are largely consistent with the notion that IOR depends on an intact SC (Posner et al., 1985; Sapir et al., 1999; Sereno et al., 2006a), again because IOR is likely a consequence of oculomotor response activation (e.g., Godijn & Theeuwes, 2002; Posner et al., 1985). If IOR does not merely occur in the aftermath of oculomotor response activation, but is actually caused by it, then conditions abolishing oculomotor facilitation would likewise be expected to abolish IOR.

In Experiment 2 we tested this proposal. By actively discouraging oculomotor responses to peripherally-presented visual signals and by reducing the frequency of oculomotor responding, we predicted that we would reduce – if not eliminate – cue-elicited oculomotor response activation (Everling et al., 1999; Ignaschenkova et al., 2004; Klein & Hilchey, 2011) and, consequently, IOR. Indeed, there was neither evidence for oculomotor facilitation nor oculomotor IOR in Experiment 2. There was, however, evidence for a late input form of ICE (to be discussed in the next section).

With respect to the origins of IOR as measured one second after the peripheral cue in Experiment 1, we propose that the input signals associated with both target types are unaffected by the prior cue but these signals converge on a neural substrate that is locally affected in the vicinity of the cue. Whether this effect is a direct inhibition or an increased threshold for initiating a response (cf Ivanoff, Klein, & Lupianez, 2002; Klein & Taylor, 1994), it takes time to be generated and replaces sensory adaptation as the cause of SRT increases at a previously cued location. It is mediated by as yet undetermined computations that probably involve both the SC and cortical modules (e.g., Tian, Klein, Satel, Xu, & Yao, 2011). The inhibition version of this proposal has

been implemented successfully in a neurophysiologically-plausible dynamic neural field model that demonstrably accounts for a range of data (Satel, Story, Hilchey, Wang, & Klein, 2013).

Although the SC is widely believed to be critical for generating IOR, there are several reasons why it is unlikely to be the neural substrate where the inhibition or response bias is implemented. For example, IOR outlasts all neurophysiological effects that have been identified with IOR in the SC (compare Fecteau & Munoz, 2005, with Samuel & Kat, 2003), and IOR is coded in environmental (Maylor & Hockey, 1985) and object or scene-based (Tipper, Driver, & Weaver, 1991) coordinates. As originally suggested by Klein (2000), the neural substrate where IOR is implemented may be parietal cortex. Mirpour, Arcizet, Ong, and Bisley (2009), for example, recorded extracellularly from the lateral intraparietal (LIP) area while rhesus monkeys searched for a target in a simple and visually stable scene. The LIP is a cortical brain region that projects directly to the SC (Andersen, Asanuma, Essick, & Siegel, 1990) and that is associated with many higher order cognitive operations like motivation, intention, the representation of saccadic response thresholds and the metrics of saccade programming (cf Gottlieb & Balan, 2010; Gottlieb, Balan, Oristaglio, & Suzuki, 2009). Mirpour et al. (2009) found that neurons in the LIP responded less vigorously when, because of a search-related eye movement, a previously fixated element entered its receptive field. These results neatly compliment investigations demonstrating that an intact LIP is essential for observing IOR behaviorally in an environmental reference frame (Sapir, Hayes, Henik, Danziger, & Rafal, 2004; van Koningsbruggen, Gabay, Sapir, Henik, &

Rafal, 2010) in addition to demonstrating that abrupt luminance onsets are not required to observe reductions in neural activation levels or IOR.

Late input-based ICEs

The results of Experiment 2 demonstrate that an ICE could be measured at the long CTOA using manual localization responses even while the reflexive oculomotor machinery was successfully suppressed (and, confirming this suppression, there was no evidence of early oculomotor activation). Using the diagnostic from Taylor and Klein (2000; see also p. 9) this ICE is entirely of the "input" type because there was no evidence of slowed saccades in the cued direction in response to central arrows. Moreover, there was no apparent behavioral evidence of any sensory adaptation effect at the short CTOA when keypress responses were required to peripheral onset targets.

The data from Experiment 2 call into question whether the late ICEs generated in covert spatial orienting paradigms *that carefully control for the presence of, and actively discourage, saccadic eye movements* should be classified as IOR, given there was no evidence in Experiment 2 for the presence of either the cause (oculomotor activation) or the effect (oculomotor inhibition) of IOR, as it was named by Posner et al. (1985). We believe that declining to call such ICEs IOR is a progressive departure from the common practice of attributing any evidence of an ICE in a covert spatial orienting paradigm to IOR**,** and represents a return to the original meaning of the term.

On the basis of the results of Experiment 2, we reject the assumption that oculomotor activation is the cause of *both* the input and output forms of "IOR" that the Taylor and Klein (2000) experiments so clearly distinguished on the basis of their

different effects. In the remainder of this section we will explore some of the implications of this rejection and we will present recent evidence that supports it.

One of the more compelling dissociations between keypress and oculomotor ICEs was demonstrated by Sumner et al. (2004). Sumner et al. took advantage of a special class of stimulus, the S-cone or short-wave-sensitive cone stimulus. S-cone stimuli are special because there are no direct projections from S-cones to the SC. In consequence, these stimuli are initially invisible to the retinotectal and magnocellular pathways that ultimately project to this midbrain structure. It is therefore difficult to see how this class of stimulation would *directly* activate an oculomotor response at the level of the SC. Sumner et al. administered either the S-cone or luminant stimuli as cues in a spatial cueing paradigm and required keypress or saccade localization responses to cued or uncued luminant peripheral targets. ICEs were obtained with both saccadic and manual responses when the cues were luminant. Critically, when the cues were S-cones, an ICE was obtained only with keypress responses. Sumner et al.'s pattern of results was sufficiently compelling to evoke, in Lupianez, Klein, and Bartolomeo (2006), the inference, "...that there are (at least) two types of IOR: one affecting both manual and ocular responses, presumably depending on the superior colliculus, and the other affecting only manual responses, presumably depending on the cortex" (p. 1010). Converging evidence for this inference comes from studies of patients with damage to different oculomotor circuitry (Gabay, Henik, & Gradstein, 2010; Smith, Rorden, & Jackson, 2004), damage to fronto-parietal and related cortical networks (Bourgeois, Chica, Migliaccio, Thiebaut de Schottan, & Bartolomeo, 2012), and from studies of

normal individuals in which the experimental situation restricted oculomotor activation (Hilchey et al., in press; Smith, Schenk, & Rorden, 2012).

Although some forms of input-based ICEs at late CTOAs may not be caused by oculomotor activation, the experiments presented here do not allow us to endorse with confidence what they are caused by. In other theoretical and empirical work (e.g., Hilchey et al., 2013), we are exploring the possibility that a process of automatic manual response activation, followed by response suppression when the activation exceeds some threshold [as has been hypothesized at times for negative compatibility effects (see Sumner, 2007, for review)], might generate an ICE that has been mistaken for IOR[4]. Another possibility is that input-based ICEs are mediated by relatively low-level visuo-cortical processes (e.g., Muller & Kleinschmidt, 2007; Satel, et al., 2013; Smith, Ball, & Ellison, 2012).

Conclusions

When the inhibitory aftereffect of orienting was named "inhibition of return" by Posner et al. (1985) - the phenomenon they were naming was: 1) Caused by activation in the oculomotor system, 2) its effect on subsequent behavior was manifest as a long-lasting response bias, 3) an intact SC was important for its normal operation and 4) its function was identified as novelty seeking. By exploring the time course of exogenous cueing effects when saccadic responses were made to both peripheral and central arrow targets, in Experiment 1 at the earliest CTOA, we observed both oculomotor facilitation

[4] Following this suggestion, the reader may wonder whether an ICE with such a motoric cause may have only presented as an input-based effect in Experiment 2 because the "inhibition" was confined to the primed (manual) effector. We remind the reader that late ICEs in manual localization experiments forbidding oculomotor responses (see Figure 2.1, conditions 1 & 2; Taylor, & Klein, 2000) have input-based effects (i.e., there is little evidence for ICEs even when manual responses are made to arrow S2s).

(central arrow targets) and sensory adaptation (peripheral targets; an early input-based ICE). In this same experiment, at the longest CTOA, a motoric form of ICE was observed (similar SRT costs with central and peripheral targets); a form that we believe deserves the moniker "inhibition of return", as originally defined by Posner et al. (1985). Experiment 2, wherein we required participants to make only manual responses to peripheral targets and the probability of central arrow targets still calling for saccades was reduced (to discourage oculomotor activation), extends and constrains these observations. First, the early oculomotor facilitation depends critically on the activation state of the reflexive oculomotor machinery at the time of the cue; this facilitation was not observed when the participant was precluded from making saccades to all peripheral events. Second, the sensory adaptation effect that was so robust at the short CTOA in Experiment 1 (when a peripherally presented saccade target repeated the pathway of the previous cue), was not observed when the same stimulus sequence required a manual response in Experiment 2. Third, and most importantly, at the longest CTOA – while manual responses to peripheral targets were under the influence of a late ICE – no such effect was observed in the saccadic responses to central arrows.

Thus, as suggested by Klein and Hilchey (2011), an input form of ICE at late CTOAs is generated by peripheral cues *only* when the reflexive oculomotor machinery is suppressed. As this form is not caused by oculomotor activation and does not manifest as a general response bias, it does not satisfy Posner et al.'s (1985) definition of "inhibition of return". Until this form is better understood, we recommend that it be referred to by the more general, and theoretically uncommitted, "inhibitory cueing effect," or ICE.

Table 2.1
The Causes, Effects, Functions and Names Attributed to the Inhibitory Aftermath of Orienting in the Two Seminal Papers of Posner.

Posner & Cohen (1984)	Defining concepts	Posner et al. (1985)
Any visual stimulus	Cause	Oculomotor activation
Reduces efficiency of target detection	Effect	Motor bias
Novelty seeking	Function	Novelty seeking
Inhibition	Name	Inhibition of return

Table 2.2
Mean SRTs (in ms) for Each Level of Each Factor and the Corresponding Cueing Effect (Uncued SRT - Cued SRT).

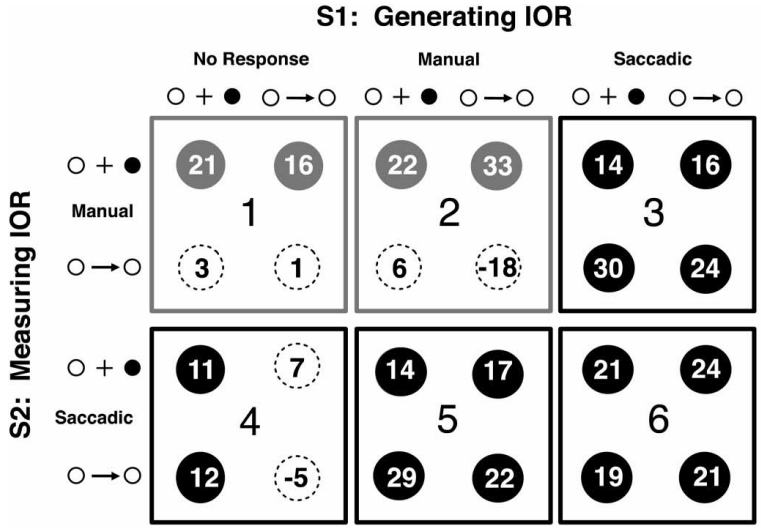
	Peripheral targets			Central targets		
CTOA(ms)	Cued SRT	Uncued SRT	Cueing effect	Cued SRT	Uncued SRT	Cueing effect
150	363	341	−22	358	378	20
250	304	282	−22	326	333	7
350	303	266	−37	309	319	10
450	304	276	−28	318	321	3
1,050	282	260	−22	328	313	−15

Table 2.3
Mean RTs/SRTs (in ms) for Each Level of Each Factor and the Corresponding Cueing Effect (Uncued - Cued).

	Peripheral targets (manual)			Central targets (saccade)		
CTOA (ms)	Cued RT	Uncued RT	Cueing effect	Cued SRT	Uncued SRT	Cueing effect
150	497	494	−3	473	464	−9
1,050	403	383	−20	428	423	−5

Figure 2.1

The matrix on the left illustrates all 24 combinations of the first and second stimulus (S1, S2) and responses required to each (no response, manual, and saccadic for S1; manual and saccadic for S2) that were explored by Taylor and Klein (2000). The iconograms, which label the columns (S1) and rows (S2), convey whether the stimuli were central arrows or peripheral events. Solid circles indicate combinations for which significant inhibitory aftereffects of S1 upon the processing leading to responses to S2 were obtained. Unfilled dotted circles indicate conditions for which no inhibitory aftereffect was observed. The two forms of ICE/IOR described in the text are denoted by gray (for the input form, ICE) and black (for the output form, IOR) filled circles. The embedded table on the right identifies the six combinations of responses that the participant was instructed to make to S1 and S2 in a session of testing. The numbers on each row of this table correspond to the numbers at the center of each box in the figure and to the six experiments (which are referred to here as conditions) reported by Taylor and Klein.



Condition	Tasks	
	S1	S2
1	No Response	Manual
2	Manual	Manual
3	Saccade	Manual
4	No Response	Saccade
5	Manual	Saccade
6	Saccade	Saccade

Figure 2.2

The sequence of events on a typical trial in both experiments. In Experiment 1, the interval (panel 4) was randomly selected from: 50, 150, 250, 350, and 950 ms; in Experiment 2, the intervals were 50 and 950 ms. In Experiment 1, saccades were made to both possible target types (panel 5), which were equiprobable. In Experiment 2, manual responses were made to peripheral targets, which were presented on 80% of the trials, while saccadic responses were made to central arrows, which were presented on 20% of the trials. Although shown here as black on white, in the actual display the background was black and all stimuli were white.

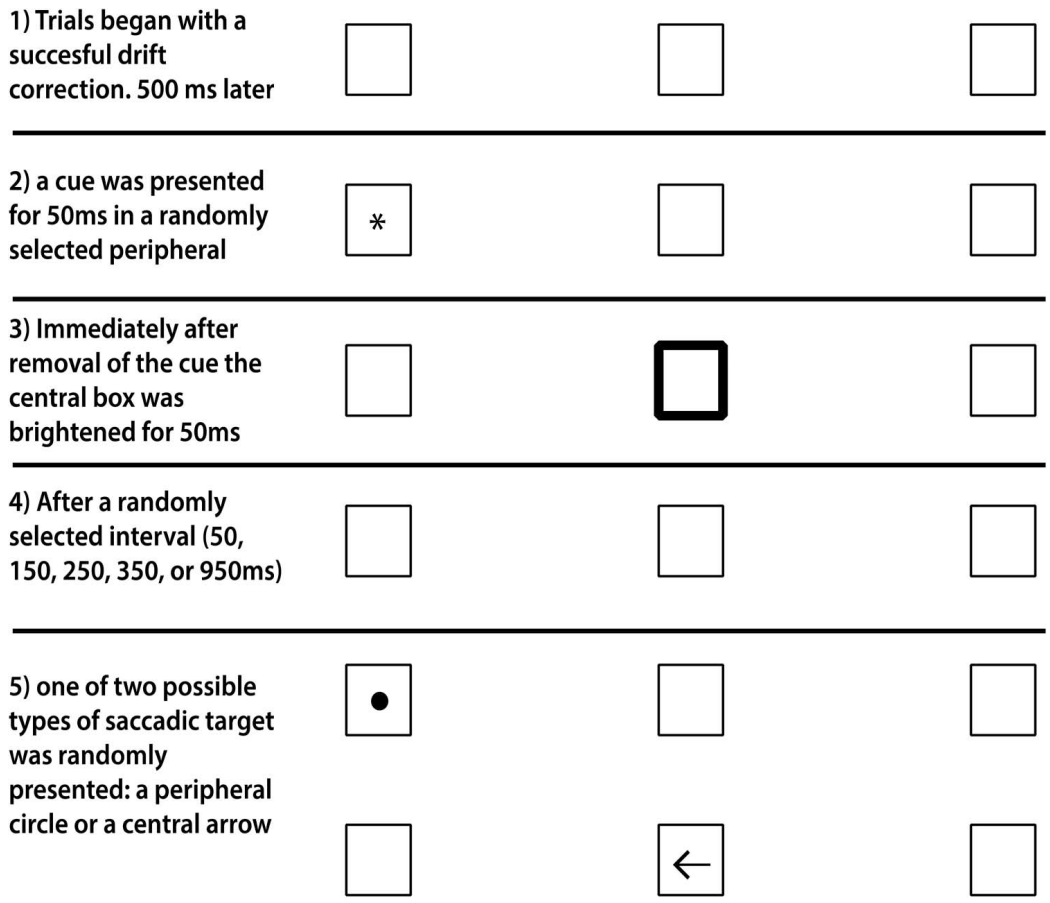


Figure 2.3

The three-way interaction among factors CTOA, Cueing, and Target Type based on saccades landing within 1° of the center of the target box (accurate saccades). The error bars are Fisher's Least Significant Differences (FLSDs), which are intended to facilitate comparisons on cueing effects between CTOAs and Target Types. Any overlap in the FLSDs signifies a nonsignificant difference. Filled circles indicate that the cueing effect is significant against zero with single sample t tests.

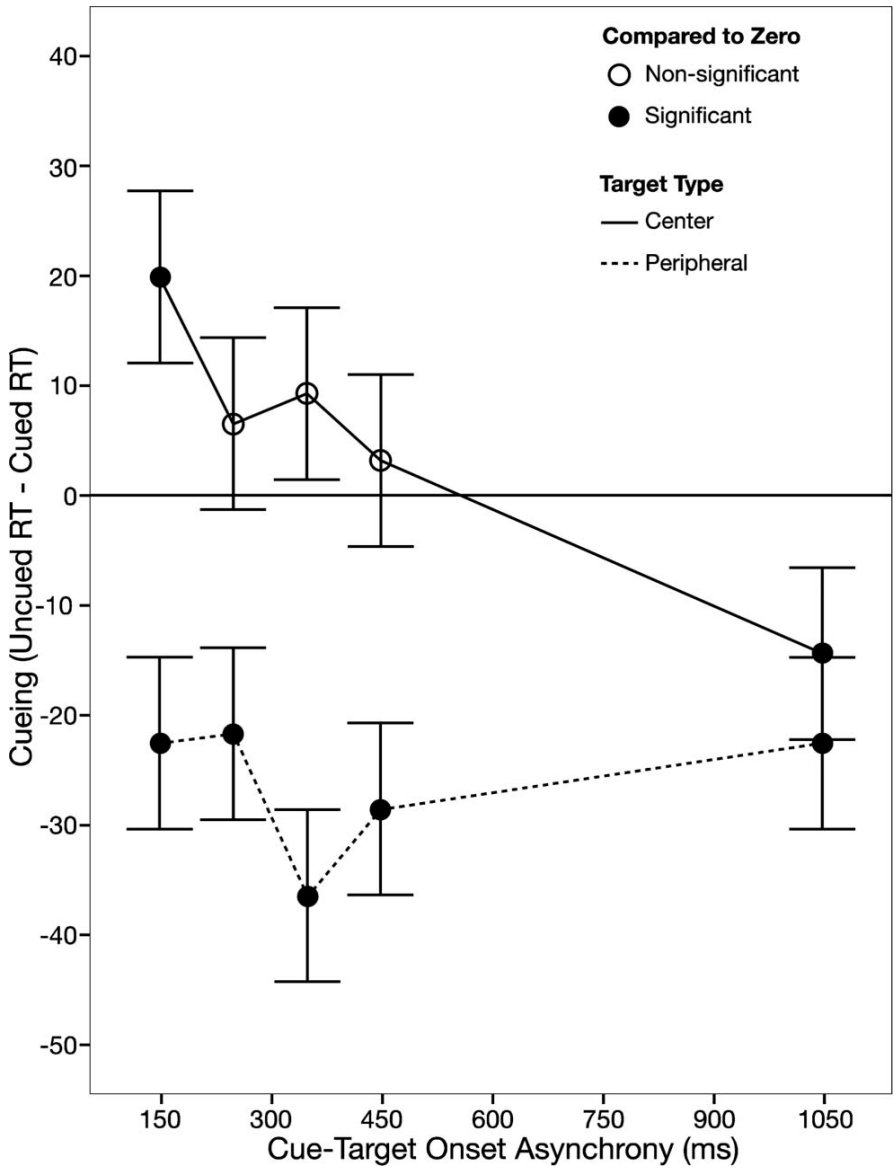
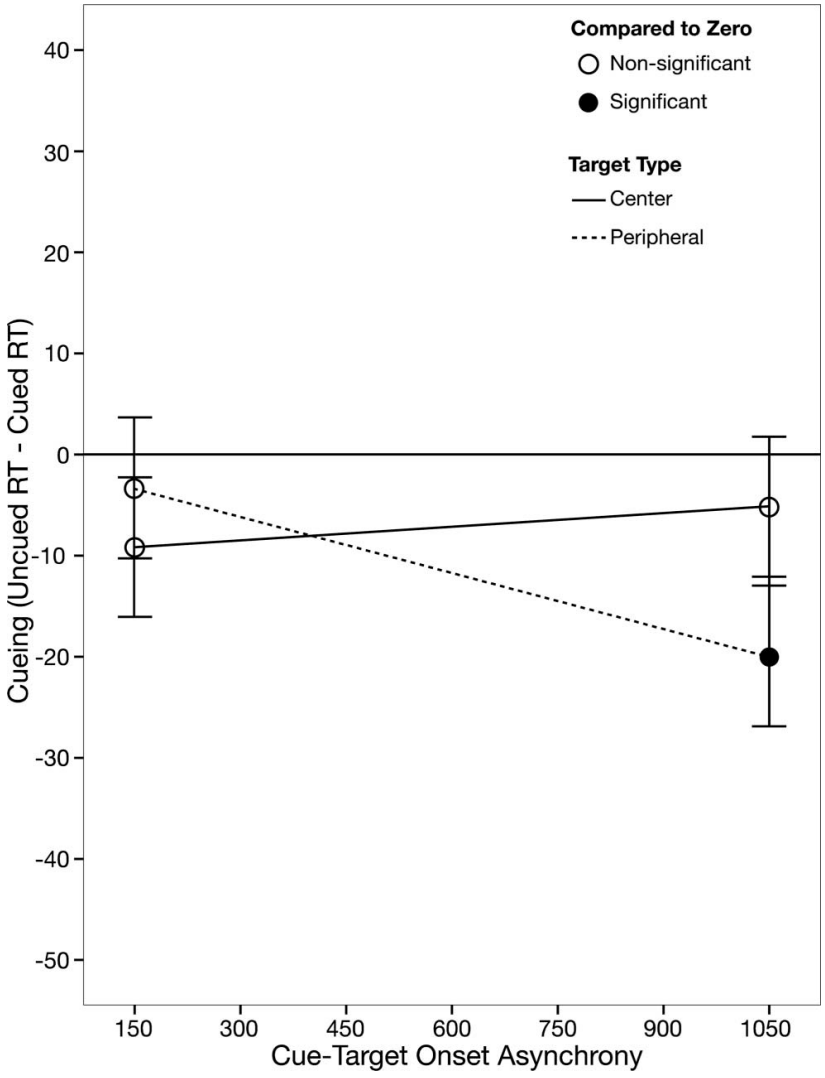


Figure 2.4

The three-way interaction among factors Cueing, CTOA, and Target Type. Error bars are FLSDs.



CHAPTER 3:
OCULOMOTOR INHIBITION OF RETURN: HOW SOON IS IT "RECODED" INTO SPATIOTOPIC COORDINATES?

The manuscript based on this study is presented below. Co-authors for this manuscript are Dr. Raymond M. Klein, Dr. Jason Satel and Dr. Zhiguo Wang, respectively.

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Abstract

When, in relation to the execution of an eye movement, does the recoding of visual information from retinotopic to spatiotopic coordinates happen? Two laboratories seeking to answer this question using oculomotor inhibition of return (IOR) have generated different answers: Mathôt and Theeuwes (2010) found evidence for initial coding of IOR to be retinotopic, while Pertzov, Zohary, and Avidan (2010) found evidence for spatiotopic IOR at even shorter post-saccadic intervals than were tested by Mathôt and Theeuwes (2010). To resolve this discrepancy we conducted two experiments that combined their methods while testing as early as possible. We found early spatiotopic IOR in both experiments, suggesting that visual events, including prior fixations, are typically coded into an abstract, allocentric representation of space either before or during eye movements. This type of coding enables IOR to encourage orienting toward novelty and consequently to perform the role of a foraging facilitator.

Introduction

Efficient sampling of the visual world when achieved by changes in gaze direction is determined by a multitude of factors. In visual search, for example, task demands (*cf.* Dodd, van der Stigchel, & Hollingworth, 2009; Smith & Henderson, 2009), bottom-up factors that concern the natural salience of objects (see Wolfe, & Horowitz, 2004, for a review) and, importantly, a "memory" of previously sampled locations (*cf.* Peterson, Beck, & Vomela, 2007) are all at play. One mechanism proposed to afford this memory function is known as *inhibition of return* (IOR; Klein, 1988; Klein & MacInnes, 1999; Itti & Koch, 2001; Posner & Cohen, 1984). IOR, cast in broad strokes, refers to the robust phenomenon of increased saccadic or manual response times to targets in previously inspected locations relative to those in previously unattended, and distance-matched, locations.

IOR is traditionally explored in a cueing paradigm in which a peripheral onset target is preceded by an uninformative peripheral cue. The peripheral cue is believed to automatically capture attention when, at short cue-target onset asynchronies (CTOAs), processing of targets presented at cued locations is faster than those at uncued locations (facilitation effect). At longer CTOAs, processing of targets at the cued location is hindered (IOR effect). Both mechanisms are ecologically important. The facilitation mechanism prioritizes the processing of salient events in the environment, while IOR encourages orienting toward novel locations (Posner & Cohen, 1984; Posner, Rafal, Choate, & Vaughan, 1985) and thus facilitates foraging behaviors (Klein, 1988; Klein & MacInnes, 1999).

With each successive eye movement, objects within the visual field are represented in new retinal coordinates. To function effectively as a foraging facilitator (Klein & Dukewich, 2006), IOR must be coded in spatiotopic rather than merely in retinotopic coordinates. Note that in using the term 'spatiotopic', we are referring to a non-retinotopic, environmental reference frame. This is exactly what Posner and Cohen (1984) found in their seminal paper on IOR (see also Maylor & Hockey, 1985). However, the spatiotopic reference frame was put in question by Golomb, Chun and Mazer (2008) who proposed that the "visual system's native or low-level representation of endogenously maintained spatial attention is retinotopic, and remapping of attention to spatiotopic coordinates occurs slowly and only when behaviorally necessary" (p. 10654). In response to this claim, two recent studies (Mathôt & Theeuwes, 2010; Pertzov et al., 2010) explored the time course of the retinotopic and spatiotopic coding of IOR with a task similar to that used in Maylor and Hockey (1985).

The sequence of events in a typical trial in both studies (*i.e*., Mathôt & Theeuwes, 2010; Pertzov, Zohary, & Avidan, 2010) was similar: IOR was generated by presenting an uninformative, to-be-ignored peripheral onset signal (*cue*). A saccade was executed in response to a peripheral onset signal (*shift stimulus)* which later allowed for saccade response time (SRT) measurements in two reference frames (*i.e.,* spatiotopic and retinotopic). Following this intervening saccadic eye movement, at varying intervals after termination of the saccade, a final saccade signal (which we will refer to as the *target*) was delivered. The target was presented at the same screen location as the cue (spatiotopic), at the same location on the retina of the participant (retinotopic), or at one

of two control locations, respectively, to the spatiotopic and retinotopic targets. Thus, the target evaluated whether any cueing effects were present and determined the reference frames within which they exist. Despite these similarities, the two investigations reported dramatically different patterns of results for the nature of the representation immediately following the first saccade (see Figure 3.1, where the empirical discrepancy is highlighted by the rectangle): Mathôt and Theeuwes (2010) reported initial retinotopic coding of IOR, whereas Pertzov et al. (2010) reported initial spatiotopic coding. As suggested by Mathôt and Theeuwes (2010), "This apparent discrepancy warrants further scrutiny" (p. 1796). Our goal in the present study is to apply such scrutiny.

Important methodological features of Mathôt and Theeuwes (2010) and Pertzov et al. (2010)

Following the methods of Golomb et al. (2008), while improving upon them (notably by having two separate target locations to provide baselines against which to measure retinotopic and spatiotopic IOR), Mathôt and Theeuwes (2010) used a *quasi-random* arrangement of shift stimuli and cues. In contrast, Pertzov et al. (2010), while also using separate baselines for estimating the two forms of IOR, used a much more *rigid* arrangement, akin to that used by Maylor and Hockey (1985). This difference is illustrated in Figures 3.2A-C and 3.2D. At the beginning of a trial in Pertzov et al. (2010), participants were fixating the center of the screen and the cues and targets could be presented at 4 or 8 fixed locations, respectively, centered around fixation (Figure 3.2A). There were only two possible shift stimuli (and, consequently, first saccades) which were 7 degrees to the left or right of fixation. In contrast, in Mathôt and Theeuwes (2010), the shift stimuli (and first saccades), which were always 6 degrees, were in

randomly selected directions (with the restriction that neither they, nor the 4 possible targets that might be delivered, could fall off the screen; see Figure 3.2D). Possible cue and target locations (always of 4.2 degrees eccentricity) were centered around fixation. The major procedural difference between these studies lies in their use of fixed versus random locations for cues, shift stimuli, and targets. Since Pertzov et al. (2011) found early spatiotopic IOR using fixed displays[5], while Mathôt and Theeuwes (2010) did not find significant spatiotopic IOR using random locations, it is possible that spatiotopic coding may be faster, more likely, and/or more powerful *when the locations where task-relevant events occur are distinct***,** as they would be when these events occur at fixed locations. This possibility will be referred to as the *locational salience hypothesis*.

A second procedural difference between the two experiments concerns the degree to which the cue and shift stimulus were distinct. This distinctiveness may have practical and conceptual significance. The practical significance concerns task difficulty: Subjects reported extreme difficulty following the instructions to ignore the cue, saccade to the shift stimulus and then saccade to the target when using the paradigm as sent to us by Mathôt and Theeuwes (2010). We hypothesized that the critical factor responsible for this difficulty was the high similarity between the cue and shift stimulus (see Figure 3.2E), both being unfilled circles in the same shade of grey. This similarity alone might

[5] At the beginning of each trial in Pertzov et al. (2010) the upcoming target might be presented at any of 8 possible fixed locations, centered on the initial fixation point (Figure 3.2A). After the cue and shift stimulus, there were only 4 possible target locations. Importantly, the combination of cue and shift stimulus locations might shift (when these were in the same direction) or not shift (when these were in opposite directions) the center of gravity of locations on the screen that might contain the target (compare Figures 3.2B,C with 3.2A). Because, particularly in the context of a study with fixed locations, the shift in attention that is warranted on the same direction trials might have somehow interfered with the natural coding of IOR, in Figure 3.1 we have only illustrated the opposite direction condition from Pertzov et al. (2010). Even when all the data from the shortest interval used by Pertzov et al. (2010) are combined, there was substantial spatiotopic IOR that was about 9 ms greater than the retinotopic IOR observed at this time period. This is almost precisely the opposite pattern reported by Mathôt and Theeuwes (2010).

not have been a problem if the cue and shift stimulus had been separated by a sufficient interval, or were always presented at different locations (as in Pertzov et al., 2010). However, in Mathôt and Theeuwes (2010), the cue and shift stimuli were close together temporally (separated by only 150 ms) and over the course of a block of trials, the cues and shift stimuli alike could appear, more or less, anywhere on the screen. None of these factors were operating in Pertzov et al. (2010). First, as illustrated in Figure 3.2E, the cue and shift stimulus were different shapes and differently filled. Second, the cue and shift stimuli were separated by 700 ms. Finally, for the entire experiment there was a tight separation between the shift stimulus locations and the locations where cues and targets could appear. It is possible that the highly demanding stimulus selection and/or response competition that characterized the Mathôt and Theeuwes (2010) methodology may have delayed the dynamic remapping of IOR from retinotopic into spatiotopic coordinates. The absence of such competition between the cue and shift stimulus in Pertzov et al. (2010), on the other hand, may have allowed for rapid pre-saccadic transformation from retinotopic into spatiotopic coordinates. This possibility will be referred to as the *featural salience hypothesis*.

There were also differences between the two studies with regard to the timing of the events of a trial. In Pertzov et al. (2010), the interval between the onset of the cue and shift stimulus was 700 ms, considerably longer than the 150 ms used by Mathôt and Theeuwes (2010). In Mathôt and Theeuwes (2010), the timing of the target was fixed in relation to the cue (CTOAs, were 500, 800, 1100 and 1400 ms) whereas in Pertzov et al. (2010) the delivery of the target was gaze contingent and occurred 10, 250 or 600 ms

after the participants had fixated the shift stimulus. Because of these methodological differences, the CTOAs were variable in Pertzov et al. (2010) (approximately 960, 1200 and 1550 ms) while the end saccade-target intervals were variable in Mathôt and Theeuwes (2010) (approximately 70, 350, 640, 920 ms). Despite these differences, there is good agreement about spatiotopic coding at long intervals. Nevertheless, there is a dramatic disagreement in how early spatiotopic coding of IOR was observed.

The experiments

The key question addressed here was whether, immediately after an eye movement, IOR generated by a cue and measured by a saccade, would be represented in spatiotopic coordinates. Even if the fixed procedure used by Pertzov et al. (2010) were not untoward, we prefer the quasi-random procedure of Mathôt and Theeuwes (2010) and Golomb et al. (2008). One reason is related to the functional significance of IOR as a novelty seeking mechanism (Posner & Cohen, 1984) and foraging facilitator (Klein, 1988). If these functions are to operate in the real world, then IOR would need to be present, and coded spatiotopically, in a richer and more random world than the one explored by Pertzov et al. (2010). Consequently, we decided to use the quasi-random procedure of Mathôt and Theeuwes (2010). Because our question was about the relative magnitude of retinotopic versus spatiotopic IOR immediately after an eye movement, we chose to use the gaze-contingent delivery of the target procedure of Pertzov et al. (2010). Finally, while keeping the timing of the cue and shift stimulus the same as in Mathôt and Theeuwes (2010), we also manipulated the degree to which these two stimuli (cue and shift stimulus) could be distinguished (see Figure 3.2E). Whereas in both of our

experiments the cue and shift stimulus differed in terms of size and fill, in Experiment 1 the cue and shift stimulus could be easily distinguished on the basis of a difference in brightness, whereas in Experiment 2 this salient difference was eliminated.

The locational salience and featural salience hypotheses described above are simultaneously tested by our combination of Mathôt and Theeuwes' (2010) quasi-random presentation of all stimuli with our manipulation of the discriminability of the cue from the shift stimulus. If early spatiotopic coding is in part dependent on an internal representation of plausible shift stimulus locations, as would be encouraged by stability in the locations of cues, shift stimuli, and targets (the locational salience hypothesis), then because we are using the quasi-random selection of cue, shift stimulus, and target locations (*cf* Pertzov et al., 2010), we should find that retinotopic IOR is greater than spatiotopic IOR (as reported by Mathôt & Theeuwes, 2010). If, however, early spatiotopic coding is dependent on the ease with which the target for a saccade (in this case the shift stimulus) can be selected (the featural salience hypothesis), then spatiotopic IOR should be greater than retinotopic IOR when selection is easy (as in Pertzov et al., 2010) and this pattern should be reduced, if not reversed, when selection is difficult (as in Mathôt & Theeuwes, 2010).

Experiment 1

Methods

Participants

Twelve naive participants (1 male) were tested in Experiment 1. Participants were university students who volunteered to participate for course credit. They all reported normal or corrected-to-normal visual acuity and their average age was 21 years.

Stimuli and procedure

The stimuli and procedure used in Experiment 1 were similar to Mathôt and Theeuwes (2010). All stimuli were presented on a 19" ViewSonic Optiquest Q95-3 CRT monitor (refresh rate = 100 Hz) connected to an Intel Core Duo processor, at a viewing distance of 22.5". Eye movement responses were recorded by way of an Eyelink II head-mounted eye tracking system that was connected to a host computer operating on a Pentium 4 processor. The velocity threshold used to determine whether a saccadic eye movement was made was set to 35°/s and the response times were computed by subtracting the time at which the eye movement exceeded the velocity threshold from the time at which the imperative stimulus appeared onscreen. To ensure an accurate recording of the gaze coordinates, a 9-point calibration procedure was administered to the observers before the experiment. To further ensure an accurate reading of gaze position, the successful completion of a drift correction phase was required at the beginning of each trial.

After the drift correction, a filled gray fixation disk (d = 0.5°) appeared onscreen. Participants were required to fixate this gray dot until it moved to a new location. 750 ms after the appearance of the fixation disk, a peripheral onset cue was presented at a location 4.2° visual angle from the fixation disk. This onset cue was a white unfilled circle (d = 1.0°) presented for 50 ms. 150 ms after the appearance of the cue, the fixation

disk (the shift stimulus) stepped to a location that was 6° from the original location and 4.2° from the cued location. If participant's eyes deviated more than 1° from the fixation disk before it moved, the trial was aborted. The termination of a successful eye movement to the shift stimulus was used to trigger the presentation of the target, which was a bright green disk (d = 0.5°). Based on our eye monitoring equipment and software, the average interval between termination of the shift stimulus-directed saccade and the appearance of the target was 42 ms. The target called for a saccadic response within 750 ms, and failure to respond to the target within this period led to the trial being aborted. As illustrated in Figure 3.2D, the target was presented with equal probability at one of four randomly selected locations: the cued location (cued spatiotopic location), a location 6° from the cued location and 4.2° from the shift stimulus location (uncued spatiotopic location), a location 6° from the cued location and 4.2° from the shift stimulus location (cued retinotopic location), and a location 6° from the uncued spatiotopic location and 4.2° from the shift stimulus location (uncued retinotopic location). An algorithm was implemented to guarantee that none of these four possible target locations would fall off the screen. All aborted trials were recycled and presented to the participant in a random order.

Results and discussion

8.4% of the trials were excluded because the final saccade deviated by more than 2.0° from the target's location. 3.6% of the trials were excluded because the saccade to the shift stimulus deviated by more than 2.0° from the shift stimulus' location. Finally, less than 1.0% of the trials were excluded because of extremely fast (SRT < 100 ms) or

slow (SRT > 500 ms) saccadic response times to the target. All SRTs and exclusions for each level of factors cueing and coordinates can be found in Table 3.1.

