Research Article

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How can we learn what attention is? Response gating via multiple direct routes kept in check by inhibitory control processes

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Abstract: To explore the time course of space- and object-based attentional selection processes I analysed the shapes of the response time (RT) and accuracy distributions of left/right arrow identification responses in the two-rectangle paradigm. After cueing one of the four ends of two horizontally or vertically oriented rectangles the arrow typically appears at the cued location (valid), or sometimes at an uncued location in the same (invalid-same) or other rectangle (invalid-different). The data point to a multiple-route model in which (a) an informative cue generates response channel activation before arrow signals emerge, (b) the task-irrelevant arrow location is represented in multiple egocentric and allocentric reference frames around 150 ms after target onset, with the former including a reference frame centered on the currently attended location, (c) the task-irrelevant spatial codes activate premature response tendencies that are actively inhibited to allow gating of arrow direction signals, (d) after an invalid cue the onset of the arrow triggers an “attention shift” – acting between 150 and 240 ms after target onset – that strongly interferes with task performance in certain conditions (invalid-same cueing with horizontal rectangles, and invalid-different cueing with vertical rectangles), and (e) participants differ in which task-irrelevant codes they preferentially inhibit. These results pave the way for future confirmatory studies to temporally characterize and disentangle the contributions of different types of response channel activation processes, from those of reactive cognitive control processes including active and selective response suppression.

Keywords: Simon effect, space-based attention, object-based attention, response times, event history analysis, hazard function, speed-accuracy tradeoff, inter-individual differences, spatial compatibility effects

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Introduction

Research on attention is ubiquitous since James (1890) claimed that “everyone knows what attention is”. Attention has been defined as “selection from available, competing environmental and internal stimuli, of specific information for conscious processing” (Posner & Rafal, 1987, p. 183). It is a multidimensional construct because it refers to a set of cognitive mechanisms used to select information to guide voluntary behavior. One dimension concerns the type of selection bias: (a) selection of task-relevant information based on current goals (top-down, endogenous, or goal-driven control), (b) reflexively orienting to physically salient stimulation (bottom-up, exogenous, or stimulus-driven), and (c) the ever-present, lingering memory effects of past selection episodes (e.g., reward and feature selection history, statistical regularities, and other inter-trial effects; Awh, Belopolsky, & Theeuwes, 2012). Another dimension concerns the content of selection: attention can select locations (space-based), surface features such as color and shape

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(feature-based), time segments (temporal attention), whole objects (object-based), and response features (motor-based attention, or intention). Finally, attentional selection seems to involve not only attentional facilitation or gating of task-relevant information, but also attentional suppression or inhibition of task-irrelevant information (Gaspelin & Luck, 2018; Geng, 2014; Houghton & Tipper, 1996; Howard, Johnson, & Pascual-Leone, 2014; MacLeod, 2007; Moher & Egeth, 2012; Neumann & Descheppe, 1992; Noreen & MacLeod, 2015; Sawaki, Geng, & Luck, 2012; Slotnick, Schwarzbach, & Yantis, 2003; Weiß, Hilkenmeier, & Scharlau, 2013). Emphasizing how these multifaceted functions of attention are embedded in a context of goal-directed motor actions, Allport (1989) characterized attention as “selection-for-action”.

However, Hommel, Chapman, Cisek, Neyedli, Song, and Welsh (2019) recently claimed that “no one knows what attention is” (p. 2288). They argue that the concept of attention is one of the most misleading and misused terms in the cognitive sciences and propose that we should instead focus on behaviorally relevant selection processes and the many neural systems that implement them. Their claim is part of a recent trend in the literature to start reinterpreting attentional studies (Awh, et al., 2012; Panis & Schmidt, 2020; Yaron & Lamy, 2020).

For example, Panis and Schmidt (2020) revisited the classic non-informative spatial cueing paradigm of Posner and Cohen (1984) who discovered the inhibition-of-return effect with longer cue-target onset asynchronies: slower target detection responses when the non-informative cue was spatially valid compared to invalid. As shown in Figure 1A, Panis and Schmidt (2020) presented a peripheral spatial cue that could be valid, invalid, or absent, followed by a central cue display that did or did not contain a central cue, and finally a peripheral target which participants had to localize by pressing one of two buttons. Each inter-stimulus interval (ISI) could last 47 or 141 ms. Panis and Schmidt (2020) analysed the response time (RT) distributions using discrete-time event history analysis and the accuracy distributions using conditional accuracy analysis (Panis & Hermens, 2014; Panis, Torfs, Gillebert, Wagemans, & Humphreys, 2017; Panis & Wagemans, 2009; Wolkersdorfer, Panis, & Schmidt, 2020). The results of a descriptive analysis of the data from one representative participant are shown in Figure 1B.

The blue, orange, and black vertical lines in Figure 1B indicate peripheral cue onset, central cue onset, and target onset, respectively. Time is divided in bins of 40 ms and I will index them by their endpoint – bin “200” refers to bin (160,200], for example. The shape of the RT distribution in each condition is described by two functions: the sample-based estimates of the discrete-time hazard and survivor functions. The hazard function of response occurrence, or $h(t) = P(T = t | T \geq t)$, gives the conditional probability that a response occurs in bin t given that it has not yet occurred in past bins. The survivor function, $S(t) = P(T > t)$, gives the probability that the response does not occur before the end of bin t . The survivor function is the complement of the cumulative distribution function and $S(t)_{.50}$ is the estimated median RT (see the extra vertical lines in the $S(t)$ plots). The shape of the accuracy distribution is described by the micro-level speed-accuracy tradeoff function, a.k.a. the discrete-time conditional accuracy function, or $ca(t) = P(\text{correct} | T = t)$. Four features should be noticed.

First, the fastest responses in the peripheral cue-present conditions emerge time-locked to peripheral cue onset (compare columns in Figure 1B). 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 and sometimes triggers overt responses.

Second, when the central cue is absent (top panels) the hazard of response occurrence 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. Both effects suggest 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 appear.

Third, when the central cue is present (bottom panels), however, the hazard function displays a temporary peak value after the fastest responses, while the conditional accuracy functions approach .5 and even cross, 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. Panis and Schmidt (2020) reasoned that the location of a stimulus is coded in different spatial reference frames (Baess, Weber, & Bermeitinger, 2018; Li, Liu, Li, Weidner, Fink, & Chen, 2019; Wang, Liu, Zou, Li, Zeng, Chen, & Chen, 2016), one of which is centered on the current focus of spatial attention (Hommel, 1993; Nicoletti & Umiltà, 1989; Stoffer, 1991). Thus, after the salient peripheral cue has attracted attention exogenously then the central cue will occupy a left or right position relative to the attentional focus when it appears on the screen. This spatial code will then lead to compatible response channel activation.

Figure 1: Uninformative cueing study of Panis and Schmidt (2020). (A) Trial design in Experiment 2 in Panis and Schmidt (2020). TRTO: time relative to target onset in milliseconds. (B) Descriptive statistics of event history analysis ($h(t)$ and $S(t)$ functions) extended with conditional accuracy functions for the results of a single participant of Experiment 2 in Panis and Schmidt (2020). The vertical black line is positioned at target display onset, the vertical blue line at peripheral cue display onset, and the orange line at central cue display onset. The extra vertical lines in the survival plots are positioned at the estimated median RTs for the valid (green), invalid (red), and no cueing (black) conditions. Error bars represent ± 1 standard error of the respective proportion.

In sum, the peripheral cue activates the spatially compatible response channel (e.g., left) which is then quickly and selectively inhibited, and the central cue then bottom-up activates 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.

Fourth, around 200 ms after target onset all hazard functions increase steadily and all $ca(t)$ functions rise to 1, indicating the emergence of task-relevant target signals. Inhibition-of-return appears in the hazard functions around this time for each ISI combination, is gone after about 120 ms, and is stronger when the central cue is present. Panis and Schmidt (2020) concluded that mean performance measures conceal crucial information about behavioral dynamics,

and that inhibition-of-return is the direct result of encoding the cue location in spatial working memory to promote change detection, instead of spatial attention leaving an inhibitory tag to promote visual search.

Current study

The current study is motivated by three goals. First, I want to follow up on the study of Panis and Schmidt (2020) by studying the role of an *informative* cue for the time-dispersed behavior of participants using discrete-time hazard functions of response occurrence (Allison, 1982, 2010; Austin, 2017; Singer & Willett, 1991, 2003; Willett & Singer, 1993), extended with discrete-time conditional accuracy functions (Pachella, 1974; van Maanen, Katsimpokis, & van Campen, 2019; Wickelgren, 1977). Event history analysis (EHA), a.k.a. survival, hazard, duration, transition, and failure-time analysis, is the standard set of statistical methods for studying the occurrence and timing of events in many scientific disciplines (Allison, 2010; Singer & Willett, 2003). Examples of time-to-event or survival data include RT data, saccade latencies, fixation durations, time-to-force-threshold data, perceptual dominance durations when viewing a bi-stable stimulus, neural inter-spike durations, etc. In general, to apply EHA one must be able to define the event-of-interest (any qualitative change that can be situated in time; here: a button-press response), to define time point zero (here: target onset), and to measure the passage of time between time zero and event occurrence in continuous or discrete units (here: discrete time bins). 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.

Second, I want to explore the within-trial time course of spatial cueing effects in the famous two-rectangle paradigm introduced by Egly, Driver, and Rafal (1994) to investigate the deployment of space- and object-based attention within the same experiment (Mozer & Vecera, 2005). In this paradigm two horizontally or vertically oriented rectangles are presented that flank the fixation cross at each side (see Figure 2). Then an informative spatial cue is shortly presented at one of the four ends of the rectangles. Finally, the target is presented at one of three locations: the cued location on 75% of the trials (the valid condition), the uncued end of the cued rectangle on 12.5% of the trials (the invalid-same condition), or the uncued equidistant end of the uncued rectangle on 12.5% of the trials (the invalid-different condition). Participants have to detect the target as fast as possible by pressing a single button. The typical result in terms of mean RT is that (a) participants are faster to detect the target at the cued location than at either of the uncued locations – a space-based effect –, and (b) participants are faster to detect the target in the invalid-same condition than in the invalid-different condition – an object-based effect (for a review, see Chen, 2012).

