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Modeling inhibition of return as short-term depression of early sensory input to the superior colliculus

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ABSTRACT

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

1. Introduction

Inhibition of return (IOR) is an orienting phenomenon characterized by slower behavioral responses to targets presented at spatially cued, relative to uncued locations, when the cue-target onset asynchrony (CTOA) is longer than approximately 200 ms (for a review, see Klein, 2000). This phenomenon was first discovered by Posner and Cohen (1984), with a model task (see Fig. 1A for an illustration) in which non-predictive peripheral cues are followed by targets that require simple detection responses. Posner and Cohen (1984) showed that reaction times (RTs) to targets appearing at previously cued locations were faster than RTs to targets appearing at uncued locations, so long as the CTOA was short. However, when CTOA was extended, this early benefit evolved into a behavioral cost, as exhibited by slower RTs for targets presented at cued locations than for targets at uncued locations (see Fig. 1B for an illustration of these effects). This later effect has been termed IOR (Posner, Rafal, Choate, & Vaughan, 1985), and has since been demonstrated by many researchers using a number of experimental paradigms (for a discussion and testing of various IOR experimental paradigms, see Taylor & Klein, 1998, 2000). Although the neural processes underlying IOR are still under investigation, previous behavioral (Rafal, Calabresi, Brennan, & Sciolto, 1989), lesion (Posner et al., 1985; Sapir, Soroker, Berger, & Henik, 1999; Sereno, Briand, Amador, & Szapiel, 2006), and developmental (Simion, Valenza, Umiltà, & Barba, 1994; Valenza, Simion, & Umiltà, 1995) studies have suggested that the oculomotor system, particularly the superior colliculus (SC), is intimately involved with the generation and processing of IOR. Neurophysiological work has further confirmed the involvement of the SC in IOR (for a review, see Fecteau & Munoz, 2006).

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

Recent single-unit recording studies using the cue-target experimental paradigm have shown that target induced neural activity in the iSC is greatly reduced for previously cued, as compared to uncued targets (Dorris, Klein, Everling, & Munoz, 2002; Fecteau & Munoz, 2005). This reduction in activity is highly correlated with saccadic reaction times (SRTs) to targets, further cementing the relationship of iSC activity to behaviorally exhibited IOR (Fecteau & Munoz, 2005). More importantly, when electrical stimulation was delivered through the recording electrode to elicit a saccade, the latency of these electrically evoked saccades was actually faster for previously cued regions (Dorris et al., 2002), suggesting that

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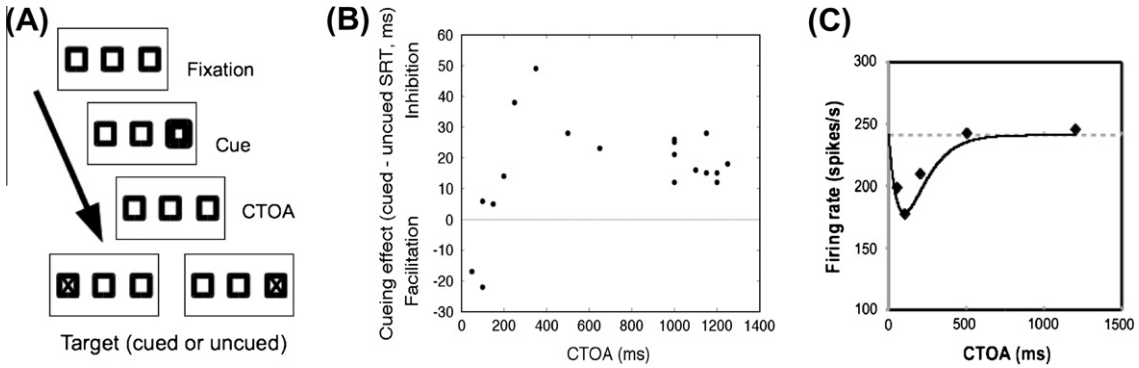

Fig. 1. (A) Sequence of events in a typical trial using a traditional cue-target IOR paradigm. (B) Human and monkey behavioral data from studies using a cue-saccade paradigm demonstrating the time course of IOR (Abrams & Dobkin, 1994, 1995; Briand, Larrison, & Sereno, 2000; Dorris, Taylor, Klein, & Munoz, 1999; Maylor & Hockey, 1985; Rafal, Egly, & Rhodes, 1994; Reuter-Lorenz et al., 1996; Taylor, 1997). The cueing effect (cued–uncued SRT, ms) is shown as a function of CTOA, with facilitation seen at short CTOAs, and IOR at long CTOAs (adapted from Klein, 2000). (C) Illustration of the sensory STD thought to underlie the behavioral observation of IOR. The diamond data points denote the response of visual neurons (in the sSC) to cued targets, the dashed line denotes the average firing rate of visual neurons to uncued targets (adapted from Fecteau and Munoz (2005)). These single-unit recordings demonstrate that target elicited early sensory input strength is reduced following a previous stimulation. In our model, an alpha function is used to approximate this sensory STD process, as illustrated by the solid line.

neural activity was not directly suppressed in the iSC following a cue.

