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What is causing “inhibition of return” in spatial cueing tasks?

Temporally disentangling multiple cue-triggered effects on multiple time scales using response history and conditional accuracy analyses

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Abstract

Research on spatial cueing has shown that uninformative cues often facilitate mean response time (RT) performance in valid- compared to invalid-cueing conditions at short cue-target stimulus-onset-asynchronies (SOAs), and robustly generate a reversed or inhibitory cueing effect at longer SOAs that is widely known as inhibition-of-return (IOR). To study the within-trial time course of IOR we employ discrete-time hazard and conditional accuracy analyses to describe and model the shapes of the RT and accuracy distributions measured in two experimental tasks. In contrast to the mean performance measures, our distributional analyses show that (a) the uninformative cue generates response channel activation, (b) which continues during the cue-target interval so that the cue location must be stored in spatial working memory, (c) the premature cue-triggered response is selectively inhibited before target onset, (d) the IOR effect (valid versus invalid cueing) emerges around 160 ms after target onset in the hazard functions when cue-target SOA exceeds ~200 ms, quickly increases and decreases in size, and is gone within 120 ms, (e) the inhibitory component does not diminish over the course of the experiment, and (f) the location of an additional central cue relative to the current focus of spatial attention can generate response channel activation as well. These distributional data show that mean performance patterns conceal crucial information about behavioral dynamics, and suggest that sensory IOR is the direct result of encoding the cue location in spatial working memory to promote change detection, instead of attention leaving an inhibitory tag to promote visual search.

Key words: inhibition of return, spatial cueing, distributional analysis, event history analysis, working memory, response inhibition, attention, habituation

Introduction

The work of Charles W. Eriksen is still an inspiration to many of us, and he was one of the first to introduce informative spatial cues to study attentional selection processes (Eriksen & Hoffman, 1973). The early studies showed that informative spatial cues facilitate various mean performance measures (Jonides, 1981; Posner, 1980). In general, this positive effect of informative cueing increases with an increasing cue-target *inter-stimulus interval* (ISI) or *stimulus-onset-asynchrony* (SOA) until an asymptote is reached, and is typically interpreted as the result of increased attentional focusing on the target location ahead of target onset.

Soon thereafter, Posner and Cohen (1984) used *uninformative* cues and discovered a reversed or negative cueing effect, the main topic of this paper. At the start of the trial they presented a central box flanked by two peripheral boxes 8 degrees to the left and right of the fixation box. Then one of the lateral boxes was cued by brightening its outline for 150 ms. Twenty percent of the trials were catch trials in which no target appeared. In the remaining trials, the target appeared in the center of one box with a cue-target SOA of either 0, 50, 100, 200, 300, or 500 ms. The supra-threshold target appeared centrally (60 percent of the trials) or peripherally (10 percent of the trials for each lateral box). If the target appeared peripherally it could appear randomly at the cued location (valid cueing) or uncued location (invalid cueing). Participants were instructed to respond to the target as quickly as possible by pressing a single key.

Based on the earlier studies, Posner and Cohen (1984) theorized that the uninformative cue would summon attention, leading to an initial *response time* (RT) advantage for the cued relative to the uncued side. “However, because targets occurred mainly at the center, subjects would be expected to keep attention there insofar as they were able to. Thus, any advantage of the cued side as a result of attention should be lost as attention was returned to the center. We would then be able to compare the two sides to observe the consequences of previous peripheral cue on target detection latencies once attention was withdrawn” (p. 534).

While responses to central targets were found to be fastest for each SOA, Posner and Cohen (1984) indeed found that for SOAs below 150 ms peripheral target detection was faster on average when the target appeared in the cued location than in the uncued location. However, for the SOAs of 300 and 500 ms, mean peripheral detection RT was slower at the cued than at the uncued location. They noted that the early facilitation “is replaced by a clear inhibition of target RT on the cued side in comparison to the uncued side after about 300 ms” (p. 535). This empirical effect is generally known as “*inhibition of return*” (IOR; Posner, Rafal, Choate, & Vaughan, 1985; see Klein, 2000, for a review)¹.

Posner and Cohen (1984) describe how Cohen (1981) replicated this basic inhibitory or negative cueing effect in mean RT with a simpler method, in which a peripheral brightening first summons attention, which is then returned to fixation by a central brightening before peripheral target onset. By using an additional central cue display, shifts of attention can presumably occur with no manipulation of target presentation probability. Based on a series of experiments (double cueing, central arrow cueing, etc.), Posner and Cohen (1984) concluded that (a) orienting of attention is not a sufficient condition for the negative cueing effect, (b) the origin of IOR is sensory rather than attentional, (c) sensory IOR lasts for about 1 to 1.5 seconds, and (d) sensory IOR occurs quite automatically. There is now substantial evidence that this sensory IOR is due to an increase in the perceptual detection threshold for the target (Lupiáñez, Martín-Arevalo, & Chica, 2013; Osugi, Takeda, & Murakami, 2016; Schönhammer & Kerzel, 2017).

However, after more than 30 years of research there is still no consensus in the literature about *why* the sensory threshold is increased at the cued location, and about whether there exist multiple types of IOR, each relying on different mechanisms so that different terms should start to be introduced (Berlucchi, 2006; Dukewich & Klein, 2015; Lupiáñez, Klein, & Bartolomeo, 2006). The textbook explanation for the increase in the sensory detection threshold is that *attentional withdrawal* from the cue leaves an inhibitory tag so that spatial attention is “inhibited to return” to

that previously attended location, thereby facilitating visual search for a target in a scene based on memorizing each previously attended location during the search (Klein, 2000). However, it might instead (also) be due to *adaptation or habituation* of sensory cells to the cue (Posner & Cohen, 1984; Satel, Wang, Trappenberg, & Klein, 2011). Finally, Pratt, Spalek, and Bradshaw (1999) proposed that sensory IOR is caused by *attentional momentum*, i.e., it is easier to keep attention moving in the same direction – from the cued to central to uncued location – than to change direction – from the cued to center and back to cued location.

Other mechanisms have also been proposed (Godijn & Theeuwes, 2002; Lupiáñez & Milliken, 1999; Taylor & Klein, 2000). For example, according to the motor account of IOR (Taylor & Klein, 2000), the inhibition arises because the cue triggers an automatic orienting motor response which must be *actively and selectively inhibited* to avoid a premature response. Participants are then slower to execute that response when a target appears in the same location.

Currently, state-of-the-art theories claim that there are independent sensory and motor mechanisms underlying IOR (Abrams and Dobkin, 1994; Hunt & Kingstone, 2003; Satel & Wang, 2012; Taylor & Klein, 2000; Wang, Satel, & Klein, 2012). For example, in a large-scale study, Taylor and Klein (2000) presented peripheral onsets or central arrows as cues and/or targets. They manipulated the response required to the cue and target: no response-manual, manual-saccadic, saccadic-saccadic, etc., respectively. Based on the presence or absence of IOR for each of the 24 conditions, they concluded that there are two forms of IOR: manual IOR reflects a sensory or attentional bias when the eyes remain fixed, and oculomotor IOR reflects an inhibitory motor bias involving the superior colliculus when eye movements are executed. On the other hand, however, Wang, et al. (2012) concluded that for saccadic responses to the cue and target, sensory and motor components can be dissociated and that their effects on saccadic RT are additive. They proposed that oculomotor IOR is based on (a) automatic habituation due to sensory stimulation by the cue, as well as (b) a passive remapping of space in the superior colliculus. Due to this remapping,

saccades in the forward direction are automatically and temporarily facilitated, while those directed back to the vicinity of the previous fixation are impeded (*saccadic momentum*). The additivity does not carry over into the manual response modality, however, suggesting the operation of a potentially different motor mechanism with manual compared to saccadic responses (Satel & Wang, 2012). Note that all these proposed sensory, attentional, and motor mechanisms are not mutually exclusive in principle.

More generally, given the sensitivity of the IOR effect to various factors, and given the rather small effect sizes and the rather large variability between studies and even between participants within studies (see below), one might be tempted to think that the only way out of this theoretical labyrinth and to disentangle the potential effects of all these proposed mechanisms is to measure additional physiological information using EEG, fMRI, etc. However, it turns out that mean performance measures conceal a lot of useful information on behavioral dynamics that can be used to strongly constrain theories and computational models (Holden, Van Orden, & Turvey, 2009; VanRullen, 2011; VanRullen & Koch, 2003; van Zoest, Hunt, & Kingstone, 2010). By using the standard longitudinal technique for time-to-event data known as *Event History Analysis*² which takes into account the effect of an additional dimension that is always present – the passage of time –, one can provide an unbiased, dynamic and probabilistic description of the time-dispersed behavior of participants in *every* cognitive paradigm (Panis & Hermens, 2014; Panis, Moran, Wolkersdorfer, Schmidt, 2020; Panis & Schmidt, 2016; Panis, Torfs, Gillebert, Wagemans, & Humphreys, 2017; Panis & Wagemans, 2009; Wolkersdorfer, Panis, & Schmidt, 2020). In other words, one can study the within-trial onset time of an effect, its duration, its shape, and how all these features might change as time passes by on longer time scales, for example due to across-trial and across-block learning effects.

Furthermore, once behavior is described at the macroscopic level as a function of different time scales it is likely that one can better (a) constrain theory development, (b) develop

microscopic descriptions such as computational models (Kelso, Dumas, & Tognoli, 2013), and (c) link the behavioral effects to the ongoing underlying physiological changes (Meyer, Osman, Irwin, & Yantis, 1988), as illustrated below. For example, reactive cognitive control processes such as selective response inhibition operate on the within-trial time scale (Panis & Schmidt, 2016; Ridderinkhof, 2002), while learning processes such as habituation, and proactive control processes operate on longer time scales (Braver, 2012). Finally, as discussed by Whelan (2008) the use of a more advanced analysis method can maximize the return from the collected data, which is important in view of the costs and time required to run an experiment.

Current study

The current study is motivated by three goals. First, we want to correctly characterize the *within-trial time course* of IOR because there are more and more indications that comparing mean RTs as a function of SOA causes misinterpretations about the time course of an effect (Panis & Schmidt, 2016; VanRullen, 2011; Wolkersdorfer, et al., 2020). For example, while the size of the human IOR effect in mean (correct) RT is typically about 30 ms, single-cell studies have revealed that the within-trial duration of neural IOR is actually about 100 ms (Fecteau, Bell, & Munoz, 2004). As shown in Figure 1, three neural stages can be identified in the shape of the time-varying firing rate of a monkey superior colliculus neuron: a cue-triggered response in valid trials (blue line), a delay stage where activity is reduced but non-zero (black to white vertical bars), and finally a target-locked stage (vertical grey bar) where the response is much stronger in invalid (red line) compared to valid (blue line) trials. Fecteau et al. (2004) concluded that the smaller target-locked response in the valid trials reflects reduced input to the superior colliculus, consistent with *sensory inhibition* or *habituation* of that location in “upstream” areas.

Is the same qualitative information present in the time-dispersed behavior of human participants? To answer this question we analyse the shape of RT distributions using discrete-time

hazard functions of response occurrence (Allison, 1982, 2010; Austin, 2017; Singer & Willett, 1991, 2003; Willett & Singer, 1993), and the shape of accuracy distributions using discrete-time conditional accuracy functions, a.k.a. micro-level speed-accuracy tradeoff functions (Pachella, 1974; van Maanen, Katsimpokis, & van Campen, 2019; Wickelgren, 1977). In two experiments we characterize the within-trial time course of the “sensory IOR” effect using the paradigm of Posner and Cohen (1984), which corresponds to the “ignore-manual” condition of Taylor and Klein (2000).

Second, what is the effect of an additional central cue presented after the peripheral cue and before target onset? According to Posner and Cohen (1984) it causes attention to return to the center after being summoned to the cued location. However, it might instead – or also – act as a contextual signal that any (here: peripheral cue-triggered) response activation is still premature and therefore activate reactive cognitive control processes (Panis & Schmidt, 2016).

Third, does the time course of IOR change depending on the task (Tipper, Weaver, & Houghton, 1994)? To answer these questions we employ a one-button detection task in Experiment 1 and manipulate cue validity and cue-target ISI. We also include a „no cue“ condition to study the effect of cue presence, and a „central cue only“ condition to study how the alerting effect of a cue might depend on the task-relevance of its location. In Experiment 2 we employ a two-button localization task and manipulate the presence of an additional central cue, next to peripheral cue validity (valid, invalid, no peripheral cue) and cue-target ISIs. The localization task allows us to also analyse the accuracy data on top of the response time data. We do not use three boxes or placeholders to avoid any object-based effects (Chen, 2012; Jordan & Tipper, 1998; Lamy & Egeth, 2002; Tipper, 1985), and the target can appear only to the left or right of fixation.