There is a broad consensus that the former space-based effect is the result of the informative cue summoning spatial attention before target onset, which then facilitates target processing compared to a cue-absent condition (Posner, 1980). However, there is less consensus about how to interpret the latter object-based effect. For example, Egly et al. (1994) suggested that the object-based effect is the result of attention spreading more quickly to other locations within the same object than between different objects. However, Brown and Denney (2007) argued that the object advantage is mainly due to disengage operations associated with object-based deployment of attention. Lamy and Egeth (2002) confirmed that object-based effects are only obtained when the task requires shifts of attention. Interestingly, object-based effects are more prevalent for horizontal rectangles, which is in accordance with the theory that attention may be allocated more easily along the horizontal meridian (Hein, Blaschke, & Rolke, 2017).

Can we distinguish between these attentional-spreading and attentional-disengagement hypotheses using a distributional analysis? The attention-spreading hypothesis predicts that the invalid-same condition will at some point in time lead to faster response occurrence – that is, a higher $h(t)$ estimate – compared to invalid-different. This point in time can be early or late, depending on the speed with which attention spreads. The attentional-disengagement hypothesis predicts that the emergence of target signals – indexed here by spatial compatibility effects (see below) – is delayed in the invalid-different condition compared to the invalid-same condition.

Third, I want to study potential inter-individual differences in behavior. For example, Pilz, Roggeveen, Creighton, Bennett, and Sekuler (2012) noted that object-based effects are typically small and found less consistently across experiments compared to space-based effects. Furthermore, they showed that object-based effects in mean RT are present in only a small subset of participants, and that they reverse into a negative object-based effect in another small subset of participants. When exactly do these reversals occur during a trial, and what is special about such participants?

Figure 2: Trial design. An example trial of condition “VA-C-V” is shown: a *valid* cue, a *compatible* relation between target location relative to the fixation cross and target direction (both “LEFT”), and *vertically* oriented rectangles.

In order to answer these questions I changed two features of the original paradigm of Egly et al. (1994). First, a cue-absent condition was included to measure the effects of the three cue types relative to a common baseline. Second, instead of a target detection task I used a left/right target arrow direction identification task in order to be able to analyse not only the hazard functions but also the conditional accuracy functions, and to be able to observe a positive spatial compatibility effect between the egocentric left/right target arrow location and effector location – or arrow direction (the Simon effect; Simon & Wolf, 1963; Simon & Rudell, 1967; Simon, 1969; for a review, see Lu and Proctor, 1995), which will index the availability of target direction information (Yaron & Lamy, 2020). A small-*N* design was employed with ten participants (Smith & Little, 2018). In contrast to the mean performance measures, the distributional results reveal shortcomings of the attentional interpretations of the space- and object-based effects, and point to a multiple-route model of response activation kept in check by inhibitory control processes.

Methods

Participants

Ten right-handed students (3 males) participated voluntarily in exchange for course credits. They had normal or corrected-to-normal vision. Their average age was 25.5 years (range: 22-36). 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. An RT box with a built-in microprocessor was used to record the RT and button identity independently from the Windows operating system (Suzhou Litong Electronic CO., LTD; <https://lobes.osu.edu/rt-box.php>). PsychToolbox (<http://psychtoolbox.org/>) and Matlab (<https://www.mathworks.com>) were used to generate the stimuli, control the timing of stimulus presentation, read-out the response box at the end of the data collection period, and save the data set.

Stimuli

A central small black fixation cross (.2 x .2 visual degrees) was flanked by two light grey rectangle-shaped surfaces that were oriented horizontally or vertically. Each rectangle measured 8.4 x 2.1 visual degrees. The background was white. The horizontal distance from the center to the inner long edge of each rectangle measured 2.1 visual degrees. The black cue consisted of a u-shaped edge located at one end of one of the rectangles. Each side of the cue measured 2.6 degrees and the thickness of the cue was .3 degrees. The black target was a double arrow pointing to the left or right (1 x 1 visual degrees). The distance from target center to fixation was always 4.4 degrees.

Experimental design

In 89% of all trials a cue appeared shortly at one of the four ends of the (vertical or horizontal) rectangles (Figure 2). In 75% of cued trials the cue was spatially valid (condition “valid” or VA) and the target appeared at the cued location. In the invalid cue conditions the target either appeared at the other end of the cued rectangle (condition “invalid-same” or IS; 12.5% of cued trials) or at one end of the uncued rectangle (condition “invalid-different” or ID; 12.5% of cued trials). The cue-target stimulus-onset-asynchrony (SOA) was 150 ms. I included trials without a cue to study the within-trial time course of the cueing effects relative to a common baseline (condition “no cue” or NC; 11% of all trials).

There were 16 experimental conditions or trial-types resulting from combining the factors Cue Type (VA, IS, ID, NC), Compatibility between the egocentric left/right location of the target relative to fixation and the left/right target arrow direction or correct response button (compatible or C, incompatible or I), and Orientation (horizontal or H, vertical or V). The VA trials were presented six times more frequently than the IS, ID, and NC trials. Thus, after each series of 144 trials (or 4 blocks; see Procedure) all possible combinations of cue location (left-up, left-down, right-up, right-down), orientation (horizontal, vertical), target direction (left, right), and cue type (VA, IS, ID, NC) were presented with the correct relative frequencies.

Procedure

Participants performed two sessions separated by a break of about 15 minutes. Each session contained one practice block and 48 experimental blocks. Each block contained 36 trials. The order of trials was randomized within each block. As shown in Figure 2 each trial began with a fixation display presented for ~354 ms (30 frames). Next the first object display was presented for ~142 ms (12 frames) which contained the fixation cross and both rectangles. The cue display was presented for ~47 ms (4 frames). The second object display was presented for ~106 ms (9 frames). The target display was presented for ~94 ms (8 frames). The third object display was presented for ~506 ms (43 frames).

Participants were seated in front of the computer, and 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 cue and objects, and to respond quickly (within 600 ms) and accurately in each trial to the direction of the double arrow target (left or right) by pressing the corresponding (left or right) button with the index finger of the corresponding (left or right) hand. In each trial the data collection period lasted 600 ms and started at target onset. 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” or “error” was displayed; if no response was detected “too slow” was displayed. The next trial started automatically after the blank screen. After each block, summary information was provided including the number of errors, percentage accuracy, and the mean correct RT. Participants could start the next block by pressing one of the two buttons.

Statistical analyses

In the first step, a number of trials had to be discarded due to technical or timing issues. First, due to technical errors in session one, nine blocks were lost for one participant, and one block and the last three trials of another block for another participant, leaving 34,197 trials (instead of 34,560 trials). Second, 147 trials were discarded because the

observed RT was negative or zero (leaving 34,050 trials). Third, 123 trials were deleted because the actual first-object display duration was longer than 200 ms. Fourth, 408 trials were deleted because the actual cue-target SOA was longer than 220 ms. Fifth, 44 trials were deleted because the actual target duration was longer than 120 ms. After applying these selection criteria there were 33,475 trials left for the analyses.

In the second step, the classic mean performance measures (error rate and mean correct RT) were analyzed using repeated-measures ANOVAs. The Greenhouse-Geisser correction was applied if Mauchly's test for sphericity was significant. All significant effects are reported.

In the third step, I used R (R Core Team, 2014) to set up life tables (see Table 1) in order to calculate the descriptive statistics provided by discrete-time EHA. The hazard function of response occurrence is one of the most diagnostic functions when describing the distribution of a sample of (right-censored) RT data (Luce, 1986; Townsend, 1990)¹. The discrete-time hazard function, $h(t) = P(T = t \mid T \geq t)$, represents for each bin 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 .

I used time bins with a width of 30 ms up until 360 ms after target onset, followed by three bins with a width of 40 ms, and ending with two bins of 60 ms. Because only a few responses occur in the late bins, using only 30 ms bins would result in highly unstable hazard estimates for the late bins. While the starting point is not part of the interval, the endpoint is – the first and last bins are (0,30] and (540,600], respectively, for example.

When I plotted the bin-specific differences in hazard between two conditions – $\Delta h(t)$ plots – I also plotted the approximate 95% confidence interval for the difference between two proportions, based on the estimated standard error: $se[h_1(t) - h_2(t)] = \text{square root of } [\{h_1(t) \cdot [1 - h_1(t)] / RS_1(t)\} + \{h_2(t) \cdot [1 - h_2(t)] / RS_2(t)\}]$, where $RS(t)$ refers to the size of the risk set in bin t .

A second useful function is the survivor function, or $S(t) = P(T > t) = [1 - h(t)] \cdot [1 - h(t-1)] \cdot \dots \cdot [1 - h(1)]$. The survivor function is the complement of the cumulative distribution function, $S(t) = 1 - F(t) = 1 - P(T \leq t)$, 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.

I 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 (see Table 1; Pachella, 1974; Wickelgren, 1977). When I plotted the bin-specific differences in $ca(t)$ between two conditions – $\Delta ca(t)$ plots – I also plotted the approximate 95% confidence interval for the difference between two proportions, based on the estimated standard error: $se[ca_1(t) - ca_2(t)] = \text{square root of } [\{ca_1(t) \cdot [1 - ca_1(t)] / E_1(t)\} + \{ca_2(t) \cdot [1 - ca_2(t)] / E_2(t)\}]$, where $E(t)$ refers to the number of observed responses in bin t .

Finally, to test *whether and when* the main and interaction effects including cue type, rectangle orientation, and spatial compatibility are significant across participants, I fitted discrete-time hazard and conditional accuracy 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 follows²:

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

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 effects of the predictors, the parameter estimates are exponentiated, resulting in a hazard ratio. In the case of a small- N design with repeated measures, 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).