Thus, the IOR effect is not caused by active inhibition of recently stimulated iSC sites, but rather by a reduction in the strength of subsequent input signals to these neurons. This hypothesis was supported by Fecteau and Munoz (2005), who found reduced responses to cued targets in the sSC (see Fig. 1C, diamonds). As mentioned earlier, this reduction of discharge in the sSC is purely sensory, hence, we label it short-term depression (STD) of sensory input. As shown in Fig. 1C, this sensory STD can be modeled with an alpha function $\alpha_{CTOA} = A\frac{t_{CTOA}}{t_{MAX}}e^{\left(1-\frac{t_{CTOA}}{t_{MAX}}\right)}$, with parameters $A = -63$ and $t_{MAX} = 100$ specifying the maximal discharge reduction and the time when this discharge reduction reaches its maximum. Also note that this STD function nicely correlates (negatively) with the behavioral IOR effects reported in previous studies (see Fig. 1B). Based on this sensory STD in the sSC, Fecteau and Munoz (2005) postulated that IOR simply "reflects a habituated sensory response occurring in early sensory areas that is subsequently transmitted through the rest of the brain" (p. 1722). Furthering this line of thought, Dukewich (2009) proposed a theory wherein IOR is simply the result of habituation-like mechanisms at multiple stages of processing, occurring anytime a pathway is repeatedly stimulated (see also Huber, 2008; Patel, Peng, & Sereno, 2010).

In sum, although IOR, as a behavioral effect, could have multiple underlying neural mechanisms, we believe a large set of IOR effects observed in the cue-target paradigm can be explained in the input domain, through STD of early sensory inputs. The primary purpose of this work is to implement and quantify this sensory STD hypothesis of IOR by expanding an established DNF model of the iSC (Trappenberg, Dorris, Munoz, & Klein, 2001) to include STD of early sensory input strength. Furthermore, this work compares the results of simulations with established experimental results (Bell & Munoz, 2008; Fecteau & Munoz, 2005; Watanabe, 2001), and makes predictions that can be investigated empirically. Although we do not expect that this model will be able to account for all manifestations of IOR, we believe that much can be learned from the boundary conditions of its successes.

2. Dynamic neural field model of the SC

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

2.1. Model architecture

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

The interaction structure within the iSC is captured by the interaction matrix, w, that depends only on the spatial distance between nodes (Trappenberg et al., 2001). This interaction profile is approximated with two Gaussians, as defined in Eq. (1), and is kept constant across all simulations. Although this lateral interaction was chosen to approximate cell recordings in the iSC of monkeys (see Trappenberg et al., 2001), it is not an exact fit to the neurophysiological data. All simulations used the following interaction matrix parameters: $a = 72$, $b = 24$, $c = 6.4$, $\sigma_a = 0.6$, and $\sigma_b = 1.8$.

$$w_{ij} = a * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_a^2}\right) + b * \exp\left(\frac{-((j-i)\Delta x)^2}{2\sigma_b^2}\right) - c \qquad (1)$$

$$\tau \frac{du_i(t)}{dt} = -du_i(t) + \sum_j w_{ij} r_j(t) \Delta x + I_i(t) + u_0 \quad (2)$$

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)} \quad (3)$$

$$I_k = d * \exp\left(\frac{((k-i)\Delta x)^2}{2\sigma_d^2}\right) \quad (4)$$

The dynamics of the internal state, $u_i(t)$, of node i is described in Eq. (2), where $\tau = 10$ ms is a time constant, w_{ij} is the connection strength (weight) between node i and node j, $r_j(t)$ is the activity level (average firing rate) of node j, $I_i(t)$ represents the external input to node i, and $u_0 = 0$, is a constant resting level. The activity of node i, $r_i(t)$, as a function of its internal state, $u_i(t)$, is defined by a sigmoidal gain function (Eq. (3)), where $\beta = 0.07$ and $\theta = 0$ were used as parameters in all simulations to define the steepness and offset of the sigmoid.

The iSC is a neural structure where bottom-up (exogenous) inputs and top-down (endogenous) inputs are integrated (for a description of projections to and from the SC, see Fecteau & Munoz, 2006; Munoz & Fecteau, 2002). Our model of the iSC receives both exogenous (I_{exo}), and endogenous (I_{endo}) inputs. Both types of input signals take on a Gaussian spatial shape, centered at location i. Thus, input to other nodes (k) in the network depends on the distance between nodes i and k, as represented by Eq. (4), where d represents the strength of the input, and σ_d represents the width of the input.