Remarkably, our results show that time-dispersed human behavior portrays the same qualitative features as the monkey single-cell data in Figure 1. Furthermore, the within-trial time course of IOR in the hazard functions (onset time, shape, and duration) is not affected much by the task. The additional central cue unexpectedly triggers localization response activation and causes a

hitherto unknown temporary negative cueing effect in the conditional accuracy distributions. We discuss how our longitudinal descriptions of behavior in both spatial cueing tasks, together with recent computational modeling studies of change detection behavior, point more clearly to a single underlying cause of sensory IOR, independent of manual motor IOR.

Experiment 1

In Experiment 1 a 50 ms cue display (left, right, central, absent) was followed by a 100 ms target display separated by one of six ISI levels (50, 150, 250, 350, or 450 ms and target presented, 250 ms and target not presented). Participants had to press one button within 600 ms whenever they detected the target. Importantly, to capture responses that occur before target display onset we started the response timer at trial onset.

Methods

Participants

Six master students (3 males, 1 left-handed) participated for course credits. They had normal or corrected-to-normal vision. Their average age was 24.3 years (range: 21-26). Each participant gave informed consent and was treated in accordance with the ethical standards of the American Psychological Association.

Apparatus and software

Stimuli were presented on a Samtron 96P monitor (36.5 x 27.5 cm) connected to a Tarox desktop computer in a darkened room. The screen refresh rate was 85 Hz. A response time box with a built-in microprocessor was used to record the response time and button identity independently from the Windows operating system (Suzhou Litong Electronic CO., LTD; <https://lobes.osu.edu/rtbox.php>). PsychToolbox (<http://psychtoolbox.org/>) and Matlab

(www.mathworks.com) were used to generate the stimuli, control the timing of stimulus presentation, read-out the response box, and save the data set.

Stimuli

A small black fixation cross (.25 x .25 visual degrees) was presented centrally on a white background (Figure 2). The black cue consisted of the outline of a square (3 x 3 visual degrees) with a line thickness of .25 visual degrees, presented 8 degrees to the left or right of the vertical midline or on the vertical midline. The target was the black letter X (1 x 1 visual degrees) presented 8 degrees to the left or right.

Experimental design

Forty-eight trial types were created by factorially combining the variables Cue Type (valid, invalid, central, absent), Target Location (left, right), and cue-target ISI (6 levels: 50, 150, 250, 350, or 450 ms and target presented, 250 ms and target not presented). Forty trials were designed for each trial type. Collapsing across target location gives 24 within-subject conditions and 80 trials per condition.

Procedure

Each individual participated in a single session of about 90 minutes and performed a practice block followed by 80 experimental blocks of 24 trials each. All trial types were presented once more after every two consecutive blocks. The order of trials was randomized within each block.

As shown in Figure 2 each trial began with a fixation display presented for ~494 ms (42 frames). Next the cue display was presented for ~47 ms (4 frames) followed by the ISI display presented for either ~47, ~141, ~247, ~341, or ~447 ms (4, 12, 21, 29, 38 frames). The target display was presented for ~94 ms (8 frames). If the target was presented it could appear to the left or right of fixation. The presence and location of the cue in the cue display depended on Cue Type

and Target Location.

Participants were seated with their midline aligned with the center of the response box and the screen. They were instructed to gaze at the fixation cross whenever it was visible, to ignore the square outline, and to respond quickly (within 600 ms) in each trial to the onset of the target by pressing the response box button with the index finger of their dominant hand, or to withhold responding when no target was presented. In each trial the data collection period started at -520 ms from target display onset – just before cue display onset in the longest ISI condition – and ended at 600 ms after target display onset. The latency of the first emitted response – if any – was recorded in ms relative to target display onset. In other words, the response timer started shortly after trial onset so that we could also time the responses that occur before target display onset, which will have a negative RT in the data set. Note that we can expect some right-censored trials even when the target is presented, i.e., trials without a response before 600 ms. Importantly, such trials are taken into account when we describe and model the shape of the RT distributions (see below).

Feedback was provided after the end of the data collection period for 300 ms, followed by a 300 ms blank screen. If a response was detected, “correct” was displayed if the target was present and “error” if the target was absent. If no response was detected, “too slow” was displayed if the target was present and “correct” if the target was absent. The next trial started automatically after the blank screen.

After each block, summary information was provided including the number of correct and error responses, and the mean RT of all hits. Participants could then take a small break and start the next block by pressing the response button. Before and after the practice block, participants were told to monitor the global decrease in mean hit RT across blocks for motivational purposes, to always try to avoid any „too slow“ messages, and not to worry at all if they happen to make some errors per block as this is to be expected due to the response deadline time of 600 ms. After

obtaining informed consent and delivering the instructions, the experimenter performed one block of trials himself to show the participant how fast the experimental trials are running, and how frequently they have to respond. After monitoring the participant's performance in the practice block – all participants immediately understood the task and performed satisfactorily – and answering any remaining questions, the experimenter left the room.

Statistical analyses

First, we analysed the mean RT data and the cueing effects using repeated-measures ANOVAs. The Greenhouse-Geisser correction was applied if Mauchly's test for sphericity was significant. All significant effects are reported.

Second, we calculated the descriptive statistics provided by discrete-time event history analysis for each combination of participant and condition, and for each condition after aggregating the data across participants, in R using life tables (R Core Team, 2014; see Table 1). The hazard function of response occurrence is one of the most diagnostic functions when describing the distribution of a sample of (right-censored) time-to-event data (Allison, 2010; Luce, 1986; Townsend, 1990)³. The discrete-time hazard function, $h(t) = P(T = t | T \geq t)$, represents for each time bin t the conditional probability that a response will occur in bin t given that it has not yet occurred in any of the past bins (Allison, 2010). It is estimated by dividing the number of responses in bin t by the risk set for bin t , i.e., the number of trials that are still response-free at the start of bin t .

Because all response times must be positive for a survival analysis, we added 520 ms to each original RT datum in the data set in order to capture many „negative“ RTs as well (and then the remaining trials with an RT at or below zero were discarded). We used bins of 40 ms except for the last 200 ms of the response collection period. To cover this stretch of time where few responses occur, we used four bins of 50 ms to minimize the instability of the estimates⁴.

When we plot the bin-specific differences in hazard between two conditions – $\Delta h(t)$

plots – we also plot the approximate 95% confidence interval for the difference between two proportions, based on the estimated standard error: $se[h_1(t) - h_2(t)] = \text{square root of } [\{h_1(t) * [1 - h_1(t)] / RS_1(t)\} + \{h_2(t) * [1 - h_2(t)] / RS_2(t)\}]$, where RS refers to the size of the risk set.

A second useful function is the survivor function or $S(t) = P(T > t) = [1 - h(t)] * [1 - h(t-1)] * ... * [1 - h(1)]$. The survivor function is the complement of the cumulative distribution function, or $1 - F(t) = 1 - P(T \leq t)$, and gives for each bin the probability that the response does not occur before the end of bin t . The estimated median RT – the time point when half of the trials have experienced a response – equals quantile $S(t)_{.50}$ and can be obtained using linear interpolation.

Third, to test *whether and when* the main and interaction effects including ISI and cue type are significant across participants, we fit discrete-time hazard models to the data. An example discrete-time hazard model with three predictors and the *complementary log-log* (cloglog) link function can be written as follows⁵:

$$\begin{aligned} \text{cloglog}[h(t)] = \ln(-\ln[1-h(t)]) = & [\alpha_0 \text{ONE} + \alpha_1(\text{TIME} - 1) + \alpha_2(\text{TIME} - 1)^2 + \alpha_3(\text{TIME} - 1)^3] \\ & + [\beta_1 X_1 + \beta_2 X_2 + \beta_3 X_2(\text{TIME} - 1)]. \end{aligned}$$

The main continuous predictor variable TIME is the time bin index t (see Table 1) which is centered on value 1 in this example. The first set of terms within brackets, the alpha parameters multiplied by their polynomial specifications of (centered) time, represents the shape of the cloglog-hazard function in the chosen baseline condition (i.e., when all predictors X_i take on a value of zero). The second set of terms (the beta parameters) represents the vertical shift in the baseline cloglog-hazard for a 1 unit increase in the respective predictor. For example, the effect of a 1 unit increase in X_1 is to vertically shift the whole baseline cloglog-hazard function with β_1 cloglog-hazard units. However, if the predictor interacts linearly with time (see X_2 in the example) then the effect of a 1 unit increase in X_2 is to vertically shift the predicted cloglog-hazard in bin 1 with β_2 cloglog-hazard units (when $\text{TIME} - 1 = 0$), in bin 2 with $\beta_2 + \beta_3$ cloglog-hazard units (when $\text{TIME} - 1 = 1$), etc. To interpret the effect of each predictor, the parameter estimate is exponentiated, resulting in a

hazard ratio (HR). In the case of a small- N design with repeated measurements the parameters of a discrete-time hazard model can be estimated using population-averaged methods (e.g., Generalized Estimating Equations), Bayesian methods, or generalized linear mixed models (Allison, 2010).

We proceeded as follows for the current IOR data set. First, we chose to fit hazard models by implementing generalized linear mixed-effects regression models in R (R Core Team, 2014; function `glmer()` of package `lme4`, Bates, Mächler, Bolker, & Walker, 2015) using the `cloglog` link function. To focus on the IOR effect we deleted all trials where the SOA did *not* equal 350 or 450 ms. We selected a time range where all participants provided enough data in each of the remaining eight conditions, and followed the general advise to create between 10 and 20 bins for modeling purposes (Singer & Willett, 2003). Thus, we selected the time range (160,600], starting with 6 bins of 40 ms and ending with 4 bins of 50 ms (see Table 1).

Second, the condition “350 ms ISI and central cue” was chosen as the baseline condition. The main continuous predictor variable TIME was the time bin rank centered on value 20 or bin (240,280], the reference bin. The intercept, the linear effect of TIME, and their correlation were treated as random effects to deal with the correlated data resulting from the repeated measures on the same participant⁶. Next to dummy-coding the relevant levels of our experimental factors (VALid, INvalid, No Cue, ISI_450) we also included BLOCK number as a continuous predictor to model across-block learning effects in the hazard of response occurrence. BLOCK was centered on value 25 and rescaled by dividing by 10. Thus, with all effects set to zero, the model’s intercept refers to the estimated $\text{cloglog}[h(t)]$ for bin (240,280] in block 25 when the central cue precedes target onset with an ISI of 350 ms (see Table 2, column 5, effect nr. 1).

Third, to estimate the parameters of a cloglog-hazard model, we must create a dataset where each row corresponds to a time bin of a trial of a participant (a person-trial-bin oriented data set). Specifically, each *time bin* that was at risk for event occurrence in a trial was scored on

the dependent variable OUTCOME (0 = no response occurred; 1 = response occurred), the covariates TIME and BLOCK, the variable PARTICIPANT, and the dummy-coded dichotomous experimental predictor variables (VAlid, INvalid, No Cue, ISI_450). Thus, all trials without an observed response were treated as right-censored observations (i.e., each of these trials contributes 10 rows, and each row has a value 0 for OUTCOME). The resulting person-trial-bin oriented data set contained 13,713 rows.

Fourth, we started with a full multilevel cloglog-hazard model (45 parameters; with bins at level 1 nested within participant at level 2) encompassing the following effects at level 1: (a) a 3rd order polynomial for the shape of the cloglog-hazard function in the baseline condition (4 parameters), (b) the effects of valid cueing ('VA'), invalid cueing ('IN'), removing the cue ('NC'), and changing the ISI from 350 to 450 ms ('ISI_450') were allowed to interact with time in a cubic fashion (16 parameters), (c) the three first-order interaction effects involving one cue type level and ISI_450 could vary over time in a quadratic fashion (9 parameters), (d) the linear and quadratic effects of BLOCK could interact with time in a quadratic fashion (6 parameters), (e) to study whether the facilitation effect in the invalid vs. central cueing conditions, and the negative cueing effect in the valid vs. central cueing conditions change in size over the time course of the experiment, and perhaps differently for both selected ISIs, we also included the interactions VA:BLOCK and IN:BLOCK that could vary linearly over time (4 parameters), the interactions VA:ISI_450:BLOCK and IN:ISI_450:BLOCK that could vary linearly over time (4 parameters), and the interaction ISI_450:BLOCK that could vary linearly over time (2 parameters).

We used an automatic backward selection procedure to select a final model to report. Specifically, during each iteration, the effect with the largest p -value that was not part of any higher-order effect was deleted, and the model refitted. This continued until each of the remaining effects that was not part of any higher-order effect had a $p < .05$.

Finally, after model selection, we refitted the selected cloglog-hazard model a number of

times with TIME centered each time on a different reference bin, to make explicit what values the parameter estimates of the effects not involving TIME take on according to the selected model in these other time bins, and whether they represent a significant effect or not.

Results

We measured all the 1,920 trials (24 conditions x 80 trials) for four participants but could measure only 1,248 and 1,839 trials for the other two participants due to technical errors.