Table 1: Life table for condition NC. Data aggregated across the four combinations of rectangle orientation and compatibility, and participants (N=10).

time bin	t	cens	E	RS	$h(t)$	$se[h(t)]$	$S(t)$	$se[S(t)]$	$P(t)$	$se[P(t)]$	NrCorr	$ca(t)$	$se[ca(t)]$
(0,30]	1	0	0	3731	0	0	1	0	0	0	0	NA	NA
(30,60]	2	0	0	3731	0	0	1	0	0	0	0	NA	NA
(60,90]	3	0	1	3731	0,00027	0,00027	0,99973	0,00027	0,00027	0,00027	0	0	0
(90,120]	4	0	0	3730	0,00000	0,00000	0,99973	0,00027	0,00000	0,00000	0	NA	NA
(120,150]	5	0	2	3730	0,00054	0,00038	0,99920	0,00046	0,00054	0,00038	0	0	0
(150,180]	6	0	1	3728	0,00027	0,00027	0,99893	0,00054	0,00027	0,00027	0	0	0
(180,210]	7	0	7	3727	0,00188	0,00071	0,99705	0,00089	0,00188	0,00071	4	0,571	0,187
(210,240]	8	0	49	3720	0,01317	0,00187	0,98392	0,00206	0,01313	0,00186	33	0,673	0,067
(240,270]	9	0	181	3671	0,04931	0,00357	0,93541	0,00400	0,04851	0,00352	152	0,840	0,027
(270,300]	10	0	417	3490	0,11948	0,00549	0,82364	0,00609	0,11177	0,00516	391	0,938	0,012
(300,330]	11	0	592	3073	0,19265	0,00711	0,66497	0,00724	0,15867	0,00598	579	0,978	0,006
(330,360]	12	0	652	2481	0,26280	0,00884	0,49022	0,00717	0,17475	0,00622	634	0,972	0,006
(360,400]	13	0	828	1829	0,45271	0,01164	0,26829	0,00560	0,22192	0,00680	819	0,989	0,004
(400,440]	14	0	496	1001	0,49550	0,01580	0,13535	0,00358	0,13294	0,00556	482	0,972	0,007
(440,480]	15	0	287	505	0,56832	0,02204	0,05843	0,00190	0,07692	0,00436	282	0,983	0,008
(480,540]	16	0	128	218	0,58716	0,03335	0,02412	0,00091	0,03431	0,00298	122	0,953	0,019
(540,600]	17	45	45	90	0,50000	0,05270	0,01206	0,00050	0,01206	0,00179	45	1	0

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 $S(t) = P(T > t) = [1 - h(t)] * [1 - h(t-1)] * \dots * [1 - h(1)]$, and the probability mass function $P(t) = P(T = t) = h(t) * S(t-1)$. The standard errors (se) for $h(t)$, $P(t)$, and $ca(t)$ are estimated using the familiar formula for a proportion p – the square root of $\{p(1-p) / N\}$ – where N equals $RS(t)$ for $h(t)$, $RS(1)$ for $P(t)$, and $E(t)$ for $ca(t)$. 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. Forty-five trials are right-censored (“cens”) at 600 ms after target onset (column 3), i.e., no response occurred for these trials during the 600 ms data collection period, so that we only know that $RT > 600$ ms; Therefore, $h(t)$ does not reach 1, $S(t)$ does not reach 0, and the $P(t)$ estimates do not sum to 1 – hence the name *subprobability* mass function (Chechile, 2003). At time point zero, $S(0) = 1$, $P(0) = 0$, and $h(0)$ is undefined. It is important to realize the difference between the (sub)probability mass and the hazard function. The (sub)probability mass function calculates the response count in a given time bin relative to the number of all trials, whereas the hazard function calculates the response count relative to the number of all trials that are still response-free up to the start of that time bin.

I proceeded as follows for the current data set. First, I 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. I selected a time range where all participants provided enough data in each of the 16 conditions, and followed the general advice to create between 10 and 20 bins for modeling purposes (Singer & Willett, 2003). I selected the time range (150,480], starting with 7 bins of 30 ms and ending with 3 bins of 40 ms (cfr. Table 1). Thus, all trials with a $RT \leq 150$ ms were discarded, and all trials were right-censored at 480 ms.

Second, the condition “IS-C-H” (invalid-same cueing with a compatible target and horizontal rectangles) was chosen as the baseline condition. The main continuous predictor variable TIME was the time bin rank centered on bin (270,300], the reference bin during model selection. 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 participant³. As well as dummy-coding the relevant levels of the experimental factors (VA, ID, NC, I, V) I also included the continuous

Table 2: Selected cloglog-hazard model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	se	z	p	PE	p	PE	p	p
1	(Intercept)	-4,00E+00	< 2e-16 ***		-3,11E+00	< 2e-16 ***		-2,32E+00	1,86E-01	-12,47	< 2e-16 ***	-1,40E+00	< 2e-16 ***	-5,06E-01	4,33e-06 ***	
2	TIME							3,94E-01	6,07E-02	6,48	8,81e-11 ***					
3	TIME ²							1,80E-02	8,90E-03	2,02	0,042684 *					
4	TIME ³							9,97E-03	2,55E-03	3,9	9,31e-05 ***					
5	TIME ⁴							-8,86E-04	3,05E-04	-2,9	0,003646 **					
6	TIME ⁵							-4,55E-04	7,67E-05	-5,93	2,94e-09 ***					
7	IC	-4,25E-01	0,065710 .		4,82E-01	5,05e-07 ***		7,85E-01	7,46E-02	10,52	< 2e-16 ***	6,85E-01	< 2e-16 ***	3,82E-01	3,12e-05 ***	
8	TIME:IC							3,40E-02	3,44E-02	0,98	0,322747					
9	TIME ² :IC							-5,04E-02	1,01E-02	-4,99	5,95e-07 ***					
10	TIME ³ :IC							4,18E-03	2,36E-03	1,77	0,076703 .					
11	Ve	-1,59E+00	1,24e-07 ***		-9,88E-02	0,366562		6,49E-01	7,69E-02	8,43	< 2e-16 ***	7,66E-01	< 2e-16 ***	3,69E-01	2,31e-05 ***	
12	TIME:Ve							2,07E-01	3,81E-02	5,42	5,75e-08 ***					
13	TIME ² :Ve							-7,87E-02	1,20E-02	-6,53	6,42e-11 ***					
14	TIME ³ :Ve							2,38E-03	2,88E-03	0,82	0,408676					
15	Ve:IC	-4,28E-01	0,323403		-8,22E-01	8,90e-08 ***		-1,06E+00	1,02E-01	-10,4	< 2e-16 ***	-1,03E+00	< 2e-16 ***	-5,90E-01	3,21e-06 ***	
16	TIME:Ve:IC							-6,15E-02	4,77E-02	-1,28	0,197384					
17	TIME ² :Ve:IC							3,46E-02	1,67E-02	2,07	0,038002 *					

continued
Table 2: Selected cloglog-hazard model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	se	z	p	PE	p	PE	p	p
18	TIME ³ :Ve:IC				2,58E-03		3,34E-03	0,77			0,440558					
19	ID	-7,05E-01	0,005676 **		-2,08E-01	0,055234	2,86E-01	7,87E-02	3,64		0,000273 ***	4,87E-01	2,65e-13 ***	1,04E-01	0,210315	
20	TIME:ID				1,98E-01		3,77E-02	5,25			1,50e-07 ***					
21	TIME ² :ID				-3,67E-02		1,06E-02	-3,45			0,000559 ***					
22	TIME ³ :ID				-6,04E-03		2,47E-03	-2,45			0,014304 *					
23	ID:IC	8,20E-01	0,008109 **		-7,18E-01	6,80e-07 ***	1,04E-01	-13,91			< 2e-16 ***	-1,38E+00	< 2e-16 ***	-5,05E-01	3,61e-05 ***	
24	TIME:ID:IC				-1,66E-01		4,04E-02	-4,09			4,23e-05 ***					
25	TIME ² :ID:IC				1,01E-01		1,25E-02	8,05			8,27e-16 ***					
26	ID:Ve	1,74E+00	8,50e-06 ***		3,77E-01	0,014717 *	-8,54E-01	1,08E-01	-7,9		2,66e-15 ***	-1,37E+00	< 2e-16 ***	-5,87E-01	1,04e-06 ***	
27	TIME:ID:Ve				-4,84E-01		5,31E-02	-9,12			< 2e-16 ***					
28	TIME ² :ID:Ve				8,95E-02		1,58E-02	5,67			1,39e-08 ***					
29	TIME ³ :ID:Ve				1,21E-02		3,56E-03	3,39			0,000685 ***					
30	ID:Ve:IC	-5,48E-01	0,245365		1,83E+00	< 2e-16 ***	2,87E+00	1,40E-01	20,4		< 2e-16 ***	2,55E+00	< 2e-16 ***	8,97E-01	5,75e-07 ***	
31	TIME:ID:Ve:IC				1,81E-01		6,13E-02	2,94			0,003247 **					
32	TIME ² :ID:Ve:IC				-1,68E-01		1,82E-02	-9,24			< 2e-16 ***					
33	NC	-5,61E+00	< 2e-16 ***		-1,24E+00	8,44e-13 ***	3,90E-01	8,06E-02	4,83		1,33e-06 ***	4,70E-01	3,68e-13 ***	1,83E-01	0,020057 *	

continued **Table 2:** Selected cloglog-hazard model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	p		PE	p		PE	p	
34	TIME:NC							3,29E-01		4,67E-02	7,04		1,82e-12			

35	TIME:NC							-1,94E-01		1,98E-02	-9,79		< 2e-16		***	
36	TIME:NC							2,47E-02		3,67E-03	6,72		1,81e-11			

37	NC:IC	-1,79E-01	0,831571		-1,54E+00	3,07e-09	***	-1,52E+00		1,17E-01	-12,94		< 2e-16	***	-3,32E-01	0,004848
													< 2e-16	***	< 2e-16	**
38	TIME:NC:IC							2,29E-01		6,31E-02	3,62		0,000286			

39	TIME:NC:IC							7,90E-02		2,77E-02	2,84		0,004388			
													**			
40	TIME:NC:IC							-1,55E-02		5,06E-03	-3,06		0,002208			
													**			
41	VA	3,15E-01	0,068628		1,07E+00	< 2e-16	***	9,92E-01		6,29E-02	15,76		< 2e-16	***	5,37E-01	0,006708
													< 2e-16	***	1,68E-01	**
42	TIME:VA							-1,72E-01		3,07E-02	-5,59		2,22e-08			