2.2. Input and output parameters

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

Endogenous move signal inputs, with an onset delay of 120 ms, were sustained until a reaction occurred. Reflecting the well-known foreperiod, or warning signal effect, reaction times vary with the interval between a warning cue and a target (Posner, Klein, Summers, & Buggie, 1973). Consequently, the strengths of endogenous input signals (the move signals) were modulated as a function of CTOA. The strength of these signals was always the same for both validly (same side) and invalidly (opposite side) cued targets, so they do not significantly affect the magnitude of the IOR effect, only the SRTs for different CTOAs. For this work, the strength of these signals have been chosen in order to fit monkey behavioral data (SRTs; Fecteau & Munoz, 2005) as accurately as possible. The foreperiod effect was simulated by using a linear equation ($y_1 = 7.3$, $m_1 = 0.3$) to increase the strength of the endogenous move signal as a function of CTOA, until CTOA = 200 ms, and a second linear equation ($y_2 = 14.5$, $m_2 = -0.0024$) to decrease the strength of this signal when CTOA was greater than 200 ms. Thus, due to temporal expectation effects, SRTs in all conditions are gradually increased as a function of CTOA until a CTOA of 200 ms, at which point they begin to decrease again. In simulations of the predictive cueing paradigm, an additional predictive, endogenous input, I_{pred}, was applied to the network after cue offset, with an initial strength of $d = 1$ and an effective time constant of $teff = dt/350$, which slowly increases the signal over time.

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

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

3. Simulations

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

3.1. Early benefits and subsequent costs following uninformative peripheral cues

When using the model cue-target IOR task, subjects often exhibit behavioral benefits (faster RTs) to cued targets at short CTOAs (Posner & Cohen, 1984). It has been proposed that attentional capture by a cue results in a brief period of enhanced processing in the vicinity of the cue, as if, during this period, the "effective contrast" (Reynolds & Chelazzi, 2004, p. 15) of stimuli presented there is increased. In our model, this phenomenon occurs, despite the reduced signal strength due to STD, as a result of the summation of

cue and target-elicited neuronal activity (for similar explanations, see Bell, Fecteau, & Munoz, 2004; Dukewich, 2009). Such an activity summation process for a cued target is illustrated in the simulated node activity seen in Fig. 2C, as well as the monkey neurophysiological data seen in Fig. 2A (Fecteau & Munoz, 2006). Because neuronal activity elicited by exogenous inputs is transient, this behavioral benefit disappears when CTOA is increased.

When CTOA is larger than about 50 ms in monkeys, or 100 ms in humans, this behavioral benefit, as measured with SRTs, reverses into a behavioral cost (i.e., IOR; Klein, 2004b). Note that we are exploring IOR using saccadic responses. It is well-known that the cross-over from benefits to costs at the cued location is quite a bit earlier when IOR is explored with saccades than when it is explored with manual responses (e.g. Briand et al., 2000; for a review, see Klein, 2004b). In our model, this later inhibitory effect is implemented in the input domain (i.e., sensory STD). Once an exogenous input reaches the iSC, the amplitude, or strength, of subsequent exogenous inputs to the same iSC location is reduced for a specified period of time (see Fig. 1C). This reduction of cued target related input strength has been demonstrated neurophysiologically in the monkey iSC (see Fig. 2B; Dorris et al., 2002; Fecteau & Munoz, 2005), as well as the sSC (which receives only early sensory inputs; see Fig. 1C; Fecteau & Munoz, 2005). Fig. 2D demonstrates that the model closely reproduces neurophysiological results at a CTOA of 200 ms. Other CTOAs were also simulated and compared to behavioral data (see Fig. 3), demonstrating that our model successfully reproduced monkey data at a number of CTOAs in a cue-target experimental paradigm.

3.2. IOR and predictive cueing

In a typical cue-target paradigm, the cue is uninformative. Early studies (e.g., Posner & Cohen, 1984) showed that the observed IOR effect disappears when the cues are predictive. A recent neurophysiological study (Bell & Munoz, 2008) sheds some light on this interesting observation. The experimental setup of this study was identical to the previously described cue-target paradigm, except that the target appeared at the cued location in 80% of the trials. Bell and Munoz (2008) found that when monkeys learned how to use the cue to predict target locations, behavioral IOR disappeared and facilitation was observed at long CTOAs (see Fig. 4C). As shown in Fig. 4A, this observation was accompanied by a pre-target build-up only for cued targets in cell recordings. This suggests that predictive, endogenous information reaches the iSC, bringing the neural activity of the expected iSC location closer to threshold before target appearance. Although the input strength for cued targets is reduced, due to sensory STD, the cued target cell still reached threshold first, leading to faster SRTs for cued targets at relatively long CTOAs (e.g., 650 ms; see Fig. 4A).