Mean performance measure

First, the mean RT was calculated for each combination of participant and target-present condition using all observed response times between 150 and 600 ms (Figure 3A). A two-way repeated-measures ANOVA revealed significant main effects of Cue Type ($F(3,15) = 51.82, p < .001, \eta^2 = .91$) and ISI ($F(4,20) = 11.27, p < .001, \eta^2 = .69$), next to a significant interaction effect ($F(12,60) = 4.71, p < .001, \eta^2 = .49$). Next, the cueing effect (invalid minus valid) was calculated for each ISI (Figure 3B). A one-way repeated-measures ANOVA showed a significant effect of ISI ($F(2.044,10.219) = 8.94, p = .006, \eta^2 = .64$). Bonferroni-corrected pairwise comparisons showed that the cueing effect was more negative for ISE 450 compared to ISI 150 ($t = 3.85, p = .01$) and ISI 50 ($t = 5.57, p < .001$), and more negative for ISI 350 compared to ISI 50 ($t = 3.89, p = .009$). We thus observe that the inhibitory cueing effect in mean RT is clearly present for ISIs 350 and 450 ms, consistent with the findings of Posner and Cohen (1984).

Note the large variability between individual participants in the shape of the function relating the cueing effect in mean RT to the cue-target ISI (Figure 3B). For example, participant 6 seems to show IOR for each ISI, and participant 1 perhaps only for the longest. Indeed, Figure 3B is very misleading when interpreted in terms of the time course of IOR, as soon will be revealed. For example, based on Figure 3B (with SOA on the horizontal axis) one might conclude – and many

have – that IOR emerges sometime around 300 ms *after cue onset* and is also present around 450 ms *after cue onset*. Similarly, a significant difference between two mean RTs does not necessarily mean that people are starting to respond earlier in the fast compared to the slow condition (Panis & Schmidt, 2016). As explained by VanRullen (2011), such interpretations should always be checked with a distributional analysis, and preferably a hazard analysis (Luce, 1986; Townsend, 1990).

Event history analysis: descriptive statistics

Figure 4 presents the sample-based estimates of the hazard and survivor functions and the median RTs for each of the 24 conditions after aggregating the data across the six participants. Focusing also on the aggregated data is justified here because the three visible main data patterns are present in all participants (see below). First, the onset of the RT distribution is time-locked to cue onset because the first responses appear around 250 ms after cue *onset*, for each ISI in the cue-present conditions. Now, imagine traveling with time starting at trial onset. Then, for example, if the waiting time has increased until -280 ms without event occurrence when the ISI is 450 ms and the cue invalid, then the conditional probability that the response is about to occur in bin (-280, -240] is estimated to be 0.043 (19 observed responses with a risk set equal to 439; Table 1). Thus, of all trials that survive until (i.e., are still response-free at) -280 ms, about 5 percent will experience a response during the next 40 ms (i.e., they will „die“, and drop out of the risk set). In short, $h(-240) = .043$. Note that from now on, we use the endpoint to refer to each bin when reporting and plotting estimates.

Second, soon after these first cue-triggered responses hazard decreases to a non-zero value during the delay period between cue and target. For example, if the waiting time has increased until 0 ms – when the target appears on the screen – without event occurrence in condition invalid/450, then the hazard probability that the response is about to occur in bin (0,40] or $h(40)$ is

estimated to be 0.019 (7 observed responses with a risk set equal to 375). This decrease might be due to selective response inhibition of the cue-triggered response. We come back to this issue after Experiment 2. The fact that hazard does not drop to zero suggests an active working memory representation of the cue location that survives the cue-target interval. We come back to this issue in the Discussion section of Experiment 1.

Third, around 200 ms after target onset all the hazard functions in the target-present conditions sharply rise toward a value of around 0.40, before leveling off. Crucially, IOR emerges around the same time in the hazard functions when cue-target ISI exceeds ~150 ms, with larger hazard estimates for the invalid compared to valid cueing condition. For example, if the waiting time has increased until +200 ms in condition invalid/450, then $h(240)$ is estimated to be 0.259, 0.185, and 0.084, for the invalid, central, and valid cueing conditions, respectively. Thus, for relatively long ISIs and relative to central cueing, there is a temporally localized *facilitation effect* with invalid cues and an *inhibitory effect* with valid cues. The total (valid minus invalid) cueing effect – IOR – represents a factor of more than 3 in terms of hazard of detection response occurrence in bin (200,240]⁷. Furthermore, the within-trial duration of IOR seems to increase somewhat with increasing ISI.

Finally, when the target is absent, the RT distribution in the central cue condition peaks in an earlier time bin compared to both conditions where the cue appears at one of the possible target locations (this feature is present in 4 participants). These three cue-triggered, target-absent response hazard distributions each cover a range of about 240 ms. Figures 5 and 6 each present the descriptive statistics of a single participant to illustrate that all these data patterns are present at the individual level. Interestingly, even though participant 4 frequently emits responses when no stimulus is presented, the main features are present (Figure 6).

To better focus on the within-trial time course of IOR, Figure 7 presents the cueing effect (valid minus invalid) in the time-varying hazard estimates for each participant, as a function of ISI.

Note that *all* participants show a clear IOR effect for the larger cue-target ISIs sometime between 160 and 280 ms after target onset (shaded area is shifted by +20 ms), except participant 6 whose IOR effect occurs just outside the shaded area. Figure 8A presents the same delta-hazard plots (valid minus invalid) for the aggregated data but after excluding participant 6. Based on the 95% confidence intervals, we observe for the first time that IOR is present (a) in bin (160,200] for ISIs above 50 ms, (b) in bin (200,240] for ISIs above 150 ms, and (c) in bin (240,280] for both ISIs above 250 ms. To better focus on the within-trial time course of the inhibitory component, Figure 8B compares the hazard estimates of the valid and central cueing conditions (valid minus central). We observe that (a) the inhibitory component reaches the same maximal value in bin (200,240] for ISIs 250, 350, and 450 ms, (b) is still present in bin (240,280] for ISIs 350 and 450 ms, and (c) is perhaps still present in bin (280,320] for an ISI of 450 ms. Also, notice the short-lived positive cueing effect in bin (160,200] for an ISI of 50 ms.

Event history analysis: inferential statistics

Because we want to focus on how the facilitatory and inhibitory components of the IOR effect change in size with the passage of time within a trial, and possibly with the passage of time across blocks due to medium-term habituation, we developed a statistical hazard model for the detection latency data for which the ISI equals either 350 or 450 ms. We focused on the time range 160-600 ms after target onset. We implemented a generalized linear mixed regression model in R including the continuous predictors TIME (time bin rank, centered on the reference bin (240,280]) and BLOCK number (centered on block number 25), and the dummy-coded levels of cue type (baseline = central) and the selected ISIs (baseline = 350), together with all relevant interactions (see *Methods, Statistical analyses*).

Table 2 presents the selected hazard model for Experiment 1, and Figure 9 the model-based (cloglog-)hazard functions as a function of both ISIs, 4 cue types, and three block numbers.

Because BLOCK is centered, the first four parameter estimates (PE) in Table 2 model the shape of the $\text{cloglog}[h(t)]$ function for the baseline condition (central cue and 350 ms ISI) in block 25 using a 3rd order polynomial function of TIME (Figure 9C, thick blue line). Because TIME is centered on the reference bin (240,280] during model selection, the estimated intercept of the selected hazard model refers to the predicted $\text{cloglog}[h(280)]$ value for the baseline condition in block 25. Converting back from cloglogs to hazards, $h(280) = .37 (= 1 - \exp[-\exp(-0.78)])$ as shown in Figure 9E (thick line). Parameters 2-4 show significant linear, quadratic, and cubic effects of TIME on this intercept estimate, such that the predicted response hazard increases and then decreases over time: $h(200) = 0.082$, $h(280) = 0.37$, $h(360) = 0.44$, and $h(450) = 0.33$. This shows that the hazard of response occurrence changes in a particular fashion on the across-bin/within-trial time scale for the baseline condition in block 25.

With respect to the manipulations of interest, we see that relative to the baseline condition, there is a main effect of valid cueing (parameter 5, reference bin PE = -0.5103, $p < .0001$). A measure of effect size for a discrete-time cloglog-hazard model can be obtained by exponentiating the parameter estimates which give us hazard ratios (HR; Allison, 2010, p. 242). Thus, compared to the $\text{cloglog}[h(280)]$ estimate in the baseline condition, a valid cue decreases the estimated $\text{cloglog}[h(280)]$ by 0.51 cloglog-hazard units, which corresponds to a decrease in response hazard by a factor of 0.6 ($HR(280) = \exp[-0.5103] = 0.6$). This main effect of valid cueing changes over time in a linear, quadratic, and cubic fashion (parameters 6-8), resulting in $HR(200) = 0.41$ ($p < .001$), $HR(280) = 0.6$ ($p < .0001$), $HR(360) = 0.95$ ($p = .53$), and $HR(450) = 1.15$ ($p = .23$; compare green and blue lines in Figure 9E). Thus, the negative effect of valid cueing on response cloglog-hazard is strongly present in bin (160,200], decreases non-linearly over the next 160 ms of within-trial time, and is gone around 360 ms after target onset in block 25 when the cue-target ISI equals 350 ms (Figure 9C).

The main effect of invalid cueing is also significant (parameter 9, reference bin PE = 0.1763,

$p = .008$) and changes linearly and quadratically over time (parameters 10 and 11): $HR(200) = 1.8$ ($p < .0001$), $HR(280) = 1.2$ ($p = .008$), $HR(360) = .9$ ($p = .25$), $HR(450) = .8$ ($p = .043$). Thus, the positive effect of invalid cueing on response cloglog-hazard is strongly present in bin (160,200], decreases non-linearly over the next 160 ms within-trial time, and is gone around 360 ms after target onset in block 25 when the cue-target ISI equals 350 ms.

The main effect of not cueing is significant relative to the baseline condition (parameter 12, reference bin $PE = -1.01$, $p < .0001$) and changes over time (parameters 13 and 14): $HR(200) = 0.15$ ($p < .0001$), $HR(280) = 0.36$ ($p < .0001$), $HR(360) = .7$ ($p < .0001$), $HR(450) = 1.008$ ($p = .95$). The alerting effect of cueing is thus gone around 450 ms after target onset. In other words, when the waiting time has increased to 450 ms after target onset without response occurrence, then the response hazard is not affected anymore by whether a (central) cue was presented or not.

The main effect of changing the ISI from 350 to 450 ms is significant (parameter 15, reference bin $PE = -0.3123$, $p < .0001$) and changes linearly over time (parameter 16): $HR(200) = 0.15$ ($p < .0001$), $HR(280) = 0.36$ ($p < .0001$), $HR(360) = .7$ ($p < .0001$), $HR(450) = 1.008$ ($p = .95$). People thus react slower for ISI 450 but only for $t < 450$ ms.

The interaction effect between ISI 450 and NC is significant (parameter 17, reference bin $PE = 0.2939$, $p < .0001$) and decreases linearly over time (parameter 18). Note that this interaction effect cancels the time-varying effect of ISI 450, as there can be no effect of ISI when no cue is presented (compare the absolute size of the parameter estimates of effects 15 and 17, separately for each bin).

One advantage of a discrete-time hazard model is that we can incorporate multiple time scales. The selected model shows that there are significant linear and quadratic effects of BLOCK number in the reference bin (parameters 19 and 21), that each change linearly over time (parameters 20 and 22). Increasing block number leads to a gradual increase of hazard in early bins, but a gradual decrease of hazard in later bins (Figure 9).

Finally, the time-invariant interaction effect IN:BLOCK was significant (parameter 23, PE = -0.0044, $p = .028$). As can be seen in Figure 9, due to this interaction effect the within-trial duration and size of the early facilitation effect in hazard for invalid compared to central cueing decreases during the passage of time on the across-block time scale. Equally interesting, all the interaction effects involving VA:BLOCK left the cloglog-hazard model during the selection process.

Discussion

To study the within-trial time course of IOR in an ignore-manual detection task (Posner & Cohen, 1984; Taylor & Klein, 2000) we employed descriptive and inferential discrete-time hazard analyses (Allison, 2010). In contrast to the mean performance measures, we observe that IOR in hazard exists in most participants for some time between 160 and 280 ms after target onset when the cue-target SOA exceeds ~200 ms (Figure 8A). Importantly, the time-dispersed human behavior (Figure 4) qualitatively matches single-cell data (Fecteau, et al., 2004; Figure 1), and the observed onset time of IOR in the hazard functions is very close to that observed with EEG data (Pierce, Crouse, & Green, 2017).

The fact that the inhibitory component of the IOR effect (valid versus central cueing) does not interact with BLOCK suggests that medium-term habituation or adaptation processes cannot (fully) explain it – future studies might also want to control for the passage of time on the across-trial/within-block time scale to study any role for short-term sensory adaptation. Also, the sequential waves of bottom-up sensorimotor responses to the cue and target – the underlying stimulus-triggered neural feedforward sweeps (Alilović, Timmermans, Reteig, van Gaal, & Slagter, 2018; Lamme & Roelfsema, 2000; Schmidt, Niehaus, & Nagel, 2006; VanRullen & Koch, 2003) – make any explanation in terms of cue-target sensory integration processes unlikely (Lupíáñez & Milliken, 1999).