SRTs to the targets (see Table 3.1) were subjected to an ANOVA which revealed significant main effects of coordinates [$F(1, 11) = 88.55$, $p < 0.001$] and cueing [$F(1, 11) = 56.31$, $p < 0.001$]. These main effects reflect that, overall, IOR was significant and SRTs testing retinotopic coding were faster than SRTs testing spatiotopic coding. The 2-way interaction between coordinates and cuing was significant [$F(1,11) = 6.35$, $p < .05$], reflecting the finding that spatiotopic IOR was greater than retinotopic IOR. This pattern demonstrates that spatiotopic IOR can be encoded before, during, or very shortly after a saccade. This pattern contrasts with that reported by Mathot and Theeuwes (2010; retinotopic IOR > spatiotopic IOR). Given that cue-target similarity is the most noteworthy methodological difference between the present investigation and Mathot and Theeuwes (2010), this finding is consistent with the featural salience hypothesis.

Experiment 2

Method

Participants

Twelve naive participants (1 male), who were not tested in Experiment 1, were tested in Experiment 2. Their average age was 21 years. Participants were selected from the university population and performed for course credit. All participants reported normal or corrected-to-normal visual acuity.

Stimuli and procedure

Experiment 2 was identical to Experiment 1 with one exception: a gray (as opposed to a white) unfilled circle (d = 1.0°) served as the onset cue (see Figure 3.2E). More explicitly, as compared to Experiment 1, the cue onset and switch stimulus were more similar on the luminance dimension.

Results and discussion

10.6% of the trials were excluded because the final saccade deviated by more than 2.0° from the target's location. 3.3% of the trials were excluded because the saccade to the shift stimulus deviated by more than 2.0° from the shift stimulus' location. Finally, less than 1.0% of the trials were excluded because of extremely fast (SRT < 100 ms) or slow (SRT > 500 ms) saccades to the target. All SRTs and exclusions for each level of factors cueing and coordinates can be found in Table 3.1.

Saccadic SRTs to the targets (see Table 3.1) were subjected to an ANOVA which revealed significant main effects of coordinates [$F(1, 11) = 25.03$, $p < 0.001$] and cueing [$F(1, 11) = 23.16$, $p < 0.001$]. These main effects reflect that, overall, IOR was significant and that SRTs testing retinotopic coding were faster than SRTs testing spatiotopic coding. In contrast to the results of Experiment 1, the 2-way interaction did not approach significance [$F(1,11) = 1.102$, $p > 0.3$]. This pattern (retinotopic equal to spatiotopic IOR) does not replicate the pattern reported by Mathôt and Theeuwes (2010; retinotopic IOR > spatiotopic IOR; see the General Discussion for an explanation), nor does it replicate the pattern reported in Experiment 1 (spatiotopic IOR > retinotopic IOR). Taken together, however, the current and extant data are consistent with the featural salience hypothesis (see section "Spatiotopic coding and the featural salience

hypothesis" below), but not against the locational salience hypothesis (see section "Spatiotopic coding and the locational salience hypothesis" below).

General discussion

There are three main findings from our experiments. First and foremost, cue-generated IOR appears to be coded spatiotopically almost immediately after a post-cue eye movement. This implies that visual events are coded into spatiotopic (and possibly allocentric) coordinates either before, or during, eye movements (see also Pertzov et al., 2011; Tatler & Land, 2011). Second, at this point in time, spatiotopic IOR can be larger than retinotopic IOR (Experiment 1). Third, we observed a consistent benefit for second saccades that moved the eyes forward (in the retinotopic direction) as opposed to backward (in the spatiotopic direction).

The discrepancy between Mathôt and Theeuwes (2010) and the present experiments

Despite our close replication of most of the methods of Mathôt and Theeuwes (2010), we never observed their pattern of results that shortly after a saccade spatiotopic IOR was numerically smaller than retinotopic IOR and was not statistically significant. To help resolve this discrepancy, we consulted with Mathôt and eventually requested and received trial-by-trial data from the Mathôt and Theeuwes (2010) experiment. Because we are more concerned about the relative magnitude of spatiotopic and retinotopic IOR immediately following the saccade to the shift stimulus, we initially applied the same data exclusion criteria of Mathôt and Theeuwes (2010) and performed an ANOVA on the SRTs of their shortest CTOA. This analysis revealed significant main effects of

coordinates [$F(1, 13) = 7.83$, $p < 0.05$] and cueing [$F(1, 13) = 5.02$, $p < 0.05$]. Consistent with, but not demonstrating, stronger retinotopic IOR, a marginally significant interaction between cueing and coordinates was also observed [$F(1, 13) = 3.73$, $p = 0.08$]. When we examined the distribution of their participants' SRTs to targets, we recognized that their lower bound of exclusion (50 ms, as reported at the beginning of their results section, p. 1795) was far too low (Figure 3.A1[6]), so we reanalyzed their data using the same lower bound as in the analysis of our data (100 ms) and as seemed appropriate for their data set given the distribution. For their shortest CTOA, the main effect of coordinates was significant [$F(1, 13) = 18.1$, $p < 0.001$] and the main effect of cueing was marginally significant [$F(1, 13) = 3.89$, $p = 0.07$]. However, the marginally significant interaction between coordinates and cueing went away [$F(1, 13) = 0.02$, n.s.], suggesting that retinotopic and spatiotopic IOR were equivalent (both were about 10 ms). With this result in hand, the original discrepancy that motivated our experiments is considerably diminished. As reported by Pertzov et al. (2010), cue-generated spatiotopic and retinotopic IOR are generally both present immediately after a saccade.

To the extent to which one believes that the pattern of data that includes extremely/impossibly fast SRTs might be theoretically meaningful, one might consider re-analyzing the data in the present investigation with the same SRT cutoffs used by Mathot and Theeuwes (2010). It is important, however, to recognize that fast (< 100 ms) SRTs were rare in our experiments. It perhaps then comes as little surprise that including these inordinately fast SRTs in our analyses does not change the pattern of results. Why/

[6] The figure is referred to as 3.A1 because it appeared as an appendix in the published report. All figures and/or tables previously published as appendices will be denoted by "A".

how Mathôt and Theeuwes' (2010) participants made so many extremely if not impossibly fast saccades to the targets (5.56%) remains a puzzle to us. In any event, we believe those SRTs should be excluded from analyses because they are unlikely to be responses to the target.

Spatiotopic coding and the featural salience hypothesis

According to the featural salience hypothesis, spatiotopic IOR may depend on cue-shift stimulus discriminability. To explore the degree to which this factor might be important, we considered on how many dimensions the cue and shift stimulus differed in each of the key studies that have examined retinotopic versus spatiotopic coding of IOR using saccadic responses (see Table 3.2). As can be seen in Figure 3.3, retinotopic IOR immediately after a saccade is relatively unaffected by the difficulty of selecting the saccade metrics, while spatiotopic IOR immediately after a saccade is substantially affected. This interesting difference is certainly worthy of further investigation. For example, new experiments would be required to determine if it is related to the nature of the spatial attention that the cue is receiving when selecting against the cue is difficult, or if it is due to the competition for limited processing resources that may be required by both this selection process and the computations assumed to be required for spatiotopic remapping.

Spatiotopic coding and the locational salience hypothesis

It is not possible to definitively rule out the “locational salience hypothesis”, despite our finding evidence that cue-target distinctiveness mediates the hypothetical retinotopic to spatiotopic remapping process. Our primary objective was to determine

which factor may have led to significant retinotopic but insignificant spatiotopic IOR soon after an intervening eye movement was made in Mathôt and Theeuwes (2010; a finding that, upon further scrutiny of their data, was questionable, and a finding that had, until re-analysis, been in conflict with the findings of Pertzov et al., 2010). Recall that this particular finding, coupled with the findings of significant spatiotopic IOR but insignificant retinotopic IOR at longer CTOAs, led Mathôt and Theeuwes (2010) to the inference that spatiotopically-coded IOR was derived from an initial retinotopically-coded representation of IOR. We employed Mathôt and Theeuwes' (2010) quasi-random stimulus presentation procedure because it was possible that this methodological aspect, unique to Mathôt and Theeuwes, may have contributed to greater retinotopic than spatiotopic IOR at their short CTOA. Thus, we preserved this particular methodological feature (which may have delayed the onset of spatiotopic coding) and manipulated the other theoretically meaningful factor (*i.e.,* cue-shift stimulus distinctiveness; which may also have delayed spatiotopic coding). Had we reproduced the reported Mathôt and Theeuwes' (2010) finding in the present investigation, there would be relatively strong evidence against the "featural salience hypothesis" and we would, consequently, be justified in following the experiment up with a direct test of the "locational salience hypothesis". Our finding of evidence in favor of the featural salience hypothesis (see Figure 3.3) does not preclude the possibility that presenting Mathôt and Theeuwes' (2010) stimuli at fixed (instead of quasi-random) locations would increase the magnitude of spatiotopic IOR at the earliest CTOA. That noted, so long as one is willing to compare across experiments from different laboratories, there is a pattern in

Figure 3.3 which suggests against the locational salience hypothesis: When cue - shift stimulus similarity is relatively low (see the data points for 3 and 4 feature differences) we (using variable locations) find almost as much IOR as did Pertzov et al. (2010; using fixed locations).

Forward bias of saccades

The third finding from our experiments was a highly significant forward bias. Although this was seen, statistically, as a main effect of the coordinate space we were testing, we believe that it is appropriate to measure this forward bias using only the baseline locations (to minimize the potential contributions of retinotopic and spatiotopic IOR). Measured this way, the forward biases were 36 and 30 ms in Experiments 1 and 2, respectively. This forward bias could be caused by IOR left behind when gaze shifts to a new location (MacInnes & Klein, 2003), or it might instead be an example of saccadic momentum (Smith & Henderson, 2009; 2011a; 2011b), a behavioral effect that may have it roots in the wiring diagram of the intermediate layers of the superior colliculus (Wang, Satel, Trappenberg, & Klein, 2011). A similar forward bias (21 ms) was present in Mathôt and Theeuwes (2010), but only at the shortest CTOA they tested. Saccades directed to the uncued retinotopic and spatiotopic locations are not precisely forward and return saccades, respectively; rather, in polar coordinates, they are directed 45 degrees away from the forward and return directions. Since IOR does not decay as rapidly as momentum, we tentatively assume that the forward bias is an example of saccadic momentum and that this momentum effect is characterized by a gradient (as has been demonstrated during viewing of complex scenes by Smith & Henderson, 2009) that

includes the forward target locations. Regardless, this effect is methodologically balanced across the cued and uncued locations used to calculate the cue-generated retinotopic and spatiotopic IOR scores that are the focus of this investigation.

Conclusions

While the debate on the link between retinotopic and spatiotopic representations of space remains largely unsettled (*cf* Mathôt & Theeuwes, 2011; Hall & Colby, 2011; Burr & Morrone, 2011, for conflicting theoretical accounts), the primary objective of the present investigation was to resolve *when*, relative to an intervening saccadic eye movement, IOR would exist in spatiotopic coordinates. On this point, the present and extant data on the dynamic remapping of oculomotor IOR are in good agreement: cue-generated IOR exists in spatiotopic coordinates at the earliest possible behavioral measurement following a post-cue saccade, a finding that is consistent with IOR's proposed function as a foraging facilitator.

Author Note

We are grateful to Sebastian Mathot and Jan Theeuwes for sharing their data and code with us. We are also grateful to Yvan Pertzov, Ana Vivas and an anonymous reader for constructive advice. Finally, we thank Julie Golomb for providing general assistance and guidance on the concept of dynamic remapping during the earlier stages of this research project. This research was made possible by an NSERC Graduate Scholarship to Matthew Hilchey and an NSERC Discovery Grant to Raymond Klein.

Table 3.1

Saccadic response times (SRTs) and numbers of trial exclusions for each level of the factors Cueing (cued or uncued) and Coordinates (retinotopic or spatiotopic).

	Retinotopic			Spatiotopic		
	Cued	Uncued	IOR	Cued	Uncued	IOR
SRT (ms)						
Experiment 1	212.2	192.5	19.7	263.3	228.7	34.6
Experiment 2	222.7	205.3	17.4	256.5	234.2	22.3
Trial Excluded (%)						
Experiment 1	15.5	14.2	–	11.0	8.8	–
Experiment 2	17.7	18.1	–	9.4	13.3	–

IOR is the difference score between cued and uncued locations.

Table 3.2

Cue–shift stimulus discriminability on the features of brightness, size, fill, shape, and relative time of occurrence, where the cue and the shift stimulus for the first saccade in each of the experiments listed was the same or similar (S) or was different (D).

Experiment	Task Characteristics					No. of Diff. Features
	Brightness	Size	Fill	Shape	Time	
Pertzov ’10	S	D	D	D	D	4
Mathôt ’10	S	D	S	S	S	1
Exp. 1	D	D	D	S	S	3
Exp. 2	S	D	D	S	S	2

Figure 3.1

The time courses of retinotopic (open symbols) and spatiotopic (filled symbols) inhibition of return (IOR; y-axis) from Pertzov et al. (2010; plotted as squares) and Mathôt and Theeuwes (2010; plotted as triangles). The x-axis refers to the time between the achievement of fixating the shift stimulus and the onset of the target stimulus. The rectangle highlights the apparent discrepancy between Pertzov et al. (2010) and Mathôt and Theeuwes (2010). The data from Pertzov et al. (2010) are restricted to their "opposite-sides" condition; see Footnote 1 for an explanation.

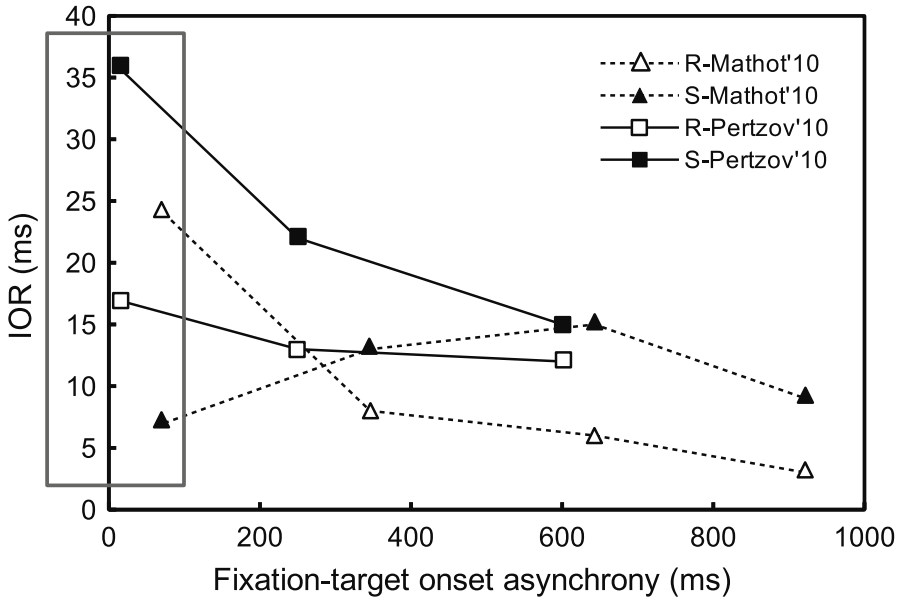


Figure 3.2

(a)–(d) Methods that have been used to explore the time course of retinotopic versus spatiotopic oculomotor IOR: (a)–(c) in Pertzov et al. (2010), and (d) in Mathôt and Theeuwes (2010). In these panels, F indicates the initial fixation on a trial, S the shift stimulus locations, and C the cued locations. Possible target locations are indicated by empty circles or circles with Cs. The circles are shown only for illustrative purposes; they were not present on the screen. (a) The possible locations of fixation, cue, and shift stimuli were fixed in Pertzov et al. (2010). Fixation always started at the center of the screen, and at that time all of the locations that might be stimulated (either by cues, shift stimuli, or targets) were similarly centered at the middle of the screen. (b) and (c) 700 ms following the onset of a cue at one of the four possible cue locations, a shift stimulus was presented at one of two possible locations. (d) In Mathôt and Theeuwes (2010), there were no fixed locations for the different types of stimuli, each of which, more or less, could appear anywhere on the screen. Illustrated here are the possible target locations, given the initial fixation stimulus, shift stimulus, and cue. The target and cue locations were selected randomly with the restriction that neither could be off the screen. (e) Cue and shift stimuli used in Mathôt and Theuwees (2010), Pertzov et

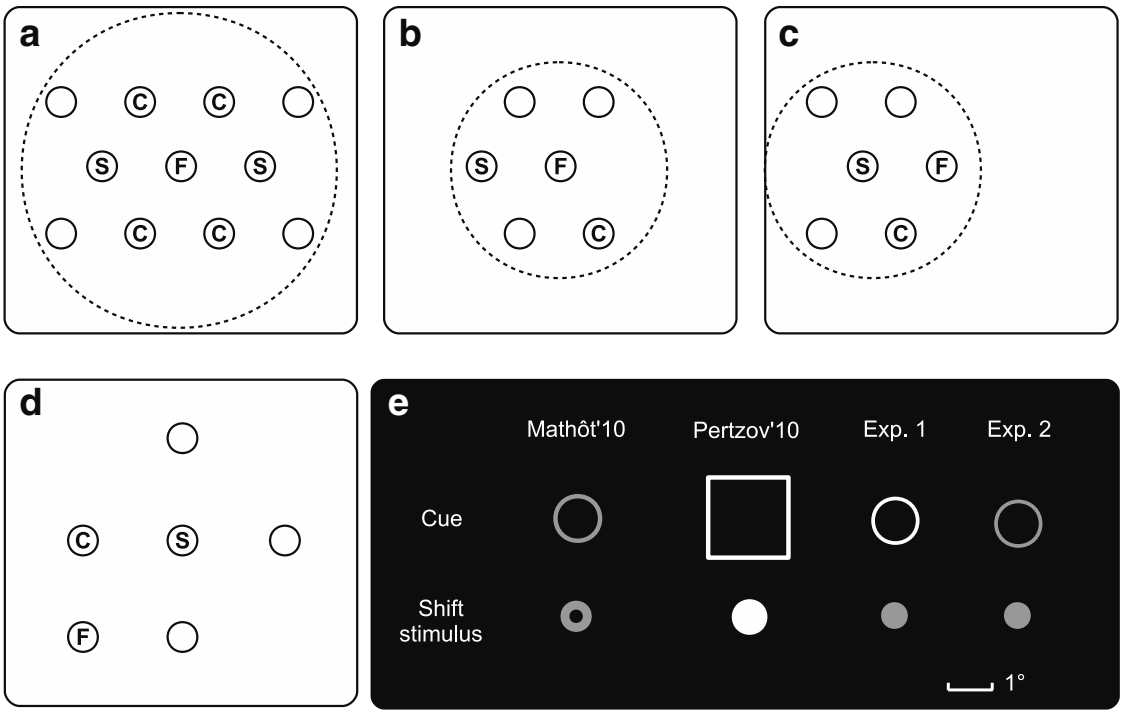


Figure 3.3

IOR scores as a function of cue–shift stimulus distinctiveness (see Table 3.2). Retinotopic IOR is plotted as open symbols, while spatiotopic IOR is plotted as filled symbols. The data from the shortest interval tested by Mathôt and Theeuwes (2010, as reanalyzed here; see Figures 3.A1 and 3.A2) are plotted as triangles, and those from Pertzov et al. (2010) are plotted as squares. Data from the present experiments are plotted as circles. The best-fitting linear functions regressing IOR against log2(-number of different features) are shown for both spatiotopic (solid line) and retinotopic (dashed line) IOR scores.

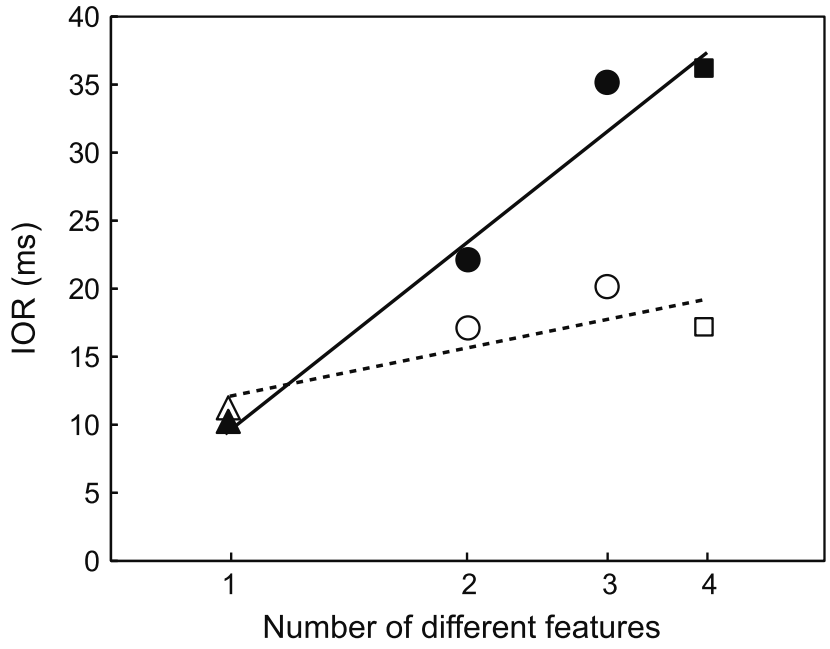


Figure 3.A1

Saccadic response time (SRT) distribution for all of the conditions of Mathôt and Theeuwes (2010). The dashed line shows the lower cutoff used by Mathôt and Theeuwes (2010), while the solid line shows the cutoff that we used in our reanalysis.

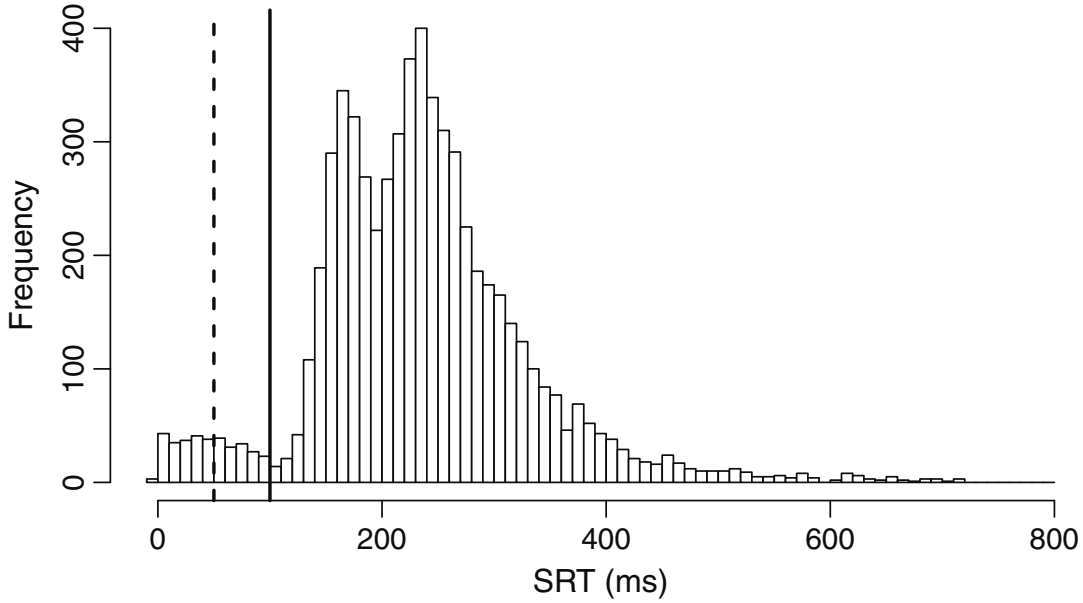
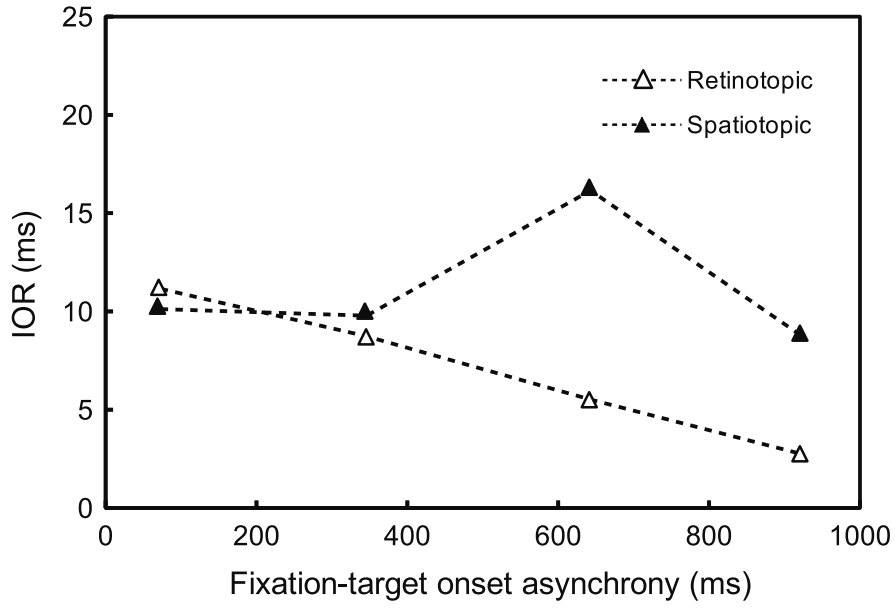


Figure 3.A2

The IOR results from Mathôt and Theeuwes (2010) when target saccadic response times (SRTs) between 0 and 100 ms were excluded. The x-axis refers to the time between the achievement of fixating the shift stimulus and the onset of the target



CHAPTER 4:
ON THE ROLE OF EYE MOVEMENT MONITORING AND DISCOURAGEMENT ON INHIBITION OF RETURN IN A GO/NO-GO TASK

The manuscript based on this study is presented below. Co-authors for this manuscript are Mahmoud Hashish, Gregory H. MacLean, Dr. Jason Satel, Dr. Jason Ivanoff and Dr. Raymond M. Klein, respectively.

Vision Research, Vol 74(6), Aug 2012, pp 1145-1153. http://www.sciencedirect.com/science/article/pii/S0042698913002824

Abstract

Inhibition of return (IOR) most often describes the finding of increased response times to cued as compared to uncued targets in the standard covert orienting paradigm. A perennial question in the IOR literature centers on whether the *effect* of IOR is on motoric/decision-making processes (output-based IOR), attentional/perceptual processes (input-based IOR), or both. Recent data converge on the idea that IOR is an output-based *effect* when eye movements are required or permitted whereas IOR is an input-based *effect* when eye movements are monitored and actively discouraged. The notion that the *effects* of IOR may be fundamentally different depending on the activation state of the oculomotor system has been challenged by several studies demonstrating that IOR exists as an output-, or output- plus input- based effect in simple keypress tasks not requiring oculomotor responses. Problematically, experiments in which keypress responses are required to visual events rarely use eye movement monitoring let alone the active discouragement of eye movement errors. Here, we return to an experimental method implemented by Ivanoff and Klein (2001) whose results demonstrated that IOR affected output-based processes when, ostensibly, only keypress responses occurred. Unlike Ivanoff and Klein, however, we assiduously monitor and discourage eye movements. We demonstrate that actively discouraging eye movements in keypress tasks changes the form of IOR from output- to input-based and, as such, we strongly encourage superior experimental control over or consideration of the contribution of eye movement activity in simple keypress tasks exploring IOR.

Introduction

As explored in a typical covert orienting paradigm, inhibition of return (IOR) refers to the phenomenon of slower response times (RTs) to previously cued locations (for reviews, see Klein, 2000; Lupianez, Klein & Bartolomeo, 2006). The effect of IOR can be separated into two broad classifications or forms: those affecting *output* (motoric or decision-making), and *input* (attentional or perceptual) pathways (e.g., Taylor, 1999; Taylor, & Klein, 2000; Klein & Hilchey, 2011). Two completely dissociable mechanisms underly these forms (*e.g*., Kingstone, & Pratt, 1999; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Bourgeois, Chica, Migliaccio, de Schotten, & Bartolomeo, 2012). Efforts (Chica, Taylor, Lupianez and Klein, 2010; Klein, Hilchey & Satel, 2012) to integrate ideas about when (*cause*; Taylor & Klein, 2000) and how (*mechanism*; Ivanoff, Lupianez & Klein, 2002) these two forms are generated have been made difficult by robustly observed output-based effects in variants of the go/no-go task in which input-based effects are predicted. Our purpose here is three-fold: 1) to assert a particular relation between the activation state of the oculomotor system and the form of IOR, 2) to illustrate a range of data that seems to conflict with this assertion, and 3) to resolve the discrepancy.

On the underlying mechanisms for the effects associated with the two forms of IOR

Taylor and Klein (2000) manipulated the nature (peripheral events; central arrows) of the stimuli that might cause (the first signal; S1) and measure (the second signal; S2) IOR. The 4 possible pairings of S1/S2 were randomly intermixed within each of 6 combinations of response modality for S1 (no response, manual or saccadic

localization responses) and S2 (manual or saccadic localization responses). Their methods and findings are illustrated in Figure 4.1. Whenever an eye movement response was required and IOR caused, the effect was observed whether S2 was a peripheral onset or central arrow. Simply, responses to S2s were slower when the direction indicated by the S1 was compatible with the response required by S2. This pattern implied that the effect of IOR in these conditions was more closely related to delayed responding (i.e., a decision or output-based effect). In contrast, when eye movements were forbidden and withheld during a trial (made neither to S1 nor S2), IOR was only observed in response to peripheral S2s. Because IOR only delayed responding when S2 was a peripheral event (occurring at the *location* indicated by S1), the pattern implied that the effect was closer to the input end of the processing continuum. Taylor and Klein (2000) suggested that the requirement to withhold eye movements altered the activation state of the oculomotor system, fundamentally changing the form of IOR.

Ivanoff, Klein and Lupianez (2002) described two distinct mechanisms (illustrated in Figure 4.2) that might lead to IOR effects on RT. An input-based mechanism delays the accumulation of information linking cued targets with their corresponding responses (Figure 4.2A). This IOR effect would result in a genuine reduction in performance for cued relative to uncued targets (e.g., Hilchey, et al., 2011; Ivanoff & Klein, 2006). In contrast, an output-based mechanism operates as a bias against responses in the direction indicated by the earlier cue (*e.g.,* Posner, Rafal, Choate, & Vaughan, 1985; Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987; Klein, & Taylor, 1994; Ivanoff, & Klein, 2001; Prime, & Jolicoeur, 2009). This IOR effect

would have no effect on the accumulation of information about the target (Figure 4.1B); instead, it increases RT by raising the criterion for responding (Klein, & Taylor, 1994; Ivanoff, & Klein, 2001; Prinzmetal, Taylor, Myers, & Nguyen-Espino, 2011). When such a criterion shift is in effect, delayed responding is accompanied by increased accuracy (Posner, 1975). Simply, the output effect is characterized by a speed-accuracy tradeoff (SAT).

These ideas about the conditions necessary to elicit the two forms of IOR and the two different mechanisms that could slow RT to cued targets were empirically linked by Chica et al. (2010). IOR was generated by a peripheral cue and measured by manual responses in a non-spatial two-alternative forced choice task. When participants were instructed to ignore the peripheral cue and, importantly, given feedback whenever an incorrect eye movement occurred (the condition represented by a dashed, rounded square in Figure 4.1A), there was a genuine decline in performance at the cued location (see the dashed arrow in Figure 4.3). In contrast, when participants made a saccade to the peripheral cue (and back to the original fixation before target onset, the condition represented by a dotted rounded square in Figure 4.1B), the delay in RT at the cued location was accompanied by an improvement in accuracy (viz., an SAT as represented by the dotted arrows in Figure 4.3).

The puzzle: An "output" form in a condition where the "input" form of IOR should exist

Ivanoff and Klein (2001)'s participants performed a go/no-go task wherein a simple keypress response was required for "go" stimuli whereas no response was required for "no-go" stimuli. Providing the first direct evidence for the suggestion that

IOR could manifest as a bias against responding to the cued location (Klein, & Taylor, 1994), they found that false alarms (FAs; *i.e.,* responses to no-go targets) were rarer on cued than uncued trials in the presence of an IOR effect on RT (dotted arrow in Figure 4.4). In the context of our effort to integrate the two mechanisms of IOR with the two forms of IOR, this finding (IOR=an SAT) is problematic because the condition tested by Ivanoff and Klein corresponds to that represented by a dashed, rounded square in Figure 4.1, where an input-based effect ought to have been observed.

This anomalous finding cannot be dismissed as a fluke. We found five, post 2001 papers[7] using a go/no-go task in which cuing effects on FA rates could be examined using methods similar to those of Ivanoff and Klein (2001). In each of these studies, IOR was expressed as an SAT. The consistency of this SAT is illustrated in Figure 4.5. As would be expected given that each of the false alarm effects were significant, the 95% confidence intervals for each study excludes zero. The overall effect is illustrated at the bottom of the figure.

Typical of most studies measuring IOR with keypress responses, Ivanoff and Klein (2001) did not monitor eye movements. Importantly, in none of the studies represented in Figure 4.5 did participants receive trial-by-trial feedback on their oculomotor behavior. This is in sharp contrast to the assiduous feedback that was provided in the experiments by Taylor and Klein (2000) and Chica et al. (2010). We thought this difference might be critical (cf Klein & Hilchey, 2011) and, when reviewing the literature to identify all studies exploring IOR using a go/no-go task, we found one

[7] These are: Ivanoff and Klein (2003, E1 and E2, no mask data only), Ivanoff and Klein (2004, E1), Prime and Ward (2006, E3), Prime and Jolicoeur (2009, E1 Hi probability of go target), Taylor and Ivanoff (2003).

that supported our conjecture. Using a go/no-go task, Cheal, Chastain and Lyon (1998, Experiment 3) monitored eye position and provided feedback to their observers whenever eye movements were detected.[8] As would be expected from the theoretical integration presented in the previous section, a robust IOR effect was obtained in RT (26 ms) while there was no evidence for an SAT: the FA rates did not differ between conditions (15.6% for uncued trials; 15.4% for cued trials). Miss rates were substantial enough in this experiment to permit the computation of d' which was also nearly identical for the cued (2.109) and uncued (2.114) conditions.

If our conjecture were correct, then when – in conditions akin to those used by Ivanoff and Klein (2001) – the eye movements of observers are actively discouraged, the effect of IOR ought to be of the input form. To test this conjecture we will replicate the methods of Ivanoff and Klein (2001) while: 1) adding eye movement monitoring, 2) providing explicit error feedback when untoward gaze shifts were detected, and 3) excluding any such eye movement trials from analysis. If the active discouragement of reflexive saccadic eye movements to peripheral visual events is the critical factor responsible for an input-based IOR effect, we should observe IOR in RT but, in contrast to Ivanoff and Klein (2001), we should *not* find an accuracy advantage at the cued location.

Although we no longer have the raw data from Ivanoff and Klein we do have the mean RTs and error rates for each participant. This will allow us to make a direct statistical comparison of the pattern of results with (the present study) and without

[8] From this experiment's methods section: "If an eye movement occurred immediately before, during or immediately after the trial, the subject was admonished by the experimenter."

(Ivanoff & Klein) active discouragement of eye movements. The critical results (identical cuing effects on RTs accompanied by significantly different in cuing effects on FA rates) are foreshadowed in Figure 4.4.

Methods

Participants

Fourteen students (10 females and 4 males) from Dalhousie University volunteered for participation and were compensated with course credit. All participants were naïve to the purposes of the experiment and reported normal or corrected-to-normal vision.

Apparatus and stimuli

Participants were tested under dim lighting conditions with stimuli presented at a viewing distance of 57 cm. Stimuli were presented in black against a white background on a 19" CRT monitor connected to an Intel Core Duo processor. An Eyelink II head-based eye monitoring system was used to monitor eye position.

The stimuli used in this experiment were as similar as possible to those administered by Ivanoff and Klein (2001). Three landmark squares (black outline with white fill), each measuring 1.5 x 1.5 degrees visual angle, formed an imaginary horizontal plane at the midpoint of the monitor. The observer's midline was approximately in line with the center of the middle placeholder. The distance between the lateral side of the middle landmark square and the inner lateral side of the peripheral landmark squares was 6.2 degrees visual angle. The fixation point was a small filled dot

(0.3 degrees visual angle in diameter) centered in the middle landmark square. The cue was a "+" or "x" symbol embedded in and encompassing the entirety of a circle (1.5 degrees visual angle in diameter). The go and no-go signals measured 1.5 x 1.5 degrees visual angle and were solid black, and checkered black and white squares, respectively.

Procedure

The procedure for this experiment was identical to that in Ivanoff and Klein (2001) except: 1) the location of the non-responding hand relative to target events was not manipulated (this is in conformity with the remaining studies represented in Figure 4.5); 2) a trial abruptly terminated if an eye movement was detected (Eyelink default settings: velocity threshold = 35°/sec and acceleration threshold = 9500°/sec^2); and 3) such termination was followed immediately by visually presented feedback: "Eye movement detected. Please refrain from making any eye movements." A keypress response was required to acknowledge the feedback and to re-initiate the trial sequences. The sequence of events (on non-terminated trials) was as follows: At the outset of each trial, three landmark squares and the fixation point appeared for 750 ms when the fixation point was extinguished and a cue occurred randomly in one of the three landmark squares for 375 ms. Ninety or 675 ms after the cue's disappearance [cue-target onset asynchronies (CTOAs) of 465 or 1050 ms, respectively], a go or no-go signal appeared randomly in one of the three landmark squares. Observers were instructed to withhold or make single keypress responses (either the "/" or "z" key with the left or right index finger, respectively) on no-go or go trials, respectively. The go and no-go signals appeared onscreen for a maximum of 1 s or until a response was detected. A

failure to respond was recorded as a miss; a FA was recorded if a response occurred during the no-go target. Following termination of the trial a blank white screen appeared for 750 ms (the inter-trial interval). The next trial was initiated following the inter-trial interval.

Design

The ratio of go to no-go trials was 2:1. Each observer participated in one practice block containing 54 trials followed by 4 experimental blocks, each containing 108 trials. Half of the observers were randomly selected to respond to go signals with the left index finger ("/" key) whereas the other half responded with the right index finger ("z" key). Observers were correctly informed that there was no spatial relationship between the location or identity of the cue the subsequent go or no-go signal. Speed and task-appropriate responding were emphasized and eye movements were explicitly discouraged.