To capture this finding in our simulations, shortly after cue onset a small, endogenous input that builds up slowly over time was transmitted to the cued iSC location (see Fig. 4B). This implementation is also justified by other studies which have demonstrated that increases in target elicited activity during the pre-target period can be linked to top-down processes (Dorris & Munoz, 1998; Fecteau, Bell, & Munoz, 2004; Ignashchenkova, Dicke, Haarmeier, & Thier, 2004).

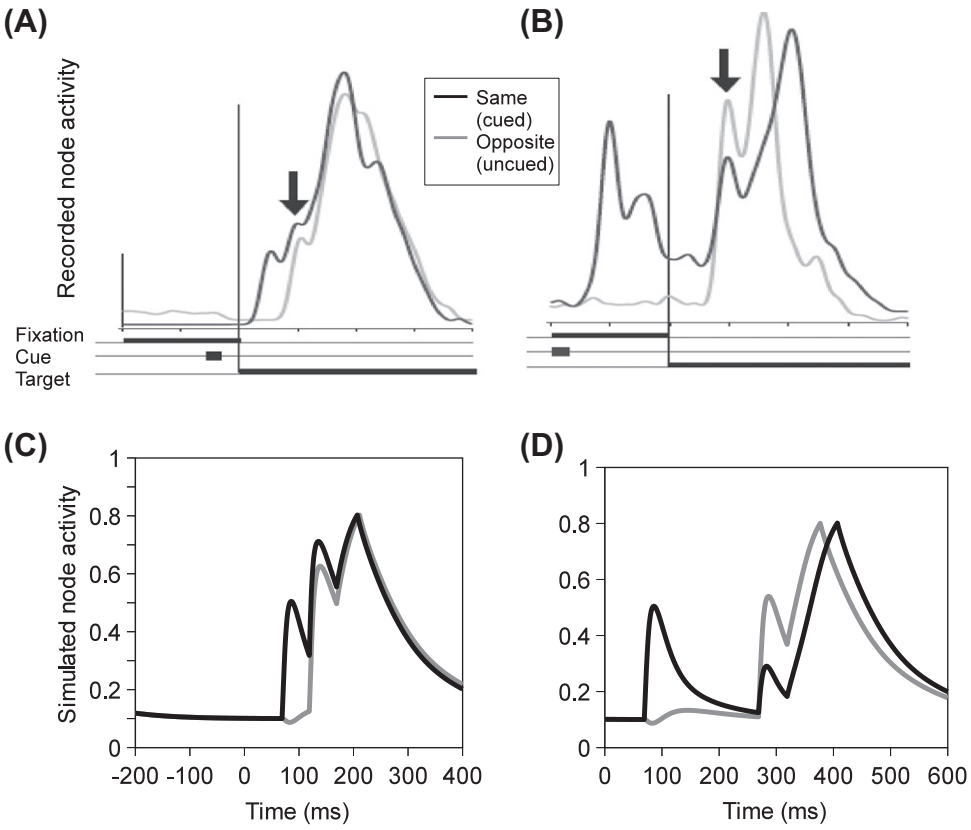

Fig. 2. (A) Neurophysiological firing rates over time when CTOA = 50 ms. As indicated by the arrow, the strength of target elicited activity is reduced for cued, as compared to uncued, targets. Cued targets still hit threshold before uncued targets because the cue elicited activity has not yet dispersed, leading to faster SRTs for cued than for uncued targets (reprinted from Fecteau & Munoz, 2006). (B) Neurophysiological firing rates over time when CTOA = 200 ms. The cue elicited activity is transient and has nearly dispersed when targets appear. Due to sensory STD, target elicited exogenous inputs are reduced when cued, so the activity elicited by an uncued target hits threshold first, leading to faster SRTs for uncued targets (reprinted from Fecteau and Munoz (2006)). (C and D) Simulated node activity over time when CTOA = 50 ms and 200 ms.