We did not find a facilitation effect in mean RT at the shortest cue-target ISI of 50 ms

(Figure 3). Reassuringly, Maruff, Yucel, Danckert, Stuart, and Currie (1999) concluded that facilitation in mean RT with short cue-target SOAs arises only when the cue and target temporally overlap, which was not the case here. However, when we descriptively compare hazard for valid with central cueing we see evidence for a short-lived facilitation effect for the shortest ISI (Figure 8B). This might be due to the fact that there is still some neural overlap despite the absence of physical overlap, and/or to the fact that the salient cue onset does attract attention temporarily; Whether attention would then shift quickly back to the central fixation cross after peripheral cue disappearance, keeps lingering at the cued location, or keeps sampling both target locations sequentially (Zoefel & VanRullen, 2017) is currently still unclear (see Experiment 2, however).

What is causing the IOR effect observed here? Several findings point to a direct role for spatial working memory. First, Castel, Pratt, and Craik (2003) introduced a secondary task between the cue and target, and found that IOR was eliminated only when the secondary task involved a spatial working memory load. Second, the non-zero hazard estimates during the cue-target interval (Figures 4, 5, and 6) suggest that a working memory representation of the cue location is kept active. Third, a dynamic field model of location change detection behavior shows that “same/no change” responses are slower than “change/different” responses due to a decrease in the baseline level of neural population activity in sensory areas at locations stored in spatial working memory (Figure 10A; Johnson, Spencer, Luck, & Schöner, 2009). Note that the uninformative spatial cueing paradigm can be regarded as a location change detection task with only one object in each consecutive scene (i.e., valid cueing would require a “same” response, and invalid cueing a “different” response).

Thus, as soon as the cue location is detected in retinotopically organized perceptual areas in the dorsal stream, it will also be encoded in allocentric spatial working memory (WM) – via an automatic and online reference frame transformation process (see chapter 7 in Schöner, Spencer, et al., 2016). As soon as the active memory forms – and as long as it exists – it will automatically

provide an inhibitory bias at the corresponding location in sensory areas. This sensory bias thus reflects the existence of an active WM representation and will lead to slower detection RT when the same spatiotopic location is stimulated after an eye-movement because no change has occurred, compared to when a location is suddenly and unexpectedly stimulated due to an external change in position. This general principle causes salient changes across eye-movements to be preferentially processed by the brain (Schöner, Spencer, et al., 2016). This would explain why multiple cued locations show IOR, why IOR is coded in spatiotopic coordinates, why IOR is quite fast and automatic, why it can still be measured with a cue-target SOA of multiple seconds (Posner & Cohen, 1984).

If this proposal is correct, then sensory IOR should be influencing the shape of RT distributions in many – if not all – paradigms that involve storing information in WM and performing a WM-relevant task at a later time point (MacLeod, 2007; Noreen & MacLeod, 2015; Tipper, 1985; Tipper & Cranston, 1985). Furthermore, IOR should not occur when attention withdraws from a location *not* stored in WM. Also, IOR should show drifts in space over time due to the existence of repulsion effects between items stored in WM (Schöner, Spencer, et al., 2016), and IOR can exist not only for space dimensions but also for feature dimensions in discrimination tasks (Lupiañez, Milán, Tornay, Madrid, & Tudela, 1997).

The idea of an inhibitory sensory bias caused by an active WM representation can explain both the presence of the facilitatory and inhibitory components, and why only the facilitation effect decreases over the course of the experiment. In Figure 10B we assume that the sensory bias is broad and deep at the start of the experiment, reflecting positional uncertainty across trials – due to the short cue duration, an initial broad WM peak, etc. After cue offset, and before target onset at position -50, the neural population activity value at position -50 is smallest when the cue has appeared at position -50 (valid, green striped curve), intermediate for central cueing at position 0 (blue striped curve), and highest when the cue has appeared at position 50 (invalid, red

striped curve). When a left target activates this sensory field at position -50, and relative to the central cueing condition, it will then take longer for the population activity at position -50 to grow and reach the output threshold at ordinate value 0 for valid cueing (visible as temporary behavioral inhibition) and less time for invalid cueing (visible as temporary behavioral facilitation). If we also assume that synaptic learning makes the bias less and less wide and deep due to a more and more precise WM representation among other factors (Schöner, Spencer, et al., 2016; Schutte & Spencer, 2009), then only the inhibitory effect in the valid relative to central cueing condition is still present at position -50 after learning (full curves in Figure 10B). Future computational modeling studies will have to test these ideas quantitatively and to check if the shapes of the $\Delta h(t)$ curves in Figure 8 can be simulated.

The superior colliculus data of Fecteau et al. (2004) in Figure 1 might thus reflect a top-down WM-triggered sensory bias due to an active WM representation. Note that we observe the general alerting effect of the cue in the mean RTs (Figure 3A and footnote 7). The fact that mean RT increases for an ISI of 450 ms compared to 350 ms (see parameter 15 in Table 2) might be due to the fact that the system assumes it is in a target-absent trial around 450 ms after cue *onset* in condition ISI 450 and starts increasing the global response threshold to prevent further overt response occurrence (Wiecki & Frank, 2013), because it is not expecting the upcoming target anymore. Experiment 2 will reveal that selective response inhibition processes can also play a role even though the eyes do not move, meaning that the cue stimuli do much more than just summoning attention.

Experiment 2

If sensory IOR does not simply arise because of the withdrawal of attention from the cued location (Maruff et al., 1999), then what is the role of the additional central cue display introduced by Cohen (1981) as reported by Posner and Cohen (1984)? And why does its presence tends to increase the size of the IOR effect in mean RT?

In Experiment 2 we switch to a two-button target localization task and manipulate the presence of an additional central cue presented between peripheral cue offset and target onset. A 50 ms peripheral cue display (valid, invalid, no peripheral cue) was followed by a 50 ms central cue display (present/absent) after a peripheral-central cue ISI (47 or 141 ms), and finally a 100 ms target display was presented (the letter X on the left or right) after some central-target ISI (47 or 141 ms). Participants had to indicate the peripheral target location by pressing one of two buttons within 400 ms.

Based on Experiment 1 we can again expect cue-triggered responses that will now all be correct in the valid cueing condition but incorrect in the invalid cueing condition, because the cue acts like a prime in a response priming experiment (Panis & Schmidt, 2016). Then at some point bottom-up target signals will take over (Schmidt et al., 2006). According to the traditional attentional interpretation, IOR in mean (correct) RT is increased with an additional central cue display because attention is switched from the cued to the central location before target onset more frequently with than without a central cue. In other words, it is assumed that IOR – presumably reflecting an inhibitory tag left by the withdrawal of attention during search – is attenuated when the central cue is absent due to more frequent lingering of attention at the peripherally cued location. Is this assumption correct?

Methods

Participants

Six naive master students (2 males, 0 left-handed) participated for course credits. They had normal or corrected-to-normal vision. Their average age was 24.8 years (range: 22-28). Each participant gave informed consent and was treated in accordance with the ethical standards of the American Psychological Association.

Apparatus and software

Same as in Experiment 1.

Stimuli

A small black fixation cross (.25 x .25 visual degrees) was presented centrally on a medium grey background (Figure 11). The stimuli appeared on a virtual horizontal line 1 visual degree above the central fixation cross. The black cue consisted of the outline of a square (1 x 1 visual degrees) with a line thickness of .25 visual degrees, presented 8 degrees to the left or right of the vertical midline or on the vertical midline. The target was the black letter X (.5 x .5 visual degrees) presented 8 degrees to the left or right.

Design

Forty-eight trial types were created by factorially combining the variables Cue Type (valid, invalid, absent), Target Location (left, right), Central Cue Presence (present, absent), and ISI1/ISI2 Combination (47/47, 47/141, 141/47, 141/141). Fifty trials were designed for each trial type. Collapsing across target location gives twenty-four within-subject conditions and 100 trials per condition. Note that when the central cue is absent, the conditions 141/47 and 47/141 are indistinguishable for the participants, and should show similar behavior.

Procedure

Each individual participated in a single session of about 90 minutes and performed a practice block followed by 50 experimental blocks of 48 trials each. All trial types were presented once in each block. The order of trials was randomized within each block.

Each trial began with a fixation display presented for ~494 ms (42 frames). As shown in Figure 11, next the peripheral cue display was presented for ~47 ms (4 frames) followed by the first ISI-display presented for either ~47 or ~141 ms (4 or 12 frames). Next the central cue display was presented for ~47 ms (4 frames) followed by the second ISI-display presented for either ~47 or ~141 ms (4 or 12 frames). The target display was presented for ~94 ms (8 frames). The target appeared always to the left or right of fixation. The presence and location of the peripheral cue depended on Cue Type (valid, invalid, absent) and Target Location.

The rest of the procedure was similar to Experiment 1, except that (a) participants were instructed to ignore the cue stimuli (square outlines) and to respond quickly (within 400 ms) in each trial to the location of the target (left/right) by pressing one of two response box buttons with the index finger of the corresponding hand, (b) the data collection period started at -400 ms from target onset – just before peripheral cue onset in the 141/141 condition – and ended at 400 ms after target onset, (c) the following feedback was presented after the data collection period: „correct“ or „error“ depending on the given response and actual target location, or „too slow“ in case no response was detected, and (d) the summary information at the end of each block included the number of correct responses, the number of error responses, the number of „too slow“ trials, and the mean correct RT.

Statistical analyses

First, we analysed the mean performance measures and the respective cueing effects using repeated-measures ANOVAs. The Greenhouse-Geisser correction was applied if Mauchly's test for

sphericity was significant. All significant effects are reported.

Second, we calculated the descriptive functions for each combination of participant and condition, and for each condition after aggregating the data across participants, using life tables. We added 400 ms to each original RT datum in the data set in order to capture „negative“ RTs as well (and then the remaining trials with an RT at or below zero were discarded). We used 20 bins of 40 ms. Next to $h(t)$ of response occurrence we also estimated the discrete-time conditional accuracy function or $ca(t) = P(\text{correct} \mid T=t)$, by dividing the number of correct responses in bin t by the total number of observed responses in bin t (Pachella, 1974; Wickelgren, 1977). The standard formula for the standard error of a proportion was used.

Third, we fitted discrete-time hazard models and conditional accuracy models to the data. We used 10 bins of 32 ms to cover the time range (80,400]. Condition “invalid peripheral cue, ISI combination 47/47, and central cue absent” was chosen as the baseline condition. The main continuous predictor variable TIME was the time bin rank centered on reference bin (208,240]. The intercept, the linear effect of TIME and their correlation were treated as random effects. Next to dummy-coding the levels of our experimental factors (ISI combination, cue type, central cue presence) we also included BLOCK number, centered on value 25 and rescaled by dividing by 10. Next, we constructed a person-trial-bin oriented data set for the hazard modeling (containing 66,937 rows).

We started with a full multilevel cloglog-hazard model (85 parameters; with bins at level 1 nested within participant at level 2) encompassing the following effects at level 1: (a) a 3rd order polynomial for the shape of the cloglog-hazard function in the baseline condition (4 parameters), (b) the effects of valid peripheral cueing (VA), no peripheral cue (NC), central cue presence (CC), and ISI combination (47/141, 141/47, 141/141) were allowed to interact with TIME in a cubic fashion (24 parameters), (c) the 11 first-order interaction effects involving VA, NC, CC, and the levels of ISI combination could vary over time in a quadratic fashion (33 parameters),

(d) the 6 second-order interaction effects involving VA, NC, CC, and the levels of ISI combination could vary over time in a linear fashion (12 parameters), (e) the linear and quadratic effects of BLOCK could interact with time in a quadratic fashion (6 parameters), (f) to study whether the “total” cueing effect in the valid vs. invalid cueing conditions change over the time course of the experiment, we also included the interactions VA:BLOCK and VA:BLOCK:BLOCK that could vary over time in a quadratic fashion (6 parameters). We used the same automatic backward selection procedure as in Experiment 1.

For the conditional accuracy models we used the same multilevel modeling procedure except that we used the original person-trial oriented data set, only trials with observed RTs within the time segment (80,400] were included (11974 trials or rows), we predicted accuracy (1/0) in each bin, and used the symmetric logit link function.

Results

Mean performance measures

First, the error-rate was calculated for each combination of participant and condition using all observed responses between 100 and 400 ms (Figure 12A). A three-way repeated-measures ANOVA revealed significant main effects of Cue Type ($F(2,10) = 9.17, p = .005, \eta^2 = .65$), ISI combination ($F(3,15) = 5.83, p = .008, \eta^2 = .54$), and Central Cue Presence ($F(1,5) = 11.84, p = .018, \eta^2 = .70$), next to significant interaction effects between ISI and Central Cue Presence ($F(3,15) = 5.30, p = .011, \eta^2 = .51$), between Cue Type and Central Cue Presence ($F(2,10) = 10.91, p = .003, \eta^2 = .69$), and between ISI, Cue Type, and Central Cue Presence ($F(6,30) = 4.37, p = .003, \eta^2 = .47$).