43	TIME:VA							-4,69E-02		7,88E-03	-5,95		2,62e-09			

44	TIME:VA							9,58E-03		2,19E-03	4,38		1,17e-05			

45	VA:IC	6,84E-01	0,005148		-9,61E-01	< 2e-16	***	-1,27E+00		8,01E-02	-15,82		< 2e-16	***	-8,34E-01	0,010345
			**										< 2e-16	***	-2,58E-01	*
46	TIME:VA:IC							8,14E-02		3,85E-02	2,11		0,034599		*	
47	TIME:VA:IC							9,26E-02		1,08E-02	8,56		< 2e-16		***	
48	TIME:VA:IC							-1,24E-02		2,70E-03	-4,6		4,17e-06			

Continued
Table 2: Selected cloglog-hazard model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	se	z	p	PE	p	PE	p	p
49	NC:Ve	1,37E+00	0,002724 **		1,58E-01	0,459122		-5,19E-02	9,99E-02	-5,19	2,10e-07 ***	-6,56E-01	2,30e-15 ***	-2,56E-01	0,030370 *	
50	TIME:NC:Ve							-2,04E-01	5,94E-02	-3,42	0,000608 ***					
51	TIME ² :NC:Ve							6,73E-02	1,39E-02	4,85	1,22e-06 ***					
52	NC:Ve:IC	2,04E+00	1,36e-08 ***		1,63E+00	6,08e-11 ***		1,22E+00	1,51E-01	8,09	5,53e-16 ***	8,11E-01	1,91e-15 ***	4,02E-01	0,011950 *	
53	TIME:NC:Ve:IC							-2,04E-01	5,83E-02	-3,5	0,000455 ***					
54	VA:Ve	-1,41E-01	0,663835		-8,05E-01	3,91e-12 ***		-8,37E-01	8,19E-02	-10,21	< 2e-16 ***	-4,86E-01	1,75e-11 ***	-1,21E-03	0,990137	
55	TIME:VA:Ve							1,01E-01	4,20E-02	2,39	0,016706 *					
56	TIME ² :VA:Ve							4,79E-02	1,31E-02	3,66	0,000246 ***					
57	TIME ³ :VA:Ve							-5,19E-03	3,26E-03	-1,59	0,111754					
58	VA:Ve:IC	-3,70E-02	0,937095		1,26E+00	1,70e-14 ***		1,48E+00	1,10E-01	13,45	< 2e-16 ***	1,03E+00	< 2e-16 ***	2,78E-01	0,047085 *	
59	TIME:VA:Ve:IC							-8,99E-02	5,38E-02	-1,67	0,094987 .					
60	TIME ² :VA:Ve:IC							-8,52E-02	1,82E-02	-4,67	3,00e-06 ***					
61	TIME ³ :VA:Ve:IC							8,08E-03	3,98E-03	2,02	0,042494 *					
62	TRIAL	-1,12E-02	1,92e-15 ***		-1,01E-02	< 2e-16 ***		-8,98E-03	6,17E-04	-14,54	< 2e-16 ***	-7,88E-03	< 2e-16 ***	-6,79E-03	3,54e-13 ***	
63	TIME:TRIAL							5,48E-04	2,54E-04	2,15	0,031076 *					
64	TRIAL ²	3,99E-03	1,54e-11 ***		3,99E-03	1,54e-11 ***		3,99E-03	5,91E-04	6,74	1,49e-11 ***	3,99E-03	1,54e-11 ***	3,99E-03	1,53e-11 ***	
65	BLOCK	-4,01E-03	0,112669		-2,02E-03	0,064092 .		-5,31E-04	8,00E-04	-0,66	0,50674	4,40E-04	0,553642	8,98E-04	0,446664	

Continued **Table 2:** Selected cloglog-hazard model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	se	z	p	PE	p	PE	p	p
66	TIME:BLOCK				6,14E-04		3,28E-04	1,87			0,060842 .					
67	TIME ² :BLOCK				-6,42E-05		1,10E-04	-0,58			0,560495					
68	BLOCK ²	-3,80E-03	0,008545 **		-1,10E-03	0,077302 .	4,56E-04	1,06			0,285526	9,71E-04	0,020610 *	3,50E-04	0,58949	
69	TIME:BLOCK ²				5,18E-04		1,87E-04	2,77			0,005461 **					
70	TIME ² :BLOCK ²				-1,38E-04		6,21E-05	-2,22			0,026124 *					
71	TRIAL:BLOCK	-2,33E-04	0,022986 *		-1,43E-04	0,040337 *	4,47E-05	-1,19			0,233686	3,64E-05	0,401444	1,26E-04	0,060835 .	
72					4,48E-05		1,85E-05	2,42			0,015191 *					
73	ssc	-1,35E-01	0,005588 **		-1,47E-01	4,27e-12 ***	1,56E-02	-9,03			< 2e-16 ***	-1,19E-01	< 2e-16 ***	-7,97E-02	0,000321 ***	
74	TIME:ssc				6,96E-03		6,32E-03	1,1			0,271163					
75	TIME ² :ssc				2,09E-03		2,11E-03	0,99			0,321215					
76	BLOCK:ssc	1,22E-02	0,000704 ***		3,52E-03	0,023505 *	1,13E-03	-1,89			0,057969 .	-4,79E-03	4,20e-06 ***	-4,43E-03	0,005843 **	
77					-2,08E-03		4,64E-04	-4,47			7,73e-06 ***					
78					3,76E-04		1,54E-04	2,43			0,014750 *					
RANDOM																
79	intercept vari- ance	1,42405			0,75296							0,08439		0,08758		
80	TIME variance	0,02811			0,02812							0,02812		0,02812		
81	correlation	-0,98			-0,96							-0,56		0,58		

Note. The model was centered on reference bin (270, 300] during model selection. The selected model was refitted three times with TIME centered each time on another reference bin: (150, 180], (210, 240], (330, 360], and (400, 440]. Parameter estimates (PE) are in cloglog-hazard units.

predictors trial-within-block number (TRIAL) and block-within-session number (BLOCK), and the dichotomous variable SESSION to model across-trial, -block, and -session changes in the hazard of response occurrence, due to synaptic learning processes (Schöner, Spencer, et al., 2016) or proactive cognitive control processes (Braver, 2012), for example, that play out on these longer time scales. TRIAL was centered on value 18, BLOCK on value 25, and SESSION on value 1. Thus, with all effects set to zero, the cloglog-hazard model's intercept refers to the estimated $\text{cloglog}[h(300)]$ in trial 18 of block 25 of session 1 when a compatible target is invalidly-same cued with horizontal rectangles (see Table 2, column 7, effect number 1).

Third, to estimate the parameters of the $h(t)$ model, one has to create a dataset where each row corresponds to a time bin of a trial of a participant (a person-trial-bin oriented dataset). 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 centered covariates TIME, TRIAL, BLOCK, and SESSION, the variable PARTICIPANT, and the dummy-coded dichotomous experimental predictor variables (VA, ID, NC, I, V). Thus, each trial without an observed response before 480 ms contributes 10 rows, and each row has a value 0 for OUTCOME. The resulting person-trial-bin oriented data set contained 209,201 rows.

Fourth, I started with a full multi-level cloglog-hazard model (93 fixed parameters; with bins at level 1 nested within participant at level 2) encompassing the following effects at level 1: (a) a 5rd order polynomial for the shape of the cloglog-hazard function in the baseline condition (6 parameters), (b) the effects of valid cueing ('VA'), invalid-different cueing ('ID'), no cueing ('NC'), incompatibility ('IC') and vertical ('Ve') were allowed to interact with time in a cubic fashion (20 parameters), (c) the seven first-order interaction effects involving VA, ID, NC, IC, and Ve could vary over time in a cubic fashion (28 parameters), (d) the three second-order interaction effects involving VA, ID, NC, IC, and Ve could vary cubically over time (12 parameters), (e) the linear and quadratic effects of TRIAL and BLOCK could interact with time in a quadratic fashion (12 parameters), with $\text{TRIAL}^2 = \text{TRIAL} \cdot \text{TRIAL} / 10$, and $\text{BLOCK}^2 = \text{BLOCK} \cdot \text{BLOCK} / 10$, (f) the effect of SESSION could vary quadratically over time (3 parameters), and (g) the interactions TRIAL:BLOCK, TRIAL:SESSION, BLOCK:SESSION, and TRIAL:BLOCK:SESSION could vary quadratically over time (12 parameters).

I used an automatic backward selection procedure to select a final model. 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, I 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 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.

To select a conditional accuracy model I used the same multi-level modeling procedure except that I used the original person-trial oriented data set. Only trials with observed RTs within the time segment (150,480] were included (31,263 trials or rows). I predicted accuracy (1/0) in each bin, and used the symmetric logit link function.

Results

Mean performance measures: error rate and mean correct RT

First, the error rate was calculated for each combination of subject and experimental condition using all observed responses between 0 and 600 ms (Figure 3A). A three-way repeated-measures ANOVA on error rates revealed a significant main effect of Cue Type ($F(3,27) = 27.12, p < .001, \eta^2 = .75$) and Orientation ($F(1,9) = 17.21, p = .002, \eta^2 = .66$), next to significant first-order interactions between Cue Type and Compatibility ($F(1.91,17.19) = 22.29, p < .001, \eta^2 = .71$) and between Cue Type and Orientation ($F(3,27) = 15.11, p < .001, \eta^2 = .63$). The second-order interaction between Cue Type, Compatibility, and Orientation was also significant ($F(1.91,17.17) = 39.05, p < .001, \eta^2 = .81$).

To focus on the elusive object-based effects (Pilz, et al., 2012) I performed four post-hoc pairwise comparisons using a Bonferroni-corrected p -value of $0.05/4 = 0.0125$. For vertical rectangles, there is a significant positive object-based effect of 26 percentage points when the target is compatible ($t(9) = 8.03$, uncorrected $p < .001$) and a significant negative object-based effect of 12 percentage points when the target is incompatible ($t(9) = -4.61$, uncorrected $p = .001$).

Figure 3: Mean performance measures. Error rate (A) and mean correct RT (B) as a function of cue type (VA = valid, IS = invalid-same, ID = invalid-different, NC = no cue), compatibility (C = compatible, I = incompatible), and orientation (H = horizontal, V = vertical). Error bars show +/- 1 standard error of the mean, corrected for between-subject variability.