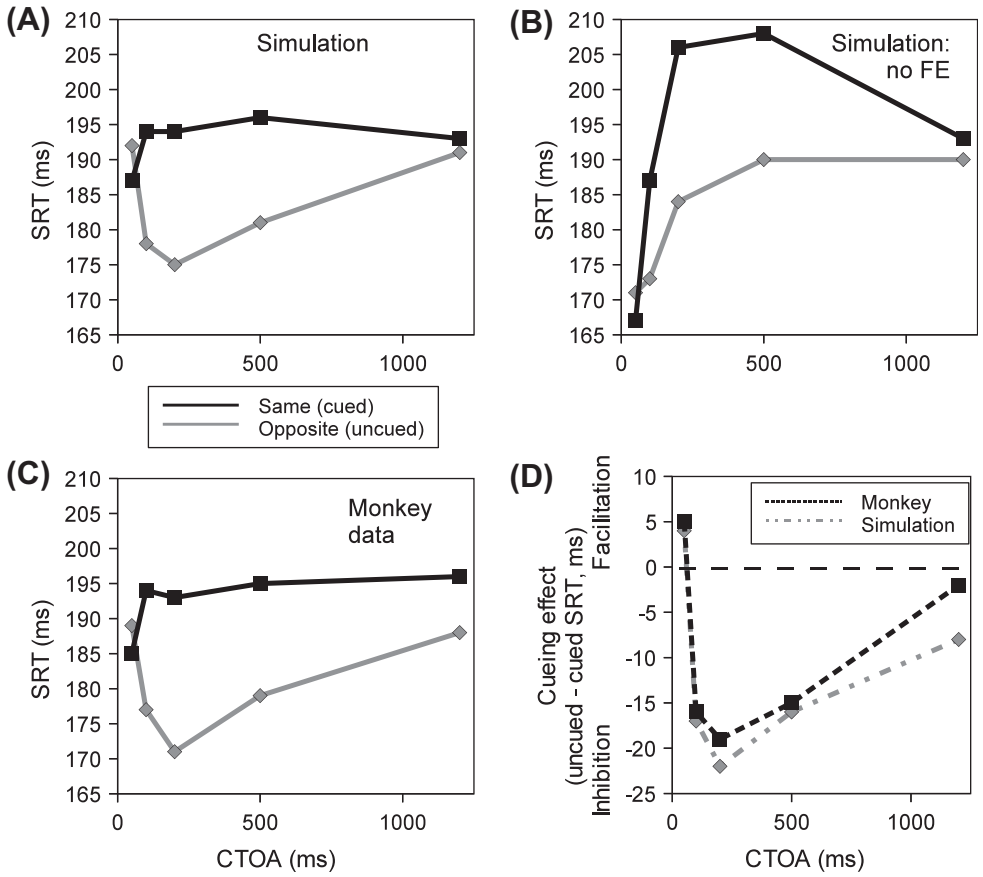

Fig. 3. (A) Simulated SRTs for cued and uncued targets at various CTOAs, with the inclusion of a foreperiod effect (FE), which modulates endogenous move signal strength to account for temporal predictability. At CTOAs less than 200 ms, endogenous move signals are reduced (relative to simulations with no FE), slowing SRTs. At longer CTOAs, move signal strength is increased, leading to faster SRTs relative to simulations with no FE effect. (B) Simulated SRTs for cued and uncued targets at various CTOAs without temporal predictability. (C) Monkey SRTs for cued and uncued targets at various CTOAs (replotted from Fecteau & Munoz, 2005). (D) Simulated and monkey data (replotted from Fecteau & Munoz, 2005) illustrating cueing effects, which are calculated as uncued SRT–cued SRT, such that positive values indicate behavioral facilitation, and negative values indicate IOR.

Simulated and monkey behavioral results (Bell & Munoz, 2008) are compared in Fig. 4C, where IOR is still exhibited at a relatively short CTOA (250 ms), but is eliminated behaviorally at a longer CTOA (650 ms). This nicely demonstrates that there are experimental conditions for which the underlying mechanisms of IOR may be occurring, even though IOR is not exhibited behaviorally, due to the competition with top-down, endogenous inputs.

3.3. Saccadic averaging and IOR

When participants make a quick saccade to one of two stimuli that are presented simultaneously and in close spatial proximity, a first saccade is often directed to an intermediate location between these two stimuli. This phenomenon has been termed saccadic averaging (Ottes, Van Gisbergen, & Eggermont, 1984), and has been previously investigated with DNF modeling techniques (Wilimzig et al., 2006). Particularly in the presence of distractors that are nearby targets, saccadic curvature has also been observed either with saccades launched in the direction of the distractor and arriving at the target or with saccades arriving at the target on a curved path initially biased away from the distractor's location (e.g., Arai, McPeek, & Keller, 2004; Theeuwes, Kramer, Hahn, & Irwin, 1998). Because our model of the iSC generates a saccade to the "winning" location when a threshold level of activation is exceeded, it cannot generate or predict curvature. Indeed, like Arai and Keller (2005), we believe that curvature is generated downstream from the iSC, perhaps at the level of the brainstem where signals from from the iSC and frontal eye fields converge.

Watanabe (2001) further demonstrated that IOR interacts with saccadic averaging. When two identical visual targets were presented closely and simultaneously in the peripheral visual field, most saccades showed a tendency to land near the mid-point between the two targets (see Fig. 5; gray bars show the 95% confidence intervals of the mean landing positions observed in Watanabe, 2001), replicating previous work (Ottes et al., 1984). However, when a non-predictive visual cue was presented 600 ms before target appearance, saccades were biased away from the cued location (see Fig. 5). This later finding was explained in terms of IOR biasing the average saccade away from previously cued (attended) locations (Watanabe, 2001). Note that behavioral results (Watanabe, 2001) tend to show a rightward bias in averaging saccades, although this bias is not incorporated into our model.