Next, the cueing effect (invalid minus valid) was calculated for each ISI (Figure 12B). A two-way repeated-measures ANOVA showed a significant main effect of Central Cue Presence ($F(1,5) = 12.98, p = .016, \eta^2 = .72$), and a significant interaction effect ($F(3,15) = 5.62, p = .009, \eta^2 = .53$). The effect of Central Cue Presence is larger when ISI1 equals 141 ms.

Second, the mean correct RT was calculated for each combination of participant and condition using all observed correct responses between 100 and 400 ms (Figure 12C). A three-way repeated-measures ANOVA revealed significant main effects of Cue Type ($F(2,10) = 18.15, p < .001, \eta^2 = .78$), ISI combination ($F(3,15) = 17.25, p < .001, \eta^2 = .78$), and Central Cue Presence ($F(1,5) = 13.79, p = .014, \eta^2 = .73$), next to significant interaction effects between ISI and Central Cue Presence ($F(3,15) = 4.27, p = .023, \eta^2 = .46$), between Cue Type and Central Cue Presence ($F(2,10) = 42.38, p < .001, \eta^2 = .89$), and between ISI and Cue Type ($F(6,30) = 3.87, p = .006, \eta^2 = .44$).

Next, the cueing effect (invalid minus valid) was calculated for each ISI (Figure 12D). A two-way repeated-measures ANOVA showed significant main effects of Central Cue Presence ($F(1,5) = 34.75, p = .002, \eta^2 = .87$) and ISI ($F(3,15) = 5.14, p = .012, \eta^2 = .51$). Instead of speculating about the signs and sizes of these cueing effects at this point in the analysis, let us have a look at the descriptive functions of within-trial time.

Event history analysis: descriptive statistics

Figure 13 presents the aggregated data, and Figure 14 the data from a single individual. Four patterns are notable. First, the fastest responses in the cue-present conditions emerge time-locked to peripheral cue onset. These early responses tend to be all correct for valid cueing, and all incorrect for invalid cueing, demonstrating that the peripheral cue location activates the spatially compatible response channel.

Second, when the central cue is absent (top panels) hazard then tends to decrease somewhat while the conditional accuracy functions start approaching chance level, from below for invalid cueing and from above for valid cueing; When the central cue is present (bottom panels) hazard then displays a temporary peak value while the conditional accuracy functions approach .5 and even reverse, revealing a temporary negative cueing effect in $ca(t)$. The localized peak in hazard and the negative cueing effect in $ca(t)$ emerge at the same time, time-locked to central cue

onset.

Third, all hazard functions increase steadily around 200 ms after target onset. IOR appears in the hazard functions around this time for each ISI combination, and is somewhat stronger when the central cue is present. At this time also all $ca(t)$ functions start to rise to 1. Finally, each individual shows these effects (Figure 14), except that one individual did not show a pronounced IOR effect in hazard.

The cueing effects (valid minus invalid) in $h(t)$ and $ca(t)$ based on the aggregated data are shown in Figure 15. The delta- $h(t)$ plots (top panels) show that IOR emerges in bin (160,200], peaks in bin (200,240], and is somewhat stronger when the central cue is present (Figure 15B). The delta- $ca(t)$ plots (bottom panels) show how the peripheral cue-triggered, maximal positive cueing effect decreases towards zero, and how there is an overshoot in this decrease when the central cue is present, generating the temporary, smaller negative cueing effect in $ca(t)$, until target signals take over (Figure 15D). Note that there is no difference in behavior between 47/141 and 141/47 when the central cue is absent (Figures 15A and 15C).

Event history analysis: inferential statistics, cloglog- $h(t)$ model

Table 3 presents the selected cloglog-hazard model and Figures 16, 17, and 18 the model-based $h(t)$ functions. Figure 16A (top) shows that hazard increases steadily in the baseline condition until bin (272,304] (thick red line; parameters 1-4 in Table 3). The main effect of valid cueing on cloglog-hazard is significant for reference bin (208,240] (parameter 5, PE = -0.48004, $p < .001$) and changes non-linearly over time (parameters 6-8): HR(112) = 1.32 ($p = .044$), HR(176) = 0.61 ($p < .001$), HR(240) = 0.62 ($p < .001$), HR(304) = 0.87 ($p = .022$), and HR(368) = 1.11 ($p = .421$). Thus, compared to invalid cueing, the effect of valid cueing on cloglog-hazard is negative and significant in time segment (144-304] (compare green and red lines in the upper panel in Figure 16A). The small positive effect in bin 112 is likely a „false positive” finding.

The effect of peripheral cue absence is of course negative and decreases in absolute value over time (parameters 9-12). The three effects of changing to another ISI combination change over time in a cubic fashion (parameters 17-28; compare red lines in the upper row of Figure 16). The interaction effects NC:47/141 and NC:141/141 are positive when significant (parameters 31-36).

The presence of a central cue increases cloglog-hazard in bin 112 (parameter 13, PE = 0.389, $p = .0035$) compared to the baseline condition, due to an interaction with cubic time (parameter 16) – reflecting the localized peak in hazard time-locked to central cue onset as observed in the descriptive plots. It also decreases cloglog-hazard in bin 368 (parameter 13, PE = -0.336, $p = .025$). Importantly, when the central cue is present the validity effect (see parameter 5) becomes more negative in bins 176 (parameter 37, PE = -0.149, $p = .052$) and 240 (parameter 37, PE = -0.206, $p = .0022$), and more positive in bin 368 (parameter 37, PE = 0.379, $p = .030$). Center cue presence also interacts in a time-varying fashion with peripheral cue absence, 47/141, 141/47, and VA:47/141 (parameters 40-49).

Finally, the linear and nonlinear influences of block number on cloglog-hazard change nonlinearly with within-trial time (parameters 50-55). As shown in Figures 17 (block 1), 16 (block 25), and 18 (block 50), hazard in early bins increases during the course of the experiment, while hazard in late bins decreases, just as in Experiment 1. Also, the negative effect of valid versus invalid cueing in bin 240 (see parameter 5) decreases in absolute sign during the course of the experiment due to the positive interaction effect VA:BLOCK (parameter 56) which changes over within-trial time in a quadratic fashion (parameters 57-58).

Event history analysis: inferential statistics, logit-ca(t) model

Table 4 presents the selected logit-ca(t) model and Figures 16, 17, and 18 the model-based ca(t) functions. Figure 16A (bottom) shows that the conditional accuracy function in the baseline condition initially increases and reaches 1 around 272 ms after target onset (thick red line;

parameters 1-4 in Table 4). The intercept of 3.5 in reference bin (208,240] corresponds to $ca(240) = 0.97 (= \exp(3.5)/\{\exp(3.5)+1\})$.

There are positive effects of valid cueing and removing the peripheral cue on $\text{logit-}ca(t)$ that change nonlinearly with time (parameters 5-11). As a result we see a positive cueing effect in the $ca(t)$ functions for responses emitted before 240 ms (Figure 16A, bottom).

The effects of changing to another ISI combination increases $\text{logit-}ca(t)$ but only in the first few bins (parameters 15-23). The interaction effects involving valid cueing and one ISI combination are typically negative in the first 3 bins (parameters 24-32). As a result, the positive cueing effect in $ca(t)$ is largest for ISI combination 47/47. The interaction effects NC:47/141 and NC:141/47 change over time in a quadratic fashion (parameters 33-38).

When a central cue is presented, the $\text{logit-}ca(t)$ increases compared to the baseline condition in each bin (parameters 12-14). There is a negative time-invariant interaction effect between central cue presence and valid cueing (parameter 39), which reverses the early positive cueing effect in $ca(t)$, and a negative time-invariant interaction between central cue presence and peripheral cue absence (parameter 40). Central cue presence also interacts with ISI combinations 47/141 and 141/141, and the interactions 141/141:TIME and VA:47/141 (parameters 40-44).

Finally, the linear and nonlinear influence of block number on $\text{logit-}ca(t)$ change nonlinearly with the passage of within-trial time (parameters 45-50). The positive effects of valid cueing in bins 304 and 368 (see parameter 5) decrease during the course of the experiment due to the negative interaction effect VA:BLOCK in those bins (parameter 51).

Discussion

Experiment 2 reveals that the within-trial time course of IOR in the hazard functions (valid minus invalid) is very similar to that in Experiment 1 (compare Figures 8A and 15A): it is present between ~160 and ~280 ms after target onset in the hazard functions. Furthermore, presenting an

additional central cue after the peripheral cue and before target onset temporarily increases hazard about 200 ms later, causes the early positive cueing effect in the conditional accuracy functions to reverse into a temporary negative cueing effect, and increases the size of the sensory IOR effect in hazard.

The positive cueing effect in $ca(t)$ for the fastest responses shows that the cue acts like a prime in a response priming experiment (Burle, van den Wildenberg, & Ridderinkhof, 2005; Panis & Schmidt, 2016). To explain the temporary decrease in hazard and the change in $ca(t)$ right after these initial responses, we assume that the cue-triggered response channel activation is selectively inhibited if no overt response has occurred yet (Panis & Schmidt, 2016). The selective response inhibition attempts to restore the balance between the response channels before target signals take over. However, when a central cue is present, an additional process comes into play.

The fact that the hazard functions show a localized peak time-locked to central cue onset suggests that the centrally presented cue activates a left/right localization response channel. According to the attentional shift hypothesis (Stoffer, 1991) the central cue pulls attention back to the center, and this shift itself might cause corresponding (left or right) response channel activation. However, there are other, more established cognitive representations and processes that could explain it. For example, there is a lot of evidence that the location of a stimulus is coded in different spatial reference frames, one of which is centered on the current focus of attention (Howard, Lupiáñez, & Tipper, 1999; Nicoletti & Umiltà, 1989). Thus, if we assume that the salient peripheral cue onset attracts attention, then the central cue will occupy a left or right position relative to the attentional focus when it appears on the screen. This task-irrelevant spatial code will then lead to compatible response channel activation.

In sum, the peripheral cue will activate the spatially compatible response channel (e.g., left) which is then quickly and selectively inhibited, and the central cue will then bottom-up activate the opposite response channel (i.e., right for a peripheral cue on the left). As a result, the balance

between the competing response channels is temporarily tilted to the correct response for invalid cueing, and to the incorrect response for valid cueing – the negative cueing effect in $ca(t)$ – before target signals take over.

Why is sensory IOR in hazard larger when the cue is present? One possibility is that the selective response inhibition of the cue-triggered response is increased because the central cue is also acting as a contextual signal that any already active response channel is prematurely activated as the target still has to appear (Panis & Schmidt, 2016). This would be consistent with the idea of Satel and Wang (2012) that the IOR effect in an ignore-manual paradigm reflects two independent components (at least for the localization task in Experiment 2).

General discussion

We revisited the classic uninformative spatial cueing paradigm of Posner and Cohen (1984) to characterize the within-trial time course of the negative cueing effect observed with longer cue-target SOAs – inhibition of return (IOR). To temporally disentangle the contributions of working memory, habituation, response inhibition, and attentional selection processes, we analysed the RT distributions with discrete-time hazard functions of response occurrence, and the accuracy distributions with micro-level speed-accuracy tradeoff functions.

Our distributional results show that (a) cue presentation triggers covert (and sometimes overt) response channel activation, (b) its location is quickly stored in WM, (c) the premature response tendency is selectively inhibited, (d) IOR (valid vs. invalid) emerges around 160 ms after target onset in the hazard functions for cue-target SOAs above ~200 ms, and (e) the maximal within-trial duration of IOR is about 120 ms (for the current tasks and ISIs used).

To explain the shape of the hazard and conditional accuracy functions, we assume that the detected location of the peripheral cue in the dorsal system is available faster than the identity of the peripheral cue in the ventral system. Thus, because the peripheral cue shares task-relevant

features with the target (abrupt onset, lateral position) it will initially be able to activate the task-relevant response channel during feedforward sensorimotor processing (a conditionalized automatic response to abrupt onsets in Experiment 1 and spatial left/right codes in Experiment 2; see also Eimer & Kiss, 2008; Kiefer, 2007), likely via the direct pathway of the basal ganglia (Boussaoud & Kermadi, 1997; McNab & Klingberg, 2008; Middleton & Strick, 2000; Nagano-Saito, Martinu, & Monchi, 2014; Wiecki & Frank, 2013; Yin, 2014). Once the identity of the cue becomes available, however, an „error“ signal gets activated and reactive cognitive control processes will start to selectively inhibit the premature cue-triggered response channel activation if no overt response has occurred yet, likely via the indirect pathway of the basal ganglia (Kim, Amita, & Hikosaka, 2017; Panis & Schmidt, 2016; Stuphorn, 2015).

In the meantime, the location of the cue has been stored in spatial WM and produces a 2D-Gaussian-shaped inhibitory sensory bias around the memorized cue location in perceptual areas which delays target detection. Thus, when the target appears there are two processes active due to cue presentation in Experiment 2: a WM representation that is inhibiting the baseline activation in sensory areas at the cued location and selective response inhibition of the cue-triggered response. The latter is probably stronger when a contextual signal in the form of a central cue is present. Overall, our data suggest that sensory IOR can be properly measured in detection tasks, while the empirical effect size of the cueing effect can also be influenced by ongoing selective response inhibition processes in localization tasks.