Methods of analysis

One observer was excluded from analysis for making almost 50% FAs on no-go trials (the next highest rate was 12.5%). All practice trials were excluded from analysis. All trials with a failure to maintain fixation at any point during the trial were excluded from analysis. Less than 1% of the trials were excluded because the observer either made a keypress response before target onset or failed to make a response to a go signal. Two correct RTs less than 200 ms were excluded from analysis. Because we are not interested in the same questions that Ivanoff and Klein (2001) were pursuing, our analyses will be constrained to the critical question for present purposes which is about

the effect of peripheral cuing upon RT and FA rates[9] and a comparison of these results against those of Ivanoff and Klein. That comparison will be made using logistic regression. A more complete presentation of the results can be found in Appendix A).

Results

A failure to maintain fixation at any point during a trial (see methods) resulted in the termination of 19.7% of the trials. Comparison with some of our other studies using similar feedback about unwanted eye movements reveals that this relatively high rate of unwanted eye movements was likely due to our low threshold for detecting them[10].

RTs following peripheral cues were significantly slower [$t_{12} = 2.99$, p=.011] for cued (422 ms) than uncued peripheral targets (408 ms). FA rates did not differ between these two conditions [$t_{12} = .51$, ns][11]. In point of fact, there were approximately 1% **more** FAs for cued (8.4%) as compared to uncued (7.4%) targets (see Appendix A for more detailed results).

Because the IOR effect in RT observed in the present study was almost identical to that reported by Ivanoff and Klein (2001;14.02 ms vs 13.74 ms, respectively) no statistical comparison was performed on these effects.

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[9] For consistency with the literature our primary analyses will be of the untransformed percentages. However, following the recommendations of Dixon (2008), we also analysed the FA data after conversion to log odds using the formula:

log odds = ln (pc/(1-pc)); where pc=(1 minus the proportion of FAs)

To avoid any divisions by zero, in any cells for which there were no FAs, following convention, it was assumed that rate of FAs was 1/64 (or 1/2 way between zero and the minimum number possible).

[10] Many fewer unwanted eye movements were detected in the manual-response experiments of Chica et al. (2010) whose threshold was much higher (to be detected an eye movement had to exceed 2 degrees of visual angle) than used here and a similar rate of unwanted eye movements were detected in Hilchey, Klein and Satel (2013, Experiment 2) who used the same threshold as we did here.

[11] This finding was replicated when we analyzed log odds of the FA rates.

To compare the cued and uncued FA rates from the two experiments these rates were subject to logistic regression analysis. The LMER package with R Environment for Statistical Computing was used to compute two LMMs (M1 & M2; see Table 4.1), with participant as a random effect and study and cue condition as fixed effects (R Development Core Team, 2011). The only difference between the two models was that M1 did not include the interaction as a fixed effect whereas M2 did. If eye tracking does not affect error rates in the IOR task the interaction should be unimportant and the simpler M1 should yield a lower or comparable AIC score. M2 (AIC = 1683.3) fit the dataset much better than M1 (AIC = 1674.5, with an AIC score reduction of 8.8. Thus, M1 falls somewhere between having considerably less support than M2 and having essentially no support given M2 (Burnham & Anderson, 2004). Within M2, the interaction between study and cue condition was highly significant ($Z= 3.285$, $p<.00102$; See also Figure 4.6)[12]. Given that the crucial difference between Ivanoff and Klein's (2001) experiment and the present one concerns whether eye movement activity was monitored and controlled we believe that observers in the present study were suppressing the natural tendency to make eye movements to peripheral stimuli while, in the absence of feedback, participants in the Ivanoff and Klein experiment were not.

Discussion

Consistent with the hypothesis that the effect of IOR is on input pathways when oculomotor responses to peripheral visual stimuli are actively discouraged (Taylor, & Klein, 2000; Chica et al., 2010), FA rates observed here were similar if not greater for

[12] When we compared the cueing effects from these two studies (uncued minus cued FA rates) using a 2-sample t-test, the difference remained significant ($t30 = -2.06$, $p<.05$).

peripherally cued as compared to uncued no-go signals in the presence of delayed responding for cued relative to uncued go-signals. This pattern of results (represented by the dashed arrow in Figure 4.4) is qualitatively different from the output-based IOR effects reported by Ivanoff and Klein (2001, represented by the dotted arrow in Figure 4.4) as well as from the studies we have found on this topic. All such studies, like Ivanoff and Klein, failed to actively discourage eye movements. Our results are consistent with the only published study we were able to find using a go/no-go task in which eye movements were actively discouraged (Cheal et al., 1998) and with the Chica et al. (2010) investigation dedicated to evaluating the effect of saccadic eye movements on the form of IOR in a non-spatial two-alternative forced choice task.

The reader might wonder[13] whether the genuine improvement that we and Cheal et al. (1998) observed while discouraging eye movements and, conversely, the SAT that is generally observed when eye movements are not discouraged (see Figure 4.5) are due to the actual eye movements made in these two conditions or to the mental set that eye movements are forbidden or permitted. There are several reasons why we are confident that mental set explains the dissociation. First, consider that the maximum possible rate of trials with unwanted eye movements in Prime and Jolicoeur (2009, Experiment 1) was about 19% (total % of trials excluded for eye movement artifacts or for responses being too fast, too slow or erroneous). Yet the SAT that they observed was similar to, if not larger than, that reported by Chica et al. (2010) when participants were instructed to

[13] We thank an anonymous reviewer for raising this concern. The same reviewer also suggested that "it is essential to replicate the current methods without providing feedback on eye movements". We disagree. All studies illustrated in Figure 4.5 demonstrated output-based forms of IOR in the absence of express feedback discouraging oculomotor responding, including Ivanoff and Klein (2001) whose methods we have closely replicated while adding eye movement feedback.

make eye movements on every trial. Second, the behavioral data in the two EEG studies by David Prime that we have reported in Figure 4.5 were generated after excluding trials with obvious eye movement artifacts (Prime, personal communication). This provides direct evidence that the SAT is not dependent on trials with eye movements. Finally, we know it is not the occurrence of eye movements per se that is responsible for the SAT form of IOR because an input form of IOR is generated by anti-saccades (e.g., Rafal, Egly & Rhodes, 1994; Khatoon, Briand, & Sereno, 2002; Fecteau, Au, Armstrong, & Munoz, 2004; Abegg, Sharma, & Barton, 2012). As we explain elsewhere (Klein, Hilchey, & Satel, 2012; Klein & Hilchey, 2011), the form of IOR that is generated does not depend on the occurrence of eye movements *per se* but rather on whether the system responsible for reflexive saccades is inhibited (as it needs to be to perform accurately in an anti-saccade task; Forbes & Klein, 1996) or not inhibited during the trial when IOR is being measured. The neurophysiological data from anti-saccade experiments requiring controlled eye movements directed to locations opposite the source of stimulation are clear in demonstrating suppression over the primitive midbrain structures responsible for reflexively-generated saccades at the cellular level (Everling, Dorris, Klein, & Munoz, 1999; Ignashchenkova, Dick, Haarmeier, & Thier, 2004).

As demonstrated here and elsewhere, the effects of IOR are dissociable on the basis of whether the task is likely to recruit the oculomotor circuits responsible for reflexively-generated saccades (for review see Klein & Hilchey, 2011). This dissociation poses challenges when it comes to integrating the extant data on IOR into a unified and coherent theoretical framework primarily because the activation state of the oculomotor

system is so often unknown in simple keypress RT tasks. More specifically, little effort is made in experimental designs to discourage untoward oculomotor response activation in covert spatial orienting paradigms. For example, Zhao, Heinke, Ivanoff, Klein and Humphreys (2011) provided some evidence that IOR affects both input- and output-processing in a discrimination task requiring only keypress responses, a result that seemed to challenge the notion that output- versus input-based effects are determined by the activation state of the oculomotor system. Yet, in that study, the presence of eye movements was not monitored let alone discouraged. This failure to discourage oculomotor responding raised the specter that the oculomotor system was shifting in and out of activation states throughout the task. Such phasic activation would yield data consistent with a two-component theory. On any given trial, however, only one form of IOR may have been in effect. It is precisely this ambiguity, rooted in the fact that the effects of IOR in keypress tasks are distinct depending on the activation state of the oculomotor system, that compels us to urge investigators of IOR to ensure adequate control over, and measurement of the activation state of, the oculomotor system. Simply, and more to the point, it is clear that research objectives dedicated to evaluating the nature of covert orienting should actively discourage oculomotor responding.

Table 4.1
LMM analyses of error rates across cue conditions in two studies.

	Probability of error			
	B	SE	*z*	Pr(>|*z*|)
Model 1				
Intercept	−3.42886	0.23078	−14.858	
Study (Ivanoff and Klein vs. Hashish et al.)	−0.41638	0.22943	−1.815	0.0695
Cueing (Cued vs. Uncued)	0.39599	0.07453	5.313	1.08E−07
Model 2				
Intercept	−3.42572	0.23122	−14.816	
Study (Ivanoff and Klein vs. Hashish et al.)	0.47057	0.23122	2.035	0.04184
Cueing (Cued vs. Uncued)	0.21680	0.09254	2.343	0.01914
Interaction (Cueing ∗ Study)	0.30396	0.09254	3.285	0.00102

Figure 4.1

A schematic illustration of the methods and results from Taylor and Klein (2000). Six experiments differed in terms of the localization task observers were required to perform in response to S1 (none, manual, saccadic) and S2 (manual, saccadic). The rows and columns within each box represent the nature of the stimuli (peripheral luminance changes and central arrows) that were randomly intermixed in each block of trials. Solid circles represent conditions in which significant IOR was obtained. IOR was not observed in the remaining (dotted) circles. The gray region illustrates the conditions for which Taylor and Klein inferred an "input" form of IOR that was characterized by a delay in attending peripheral inputs or linking them with their correct responses. The black region represents the conditions for which Taylor and Klein inferred a "motoric" form of IOR that was characterized by a bias against responding in the originally cued direction. The conditions highlighted by red and green boxes (in the on-line version and which are rendered using dashed and dotted lines, respectively, in the print version) are discussed in the text.

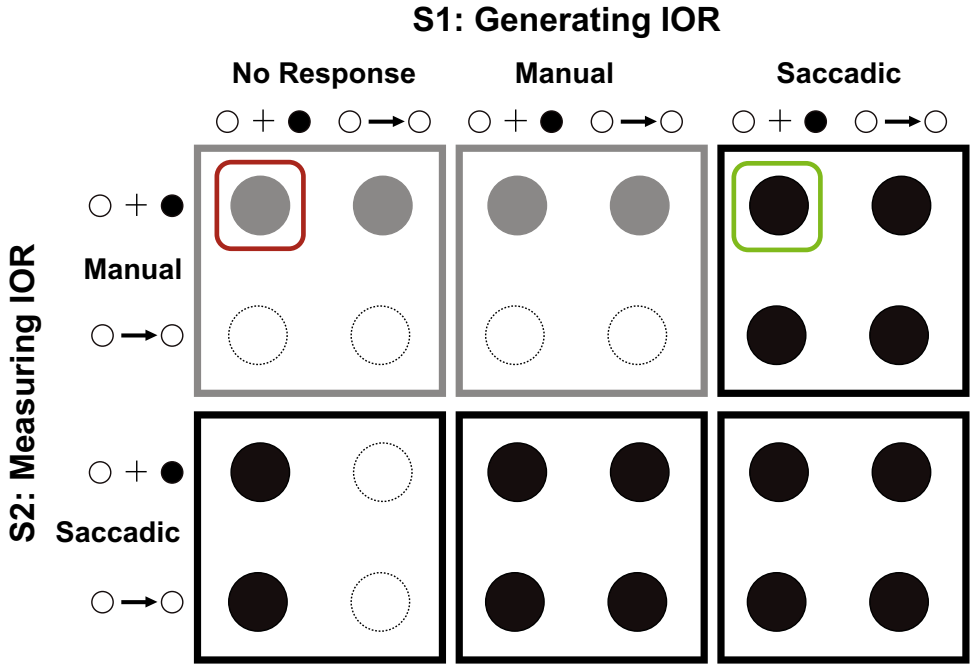


Figure 4.2

Two accounts for how IOR might slow response times. The temporal dynamics of information processing is illustrated in both panels by SAT functions with accuracy plotted as a function of RT. The solid function represents the monotonic accumulation of information needed to make a correct response to the target, and the solid horizontal line represents the average criterion amount of evidence the observer requires to initiate a response. According to the input-based account (panel A) IOR delays the accumulation of task-relevant information (cf Hilchey et al., 2011) as represented by the dotted SAT function. The typical effect of input-IOR on performance (a genuine improvement in speed, or accuracy, or both) is represented by the red/dashed arrows. According to the output-based account (panel B) IOR increases the amount of evidence required to initiate a response (dotted horizontal line. The typical effect of output-IOR (slower and more accurate responding; viz a speed–accuracy tradeoff) is represented

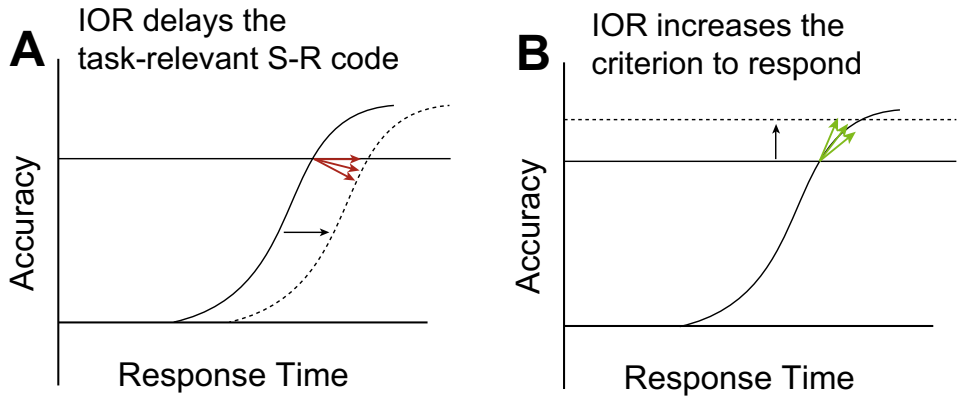


Figure 4.3

Results from Chica et al.'s (2010) manual discrimination task plotted in speed–accuracy space (see Fig. 4.2). Data from the experiment when participants were instructed to ignore the cue are plotted in the left panel; data from the experiments when participants made an eye movement to the cue (and back to fixation) are plotted

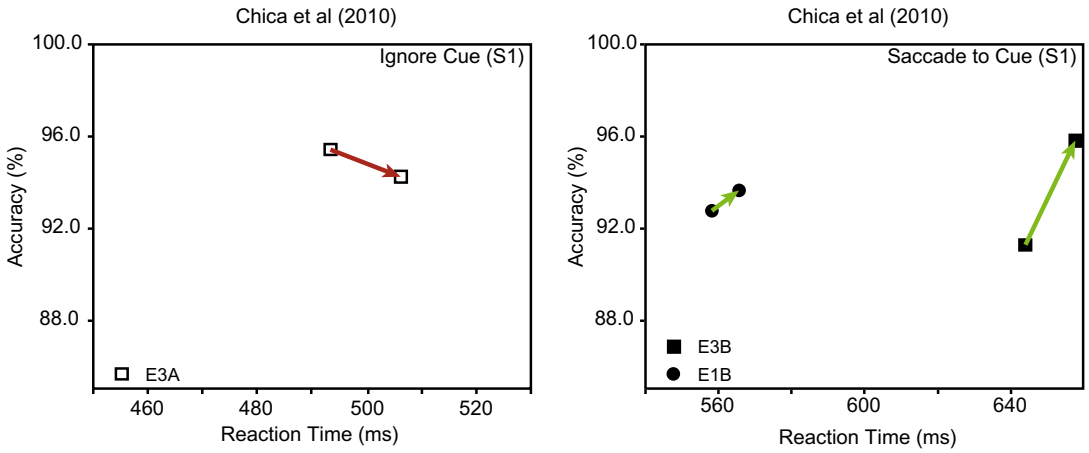


Figure 4.4

The results from peripheral targets in Ivanoff and Klein (2001; green/dotted arrows in this illustration) and the present experiment (red/dashed arrows) represented in speed–accuracy space (see Fig. 4.2). The data from both experiments have been collapsed across CTOA, T-R correspondence, and in the case of Ivanoff and Klein, position of the non-responding effector). In both experiments the faster data point is from peripheral targets preceded by a peripheral cue at the opposite location; the slower data point is from peripheral targets preceded by a cue at the same location as the target; in both cases the IOR effect in RT was 14 ms. The dramatic difference is in accuracy with a speed– accuracy tradeoff in Ivanoff and Klein and a genuine change in the quality of performance in the present study.

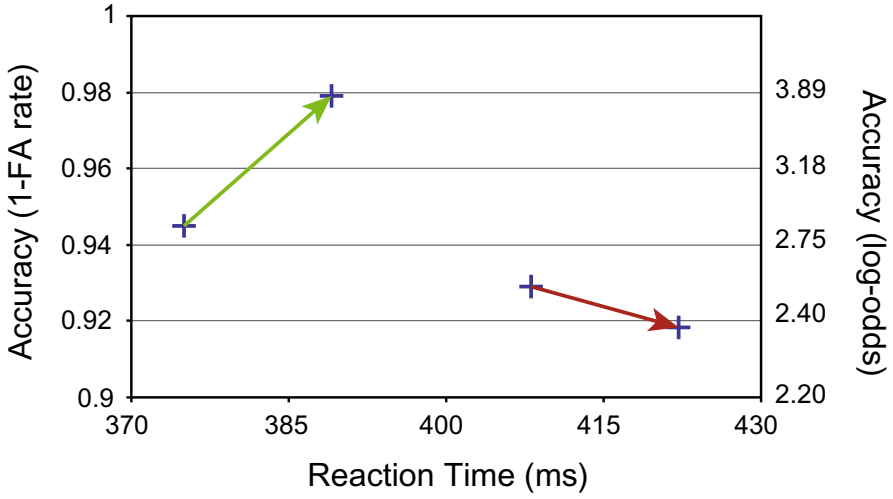


Figure 4.5

This forest plot was generated using the metafor package (Viechtbauer, 2010) within R version 2.12.1 (R Development Core Team, 2011). Each symbol in the upper panel of the figure represents the data from one of the 6 studies in the literature that explored IOR using a go/no-go task, reported separately the FA rates for cued and uncued no-go targets, and did not provide trial-by-trial feedback on eye movements. The X-axis projections of the symbols in the upper panel of the figure represent the mean FA rate difference: Uncued FA minus Cued FA. The sizes of these symbols are positively related to the number of participants in each study. Studies were weighted equally to generate an estimate of the overall effect of cuing upon FA rates which is illustrated by the diamond (whose width represents the 95% confidence interval) in the bottom portion of the figure. Mean cuing effects and 95% confidence intervals for each of the six studies

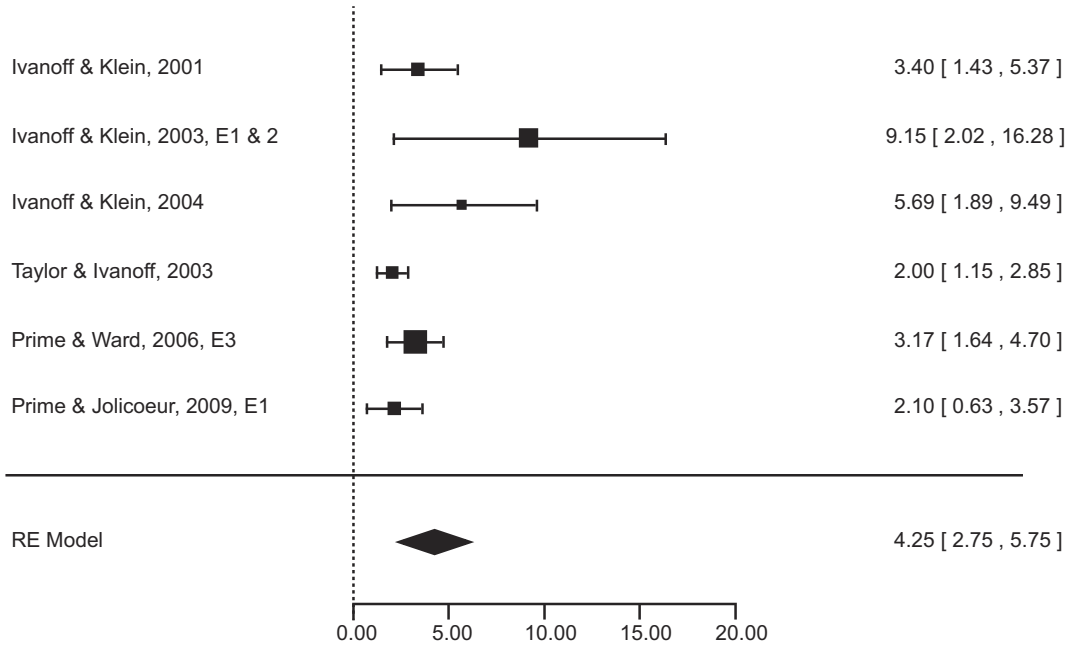
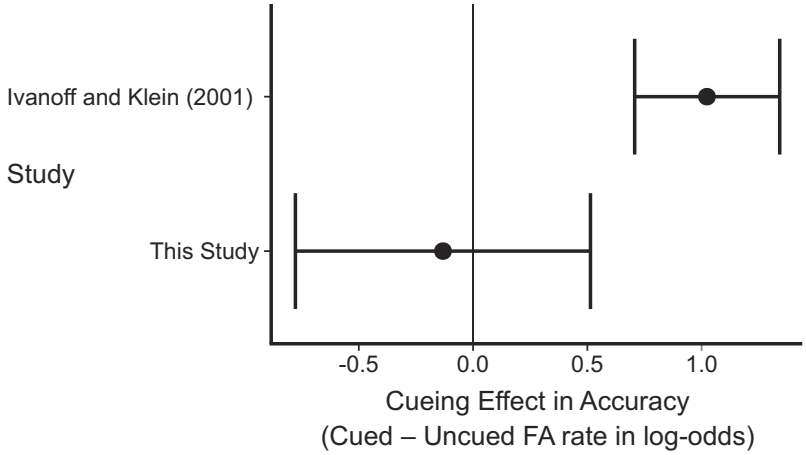


Figure 4.6

The effect of cues on FA rates in the present experiment (left) and in Ivanoff and Klein's (2001) previous investigation (right) computed from the predicted values of linear mixed model "M2". The ezPreds function from the ez package within the R Environment for Statistical Computing (R Development Core Team, 2011) was used to compute the predicted values (Lawrence, 2012). Ivanoff and Klein's participants produced significantly fewer FAs when targets appeared at the cued location than when they appeared at the uncued location. This effect differed significantly from the present study, wherein cues did not influence FA rates. Error bars represent 95%



CHAPTER 5:
ARE THERE MULTIPLE FORMS OF INHIBITION OF RETURN? IT DEPENDS ON HOW YOU LOOK AT IT.

The manuscript based on this study is presented below. Co-authors for this manuscript are Deniz Dohman, Dr. Nathan Crowder and Dr. Raymond M. Klein, respectively.

Canadian Journal of Experimental Psychology/Revue canadienne de psychologie experimentale, advance online publication. http://psycnet.apa.org/index.cfm?fa=buy.optionToBuy&id=2015-55835-001

Abstract

Two important diagnostics have been used to determine whether the effect of inhibition of return, when preceded by a saccade, is primarily upon input (*i.e.,* attentional/ perceptual level) or output (*i.e.,* response/decision level) processes. Data from anti-saccade paradigms suggest input effects whereas data from manual responses to centrally presented arrow targets suggest output effects. Here, we combine these diagnostics to resolve the discrepancy. In separate conditions participants made a pro- or anti-saccade to a peripheral stimulus. Upon returning gaze to the original fixation, left and right manual responses were made to left- and right- pointing arrows at fixation, respectively. The primary objective of the pro-saccade condition was to determine whether an eye movement toward a visual stimulus that was not associated with a manual localization response would bias spatially compatible manual responses against the prior saccade vector. Manual responses were slowest in the direction of the prior saccade, consistent with an output-based attribution (e.g., Posner, Rafal, Choate and Vaughan,1985). The primary objective of the anti-saccade condition was to determine whether an eye movement away from a visual stimulus would also bias subsequent manual responses. No apparent response bias was detected, consistent with an input-based attribution (e.g., Fecteau, Au, Armstrong, & Munoz, 2004). Collectively, the findings indicate that there are two, dissociable forms of inhibition depending on saccadic response demands. Converging evidence from other paradigms is discussed.

Introduction

A putative inhibitory aftereffect of visuospatial orienting was first explored by Posner and Cohen (1984) and later named "inhibition of return" (IOR) by Posner, Rafal, Choate and Vaughan (1985). IOR is commonly studied using variations of the model task developed by Posner (Posner, 1980; Posner & Cohen, 1984). In a typical implementation, observers fixate on a central location commonly demarcated by a box ("fixation") amid two others, one to the right and the other to the left of fixation. The first event (S1) is the presentation of a stimulus that is uninformative about the location or direction of the subsequent stimulus (S2). The observer may be instructed to ignore (Posner, & Cohen, 1984) or respond (Vaughan, 1984; Maylor, & Hockey, 1985) to S1 but is informed correctly that the location of S1 is uncorrelated with the location or direction of a second event (S2). S2 serves as a response stimulus [commonly called the "target"] from which the effect of S1 is measured. The cue-target onset asynchrony (CTOA) is often held constant at ~1 s, an interval sufficiently long for observing IOR: operationally, the costs in processing that occur when the location indicated by S1 is shared by the location of (Posner, & Cohen, 1984; Posner, Cohen, Choate, Hockey, & Maylor, 1984; Posner, Cohen, & Rafal, 1982) or response to (Posner, Rafal, Choate, & Vaughan, 1985; Rafal, Calabresi, Brennan, & Sciolto, 1989) S2.

This inhibitory phenomenon has attracted considerable interest in part because of IOR's presumed role as a novelty seeking mechanism (Posner & Cohen, 1984; Posner, Rafal, Choate, & Vaughan, 1985) that can facilitate foraging behaviour (Klein, 1988) and because of studies pointing to its neural implementation (Dorris, Klein, Everling, &

Munoz, 2002; Fecteau & Munoz, 2005; Mirpour, Arcizet, Ong, & Bisley, 2009; Sapir, Soroker, Berger, & Henik, 1999) in the machinery mediating oculomotor behaviour. One of the enduring questions for scholars interested in this phenomenon is "What is inhibited in inhibition of return?" (e.g., Reuter-Lorenz, Jha, & Rosenquist, 1996): Is the delay in responding due to effects operating upon “input” (i.e., attentional/perceptual level) or “output” (i.e., motor/decision level) processes (see next paragraph)? The interested reader is referred to Taylor and Klein (2000) for empirical evidence for both types of effect, and Ivanoff, Klein and Lupianez (2002), for theoretical interpretations.

In the context of this report, we use "input" and "output" to refer to effects that occur early as opposed to late in the sequence of processes between an imperative stimulus and the response to it. Whereas, by definition, input effects begin with sensory processing there are reasons to believe that IOR's input effects are not due to delayed sensory processing generally because, for example, IOR has no effect on perceptual tasks like temporal order judgments or illusory line motion (Posner & Cohen, 1984; Maylor, 1985; Schmidt, 1996). Taylor and Klein (2000, p. 1652) identified two alternative early, yet post-sensory, effects that might be generated when the eyes remain fixed: "IOR operates specifically at the interface of visual processing and manual programming or execution...[or]...IOR reflects slowed reflexive reorienting of covert attention." Either of these possibilities would be consistent with the input form of inhibition described by Ivanoff, Klein and Lupianez (2002) under which "activation of the task-relevant response code is delayed." The output form of inhibition,as first discovered by Posner et al. (1985), was defined by them as "inhibition of return" (see

also Hilchey, Klein & Satel, 2014). It was later described by Klein and Taylor (1994) and Ivanoff et al. (2002) as a response bias which, importantly, is more closely related to decision making than to post-decision motor programming processes.

Two important diagnostics have been used in the IOR literature to determine whether IOR's effects are primarily input- or output- based. One diagnostic, pioneered by Abrams (Abrams & Dobkin, 1994a) and Rafal (Rafal, Egly & Rhodes, 1994, Experiment 4) measures the inhibitory aftereffect of a peripheral stimulus using saccadic responses to centrally presented arrows. Since a centrally presented arrow target requires a spatial response but does not repeat the pathway of the earlier peripheral stimulus, a delay in responding in the direction of the original stimulus cannot be due to a delay in detecting the target. An inhibitory aftereffect of a cue when measured by a central arrow stimulus is therefore interpreted as a post-sensory effect operating at the decision (response bias) or output stage of processing.

Another diagnostic, first used by Rafal et al. (1994) requires the participant to make a movement in the opposite direction of a stimulus. When the response is an eye movement, this is called an anti-saccade. The anti-saccade might be used to generate or to measure inhibitory aftereffects. To see how this diagnostic works, consider the former case in which the participant first makes an anti-saccade to a peripheral target and then is required to make a compatible (pro-) saccadic response to a subsequent peripheral target. If the location to which an anti-saccade was made generated a response bias against the response vector, responding would be slowest when S2 appeared opposite S1 On the other hand, if the original stimulus – and not the saccade vector – generated an input

effect (as described above) then responding would be slowest when S2 and S1 occupied the same location. As we will show in the next two sections, the results from these two diagnostics have generated discrepant conclusions.

Saccadic responding generates output-based IOR effects: Data from the central arrow diagnostic

In the late 1990s, Taylor and Klein (1998; 2000) set out to determine the contribution of input- and output- based processes to IOR by systematically investigating factorial combinations of responding to S1 (no response, manual localization or saccade localization) and S2 (manual localization or saccade localization) in the model task. These combinations yielded six unique conditions. In each of these conditions, visual stimuli were randomly either transient perturbations in peripheral vision or centrally presented arrows (pointing left or right). As instructed, the observer made saccadic and manual localization responses to peripheral visual stimuli and direction-compatible responses to central arrows. In all six conditions, the CTOA was held constant at 1 s and five hundred milliseconds after the onset of S1, the fixation box brightened (i.e., a fixation cue; Pratt, & Fischer, 2002) to return gaze and/or attention back to fixation before the presentation of S2, which appeared 1 second after the onset of S1.Taylor and Klein demonstrated that the effect of S1 on S2 processing depended critically on the response characteristics of the tasks. Whenever a saccade was required to either S1, S2 or both signals, approximately equivalent inhibition was observed whether S2 was a central arrow or peripheral onset. This pattern was interpreted as evidence for an effect of IOR nearest the output end of the processing continuum

(Posner et al., 1985; Klein, & Taylor, 1994) because, while there was no repeated stimulation along the input pathway of S1, responding was delayed whenever the response required by S2 was compatible with that required (e.g., saccade - manual) and/or indicated (e.g., no response - saccade) by S1. In contrast, inhibitory effects in the no eye movement conditions (i.e., no response - manual or manual - manual) were absent when measured by centrally presented arrows. Instead, inhibitory effects were observed only when a peripheral onset S2 appeared at the location indicated by S1. Based on the overall pattern (see Figure 5.1), Taylor and Klein (2000) concluded that there must be two dissociable forms of IOR, and that which form is generated "...depends critically on whether or not saccades are required ..." (Taylor & Klein, p. 1652). When eye movements were required the effect was purely decisional or output related (i.e., could be measured either by an arrow S2 at fixation commanding a response compatible with the direction indicated by S1 or by an onset S2 at the location indicated by S1). When eye movements were not required (and not made), the effect was input-based (i.e., could *only* be measured by a stimulus in peripheral vision at the location indicated by either the arrow or onset S1).

Saccadic responding generates input-based IOR effects: Data from the anti-saccade diagnostic

The idea that the effect of IOR is purely on output processes when saccadic eye movements are required is challenged by studies exploring IOR when generated and/or measured by an anti-saccade. In such tasks, whether, in response to S1 and S2, respectively, an anti- and pro-saccade are required (e.g., Abegg, Sharma, & Barton,

2012; Barton, Goff, & Manoach, 2006; Fecteau, Au, Armstrong, & Munoz, 2004), a pro- and anti- saccade are required (e.g., Fecteau et al., 2004; Rafal, Egly, & Rhodes, 1994), an anti- and an anti- saccade are required (e.g., Fecteau et al., 2004) or no response and an anti-saccade are required (Khatoon, Briand, & Sereno, 2002; Rafal, Egly, & Rhodes, 1994), responding is almost always (see Barton, Goff & Manoach, 2006, for an exception) slowest when the cue and target occupy the same location. The nearly ubiquitous finding that responding is slowest when the cue and target traverse the same input pathway when anti-saccades are required implies an effect nearer the input end of the processing continuum (e.g., Fecteau, Au, Armstrong, & Munoz, 2004).

Resolving the discrepancy

Our purpose in this paper is to resolve the discrepancy between these two diagnostics. One possibility is that Taylor and Klein's (2000) finding that an output form of IOR had been generated by a prosaccade may have been challenged by their random intermixing of responses to arrow and peripheral S1s and S2s. The other possibility is that Taylor and Klein's (2000) findings are not so challenged but their conclusion that the input form of IOR is observed "only when eye movements were not made" must be qualified. In the remainder of this section, we elaborate on these two ways to resolve the discrepancy.

Taylor and Klein (2000)'s findings in their "saccade-manual" conditions (see Figure 5.1) suggest that an oculomotor response to either an arrow or peripheral S1 will bias subsequent manual responding against the prior saccade vector 1 second later, consistent with the canonical, output-based, account of IOR (Posner, Rafal, Choate &

Vaughan, 1985). An alternative account, however, is made possible by the fact that the two types of imperative stimuli (central arrows and peripheral luminance changes), for both the first (saccade) and second (manual) responses on a trial, were randomly intermixed. Under this account, the delayed manual responding in this "saccade-manual" condition was not caused by the oculomotor response per se, but rather by the active suppression of the task-irrelevant manual response activation elicited by S1 (see also, e.g., Harvey, 1980; Maylor & Hockey, 1985). According to the activation-suppression model (e.g., Eimer, 1999; Ridderinkohf, 2002; Burle, van der Wildenberg, & Ridderinkohf, 2005; Wijnen & Ridderinkohf, 2007; Hilchey, Satel, Ivanoff & Klein, 2013), a reversal of the standard, facilitatory spatial orienting effect in the model task would be expected at later CTOAs. This point is made succinctly by Burle et al (2005, p. 623):

> "the cue first transiently activates the associated response. For short [CTOAs], this leads to a direct positive congruency effect, characterized by slower RTs [response times] to the target on incongruent trials compared to congruent trials. However, as time passes, this activation is selectively suppressed, as becomes evident with long [CTOAs]. Consequently, if cue and target are separated by a long delay, the response associated with the cue will be disfacilitated [sic]."

The activation-suppression model is a viable account for the data from Taylor and Klein (2000)'s "saccade-manual" design because *both* manual localization responses and pro-saccadic oculomotor responses were associated with arrow and peripheral onset S1s. The manual response association occurred because the oculomotor S1s doubled as manual S2s. According to the activation-suppression model, delayed manual responding to S2 may have been a consequence of the late suppression hypothesized to follow task-

irrelevant S1-elicited manual response activation and not the task-relevant S1-elicited oculomotor response as proposed by Taylor and Klein (2000).

This possibility is tested in the pro-saccade condition of our experiment by eliminating the kind of manual response priming by S1 that was possible in Taylor and Klein (2000). Specifically participants *only* perform the combination of stimuli and responses highlighted by the solid blue circle in Figure 5.1: a pro-saccade to a peripheral S1 followed 1 second later by a manual response spatially compatible with the direction indicated by a central arrow S2. Thus, in this design, and unlike that in Taylor and Klein (2000), the peripheral S1 is only associated with a pro-saccadic response, not a manual localization response, and the central S2 is only associated with a spatially-compatible manual response. A failure to observe output-based inhibition in this design would resolve the discrepancy by suggesting that Taylor and Klein's "saccade-manual" findings were an artifact of S1-elicited manual response activation and subsequent suppression.

Assuming that the motor bias in Taylor and Klein (2000)'s "saccade-manual" cell is caused by the pro-saccade, and not by manual response activation-suppression, how would such data be reconciled with the data from the anti-saccade paradigms clearly demonstrating input-based IOR effects? Klein and Hilchey (2011) provided a rapprochement between the anti-saccade results and the theoretical framework advanced by Taylor and Klein (2000) by suggesting that the input-based form of IOR is generated not simply when eye movements are not being made during the task (as proposed by Taylor & Klein) but rather only when the reflexive machinery for generating saccadic eye movements is tonically suppressed. Klein and Hilchey note that even though eye

movements are permitted (indeed, required) in an anti-saccade task, the circuitry (specifically the superior colliculus) responsible for generating reflexive saccadic eye movements to the source of stimulation must be inhibited (*cf* Forbes, & Klein, 1996; Everling, Dorris, Klein, & Munoz, 1999; Ignashchenkova, Dicke, Haarmeier & Thier, 2004) for satisfactory performance on this task because, in the absence of this inhibition, incorrect pro-saccades would often be made.

To test this proposal we explore IOR using the same combination of stimuli and responses as described above for the pro-saccade condition except that participants are required to make an anti-saccade to S1. This allows us to determine whether tonic suppression of the oculomotor system responsible for *reflexively generated* saccades abolishes output-based IOR in much the same way that forbidding pro-saccadic oculomotor responses does (see the dashed blue circles in Figure 5.1). If Klein and Hilchey (2011) are correct in their assertion that the input-based form of IOR depends critically on whether the reflexive oculomotor system is actively suppressed – and if IOR is indeed more input-based in the anti-saccade paradigm, as suggested by prior experimental results (e.g., Fecteau et al., 2004) – there should be no IOR effect from an anti-saccade upon manual responses to central arrows.

Experiment and predictions

The methods of the experiment described above are illustrated in Figure 5.2. Participants make a pro- or anti-saccade (in different blocks) to a peripheral cue, return gaze to center, and then, in response to a centrally presented arrow, make a manual response either spatially compatible or incompatible with the saccade to S1. We

consider two possible patterns of results in Figure 5.3. As illustrated in panel A, if the IOR generated by a saccade in response to a peripheral target (whether pro-or anti-) has its effect along the input pathway, inhibition should be observed in neither condition (because there is no sensory overlap between the peripheral target for the first saccade and the central arrow target). Such a finding would also imply the motoric inhibition observed in this cell by Taylor and Klein (2000) had been generated by "active suppression of the task-irrelevant manual response activation elicited by the first target" (see p. 8-9). As illustrated in panel B, if the refinement to Taylor and Klein (2000) offered by Klein and Hilchey (2011) is correct, inhibition should be observed with pro-saccades but not with anti-saccades. Klein and Hilchey suggested that the suppression of the reflexive oculomotor system is a pre-requisite for generating the input form of IOR (and conversely, that generation of the output form of IOR depends on this system being in an active state).