Saccadic averaging occurs due to the proximal locations of the two target stimuli (Watanabe, 2001; see also Chou, Sommer, & Schiller, 1999). In our model, when two closely located nodes are equally stimulated, due to the dynamic interaction of the iSC, neurons located around the middle of the two stimulated nodes will eventually become the most excited nodes, reaching the saccade initiation threshold first, and resulting in saccades landing in the middle of the two target locations. However, when one of the targets is cued, due to sensory STD, the actual visual input for the cued target will be reduced. That is, in our model, in the cue-double-target paradigm, two spatially proximal nodes will receive

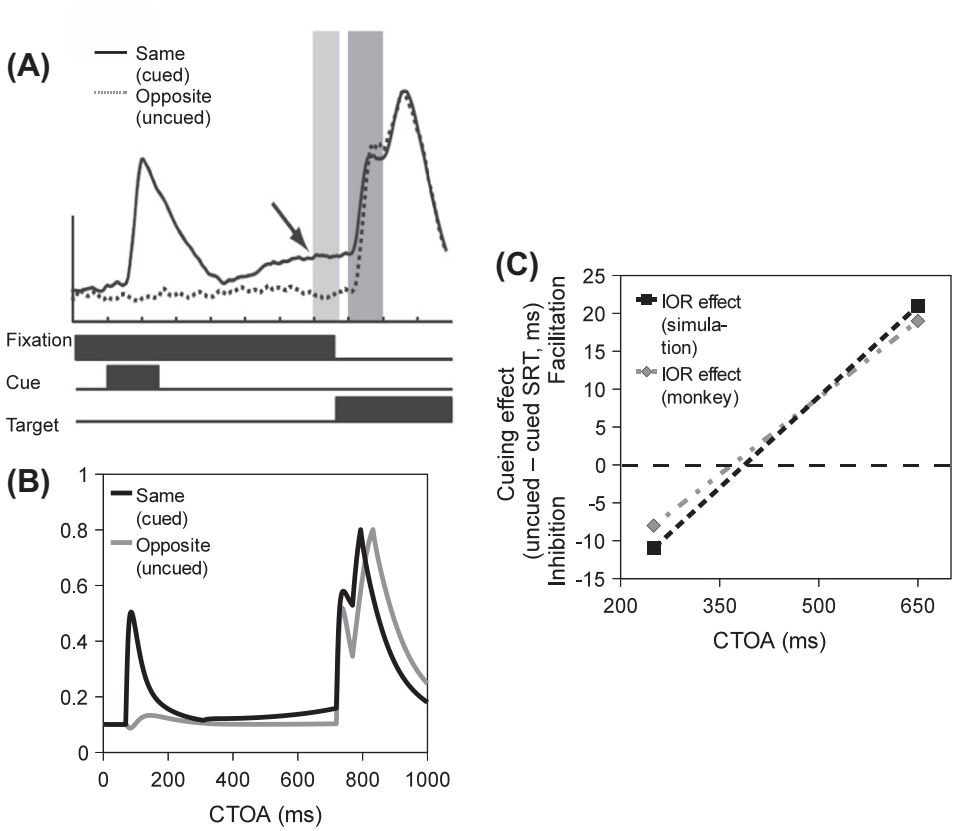

Fig. 4. (A) Neurophysiological firing rates over time when CTOA = 650 ms in a predictive cueing paradigm (reprinted from Bell and Munoz (2008)). The arrow indicates that target cell activity is increased during the pre-target period (light gray bar time period), due to the predictive nature of the cue. The darker gray bar (post-target period) indicates the time period when target-elicited inputs arrive at the iSC, and demonstrates that cued inputs are reduced in strength as compared to uncued inputs. Even though cued target-related inputs are reduced (STD), cued responses are still faster than uncued responses, due to the overwhelming strength of the top-down, predictive, cue elicited input, which builds up during the cue-target interval. (B) Corresponding simulation of node activity over time when CTOA = 650 ms. (C) Simulated and monkey data (replotted from Bell and Munoz (2008)) illustrating cueing effects (uncued SRT–cued SRT) in a predictive cueing paradigm.