Future studies using saccadic responses can now investigate whether oculomotor IOR portrays a different time course (within-trial, across-trial, and/or across-block) compared to manual responses to study potential differences in the underlying mechanisms (Briand, Larrison, & Sereno, 2000; Godijn & Theeuwes, 2004; Hilchey, Klein, & Satel, 2014; Hilchey, Klein, Satel, & Wang, 2012; Lisi & Cavanagh, 2017; McSorley, Haggard, & Walker, 2006, 2009; Satel & Wang, 2012). For example, an active motor intention in areas “downstream” of the superior colliculus

(e.g., the frontal eye fields) might influence the within-trial time course of oculomotor IOR. Note that different events of interest can be identified in movement trajectories, next to onset times (Theeuwes & Van der Stigchel, 2009). Also, measures of working memory performance can be included in the multilevel models at the participant level, together with cross-level interactions to study, for example, whether and when the effect of valid cueing on the within-trial time scale is affected by these working memory performance measures.

Other theoretical benefits of event history analysis

To even better appreciate what EHA can tell us about the nature of cognition, one can ask one-self the question: Why did it take more than 40 years to appreciate the rich temporal information present in RT and accuracy distributions? Two factors immediately come to our minds.

First, comparing mean RTs provides useful indices of cognitive processing durations when one assumes that the brain is a serial information processing system (Donders, 1869/1969; Sternberg, 1969). This philosophical assumption is very likely false and caused by the still popular computer metaphor of cognition (van Gelder, 1995). Instead, cognition is likely the behavior of a dynamical system that continuously makes *sequential transitions* from one stable state to another in separate but typically bi-directionally coupled, often topographically organized, neuronal populations (Cisek & Kalaska, 2010; Perone & Simmering, 2017; Schöner, Spencer, et al., 2016). A stable „peak“ state of population activity represents, for example, a memorized location (see Figure 10A), a sensory detection of the color red at position (x,y) in a two-dimensional color-position field, the (x,y) position of the current attentional focus, a motor intention of 30 degrees to the right, etc. The exact sequence of within-trial peaks across all coupled (sensory, central, motor) populations depends on the time-varying sensory input, the ongoing motor states, the history of detection and motor decisions, and the active instructions-implementing top-down intentions (Schöner, Spencer, et al., 2016). In such a view, exactly when a stimulus is presented is important

for behavior (in contrast to algorithms), history matters (not cyclic), there is circular input-response causality (not one-way causality), and the underlying neural system is a recurrent one (not feedforward only; van Gelder, 1995). On a longer time scale learning processes such as synaptic adaptation (which slowly change the Gaussian-shaped kernels that couple neurons of different populations, as well as neurons of the same population) and proactive cognitive control processes come into play (Braver, 2012; Schöner, Spencer, et al., 2016).

This would mean indeed that differently timed responses can reflect different, stable within-trial motor states, as observed in the $ca(t)$ functions here and elsewhere (Panis, et al., 2020; Panis & Schmidt, 2016; Wolkersdorfer, et al., 2020). Statistically controlling for the passage of time on multiple time scales during data analysis is therefore *equally* important as experimental control during the design of an experiment, in order to understand human behavior in our experimental paradigms.

A second factor that comes to mind is the fact that the concept of attention has been used so flexibly that it has often sufficed to easily explain mean performance patterns post hoc. There is a recent trend in the literature, however, to reinterpret attentional studies (Awh, Belopolsky, & Theeuwes, 2012; Hommel, Chapman, Cisek, Neyedli, Song, & Welsh, 2019; Yaron & Lamy, 2020). Hommel et al. (2019) claim that the concept of “attention” is one of the most misleading and misused terms in the cognitive sciences, and argue that we should instead focus on behaviorally relevant selection processes and the many systems that implement them.

For example, we can imagine that many readers were surprised to see the presence of the cue-triggered overt responses in Figures 4 and 13. When blindly trimming RT data, such unexpected and undetected behavior can strongly influence the small effect sizes of differences in mean (correct) RT, leading to unnecessary variation between laboratories. Relatedly, theoretically important inter-individual differences can easily be masked when focusing on general behavior by blindly averaging across participants (Panis et al., 2020). We therefore recommend to always first

plot the empirical hazard and conditional accuracy functions of each individual before making any further data-analytical or computational modeling decision. Issues about bin size optimality play a secondary role at this moment in time in our view, because by working in discrete time – or using interval censored data – we can make an informed tradeoff between the availability of temporal information (smaller bins increase temporal resolution) with the feasibility to perform expensive data collection efforts (continuous-time methods require in general more repeated measures per condition). In other words, the number and sizes of the time bins used for the analyses can be optimally adapted to each situation, depending on the duration of the data collection period, the rarity of event occurrence, the shape of the empirically observed hazard function, and whether one is using a large- or small- N design as done here (Smith & Little, 2018).

We have tried to use a minimum number of assumptions about attention when discussing the empirical patterns. To explain our results we only had to assume that attention is summoned to the salient peripheral cue location, and that the focus of attention forms the center of a spatial reference frame. However, a cue is also stored in working memory, it can activate a response channel, and it can probably act as a contextual cue for reactive cognitive control processes. This means that we should also revisit the informative cueing studies (Eriksen & Hoffman, 1973; Jonides, 1981; Posner, 1980; McCormick & Klein, 1990; Umiltà, Riggio, Dascola, & Rizzolatti, 1991) because the informative cue might also act as a contextual cue and lower the general response threshold, for example (Wiecki & Frank, 2013). By studying how empirical and simulated hazard estimates change over multiple time scales, we can now efficiently test dynamic theories of attention (Bundesen, 1990; Duncan, 2013; Sperling & Weichselgartner, 1995).

Finally, event history analysis can not only be used to distinguish behavioral responses triggered by the feedforward sweep or after time-consuming recurrent processing (VanRullen & Koch, 2003), but also to study the effect of time-varying predictors like eye gaze position, heart rate, and the amplitude of alpha power in occipital regions. And again, the effect of such a time-

varying predictor on the hazard of (behavioral or neural) event occurrence can be linear or nonlinear, and might change over time. We are positive that accepting the critical role of time scales when studying behavior will allow us to make a big step forward toward more unifying theories and interpretations of the behavioral effects people display in different paradigms.

Conclusions

Mean performance measures conceal crucial information on behavioral dynamics on multiple time scales. Our results suggest that sensory IOR is a direct result of the encoding of the cue location in spatial working memory – a process modulated by synaptic learning processes on the across-block time scale –, although the behaviorally measured and timed effect sizes can also be affected by response inhibition processes. Sensory IOR effects can therefore be expected in multiple paradigms and have perhaps already been measured but reported under different names. Given the statistical and theoretical advantages, and the free availability of relevant software 151 years after Donders (1869/1969), there is currently no reason anymore whatsoever to not start using event history analysis for repeated-measures time-to-response data, and other types of time-to-event data.

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Open practices statement

Data and R code are available; please email the first author.

Footnotes

1 Hilchey, Klein, and Satel (2014) argue that the late inhibitory cueing effect observed by Posner and Cohen (1984) should be distinguished from IOR (proper) as defined by Posner, Rafal, Choate, and Vaughan (1985) because the effect observed by Posner and Cohen (1984) is not believed to be caused by oculomotor activation and reflect a motor bias. We will use the term “oculomotor IOR” to refer to the effect observed by Posner et al. (1985) and the terms “IOR” and “sensory IOR” to refer to the effect observed by Posner and Cohen (1984). We come back to this issue of naming and definition in the General discussion.

2 Event history analysis is also known as survival analysis, hazard analysis, failure-time analysis, duration analysis, and transition analysis, and refers to a set of statistical methods to study the occurrence and timing of events; To apply it you must be able to define the event of interest (any qualitative change that can be situated in time; here: a button-press), to identify time point zero (here: target display onset), and to measure the passage of time between time zero and event occurrence in discrete or continuous units (Allison, 2010; Singer & Willett, 2003).

3 Right-censoring occurs when all you know about an observation on a variable T is that it is *larger* than some value. Interval censoring means that all you know about T is that $a < T < b$, for some values of a and b – as in Table 1 (Allison, 2010). The most common type of right-censoring is “singly Type I censoring” which applies when the experiment uses a fixed response deadline for all trials. “Type I” means that the censoring time is fixed and under the control of the experimenter, and “singly” refers to the fact that all observations have the same censoring time (Allison, 2010).

4 Thus, the endpoints of the bins for plotting the descriptive statistics are: -480, -440, -400, -360, -

320, -280, -240, -200, -160, -120, -80, -40, 0, 40, 80, 120, 160, 200, 240, 280, 320, 360, 400, 450, 500, 550 and 600 ms, relative to target display onset. While the starting point is not part of the interval, the endpoint is – the first and last bins are (-520,-480] and (550,600], respectively, for example.

5 The complementary log-log link is preferred over the logit link for a discrete-time hazard model when the events can in principle occur at any time during each time bin (Allison, 2010), which is the case for RT data: $\text{cloglog}[h(t)] = \ln\{-\ln[1-h(t)]\}$. Inverse of the link: $h(t) = 1 - \exp\{-\exp\{\text{cloglog}[h(t)]\}\}$. Although the cloglog link function is asymmetrical it is similar to the logit link function for proportions below about 0.4.

6 Barr, Levy, Scheepers, and Tily (2013) claim that linear mixed-effects models generalize best when they include the maximal random effects structure justified by the design. However, fitting a maximal model can lead to a significant loss of power, as shown by Matuschek, Kliegl, Vasishth, Baayen, and Bates (2017). Although there are guidelines for linear mixed models (Cunnings, 2012; Zuur & Ieno, 2016), there is a need for simulation studies and guidelines for generalized linear mixed models – especially what to do when the originally planned model does not converge. Current practices of switching ad hoc to a simpler model, sometimes with drastically different properties, are concerning. Furthermore, it is more difficult for getting maximal models to converge in the case of categorical response variables (Barr et al., 2013) – fitting a hazard model with an intermediate complex random effects structure to our IOR detection data took at least 11 hours as the model never converged. For these reasons, we opted for a simple random effects structure with three parameters (intercept, slope of TIME, and their correlation).

7 After comparing the results of several uninformative cueing experiments, Posner and Cohen (1984) mention in their footnote 1 on page 536, that for one experiment “It should be noted ... that the cued side is relatively flat, while the uncued side shows a reduction in RT with time. It might be thought that the uncued side is facilitated rather than the cued side being inhibited. However, in most experiments a cue produced a reduction in RT with SOA due to a general alerting change ... Thus the relatively flat function for the cued side indicates that something is preventing the usual decrease of RT with SOA. ... However, irrespective of the differing overall shapes ... the early facilitation and late inhibition of the cued side with respect to the uncued side is always found.” We observe here that the total IOR effect (valid compared to invalid) consists of a facilitatory and an inhibitory component when compared to central cueing. The fact that the facilitatory component decreases in size over the course of Experiment 1 (see section *Event history analysis: inferential statistics*) can explain why Posner and Cohen (1984) decided to measure IOR (“late inhibition”) as the difference between valid and invalid cueing conditions.