For horizontal rectangles, there is a significant positive object-based effect of 16 percentage points when the target is incompatible ($t(9) = 6.06$, uncorrected $p < .001$) and a significant negative object-based effect of 18 percentage points when the target is compatible ($t(9) = -6.14$, uncorrected $p < .001$).

Second, the mean correct RT was calculated for each combination of subject and experimental condition using all observed *correct* responses between 0 and 600 ms (Figure 3B). A three-way repeated-measures ANOVA revealed a significant main effect of Cue Type ($F(1.50, 13.54) = 29.87$, $p < .001$, $\eta^2 = .77$) and Orientation ($F(1, 9) = 7.53$, $p = .023$, $\eta^2 = .46$), next to significant first-order interactions between Cue Type and Compatibility ($F(3, 27) = 24.56$, $p < .001$, $\eta^2 = .73$) and between Cue Type and Orientation ($F(3, 27) = 14.00$, $p < .001$, $\eta^2 = .61$). The second-order interaction between Cue Type, Compatibility, and Orientation was also significant ($F(3, 27) = 68.68$, $p < .001$, $\eta^2 = .88$).

The Bonferroni-corrected post-hoc pairwise comparisons revealed the same patterns as in the error rates. For vertical rectangles, there is a significant positive object-based effect of 62 ms when the target is compatible ($t(9) = 10.55$, uncorrected $p < .001$) and a significant negative object-based effect of 52 ms when the target is incompatible ($t(9) = -7.41$, uncorrected $p < .001$). For horizontal rectangles, there is a significant positive object-based effect of 74 ms when the target is incompatible ($t(9) = 14.47$, uncorrected $p < .001$) and a significant negative object-based effect of 30 ms when the target is compatible ($t(9) = -7.72$, uncorrected $p < .001$).

Note that for both performance measures a regular Simon effect or positive compatibility effect is present for each orientation in conditions VA and NC, and in conditions IS-x-V and ID-x-H. Reversed Simon effects or negative compatibility effects are present in conditions IS-x-H and ID-x-V.

Figure 4: Descriptive statistics: Time-varying main effects, aggregated data. (A) Effect of cue type after aggregating across orientations, compatibility levels, and participants. (B) Effect of rectangle orientation after aggregating across cue types, compatibility levels, and participants. (C) Effect of compatibility after aggregating across orientations, cue types, and participants.

Response history and conditional accuracy analyses: Descriptive statistics

To gradually introduce the reader to the informative shapes of the $h(t)$ and $ca(t)$ functions in the 16 conditions, and some interesting inter-individual differences, Figure 4 presents the sample-based estimates of the median RT, hazard, $\delta h(t)$, and $ca(t)$ functions for the main effects of cue type (Figure 4A), rectangle orientation (Figure 4B), and compatibility (Figure 4C), each based on data aggregated across participants and the levels of the other two variables.

Let's focus first on what happens when no cue is presented (condition NC in Figure 4A; Table 1). Imagine traveling with time starting at target onset. Then, for example, if the waiting time has increased to 210 ms without event occurrence, then the conditional probability that the response occurs sometime in bin (210,240] is estimated to be 0.013 (49 observed responses with a risk set equal to 3720). Thus, of all trials that survive to (i.e., are still response-free at)

210 ms, about 1 percent will experience a response during the next 30 ms (i.e., they will “die”, and drop out of the risk set). In short, $h(240) = .013$. Also, if a response occurs in bin (210,240] then the probability that it will be correct equals $.67 = ca(240)$. However, if the waiting time has increased to 300 ms, for example, then $h(330) = .20$ and $ca(330) = .98$.

The hazard function of response occurrence in condition NC thus starts to increase around 240 ms after target onset until it reaches a plateau around 400 ms. While the accuracy of the fastest responses, around 210 ms, is at chance level, most responses emitted after about 300 ms are correct. The thick grey surface area is positioned around those bins that reveal a decrease in response competition in condition NC due to emerging target signals, i.e., bins 240 to 300 (see Figure 4A, bottom panel). It will appear in all forthcoming Figure panels to help with interpreting the shape of the functions.

When a cue is present, hazard starts to increase visibly above 0, around 150 ms after target onset in the aggregated data (Figure 4A, top), which is indicated by the thin grey surface area. Note that the median RTs show the expected space-based (VA faster than IS and ID) and object-based effects (IS faster than ID). The delta- $h(t)$ plot of the cueing effects relative to NC (Figure 4A, middle) shows that the size of the positive cueing effect in bin (120,150] is similar for each cue type. The cueing effects become sensitive to the spatial validity of the cue from bin (180,210] onwards. The space-based effect emerges around 150 ms and lasts about 300 ms (blue shaded area) and the object-based effect emerges around 300 ms and lasts about 120 ms (white shaded area).

Figure 4B shows how hazard in early bins is overall higher for horizontal compared to vertical orientations, and how this effect reverses for later bins. Figure 4C shows that a positive spatial compatibility effect is present in the hazard estimates between 240 and 400 ms. A positive spatial compatibility effect is present in $ca(t)$ for $t < 300$ ms.

The delta- $h(t)$ plots of the cueing effects look compatible at first sight with attentional interpretations in terms of the focusing of space-based attention on the cued location, and the attentional-spreading hypothesis. But why is $ca(t)$ not higher for VA compared to NC in any bin (Figure 4A, bottom), for example? We will soon see that Figure 4 conceals more complex time-varying effects of our experimental manipulations.

Figure 5A presents the time-varying cueing effects separately for horizontal and vertical rectangle orientations. For both orientations the object-based effect now emerges at around 210 ms, and it is positive throughout for horizontal rectangles, but it begins as a negative effect that reverses into a positive one for vertical rectangles. Figure 5B shows the time-varying positive compatibility effect for horizontal (black shaded area) and vertical rectangles (green shaded area). Again, Figure 5 conceals more complex time-varying effects as shown in Figure 6. For horizontal rectangles, there is a small positive object-based effect that reverses into a negative one for compatible targets (Figure 6A, middle), and a large positive object-based effect for incompatible targets (Figure 6B). In contrast, for vertical rectangles, there is a small negative object-based effect that reverses into a positive one for compatible targets (Figure 6C), and a large negative object-based effect for incompatible targets (Figure 6D). While it is difficult – if not impossible – to explain these patterns solely in terms of space- and object-based attentional selection processes, data interpretation becomes easier when the data are plotted differently.

Figure 7 again shows the data for each experimental condition, aggregated across participants, but including the survivor functions and delta- $ca(t)$ plots. I grouped NC-x-H with VA-x-H (column 1), NC-x-V with VA-x-V (column 2), IS-x-H with ID-x-V (column 3), and IS-x-V with ID-x-H (column 4). In condition NC-x-H we observe how a positive compatibility effect (PCE) in $h(t)$ emerges around 240 ms, peaks in bin 330, and then levels off (black and grey lines in Figure 7A). The PCE in $ca(240)$ has a maximal size and is gone for $t > 300$ ms (Figure 7C). More or less the same is happening for vertical orientations (black and grey lines in Figures 7D and F). These data reveal the presence of the Simon effect where responses around 240 ms are still reflecting the task-irrelevant arrow location, while virtually all responses emitted after 300 ms reflect the correct arrow direction.

In condition VA we observe a PCE in $h(t)$ for horizontal rectangles (blue and cyan lines in Figure 7A) and a hint of a tiny PCE for vertical orientations followed by a larger effect around 400 ms (Figure 7D). As we will soon see, this tiny PCE around 270 ms in the aggregated data is due to inter-individual differences. The $ca(t)$ functions show that in condition VA responses are sometimes emitted well before 210 ms (Figures 7C and 7F), and if so they exhibit a non-maximal PCE, again due to inter-individual differences (see below). Nevertheless, these data reveal that the informative cue triggers response channel activation that can lead to overt responses around 60 ms after target onset, that is, 210 ms after cue onset – the time required for the first overt sensorimotor signals to appear in condition NC. And this premature response activation then seems to be selectively inhibited (Panis & Schmidt, 2016), resulting in $ca(t)$ functions approaching chance level just as in Figure 1B (Panis & Schmidt, 2020).

Figure 5: Descriptive statistics: Time-varying first-order interaction effects, aggregated data. (A) Cueing effects with horizontal (left) and vertical (right) rectangles, after aggregating across compatibility levels and participants. (B) Compatibility effects for horizontal and vertical rectangles, after aggregating across cue types and participants.

In conditions IS-x-H and ID-x-V (third column) we observe a large negative compatibility effect (NCE) in the hazard functions emerging around 270 ms (Figure 7G) and around 210 ms in the $ca(t)$ functions (Figure 7I). More specifically, in the aggregated data, responses emitted around 150 ms show a small PCE for condition IS-x-H (green lines in Figure 7I) which then quickly reverses into a large NCE around 210 ms. As we will soon see, participants differ in whether or not they emit responses around 150 ms in these conditions. Finally, in conditions IS-x-V and ID-x-H (last column) we observe a large PCE in the hazard functions emerging around 300 ms (Figure 7J) and around 150 ms in the $ca(t)$ functions (Figure 7L).

After inspecting the behavior of each participant I noticed that they acted very similar in the NC conditions – they all tend to show a PCE in $h(t)$ and $ca(t)$ for at least one orientation – and in conditions IS-x-H and ID-x-V – they all show a strong NCE in $ca(t)$ around 240 ms, and most participants show a NCE in $h(t)$ around 300 ms. Figure 8 presents the

Figure 6: Descriptive statistics: Time-varying second-order interaction effects, aggregated data.

data for Participant 9 whose data patterns are rather similar to the aggregated patterns in Figure 7. Notice the PCEs in $h(t)$ and $ca(t)$ for conditions NC and VA (both orientations), and conditions IS-x-V and ID-x-H, and the NCEs in $h(t)$ and $ca(t)$ for conditions IS-x-H and ID-x-V (third column). This participant emits some responses around 150 ms in condition IS-x-H and those show a maximal PCE; no responses are emitted around 150 ms in condition ID-x-V by this participant.