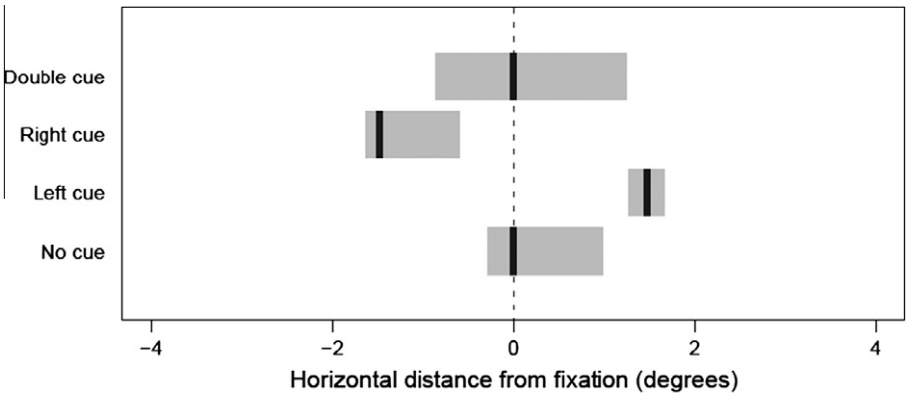

Fig. 5. Saccadic averaging and IOR. When two targets are presented simultaneously, the majority of saccades land at the mid-point between both targets (no cue and double cue conditions). If one of the potential target locations is pre-cued (left and right cue conditions), saccades are biased toward the opposite location, due to the influence of IOR at the cued location. Gray bars denote the 95% confidence intervals of the mean landing positions in each condition calculated from the behavioral results reported in Watanabe (2001). Black lines denote the simulated mean landing position in each condition, obtained in our model without including any noise.

unbalanced inputs. Although these two inputs merge into one activity packet, the peak activity will be located closer to the nodes which received stronger input (i.e., the uncued target). Note that saccadic averaging will not necessarily occur in all experimental paradigms. For example, if target locations are far enough apart, there will be a single winner, rather than an averaged landing point (for a more detailed investigation of saccadic averaging using DNF modeling, see Wilimzig et al., 2006). Some authors (e.g., Arai & Keller, 2005) have suggested that the iSC may not be characterized by a Mexican-hat lateral interaction profile and that saccadic averaging behavior may be the result of downstream (brainstem) processes rather than merging bubbles in the iSC. However, these issues are still unresolved empirically, and we believe, as described above, that there is sufficient evidence for our theoretical interpretation.

Simulations were performed to demonstrate saccadic averaging (without cueing and with double cues), as well as how IOR interacts with saccade averaging (as in Watanabe, 2001; one of

the targets was cued). In each simulation, transient exogenous inputs representing the appearance of cue and target stimuli were presented to the network with amplitudes of $d = 60$, widths of $\sigma_d = 0.45$ and sustained endogenous inputs which represent the move signal had amplitudes of $d = 10$. The distance between the two target locations was set to 5° (as in Watanabe, 2001) and the CTOA was set to 600 ms since IOR was observed in both previous behavioral studies and our simulations with this CTOA (see Figs. 1 and 3). The purpose of these simulations was not to precisely reproduce behavioral findings, but rather to demonstrate how IOR interacts with saccadic averaging (Watanabe, 2001). The parameters of our model, such as the noise level, could be modified to replicate the landing position distribution behavioral observations in Watanabe (2001).

The mean center of gravity of the saccade landing locations in all simulations are plotted in Fig. 5 (solid black lines). It is clear from these results that saccades tend to land around the mid-point between two targets when no cues, or double cues are presented, reproducing previous behavioral results (as seen in Fig. 5; simulation means are all within the 95% confidence intervals of behavioral results). When one of the targets is cued, both real saccades, and those of our simulations (see Fig. 5), tend to be biased away from the mid-line toward the uncued target, reflecting the effects of IOR on the cued location. These results clearly demonstrate that the interaction structure of the iSC is important to the behavioral exhibition of IOR, since saccadic averaging (which depends on the lateral interaction of the iSC) interacts with IOR, and can be explained with the sensory STD hypothesis of IOR.

4. Discussion

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

These findings suggest that, with repeated stimulation, sensory input to the iSC will be reduced. To reflect this fact, our model reduces target-related exogenous input strengths when they are preceded by a cue at the same location. Thus, for a period of time following peripheral cues, subsequent exogenous inputs to cued locations are reduced in strength. With this simple manipulation, our model reproduces the prototypical experimental effects in the Posner cueing paradigm. Capture of attention, as manifested by shortened SRTs to cued, relative to uncued targets, was observed at short CTOAs, and IOR, characterized by longer SRTs to cued than to uncued targets, was observed at longer CTOAs.