Tables

Table 1. Life table for condition “invalid / 450 ms ISI” in Experiment 1, aggregated data (N=6).

time bin	t	cens	E	RS	h(t)	se[h(t)]	S(t)	se[S(t)]	P(t)	se[P(t)]
(-520,-480]	1	0	0	443	0	0	1	0	0	0
(-480,-440]	2	0	1	443	0.002	0.002	0.998	0.002	0.002	0.002
(-440,-400]	3	0	1	442	0.002	0.002	0.995	0.003	0.002	0.002
(-400,-360]	4	0	0	441	0	0	0.995	0.003	0	0
(-360,-320]	5	0	0	441	0	0	0.995	0.003	0	0
(-320,-280]	6	0	2	441	0.005	0.003	0.991	0.004	0.005	0.003
(-280,-240]	7	0	19	439	0.043	0.01	0.948	0.01	0.043	0.01
(-240,-200]	8	0	16	420	0.038	0.009	0.912	0.013	0.036	0.009
(-200,-160]	9	0	7	404	0.017	0.006	0.896	0.014	0.016	0.006
(-160,-120]	10	0	6	397	0.015	0.006	0.883	0.015	0.014	0.005
(-120,-80]	11	0	10	391	0.026	0.008	0.86	0.016	0.023	0.007
(-80,-40]	12	0	1	381	0.003	0.003	0.858	0.016	0.002	0.002
(-40,0]	13	0	5	380	0.013	0.006	0.847	0.017	0.011	0.005
(0,40]	14	0	7	375	0.019	0.007	0.831	0.017	0.016	0.006
(40,80]	15	0	5	368	0.014	0.006	0.819	0.017	0.011	0.005
(80,120]	16	0	3	363	0.008	0.005	0.813	0.018	0.007	0.004
(120,160]	17	0	6	360	0.017	0.007	0.799	0.018	0.014	0.005
(160,200]	18	0	33	354	0.093	0.015	0.725	0.02	0.074	0.012
(200,240]	19	0	83	321	0.259	0.024	0.537	0.021	0.187	0.019
(240,280]	20	0	77	238	0.324	0.03	0.363	0.019	0.174	0.018
(280,320]	21	0	55	161	0.342	0.037	0.239	0.015	0.124	0.016
(320,360]	22	0	34	106	0.321	0.045	0.163	0.011	0.077	0.013
(360,400]	23	0	18	72	0.25	0.051	0.122	0.009	0.041	0.009
(400,450]	24	0	19	54	0.352	0.065	0.079	0.007	0.043	0.01
(450,500]	25	0	12	35	0.343	0.08	0.052	0.005	0.027	0.008
(500,550]	26	0	7	23	0.304	0.096	0.036	0.003	0.016	0.006
(550,600]	27	13	3	16	0.188	0.098	0.029	0.003	0.007	0.004

Note. For each time bin (column 1) with rank t (column 2) the number of observed responses (E) are counted and the risk set (RS) is determined, before estimating the discrete-time hazard function $h(t) = P(T = t | T \geq t)$ as E/RS . The survivor function $S(t) = P(T > t) = [1-h(t)] * [1-h(t-1)] * ... * [1-h(1)]$, and the probability mass function $P(t) = P(T = t) = h(t) * S(t-1)$. The standard errors (se) for $h(t)$ and $P(t)$ are estimated using the familiar formula for a proportion p – the square root of $\{p(1-p) / N\}$ – where N equals $RS(t)$ for $h(t)$, and $RS(1)$ for $P(t)$. The standard errors for $S(t)$ are estimated using the recurrent formula on page 350 of Singer and Willett (2003). Note that $P(t)$ also equals

the number of events in bin t divided by the risk set of the first bin. Thirteen trials are right-censored (“cens”) at 600 ms after target onset (column 3).

Table 2. Selected cloglog-hazard model for Experiment 1 (columns 5 to 8).

		(160,200]		(240,280]				(320,360]		(400,450]	
Nr.	Effect	PE	p	PE	se	z	p	PE	p	PE	p
1	Intercept	-2.4567	8.42e-12 ***	-0.784	0.25644	-3.05	0.002234 **	-0.5268	0.004943 **	-0.8986	6.73e-07 ***
2	TIME			0.417	0.06332	6.58	4.54e-11 ***				
3	TIME ²			-0.177	0.01227	14.42	<2e-16 ***				
4	TIME ³			0.0164	0.00161	10.12	<2e-16 ***				
5	VA	-0.6398	0.000114 ***	-0.5103	0.07304	-6.98	2.81e-12 ***	-0.0478	0.52904	0.1399	0.228706
6	VA:TIME			0.1987	0.0455	4.36	1.26e-05 ***				
7	VA:TIME ²			0.0415	0.02404	1.72	0.083980 .				
8	VA:TIME ³			-0.0126	0.00358	-3.52	0.000423 ***				
9	IN	0.6047	4.88e-07 ***	0.1763	0.06652	2.65	0.008011 **	-0.0968	0.249921	-0.2147	0.043364 *
10	IN:TIME			-0.1754	0.04091	-4.28	1.81e-05 ***				
11	IN:TIME ²			0.0194	0.00967	2	0.044864 *				
12	NC	-1.9335	<2e-16 ***	-1.0102	0.0837	12.06	<2e-16 ***	-0.3627	2.36e-05 ***	0.0076	0.946545
13	NC:TIME			0.3929	0.05303	7.4	1.28e-13 ***				
14	NC:TIME ²			-0.0346	0.00985	-3.51	0.000446 ***				
15	ISI_450	-0.5261	6.87e-12 ***	-0.3123	0.04801	-6.5	7.76e-11 ***	-0.0984	0.057073 .	0.1153	0.167957
16	ISI_450:TIME			0.1069	0.02221	4.81	1.49e-06 ***				
17	NC:ISI_450	0.5124	0.002877 **	0.2939	0.10816	2.71	0.006568 **	0.0752	0.391572	-0.1433	0.280197
18	NC:ISI_450:TIME			-0.1093	0.04165	-2.62	0.008649 **				
19	BLOCK	0.0194	4.33e-12 ***	0.0084	0.00179	4.71	2.43e-06 ***	-0.0024	0.116915	-0.0134	1.62e-08 ***
20	BLOCK:TIME			-0.0054	0.0007	-7.81	5.57e-15 ***				
21	BLOCK ²	-0.0029	4.55e-05 ***	-0.0013	0.00045	-2.88	0.003911 **	0.0003	0.45695	0.0019	0.004018 **
22	BLOCK ² :TIME			0.0008	0.00019	4.22	2.38e-05 ***				
23	BLOCK:IN	-0.0044	0.027616 *	-0.0044	0.00201	-2.2	0.027662 *	-0.0044	0.027640 *	-0.0044	0.027618 *
	RANDOM										
24	variance intercept	0.709		0.376				0.184		0.133	
25	variance TIME	0.017		0.017				0.017		0.017	
26	correlation	-0.9		-0.81				-0.53		0.1	

Note. The model was centered on reference bin (240,280] during model selection. The selected model was refitted three times with TIME centered each time on another reference bin: (160,200], (320,360], and (400,450]. Parameter estimates (PE) are in cloglog-hazard units.

Table 3. Selected cloglog-hazard model for Experiment 2 (columns 7 to 10).

	(80,112]		(144,176]		(208,240]				(272,304]		(336,368]		
Nr	effect	PE	p	PE	p	PE	se	z	p	PE	p	PE	p
1	(Intercept)	-3.71172	< 2e-16 ***	-2.27E+00	< 2e-16 ***	-0.88024	0.097019	-9.07	< 2e-16 ***	-0.1242	0.333843	-0.58228	0.010361 *
2	TIME					0.58476	0.054242	10.78	< 2e-16 ***				
3	TIME ²					-0.07916	0.006834	11.58	< 2e-16 ***				
4	TIME ³					-0.0121	0.002162	-5.59	2.21e-08 ***				
5	VA	0.27588	0.044383 *	-4.89E-01	4.30e-16 ***	-0.48004	0.04631	10.36	< 2e-16 ***	-0.1359	0.021612 *	0.10388	0.420618
6	TIME:VA					0.12496	0.027972	4.46	7.92e-06 ***				
7	TIME ² :VA					0.04186	0.007082	5.91	3.38e-09 ***				
8	TIME ³ :VA					-0.00915	0.002031	-4.5	6.57e-06 ***				
9	NC	-7.09315	< 2e-16 ***	-3.05E+00	< 2e-16 ***	-0.97806	0.050834	19.24	< 2e-16 ***	-0.18991	0.000815 ***	0.01481	0.908046
10	TIME:NC					0.65612	0.038466	17.05	< 2e-16 ***				
11	TIME ² :NC					-0.16006	0.015332	10.44	< 2e-16 ***				
12	TIME ³ :NC					0.01452	0.002466	5.88	3.88e-09 ***				
13	CC	0.3888	0.003493 **	2.47E-02	0.680296	0.03169	0.048554	0.65	0.513878	0.03588	0.568714	-0.33603	0.025283 *
14	TIME:CC					0.03391	0.027163	1.24	0.2118				
15	TIME ² :CC					-0.00034	0.007826	-0.04	0.965036				
16	TIME ³ :CC					-0.00778	0.001772	-4.39	1.13e-05 ***				
17	47/141	-0.09599	0.573978	5.49E-01	4.28e-12 ***	0.48916	0.055127	8.87	< 2e-16 ***	0.08196	0.312766	-0.31596	0.060198 .
18	TIME:47/141					-0.14637	0.035845	-4.08	4.44e-05 ***				
19	TIME ² :47/141					-0.04345	0.007004	-6.2	5.50e-10 ***				
20	TIME ³ :47/141					0.00742	0.002433	3.05	0.002270 **				
21	141/47	0.11659	0.483905	5.86E-01	< 2e-16 ***	0.45711	0.044333	10.31	< 2e-16 ***	0.10253	0.037096 *	-0.1062	0.323935
22	TIME:141/47					-0.15173	0.027938	-5.43	5.60e-08 ***				
23	TIME ² :141/47					-0.02825	0.007572	-3.73	0.000190 ***				
24	TIME ³ :141/47					0.00774	0.002161	3.58	0.000340 ***				
25	141/141	0.86079	1.84e-12 ***	9.03E-01	< 2e-16 ***	0.39469	0.043886	8.99	< 2e-16 ***	-0.14731	0.007279 **	-0.20547	0.073928 .
26	TIME:141/141					-0.3057	0.02901	10.53	< 2e-16 ***				
27	TIME ² :141/141					-0.00419	0.006522	-0.64	0.520059				
28	TIME ³ :141/141					0.01077	0.002272	4.74	2.12e-06 ***				
29	VA:47/141	0.15058	0.38193	6.69E-02	0.529233	-0.01677	0.072048	-0.23	0.815884	-0.10043	0.342088	-0.18433	0.280846
30	TIME:VA:47/141					-0.04183	0.038902	-1.07	0.282148				
31	NC:47/141	1.86928	1.60e-08 ***	6.99E-01	2.79e-08 ***	0.06194	0.065954	0.93	0.347593	-0.04172	0.619443	0.38775	0.036955 *
32	TIME:NC:47/141					-0.1852	0.041286	-4.48	7.26e-06 ***				
33	TIME ² :NC:47/141					0.06666	0.014094	4.73	2.24e-06 ***				
34	NC:141/141	2.05276	1.21e-11 ***	9.07E-01	4.10e-15 ***	0.22278	0.062155	3.58	0.000338 ***	0.00107	0.987279	0.24141	0.102229
35	TIME:NC:141/141					-0.22643	0.036563	-6.19	5.91e-10 ***				
36	TIME ² :NC:141/141					0.05777	0.012868	4.49	7.14e-06 ***				
37	VA:CC	0.14003	0.385499	-1.49E-01	0.051470 .	-0.20561	0.067124	-3.06	0.002190 **	-0.02964	0.693661	0.37854	0.030217 *
38	TIME:VA:CC					0.02984	0.026663	1.11	0.263067				
39	TIME ² :VA:CC					0.02906	0.009876	2.94	0.003249 **				
40	NC:CC	3.20655	< 2e-16 ***	1.61E+00	< 2e-16 ***	0.60431	0.058426	10.34	< 2e-16 ***	0.18339	0.007924 **	0.34909	0.037404 *
41	TIME:NC:CC					-0.35715	0.038895	-9.18	< 2e-16 ***				
42	TIME ² :NC:CC					0.07334	0.014063	5.21	1.84e-07 ***				
43	CC:47/141	0.3727	0.010464 *	2.34E-01	0.009732 **	0.09433	0.056428	1.67	0.094582 .	-0.04482	0.568324	-0.18413	0.15922
44	TIME:CC:47/141					-0.06957	0.031571	-2.2	0.027541 *				
45	CC:141/47	0.62524	0.000214 ***	7.16E-02	0.328835	-0.16423	0.057024	-2.88	0.003975 **	-0.08183	0.17747	0.31838	0.024103 *
46	TIME:CC:141/47					-0.03835	0.024205	-1.58	0.113066				
47	TIME ² :CC:141/47					0.03977	0.009081	4.37	1.19e-05 ***				
48	VA:CC:47/141	-0.62712	0.006900 **	-3.03E-01	0.036979 *	0.02091	0.095107	0.22	0.825919	0.34499	0.008594 **	0.66916	0.001721 **
49	TIME:VA:CC:47/141					0.162	0.050379	3.21	0.001301 **				
50	BLOCK	0.03706	< 2e-16 ***	2.83E-02	< 2e-16 ***	0.01698	0.001003	16.92	< 2e-16 ***	0.00318	0.002595 **	-0.01311	8.82e-08 ***
51	TIME:BLOCK					-0.00627	0.000472	13.27	< 2e-16 ***				
52	TIME ² :BLOCK					-0.00031	0.000179	-1.74	0.080765 .				
53	BLOCK ²	-0.00597	0.002034 **	-7.37E-03	< 2e-16 ***	-0.00571	0.000633	-9.01	< 2e-16 ***	-0.00103	0.115799	0.00669	1.01e-05 ***
54	TIME:BLOCK ²					0.00158	0.000268	5.9	3.57e-09 ***				
55	TIME ² :BLOCK ²					0.00037	0.000102	3.71	0.000201 ***				
56	BLOCK:VA	-0.00752	0.140752	8.14E-04	0.717779	0.00433	0.001831	2.36	0.017900 *	0.00304	0.100711	-0.00307	0.484011
57	TIME:BLOCK:VA					0.00055	0.000731	0.76	0.446861				
58	TIME ² :BLOCK:VA					-0.0006	0.000285	-2.1	0.035173 *				
	RANDOM												
59	variance intercept	0.29042		0.11592		0.04654				0.08225		0.22308	
60	variance TIME	0.01314		0.01314		0.01314				0.01313		0.01314	
61	correlation	-0.92		-0.78		-0.17				0.67		0.89	

Note. The model was centered on reference bin (208,240] during model selection. The selected model was refitted four times with TIME centered each time on another reference bin: (80,112], (144,176], (272,304], and (336,368]. Parameter estimates (PE) are in cloglog-hazard units.