The largest differences in behavior between participants were found in the conditions VA, IS-x-V, and ID-x-H. First, some participants show stronger signs of inhibition for horizontal compared to vertical orientations (Participants 1, 2, 4, and 6), whereas others show stronger signs of inhibition for vertical compared to horizontal orientations (Participants 3, 5, 7 and 8). One participant shows signs of inhibition for both orientations (Participant 10) and one participant shows minimal signs (Participant 9, Figure 8).

In Figure 9 I present the data from Participant 2. Notice the NCE in the $\delta h(t)$ plots for condition VA when rectangles are oriented horizontally (Figure 9A), and the NCE in the $\delta ca(t)$ plot (Figure 9C). Interestingly, the four

Figure 7: Descriptive statistics: Time-varying second-order interaction effects, aggregated data. Hazard, $\delta h(t)$, survivor, $\delta ca(t)$, and $ca(t)$ plots are depicted in rows one to five, respectively, for the 16 experimental conditions. The delta plots show the difference between the estimated proportions $[h(t) \text{ or } ca(t)]$ in the compatible and incompatible conditions. Error bars in the hazard, survivor, and conditional accuracy functions display ± 1 standard error of the respective proportion. Error bars in the delta plots display the approximate 95% confidence interval for the difference in proportions between compatible and incompatible conditions. The estimated median RT – or quantile $S(t)_{.50}$ – is indicated for each experimental condition by vertical colored lines in the plots of the hazard and survivor functions.

Figure 8: Descriptive statistics: Time-varying second-order interaction effects, Participant 9. Same conventions as in Figure 7.

participants who show signs of inhibition for VA-x-H also show signs of inhibition in condition ID-x-H (a short-lived temporary NCE in $ca(t)$ in Figure 9L).

Figure 10 shows the data from Participant 7. Notice the NCE in the $\delta h(t)$ plots for condition VA when rectangles are oriented vertically (Figure 10D), and the NCE in the $\delta ca(t)$ plot (Figure 10F). Interestingly, the four participants who show signs of inhibition for VA-x-V also show signs of inhibition in condition IS-x-V (a temporary NCE in $ca(t)$

Figure 9: Descriptive statistics: Time-varying second-order interaction effects, Participant 2. Same conventions as in Figure 7.

in Figure 10L). Figure 11 shows the data from Participant 3, who emits enough early responses around 150 ms in the invalid-cue conditions; note the presence of a maximal PCE in $ca(150)$ for conditions IS-x-H and ID-x-V.

Second, in the invalid cue conditions, early PCEs in $ca(t)$ around 150 ms are only visible if the participant emits (enough) responses around that time (Participants 1, 2, 4 and 8 do not emit enough early responses in the invalid cue conditions).

Figure 10: Descriptive statistics: Time-varying second-order interaction effects, Participant 7. Same conventions as in Figure 7.

Third, all participants except one show a hazard advantage around 150-200 ms in the cue-present conditions for horizontal compared to vertical orientations. As we will discuss below, this strongly suggests the involvement of a reference frame that is centered on the rectangular object, because the location of the target arrow will activate a task-irrelevant spatial code in this object-centered reference frame for horizontally oriented rectangles but not for vertically oriented rectangles. Figure 12 illustrates the spatial codes that are activated by the cue and target location in egocentric (e.g., gaze-centered) and allocentric (e.g., rectangle-centered) reference frames, including an egocentric frame centered

Figure 11: Descriptive statistics: Time-varying second-order interaction effects, Participant 3. Same conventions as in Figure 7.

on the location of covert spatial attention just before target onset (for those trials where the target is presented in the “LEFT-UP” location). We can also identify a possible reason why only conditions IS-x-H and ID-x-V show a strong and systematic NCE: the target occupies a left/right position relative to the cued location only in these two combinations of rectangle orientation and invalid cue type.

To sum up, the following list of effects observed in the descriptive statistics for the aggregated data (Figure 7) will be tested for significance in the next section: (A) the presence of a NCE in $h(t)$ and $ca(t)$ for conditions IS-x-H and ID-x-V

Figure 12: Experimental design and spatial codes. The spatial codes of the cue location and target location generated in different reference frames are printed above the location of the correct response button, for both horizontal (H) and vertical (V) orientations. The sixteen trial-types are illustrated on top for the cases where the target appears in the upper-left location. Spatial codes for the horizontally oriented rectangles are printed in bold. VA = valid, IS = invalid-same, ID = invalid-different, NC = no cue, C = compatible, I = incompatible.

(indicative of the involvement of a reference frame centered on the cued location), (B) the hazard advantage around 150-200 ms in the cue-present conditions for horizontal compared to vertical orientations (indicative of the involvement of a rectangle-centered reference frame), (C) the presence of a PCE in $h(t)$ and $ca(t)$ for conditions IS-x-V and ID-x-H, and (D) the presence of a PCE in $h(t)$ and $ca(t)$ for conditions NC and VA.

Response history and conditional accuracy analyses: Inferential statistics

The selected cloglog-hazard model is presented in Table 2 and the selected logit- $ca(t)$ model in Table 3. The predicted functions for trial 18 of block 25 in session 1 are shown in Figure 13. Note that I modeled behavior for the within-trial time range (150,480].

Cloglog[$h(t)$] model

Because TRIAL, BLOCK, and SESSION are centered, the first six parameter estimates (PE) in Table 2 model the shape of the cloglog[$h(t)$] function for the baseline condition (IS-C-H) in trial 18 of block 25 of session 1 using a 5th order polynomial function of TIME (Figure 13C, top panel, dark green line). Because TIME is centered on the reference bin (270,300] during model selection, the estimated intercept of the selected model refers to the predicted cloglog[$h(300)$] value for the baseline condition. Converting back from cloglogs to hazards, $h(300)$ equals .09 ($= 1 - \exp[-\exp(-2.32)]$) as shown in Figure 13C (second row, dark green line, bin 300). Parameters 2-5 show significant linear, quadratic, cubic,

Figure 13: Model predictions. Predicted (cloglog-) hazard and delta- $h(t)$ functions, and (logit-) $ca(t)$ and delta- $ca(t)$ functions for the 16 experimental conditions in trial 18 of block 25 of session 1.

quartic, and quintic effects of TIME on this intercept estimate, such that the predicted response hazard mostly increases over time: $h(180) = 0.018$, $h(240) = 0.044$, $h(300) = 0.094$, $h(360) = 0.22$, and $h(440) = 0.45$. 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.

With respect to the manipulations of interest, we see that relative to the baseline condition, there is a main effect of Incompatibility (parameter 7, reference bin PE = 0.7846, $p < .0001$). A measure of effect size for a discrete-time cloglog-hazard model can be obtained by exponentiating the parameter estimate which gives us a hazard ratio (HR; Allison, 2010, p. 242). Thus, compared to the cloglog[$h(300)$] estimate in the baseline condition, an incompatible target increases the estimated cloglog[$h(300)$] by 0.785 cloglog-hazard units, which corresponds to an increase in response hazard by a factor of 2.2 ($= \text{HR}(300) = \exp[0.785]$). This main effect of Incompatibility changes over time in a linear, quadratic, and

Table 3: Selected logit-*ca*(t) model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	se	z	p	PE	p	PE	p	p
1	(Intercept)	-6,63E-01	0,11532		-1,5092639	3.19e-11 ***		0,1868001	0,2408669	0,77	0,438025	2,2076638	1.06e-11 ***	3,62E+00	2.66e-15 ***	
2	TIME							1,1035081	0,1093209	10,09	< 2e-16 ***					
3	TIME ²							0,0271163	0,0247175	1,09	0,272621					
4	TIME ³							-0,0462802	0,0099638	-4,64	3.40e-06 ***					
5	TIME ⁴							0,0033501	0,0009067	3,69	0.000220 ***					
6	TIME ⁵							0,0006756	0,0003047	2,21	0.026615 *					
7	IC	8,75E-01	0,107757		4,0680233	< 2e-16 ***		3,6555701	0,2890551	12,64	< 2e-16 ***	1,6333699	1.23e-07 ***	-2,28E-04	0,999597	
8	TIME:IC							-0,7751374	0,1592499	-4,86	1.13e-06 ***					
9	TIME ² :IC							-0,2009889	0,0313841	-6,4	1.51e-10 ***					
10	TIME ³ :IC							0,0415572	0,0111642	3,72	0.000197 ***					
11	Ve	1,58E+00	0.025586 *		2,8674872	< 2e-16 ***		2,7673264	0,2561904	10,8	< 2e-16 ***	2,184079	8.85e-10 ***	2,02E+00	0.005486 **	
12	TIME:Ve							-0,2458228	0,1697817	-1,44	0,147652					
13	TIME ² :Ve							-0,0603906	0,0382848	-1,57	0,114703					
14	TIME ³ :Ve							0,0187178	0,012495	1,49	0,134129					
15	Ve:IC	-2,96E+00	0.008011 **		-6,2834095	< 2e-16 ***		-5,4647446	0,3854089	-14,17	< 2e-16 ***	-2,8740168	2.12e-09 ***	-8,80E-01	0,328656	