Many IOR researchers believe that the effect is primarily upon input processes and therefore would predict the pattern illustrated in panel A. This view begins with Posner and Cohen (1984) who asserted that the "...inhibition depends primarily or perhaps exclusively upon the sensory information" (p. 541). And it has been regularly endorsed in the following decades by various scholars: "when inhibition of return is activated by a peripheral precue, it functions as a location tagging mechanism which inhibits detection of signals at the tagged location" (Rafal, Egly & Rhodes, 1994, p. 294); "...the delay of a saccade to a cued location, a seemingly motor impairment, could be explained by a perceptual-level mechanism characterized by interactions between

stimuli presented successively at the same spatial location" (Li & Lin, 2002b, p. 273); "we propose that a sensory-based mechanism is at work, causing the participant to be slower at processing information in a region of space that was recently stimulated." (Fecteau et al., 2004, p. 90). In this context, results in accordance with panel B would be surprising to many and therefore highly informative.

Method

Participants

Fourteen Dalhousie University undergraduates (11 females) ranging in age from 18-24 were tested in a single 75-90 minute session for course credit or monetary compensation ($15 CDN) in two saccade tasks: pro-saccade to S1 and anti-saccade to S1 (see below). All participants were naive to the purposes of the experiment and reported normal or corrected-to-normal vision.

Apparatus and procedure

Participants were tested in a dimly-lit room and all stimuli were presented against a black background on a 19” Hitachi CRT stimuli. The viewing distance was 22.5”. An EyeLink II head-based eye monitoring system was used to monitor eye movements. The eye-tracker’s precision was ensured via a 9-point calibration procedure for each observer in each task. A Python program written by Matthew Hilchey was used to coordinate stimulus presentation and participants’ responses with the eye tracking equipment.

The sequence of events on a typical trial is illustrated in Figure 5.2. Each trial began with the presentation of three white boxes [measuring 2.3 x 1.8 degrees of visual angle (DVA)], separated by 7.5 DVA, along an imaginary horizontal plane at the center of the monitor. The middle box contained a fixation stimulus, a red "+" symbol, measuring .5 x .5 DVA. To initialize the trial, the observer was required to depress the spacebar key when fixating the "+" symbol; if the observer was not fixating the "+" symbol, a tone would inform them and the fixation stimulus would remain red. If the observer successfully fixated the "+" symbol, no tone was presented and the symbol spontaneously turned white to indicate that the trial was successfully initialized.

Two hundred fifty milliseconds (ms) after the successful initiation of a trial, a white dot (S1) with a radius of .5 DVA appeared for 50 ms centered in either the left or right box. In the pro-saccade task, the observer was required to make a saccadic eye movement to the box wherein S1 appeared. In the anti-saccade task, the observer was required to make an eye movement to the mirror opposite box. In both tasks, observers were instructed to make precise and rapid eye movements to the boxes and to return their gaze endogenously to fixation as soon as possible after their gaze had arrived at the target box. If an eye movement was made before the onset of S1, the trial was spontaneously aborted and reshuffled back into the list of trials. One second after the onset of S1, a leftward or rightward pointing arrow (S2, measuring 1 DVA in width) appeared at fixation for 1.5 s. The direction of the arrow could not be predicted from S1 and observers were apprised accordingly. Observers were required to report the direction of the arrow with a manual response (pressing the "z" or "/" key on a standard

QWERTY keyboard with their left or right index finger for left- and right- pointing arrows, respectively).

The pro- and anti-saccade tasks were run in separate blocks (of 232 complete trials), the order of which was counterbalanced across participants. The first 32 trials in each block were considered practice. Between tasks, the observer took a short break, re-calibration was performed and the observer was informed of the new saccadic response code to S1. In essence, this is a simple 2 (saccade task: pro- or anti-) x 2 (cue condition: cued or uncued) within-subject design. Remaining faithful to nomenclature from the IOR literature, a trial was classified as "cued" if S1 had been presented at the location toward which S2 (the arrow) later pointed.

Results

One observer was excluded from analysis because of a failure to obey task instructions. Ten percent of the experimental trials were excluded because the two saccades (in response to S1 and back to the fixation stimulus) were not made within the allotted 1 second window. The expected effects of saccade task performed in response to S1 were obtained (see SRT column of Table 5.1): participants were significantly slower [$t(12) = 7.03$, $p < 0.01$] and less accurate [saccade in the wrong direction indicated by S1; $t(12) = 3.76$, $p < 0.01$] in the anti-saccade block (M=308 ms, %error=17.5) than in the pro-saccade block (M=209ms, %error=5.4).

After excluding saccadic eye movement errors of omission and commission (as described above), an additional ~8% of the trials were excluded, in the following order, for various reasons. An eye movement was detected during S2 on 1.3% of trials. No

manual response was made to S2 on 0.8% of trials. Anticipatory responses (manual response times < 100 ms) were detected on 0.4% of trials. Uncharacteristically long manual response times (RTs > 800 ms) not likely to reflect the phenomenon of interest were detected on 2.6% of trials. Manual response errors were made on 2.5% of trials (See Table 5.1 for more information); analysis of these error rates revealed no significant effects.

The key dependent variable from the experimental trials was performance on the manual RT task (S2) on trials in which the initial saccade was made in the direction indicated by the instructions for S1 (pro- or anti-saccade) and a correct manual response was made to S2. The manual RT data from these "correct" trials were submitted to a 2 (Cueing: cued or uncued) x 2 (Task: pro-saccade or anti-saccade) repeated measures analysis of variance (RMANOVA). There was no overall effect of Task on RT ($F < 1$). There was no effect of Cueing [$F(1, 12) = 2.39$, $p > 0.10$]. Importantly, there was a two-way interaction between Cueing and Task [$F(1, 12) = 12.72$, $p < 0.01$]. Simply, and as can be seen in Figure 5.4, the results conform to the predictions in panel B of Figure 5.3: Responses were slower when cued ($M = 432$ ms) as compared to uncued ($M = 407$ ms) in the pro-saccade task [$t(12) = 3.57$, $p < 0.01$] whereas no difference was observed between cued ($M = 421$ ms) and uncued ($M = 430$ ms) responses in the anti-saccade task [$t(12) = -1.23$, $p > 0.10$].

Discussion

In the pro-saccade task, keypress responses to central arrow S2s were on average 25 ms slower when spatially compatible with the saccadic response to S1. This finding

compellingly demonstrates that a pro-saccade to a peripheral S1 generates a response bias against the prior saccadic response vector, as suggested by Posner et al (1985). Importantly, this response bias is present even though (in contrast with Taylor & Klein's methods) peripheral onsets were not associated with manual responses and arrows were not associated with saccadic responses[14]. In the complimentary condition, the methods were the same except that each trial began with an anti-saccade. As predicted by Klein and Hilchey's (2011) proposal that the output form of IOR depends on the reflexive oculomotor system's being in an active state (and the well-established idea that requirement to make an anti-saccade entails the suppression of this system), the output form of IOR was not observed (keypress responses to central arrows were statistically indistinguishable on cued and on uncued trials).

Pro-saccades generate output-based IOR

The inhibitory aftereffect in the pro-saccade condition also repudiates the notion that repeat sensory stimuli are necessary for IOR (e.g., Fecteau, & Munoz, 2006; Satel, Wang, Trappenberg, & Klein, 2011) because with a central arrow S2 no such repetition occurs. The data accord neatly with results from other studies, not involving anti-saccades, that generated IOR by pro-saccadic responses: Speed-accuracy tradeoffs are observed at the cued locations (Chica, Taylor, Lupianez, & Klein, 2010; Redden, Hilchey, & Klein, 2014a; the magnitude of the event-related component in electroencephalography reflecting low-level sensory processes does not correlate with

[14] Although this finding cannot be accommodated by the activation-suppression model in its current form (Burle, van der Wildenberg, & Ridderinkohf, 2005), we nevertheless believe that this model has merit for understanding results from other conditions represented in Figure 5.1 (see, e.g., Hilchey et al, 2013).

the magnitude of the output-based IOR effect (Satel, Hilchey, Wang, Story, & Klein, 2013); sensory adaptation at the level of the visual neuron in the SC is not sufficiently long-lasting to account for the phenomenon (Fecteau, & Munoz, 2005) and, as noted, repeated stimulation along the input pathway is not required to observe the effect (e.g., Posner et al., 1985; Rafal et al., 1989; Machado, & Rafal, 2004; Chica, Klein, Rafal, & Hopfinger, 2010; Hilchey, Klein, & Ivanoff, 2012; Hilchey, Klein &, Satel, 2014; Satel, Hilchey, Wang, Reiss, & Klein, 2014).

Anti-saccades generate input-based IOR

The absence of output-based IOR following an anti-saccade (and measured in response to a central arrow) is perfectly consistent with findings from earlier reports demonstrating robust IOR effects in anti-saccade paradigms when the cues and targets occupy the same locations (Rafal, Egly, & Rhodes, 1994; Khatoon, Briand, & Sereno, 2002; Fecteau et al., 2004; Abegg, Sharma, & Barton, 2012). Such findings also accord with recent data from spatial cueing paradigms in which pro-saccadic responses were forbidden or actively discouraged and manual detection or localization responses were required to peripheral S2s (Chica et al., 2010; Hilchey et al., 2014). In these cases, IOR is expressed as a genuine reduction in performance and not as a speed-accuracy tradeoff. Input-based IOR correlates with low-level sensory processes when oculomotor responses are expressly forbidden (Satel et al., 2013), impairs the detection of phosphenes when induced via transcranial magnetic stimulation (Smith, Ball, & Ellison, 2012), and is represented by visual cortices in neuroimaging studies controlling for the presence of eye movements (Muller, & Kleinschmidt, 2007). Moreover, recall that

manual responding is not delayed by cueing when measured by a central arrow S2 in Taylor and Klein (2000)'s no response-manual, and manual-manual cells (see dashed blue circles in Figure 5.1) when saccadic responses were expressly discouraged.

On the possibility of approximately equivalent and opposing inhibitions in the anti-saccade condition.

Before concluding, we would like to address a possibility that might occur to some readers[15]. It might be suggested that there are two effects of IOR following an anti-saccade: an input-based effect caused by the stimulus at the cued location, and an output-based effect caused by the response toward the saccade location. If these effects were simultaneous and additive as proposed by Abrams and Dobkin (1994a; see also Wang, Satel & Klein, 2012), and approximately equal, it might be imagined that they would roughly cancel each other – yielding the null result we have reported. There are a couple of difficulties for this proposal. First, Hilchey, Klein and Ivanoff (2012) have undermined the "simultaneous and additive" attributions by showing that the pattern of results that led to them was not obtained when an attentional control setting confound was eliminated. Second, and more challengingly, because S1 (a peripheral stimulus) and S2 (a central arrow) do not share an input pathway in the present design responses to central arrows are not likely to measure an input-based effect.

Conclusions

We, like many others, have identified a response- or task-based dissociation on the effect of IOR. Whereas such response-based dissociations are commonly observed when comparing between keypress and saccadic responses to S2 (e.g., Kingstone, &

[15] We thank Juan Lupianez for raising this possibility.

Pratt, 1999; Taylor, & Klein, 2000; Hunt, & Kingstone, 2003; Sumner, Nachev, Vora, Husain, & Kennard, 2004; Pratt & Neggers, 2008; Zhang, & Zhang, 2011; MacInnes, Kruger & Hunt, 2015), Klein and Hilchey (2011) hypothesized further that the type of eye movement (pro- or anti-) would determine the form of IOR. The present results provide support for this hypothesis, which is further reinforced by converging evidence from other studies in our laboratory (e.g., Chica et al., 2010; Satel et al., 2012; Hilchey et al., 2014). Recapitulating, we note that when a task requires anti-saccades or forbids pro-saccade responses, tonic inhibition is applied over midbrain eye movement structures responsible for reflexively-generated saccades (e.g., Everling et al., 1999). Co-occurring with this suppression is a form of IOR that is nearer the input end of the processing continuum. When a task requires pro-saccades or reflexive eye movements are not actively suppressed, the midbrain eye movement structures responsible for reflexively-generated saccades are in a more active state, and a form of IOR that is nearer the output end of the processing continuum is generated. This latter form of IOR is caused by reflexive oculomotor response activation and serves to bias subsequent responding, oculomotor or otherwise, against the prior saccade vector (e.g., Posner, Rafal, Choate & Vaughan, 1985; Hilchey, Klein & Satel, 2013). The exact processes underlying the input-based form of IOR at 1 s CTOAs remain unclear though the effects may be induced experimentally by spatially repetitive sensory stimulation (e.g., Fecteau, Au, Armstrong & Munoz, 2004; Smith, Ball & Ellison, 2012) and/or non-oculomotor response priming (e.g., Hilchey, Satel, Ivanoff & Klein, 2013, for consideration; de Jong,

Liang & Lauber, 1994), and may involve higher level object-based representations (e.g., Jordan & Tipper, 1998; Grison, Kessler, Paul, Jordan & Tipper, 2004).

Despite our having observed an output- based IOR effect in the pro-saccade task and no discernible effect in the anti-saccade task – as expected – we recognize that response-based dissociations alone do not provide a complete account of the nature and implementation of IOR. Consider, for example, that damage to the SC (a key component of the reflexive oculomotor circuitry) is sufficient to disrupt the form of IOR that is measured when only keypress responses are required (Posner, Rafal, Choate, & Vaughan, 1985; Sapir, Soroker, Berger, & Henik, 1999; Sereno, Briand, Amador, & Szapiel, 2006)[16]. Evidently then, there is a common denominator between the input and output forms of inhibition, a commonality that qualifies the idea that the two forms are completely dissociable. Determining precisely how the reflexive oculomotor circuitry – and by extension, the activation state of this system – plays a role in both forms of inhibition should remain a high priority for future research. In the pursuit of this objective we recommend an interdisciplinary approach in which behavioral measures and the recordings of brain activity are collected during cleverly designed experimental tasks and the data from both domains is accommodated in a computationally explicit and neurophysiologically plausible model (e.g. Sereno, Lehky, Patel & Peng, 2010; Satel, Wang, Trappenberg & Klein, 2011; Wang, Satel, Hilchey & Klein, 2012).

[16] The reader may wonder why we continue to endorse the framework presented here if primitive oculomotor response pathways are required for both forms of IOR. Simply, lesion data do not necessarily imply that both forms of IOR are fundamentally caused by saccadic eye movements. As an alternative, consider the possibility that the SC needn't be suppressed when compromised, a suppression which may ultimately be required to invoke the input-based form of IOR.

Acknowledgment

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Table 5.1

Reaction times (and standard deviations) for correct responses and error rates from all conditions of the experiment.

S1 Task	SRT to S1			Cued: Man. RT to S2			Uncued: Man. RT to S2		
	RT	SD	%E	RT	SD	%E	RT	SD	%E
Pro-saccade	209	25	5	432	69	3	407	69	1.67
Anti-saccade	308	56	18	421	61	3	430	67	3.4

Figure 5.1.

Schematic representation of the methods and results from Taylor and Klein (2000). Filled circles indicate conditions for which significant inhibition was obtained; unfilled dotted circles indicate conditions for which inhibition was not observed. The two forms of inhibition described in the text are denoted by grey (for the input form) and black (for the output form) filled circles. The solid blue circle represents the stimulus and response conditions from Taylor and Klein that are explored in the present study: the first stimulus (S1) is peripheral and it requires a saccadic response; the second stimulus (S2) is a centrally presented arrow that it requires a manual

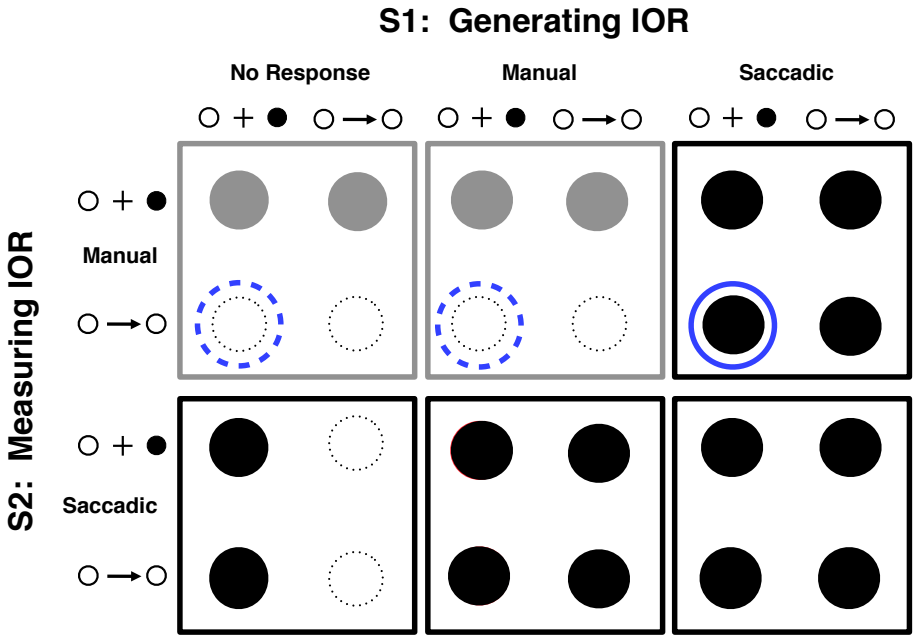


Figure 5.2

Sequence of events in the two conditions (pro-saccade and anti-saccade) of the experiment. The dotted arrows indicate correct saccades given the location of the first stimulus (S1) and the participant's tasks (pro- vs anti-saccade) which were run in separate blocks. Participants made directionally compatible button press responses to the central arrow stimuli (S2) shown in the bottom panel.

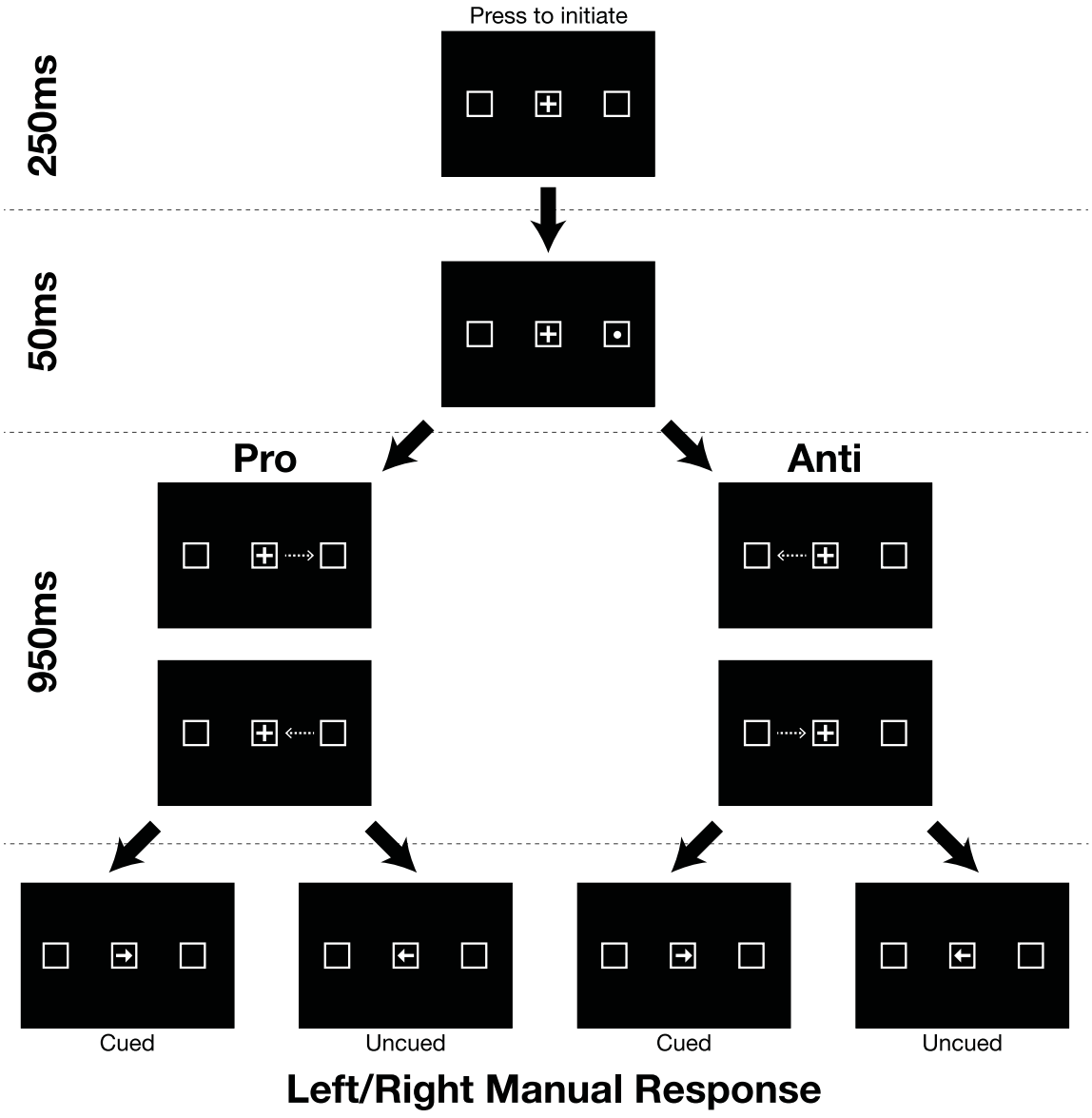


Figure 5.3.
Possible patterns of results for the present experiment with manual responses to centrally presented arrows following pro- and anti-saccades. See text for an explanation.

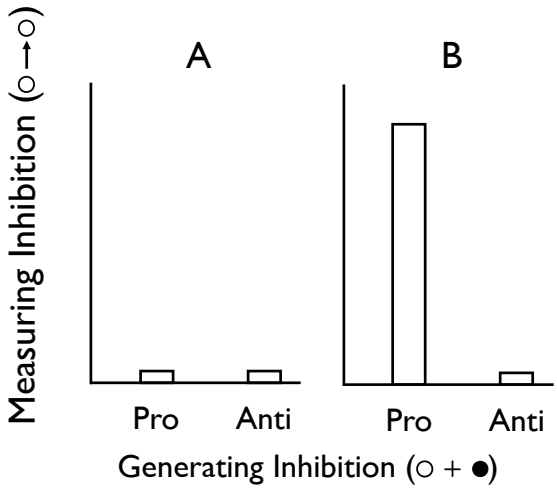
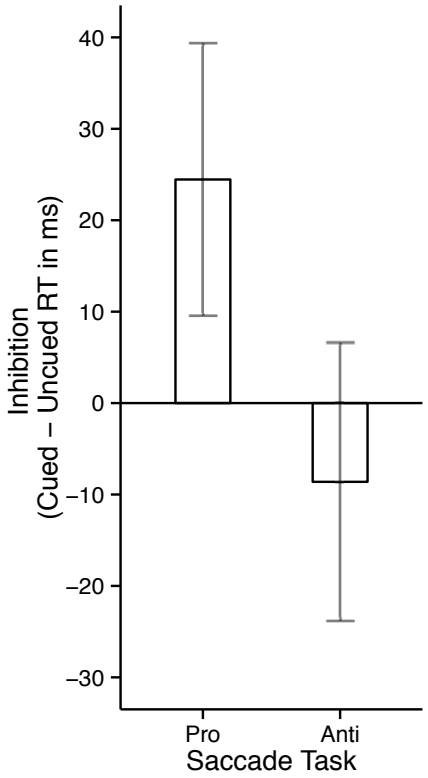


Figure 5.4

Mean IOR effects (Cued - Uncued RT) for manual responses to central arrows after peripheral stimuli calling for a pro- or anti-saccade. "Cued" refers to trials on which the location of S1 (peripheral stimulus) is in the same direction as the response required to S2 (the central arrow). "Uncued" refers to when these two spatial codes mismatch. Error bars are 95% confidence intervals of the effect.



CHAPTER 6:
ON THE NATURE OF THE DELAYED "INHIBITORY" CUEING EFFECTS GENERATED BY UNINFORMATIVE ARROWS AT FIXATION

The manuscript based on this study is presented below. Co-authors for this manuscript are Dr. Jason Satel, Dr. Jason Ivanoff, and Dr. Raymond M. Klein, respectively.

Psychonomic Bulletin & Review, Vol 20, Jan 2013, pp. 593-600. http://link.springer.com/article/10.3758/s13423-013-0376-5#page-1

Abstract

When the interval between a spatially uninformative arrow and a visual target is short (< 500 ms), response times (RTs) are fastest when the arrow points to the target. When this interval exceeds 500 ms, there is a near-universal absence of an effect of the arrow on RTs. Contrary to this expected pattern of results, Taylor and Klein (2000) observed that RTs were slowest when a to-be-localized visual target occurred in the direction of a fixated arrow presented 1 s earlier (*i.e.,* an 'inhibitory' cueing effect; ICE). Here we examine which factor(s) may have allowed the arrow to generate an ICE. Our experiments indicate that the ICE was a side-effect of sub-threshold response activation attributable to a task-induced association between the arrow and a keypress response. Because the cause of this ICE was more closely related to sub-threshold keypress activation than oculomotor activation, we considered that this effect might be more similar to the negative compatibility effect (NCE) than inhibition of return (IOR). This similarity raises the possibility that classical IOR, when caused by a spatially uninformative peripheral onset event and measured by a keypress response to a subsequent onset, might represent, in part, another instance of an NCE. Serendipitously, we discovered that context (*i.e.,* whether an uninformative peripheral onset *could* occur at the time of an uninformative central arrow) ultimately determined whether the 'inhibitory' aftermath of automatic response activation was on output or input pathways.

Introduction

Uninformative peripheral cues generally have a biphasic effect on target processing. At short cue-target onset asynchronies (CTOAs), response times (RTs) to cued targets are typically faster than to uncued targets. At long CTOAs, responses to cued targets are generally *slower* than to uncued targets (Klein, 2000; Posner & Cohen, 1984). The explanation for this later *"inhibitory" cueing effect* (ICE) has been hotly debated. Posner, Rafal, Choate and Vaughan (1985) referred to it as *inhibition of return* (IOR[17]), a name that conveyed their proposal that orienting was inhibited from locations that had been recently attended. In contrast to the inhibited attention account, this ICE has also been attributed to inhibited responding (Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987), response biases (Klein & Taylor, 1994; Ivanoff & Klein, 2001), habituation (Dukewich, 2009), sensory adaptation (Fecteau & Munoz, 2005), and onset detection cost (Lupianez, 2010). These ideas about the nature of the effect can be classified into two broad categories: those affecting information processes more closely related to the input (e.g., inhibited attention, sensory adaptation, habituation) and those tied to the output (e.g., response inhibition, response biases).

Support for both of these categories was provided in Taylor and Klein's (2000) thorough extension of methods used by Posner and Cohen (1984) in their discovery of IOR. Taylor and Klein manipulated cue type (S1: peripheral luminance change or central arrow), target type (S2: peripheral luminance change or central arrow), cue response

[17] We reserve the term “ICE” to refer to an inhibitory effect of a cue that hinders performance in responding to subsequently presented targets. ICE is neutral in the sense that it does not imply a particular mechanism or function. IOR, on the other hand, is not neutral (e.g., Berlucchi, 2006). We consider IOR as one type of ICE, perhaps with subtypes (i.e., perceptual or motoric). Thus, while IOR is an ICE, not all ICEs are IOR.

modality (no-response, manual localization or pro-saccades), and target response modality (manual localization or pro-saccades). They generated a pattern of results (see Figures 6.1 and [18]) suggesting that IOR comes in two mutually exclusive forms depending on the state of the oculomotor system. Their use of central arrow targets was instrumental for drawing this conclusion as the sensory features of a central arrow do not overlap with those of a peripheral stimulus. An IOR effect was seen in almost all conditions for which an eye movement was made to either the cue or target. Importantly, the magnitude of the effect measured in response to a central arrow was as large as that measured in response to a peripheral onset (see also Hilchey, Klein, & Ivanoff, 2012), suggesting that the effect was entirely on the output end of the processing continuum. In the remaining conditions, when no eye movements were made and the reflexive oculomotor machinery was tonically inhibited (Klein & Hilchey, 2012), IOR was observed only when measured by peripheral stimulation, suggesting an effect along the input pathway.

A surprising result among the conditions from which Taylor and Klein (2000) inferred an input form of IOR, and one that is the focus of this paper, was that IOR was seemingly generated by an ignored, and uninformative, central arrow cue (blue circle in Figure 6.1). This was surprising, circa 2000, because it had already been demonstrated that IOR was not observed after a central arrow cue had been used to generate a voluntary shift of attention that was subsequently cancelled by an event indicating that the probabilistic meaning attached to the central arrow no longer applied (Posner &

[18] This figure is referenced as 6.S1 because it appeared as a supplementary material to the original publication.

Cohen, 1984; Rafal et al., 1989). If IOR was not generated in the aftermath of orienting to a previously informative arrow, then why should it be generated by an uninformative one?

In the years that followed Taylor and Klein's (2000) comprehensive exploration of the causes and effects of IOR, it was demonstrated in several laboratories that *uninformative* arrow cues elicit orienting in a reflexive manner (e.g., Ristic, Freisen, & Kingstone, 2002; Ristic & Kingstone, 2006; Stevens, West, Al-Aidroos, Weger, & Pratt, 2008; Tipples, 2002; see also Ivanoff & Saoud, 2009; Ristic & Kingstone, 2012). In contrast to the findings of Taylor and Klein in this same condition, those studies that used relatively long CTOAs reported little evidence of IOR from uninformative central arrows. In other words, the biphasic pattern (a shift from facilitation to inhibition) as the CTOA is lengthened is rarely observed; the typical pattern is early facilitation in the direction of the arrow and a decay of this effect towards zero as the CTOA increases (see Figure 6.2)[19]. With that in mind, the Taylor and Klein finding of slower responding to peripheral targets presented approximately 1 s after the presentation of an uninformative central arrow is rather anomalous.

To resolve this anomaly, we first compared the methods used by Taylor and Klein (2000) with the aforementioned cases in the arrow-cueing literature where no ICE was observed at a long CTOA. There are two noteworthy differences between Taylor and

[19] Excluding Taylor and Klein's (2000) finding, the literature provides 21 separate measurements of uninformative arrow cueing effects in young adults at CTOAs of 600 ms and greater. The mean of these is about 7 ms of facilitation; only 4 of the 21 measures were numerically negative (inhibition) and only one of these was reported to be significant (a 7 ms ICE from Taylor and Ivanoff [2005], Exp. 2).

Klein and most of the studies exploring the time course of the automatic effect of uninformative arrow cues on peripheral target processing.

First, Taylor and Klein used a "cue-back to fixation" procedure (brightening of fixation between the cue and target) which has rarely been used by researchers studying the orienting effects of uninformative arrows. Typically, the cue-back is administered in an attempt to ensure that attention, after having been captured by the cue, is disengaged from it, and returned to a neutral state (Klein, 2005; Posner & Cohen, 1984). In the arrow-cueing literature, where the cue-back is rarely used, an IOR effect may have been obscured to the extent that attention dwelled at the cued location. The possibility that Taylor and Klein observed an ICE following uninformative and ignored central arrows because they used a cue-back to fixation will be referred to as the *attentional disengagement hypothesis*.

Second, and also in contrast to the arrow-cueing literature, localization responses were often made to arrows in Taylor and Klein's (2000) study. Consequently, when arrows were presented as 'to-be-ignored' cues they may have covertly activated a spatial response in their direction. This response tendency would have to be suppressed (e.g., Ridderinkhof, 2002) to discourage responding to the 'to-be-ignored' central arrow cue. Note, however, suppression also occurs even if the response is executed to the cue, as with target-target designs (Pratt & Welsh, 2006; Taylor & Klein, 2000). A negative aftereffect of this suppression may have masqueraded as IOR in Taylor and Klein. We will refer to this as the *stimulus-response (S-R) activation-inhibition* hypothesis.

The experiments

The goal of the present experiments is to determine which of these hypotheses might be responsible for the anomalous ICE that Taylor and Klein (2000) observed with uninformative central arrows (S1) and peripheral targets (S2) calling for a localization response. In Experiment 1, we mixed the cue types used by Taylor and Klein while using a cue back and peripheral targets. As in the literature where central arrows were not responded to, in this experiment arrows have no experiment-specific S-R associations (see Figure 6.2). Finding an ICE in this condition would be support for the *attentional disengagement hypothesis* and against the *stimulus-response (S-R) activation-inhibition.* In Experiment 2, all cues were central arrows, while targets were central arrows or peripheral onsets. Because the central arrows serve as targets on 1/2 of the trials, an ICE would be predicted by either hypothesis.

Methods

Participants

Twenty students took part in Experiment 1, and twenty-six in Experiment 2, in exchange for extra course credit. All subjects reported normal, or corrected to normal, vision.

Apparatus and stimuli

Presentation of stimuli, timing operations, and behavioral data collection was controlled by a personal computer running Python scripts. Stimuli were presented on a 19" Asus LCD monitor located 57 cm in front of participants, and responses were made using a Microsoft keyboard. All stimuli were presented in white on a black background.

Three boxes (4.5°×4.5°) 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 either a brightening and thickening of one of the peripheral boxes, or as a central arrow pointing to one of the landmark boxes. Targets were bright disks with a diameter of 2.4°. Participants were tested in a dark room, with their head resting on a chin rest.

Eye position was monitored throughout the experiment using a desk-mounted eye tracking system (EyeLink 1000) sampling at 250 Hz. EEG data was also collected, though it is not reported here.

Design and procedure

The sequence of events on a trial is illustrated in Figure 6.3. Each trial began with the display of the three landmark boxes and a central red cross. Immediately following a successful self-paced drift correction, the cross turned white, and 500 ms later a cue (non-predictive of target location or direction) was presented for 300 ms. The cue was the thickening of one of the peripheral boxes or a central arrow. Both cues were used in Experiment 1; only central arrow cues were used in Experiment 2. A central cue-back (a thickening of the middle box) was presented 200 ms after the cue offset for 300 ms. One second after the onset cue, the target was presented for 1500 ms or until a response was detected. The target was either the appearance of a filled circle in one of the two peripheral boxes or a central arrow. Both targets were used in Experiment 2; only peripheral targets were used in Experiment 1. Subjects were required to discriminate the location or direction of the circle or arrow targets, respectively, and to

indicate the result of their leftward/rightward discrimination with a corresponding keypress response (using the 'z' key for "left" and '/' key for "right"). After an inter-trial interval of 1000-1500 ms, another trial began. Each subject was run in 400 experimental trials after the observers indicated that they were comfortable performing the task. Since task instructions required central fixation throughout trials, if gaze position was ever outside an imaginary 1.5° circle centered at fixation, the trial was spontaneously aborted and recycled, with an error message instructing subjects that they must maintain central fixation. Finally, as in Taylor and Klein (2000), a target was considered "cued" if the location or direction of the peripheral onset or central arrow cue, respectively, categorically matched the location or direction of the target.

Results

Trials with incorrect responses were excluded from analysis (<1.5%). Examination of the RT distribution led to the removal of trials in which the manual RTs were faster than 200 ms (< 1%) or slower than 800 ms (< 1.5%).

Experiment 1 (mixed cues)

A repeated measures ANOVA was performed on the remaining RTs (see Appendix), with factors Cue Type (central versus peripheral cues) and Cueing (cued versus uncued). There was a main effect of Cueing [$F(1, 19) = 17.32$, $p < 0.001$, $MSe = 157.24$], indicating slower responses to cued as compared to uncued targets. There was also an effect of Cue Type [$F(1, 19) = 6.44$, $p < 0.05$, $MSe = 166.31$], indicating the RTs were slower, in general, when cues were peripheral as compared to central. Importantly, the main effects were qualified by a two-way interaction between factors Cueing and

Cue Type [$F(1, 19) = 44.21$, $p < 0.001$, $MSe = 92.45$]. Simply, an ICE (see Figure 6.4, left panel) was observed when the cues were peripheral events (ICE = 26 ms), but not when cues were central arrows (ICE = -3 ms).

Experiment 2 (mixed targets)

A repeated measures ANOVA on the remaining RTs (see Appendix), with factors Target Type (central versus peripheral targets) and Cueing (cued versus uncued), revealed an effect of Cueing [$F(1, 25) = 38.60$, $p < 0.001$, $MSe = 76.07$], indicating slower responses to cued as compared to uncued targets. A main effect of Target Type was also observed [$F(1, 20) = 96.80$, $p < 0.001$, $MSe = 554.04$]. RTs were faster for peripheral as compared to central targets. There was no two-way interaction between factors Target Type and Cueing [$F(1, 25) = 1.64$, $p > .20$, $MSe = 89.60$], suggesting that the ICE scores for peripheral onset (ICE = 8 ms) and central arrow targets (ICE = 13 ms) were statistically non-discriminable (see Figure 6.4, middle panel).

Discussion

In Experiment 1, we evaluated the *attentional disengagement hypothesis*. This hypothesis asserts that an uninformative central arrow intrinsically generates an ICE in the spatial cueing paradigm, but that its effect is commonly obscured by long-lasting, residual attentional orienting at the cued location. By administering a cue-back to fixation we reasoned that we could successfully remove any residual attentional effects and expose any latent ICE. The results were clear: there was no apparent effect of the uninformative arrow signal on localization decisions (ICE = - 3 ms). In contrast, an ICE was observed when caused and measured by peripheral events (ICE = 26 ms). A cue-

back to fixation is not sufficient to observe an ICE that might have been generated by an uninformative arrow signal.

In Experiment 2, we evaluated the *S-R activation-inhibition hypothesis*. This hypothesis asserts that assigning a response to the arrow stimuli will generate a behaviorally measurable ICE at the CTOA used in this study. In this design, we removed the peripheral onset cues that were used in Experiment 1 and we added central arrow targets (as in Taylor & Klein, 2000). Since participants were responding to arrows on 1/2 of the trials, if the *stimulus-response (S-R) activation-inhibition* hypothesis were correct we would expect to find an ICE when observers responded to peripheral targets after an ignored central arrow. The results were clear: an ICE was present following a central arrow when measured by a peripheral onset (ICE = 8 ms). Unlike in Taylor and Klein (2000), an ICE was also observed, in comparable magnitude, when measured by central arrow targets (ICE = 13 ms).

Is the ICE in Experiment 2 an example of a "negative compatibility effect"?