Table 4. Selected logit- $ca(t)$ model for Experiment 2 (columns 7 to 10).

		(80,112]		(144,176]		(208,240]			(272,304]		(336,368]		
Nr	effect	PE	<i>p</i>	PE	<i>p</i>	PE	se	<i>z</i>	<i>p</i>	PE	<i>p</i>	PE	<i>p</i>
1	(Intercept)	-2.96545	6.12e-15 ***	2.09E-01	0.443095	3.50795	0.500558	7	2.42e-12 ***	5.69722	3.17e-13 ***	5.4991	1.10e-06 ***
2	TIME					1.47595	0.165285	8.93	< 2e-16 ***				
3	TIME ²					-0.13958	0.025582	-5.45	4.86e-08 ***				
4	TIME ³					-0.02595	0.004749	-5.46	4.62e-08 ***				
5	VA	4.77644	< 2e-16 ***	1.71E+00	6.12e-11 ***	0.37271	0.302649	1.23	0.218137	0.7758	0.122144	2.90816	0.007940 **
6	TIME:VA					-0.23324	0.13586	-1.71	0.086013 .				
7	TIME ² :VA					0.21738	0.041827	5.19	2.02e-07 ***				
8	NC	1.03627	0.040639 *	1.13E+00	0.000182 ***	0.48128	0.276396	1.74	0.081632 .	-0.01558	0.967752	0.51937	0.411373
9	TIME:NC					-0.35859	0.140572	-2.55	0.010743 *				
10	TIME ² :NC					0.01848	0.028848	0.64	0.521637				
11	TIME ³ :NC					0.01833	0.007837	2.33	0.019346 *				
12	CC	1.72498	5.73e-12 ***	1.13E+00	1.86e-10 ***	0.89279	0.211026	4.23	2.33e-05 ***	1.01514	0.000442 ***	1.501	0.002403 **
13	TIME:CC					-0.02823	0.064193	-0.44	0.660002				
14	TIME ² :CC					0.04495	0.017341	2.59	0.009527 **				
15	47/141	2.78541	2.23e-10 ***	5.18E-01	0.027274 *	-0.53304	0.297277	-1.79	0.072958 .	-0.3721	0.601223	1.00588	0.527102
16	TIME:47/141					-0.22125	0.194412	-1.13	0.255091				
17	TIME ² :47/141					0.15245	0.051596	2.95	0.003128 **				
18	141/47	2.25342	2.79e-09 ***	6.13E-01	0.002176 **	-0.36577	0.280726	-1.3	0.192589	-0.67613	0.14273	-0.32669	0.724052
19	TIME:141/47					-0.32322	0.120252	-2.68	0.007191 **				
20	TIME ² :141/47					0.08315	0.034799	2.39	0.016871 *				
21	141/141	2.43179	2.96e-09 ***	5.82E-01	0.005739 **	-0.35752	0.255292	-1.4	0.161382	-0.38767	0.273838	0.49504	0.471095
22	TIME:141/141					-0.24253	0.095758	-2.53	0.011317 *				
23	TIME ² :141/141					0.11394	0.030235	3.76	0.000164 ***				
24	VA:47/141	-5.20981	< 2e-16 ***	-8.26E-01	0.032802 *	1.04848	0.479851	2.18	0.028887 *	0.41263	0.67437	-2.72958	0.207896
25	TIME:VA:47/141					0.30848	0.262352	1.17	0.239663				
26	TIME ² :VA:47/141					-0.31467	0.073016	-4.31	1.63e-05 ***				
27	VA:141/47	-3.86032	1.93e-13 ***	-9.72E-01	0.001756 **	0.66927	0.394453	1.69	0.089750 .	1.06221	0.183681	0.2067	0.909381
28	TIME:VA:141/47					0.50809	0.222708	2.28	0.022523 *				
29	TIME ² :VA:141/47					-0.15656	0.063931	-2.44	0.014329 *				
30	VA:141/141	-3.81044	7.89e-13 ***	-2.99E-01	0.316476	0.92944	0.404532	2.29	0.021586 *	-0.12731	0.848632	-3.46173	0.014305 *
31	TIME:VA:141/141					0.04268	0.176485	0.24	0.80891				
32	TIME ² :VA:141/141					-0.28604	0.054279	-5.27	1.37e-07 ***				
33	NC:47/141	-1.94906	0.003601 **	2.21E-01	0.49594	1.03489	0.399885	2.58	0.009654 **	0.49358	0.52718	-1.4062	0.4056
34	TIME:NC:47/141					0.06645	0.214252	0.31	0.756428				
35	TIME ² :NC:47/141					-0.17011	0.060818	-2.79	0.005155 **				
36	NC:141/47	-2.03055	0.013594 *	-2.83E-01	0.445821	0.58493	0.387933	1.5	0.1316	0.56759	0.305943	-0.32695	0.759989
37	TIME:NC:141/47					0.21351	0.162596	1.31	0.189126				
38	TIME ² :NC:141/47					-0.11017	0.049687	-2.21	0.026595 *				
39	VA:CC	-2.77867	< 2e-16 ***	-2.78E+00	< 2e-16 ***	-2.77922	0.22562	12.31	< 2e-16 ***	-2.77918	< 2e-16 ***	-2.77892	< 2e-16 ***
40	NC:CC	-1.35802	1.21e-06 ***	-1.36E+00	1.23e-06 ***	-1.35806	0.279775	-4.85	1.21e-06 ***	-1.35784	1.22e-06 ***	-1.35842	1.20e-06 ***
41	CC:47/141	-0.2421	0.359876	-2.42E-01	0.360438	-0.24322	0.264461	-0.92	0.357725	-0.24214	0.359979	-0.24255	0.359063
42	CC:141/141	-1.32232	7.78e-05 ***	-5.08E-01	0.018855 *	0.30482	0.319188	0.95	0.339574	1.12224	0.033078 *	1.93424	0.010652 *
43	TIME:CC:141/141					0.40697	0.122603	3.31	0.000902 ***				
44	VA:CC:47/141	0.98007	0.018624 *	9.81E-01	0.018580 *	0.98134	0.416522	2.35	0.018471 *	0.98038	0.018638 *	0.9803	0.018625 *
45	BLOCK	0.00161	0.853686	-2.13E-03	0.635342	-0.00951	0.005569	-1.7	0.087505 .	-0.02052	0.006469 **	-0.0352	0.009369 **
46	TIME:BLOCK					-0.00459	0.002017	-2.27	0.022642 *				
47	TIME ² :BLOCK					-0.00045	0.000612	-0.74	0.45897				
48	BLOCK ²	-0.00534	0.228382	5.76E-03	0.029376 *	0.00828	0.003445	2.4	0.016231 *	0.00221	0.651426	-0.01238	0.18007
49	TIME:BLOCK ²					-0.00088	0.001296	-0.68	0.494369				
50	TIME ² :BLOCK ²					-0.00107	0.000416	-2.57	0.009929 **				
51	BLOCK:VA	0.01573	0.128758	9.58E-06	0.998813	-0.01571	0.008948	-1.75	0.079115 .	-0.03137	0.035870 *	-0.04689	0.030514 *
52	TIME:BLOCK:VA					-0.00786	0.003617	-2.17	0.029685 *				
	RANDOM												
53	variance intercept	0.04079		0.24609		1.18425				2.87064		5.30802	
54	variance TIME	0.09236		0.09236		0.09184				0.09224		0.09259	
55	correlation	-0.67		0.95		0.99				1		1	

Note. The model was centered on reference bin (208,240] during model selection. The selected model was refitted four times with TIME centered each time on another reference bin: (80,112], (144,176], (272,304], and (336,368]. Parameter estimates (PE) are in logit[$ca(t)$] units.

Figure captions

Figure 1. Single-cell superior colliculus activity in spatial cueing tasks. A valid (blue) or invalid (red) cue (C) was presented together with a fixation point (F) and followed by the target (T). Copy of panel B (bottom) of Figure 3 of Fecteau, Bell, and Munoz (2004). CTOA, cue-target onset asynchrony.

Figure 2. Trial design in Experiment 1. TRTO, time relative to target display onset.

Figure 3. (A) Mean detection RT in Experiment 1, as a function of cue type and target-present ISI. Error bars represent ± 1 standard error of the mean. (B) Cueing effect (CE) in mean RT, as a function of ISI (red). Error bars represent the estimated 95% confidence interval for the difference between two means. Black lines represent the CEs for each individual.

Figure 4. Descriptive statistics of event history analysis for Experiment 1 (hazard and survivor functions), based on the aggregated data. The vertical black striped line is positioned at target onset, and the vertical red striped line at cue offset. The thin vertical lines in the survival plots are positioned at the estimated median RTs. Error bars represent ± 1 standard error of the respective proportion.

Figure 5. Descriptive statistics of event history analysis for Experiment 1, participant 2. Same conventions as in Figure 4.

Figure 6. Descriptive statistics of event history analysis for Experiment 1, participant 4. Same conventions as in Figure 4.

Figure 7. Individual cueing effects in the hazard functions (valid minus invalid) in Experiment 1. The grey area covering time segment (160,280] is shifted with +20 ms for aesthetic purposes. The black vertical striped line is located at target display onset. Error bars indicate the 95% confidence interval for a difference between two proportions.

Figure 8. (A) Cueing effects (valid minus invalid) in hazard based on aggregated data (excluding participant 6) in Experiment 1. (B) Cueing effects (valid minus central) in hazard based on aggregated data (excluding participant 6). The grey area covering time segment (160,280] is shifted with +20 ms. The black vertical striped line is located at target display onset. Error bars indicate the 95% confidence interval for a difference between two proportions.

Figure 9. Model-based (cloglog-)hazard functions, as a function of ISI (columns), cue type (see legend), and block number (rows).

Figure 10. (A). Illustration of two layers from the multi-layered dynamic field model of change detection by Johnson, Spencer, Luck, and Schöner (2009). The top panel shows a snapshot of the working memory layer after cue offset. The population activity (blue) displays a stable “peak” reflecting the memorized cue location. The peak is kept active due to reverberating activity along short-range excitatory lateral connections, while it’s growth is kept in check by longer-range inhibitory lateral connections, resulting in the Mexican hat-shaped activity profile. Output activity is indicated by the red line and provides a (broader) inhibitory bias to the perceptual field (bottom panel). No input is currently presented to each layer (green lines). The figure was created with the cosivina software (<https://dynamicfieldtheory.org/>). (B) The facilitatory and inhibitory component of the IOR effect can be explained based on a WM-triggered inhibitory sensory bias, as well as the decrease of the facilitation effect over the course of the experiment. The cue was presented either

at position -50 (green), 0 (blue), or 50 (red), and the target is about to appear at position -50. The curves show the population activity for a single spatial dimension and the sensory bias present before target onset, before and after synaptic learning.

Figure 11. Trial design in Experiment 2. TRTO, time relative to target display onset.

Figure 12. Mean performance results and cueing effects in mean performance measures in Experiment 2, as a function of ISI combination, peripheral cue type, and central cue presence. (A) Mean error-rates. (B) Mean correct RTs. (C) Cueing effects in mean error-rates. (D) Cueing effects in mean correct RTs.

Figure 13. Descriptive statistics of event history analysis for Experiment 2 (hazard and survivor functions) extended with conditional accuracy functions, based on the aggregated data. The vertical black line is positioned at target display onset, the vertical blue line at peripheral cue display onset, and the orange line in the bottom panels at central cue display onset. The thin vertical lines in the survival plots are positioned at the estimated median RTs. Error bars represent ± 1 standard error of the respective proportion.

Figure 14. Descriptive statistics for Experiment 2, participant 2. Same conventions as in Figure 13.

Figure 15. Cueing effects (valid minus invalid) based on aggregated data in Experiment 2. (A,B) Cueing effects in $h(t)$ for each ISI combination when the central cue is absent (A) or present (B). (C,D) Cueing effects in $ca(t)$ for each ISI combination when the central cue is absent (C) or present (D). The grey area covering time segment (160,280] is shifted with +20 ms. Error bars indicate the 95% confidence interval for a difference between two proportions.

Figure 16. Model-based hazard and conditional accuracy functions in Experiment 2, as a function of ISI combination (columns), peripheral cue type (see legend), and central cue presence (top versus bottom panels), for block 25. VA, valid; IN, invalid; NC, no peripheral cue.

Figure 17. Model-based hazard and conditional accuracy functions in Experiment 2 for block 1.

Same conventions as in Figure 16.

Figure 18. Model-based hazard and conditional accuracy functions in Experiment 2 for block 50.

Same conventions as in Figure 16.

Figures

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6

Figure 7

Figure 8

Figure 9

Figure 10

Figure 11

Figure 12

Figure 13

Figure 14

Figure 15

Figure 16

Figure 17

Figure 18