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

4.1. Mechanisms underlying behaviorally exhibited IOR

There has been extensive discussion regarding the underlying neurodynamics and the stages of cognitive processing involved in IOR, particularly regarding the relative contributions of early sensory and later attentional processes (Klein, 2000, 2004b). In most cases, different theories are exclusive, in the sense that each particular theory proposes a process that attempts to explain all of IOR, without allowing for the possibility of other independent processes contributing to the phenomenon. A number of researchers have proposed that IOR is associated with relatively late attentional processes in neocortical areas of the brain (Godijn & Theeuwes, 2002; Ivanoff & Klein, 2001; Klein & Taylor, 1994; Rafal et al., 1989; Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987). These ideas seem to have been strongly influenced by early observations of behaviorally exhibited IOR in experimental paradigms that ensure little or no SC involvement (e.g., Tipper, Driver, & Weaver, 1991; Tipper, Weaver, & Watson, 1996). Other researchers have suggested that IOR is the result of early sensory processes, as demonstrated by decreased early sensory signals associated with validly cued IOR trials (Fecteau & Munoz, 2005; Hopfinger & Mangun, 2001; Ivanoff & Klein, 2006; Posner & Cohen, 1984; Prime & Ward, 2004; Reuter-Lorenz, Jha, & Rosenquist, 1996). Early behavioral studies also demonstrated that IOR did not follow voluntary shifts of attention without peripheral stimulation (Posner & Cohen, 1984; Rafal et al., 1989), suggesting that what generates IOR is peripheral stimulation, oculomotor activation, or both (Klein, 2004b). The involvement of early sensory processes in the generation of IOR was further supported by recent electroencephalographic (EEG) studies (for a review, see Prime & Ward, 2006). Several EEG studies have shown that behavioral IOR was accompanied by an amplitude reduction of the early visual P1 component. More importantly, neurophysiological investigations have found that both visual neurons located in the sSC (which only receives early sensory inputs), and visuomotor neurons in the iSC (which control the initiation of saccades), show reduced activation to cued targets in typical IOR tasks (Dorris et al., 2002; Fecteau & Munoz, 2005). It has further been shown that cells in the iSC are not directly inhibited on validly cued trials, but receive reduced target-related inputs (Dorris et al., 2002; Fecteau & Munoz, 2005). The amount of this reduction in signal amplitude is a function of CTOA, as demonstrated through single-unit recordings of the sSC and iSC (Fecteau & Munoz, 2005).

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

saccadic averaging of IOR (Watanabe, 2001), and a predictive cueing paradigm demonstrating the simultaneous presence of IOR and endogenous attention at the same location (Bell & Munoz, 2008).

4.2. Cortically-based IOR

A number of experiments have demonstrated IOR in cases with little or no SC involvement (Sumner, Nachev, Vora, Husain, & Kennard, 2004; Tipper et al., 1991, 1996), or with endogenous signals (e.g., Taylor & Klein, 2000). Results from these studies indicate that there may be additional, cortical processes contributing to IOR, in addition to sensory STD. A potential explanation of these results is that additional habituation-like processes could occur at, or be propagated to, multiple levels of processing (Dukewich, 2009; Fecteau & Munoz, 2005). For example, STD of inputs to saliency maps with environmental or object-based coordinates in posterior parietal cortex (PPC) may be able to explain certain experimental results (Tipper et al., 1991, 1996) that the current implementation cannot. This proposal could be examined in future computational work by extending the current model to include more detailed, dynamic, endogenous input modulation. A modular implementation could also be developed with the inclusion of multiple dynamic networks representing different areas of the brain involved in orienting responses, including, for example, sSC, PPC, the frontal and supplementary eye fields, and prefrontal cortex.

4.3. Predictions

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

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

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

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

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

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

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

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

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

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

The current simulations and the behavioral study of Watanabe (2001) demonstrated that saccades tend to be biased away from a cued location at a CTOA long enough to generate behavioral IOR. This is due to the interaction of IOR with the dynamic lateral interaction of the iSC which causes saccadic averaging in some conditions. At shorter CTOAs known to cause behavioral facilitation, our model predicts that saccades will be biased toward the cued location, since the cue elicited exogenous input in the SC has not yet completely decayed, and consequently the cued target node will reach threshold and initiate a saccade before the uncued target node.

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

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

It should be mentioned that such top-down modulation of IOR has long been discussed and explored by IOR scholars. In a review of IOR (Klein, 2000), such top-down modulation was referred to as an attentional control setting (ACS). The ACS theory states that the deployment of attention (both spatial and temporal) depends on the cognitive task requirements. In the Posner task with predictive cues, ACS would predict that more attentional resources would be placed at the cued location following a cue. Such endogenous input would summate with the target related input, bringing the cell activity to threshold more quickly. As demonstrated in Fecteau

and Munoz (2005), target-related exogenous inputs are reduced because the target location has been previously stimulated by the cue, due to STD. However, such STD reaches its maximum effect shortly after the onset of the cue and then decays over time. As a result, at short CTOAs, while the STD is still strong, endogenous inputs will not cancel all the effects caused by the STD. However, at longer CTOAs, when the STD process has further decayed, the behavioral observation of IOR disappears.

5. Conclusion

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

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

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