It is noteworthy that the arrow-induced ICE that was observed in Experiment 2 and Taylor and Klein (2000) appears, in one important respect, more similar to the *negative compatibility effect* (NCE, see Sumner, 2007, for review) than to IOR. A quintessential procedure for eliciting an NCE is as follows: a meaningless arrow (the prime) occurs briefly (~30-50 ms) – usually at a foveated location – and is immediately masked (~100 ms). Following the offset of the mask, a to-be-discriminated arrow target occurs that either matches (compatible) or mismatches (incompatible) the prime. The NCE refers to the hallmark finding that RTs are typically faster for prime-incompatible

than prime-compatible targets. It is commonly accepted that the NCE depends critically on whether the prime is associated with a response (e.g., Schlaghecken & Eimer, 2004; Jaskowski & Slosarek, 2007; Jaskowski, 2007; Boy & Sumner, 2010; but see Cole & Kuhn, 2010). As long as the root cause of the NCE remains more closely related to the response activation spurred on by the prime than to any other factor, there could very well be a common cause between the arrow-induced ICE that we report here and the NCE. Both effects appear to share a cause: the tendency to suppress (inhibit) arrow-induced response activation.

Whereas the common cause (response activation) provides a conceptual link between the ICE we have observed and the well-studied NCE, paradigmatic differences would appear to challenge the linkage. For example, we tested at a relatively long CTOA using unmasked cues whereas in a typical NCE study the CTOA is short and the cue/prime is masked. Partially addressing this challenge, recent studies suggest that NCEs may persist at long CTOAs and without a mask (e.g., Jaskowski & Slosarek, 2007; Machado, Wyatt, Devine, & Knight, 2007[20]). Conversely, ICEs may be observed with subliminal cues (e.g., Ivanoff & Klein, 2003) and at short CTOAs (e.g., Danziger & Kingstone, 1999) in Posner cueing paradigms.

Do negative compatibility effects play a role in "classical" IOR?

Because of the natural tendency to respond toward the source of stimulation (e.g., Simon, 1969; de Jong, Liang & Lauber, 1994; Kornblum & Lee, 1995) when

[20] Two reviewers saw similarities between our investigation and that of Machado et al. (2007). In their E4, Machado et al. attempted to determine whether their ICE (E1-3) was on perceptual or response processes. Unfortunately, their manipulations abolished the ICE and, consequently, the contribution of perceptual and response processes to their ICE remained unknown.

making keypress responses, perhaps peripheral cues (as in the classical IOR paradigm) automatically activate corresponding S-R mappings in the hands. To avoid an untoward response to the cue such sub-threshold skeletal-motor (hand) activation might be inhibited (e.g., Schlaghecken & Eimer, 2002; Ridderinkhof, 2002). This proposal implies that in some situations manual response suppression leads to N/ICEs[21] that may masquerade as IOR (see also, Tassinari et al., 1987, for a different response-based explanation for the "classical" IOR effect), especially perhaps when task requires keypress responses to onset stimuli. Future research will be necessary to distinguish "IOR proper" (Posner et al., 1985) – which traditionally emphasizes oculomotor response activation and/or exogenous orienting – from those mechanisms responsible for NCEs.

Why did we see an N/ICE when central arrows were targets in Experiment 2?

Finally, we draw attention to an unanticipated but intriguing difference between our pattern of results in Experiment 2 and those of Taylor and Klein (2000). In Experiment 2, we found a significant N/ICE following a central arrow when measured using either peripheral onset or central arrow *targets*, whereas Taylor and Klein found no cueing effects when measured with central arrow targets. In experimental design, we retained almost every feature of the Taylor and Klein procedure, except, in contrast to Taylor and Klein, we did not include peripheral onset cues in Experiment 2. We will offer, below, a speculative explanation for how this factor might be responsible for

[21] Having proposed that the ICE from an uninformative central arrow might be an example of an NCE, we hereafter will use the abbreviation N/ICE to reflect this possibility.

eliciting motoric and perceptual forms of N/ICE in our study and in Taylor and Klein, respectively.

Our tentative explanation depends on the following postulates:

1) Despite instructions about the spatial non-informativeness of the cues, they nevertheless receive some attention because they indicate that the next stimulus will occur in 1 s and must be responded to;
2) When central and peripheral cues are intermixed (as in Taylor & Klein, 2000), the participant's spatial attentional control setting (ACS) at the time of the cue will, because of (1), include the periphery (see Hilchey, Klein, & Ivanoff, 2012; Wang & Klein, 2011). In the absence of peripheral cues (as in our Experiment 2), attention will be more tightly focussed on fixation;
3) When the periphery is attended at the time of a cue, the inhibition that follows cue-generated, automatically activated, spatial responses (see the previous section), is integrated into the input pathway of the prospective target location; however,
4) A central ACS precludes peripheral attention and hence reduces, if not completely eliminates, the possibility of this action-perception integration (3), leaving the inhibition unattached to an input pathway and hence purely motoric.

The role of spatial attention in action-perception integration that is entailed in postulates (3) and (4) might be viewed as akin to its role in Treisman and Gelade's (1980) "feature integration theory" except that what is being integrated are input

pathways and inhibited responses. When combined with postulates (1) and (2) (which determine whether attention at the time of the cue will be focussed on fixation or will encompass the periphery), the hypothesized action-perception integration process will ensure that the N/ICE generated by an uninformative central arrow will be motoric (unattached to input pathways) when there are no peripheral cues (4), and input-based when the central cues are mixed with peripheral cues (3). Further work will be required to evaluate the relationship between task-invoked spatial processing strategies and action-perception integration processes. Whether or not our proposal turns out to be correct, that the nature of the N/ICE generated by uninformative central arrows is clearly dependent on contextual factors.

Conclusion

The discrepancy between Taylor and Klein's (2000) finding that uninformative central arrow cues generated ‘inhibition’ when measured with manual responses to peripheral onsets and the near-universal absence of inhibitory effects in the arrow-cueing literature (Figure 6.2) can now be confidently attributed to the fact that Taylor and Klein's participants were often presented with central arrows as targets. In this context, the arrow cues would have generated automatic spatial response tendencies, the suppression of which resulted in a negative or inhibitory aftereffect. A common cause led us to suggest that the ICE might be an example of an NCE. It is even possible to extend this proposal to the more classical "IOR" that is generated when uninformative peripheral cues are followed by peripheral targets calling for a manual response. Finally, the unexpected finding that context (whether the central cues are mixed with peripheral

cues) affects whether inhibited response activations remain purely motoric or are integrated with input pathways, led us to propose that spatial attention plays a role in action-perception integration.

Acknowledgements

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Figure 6.1

Methods and results from Taylor and Klein (2000). The iconograms that label the rows and columns convey whether the stimuli were central arrows or peripheral onsets (irrespective of form). Solid circles indicate conditions in which significant inhibition of return (IOR) was obtained; unfilled dotted circles indicate conditions in which IOR was not observed. The two forms of IOR described in the text are denoted by gray (for the input form) and black (for the output form) filled circles. The outer circle surrounding the gray filled circle at the upper left represents the stimulus and response conditions from Taylor and Klein in which, in comparison with the literature, an anomalous inhibitory effect (see Fig. 6.2) was obtained: The first stimulus (S1), or cue, is an uninformative central arrow calling for no response, which is followed by a second stimulus (S2), or target, that is a peripheral onset requiring a manual localization response. The conditions explored here, in carefully selected combinations, are enclosed by the hashed polygon.

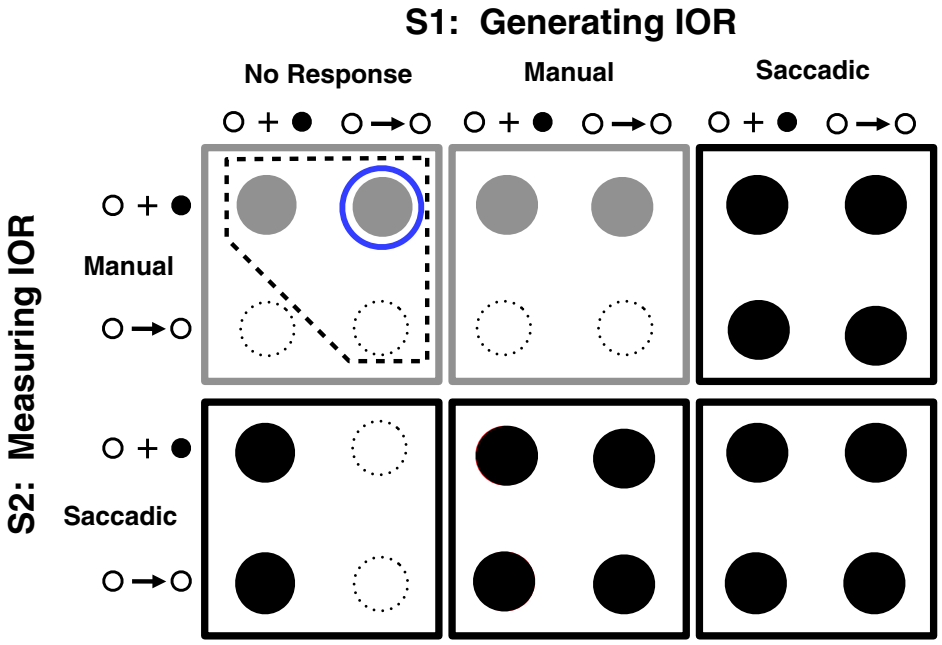

Figure 6.2

Time course of Cueing effects (facilitation is plotted as a positive score; inhibition as negative) generated by uninformative arrow cues and measured by manual responses to peripheral targets. Each unfilled circle represents a data point from one of the studies in the literature (a more detailed version of this figure with references to the literature can be found in Fig. 6.S1) that has examined response times (RTs) to targets following uninformative arrow cues (best-fit linear function, r = .4) in young adults. The finding from Taylor and Klein (2000; double circle in Fig. 6.1) is plotted here as the filled outlier disk.

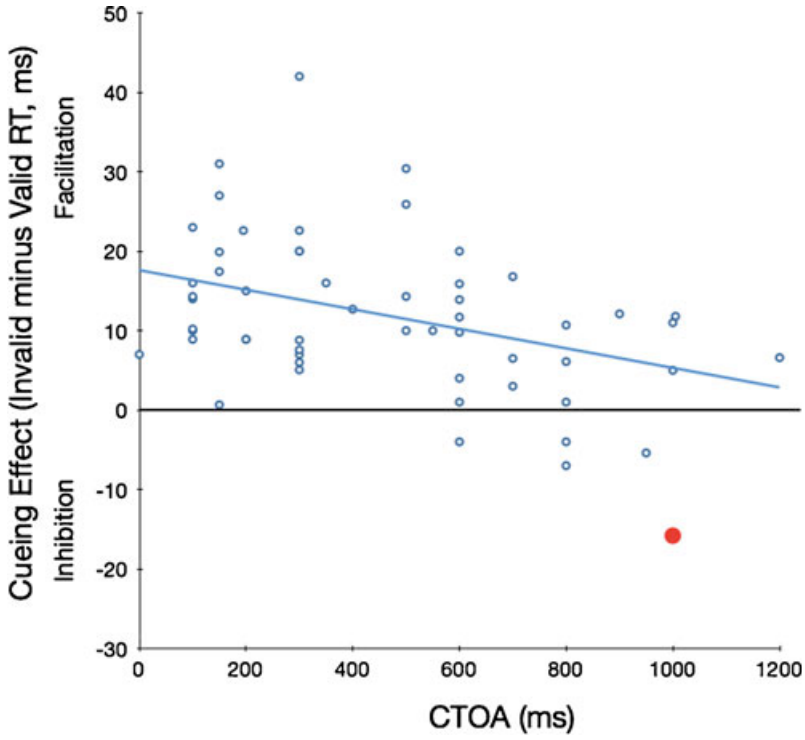

Figure 6.3

Experimental design. Participants were required to maintain central fixation throughout trials. In Experiment 1, half of the cues were central arrows, and the other half were peripheral thickenings of landmark boxes, while the targets were peripheral circles. In Experiment 2, the cues were all central arrows, while the targets were mixed peripheral circles and central arrows. A cue-back at central fixation also occurred between the cue and target onsets. Manual localization responses to targets were required after a 1-s cue– target onset asynchrony.

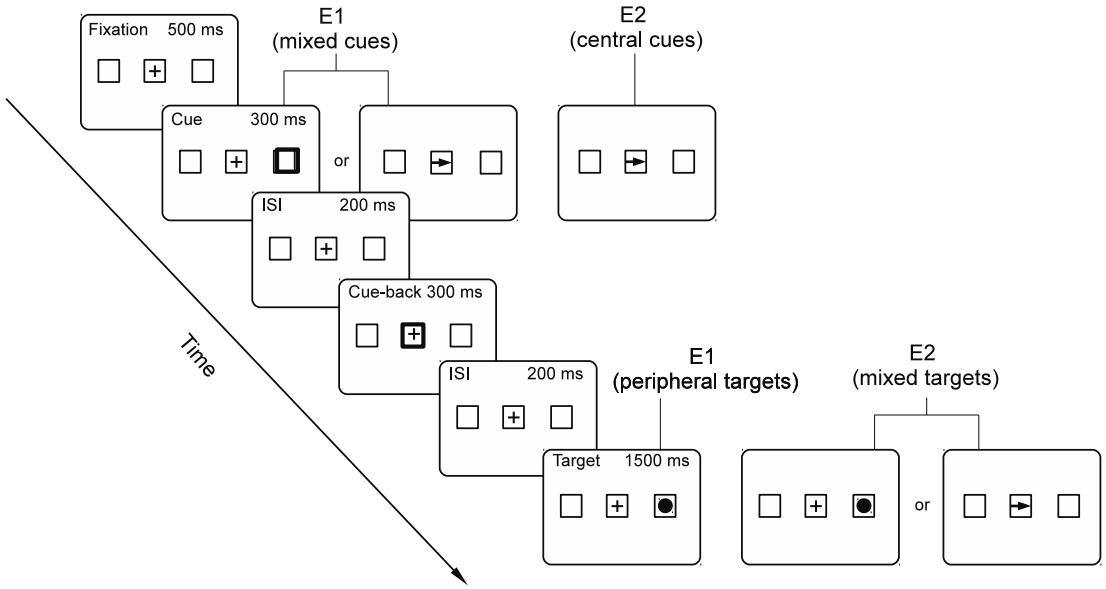

Figure 6.4

The methods and findings (cued minus uncued response times, in milliseconds) of the present experiments, along with the corresponding information from Taylor and Klein (2000). Black circles indicate conditions in which a significant inhibitory Cueing effect (ICE) was obtained; for white circles, ICE was not observed; gray circles indicate conditions not tested.

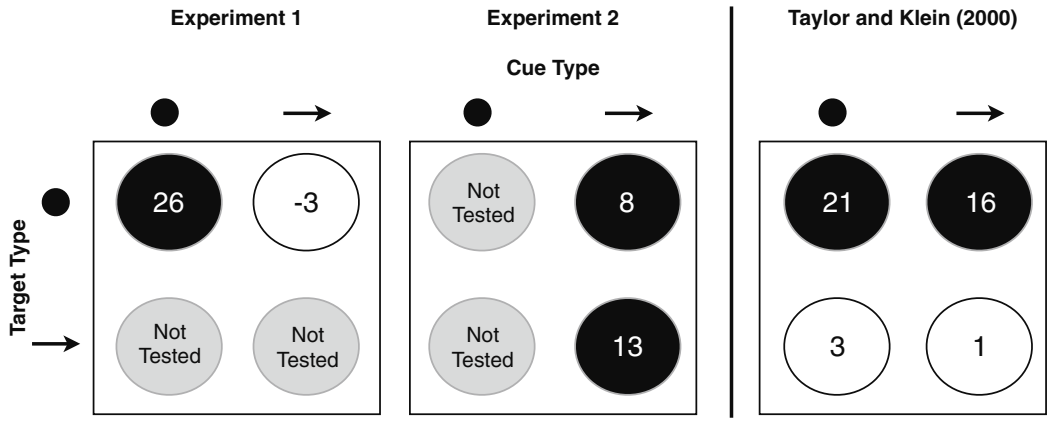

Figure 6.S1

Cuing effects (uncued minus cued reaction time) from all of the experiments of which we are aware that explored the effect of uninformative arrow cues on the perception of peripheral targets with at least one cue-target SOA of 500 ms or greater are represented in the figure below. One of these experiments (from Friedrich et al, 1997) is from an unpublished manuscript the data from which have been reproduced with the permission of the senior author. The remainder are published. Excluding the exceptional data point from Taylor and Klein (2000) which is plotted here as a filled red circle, the data show a gradual decline in facilitation toward zero as SOA is increased (r=.393 for the best fitting linear function).

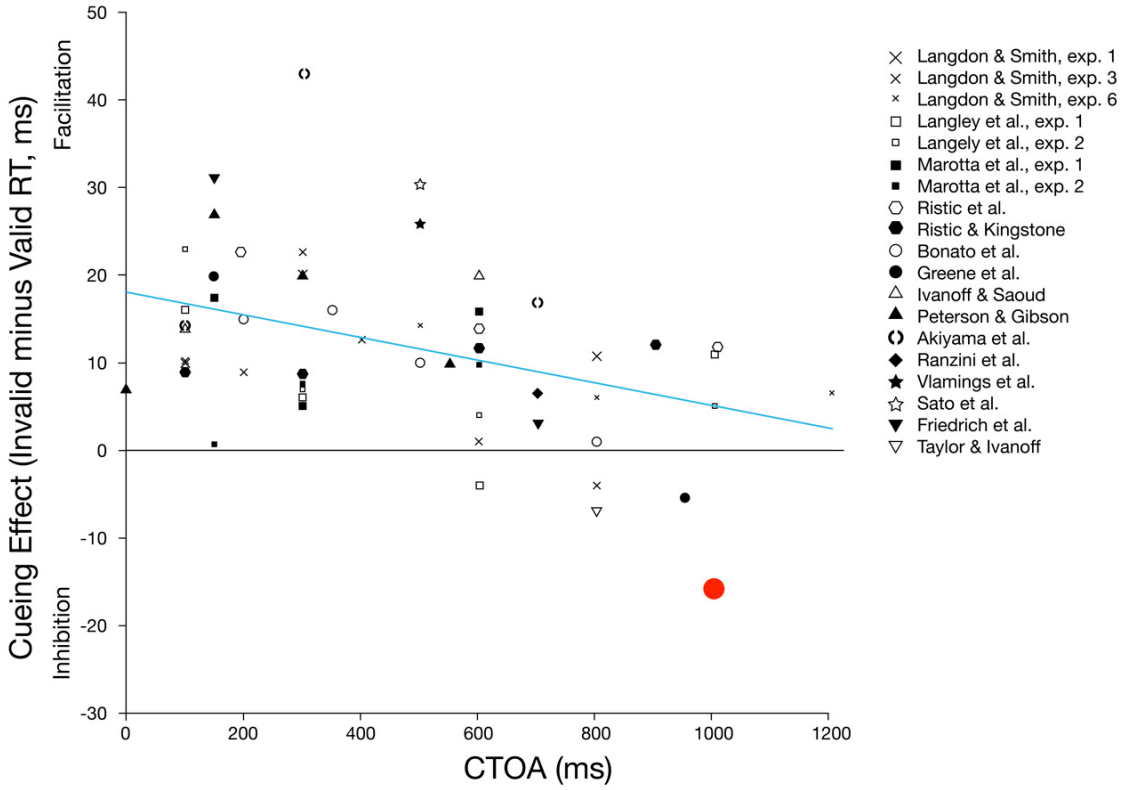

Table 6.A1
Summary of mean response times (in milliseconds) from each experiment, with standard deviations in parentheses.

	Cued	Uncued
Experiment 1 (Mixed Cues)		
Peripheral onset cue	355 (49)	329 (48)
Central arrow cue	333 (42)	336 (48)
Experiment 2 (Mixed Targets)		
Peripheral onset target	357 (41)	349 (45)
Central arrow target	405 (48)	392 (46)

CHAPTER 7:
THE INPUT FORM OF "INHIBITION OF RETURN" REQUIRES THE PRESENCE OF OBJECTS

The (unpublished) manuscript based on this study is presented below. Co-author for this manuscript is Dr. John Christie.

Abstract

One form of inhibition – commonly referred to as inhibition of return (IOR) – occurs in the aftermath of covert or overt oculomotor orienting and serves to lastingly bias responses against previously targeted locations. A neuroanatomically and functionally distinct form of inhibition – also commonly referred to as IOR – occurs in the context of covert visuospatial orienting and lastingly biases processing against previously accessed visual inputs. The extent to which IOR appears as more output- or input-based in the oft-studied cue-target paradigm depends on whether the task used to generate and measure IOR involves reflexively-generated saccadic eye movements or not, respectively. Despite the clear effect of oculomotor orienting on the forms of inhibition (e.g., Taylor & Klein, 2000), it remains to be seen whether the more input-based form of IOR requires the presence of objects. By manipulating the presence of objects across experiments and expressly forbidding reflexively-generated saccadic responses, we demonstrate an input-based form of IOR that is encoded exclusively in object-centered coordinates. The results are considered in the context of extant theory and a quantitative meta-analysis of IOR in static cue-target paradigms.

Introduction

Theories of visuospatial orienting (Posner, Snyder & Davidson, 1980) – a class of attention (Posner & Petersen, 1990; Petersen & Posner, 2012) – have been informed by scores of behavioral and neuropsychological observations from variations on the "simple model task" (Posner, 1980), also known as the "cue-target paradigm" (see Chica, Martin-Arevalo, Botta & Lupianez, 2014, for review). In classic variations of the simple model task, a to-be-ignored, spatially uninformative stimulus (a "cue") appears briefly in a marked peripheral location followed at variable intervals by an imperative manual response stimulus (a "target") at either the cued or uncued location. This method typically yields a biphasic pattern of response time (RT): Manual responding is fastest to targets appearing at cued locations within ~ 200 ms of cue appearance upon which there is a prolonged period of delayed responding toward targets at cued locations (*e.g.,* Posner & Cohen, 1984). The initial facilitatory effect is often considered a consequence of covert, exogenous (involuntary) visuospatial orienting toward the cued location whereas the later inhibitory effect is often presumed to reflect long-lasting biases against returning to exogenously attended locations (see Klein, 2000, for review). The late *inhibitory aftereffect* (Tassinari, Aglioti, Chelazzi, Marzi & Berlucchi, 1987), which is the focus of the present investigation, is frequently referred to as *inhibition of return* (IOR; Posner, Rafal, Choate & Vaughan, 1985), a phrase reflecting the popular belief that attention is inhibited against returning to previously attended locations (Lupianez, 2010).

In the flagship paper on the nature of IOR, Posner et al. (1985) defined and demonstrated IOR's properties. IOR is generated by oculomotor orienting or, in absence its overt expression, covert oculomotor orienting. The late consequence of oculomotor orienting is on biasing output (response/decision-making) pathways – not input (sensory/ perceptual) pathways– against locations to which an oculomotor orienting response had been programmed. The IOR effect is long-lasting and may be expressed in environmental as opposed to retinotopic coordinates (Posner & Cohen, 1984; Vaughan, 1984; Maylor & Hockey, 1985). This seminal characterization of IOR's properties – despite clashing with the unmistakably sensory characterization of inhibition advanced by Posner and Cohen (1980; 1984; see Hilchey, Klein, & Satel, 2014) – ultimately allows for a compelling ecological account; the mechanisms underlying IOR likely operate fundamentally to bias visuospatial analysis toward novelty, thereby improving visual search efficiency (Klein, 1988; see Wang & Klein, 2010, for review). Because of IOR's presumed functional utility, much research has been dedicated to better-understanding the mechanisms underlying it. Accordingly, contemporary thinking dictates that phylogenically primitive substrates of the oculomotor response system have evolved to maximize sampling of the visual environment (e.g., Simion, Valenza, Umilta, & Dalla Barba, 1995; Sapir, Soroker, Berger, & Henik, 1999; Gabay, Leibovich, Ben-Simon, Henik, & Segev, 2013). Environmental encoding is thought to be made possible via remapping contributions from the right lateral intraparietal sulcus (Sapir, Hayes, Henik, Danziger, & Rafal, 2004; van Koningsbruggen, Gabbay, Sapir, Henik, & Rafal, 2010).

Despite the surface appeal of the foregoing account, many have challenged whether it captures all behavioral expressions of IOR, especially in light of evidence that different, dissociable, forms of inhibition can occur in the aftermath of a cue (*e.g.,* Tassinari & Berlucchi, 1993; Tassinari & Berlucchi, 1995). One factor now known to modulate the form of inhibition relates to the oculomotor response demands imposed by the task (Kingstone & Pratt, 1999; Taylor & Klein, 2000; Hunt & Kingstone, 2003; Sumner, Nachev, Vora, Hussain & Kennard, 2004; Berlucchi, 2006; Pratt & Neggers, 2008; Chica, Taylor, Lupianez & Klein, 2010; Zhang & Zhang, 2011; Bourgeois, Chica, Migliaccio, Thiebaut de Schotten, & Bartolomeo, 2012; Bourgeois, Chica, Valero-Cabre & Bartolomeo, 2013; Hilchey, Klein, & Satel, 2014; Hilchey, Hashish, et al., 2014; MacInnes, Kruger, & Hunt, 2015). When reflexive oculomotor orienting is *not* suppressed, as in when saccadic responses are required or perhaps permitted (Hilchey, Hashish et al., 2014), the oculomotor response system responsible for reflexive eye movements generates responses to the cue (Hilchey, Klein &, Satel, 2014); the ensuing form of inhibition has its effect on biasing responding or decision-making against the vector of cue-elicited oculomotor orienting (Taylor & Klein, 2000; Chica et al., 2010; Hilchey, Klein, & Satel, 2014), as suggested by Posner et al. (1985). Because of the nature of the effect, this particular inhibition – or IOR – is often described as nearer the *output-end* of the processing continuum (e.g., Hilchey, Klein & Ivanoff, 2012) and is thought to be mediated principally by retinotectal pathways (Bourgeois et al., 2012). By contrast, when oculomotor responding is expressly discouraged to visual stimuli by way of immediate visual feedback, the inhibitory aftereffect arises outside of the reflexive

oculomotor response system and has its effect on biasing processing specifically against previously accessed visual inputs (Hilchey, Klein & Satel, 2014). Because of the nature of the effect, this particular inhibition is described as nearer the *input-end* of the processing continuum (see Table 7.1) and is thought to relate closely to visuospatial working memory (Zhang & Zhang, 2011) and posterior parietal networks (Bourgeois et al., 2013).

In addition to the input- and output-based dichotomy of IOR, there is the object- and space-based dichotomy of IOR (see Grison, Kessler, Paul, Jordan, & Tipper, 2004, for review). Tipper, Jordan and Weaver (1999) demonstrated that a cue in peripheral vision could generate two dissociable forms of inhibition in dynamic visual displays (see also, e.g., Tipper, Driver, & Weaver, 1991; Tipper, Weaver, Jerreat, & Burak, 1994; Tipper et al., 1997; Weaver, Lupianez, & Watson, 1998; see Reppa, Schmidt, & Leek, 2012, for review). In one version of their paradigm, one of three spatially discrete boxes about a central fixation stimulus was briefly cued. After cueing, the boxes rotated 120 degrees around the circumference of an imaginary circle after which a simple manual detection response was required to visual targets presented in one of the boxes. In addition to demonstrating IOR at the location of the cue, responses were also delayed to targets appearing at the previously cued – and now displaced – object. The results were important insofar as they implied the co-existence of inhibitory mechanisms for both object- and space-based reference frames in dynamic simple model tasks (see also e.g., Weaver, Lupianez, & Watson, 1998; McCrae & Abrams, 2001; Theeuwes, Mathot, & Grainger, 2014), which extended the functional utility of inhibition beyond the

prototypically static (i.e., without moving objects) cue-target paradigm. Interestingly, object-based inhibitory aftereffects – like input-based inhibitory aftereffects – are thought to be mediated by more sophisticated and phylogenically newer cortical networks dedicated to visuospatial working memory (Tipper et al., 1997; Vivas, Humphreys, & Fuentes, 2008). By contrast, space-based inhibitory aftereffects – like output-based inhibitory aftereffects – are thought to be driven principally by low-level oculomotor response systems (see Grison et al., 2004, for review). Contemplation of the origins of these inhibitory aftereffects, and recognition of the crucial role of the oculomotor response system in generating IOR, have prompted statements that, similar to those made in the input- and output-based dichotomy, suggest that the oculomotor response system is critical in gating the generation of inhibition between reference frames. Abrams and Pratt (2000, p. 783), for example, acknowledge that "in particular, when it is important to inhibit covert orienting, an object-based reference frame is invoked perhaps because there is extensive use of such a reference frame in a variety of different covert-orienting situations... When the task involves overt eye movements, a reference frame is used that is consistent with the manner in which eye movements are coded – relative to the retina, or oculocentric".

Despite widespread recognition that space- and object-based IOR effects are dissociable (*e.g.,* Grison et al., 2004), the experimental boundaries on whether the inhibitory aftereffect will be expressed in space-based (*e.g*., McAuliffe, Pratt, & O'Donnell, 2001; Taylor, Chan, Bennett, & Pratt, 2015), object-based (*e.g.,* Birmingham & Pratt, 2005) or, most commonly, both reference frames (*e.g.,* as above, Tipper, Jordan,

& Weaver, 1999; Leek & Reppa, 2003) in static variations on the simple model task remain largely undetermined (see Reppa, Schmidt, & Leek, 2012, for review). At least part of the variability, however, may be accounted for by insufficient experimenter control over involuntarily-generated saccadic eye movements. Indeed, given the shared perspective that the reference frame/expression of IOR depends crucially on the extent to which the task involves oculomotor orienting responses, both literatures predict that at least some variability should be accounted for by uncontrolled oculomotor orienting responses. In other words, space- and output-based effects should be observed *only* if the task involves low-level oculomotor orienting reflexes. Problematically, however, not a single study exploring the consequences of (putatively) covert visuospatial orienting on object-based IOR in static versions of the simple model task with and without objects has both actively (and reliably) monitored and discouraged saccadic eye movement responses via online, trial-by-trial feedback (e.g., Jordan & Tipper, 1998; McAuliffe et al., 2001; Leek, Reppa & Tipper, 2003; Paul & Tipper, 2003; Birmingham & Pratt, 2005; McAuliffe, Chasteen & Pratt, 2006; Pratt & Chasteen, 2007; Possin, Filoteo, Song, & Salmon, 2009; but see, for example, Christ, McCrae, & Abrams, 2002, for an example of eye movement monitoring and discouragement in a dynamic simple model task).

In light of the established role of the activation state of the oculomotor response system in generating distinct, dissociable forms of IOR, it is thus important to evaluate the input-/output- and object-/space-based composition of late cue-elicited inhibition in the covert simple model task under conditions of stringent eye movement control/ discouragement. Thus, the principal objective of the present investigation is to shed light

on the role of object presence on the late inhibitory aftereffect (~1 s post-cue) in a strictly covert cue-target paradigm, which should give rise to a distinctly input-based inhibitory aftereffect.

To address the principal objective, in Experiment 1, standard square placeholders (e.g., Posner & Cohen, 1984) – objects – flank a central fixation placeholder on the left and right side. In Experiment 2, the experimental conditions are identical to Experiment 1 except the peripheral placeholder objects are removed from the design. As is customary, a spatially uninformative peripheral cue requiring no response precedes the manual response target at variable cue-target onset asynchronies (CTOAs). Although our focus is principally a comparison between experiments at the latest ~1 s CTOA, at which IOR is commonly studied, we included shorter CTOAs, to provide a more complete picture of the processing dynamics over time (e.g., Posner & Cohen, 1984; Maylor, 1985; Maylor & Hockey, 1985; Posner, Rafal, Choate & Vaughan, 1985). Fixation brightening occurs between the cue and target (e.g, Briand, Larrison & Sereno, 2000; Pratt & Fischer, 2002; MacPherson, Klein, & Moore, 2003) in order to ensure minimal contribution from cue-elicited attentional facilitation (e.g., which is modulated by placeholder presence, *e.g.*, Taylor et al., 2015). Finally, although we expect that the inhibition in paradigms expressly discouraging reflexive oculomotor orienting will be input-based (*e.g.,* Taylor & Klein, 2000; Hilchey, Klein, & Satel, 2014), we nevertheless randomly intermix two classes of target stimuli (central arrows or peripheral onsets, each requiring left or right manual responses depending on the direction indicated or onset location, respectively) in the present experiments to confirm this expectation. That is, if

(contra our expectations) we observe inhibition from the peripheral cues in response to these centrally presented arrows, then this would imply that responding had been biased against the direction indicated by the cue and thus the IOR would be deemed output-based (*cf* Taylor & Klein, 2000; Hilchey, Klein & Satel, 2014).

Experiment 1

A to-be-ignored spatially uninformative peripheral cue appears at a placeholder object left or right of fixation. At variable CTOAs (150, 250, 350, 450 and 1050 ms), an arrow at fixation (pointing left or right) or peripheral onset target (appearing left or right of fixation) commands a manual response compatible with the direction indicated, or the location of the stimulus. Saccadic eye movements are expressly forbidden by way of immediate visual feedback if eye movements occur at any time during experimental trials. The purpose of the present experiment is two-fold. First and foremost, we require a pattern of data suggesting input-based inhibition from a version of the spatial cueing paradigm in which placeholder objects exist so as to compare the findings against those in an identical condition in which placeholder objects do not exist (i.e., Experiment 2). Second, the sequence of stimuli is virtually identical to that used previously by Hilchey, Klein and Satel (2014; Experiment 1) except the final response is manual, not saccadic; thus, a secondary objective of the present investigation is to evaluate the extent to which the time course of cueing is differentially modulated by response modality.

Methods

Participants

Twelve (6 female and 6 male) undergraduate psychology students from Dalhousie University took part for course credit. All were naive to the purposes of the experiment and reported normal or corrected-to-normal vision. The mean age was 19.5 years.

Apparatus and procedure

The apparatus and procedure were generally similar to that used by Hilchey, Klein and Satel (2014) except manual instead of saccadic localization responses were required to the imperative response stimuli. Note that the sequence of stimulus events, as noted below, is identical to that reported by Hilchey, Klein and Satel (2014).

The experiment was conducted in a dimly lit room. All visual stimuli were white and presented against the black background of a 19" ViewSonic Optiquest (Q95-3) CRT monitor connected to a Mac mini 2,1 with an Intel Core Duo processor at a viewing distance of 22.5". Participant gaze was continuously monitored by way of an EyeLink II head-mounted eye tracking system. A 9-point calibration and validation procedure was implemented to ensure that the eye movement metrics were precise within half a degree of visual angle. Gaze information was recorded every 4 ms. The saccade velocity and acceleration thresholds were set to 30°/s and 8000°/s^2 , respectively. Trials on which these thresholds were exceeded were spontaneously aborted. Participants were apprised of all eye movement (and manual response) errors by way of immediate visual feedback. Manual response time (RT) was computed by subtracting the time at which a keypress response was registered on a standard QWERTY keyboard from the time at which the imperative stimulus appeared onscreen.

See Figure 7.1 for the possible sequence of stimulus events on any given trial. Each trial began with the presentation of three white-outlined placeholder objects (measuring 1.6° x 1.6° of visual angle), separated center to center by 7.9° of visual angle horizontally. To initiate the sequence of stimulus events, the participant performed a drift correction by pressing the spacebar while fixating the center placeholder. The trial did not begin if the participant's gaze did not align with the fixation location. Auditory feedback (a beep) signaled unsuccessful drift correction; in this case, the participant knew to refixate the center placeholder and press the spacebar again.

Successful drift correction triggered a sequence of stimulus events (i.e., a trial). Five hundred ms after drift correction, a to-be-ignored peripheral cue appeared onscreen for 50 ms randomly in the center of the left or right placeholder object. The cue was an asterisk measuring 0.3° in diameter of visual angle. Observers were explicitly informed that the cue was "irrelevant", "meaningless" and "uninformative" to help ensure that they appreciated the inutility of the stimulus. All responses (whether manual or saccadic) were forbidden to the cue. All incorrect responses yielded response-specific warning messages. When an eye movement was registered to the cue (or at any other point during the trial), the trial was terminated by a black screen with the following visual feedback in white "Eye movement detected! Do not move your eyes." When a keypress response was registered before target onset, the trial was replaced by a black screen with the following visual feedback in white "Keypress response detected! Do not make a keypress response unless instructed by a peripheral onset circle or arrow."

Immediately after the cue was extinguished, a cue-back appeared at fixation for 50 ms. The cue-back was generated by increasing the line-width of the center

placeholder object three-fold and served principally to summon any attention drawn to the cue back to fixation. Participants were also instructed to rely on the cue-back as an indicator that the next visual stimulus would require a manual response. Following the offset of the cue-back, a randomly selected 50, 150, 250, 350 or 950 ms interval elapsed upon which a directional arrow appeared at fixation or a visual onset stimulus appeared at the midpoint of a peripheral object location. Both target types required manual responses and occurred at chance levels. In the event of a left- or right- pointing central arrow (1° of visual angle in width), participants were required to press the Q or P key with their left and right index fingers, respectively. In the event of a left- or right-appearing visual onset stimulus (a peripheral target disk measuring 0.3° of visual angle in diameter), participants were required to press the Q or P key with their left or right index finger, respectively. Incorrect manual response at the time of target onset terminated the trial and led to the following visual feedback: "'Keypress Error Detected! Make the response indicated by the onset circle or arrow." All warning messages were acknowledged by the participant by way of a spacebar keypress response which triggered the next trial after a 1 s delay (black screen). If a response was not made to the target within 2 s of its appearance or if the correct response was made, all stimuli were simply removed from the display (i.e., there was a black screen) for 1 s upon which the drift correction phase began anew.

Design

Each participant was exposed to 32 practice trials followed by 480 experimental trials (2 blocks of 240 trials). The design was completely within-subject and comprised the following 3 factors: CTOA (with 5 levels: 150, 250, 350, 450 or 1050 ms), Cueing

(with 2 levels: cued or uncued) and Target Type (with 2 levels: central arrow or peripheral onset). Following Taylor and Klein (2000), a trial was considered "cued" if the location of the cue was compatible with the response required by the target (e.g., left cue followed by a leftward-pointing arrow or left-appearing peripheral onset target).