Continued **Table 3:** Selected logit-*ca*(t) model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	p		PE	p		PE	p	
16	TIME:Ve:IC						4,48	1,050087	0,234025			7.22e-06 ***				
17	TIME ² :Ve:IC						4,04	0,221379	0,0546795			5.15e-05 ***				
18	TIME ³ :Ve:IC						-2,81	-0,0493377	0,0175463			0.004926 **				
19	ID	1,89E+00	0.002018 **		2,4089408	3.71e-12 ***	8,81	2,204544	0,2500628		1,8128983	< 2e-16 ***	1.49e-07 ***	1,77E+00	0.022074 *	
20	TIME:ID						-0,99	-0,193296	0,1935454			0,317934				
21	TIME ² :ID						-0,63	-0,0233576	0,0367972			0,525581				
22	TIME ³ :ID						0,71	0,0111006	0,0155603			0,475602				
23	ID:IC	-3,22E+00	0.000190 ***		-7,133706	< 2e-16 ***	-15,92	-6,2754751	0,3940718		-3,5078155	< 2e-16 ***	4,40e-14 ***	-1,69E+00	0.060483 .	
24	TIME:ID:IC						4,38	1,1449633	0,2610747			1.16e-05 ***				
25	TIME ² :ID:IC						4,81	0,2385334	0,0495569			1.48e-06 ***				
26	TIME ³ :ID:IC						-3,01	-0,0596055	0,0197716			0.002572 **				
27	ID:Ve	-3,89E+00	0.000294 ***		-6,6965088	< 2e-16 ***	-16,25	-6,03194	0,3711507		-4,2221176	< 2e-16 ***	< 2e-16 ***	-3,59E+00	0.000813 ***	
28	TIME:ID:Ve						3,04	0,8122038	0,2671456			0.002363 **				
29	TIME ² :ID:Ve						2,53	0,1432237	0,0565953			0.011385 *				
30	TIME ³ :ID:Ve						-2,3	-0,0484081	0,0209861			0.021073 *				

continued **Table 3:** Selected logit-ca(t) model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	p		PE	p		PE	p	
31	ID:Ve:IC	7,90E+00	1.41e-06 ***			< 2e-16 ***						< 2e-16 ***		3,28E+00	< 2e-16 ***	0.016270 *
32	TIME:ID:Ve:IC							-2,5984908	0,4131431	-6,29		3.18e-10 ***				
33	TIME ² :ID:Ve:IC							-0,5285095	0,0849648	-6,22		4.96e-10 ***				
34	TIME ³ :ID:Ve:IC							0,1262887	0,0311278	4,05		4.97e-05 ***				
35	NC	3,61E+00	0.005932 **		4,4381673	2.08e-10 ***		4,347271	0,4358203	9,97		< 2e-16 ***	3,3315292	< 2e-16 ***	1,39E+00	0.007026 **
36	TIME:NC							-0,2767861	0,1887549	-1,46		0,142545				
37	TIME ² :NC							-0,1155425	0,0375573	-3,07		0.002095 **				
38	NC:IC	-1,31E+01	< 2e-16 ***		-9,8590313	< 2e-16 ***		-6,6649723	0,5306685	-12,56		< 2e-16 ***	-3,4695277	4.03e-14 ***	-2,75E-01	0.699332
39	TIME:NC:IC							1,5977756	0,2101964	7,6		2.93e-14 ***				
40	VA	2,11E+00	9.64e-07 ***		3,2988896	< 2e-16 ***		2,7467466	0,166868	16,46		< 2e-16 ***	1,6103222	2.01e-14 ***	1,05E+00	0.002685 **
41	TIME:VA							-0,5182252	0,1099024	-4,71		2.41e-06 ***				
42	TIME ² :VA							-0,0729306	0,0222448	-3,27		0.001043 **				
43	TIME ³ :VA							0,0240253	0,0084774	2,83		0.004596 **				

Continued **Table 3:** Selected logit-ca(t) model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	p		PE	p		PE	p	
44	VA:IC	-3,57E+00	1.05e-09 ***		-6,9949497	< 2e-16 ***		-4,8648003	0,3098606	-15,7	< 2e-16 ***	-1,0745867	0.002662 **	4,83E-01	0,38914	
45	TIME:VA:IC							1,8046247	0,1779668	10,14	< 2e-16 ***					
46	TIME ² :VA:IC							0,2073212	0,0348234	5,95	2.62e-09 ***					
47	TIME ³ :VA:IC							-0,0810555	0,012794	-6,33	2.37e-10 ***					
48	NC:Ve	-2,70E+00	0.054815.		-2,7051501	0.004492 **		-2,7132573	0,5985908	-4,53	5.82e-06 ***	-2,7227967	1.61e-06 ***	-2,73E+00	0.002665 **	
49	TIME:NC:Ve							-0,0046511	0,2532415	-0,01	0.985347					
50	NC:Ve:IC	9,97E+00	5.03e-09 ***		7,7862463	1.14e-11 ***		5,6058251	0,7097453	7,89	2.83e-15 ***	3,4257118	4.99e-07 ***	1,24E+00	0,266985	
51	TIME:NC:Ve:IC							-1,0902593	0,3111881	-3,5	0.000459 ***					
52	VA:Ve	-2,15E+00	0.005514 **		-3,7405761	< 2e-16 ***		-2,5813259	0,2850611	-9,05	< 2e-16 ***	-1,0238702	0.012879 *	-1,42E+00	0.079332.	
53	TIME:VA:Ve							0,8755196	0,1923385	4,55	5.31e-06 ***					
54	TIME ² :VA:Ve							0,0498393	0,0421705	1,18	0.237264					
55	TIME ³ :VA:Ve							-0,049026	0,0144583	-3,39	0.000697 ***					
56	VA:Ve:IC	4,81E+00	5.61e-05 ***		7,7620572	< 2e-16 ***		5,4562828	0,4160097	13,11	< 2e-16 ***	1,5377445	0.005172 **	-3,62E-01	0,720879	
57	TIME:VA:Ve:IC							-1,8596606	0,2621338	-7,09	1.30e-12 ***					

Continued **Table 3:** Selected logit-ca(t) model (columns 7 to 10).

Nr.	Effect	(150,180]			(210,240]			(270,300]			(330,360]			(400,440]		
		PE	p		PE	p		PE	se	z	p	PE	p	PE	p	p
58	TIME ² :VA:Ve:IC							-0,2015736	0,059425	-3,39	0,000694 ***					
59	TIME ³ :VA:Ve:IC							0,0757303	0,0198873	3,8	0,000140 ***					
60	TRIAL	-5,40E-03	0,013713 *	-0,0054005	0,013762 *	0,002191	-2,46	-0,0054012	0,002191	-2,46	0,013693 *	-0,0053994	0,013725 *	-5,41E-03	0,013598 *	
61	TRIAL ²	5,26E-03	0,026476 *	0,0052636	0,026427 *	0,0023696	2,22	0,0052629	0,0023696	2,22	0,026348 *	0,0052628	0,026349 *	5,27E-03	0,026251 *	
62	BLOCK	5,91E-03	0,244177	-0,0020743	0,320621		-2,95	-0,0065802	0,0022263	-2,95	0,003120 **	-0,00763	0,003337 **	-5,21E-03	0,312929	
63	TIME:BLOCK							-0,0013902	0,0008091	-1,71	0,085772 .					
64	TIME ² :BLOCK							0,0004325	0,0003135	1,38	0,167728					
65	BLOCK ²	-5,82E-03	0,147851	0,0013176	0,43188	0,0017837	2,16	0,003864	0,0017837	2,16	0,030292 *	0,0018168	0,381589	-4,80E-03	0,244335	
66	TIME:BLOCK ²							0,0001239	0,0006456	0,19	0,847791					
67	TIME ² :BLOCK ²							-0,0005744	0,0002495	-2,3	0,021310 *					
68	SESSION	-2,36E-01	2,56e-07 ***	-0,2355197	2,57e-07 ***	-5,15		-0,2355779	0,045682	-5,15	2,51e-07 ***	-0,235616	2,50e-07 ***	-2,36E-01	2,51e-07 ***	
RANDOM																
69	intercept variance	0,058		0,118		0,347						0,746		1,316		
70	TIME variance	0,021		0,021		0,021						0,021		0,021		
71	correlation	-0,18		0,72		0,92						0,96		0,98		

Note. The model was centered on reference bin (270,300] during model selection. The selected model was refitted four times with TIME centered each time on another reference bin: (150,180], (210,240], (330,360], and (400,440]. Parameter estimates (PE) are in logit-ca(t) units.

cubic fashion (parameters 8-10), resulting in $HR(180) = 0.65$ ($p = .066$), $HR(240) = 1.62$ ($p < .001$), $HR(300) = 2.2$ ($p < .001$), $HR(360) = 1.98$ ($p < .001$), and $HR(440) = 1.47$ ($p < .001$; compare dark and light green lines in Figure 13C, top panels). Thus, a significant NCE appears in the hazard functions for condition IS-x-H around 240 ms (see green line in the delta- $h(t)$ plot in Figure 13C), consistent with effect A listed above.

The main effect of changing the rectangle orientations to Vertical is also significant (parameter 11, reference bin $PE = 0.6486$, $p < .001$) and changes over time (parameters 12-14 in Table 2): $HR(180) = 0.2$ ($p < .0001$), $HR(240) = 0.9$ ($p = .37$), $HR(300) = 1.9$ ($p < .001$), $HR(360) = 2.2$ ($p < .001$), and $HR(440) = 1.4$ ($p < .001$). Consistent with Figure 4B and effect B listed above, hazard is significantly higher for horizontal rectangles in early bins and this effect reverses to an advantage for vertical rectangles (compare dark green cloglog-hazard lines in Figures 13C and 13D). The time-varying interaction effect between Incompatibility and Vertical is highly significant and negative for bins after 180 ms (parameters 15-18). As a result, the large NCE in $h(t)$ for IS-x-H is reversed to a smaller PCE in $h(t)$ for IS-x-V (compare green lines in Figures 13C and 13D, top panels), consistent with effect C listed above.

The main effect of ID is significant in the reference bin ($PE = 0.2863$, $p < .001$) and changes over time (parameters 19-22): $HR(180) = 0.49$ ($p = .0057$), $HR(240) = 0.81$ ($p = .055$), $HR(300) = 1.33$ ($p < .001$), $HR(360) = 1.63$ ($p < .001$), and $HR(440) = 1.11$ ($p = .21$). The interaction effect between ID and Incompatibility is highly significant and negative for bins after 180 ms (parameters 23-25). As a result, there is a PCE in $h(t)$ for ID-x-H (red and orange lines in Figure 13D), consistent with effect C listed above.

For condition ID-x-V, two extra effects come into play. First, the interaction effect between ID and Vertical changes from being positive to negative over time (parameter 26-29). Second, the interaction effect between ID, Incompatibility, and Vertical quickly changes from being negative to positive over time (parameters 30-32). As a result, there is a strong NCE in $h(t)$ for condition ID-x-V (Figure 13C, top panels), consistent with effect A listed above.