Results

Data were excluded from analyses for various reasons. Saccadic eye movement artifacts were detected on 22.1% of trials; these trials were not considered further. Of the remaining trials, 0.4% were excluded because no response was registered within the 2 second target response window. Given the skewed and sparse long tail of the RT distribution, a formal assessment of the upper and lower RT cutoffs was undertaken by evaluating mean response accuracy in each of 50 ms RT bins. For the shorter RTs, performance was not at above chance levels until the 200 - 250 ms RT bin. Accordingly, all RTs < 200 ms (1% of trials) were considered anticipatory and were therefore excluded from subsequent analyses. Given the diminishing amounts of data and a decline in response accuracy in the 750-800 ms bins, we reasoned that responses with latencies > 750 ms were not necessarily to the appearance of the target but rather to the recognition of stimulus presence at a later time. As such, all RTs > 750 ms (4.2% of trials) were excluded from analyses. Finally, manual localization accuracy (98.3%) was not formally analyzed because many individuals in multiple cells of the design did not make manual localization errors and, at the group level, several conditions didn't contain any manual localization errors (e.g., peripheral targets at CTOAs> 250). Analyses were conducted only on correct RTs.

Mean correct RTs at each level of factors Cueing (cued or uncued), CTOA (150, 250, 350, 450 and 1050 ms) and Target Type (central arrow or peripheral onset) can be found in Table 7.2. Correct mean RTs were submitted to a 2 x 5 x 2 repeated measures analysis of variance (ANOVA). There was an effect of CTOA [$F(4, 44) = 29.48$, $p = 6.06 \times 10^{-12}$, $MSE = 521$] but not of Target Type or Cueing [$F(1, 11) = 0.03$, $p = 8.72 \times 10^{-1}$, $MSE = 1373$ and $F(1, 11) = 0.01$, $p = 9.63 \times 10^{-1}$, $MSE = 542$, respectively]. An interaction between CTOA and Target Type [$F(4, 44) = 7.24$, $p = 1.43 \times 10^{-4}$, $MSE = 438$] revealed typical preparation functions with fastest RTs for the intermediate CTOAs but the overall effect of CTOA was greater for the peripheral targets and the benefit associated with increasing CTOA was longer lasting with peripheral onset targets. Crucially, Cueing interacted with both Target Type [$F(1, 11) = 8.18$, $p = 1.55 \times 10^{-2}$, $MSE = 556$] and CTOA [$F(4, 44) = 5.75$, $p = 8.18 \times 10^{-4}$ $MSE = 274$]. Generally, there was a tendency of faster responses toward cued relative to uncued locations at shorter CTOAs but slower responses toward cued relative to uncued locations at later CTOAs. Moreover, there was a tendency for slower responses toward cued peripheral onset targets whereas this pattern reversed when measured by central arrow targets (see Figure 7.2 for illustration of the foregoing two-way interactions). There was little evidence for a three-way interaction as indicated by nearly exactly parallel effects in Figure 7.2, save for the 250 ms CTOA [$F(4, 44) = 0.46$, $p = 7.63 \times 10^{-1}$, $MSE = 311$].

Discussion

For present investigative purposes, the most relevant observation is of delayed responding to stimuli appearing at the cued object/location at the 1050 ms CTOA

whereas, at the same CTOA, there is minimal effect of the cue on manual responses to arrows. Thus, consistent with Taylor and Klein (2000), there is limited evidence that manual responding is biased against the cued location at a ~ 1 s CTOA when oculomotor responding is actively discouraged (see also Experiment 2). The finding thus contrasts with conditions in which saccadic eye movements are made to either the cue and/or target, in which case saccadic eye movements (Taylor & Klein, 2000; Hilchey, Klein, & Ivanoff, 2012; Hilchey, Klein, & Satel, 2014) and manual responses (Taylor & Klein, 2000) are biased against the cued location when commanded by central arrows. At this late CTOA, it appears as if the target must appear specifically at the cued location/object in order for us to conclude an input-based form of inhibition.

As can be seen in Figure 7.2, at the early CTOAs (150 - 450 ms), manual responding is facilitated toward the location of a prior spatially uninformative peripheral onset cue when commanded by a central arrow stimulus. These findings compliment a long history of research on manual response/spatial priming demonstrating that a stimulus associated with a response (in this case the peripheral onset cue which doubles as a target) generates a tendency to react toward the source of stimulation with the nearest corresponding effector and/or in this particular case a tendency to prime the task-relevant conditioned response pathway (e.g., Simon, Acosta, Mewaldt & Speidel, 1976; de Jong, Liang, & Lauber, 1994; Kornblum & Lee, 1995; Prinz, 1997; Valle-Inclan & Redondo, 1998). In the context of dual-route models (e.g., de Jong, Liang, & Lauber, 1994), the findings are aptly accounted for by residual, cue-elicited activation of the unconditioned response pathway (i.e., the natural tendency to respond toward the source

of stimulation) and/or the conditioned response pathway (i.e., the manual responses associated with the locations of peripheral onset stimuli). We note also that unlike when saccadic eye movements are required to peripheral onset stimuli (Hilchey, Klein, & Satel, 2014; see also, e.g., Briand, Larrison, & Sereno, 2000; Khatoon, Briand, & Sereno, 2002), there is no robust inhibitory consequence (in behavior) of repeated stimulation of input pathways at CTOAs < 450 ms when oculomotor responding is forbidden (see also Hilchey, Klein, & Satel, 2014; Experiment 2, for discussion). These latter findings conflict somewhat with prior investigations requiring 2-alternative forced choice manual localization responses to cued peripheral stimuli at earlier CTOAs (<450 ms) which have demonstrated evidence of inhibition without eye movement monitoring or cue-backs (e.g., Maylor, 1985; Tanaka & Shimojo, 1996). We speculate that there is an effect of repeat sensory stimulation at CTOAs < 450 ms that is weaker for manual than saccadic responding and that this effect, in the present design, may be overshadowed by cue-elicited manual response/spatial priming.

Finally, although the findings at the 1050 ms CTOA are consistent with an input-based inhibitory attribution, we note that the present findings do not yet require this attribution. Consideration of the nearly parallel lines in Figure 7.2 allows for the possibility that responding over time simply becomes delayed toward the cued location, regardless of target type. This particular characterization of the data ultimately ascribes the inhibition at the 1050 ms CTOA to a more output-based effect (e.g., Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987; Pastotter, Hanslmayr, & Bauml, 2008).

While such an interpretation is perhaps consideration-worthy in the absence of a three-way interaction, the results of Experiment 2 undermine it.

Experiment 2

Assuming that the inhibition at the 1050 ms CTOA in Experiment 1 is not output-based, what remains to be addressed is whether this inhibition requires the presence of peripheral placeholder objects (*i.e.*, is it object-based?). To the extent that both the space- and output-based effects are caused by oculomotor orienting reflexes (e.g., Hilchey, Klein & Satel, 2014; Grison et al., 2004), there should be little evidence of an inhibitory aftereffect at the 1050 ms CTOA in the present experiment. To evaluate this possibility, we preserved every aspect of the procedure in Experiment 1 except that there were no placeholder objects in Experiment 2.

Methods

Participants

Twelve (9 female and 3 male) undergraduate psychology students from Dalhousie University took part for course credit. All were naive to the purposes of the experiment, had not participated in Experiment 1, and reported normal or corrected-to-normal vision. The mean age was 19.5 years.

Apparatus and procedure

All aspects of the Experiment were identical to Experiment 1 except there were no peripheral placeholder objects.

Design

This was identical to Experiment 1.

Results

Data were excluded from analyses for various reasons. Saccadic eye movement artifacts were detected on 21.2% of trials; these trials were not considered further. Of the remaining trials, 3.5% were excluded because no response was registered within the 2 second target response window. As in Experiment 1, RT cutoffs were established by evaluating mean response accuracy in each of 50 ms RT bins. Excluding all RTs < 200 ms again appeared judicious, leading to the exclusion of 1.4% of trials. Determining the upper bound was more challenging. Accuracy declined in the 650-700 ms bin as well as the 850-900 ms bin. In the interest of maintaining similar cutoffs between Experiments we again opted for the 750 ms cutoff, leading to the exclusion of 5.9% of trials. Again, accuracy on the remaining (>4300) trials was high (98.9%); analyses were conducted only on correct RTs.

Mean correct RTs at each level of factors Cueing (cued or uncued), CTOA (150, 250, 350, 450 and 1050 ms) and Target Type (central arrow or peripheral onset) can be found in Table 7.2. Correct mean RTs were submitted to a 2 x 5 x 2 repeated measures analysis of variance (ANOVA). As in Experiment 1, there was an effect of CTOA [$F(4, 44) = 10.89$, $p = 3.17 \times 10^{-6}$, $MSE = 980$] but little evidence of an effect of Target Type or Cueing [$F(1, 11) = 0.07$, $p = 8.04 \times 10^{-1}$, $MSE = 3756$ and $F(1, 11) = 0.39$, $p = 5.44 \times 10^{-1}$, $MSE = 1719$, respectively]. There was again an interaction between Target Type and CTOA [$F(4, 44) = 2.65$, $p = 4.58 \times 10^{-2}$, $MSE = 484$] of the sort acknowledged

earlier. Cueing did not appear to be modulated by Target Type [$F(1, 11) = 2.37$, $p = 1.52 \times 10^{-1}$, $MSE = 2350$] or CTOA [$F(4, 44) = 1.44$, $p = 2.36 \times 10^{-1}$, $MSE = 591$].

Intriguingly, there was a three-way interaction [$F(4, 44) = 2.93$, $p = 3.14 \times 10^{-2}$, $MSE = 526$]. As can be seen in Figure 7.3, cued central arrow targets were responded to more quickly than uncued central arrow targets at the 150 ms, 250 ms, and 350 ms CTOA whereas there was no evidence of a facilitatory effect of the cue on central arrow targets at the 450 ms and 1050 ms CTOAs. By contrast, there was little evidence that Cueing modulated responding to peripheral targets, though mathematically, the trend was generally opposite the trend that was observed with central arrow targets. Finally, given that the most analytical condition for the principal question of the present investigation concerns whether object-presence matters for input-based IOR, we note that a planned comparison between experiments on the Cueing effects for peripheral onset targets at the latest CTOA revealed greater inhibition with objects [$t(22) = 2.39$, $p = 0.026$, 95% CI = 5.08 - 72.59 ms].

Discussion

In many respects, the early (CTOA < = 450 ms) cueing effects in Experiment 2 are reminiscent of those in Experiment 1 (see Figures 7.2 and 7.3). Early on, responding is faster toward the cued location when prompted by a central arrow target whereas there is little indication of an effect on responding to peripheral targets. However, contrasting with the results of Experiment 1, at the 1050 ms CTOA there is no evidence for inhibition. This pattern undermines the alternative hypothesis concerning the possibility of an output-based inhibition at the 1050 ms CTOA in Experiment 1 (i.e., it was in fact

input-based) while simultaneously elucidating that the inhibition in the prior experiment was due to placeholder object presence. In other words, the input-based inhibitory aftereffect at the late CTOA was object-based and did not co-occur with space- and/or output-based inhibitory aftereffects. Given the emphasis that has historically been placed on low-level sensory/attentional mechanisms for input-based IOR (e.g., Posner & Cohen, 1980; 1984; Handy, Jha, & Mangun, 1999; Taylor & Klein, 2000; Li & Lin, 2002a; 2002b; Hunt & Kingstone, 2003; Fecteau, Bell, & Munoz, 2004; Fecteau & Munoz, 2005; 2006; Muller & Kleinschmidt, 2007; Dukewich & Boehnke, 2008; Prime & Jolicoeur, 2009; Satel, Wang, Trappenberg, & Klein, 2011; Smith, Ball, & Ellison, 2012), we note briefly that our findings suggest that repeat stimulation of an input pathway is not sufficient for the inhibitory aftereffect.

General discussion

Taken together, the data from Experiments 1 and 2 strongly support the contention that late input-based, cue-elicited inhibition is object-based. Responding was *not* generally biased against the cued location and there was no evidence of inhibition in the absence of placeholder objects at the late CTOA. In other words, the observed inhibition was distinctly object- and input-based with little evidence of output- and/or space-based contributions.

Our key finding generally compliments inferences drawn by others: Object-presence matters for IOR (e.g., Jordan & Tipper, 1998); but the present paper goes a step further by identifying that object-presence matters for the input-based form of it and by demonstrating that this form may not comprise space-based properties, as suggested by

others. Although this investigation is the first of its kind to directly target space- and object-based properties in the context of an empirically-validated input-based form of IOR, we note that a swath of studies have nevertheless reported reduced cue-elicited inhibition in object-absent as compared to object-present conditions when measured by way of simple keypress responding in variations on static, "covert", simple model tasks (Possin, Filoteo, Song, & Salmon, 2009; Pratt & Chasteen, 2007; McAuliffe, Chasteen & Pratt, 2006; Birmingham & Pratt, 2005; Paul & Tipper, 2003; Leek, Reppa & Tipper, 2003; Jordan & Tipper, 1998; but see McAuliffe, Pratt & O'Donnell, 2001). As noted at the outset, there is disagreement among these studies, but to determine whether object presence generally modulates the magnitude of cue-elicited inhibition in these paradigms, cueing effects and their standard errors were extracted from research reports using late CTOAs in which a manual response target appeared randomly at distance-matched cued or uncued locations marked or unmarked by objects, or apparent objects (e.g., Jordan & Tipper, 1998). A meta-analysis was then performed, illustrated by the forest plot in Figure 7.4.

As shown by Figure 7.4, there is compelling support that the magnitude of the cue-elicited inhibition is greater in object- present than object- absent designs. Note, however, that most papers report a diminished, not non-existent, IOR effect in the absence of objects and that there is more heterogeneity among the effects than would be expected by the sampling variances, as foreshadowed during the introduction and now demonstrated by the funnel plot in Figure 7.5. Thus, evidently one or more unaccounted factors are modulating effects (see Reppa, Schmidt, & Leek, 2012, for discussion of

candidate moderators). Intriguingly, the lone experiment (barring the current one) in this literature failing to observe any compelling inhibitory aftereffect from a peripheral cue in an object-absent condition is also the only experiment that monitored observer gaze (Birmingham & Pratt, 2005; but see also, e.g., Paul & Tipper, 2003, for considerably diminished or absent effects in sequential cueing paradigms at the 2, 3, 4 and 5 -back cued locations). Although it is not clear from the methods whether erroneous eye movements were strictly discouraged, when considered together with the present results, it seems reasonable to suggest that at least some heterogeneity may be due to insufficient experimental control over the activation state of the oculomotor response system, as already noted by others (*e.g.,* Abrams & Pratt, 2000; Grison et al., 2004). In that way, it becomes tenable that previous investigations *not* actively discouraging task-inappropriate saccadic eye movements obtained evidence for what appeared to be concurrent, additive space- and object-based effects because, on some fraction of the trials (or in some individuals), only object-based inhibition was in effect, whereas on the remaining trials (or in some individuals), only space-based inhibition was in effect (see also Hilchey, Hashish et al, 2014, for further discussion).

Indeed, returning to the theme of the present investigation, the reader likely wonders whether the space- and object-based dissociation on cue-elicited inhibition can be *completely* reconciled with the output- and input- (object-) based dissociation. We offer a tentative affirmative response at this point. The present findings strongly suggests that input-based inhibition is object-, not space-based. If the oculomotor response system is not suppressed, output-/space-based inhibition likely occurs (*e.g.,*

Taylor & Klein, 2000; Hilchey, Klein, & Satel, 2014) but whether this inhibition can be/is (*e.g.,* Abrams & Dobkin, 1994a; Tas, Dodd & Hollingworth, 2012) or cannot be/is not (*e.g.,* Abrams & Pratt, 2000; Souto & Kerzel, 2009; Redden, Hilchey, & Klein, 2014b) recoded into object-centered coordinates remains controversial.

In the interest of forward progress, we consider also that in visual search paradigms without cueing there is strong support that IOR depends on scene presence (e.g. Muller & von Muhlenen, 2000; Takeda & Yagi, 2000), even when visual search is definitively accomplished via overt oculomotor orienting responses (Klein & MacInnes, 1999; MacInnes & Klein, 2003; Hooge, Over, van Wezel, & Frens, 2005); ultimately, there may not be inhibitory aftereffect without a scene in visual search, which could be interpreted to suggest that object presence matters even for output-based IOR. But consider also that in cue-target paradigms involving saccadic responses, IOR is frequently observed in the absence of a visual scene and placeholder objects (Vaughan, 1984; Abrams & Dobkin, 1994a; Abrams & Dobkin, 1994b; Watanabe, 2001; Pertzov, Zohary, & Avidan, 2010; Mathot & Theeuwes, 2012; Hilchey, Klein, Satel & Wang, 2012; Wang, Satel, Hilchey, & Klein, 2012; Christie, Hilchey, & Klein, 2013). The discrepancy is palpable. One possibility is that complex visual environments containing many salient objects (see, e.g.,Tipper, Jordan & Weaver, 1999; Jordan & Tipper, 1999; Kessler & Tipper, 2004, for manipulations of object salience) invoke object-based inhibition (e.g., Tipper, Jordan, & Weaver, 1999; Klein, 2004; Boot, McCarley, Kramer, & Peterson, 2004). But consider also the possibility that visual search tasks might fundamentally alter the activation state of the oculomotor response system, thereby

generating input-based effects. It may be that exogenously-generated oculomotor orienting in more complex, conjunctive visual search tasks is the exception rather than the rule (e.g., Smith, Ball, & Ellison, 2014; Klein & Farrell, 1989); therefore the inhibition in such tasks may not arise from exogenous oculomotor orienting (and, ironically, might not be classified as IOR proper as defined by Posner et al., 1985). Rather, phasic activation or suppression of oculomotor orienting, in accordance with top-down goals, would allow object-based encoding to take priority and its inhibition to be generated in neuroanatomically and functionally distinct cortical pathways. Relatedly, consider a key difference between natural visual search tasks and the spatial cueing paradigm. Experimentally-induced saccadic eye movements in the simple model task – for example, no response to the cue - saccade to the target or saccade to the cue -manual response to the target – are seldom in the service of perceptual information processing. Rather, the oculomotor response is the goal in and of itself, not the perceptual information that the eye movement brings into focus, which is often either non-existent at the time of foveation or immaterial. By contrast, during natural search, scanning of the visual environment is expressly for the purposes of evaluating potentially relevant behavioral inputs in accordance with observer goals. In light of these considerations, further research manipulating observer objectives during more complex, real-world visual search (see, e.g., Thomas & Lleras, 2009; Dodd, van der Stigchel, & Hollingworth, 2009) and in variations on the simple model task is needed.

Finally, we wish to conclude first by noting that the present findings and first quantitative meta-analysis in this literature accord with the assertion that object presence

matters for late cue-elicited inhibition in variations on the "covert" simple model task. Beyond the literature – and having benefited from over a decade of research since many of these seminal investigations – we recognized that there are at least two dissociable forms of IOR, input- and output-based, which had been confounded and largely neglected experimentally by the literature on object- and space-based IOR. We therefore deliberately generated an input-based inhibitory aftereffect – which was empirically-validated by the central arrow target diagnostic – so as to unequivocally evaluate its object- and/or space- based properties. This distinctly input-based inhibitory aftereffect was clearly object-based, without a trace of the space-based component that is presumed generated by the reflexive oculomotor orienting system. Future research is encouraged and required to determine the relationships between definitively output-based IOR and its capacity for object-based representation.

Acknowledgment

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Table 7.1

Characterization of two dissociable forms of inhibitory aftereffects that follow from a spatially uninformative cue, gated by the activation state of the oculomotor response system responsible for reflexively generated oculomotor responses.

Input	Defining concepts	Output
any visual stimulus	cause	oculomotor activation
reduces efficiency of target detection	effect	motor bias
suppressed	state of reflexive oculomotor response system	not suppressed
inhibition	name	inhibition of return
object-based?	reference frame	space-based

Table 7.2
Mean RTs for all combinations of factors Cueing, CTOA, Target Type and Experiment.

Experiment 1: Placeholders Present

	Central Targets			Peripheral Targets		
CTOA (ms)	Cued RT	Uncued RT	Effect	Cued RT	Uncued RT	Effect
150	422	439	17	460	452	-8
250	398	414	16	404	412	8
350	395	409	14	397	387	-10
450	403	408	5	406	396	-10
1050	425	414	-11	415	389	-26

Experiment 2: Placeholders Absent

	Central Targets			Peripheral Targets		
CTOA (ms)	Cued RT	Uncued RT	Effect	Cued RT	Uncued RT	Effect
150	436	469	33	479	465	-14
250	418	440	22	431	429	-2
350	421	436	15	433	422	-11
450	429	424	-5	435	419	-16
1050	445	445	0	429	442	13

Figure 7.1

Visual representation of the methods for Experiments 1 and 2 (see Method for more detail).

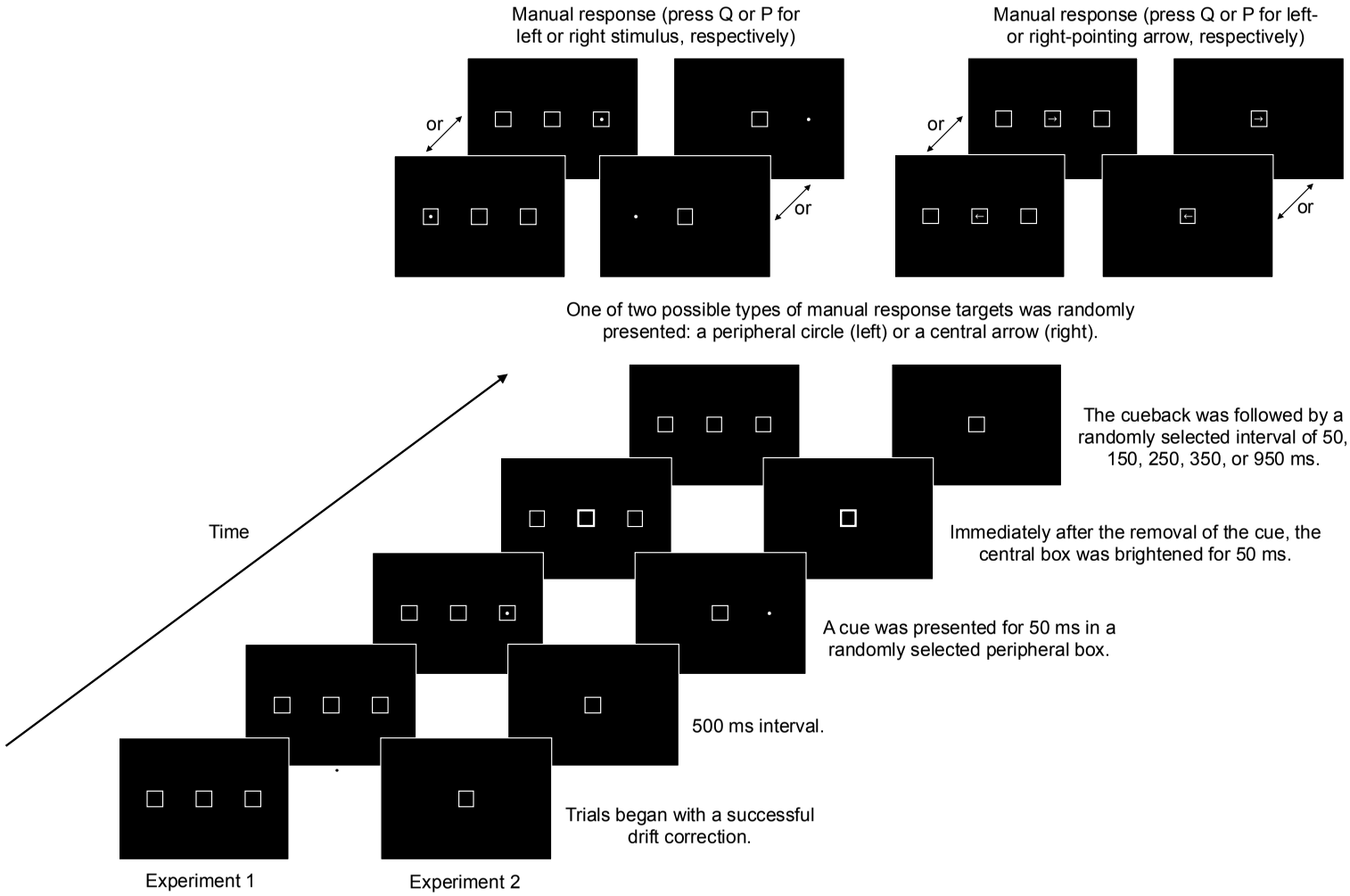


Figure 7.2

Mean Cueing effects (uncued RT - cued RT) for all combinations of factors CTOA and Target Type in Experiment 1 (placeholder present design). Errors bars are 95% confidence intervals (CIs). Note that the data points for central arrow targets have been shifted 20 ms to the right to facilitate examination of the CIs.

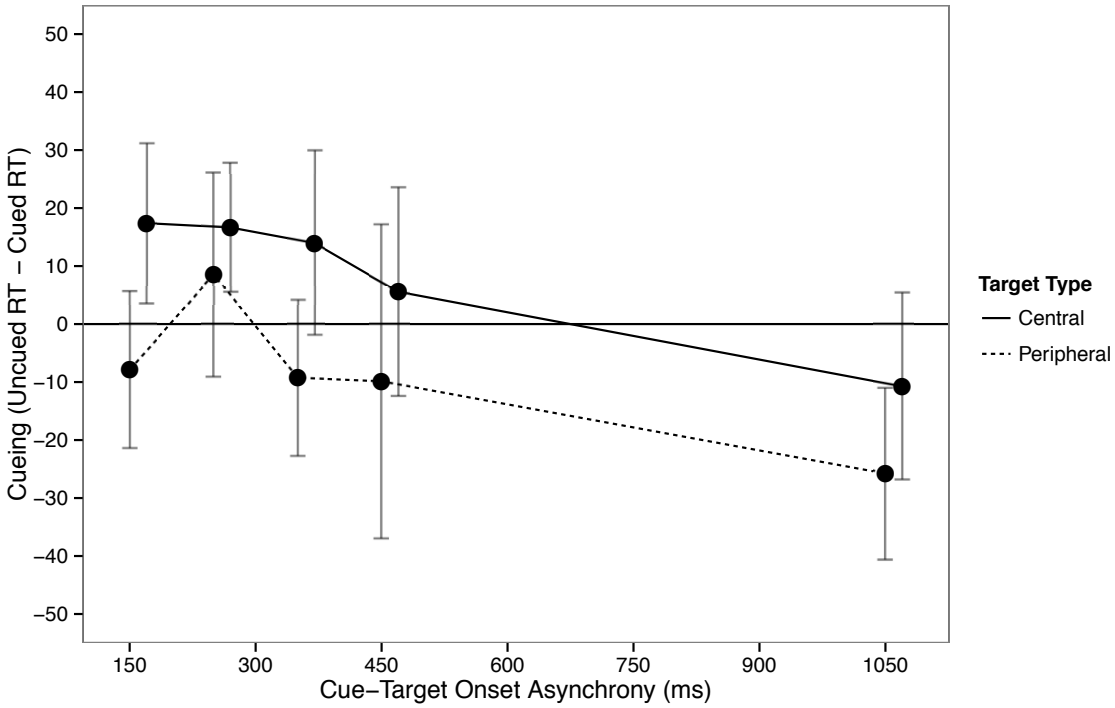


Figure 7.3

Mean Cueing effects (uncued RT - cued RT) for all combinations of factors CTOA and Target Type in Experiment 2 (placeholder absent design). Errors bars are 95% CIs. Note that the data points for central arrow targets have been shifted 20 ms to the right to facilitate examination of the CIs.

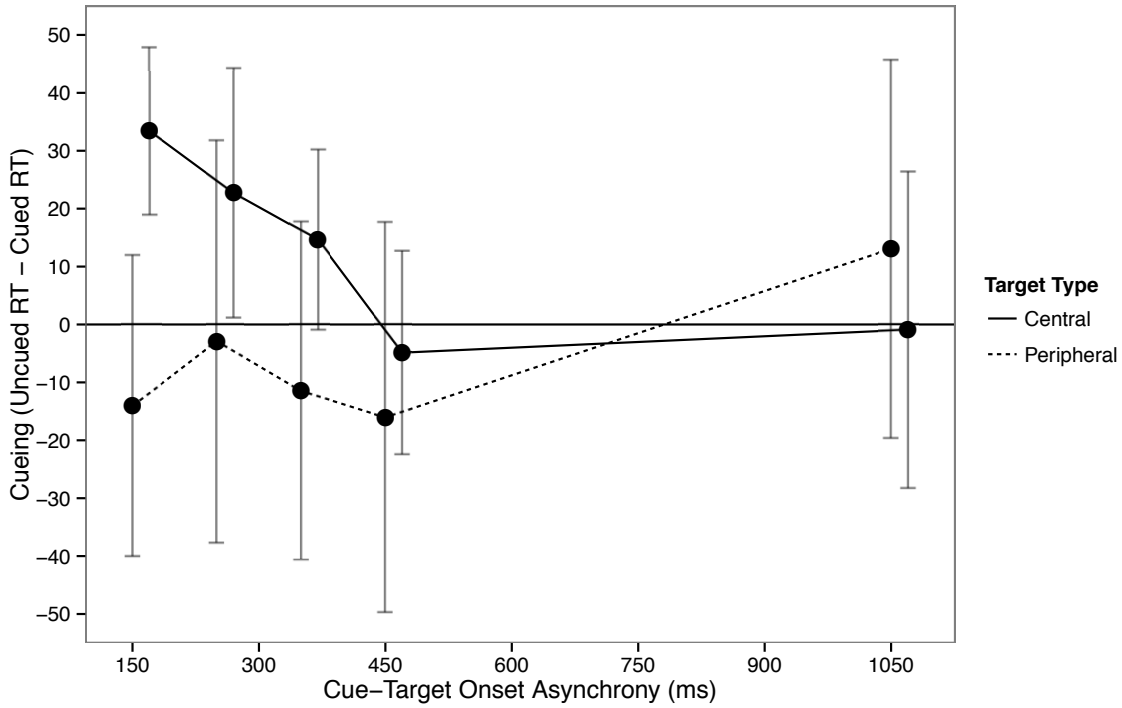


Figure 7.4

A forest plot of research reports administering stationary versions of the cue-target paradigms in which placeholder presence was manipulated, generated by the 'metafor' package in R (Viechtbauer, 2010). A positive mean difference denotes a larger cue-elicited inhibitory effect with placeholder presence than absence. Response type is simple manual detection. All CTOAs > 400 ms. In the event of a sequential cueing procedure (e.g., Paul & Tipper, 2003; Pratt & Chasteen, 2007), only the most recently cued location was considered. In the event of multiple CTOAs => 400 ms (McAuliffe et al., 2001), the data were collapsed across CTOAs. The size of the effect squares denotes relative weighting. The best estimate of the population mean difference between cue-elicited inhibition in placeholder present versus absent designs as determined by a random mixed effects model fitted by restricted maximum likelihood was 12.93 ms (SE = 3.02; z = 4.28, p < 0.05). Although the model favored a larger inhibitory aftereffect of the cue in placeholder present design, caution is advised given that the extant literature is characterized by significant heterogeneity among the sampling means [Q(11) = 24.52, p < 0.05, 54.35% (I^2) of the variability in the effect size estimate is due to heterogeneity in the effect), suggesting the involvement of moderating factor(s).

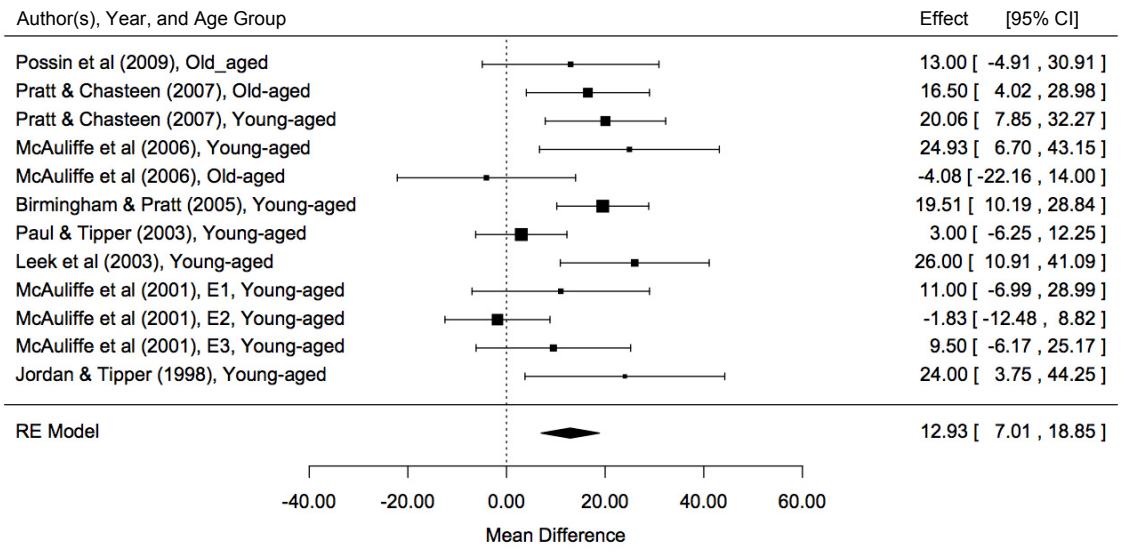
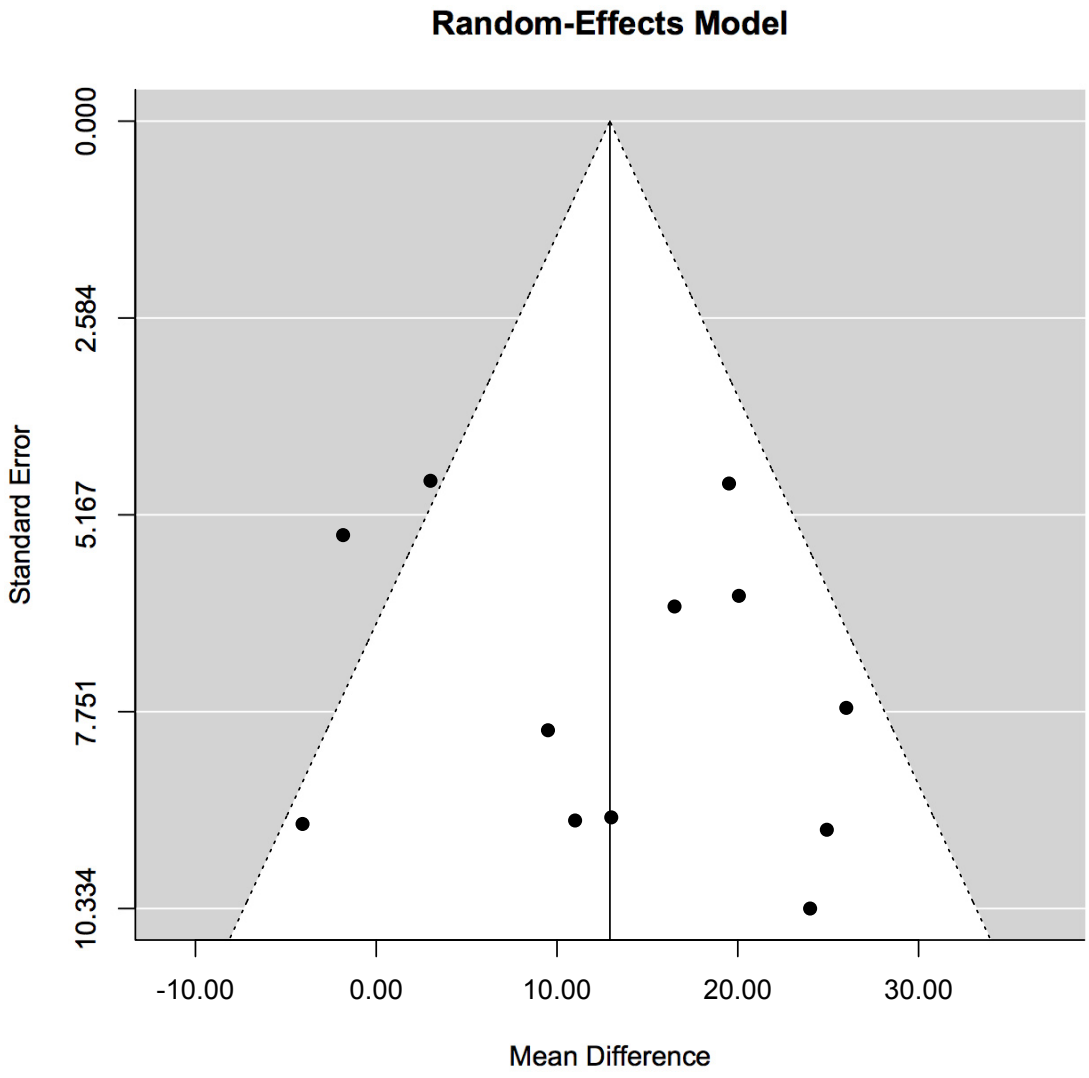


Figure 7.5

A funnel plot (based on a random effects model) illustrating the observed study outcomes and their corresponding standard errors. The vertical line denotes the estimated mean difference from the random effects model and the funnel represents hypothetical confidence intervals for given standard errors.



CHAPTER 8: CONCLUSION

The principal objective of this dissertation was to experimentally establish the boundaries on and properties of the dissociable forms of late [~ 1 s cue-target onset asynchrony (CTOA)] cue-elicited "inhibition" that arise in variations of the cueing paradigm (Taylor & Klein, 2000). Sensitive to Berlucchi (2006)'s concern that different forms of inhibition are conflated by the locution "inhibition of return" (IOR) and cognizant of confusion that arises when fundamentally distinct phenomena are united under the guise of IOR (Dukewich & Klein, 2015), special pains were taken to determine which form(s) of inhibition did and did not conform to the parameters of IOR, as originally described and demonstrated by Posner, Rafal, Choate and Vaughan (1985). Herein, clear lines of demarcation could be (and were) drawn between IOR proper – in the sense intended by Posner et al (1985) – and the forms of inhibition that are generated and/or expressed in ways that do not warrant the monicker IOR. One such line – occurring between the decidedly more input- and output- based late inhibitory aftermaths of cueing – dovetails neatly with the line hypothesized by Taylor and Klein (2000) involving the activation state of the oculomotor response system, which was later refined by Klein and Hilchey (2011). Because this dissertation has focussed most strictly on empirically-validating the boundary hypothesized by Taylor and Klein (2000), the boundaries – as they appear – will be discussed most thoroughly. Nevertheless, each form of inhibition identified by the present anthology of reports and its relationship (or lack thereof) to other forms of inhibition, especially IOR, will be reviewed. Because several reports herein manipulated the interval between cue and target onset to develop a

better sense of the processing dynamics of cueing over time, the relationships between early cue-elicited facilitatory effects and later and/or parallel forms of inhibition will likewise be discussed.

Late output-based inhibition (IOR)

As explained at the outset, one might reasonably refer to an inhibitory consequence as IOR if caused by covert or overt oculomotor orienting and expressed as a long-lasting motor/decision bias against responding (oculomotor or otherwise) against the location(s) of prior oculomotor orienting (Posner et al., 1985). Imported from Posner and Cohen (1984) and Maylor and Hockey (1985), is the wisdom that this form of IOR *should* be encoded in spatiotopic as opposed to retinotopic reference frames, a functional prerequisite that allows IOR to operate efficiently by biasing responding/orienting against already-traversed, physical space.

Using the arrow target diagnostic whenever possible to evaluate whether the form of inhibition generated by the cue resulted in a response/decision bias – and therefore conformed to the output effect criterion ascribed to IOR – I reported two instances in which this criterion was satisfied and in which this bias was plausibly caused by either covert or overt oculomotor orienting to the cue. In this dissertation's maiden experiment, saccadic eye movements were biased against a cued location at the ~ 1 s CTOA by roughly equivalent amounts regardless of whether commanded by an arrow appearing on the fovea or a target appearing in peripheral vision (see also Taylor & Klein, 2000; Hilchey, Klein & Ivanoff, 2012). The finding suggested that oculomotor responding was generally biased against the direction indicated by the cue and strongly

undermined prevailing input-based interpretations of the effect positing a major role of repeat stimulation of input pathways (*e.g.,* Posner & Cohen, 1984; Rafal, Egly & Rhodes, 1994; Abrams & Dobkin, 1994a; Fecteau & Munoz, 2005; 2006). Importantly, whereas oculomotor orienting was later biased against the location indicated by the cue, oculomotor orienting was first biased toward the cue, as foreshadowed by extracellular recordings in the monkey superior colliculus (Fecteau & Munoz, 2005; Dorris et al., 2002). Thus, in cause and effect, the inhibition in Experiment 1 of Chapter 2 could have plausibly been referred to as IOR. However, in earnest, whether the late inhibition observed in Experiment 1 was actually IOR was not clear until the subsequent experiment. In Experiment 2, I expressly forbade eye movement responses to all peripheral stimuli, instead requiring manual localization responses, but continued to probe the oculomotor response system with occasional central arrow targets. In this case, the cue did not bias arrow-elicited oculomotor responding at early or late CTOAs. Thus, strict discouragement of eye movement responses to stimuli appearing in peripheral vision was sufficient for abolishing covert, cue-elicited oculomotor orienting and, consequently, IOR.

The second instance of an output-based inhibition appeared in Chapter 6. Whereas the initial experiments were instrumental for demonstrating a link between cue-elicited oculomotor facilitation and inhibition, they did not speak to another important criterion for establishing IOR. Recall that whereas IOR ought to be generated fundamentally by oculomotor orienting, it should not be confined to oculomotor responses. That is to say that IOR, when generated, should also bias directionally-

compatible non-oculomotor responses against the location of the cue. Foreknowledge that manual responses (left or right) to arrow targets (pointing left or right) are not biased against the location of the cue (*e.g.,* Fischer, Pratt & Neggers, 2003) when oculomotor responses are expressly forbidden (Chapter 2) whereas they are when oculomotor responses are required (Taylor & Klein, 2000) provided a reasonably solid empirical foundation for believing that inhibition, when involving oculomotor orienting, would affect the skeletal-motor system (see also Posner et al., 1985, Experiment 3). However, the most apt (and only) demonstration of inhibited manual responding to central arrow targets commanding responses either compatible or incompatible with a prior saccade vector was one in which the cues and targets shared saccadic and manual response associations (*i.e.,* Taylor & Klein, 2000); simply, in Taylor and Klein, central arrow and peripheral onset saccade cues doubled as manual response targets 1 s later. When one considers further that manual response/spatial priming can generate inhibition (*cf* Chapter 5), in line with activation-suppression models, it became tenable that the inhibition that Taylor and Klein (2000) reported in their "saccade-manual" cell was intrinsically generated by the skeletal-motor system and thus, may not be classifiable as IOR proper. This ambiguity, however, was easily resolved in Chapter 6 in the "pro-saccade" condition. When central arrow saccade cues and peripheral onset manual response targets were removed from Taylor and Klein (2000)'s design, manual responding to arrow targets was nevertheless biased against the saccade direction generated by a peripheral onset cue, consistent with an output-based attribution. Because this output-based form of inhibition does not occur when eye movements are

discouraged to spatially uninformative peripheral cues, it becomes obvious that the inhibition was caused by the eye movement. Clearly then, oculomotor responding toward the source of stimulation is sufficient for engendering response biases – inhibition – that go on to affect non-oculomotor response systems, simpatico with IOR proper. Because such response/decision biases are not manifest when oculomotor orienting responses are either expressly discouraged to stimuli appearing in peripheral vision (Chapter 2, Experiment 2; Chapter 7, Experiments 1 and 2) or in which pro-saccadic orienting responses are especially maladaptive (Chapter 6, "anti-saccade" condition), it becomes clear that IOR occurs principally when the oculomotor response system responsible for reflexively-generated saccadic eye movements is *not* in a tonically suppressed state (*i.e.,* Klein & Hilchey, 2011).

Having established in Chapter 2 – later reinforced by Chapters 4 and 6 – that the inhibition generated by spatially uninformative cues is likely IOR when reflexive, but not non-reflexive, eye movements are permitted/required to visual stimuli appearing in peripheral vision, it became increasingly important to evaluate 1) whether this form of inhibition could be encoded in spatiotopic coordinates, a *sine qua non* for IOR but also 2) the expediency of spatiotopic encoding, which was challenged empirically by Mathot and Theeuwes (2010) but also theoretically by the gradual retinotopic-spatiotopic remapping framework for visual stability. Thus, we simply required two pro-saccadic responses for select visual stimuli appearing in peripheral vision. The initial pro-saccadic eye movement, between the cue and target, dissociated the retinotopic from spatiotopic reference frame. The subsequent eye movement was to an ocuolomotor response probe –

whose appearance was contingent on the execution of the first eye movement – that appeared at cued or uncued locations in spatiotopic or retinotopic coordinates (see Chapter 3). Because only pro-saccadic responses were involved, the experimental conditions were favorable to IOR proper. Because spatiotopic coordinates were dissociated from retinotopic coordinates, we had the opportunity to evaluate whether the inhibition conformed to the environmental-referencing criterion of IOR (as shown by others, *e.g.,* Vaughan, 1984; Boot et al., 2004; Pertzov et al., 2010; Mathot & Theeuwes, 2010). And because the oculomotor response probe was contingent on the execution of the first saccadic eye movement, we had the opportunity to evaluate whether an inhibitory tag was available in spatiotopic coordinates at the earliest possible testing interval subsequent to the first eye movement. For present purposes, the key findings were that spatiotopic inhibition was as great if not greater than retinotopic inhibition at the earliest possible behavioral testing intervals and retinotopic and spatiotopic inhibition were completely dissociable from saccadic/attentional momentum, two findings that are consistent with IOR and its ecological significance.

Late input-based inhibition

But not all forms of inhibition generated by spatially uninformative peripheral cues satisfy criteria of IOR. In Chapter 2, Experiment 2, oculomotor responding to central arrows was largely unaffected by peripheral cues when eye movements responses to all peripheral stimuli were expressly forbidden; yet, manual localization responses were retarded when the cue and target occupied the same location. In Chapter 4, in which the effect of peripheral cueing was evaluated at ~ 500 ms and ~1 s CTOAs in a go

no-go task strictly discouraging oculomotor responses, the inhibition was not expressed as a speed-accuracy tradeoff as would be expected by a mechanism that biased responding and/or decision making but left intact the accrual of sensory/perceptual information (i.e., IOR); rather, false alarm rates were mathematically greater at the cued location despite slower responding, consistent with a more input-based interpretation. The foregoing finding contrasts markedly with prior go no-go tasks (e.g., Ivanoff & Klein, 2001) not actively discouraging untoward oculomotor responding, in which case the inhibition is expressed as a speed-accuracy tradeoff. In Chapter 6, I could detect little to no evidence of a response bias using the central arrow target diagnostic at a 1 s CTOA when an anti-saccade preceded a central arrow, manual response target. In Chapter 7, in which the experimental conditions were identical to those in Chapter 2 involving saccadic eye movements except placeholder/object presence was manipulated and manual instead of saccadic responses were required, there was again little evidence that the cue engendered an output-based inhibitory aftereffect. That is to say that when pro-saccadic eye movements were expressly discouraged to all visual stimuli appearing in peripheral vision, manual responding to central arrows appeared to be largely unaffected by cueing. All of these decidedly more input-based inhibitory aftereffects occurred specifically when reflexive oculomotor responding to peripheral visual stimuli was strictly forbidden. Thus, collectively, the findings lend credence to the notion that tonic suppression over the oculomotor response systems responsible for reflexive saccades generates a fundamentally distinct form of inhibition, easily dissociated from IOR proper.

But what, fundamentally, is the cause of input-based inhibition? Whereas the cause and effect of IOR proper appears reasonably well-established, the cause of the input-based form of inhibition remains somewhat elusive. Nevertheless, clues about the genesis of this effect can be found in Chapters 5 and 7. In Chapter 5, I was intrigued by Taylor and Klein (2000)'s finding of slowed manual responding to peripheral onset targets when preceded by a to-be-ignored central arrow cue at a 1 s CTOA. I was intrigued by this finding principally because to-be-ignored spatially uninformative arrows very rarely generate inhibitory aftereffects at 1 s CTOAs. Rather, at short CTOAs, responding is typically advantaged to locations indicated by the arrow and, over time, this facilitatory effect decays to nil, not reverses. Reasoning that inhibition may have been observed in Taylor and Klein (2000) because the arrow stimuli were associated with manual responses, we largely replicated Taylor and Klein (2000)'s design except removed the central arrow, manual response target (i.e., the arrow cue was no longer associated with a manual response). The experiment indicated that the direction of the arrow cue had little discernible impact on later manual localization responses to left- or right- peripheral targets whereas, as per usual, peripheral cueing generated inhibition. In the ensuing experiment, we largely replicated Taylor and Klein (2000)'s no response - manual cell except we removed the peripheral cues; simply, there were central cues and peripheral and central manual response targets. Therefore in this particular experiment, central arrow cues were associated with manual localization responses. In this case, there was clear evidence that the arrow generated inhibition but, intriguingly, it expressed itself nearer the output end of the processing continuum,

consistent with negative compatibility effects – and superficially IOR, in effect – but very much unlike the input-based arrow-elicited inhibition generated by Taylor and Klein (2000). Juxtaposition of Taylor and Klein (2000)'s findings with those in Chapter 5 makes clear that inhibition can arise fundamentally from manual response/spatial priming and that this inhibition, perhaps via re-mapping mechanisms, can go on to affect more input-based or sensorimotor processes.

Over time and contemplative of the ideas in Chapter 5, it began to occur to me that Taylor and Klein (2000)'s perceptual/attention – input-based – inhibitory aftereffects were all likely object-based and caused fundamentally by manual response/spatial priming. As reviewed by Chapter 7, there was already very strong empirical support from the extant literature that simple model tasks involving manual responses tended to generate object-based forms of inhibition. And, after all, unlike in conditions involving saccadic responding, the Taylor and Klein (2000) exclusively-manual response findings seemed to suggest that a target must appear at cued object in order to observe evidence of inhibition. With this in mind, it seemed important to evaluate whether the inhibition from a peripheral cue in a strictly covert simple model task would survive the absence of placeholder objects; it didn't. But there was inhibition when placeholder objects were present and the effect was input-based, as expected, given that cueing had no discernible impact on manual responses to central arrow targets. This finding strongly suggested that the indisputably input-based form of inhibition was object-based. In part for neatness but also because I was genuinely interested in whether a peripheral cue would prime a spatially-compatible manual response when manual responses were required to

an eventual arrow or peripheral onset target, I included precisely the same CTOAs in these experiments that I used in Chapter 2, Experiment 1. Similar to the effect of the cue on saccadic responding, the cue primed the directionally compatible manual response at early CTOAs. Given that manual response/spatial priming appears sufficient, on some level, for late inhibition, it appeared clear that some form of inhibition should have been generated by the cue. I had already postulated (resolved really) in Chapter 5 that the inhibition generated by manual response/spatial priming must be acted on by some remapping mechanism that comes into play principally when there is incentive to process items in peripheral vision at the time of the cue (as in when a cue appears in peripheral vision) so as to explain the anomaly between Taylor and Klein (2000) and Chapter 5. Thus, I considered it likely that there is an object-based form of inhibition, generated fundamentally by a primed manual/spatial response that is re-mapped via parietal networks into object-based coordinates when there is incentive to process peripheral items at the time of or before the inhibition is generated. For now however, this remains a working hypothesis that, for the most part, relates both input- and output-based inhibition very closely to the premotor theory of attention (Craighero & Rizzolatti, 1997; Smith & Schenk, 2012).

Early sensory inhibition(s)

Another form of inhibition arises specifically when input pathways are repeatedly stimulated at short CTOAs (< ~500 ms), as was apparent in Chapter 2, Experiment 1, in which the time course of cueing on saccadic responding was studied and also in Chapter 7, in which the time course of cueing on manual responding was

studied. As demonstrated by the central target arrow diagnostic, in tasks involving spatially-compatible saccadic or manual responses to stimuli in peripheral vision, a spatially uninformative peripheral cue initially – and transiently – biases the task-relevant response system toward the source of stimulation. However, at these same CTOAs, this initial bias toward the source of stimulation is either overshadowed or abolished by short-term sensory effects that occur when the cue and target occupy the same location. I note further that the cost of repeatedly stimulating an input pathway is greater for saccadic than manual responses. This short-term "inhibitory" sensory effect – and perhaps even the asymmetry between saccadic and manual responses on it – is likely the behavioral consequence of a short-term refractoriness that occurs when the receptive fields of visual and visuomotor cells residing in the superior colliculus are repeatedly stimulated (e.g., Cynader & Berman, 1972; Goldberg & Wurtz, 1972; Robinson & Kertzman, 1995; Fecteau & Munoz, 2005) and may relate closely to sensory adaptation (Boehnke et al., 2011), as has been established from extracellular recordings in both felines and primates. Because of the short-lasting, retinotopic and sensory nature of the effect, the phenomenon is certainly not what Posner et al (1985) intended by IOR. This effect, however, may relate more closely to the sensory inhibition discussed by Posner and Cohen (1984; 1980). In fact, Posner and Cohen (1984) noted extensive similarities between their inhibition and that studied by single-cell recordings:

> There is also evidence of an inhibitory effect on individual neural cells that occurs with the presentation of a second signal in the visual field occupied by a target... This effect resembles the cost in RT we find when attention is cued to a position other than the target. Our explanation for the RT effect is that orienting to the cue reduces the efficiency of taking in information from the target. It is possible that [sic] single-cell inhibition effect is related to the inhibitory effect discussed in this chapter. If animals

> covertly orient to a new signal, it is possible that cells responding to the target would now exhibit the inhibition that accompanies any change in light energy. Wurtz et al. (1980) argue that their cellular inhibition effect is sensory, not attentional, in origin in much the same way we have discussed here. Nevertheless, we believe our inhibition effect plays a role in determining the location of future covert and overt orienting. (pp. 552).

What is made clear by the foregoing paragraph is awareness by Posner and Cohen (1984) that their effect was likely sensory but also a desire for an inhibition that extends beyond low-level sensory processes into the domain of "attentional" orienting, more generally. To this end, it is noteworthy that whereas spatially-repetitive visual perturbations in the natural world are closer to the exception than the rule, short-term attenuation of the input-signal would, in effect, minimize the likelihood of an oculomotor orienting response (e.g., Satel, Wang, Trappenberg & Klein, 2011). This perspective is shared by monkey neurophysiologists; Boehnke, Berg, Marino, Baldi, Itti and Munoz (2011) note, for example, that "[sensory] adaptation in the SC serves to rapidly decrease the neural representation of repeating visual events at a particular spatial location (reducing the chance of reflexive orienting to that location), and to increase the representation of temporal outlier" (pp. 777). Nevertheless, sensory adaptation is not IOR because IOR, by definition, is not bound to exceptional instances of frequently-occurring, spatially-coincident visual stimuli.

Conclusions

IOR is a robust, experimentally-demonstrable, and relatively long-lasting and cross-modal response/decision bias that occurs in the aftermath of reflexive oculomotor orienting responses; it is encoded or remapped very rapidly into environmental

coordinates, a representation that theoretically optimizes efficient sampling of the visual environment. This effect is easily obtained in variations of the simple model task when the oculomotor response system for reflexively-generated saccades is not in a task-induced, tonically-suppressed state and can be measured by both saccadic and manual responses that are compatible with the direction(s) or prior, reflexive, oculomotor orienting responses. As such, repeat stimulation of an input pathway is *not* required, as demonstrated here and by Posner et al. (1985). Despite this, common experimental practice is to study "IOR" behaviourally in the context of traditional cue-target (sensory) overlap paradigm which, under many circumstances and at this time, can make it very challenging to distinguish among late input-based, late output-based (IOR), and early sensory inhibitory aftereffects[22]. An availability heuristic thus may, in part, spur on the common beliefs that inhibition – or IOR for that matter – is little more than a consequence of repeat sensory stimulation and/or a delay in returning attention. But in light of demonstrable differences between the forms of inhibition when the simple model task is adapted in (arguably) non-traditional ways, it appears clear that future research must be dedicated to pushing the boundaries of the simple model task – and by proxy orienting – so that the complex ways in which both sensory and motor processes conspire to drive human behaviour can be uncovered. In this regard, by no means was the set of experiments appearing herein an exhaustive set – I have not as Newell (1973) suggested, "accept[ed] a single complex task and do[ne] all of it" (pp. 303) – but I do

[22] This methodological hegemony is particularly surprising in the context of IOR given that its properties were ultimately established in variations of the cue-target paradigm not involving asymmetric visual stimulation (see also, e.g., Satel, Hilchey, Wang, Reiss & Klein, 2014, for a demonstration of an inhibition caused by eye movements without spatially uninformative peripheral cues).

hope that this dissertation serves as a blueprint for demonstrating and studying the ways in which unique processing demands can spur on completely dissociable forms of inhibition.

Finally, I return to what is to be done with the phrase IOR. Although I prefer to reserve the phrase IOR for the particular class of inhibition identified by Posner et al. (1985) – which implies a particular mechanical structure that is demonstrably distinct from that of other forms – in common among all forms of "inhibition" identified by this dissertation is their vulnerability in behavioral expression to variations of the simple model task. To this end however, I can think of little else more adaptive – or functional – than the possibility of distinct forms of inhibition that are configured or invoked specifically to meet the unique sensorimotor processing demands imposed by the task, on the individual, to selectively bias orienting. Functionally then, there may be a common thread among all forms of inhibition – arising from a rich constellation of dissociable mechanisms that operate to selectively bias orienting – each of which may be subservient to the attention required for the task.

REFERENCES

Abegg, M., Sharma, N., & Barton, J. J. S. (2012). Antisaccades generate two types of saccadic inhibition. *Biological Psychology, 89*, 191-194.

Abrams, R. A., & Dobkin, R. S. (1994a). Inhibition of return: Effects of attentional cuing on eye movement latencies. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 467–477.

Abrams, R. A., & Dobkin, R. S. (1994b). The gap effect and inhibition of return: Interactive effects on eye movement latencies. *Experimental Brain Research*, *98*(3), 483-487.

Abrams, R. A., & Pratt, J. (2000). Oculocentric coding of inhibited eye movements to recently attended locations. *Journal of Experimental Psychology: Human Perception and Performance*, *26*(2), 776.

* Akiyama, T., Motoichiro, K., Muramatsu, T., Saito, F., Umeda, S., Kashima, H. (2006). Gaze but not arrows: A dissociative impairment after right superior temporal gyrus damage. *Neuropsychologia*, *44*, 1804- 1810.

Andersen, R. A., Asanuma, C., Essick, G., & Siegel, R. M. (1990). Corticocortical connections of anatomically and physiologically defined subdivisions within the inferior parietal lobule. *The Journal of Comparative Neurology*, *296*, 65-113.

Barton, J. S., Goff, D. C., & Manoach D. S. (2006). The inter-trial effects of stimulus and saccadic direction on prosaccades and antisaccades, in controls and schizophrenia patients *Experimental Brain Research, 174,* 487–498.

Berger, A., Henik, A., & Rafal, R. (2005). Competition between endogenous and exogenous orienting of visual attention. *Journal of Experimental Psychology: General*, *134*(2), 207-221.

Berlucchi, G. (2006). Inhibition of return: A phenomenon in search of a mechanism and a better name. *Cognitive Neuropsychology*, *23*, 1065-1074.

Berlucchi, G., Chelazzi, L., & Tassinari, G. (2000). Volitional covert orienting to a peripheral cue does not suppress cue-induced inhibition of return. *Journal of Cognitive Neuroscience*, *12*(4), 648-663.

Berlucchi, G., Di Stefani, M., Marzi, C. A., Morelli, M., & Tassinari, G. (1981). Direction of attention in the visual field as measured by a reaction time paradigm. *Behavioral Brain Research*, *2*(2), 244-245.

Berlucchi, G., Tassinari, G., Marzi, C. A., & Di Stefano, M. (1989). Spatial distribution of the inhibitory effect of peripheral non-informative cues on simple reaction time to non-fixated visual targets. *Neuropsychologia*, *27*(2), 201-221.

* Birmingham, E., & Pratt, J. (2005). Examining inhibition of return with onset and offset cues in the multiple-cuing paradigm. *Acta Psychologica*, *118*(1), 101-121.

Bisley, J. W., & Goldberg, M. E. (2009). Attention, intention, and priority in the parietal lobe. *Annual Review of Neuroscience*, *33*, 1-21.

Boehnke, S. E., Berg, D., Marino, R. A., Baldi, P. F., Itti, L., & Munoz, D. P. (2011). Visual adaptation and novelty responses in the superior colliculus. *European Journal of Neuroscience*, *34*, 766-779.

* Bonato, M., Konstantinos, P., Marenzi, R., & Zorzi, M. (2008). Normal and impaired reflexive orienting of attention after central nonpredictive cues. *Journal of Cognitive Neuroscience*, *21*(4), 745–759.

Boot, W. R., McCarley, J. S., Kramer, A. F., & Peterson, M. S. (2004). Automatic and intentional memory processes in visual search. *Psychonomic Bulletin & Review*, *11*(5), 854-861.

Bourgeois, A., Chica, A. Migliaccio, R., Thiebaut de Schotten, M., & Bartolomeo, P. (2012). Cortical control of inhibition of return: Evidence from patients with inferior parietal damage and visual neglect. *Neuropsychologia*, *50*, 800-809.

Bourgeois, A., Chica, A. B., Valero-Cabré, A., & Bartolomeo, P. (2013). Cortical control of inhibition of return: causal evidence for task-dependent modulations by dorsal and ventral parietal regions. *Cortex*, *49*(8), 2229-2238.

Boy, F. & Sumner, P. (2010). Tight coupling between positive and reversed priming in the masked prime paradigm. *Journal of Experimental Psychology-Human Perception and Performance, 36*(4), 892-905.

Briand, K. A., Larrison, A. L., & Sereno, A. B. (2000). Inhibition of return in manual and saccadic response systems. *Perception & Psychophysics*, *62*, 1512-1524.

Burle, B., van den Wildenberg, W. P. M., & Ridderinkhof, K. R. (2005). Dynamics of facilitation and interference in cue-priming and Simon tasks. *European Journal of Cognitive Psychology, 17,* 619–641.

Burnham, K.P, & Anderson, D.R. (2004). Multimodel inference: Understanding AIC and BIC in Model Selection. *Sociological Methods and Research, 33*, 261-304.

Burr, D. C., & Morrone, M. C. (2011). Spatiotopic coding and remapping in humans. *Philosophial Transactions of the Royal Society B- Biological Sciences*, *366*(1564), 504-515.

Cheal, M., Chastain, G., & Lyon, D. R. (1998). Inhibition of return in visual identification tasks. *Visual Cognition*, *5*, 365-388.

Chica, A. B., Klein, R. M., Rafal, R. D., & Hopfinger, J. B. (2010). Endogenous saccade preparation does not produce inhibition of return: Failure to replicate Rafal, Calabresi, Brennan & Sciolto (1989). *Journal of Experimental Psychology: Human Perception and Performance*, *36,* 1193–1206.

Chica, A. B., Martín-Arévalo, E., Botta, F., & Lupiánez, J. (2014). The Spatial Orienting paradigm: how to design and interpret spatial attention experiments. *Neuroscience & Biobehavioral Reviews*, *40*, 35-51.

Chica, A. B., Taylor, T. L., Lupianez, Y., & Klein, R. M. (2010). Two mechanisms underlying inhibition of return. *Experimental Brain Research*, *201*, 25-35.

Christ, S. E., Mccrae, C. S., & Abrams, R. A. (2002). Inhibition of return in static and dynamic displays. *Psychonomic Bulletin & Review*, *9*(1), 80-85.

Christie, J., Hilchey, M. D., & Klein, R. M. (2013). Inhibition of return is at the midpoint of simultaneous cues. *Attention, Perception, & Psychophysics*, *75*(8), 1610-1618.

Christie, J., Hilchey, M. D., Mishra, R., & Klein, R. M. (2015). Eye movements are primed toward the center of multiple stimuli even when the interstimulus distances are too large to generate saccade averaging. *Experimental brain research*, *233*(5), 1541-1549.

Cohen, Y. (1981). *Internal and external control of orienting*. Unpublished doctoral dissertation. *University of Oregon.*

Cole G. & Kuhn G. (2010). What the experimenter's prime tells the observer's brain. *Attention Perception & Psychophysics, 72*(5), 1367-1376.

Coward, R. S., Poliakoff, E., O'Boyle, D. J., & Lowe, C. (2004). The contribution of non-ocular response inhibition to visual inhibition of return. *Experimental brain research*, *155*(1), 124-128.

Sheliga, B. M., Craighero, L., Riggio, L., & Rizzolatti, G. (1997). Effects of spatial attention on directional manual and ocular responses. *Experimental brain research*, *114*(2), 339-351.

Cynader, M., & Berman, N. (1972). Receptive-field organization of monkey superior colliculus. *Journal of Neurophysiology, 35*, 187-201.

Danziger, S. & Kingstone, S. (1999). Unmasking the inhibition of return phenomenon. *Perception & Psychophysics*, *61*, 1024-1037.

de Jong, R., Liang, C. C. & Lauber, E. (1994). Conditional and unconditional automaticity - A dual-process model of effects of spatial stimulus - response correspondence. *Journal of Experimental Psychology-Human Perception and Performance, 20*(4), 731-750.

Dodd, M. D., & Pratt, J. (2007). The effect of previous trial type on inhibition of return. *Psychological Research*, *71*(4), 411-417.

Dodd, M. D., van der Stigchel, S., & Hollingworth, A. (2009). Novelty is not always the best policy: Inhibition of return and facilitation of return as a function of visual task. *Psychological Science, 20*(3), 333-339.

Dorris, M. C., Klein, R. M., Everling, S., & Munoz, D. P. (2002). Contribution of the primate superior colliculus to inhibition of return. *Journal of Cognitive Neuroscience*, *14*(8), 1256-1263.

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

Dukewich, K. (2009). Reconceptualizing inhibition of return as habituation of the orienting response. *Psychonomic Bulletin & Review, 16*, 238-251.

Dukewich, K. R. & Boehnke, S. (2008). Cue repetition increases inhibition of return. *Neuroscience Letters*, *448*(3), 231-235.

Dukewich, K. R., & Klein, R. M. (2015). Inhibition of return: a phenomenon in search of a definition and a theoretical framework. *Attention, Perception, & Psychophysics*, 1-12.

Eimer, M. (1999). Facilitatory and inhibitory effects of masked prime stimuli on motor activation and behavioral performance. *Acta Psychologica*, *101,* 293–313.

Engell, A. D., Nummenmaa, L., Oosterhof, N.N., Henson, R. N., Haxby, J. V., & Calder, A. J. (2010). Differential activation of frontoparietal attention networks by social and symbolic spatial cues. *Oxford University Press*. http://scan.oxfordjournals.org, July 17, 2012.

Everling S., Dorris, M. C., Klein, R. M., & Munoz, D. P. (1999). Role of primate superior colliculus in preparation and execution of anti-saccade and pro-saccades. *Journal of Neuroscience*, *19*, 2740-2754.

Fecteau, J. H., Au, C., Armstrong, I. T., & Munoz, D. P. (2004). Sensory biases produce alternation advantage found in sequential saccadic eye movement tasks. *Experimental Brain Research*, *159*, 84-91.

Fecteau, J. H., Bell, A. H., & Munoz, D. P. (2004). Neural correlates of the automatic and goal-driven biases in orienting spatial attention. *Journal of Neurophysiology*, *92*(3), 1728-1737.

Fecteau, J. H. & Munoz, D. P. (2005). Correlates of capture of attention and inhibition of return across stages of visual processing. *Journal of Cognitive Neuroscience*, *17*(11), 1714-1727.

Fecteau, J. H. & Munoz, D. (2006). Salience, relevance, and firing: a priority map for target selection. *Trends in Cognitive Sciences*, *10*, 382-390.

Forbes, K., & Klein, R. (1996). The magnitude of the fixation offset effect with endogenously and exogenously controlled saccades. *Journal of Cognitive Neuroscience*, *8*, 344-352.

* Friedrich, F. J, Rader, S. D. & Knauts, S.P., (1997). Expectancy effects on the orienting of spatial attention. Unpublished manuscript.

Fuentes, L., Vivas, A., & Humphreys, G. (1999). Inhibitory mechanisms of attentional networks: Spatial and semantic inhibitory processing. *Journal of Experimental Psychology. Human Perception and Performance, 25*(4), 1114-1126.

Gabay, S., Chica, A. B., Charras, P., Funes, M. J., & Henik, A. (2012). Cue and target processing modulate the onset of inhibition of return. *Journal of Experimental Psychology. Human Perception and Performance*, *38*(1), 42-52.

Gabay, S., Leibovich, T., Ben-Simon, A., Henik, A., & Segev, R. (2013). Inhibition of return in the archer fish. *Nature Communications*, *4*, 1657.

Gabay, S., & Henik, A. (2010). Temporal expectancy modulates inhibition of return in a discrimination task. *Psychonomic Bulletin & Review, 17*, 47-51.

Gabay, S., Henik, A., & Gradstein, L. (2010). Ocular motor ability and covert attention in patients with Duane Retraction Syndrome. *Neuropsychologia, 48*, 3102-3109.

Godijn, R. & Theeuwes, J. (2002). Oculomotor capture and inhibition of return: evidence for an oculomotor suppression account of IOR. *Psychological Research*, *66*(4), 234-246.

Goldberg, M. E., & Wurtz, R. H. (1972). Activity of superior colliculus in behaving monkey. I. Visual receptive fields of single neurons. *Journal of Neurophysiology*, *35*(4), 542-559.

Golomb, J. D., Chun, M. M., & Mazer, J. A. (2008). The native coordinate system of spatial attention is retinotopic. *Journal of Neuroscience*, *28*(42), 10654-10662.

Gottlieb, J. & Balan, P. (2010). Attention as a decision in information space. *Trends in Cognitive Sciences*, *14*, 240-248.

Gottlieb, J., Balan, P., Oristaglio, J., & Suzuki, M. (2009). Parietal control of attentional guidance: The significance of sensory, motivational and motor factors. *Neurobiology of Learning and Memory*, *91*, 121-128.

* Greene, D. J., Mooshagian, E., Kaplan, J. T., Zaidel, E. & Iacoboni, M. (2009). The neural correlates of social attention: automatic orienting to social and nonsocial cues. *Psychological Research*, *73*, 499–511.

Grison, S., Kessler, K., Paul, M. A., Jordan, H., & Tipper, S. P. (2004). Object-and location-based inhibition in goal-directed action: Inhibition of return reveals behavioural and anatomical dissociations and interactions with memory processes. In G.W. Humphreys and M. J. Riddoch (Eds.), *Attention in action: Advances from cognitive neuroscience* (pp. 171-208). Hove, UK: Psychology Press.

Hale, D. J. (1967). Sequential effects in a two-choice serial reaction task. *The Quarterly Journal of Experimental Psychology*, *19*(2), 133-141.

Hall, N. J., & Colby, C. L. (2011). Remapping for visual stability. *Philosophical Transactions of the Royal Society B- Biological Sciences*, *366*(1564), 528-539.

Handy, T. C., Jha, A. P., & Mangun, G. R. (1999). Promoting novelty in vision: Inhibition of return modulates perceptual-level processing. *Psychological Science*, *10*(2), 157-161.

Harvey, N. (1980). Non-informative effects of stimuli functioning as cues. *Quarterly Journal of Experimental Psychology, 32,* 413–425.

Hietanen, J. K., Nummenmaa, L., Nyman, M. J., Parkkola, R. & Hämäläinen, H.. (2006). Automatic attention orienting by social and symbolic cues activates different neural networks: An fMRI study. *NeuroImage, 33*, 406–413.

Hilchey, M. D., Hashish, M., MacLean, G. H., Satel, J., Ivanoff, J., & Klein, R. M. (2014). On the role of eye movement monitoring and discouragement on inhibition of return in a go/no-go task. *Vision Research*, *96*, 133-139.

Hilchey, M. D., Ivanoff, J., Taylor, T. L., & Klein, R. M. (2011). The effect of inhibition of return on the temporal dynamics of spatial information processing, *Acta Psychologica*, *136*, 235-244.

Hilchey, M. D., Klein, R. M., & Ivanoff, J. (2012). Perceptual and motor IOR: Components or flavors? *Attention, Perception & Psychophysics*, *74*, 1416-1429.

Hilchey, M. D., Klein, R. M., & Satel, J. (2014). Returning to" inhibition of return" by dissociating long-term oculomotor IOR from short-term sensory adaptation and other nonoculomotor" inhibitory" cueing effects. *Journal of Experimental Psychology. Human Perception and Performance, 40*(4), 1603-1616.

Hilchey, M. D., Klein, R. M., Satel, J., & Wang, Z. (2012). Oculomotor inhibition of return: How soon is it “recoded” into spatiotopic coordinates? *Attention, Perception, & Psychophysics*, *74*(6), 1145-1153.

Hilchey, M. D., Satel, J., Ivanoff, J., & Klein, R. M. (2013). On the nature of the delayed ‘inhibitory’ cueing effects generated by uninformative arrows at fixation. *Psychonomic Bulletin & Review*, *20*: 593-600.

Hoffman, J. E., & Subramaniam, B. (1995). The role of visual attention in saccadic eye movements. *Perception & Psychophysics*, *57*(6), 787-795.

Hooge, I. T. & Frens, M. A. (2000). Inhibition of saccade return (ISR): Spatial-temporal properties of saccade programming. *Vision Research*, *40*, 3415-3426.

Hooge, I. T. C., Over, E. A., van Wezel, R. J., & Frens, M. A. (2005). Inhibition of return is not a foraging facilitator in saccadic search and free viewing. *Vision Research*, *45*(14), 1901-1908.

Hu, F. K., Samuel, A. G., & Chan, A. S. (2011). Eliminating inhibition of return by changing salient nonspatial attributes in a complex environment. *Journal of Experimental Psychology. General*, *140*(1), 35-50.

Hu, F. K., Fan, Z., Samuel, A. G., & He, S. (2013). Effects of display complexity on location and feature inhibition. *Attention, Perception, & Psychophysics, 75*(8), 1619-1632.

Hunt, A. R. & Kingstone, A. (2003). Inhibition of return: Dissociating attentional and oculomotor components. *Journal of Experimental Psychology: Human Perception and Performance*, *29*(5), 1068-1074.

Ignaschenkova, A., Dicke, P. W., Haarmeier, T., & Thier, P. (2004). Neuron-specific contribution of the superior colliculus to overt and covert shifts of attention. *Nature Neurosciecne*, *7*, 56-64.

Itti, L., & Koch, C. (2001). Computational modelling of visual attention. *Nature Reviews Neuroscience*, *2*(3), 194-203.

Ivanoff, J., & Klein, R. M. (2001). The presence of a non-responding effector increases inhibition of return. *Psychonomic Bulletin & Review*, *8*, 307-314.

Ivanoff, J., & Klein, R. M. (2003). Orienting of attention without awareness is affected by measurement-induced attentional control settings. *Journal of Vision*, *3*(*1*), 32–40.

Ivanoff, J. & Klein, R. M. (2004) Stimulus-response probability and inhibition of return. *Psychonomic Bulletin & Review*, *11*(*3*), 542-550.

Ivanoff, J., & Klein, R. M. (2006). Inhibition of return: sensitivity and criterion as a function of response time. *Journal of Experimental Psychology*: *Human Perception and Performance*, *34*, 908-919.

Ivanoff, J., Klein, R. M., & Lupianez, J. (2002). Inhibition of return interacts with the Simon effect: an omnibus analysis and its implications. *Perception & Psychophysics*, *64*, 318-327.

* Ivanoff, J. & Saoud, W. (2009). Nonattentional effects of nonpredictive central cues. *Attention Perception & Psychophysics, 71*, 4, 872-880.

* Jordan, H., & Tipper, S. P. (1998). Object-based inhibition of return in static displays. *Psychonomic Bulletin & Review*, *5*(3), 504-509.

Jordan, H., & Tipper, S. P. (1999). Spread of inhibition across an object's surface. *British Journal of Psychology*, *90*(4), 495-507.

Jaskowski, P. (2007). The effect of nonmasking distractors on the priming of motor responses. *Journal of Experimental Psychology-Human Perception and Performance, 33*(2), 456-468.

Jaskowski, P. & Slosarek, M. (2007). How important is a prime's gestalt for subliminal priming?. *Consciousness and Cognition, 16*(2), 485-497.

Kessler, K., & Tipper, S. (2004). Retrieval of implicit inhibitory processes: The impact of visual field, object-identity, and memory dynamics. *Visual Cognition, 11*(8), 965-995.

Khatoon, S., Braind, K. A., & Sereno, A. B. (2002). The role of response in spatial attention: Direct versus indirect stimulus-response mappings. *Vision Research*, *42*, 2693-2708.

Kingstone, A. & Pratt, J. (1999). Inhibition of return is composed of attentional and oculomotor processes. *Perception & Psychophysics*, *61*, 1046-1054.

Kirby, N. H. (1972). Sequential effects of serial reaction time. *Journal of Experimental Psychology*, *96*(1), 32.

Kirby, N. H. (1976). Sequential effects in two-choice reaction time: Automatic facilitation or subjective expectancy?. *Journal of Experimental Psychology: Human Perception and Performance*, *2*(4), 567-77.

Klein, R. M. (1980). Does oculomotor readiness mediate cognitive control of visual attention? In R. Nickerson (Ed.), *Attention and Performance VIII* (pp. 259–276). Hillsdale, NJ: Erlbaum.

Klein, R. (1988). Inhibitory tagging system facilitates visual search. *Nature*, *334*(6181), 430-431.

Klein, R. M. (2000). Inhibition of return. *Trends in Cognitive Sciences*, *4*(4), 138-147.

Klein, R. M. (2004). Orienting and inhibition of return. In M. S. Gazzaniga (Ed.), *The Cognitive Neurosciences 3rd Edition* (pp. 545-559). Cambridge, MA: MIT Press.

Klein, R. M. (2005). On the role of endogenous orienting in the inhibitory aftermath of

exogenous orienting. In U. Mayr, E. Awh, & S. Keele (Eds.), *Developing individuality in the human brain: A tribute to Michael I. Posner* (pp. 45-64). Washington, DC:American Psychological Association.

Klein, R. M., Christie, J., & Morris, E. P. (2005). Vector averaging of inhibition of return. *Psychonomic Bulletin & Review*, *12*(2), 295-300.

Klein, R., & Dukewich, K. (2006). Does the inspector have a memory? *Visual Cognition*, *14*(4-8), 648-667.

Klein, R., & Farrell, M. (1989). Search performance without eye movements. *Perception & Psychophysics*, *46*(5), 476-482.

Klein, R. M. & Hilchey, M. D. (2011). Oculomotor inhibition of return. In S. Liversedge, I. D. Gilchrist & S. Everling (Eds). *The Oxford Handbook of Eye Movements*. Oxford: Oxford University Press (pp. 471-492).

Klein, R. M., Hilchey, M. D. & Satel, J. (2012, Nov) On the flavors of inhibition of return: Will the real IOR please stand up?, Paper presented at the 53nd Annual meeting of the Psychonomic Society (Minneapolis, USA)

Klein, R. M., & Macinnes, J. W. (1999). Inhibition of return is a foraging facilitator in visual search. *Psychological Science*, *10*(4), 346-352.

Klein, R. M. & Pontefract, A. (1994). Does oculomotor capture mediate cognitive control of visual attention? Revisited! In R. Nickerson (Ed.), *Attention and performance XV: Conscious and nonconscious information processing* (pp. 333–350). Hillsdale, NJ: Erlbaum.

Klein, R. M., Schmidt, W. C., & Müller, H. K. (1998). Disinhibition of return: Unnecessary and unlikely, *Perception & Psychophysics*, *60*, 862-872.

Klein, R. M. & Taylor, T. L. (1994). Categories of cognitive inhibition, with reference to attention. In D. Dagenbach & T. Carr (Eds.). *Inhibitory processes in attention, memory, and language*. Academic Press (pp. 113-150).

Kornblum, S. & Lee, J. W. (1995). Stimulus-response compatibility with relevant and irrelevant stimulus dimensions that do and do not overlap with the response. *Journal of Experimental Psychology-Human Perception and Performance, 21*(4), 855-875.

Krüger, H. M., & Hunt, A. R. (2013). Inhibition of return across eye and object movements: the role of prediction. *Journal of Experimental Psychology. Human Perception and Performance*, *39*(3), 735-744.

* Langdon, R. & Smith, P. (2005). Spatial cueing by social versus nonsocial directional signals. *Visual Cognition*, *21*(8), 1497-1527.

* Langley, L.K., Friesen, C.K., Saville, A. L., & Ciernia, A. T. (2011). Timing of reflexive visuospatial orienting in young, young-old, and old-old adults. *Attention, Perception, & Psychophysics*, *73*, 1546–1561.

Langley, L. K., Gayzur, N. D., Saville, A. L., Morlock, S. L., & Bagne, A. G. (2011). Spatial distribution of attentional inhibition is not altered in healthy aging. *Attention, Perception & Psychophysics,* 73, 766-783.

Lawrence, M.A. (2012). ez: Easy analysis and visualization of factorial experiments (R package Ver. 4.1-1). *Halifax, Canada*.

* Leek, E. C., Reppa, I., & Tipper, S. P. (2003). Inhibition of return for objects and locations in static displays. *Perception & Psychophysics*, *65*(3), 388-395.

Li, C. S. R., & Lin, S. C. (2002a). Inhibition of return in temporal order saccades. *Vision Research*, *42*(17), 2089-2093.

Li, C. S. R., & Lin, S. C. (2002b). A perceptual level mechanism of the inhibition of return in oculomotor planning. *Cognitive Brain Research, 14(2),* 269–276.

Lupianez, J. (2010). Inhibition of return. In H. Nobre & J. T. Coull (Eds.), *Attention and time* (pp. 17-34). Oxford, UK: Oxford University Press.

Lupianez, J., Klein, R. M., & Bartolomeo, P. (2006). Inhibition of return: Twenty years after. *Cognitive Neuropsychology*, *23*, 1003-1014.

Lupiáñez, J., Milliken, B., Solano, C., Weaver, B., & Tipper, S. P. (2001). On the strategic modulation of the time course of facilitation and inhibition of return. *The Quarterly Journal of Experimental Psychology: Section A*, *54*(3), 753-773.

Lupianez, J., Ruz, M., Funes, M. J. & Milliken, B. (2007). The manifestation of attentional capture: facilitation or IOR depending on task demands, *Psychological Research*, 71, 77-91.

Lupiáñez, J., & Weaver, B. (1998). On the time course of exogenous cueing effects: a commentary on Tassinari et al.(1994). *Vision Research*, *38*(11), 1621-1628.

Machado, L., & Rafal, R. D. (2004). Inhibition of return generated by voluntary saccades is independent of attentional momentum. *The Quarterly Journal of Experimental Psychology Section A: Human Experimental Psychology, 57,* 789–796.

Machado, L., Wyatt, N., Devine, A. & Knight, B. Action planning in the presence of distracting stimuli: An investigation into the time course of distractor effects. *Journal of Experimental Psychology: Human Perception and Performance*, *33*, 1045-1061.

MacInnes, J. W., & Klein, R. M. (2003). Inhibition of return biases orienting during the search of complex scenes. *The Scientific World Journal*, *3*, 75-86.

MacInnes, W. J., Hunt, A. R., Hilchey, M. D., & Klein, R. M. (2014). Driving forces in free visual search: An ethology. *Attention, Perception, & Psychophysics*, *76*(2), 280-295.

MacInnes, J., Kruger, H. M., & Hunt, A. R. (2015). Just passing through? Inhibition of return in saccadic sequences. *The Quarterly Journal of Experimental Psychology*, *68*, 402-416.

MacLean, G. H., Klein, R. M., & Hilchey, M. D. (2015). Does oculomotor readiness mediate exogenous capture of visual attention? *Journal of Experimental Psychology: Human Perception and Performance*. Retrieved at http://dx.doi.org/10.1037/xhp0000064

MacPherson, A., Klein, R. M. & Moore, C. (2003) Inhibition of return in children and adolescents. *Journal of Experimental Child Psychology*, *85*, 337-351.

* Marotta, A., Lupianez, J., Martella, D., & Casagrande, M. (2012). Eye gaze versus arrows as spatial cues: Two qualitatively different modes of attentional selection. *Journal of Experimental Psychology-Human Perception and Performance*, *38*(2), 326–335.

Mathôt, S., & Theeuwes, J. (2010). Gradual remapping results in early retinotopic and late spatiotopic inhibition of return. *Psychological Science*, *21*(12), 1793-1798.

Mathôt, S., & Theeuwes, J. (2011). Visual attention and stability. *Philosophical Transactions of the Royal Society B-Biological Sciences*, *366*(1564), 516-527.

Maylor, E. A. (1985). Facilitatory and inhibitory components of orienting in visual space. In M. I. Posner & O. S. M. Marin (Eds.), *Attention and performance XI* (pp. 189–204). Hillsdale, NJ: Erlbaum.

Maylor, E. A., & Hockey, R. (1985). Inhibitory component of externally controlled covert orienting in visual space. *Journal of Experimental Psychology: Human Perception and Performance*, *11*, 777-787.

Maylor, E. A., & Hockey, R. (1987). Effects of repetition on the facilitatory and inhibitory components of orienting in visual space. *Neuropsychologia*, *25*(1), 41-54.

* McAuliffe, J., Chasteen, A. L., & Pratt, J. (2006). Object-and location-based inhibition of return in younger and older adults. *Psychology and Aging*, *21*(2), 406-410.

* Mcauliffe, J., Pratt, J., & O'Donnell, C. (2001). Examining location-based and object-based components of inhibition of return in static displays. *Perception & Psychophysics*, *63*(6), 1072-1082.

McCrae, C. S., & Abrams, R. A. (2001). Age-related differences in object-and location-based inhibition of return of attention. *Psychology and Aging*, *16*(3), 437.

Mele, S., Berlucchi, G., & Peru, A. (2012). Inhibition of return at foveal and extrafoveal locations: Re-assessing the evidence. *Acta psychologica*, *141*(3), 281-286.

Mirpour, K., Arcizet, F., Ong, W. S., & Bisley, J. W. (2009). Been there, seen that: A neural mechanism for performing efficient visual search. *Journal of Neuorphysiology*, *102*, 3481-3491.

Müller, N. G., & Kleinschmidt, A. (2007). Temporal dynamics of the attentional spotlight: neural correlates of attentional capture and inhibition of return in early visual cortex. *Journal of Cognitive Neuroscience*, *19*, 587-593.

Müller, H. J., & Mühlenen, A. V. (2000). Probing distractor inhibition in visual search: inhibition of return. *Journal of Experimental Psychology: Human Perception and Performance*, *26*(5), 1591-1605.

Newell, A. (1973). You can't play 20 questions with nature and win: Projective comments on the papers of this symposium.

Pastötter, B., Hanslmayr, S., & Bäuml, K. H. (2008). Inhibition of return arises from inhibition of response processes: an analysis of oscillatory beta activity. *Journal of Cognitive Neuroscience*, *20*, 65-75.

* Paul, M. A., & Tipper, S. P. (2003). Object-based representations facilitate memory for inhibitory processes. *Experimental Brain Research*, *148*(3), 283-289.

Pertzov, Y., Zohary, E., & Avidan, G. (2010). Rapid formation of spatiotopic representations as revealed by inhibition of return. *Journal of Neuroscience*, *30*(26), 8882-8887.

Pertzov, Y., Avidan, G., & Zohary, E. (2011). Multiple reference frames for saccadic planning in the human parietal cortex. *Journal of Neuroscience*, *31*(3), 1059-1068.

Peterson, M., Beck, M., & Vomela, V. (2007). Visual search is guided by prospective and retrospective memory. *Attention, Perception, & Psychophysics*, *69*(1), 123-135.

* Peterson, S.A. & Gibson, T. N. (2011). Implicit attentional orienting in a target detection task with central cues. *Consciousness and Cognition*, *20*, 1532–1547.

Petersen, S. E., & Posner, M. I. (2012). The Attention System of the Human Brain: 20 Years After. *Annual Review of Neuroscience*, *35*(1), 73-89.

Posner, M. I. (1975). Psychobiology of attention. In M. Gazzaniga & C. Blakemore (Eds.), *Handbook of Psychobiology* (p. 441-480). Academic Press.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology*, *32*, 3-25.

Posner, M. I. (1982). Cumulative Development of Attentional Theory. *American Psychologist, 37*(2), 168-179.

Posner, M. I., & Cohen, Y. A. (1980, November). *Consequences of visual orienting*. Paper presented at the annual meeting of the Psychonomic Society, St. Louis.

Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. In H. Bouma & D. G. Bouwhuis (Eds.), *Attention and performance X: Control of language processes* (pp. 531-556). Hove, UK: Lawrence Erlbaum Associates Ltd.

Posner, M. I., Cohen, Y., Choate, L., Hockey, R., & Maylor, E. A. (1984). Sustained concentration: Passive filtering or active orienting? In S. Kornblum & J. Requin (Eds.) *Preparatory states and processes* (pp. 49-65). Hillside, NJ: Erlbaum.

Posner, M. I., Cohen, Y., & Rafal, R. D. (1982). Neural systems control of spatial orienting. *Philosophical Transaction of the Royal Society of London*, *298*, 187-198.

Posner, M. I., Inhoff, A. W., Friedrich, F. J., & Cohen, A. (1987). Isolating attentional systems: A cognitive-anatomical analysis. *Psychobiology*, *15*(2), 107-121.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*(1), 25-42.

Posner, M. I., Rafal, R. D., Choate, L., & Vaughan, J. (1985). Inhibition of return: neural basis and function. *Cognitive Neuropsychologia*, *2*(3), 211-228.

Posner, M. I., Snyder, C. R., & Davidson, B. J. (1980). Attention and the Detection of Signals. *Journal of Experimental Psychology: General*, *109*(2), 160-174.

Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *The Journal of Neuroscience*, *4*(7), 1863-1874.

Possamaï, C. A. (1986). Relationship between inhibition and facilitation following a visual cue. *Acta Psychologica*, *61*(3), 243-258.

Possamaï, C. A. (1992). The effect of a non-informative cueing signal in a three-choice reaction-time task. *Psychological Research*, *54*(2), 72-79.

* Possin, K. L., Filoteo, J. V., Song, D. D., & Salmon, D. P. (2009). Space-based but not object-based inhibition of return is impaired in Parkinson's disease. *Neuropsychologia*, *47*(7), 1694-1700.

* Pratt, J., & Chasteen, A. L. (2007). Examining inhibition of return with multiple sequential cues in younger and older adults. *Psychology and Aging*, *22*(2), 404-409.

Pratt, J. & Fischer, M. H. (2002). Examining the role of the fixation cue in inhibition of return. *Canadian Journal of Experimental Psychology*, *56*, 294-301.

Pratt, J. & Neggers, B. (2008). Inhibition of return in single and dual tasks: Examining saccadic, keypress, and pointing responses. *Perception & Psychophysics*, *70(2)*, 257-265.

Pratt, J., Spalek, T. M., & Bradshaw, F. (1999). The time to detect targets at inhibited and noninhibited locations: Preliminary evidence for attentional momentum. *Journal of Experimental Psychology: Human Perception and Performance*, *25*(3), 730.

Prime, D. J., & Jolicoeur, P. J. (2009). Response-selection conflict contribute to inhibition of return. *Journal of Cognitive Neuroscience*, *21*, 991-999.

Prime, D. J., & Ward, L. M. (2006). Cortical expressions of inhibition of return. *Brain research*, *1072*(1), 161–74.

Prinz, W. (1997). Perception and action planning. *European Journal of Cognitive Psychology*, *9*(2), 129-154.

Prinzmetal, W., Taylor, J.A., Myers, L.B., & Nguyen-Espino, J. (2011). Contingent capture and inhibition of return: a comparison of mechanisms. *Experimental Brain Research, 214*(1)*,* 47-60.

Qian, Q., Shinomori, K., & Song, M. (2012). Sequence effects by non-predictive arrow cues. *Psychological Research*, *76*, 253-262.

R development Core Team, R. (2011). R: a language and environment for statistical computing. *R Foundation for Statistical Computing.*

Rafal, R. D., Calabresi, P. A., Brennan, C. W. & Sciolto, T. K. (1989). Saccade preparation inhibits reorienting to recently attended locations. *Journal of Experimental Psychology: Human Perception and Performance, 15*(4), 673-685.

Rafal, R., Davies, J., & Lauder, J. (2006). Inhibitory tagging at subsequently fixated locations: Generation of "inhibition of return" without saccade inhibition. *Visual Cognition*, *13*(3), 308-323.

Rafal, R. D., Egly, R., & Rhodes, D. (1994). Effects of inhibition of return on voluntary and visually guided saccades. *Canadian Journal of Experimental Psychology*, *48*, 284-300.

* Ranzini, M., Dehaene, S., Piazza, M. & Hubbard, E. M. (2009). Neural mechanisms of attentional shifts due to irrelevant spatial and numerical cues. *Neuropsychologia*, *47*, 2615– 2624.

Redden, R. S., Hilchey, M. D., & Klein, R. M. (2014a). When caused by an eye movement, inhibition of return's effect is post-perceptual: Evidence from SAT functions. Poster presented at the 13th annual meeting of the Vision Sciences Society. St. Pete's Beach, Florida.

Redden, R.S., Hilchey, M.D., and Klein, R.M. (2014b). No Evidence for Object-Based Oculomotor Inhibition of Return: Abrams and Dobkins (1994) Revisited. Poster presented at the 55th Annual Meeting of the Psychonomic Society, Long Beach, CA.

Reppa, I., Schmidt, W. C., & Leek, E. C. (2012). Successes and failures in producing attentional object-based cueing effects. *Attention, Perception, & Psychophysics*, *74*(1), 43-69.

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

Ridderinkhof, K. R. (2002). Micro- and macro-adjustments of task set: Activation and suppression in conflict tasks. *Psychological Research*, *66*(4), 312-323.

Riggio, L., Patteri, I., & Umiltà, C. (2004). Location and shape in inhibition of return. *Psychological Research*, *68*(1), 41-54.

* Ristic, J. & Kingstone, A. (2006). Attention to arrows: Pointing to a new direction. *Quarterly Journal of Experimental Psychology, 59*(11), 1921-1930.

Ristic, J. & Kingstone, A. (2012). A new form of human spatial attention: Automated symbolic orienting. *Visual Cognition, 20*(3), 244-264.

* Ristic, J., Freisen, C. K. & Kingstone, A. (2002). Are eyes special? It depends on how you look at it. *Psychonomic Bulletin & Review,* 9, 3, 507-513.

Samuel, A. G. & Kat, D. (2003). Inhibition of return: A graphical meta-analysis of its timecourse, and an empirical test of its temporal and spatial properties. *Psychonomic Bulletin & Review*, *10*, 897-906.

Sapir, A., Hayes, A., Henik, A., Danziger, S., & Rafal, R. (2004). Parietal lobe lesions disrupt saccadic remapping of inhibitory location tagging. *Journal of Cognitive Neuroscience*, 16, 503-509.

Sapir, A., Soroker, N., Berger, A., & Henik, A. (1999). Inhibition of return in spatial attention: direct evidence for collicular generation. *Nature Neuroscience*, *2*(12), 1053-1054.

Satel, J., Hilchey, M. D., Wang, Z., Reiss, C. S., & Klein, R. M. (2014). In search of a reliable electrophysiological marker of oculomotor inhibition of return. *Psychophysiology, 51*(10)*,* 1037–1045.

Satel, J., Hilchey, M. D., Wang, Z., Story, R., & Klein, R. M. (2013). The effects of ignored versus foveated cues upon inhibition of return: An event-related potential study. *Attention, Perception, & Psychophysics*, *75*(1): 29-40.

Satel, J., Story, R., Hilchey, M. D., Wang, Z., & Klein, R. M. (2013). Using a dynamic neural field model to explore a direct collicular inhibition account of inhibition of return. *Proceedings of the 2013 International Symposium on Attention in Cognitive Systems*. arXiv:1307.5684

Satel, J., Wang, Z., Trappenberg, T. P., & Klein, R. M. (2011). Modeling inhibition of return as short-term depression of early sensory input to the superior colliculus. *Vision Research*, *51*, 987-996.

Satel, J., Wang, Z., Hilchey, M. D., & Klein, R. M. (2012). Examining the dissociation of retinotopic and spatiotopic inhibition of return with event-related potentials. *Neuroscience Letters, 524*, 40-44.

Satel, J. & Wang, Z. (2012). Investigating a two causes theory of inhibition of return. *Experimental Brain Research*, *223*(4), 469-478.

* Sato, W., Kochiyama, T., Uono, S., & Yoshikawa, S. (2009). Commonalities in the neural mechanisms underlying automatic attentional shifts by gaze, gestures, and symbols. *NeuroImage*, *45*, 984–992.

Schlaghecken, F. & Eimer, M. (2002). Motor activation with and without inhibition: Evidence for a threshold mechanism in motor control. *Perception & Psychophysics, 64*(1), 148-162.

Schlaghecken, F. & Eimer, M. (2004). Masked prime stimuli can bias "free" choices between response alternatives. *Psychonomic Bulletin & Review,* 11, 3, 463-468.

Schmidt, W. C. (1996). Inhibition of return is not detected using illusory line motion. *Perception & Psychophysics*, *58*, 883-898.

Sereno, A. B., Briand, K. A., Amador, S. C., & Szapiel, S. V. (2006a) Disruption of reflexive attention and eye movements in an individual with a collicular lesion, *Journal of Clinical and Experimental Neuropsychology*, *28*, 145-166.

Sereno, A. B., Jeter, C. B., Pariyadath, V., & Briand, K. A. (2006b). Dissociating sensory and motor components of inhibition of return. *The Scientific World Journal*, *6*, 862-887.

Sereno, A. B., Lehky, S. R., Patel, S. S., & Peng, X. (2010). A neurophysiological correlate and model of reflexive spatial attention. *Advances in Cognitive Science, 2,* 104–131.

Simion, F., Valenza, E., Umiltà, C., & Dalla Barba, B. (1995). Inhibition of return in newborns is temporo-nasal asymmetrical. *Infant Behavior and Development*, *18*(2), 189-194.

Simon, J. R. (1969). Reactions towards the source of stimulation. *Journal of Experimental Psychology*, *81*, 174-176.

Simon, J. R., Acosta, E., Mewaldt, S. P., & Speidel, C. R. (1976). The effect of an irrelevant directional cue on choice reaction time: Duration of the phenomenon and its relation to stages of processing. *Perception & Psychophysics*, *19*(1), 16-22.

Smith, D. T., Ball, K., & Ellison, A. (2012). Inhibition of return impairs phosphene detection. *Journal of Cognitive Neuroscience, 24,* 2262–2267.

Smith, D. T., Ball, K., & Ellison, A. (2014). Covert visual search within and beyond the effective oculomotor range. *Vision Research*, *95*, 11-17.

Smith, T. J., & Henderson, J. M. (2009). Facilitation of return during scene viewing. *Visual Cognition*, *17*(6-7), 1083-1108.

Smith, T. J., & Henderson, J. M. (2011a). Does oculomotor inhibition of return influence fixation probability during scene search? *Attention, Perception, & Psychophysics,73*, 2384-2398 .

Smith, T. J., & Henderson, J. M. (2011b). Looking back at Waldo: Oculomotor inhibition of return does not prevent return fixations. *Journal of Vision*, *11*(1), 1-11.

Smith, D. T., Jackson, S. R., & Rorden, C. (2009). An intact eye−movement system is not required to generate inhibition of return. *Journal of Neuropsychology*, *3*(2), 267-271.

Smith, D. T., Rorden, C., & Jackson, S. R. (2004). Exogenous orienting of attention depends on the ability to execute eye movements. *Current Biology*, *14*, 792-795.

Smith, D. T., & Schenk, T. (2012). The premotor theory of attention: time to move on?. *Neuropsychologia*, *50*(6), 1104-1114.

Smith, D. T., Schenk, C., & Rorden, C. (2012). Saccade preparation is required for exogenous attention but not endogenous attention or IOR. *Journal of Experimental Psychology: Human Perception and Performance*, *38*, 1438-1447.

Snyder, J. J., & Kingstone, A. (2000). Inhibition of return and visual search: How many separate loci are inhibited?. *Perception & Psychophysics*, *62*(3), 452-458.

Snyder, J. J., & Kingstone, A. (2001). Inhibition of return at multiple locations in visual search: When you see it and when you don't. *The Quarterly Journal of Experimental Psychology: Section A*, *54*(4), 1221-1237.

Snyder, J. J., & Schmidt, W. C. (2014). No evidence for directional biases in inhibition of return. *Psychonomic Bulletin & Review*, *21*(2), 432-435.

Snyder, J. J., Kingstone, A., & Schmidt, W. C. (2001). Attentional momentum does not underlie the inhibition of return effect. *Journal of Experimental Psychology. Human Perception and Performance*, *27*(6), 1420-1432.

Soetens, E. (1998). Localizing sequential effects in serial choice reaction time with the information reduction procedure. *Journal of Experimental Psychology. Human Perception and Performance, 24*(2), 547-568.

Soetens, E., Boer, L. C., & Hueting, J. E. (1985). Expectancy or automatic facilitation? Separating sequential effects in two-choice reaction time. *Journal of Experimental Psychology: Human Perception and Performance*, *11*(5), 598.

Souto, D., & Kerzel, D. (2009). Involuntary cueing effects during smooth pursuit: facilitation and inhibition of return in oculocentric coordinates. *Experimental brain research*, *192*(1), 25-31.

Spalek, T. M. (2007). A direct assessment of the role of expectation in inhibition of return. *Psychological Science*, *18*(9), 783-787.

Spalek, T. M., & Hammad, S. (2004). Supporting the attentional momentum view of IOR: Is attention biased to go right?. *Perception & Psychophysics*, *66*(2), 219-233.

Spalek, T. M., & Hammad, S. (2005). The left-to-right bias in inhibition of return is due to the direction of reading. *Psychological Science*, *16*(1), 15-18.

Stevens, S. A, West, G. L, Al-Aidroos, N., Weger, U. W. & Pratt, J.. (2008). Testing whether gaze cues and arrow cues produce reflexive or volitional shifts of attention. *Psychonomic Bulletin & Review, 15*(6), 1148-1153.

Sumner, P. (2006). Inhibition versus attentional momentum in cortical and collicular mechanisms of IOR. *Cognitive Neuropsychology*, *23*(7), 1035-1048.

Sumner, P. (2007). Negative and positive masked-priming - implications for motor inhibition. *Advances in Cognitive Psychology*, *3*, 317-326.

Sumner, P., Nachev, P., Vora, N., Husain, M., & Kennard, C. (2004). Distinct cortical and collicular mechanisms of inhibition of return revealed with S cone stimuli. *Current Biology, 14*(24), 2259-2263.

Takeda, Y., & Yagi, A. (2000). Inhibitory tagging in visual search can be found if search stimuli remain visible. *Perception & Psychophysics*, *62*(5), 927-934.

Tanaka, Y., & Shimojo, S. (1996). Location vs feature: Reaction time reveals dissociation between two visual functions. *Vision Research*, *36*(14), 2125-2140.

Tas, A. C., Dodd, M. D., & Hollingworth, A. (2012). The role of surface feature continuity in object-based inhibition of return. *Visual Cognition*, *20*(1), 29-47.

Tassinari, G., Aglioti, S., Chelazzi, L., Marzi, C. A., & Berlucchi, G. (1987). Distribution in the visual field of the costs of voluntarily allocated attention and of the inhibitory after-effects of covert orienting. *Acta Psychologica*, 55-71.

Tassinari, G., Aglioti, S., Chelazzi, L., Peru, A., & Berlucchi, G. (1994). Do peripheral non-informative cues induce early facilitation of target detection?. *Vision Research*, *34*(2), 179-189.

Tassinari, G., & Berlucchi, G. (1993). Sensory and attentional components of slowing of manual reaction time to non-fixated visual targets by ipsilateral primes. *Vision Research*, *33*(11), 1525-1534.

Tatler, B. W., & Land, M. (2011). Vision and the representation of the surroundings in spatial memory. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 366(1564), 596-610.

Taylor, T. L. (1999). Generating and measuring inhibition of return (Doctoral dissertation, Dalhousie University, 1997). *Dissertation Abstracts International*, 60, 1326B.

Taylor, J. E. T., Chan, D., Bennett, P. J., & Pratt, J. (2015). Attentional cartography: mapping the distribution of attention across time and space. *Attention, Perception, & Psychophysics*, 1-7.

Taylor, T. L., & Donnelly, M. P. (2002). Inhibition of return for target discriminations: The effect of repeating discriminated and irrelevant stimulus dimensions. *Perception & Psychophysics*, *64*(2), 292-317.

Taylor, T. L., & Ivanoff, J. (2003). The interplay of stop signal inhibition and inhibition of return. *The Quarterly Journal of Experimental Psychology. A, Human Experimental Psychology*, *56*(*8*), 1349–71.

* Taylor, T. L. & Ivanoff, J. (2005). Inhibition of return and repetition priming effects in localization and discrimination tasks. *Canadian Journal of Experimental Psychology Revue Canadienne de Psychologie Experimentale, 59*(2), 75-89.

Klein, R. M., & Taylor, T. L. (1994). Categories of cognitive inhibition with reference to attention. *Inhibitory Processes in Attention, Memory, and Language*, 113-150.

Taylor, T., & Klein, R. (1998). On the causes and effects of inhibition of return. *Psychonomic Bulletin & Review*, *5*(4), 625-643.

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

Theeuwes, J., Mathôt, S., & Grainger, J. (2014). Object-centered orienting and IOR. *Attention, Perception, & Psychophysics*, *76*(8), 2249-2255.

Thomas, L. E., & Lleras, A. (2009). Inhibitory tagging in an interrupted visual search. *Attention, Perception, & Psychophysics*, *71*(6), 1241-1250.

Tian, Y., Klein, R. M., Satel, J., Xu, P., & Yao, D. (2011). Electrophysiological explorations of the cause and effect of inhibition of return in a cue-target paradigm. *Brain Topography*, *24*(2), 164-182.

Tipper, S. P., Driver, J., & Weaver, B. (1991). Object-centred inhibition of return of visual attention. *Quarterly Journal of Experimental Psychology A*, *43*, 289-298.

Tipper, S. P., Jordan, H., & Weaver, B. (1999). Scene-based and object-centered inhibition of return: Evidence for dual orienting mechanisms. *Perception & Psychophysics*, *61*(1), 50-60.

Tipper, S. P., Rafal, R., Reuter-Lorenz, P. A., Starrveldt, Y., Ro, T., Egly, R., Danziger, S., & Weaver, B. (1997). Object-based facilitation and inhibition from visual orienting in the human split-brain. *Journal of Experimental Psychology: Human Perception and Performance*, *23*(5), 1522-1532.

Tipper, S. P., Weaver, B., Jerreat, L. M., & Burak, A. L. (1994). Object-based and environment-based inhibition of return of visual attention. *Journal of Experimental Psychology: Human Perception and Performance*, *20*(3), 478-494.

Tipples, J. (2002). Eye gaze is not unique: Automatic orienting in response to uninformative arrows. *Psychonomic Bulletin & Review, 9*(2), 314-318.

Trappenberg, T. P., Dorris, M. C., Munoz, D. P., & Klein, R. M. (2001). A model of saccade initiation based on the competitive integration of *exogenous and endogenous signals in the superior colliculus*. *Journal of Cognitive Neuroscience, 13*, 256-271.

Treisman, A. & Gelade, G. (1980). A feature integration theory of attention. *Cognitive Psychology*, *12*, 97-136.

Valle−Inclán, F., & Redondo, M. (1998). On the automaticity of ipsilateral response activation in the Simon effect. *Psychophysiology*, *35*(4), 366-371.

van Koningsbruggen, M. G., Gabay, S., Sapir, A., Henik, A., & Rafal, R. D. (2010). Hemispheric asymmetry in the remapping and maintenance of visual saliency maps: A TMS study. *Journal of Cognitive Neuroscience*, *22*(8), 1730-1738.

Vaughan, J. (1984). Saccades directed at previously attended locations in space. In A. J. Gale & C. W. Johnson (Eds.), *Theoretical and applied aspects of eye movement research* (pp. 143-150). North Holland: Elsevier.

Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software*, *36*(*3*), 1-48.

Vivas, A. B., Humphreys, G. W., & Fuentes, L. J. (2008). Object-based inhibition of return in patients with posterior parietal damage. *Neuropsychology*, *22*(2), 169-176.

* Vlamings, P. H. J. M., Stauder, J. E. A., van Son, I. A. M. & Mottron, L. (2005). Atypical visual orienting to gaze- and arrow-cues in adults with high functioning autism. *Journal of Autism and Developmental Disorders*, *35*(3), 267-277.

Wang, B., Hilchey, M. D., Cao, X., & Wang, Z. (2014). The spatial distribution of inhibition of return revisited: No difference found between manual and saccadic responses. *Neuroscience letters*, *578*, 128-132.

Wang, Z., & Klein, R. M. (2010). Searching for inhibition of return in visual search: A review. *Vision Research*, *50*(2), 220-228.

Wang, Z., Satel, J., Hilchey, M. D., & Klein, R. M. (2012). Averaging saccades are repelled by prior uninformative cues at both short and long intervals. *Visual Cognition, 20(7),* 825–847.

Wang, Z., Satel, J., & Klein, R. M. (2012). Sensory and motor mechanisms of oculomotor inhibition of return. *Experimental Brain Research*, *218*(3), 441-453.

Wang, Z., Satel, J., Trappenberg, T. P., & Klein, R. M. (2011). Aftereffects of saccades explored in a dynamic neural field model of the superior colliculus. *Journal of Eye Movement Research*, 4, 1-16.

Wascher, E., Schneider, D. and Hoffmann, S. (2015), Does response selection contribute to inhibition of return?. *Psychophysiology*, *52*, 942–950.

Watanabe, K. (2001). Inhibition of return in averaging saccades. *Experimental brain research*, *138*(3), 330-342.

Weaver, B., Lupiáñez, J., & Watson, F. L. (1998). The effects of practice on object-based, location-based, and static-display inhibition of return. *Perception & Psychophysics*, *60*(6), 993-1003.

Welsh, T. & Pratt, J. (2006). Inhibition of return in cue-target and target-target tasks. *Experimental Brain Research*, *174*, 167-175.

Wijnen, J. G., & Ridderinkhof, K. R. (2007). Response inhibition in motor and oculomotor conflict tasks: Different mechanisms, different dynamics? *Brain and Cognition, 63(3),* 260–270.

Wolfe, J. M., & Horowitz, T. S. (2004). What attributes guide the deployment of visual attention and how does they do it? *Nature Reviews Neuroscience*, 5, 1-7.

Wurtz, R. H. & Mohler, C. W. (1974). Selection of visual targets for the initiation of saccade eye movements. *Brain Research*, *71*, 209-214.

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

Zhang, Y. & Zhang, M. (2011). Spatial working memory load impairs manual but not saccadic inhibition of return. *Vision Research*, *51*, 147-153.

Zhao, Y, Heinke, D., Ivanoff, J., Klein, R. M., & Humphreys, G. W. (2011). Two components in IOR: evidence for response bias and perceptual processing delays using the SAT methodology. *Attention, Perception, & Psychophysics*, *73*, 2143-2159.

APPENDIX A
ON-LINE RESOURCE: DETAILED RESULTS SECTION

Central targets

RTs to central targets were submitted to a repeated measures ANOVA with factors Cueing (cued vs uncued), where 'cued' indicates that the target occurred at the same location as the cue, and CTOA (465 vs 1050 ms; see Table A1 for the mean scores from all conditions). RTs were slower to cued (M = 400 ms) as compared to uncued (M = 374.5 ms) targets [$F(1, 12) = 50.9$, $p < 0.01$]. There was no effect of CTOA ($F < 1$) and no interaction between Cueing and CTOA ($F < 1$). Analysis of FA rates (percentage of responses on no-go trials) revealed a main effect of Cueing [$F(1, 12) = 11.48$, $p < 0.01$]: The mean FA rate was less for cued (4.9%) as compared to uncued (13.9%) targets[23]. There was no effect of CTOA ($F < 1$) and no CTOA x Cueing interaction [$F(1, 12) = 1.79$, $p > 0.20$][24].

Peripheral targets

RTs to peripheral targets were submitted to a repeated measures ANOVA with factors Cueing (cued, uncued and central cue), CTOA (465 vs 1050 ms) and Target-Response (T-R) Compatibility (correspondence vs non-correspondence), where 'correspondence' indicates that the target occurred on the same side as the responding

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[23] This accuracy advantage for central targets when the central location was cued is larger than in Ivanoff and Klein (2001). Accompanied by an RT delay for cued, central targets (which Ivanoff & Klein considered might be an example of central IOR) this pattern represents a striking SAT. Why this output-based effect might have been magnified by our careful monitoring of and feedback on our participants' oculomotor behavior (while, as will be seen in the next section, the SAT effect observed by Ivanoff and Klein with peripheral targets was eliminated) cannot be resolved until further experiments are carefully aimed answering it.

[24] Analysis of log odds of the FA rates (percentage of responses on no-go trials) revealed the same effects (a significant main effect of Cueing [$F(1, 12) = 13.41$, $p < 0.01$], no effect of CTOA ($F < 1$), and no CTOA x Cueing interaction [$F(1, 12) = 2.41$, $p > 0.10$].

hand (see Table A2 for the mean scores from all conditions). RTs were faster at long (M = 407 ms) as compared to short (M = 425 ms) CTOAs [$F(1, 12) = 8.26$, $p < 0.05$]. There was a main effect of Cueing [$F(2, 24) = 5.95$, $p < 0.01$]. As in Ivanoff and Klein (2001), RTs following central cues (M = 408 ms) were not statistically different from the uncued RTs [M = 413 ms; $t(12) = 1.09$, $p > 0.25$], while RTs following cued (M = 427 ms) trials were slower than the RTs following uncued [$t(12) = 2.45$, $p < .05$] and center cue [$t(12) = 2.99$, $p < 0.05$] trials. There were no other significant or marginally significant effects or interactions. The analysis of the FA rates revealed neither effects nor interactions in a repeated measures ANOVA considering the aforementioned factors. Most critically, and as predicted, there was no effect of Cueing ($F < 1$) on the FA rate[25]. In point of fact, there were approximately 1% more FAs for cued as compared to uncued targets.

[25] None of these findings changed when we analyzed log odds of the FA rates.

Table A1

Central targets; Mean RTs (in ms) and FA (in %) for each level of factors Cueing and CTOA.

	Cueing			
	Cued		Uncued	
CTOA	RT	FA	RT	FA
Short	405	5.9%	377	11.4%
Long	395	3.8%	372	16.6%

Table A2

Peripheral targets; Mean RTs (in ms) and FA (in %) for each level of factors Cueing, T-R Compatibility and CTOA.

		Cueing					
		Cued		Uncued		Center Cue	
T-R Compatibility	CTOA	RT	FA	RT	FA	RT	FA
Corresponding	Short	420	6.5%	420	3.3%	413	10.3%
	Long	410	11.7%	400	9.7%	394	5.0%
Non-corresponding	Short	434	3.7%	421	6.2%	421	6.5%
	Long	424	10.8%	392	9.0%	393	8.5%

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