The main effect of NC is significant in the reference bin (parameter 33, $PE = 0.3899$, $p < .001$) and changes over time (parameters 34-36): $HR(180) = 0.004$ ($p < .001$), $HR(240) = 0.29$ ($p < .001$), $HR(300) = 1.47$ ($p < .001$), $HR(360) = 1.6$ ($p < .001$), and $HR(440) = 1.2$ ($p = .02$). Thus, for early bins hazard is lower without compared to with an IS cue, while this effect reverses for later bins, consistent with the cueing effects turning from positive to negative in Figures 4A, 5A, and 6. Due to the interaction effect between NC and Incompatibility (parameters 37-40), there is a PCE in $h(t)$ for condition NC-x-H (black and grey lines in Figure 13A), consistent with effect D listed above.

The main effect of VA is significant in the reference bin (parameter 41, $PE = 0.9916$, $p < .001$) and changes over time (parameters 42-44): $HR(180) = 1.4$ ($p = .069$), $HR(240) = 2.9$ ($p < .001$), $HR(300) = 1.47$ ($p < .001$), $HR(360) = 1.6$ ($p < .001$), and $HR(440) = 1.2$ ($p = .0067$). Due to the interaction effect between VA and Incompatibility (parameters 45-48), there is a PCE in $h(t)$ for condition VA-x-H (blue and cyan lines in Figure 13A), consistent with effect D listed above.

The effect of Vertical interacts in a time-varying fashion with NC (parameters 49-51), VA (parameters 54-47), NC:IC (parameters 52-53), and VA:IC (parameters 58-61). As a result, hazard for condition VA is lower for vertical compared to horizontal rectangles for early bins, and we see PCEs in hazard for conditions NC-x-V and VA-x-V (Figure 13B, top panels), consistent with effect D listed above.

One advantage of a discrete-time hazard model is that we can incorporate multiple time scales. In other words, the hazard of response occurrence not only varies across bins within a trial, but also with the passage of time across trials, blocks, and sessions. The selected model shows that the effects of TRIAL, BLOCK, and SESSION change over time (parameters 62-63, 65-67, 73-75). Furthermore, the effect of TRIAL² is time-invariant (parameter 64) and the effect of BLOCK² changes quadratically with TIME (parameters 68-70). Finally, TRIAL interacts with BLOCK (parameters 71-72) and BLOCK with SESSION (parameters 76-78). These effects are shown in Figure 14 (top panels) for the baseline condition IS-C-H.

Logit[ca(t)] model

Figure 13C (bottom panel) shows that the logit- $ca(t)$ in the baseline condition IS-C-H initially decreases and then increases from 270 ms onwards (dark green line; parameters 1-6 in Table 3). The intercept of 0.19 in reference bin (270,300] corresponds to $ca(300) = 0.55$ ($= \exp(0.19)/\{\exp(0.19)+1\}$).

The effect of Incompatibility is strongly positive for $180 < t < 400$ ms (parameters 7-10) so that there is a large NCE in $ca(t)$ for condition IS-x-H, consistent with effect A listed above. A measure of effect size for a logit- $ca(t)$ model

Figure 14: Model-based learning effects on multiple time scales for condition IS-C-H. Predictions for session 1 (A) and session 2 (B). T1-B1, first trial of block 1; T36-B1, trial 36 of block 1; T1-B50, first trial of block 50; T36-B50, trial 36 of block 50.

can be obtained by exponentiating the parameter estimate which gives us an odds ratio (OR). Thus, compared to the $\text{logit}[ca(300)]$ estimate in the baseline condition, an incompatible target increases this estimate by 3.66 $\text{logit-}ca(t)$ units (parameter 7, reference bin, $p < .001$), which corresponds to an increase of the odds of a correct response by a factor of 39 ($= \text{OR}(300) = \exp[3.66]$). Similarly: $\text{OR}(180) = 2.4$ ($p = .11$), $\text{OR}(240) = 58$ ($p < .001$), $\text{OR}(360) = 5.1$ ($p < .001$), and $\text{OR}(440) = 0.99$ ($p = .99$; compare green lines in Figure 13C, bottom panels).

The effect of Vertical is positive and significant in all reported bins (parameters 11-14), and the interaction effect between Vertical and Incompatibility is strongly negative for $t < 400$ ms (parameters 15-18). As a result, there is an early PCE in $ca(t)$ for condition IS-x-V (green lines in Figure 13D, bottom panels), consistent with effect C listed above.

The effect of ID is positive in all reported bins (parameters 19-22) and the interaction effect between ID and Incompatibility is strongly negative for $t < 400$ ms (parameters 23-26). As a result, there is an early PCE in $ca(t)$ for condition ID-x-H (red and orange lines in Figure 13D, bottom panels), consistent with effect C listed above. In contrast, for condition ID-x-V we see a NCE (parameters 27-34; effect A listed above). In the remaining conditions (NC-x-H, VA-x-H, NC-x-V, VA-x-V) we see an early PCE (parameters 35-59; effect D listed above). Finally, $\text{logit-}ca(t)$ changes not only on the

within-trial time scale, but also on the across trials/within-block time scale (TRIAL), the across-block/within-session time scale (BLOCK) and the across-sessions/within-experiment time scale (SESSION; parameters 60-68; Figure 14, bottom panels).

Discussion

To explore the effect of an informative cue and characterize the within-trial time course of space- and object-based effects, I statistically described and modeled the shape of RT and accuracy distributions from the famous two-rectangle paradigm of Egly et al. (1994) using response history analysis extended with micro-level speed-accuracy tradeoff analysis. These distributional analyses allow us to focus better on *when* various behaviorally relevant selection processes are active (Hommel, et al., 2019).

Neuroscientific studies have identified the basal ganglia (BG) as an important part of the vertebrate solution to the selection problem (Redgrave, Prescott, & Gurney, 1999), in both the motor and cognitive domains (Alexander & Crutcher, 1990; Boussaoud & Kermadi, 1997; McNab & Klingberg, 2008; Meyer & Bucci, 2016; Middlebrooks & Schall, 2014; Middleton & Strick, 2000; Nagano-Saito, Martinu, & Monchi, 2014; Stuphorn, 2015; van den Wildenberg & van der Molen, 2004; van Schouwenburg, den Ouden, & Cools, 2013; Yin, 2014). These and the current distributional results are more compatible with an ethologically-inspired view of dynamic behavior as determined by simultaneous processes that quickly specify potential motor actions and then select between them (Cisek & Kalaska, 2010).

Figure 15 provides a circuit diagram of some of the neural systems that implement selection processes, inspired by dynamic field theory (Schöner, et al., 2016). Figure 15 shows how the BG receive information from many parts of the brain (single-headed arrows), such as motor cortex (M1), high-level perceptual areas (IT, Body, Gaze, Exo), and frontal areas (e.g., WM, Endo, Task set). For example, I assume that the parietal cortex hosts various neuronal populations that code stimulus location in body- and hand-centered frames, and in an exogenous attention-centered frame (Exo), the superior colliculus hosts an egocentric gaze-centered frame (Gaze), neurons in the inferotemporal cortex (IT) are coding shape features such as curvature at a certain location, and neuronal populations in the frontal cortex provide an allocentric scene representation in allocentric working memory (WM) or code the current location of endogenous attention in the allocentric scene representation (Endo). Another population lays down memory traces and keeps track of where previous target detections occurred (Mem).

Figure 15 shows (only) five parallel cortico-ganglio-thalamo-cortico loops, which allow inhibitory control by the BG. There are for example (a) the dorsal and ventral working memory (WM) loops involving frontal areas to control access to WM, (b) the motor loop involving M1 to control skeletomotor movements, (c) the high-level object loop involving IT for object-based selection, (d) egocentric loops involving parietal cortex, and (e) the saccade loop involving the superior colliculus and frontal eye fields (not shown). Furthermore, the motor loop consists of three pathways: (a) the direct pathway gates response channel activation by selective disinhibition, (b) the indirect pathway reduces the gating by selective inhibition, and (c) the hyperdirect pathway can change the global response threshold level by aselectively (dis)inhibiting the baseline activity level (Frank, 2006; Panis & Schmidt, 2016; Schöner, et al., 2016; Wiecki & Frank, 2013).

A Simon effect is observed when the cue is absent together with response inhibition effects

When the cue is absent (NC) there is a positive compatibility effect present in the hazard and conditional accuracy functions for both rectangle orientations – the Simon effect. In other words, while the first target-triggered responses emitted around 210 ms still reflect the arrow location relative to the fixation cross (or the gaze location, or body), responses appearing around 240 ms start to reflect the arrow direction information, and all responses emitted after about 300 ms are correct.

Most authors agree that the Simon effect has its origin in response selection processes (Lu & Proctor, 1995). According to the dual-route hypothesis (De Jong, Liang, & Lauber, 1994; Kornblum, Hasbroucq, & Osman, 1990), the irrelevant arrow location and relevant direction attribute are processed in parallel along different routes. A fast,

Figure 15: Multiple-route circuit diagram including basal ganglia loops. Circles indicate various cortical populations that can form cortico-ganglio-thalamo-cortico loops. V1 is closest to the sensory surface, M1 to the motor surface. Cortical areas are topographically coupled (double-headed arrows). The basal ganglia (BG) receive input from many areas (single-headed arrows) and can provide inhibitory control (lines ending in a dot). The panel labeled “M1” shows the population activity for a single response dimension (x-axis), where activity for the right (R) response channel peaks and exceeds the threshold (dotted line). The small circles with crosshairs illustrate different spatial reference frames, and show population activity across the 2 dimensions of space viewed from above (with an above-threshold peak of activity indicated by a small black dot). The panel labeled “Task set” shows the memorized task-relevant stimulus-response bindings (Wilimzig & Schöner, 2005), and the panel labeled “IT” shows a three-dimensional perceptual neural field sensitive to 2D location and curvature. Finally, the two panels labeled “rectangle-centered” and “target-centered” illustrate how spatial codes are generated in object-centered reference frames. Note that only a single above-threshold peak can be formed in the fields M1, Exo, and Endo, while multiple above-threshold peaks can be present in the other fields (Schöner, et al., 2016). IT, inferotemporal cortex; BG, basal ganglia; V1, primary visual cortex; M1, primary motor cortex; WM, working memory; Mem, memory traces of target detections in previous trials.

direct route processes the task-irrelevant (left/right) position and automatically activates the ipsilateral response. A slower, controlled route processes and identifies the task-relevant stimulus attribute (e.g., shape, color) and activates the response assigned to this attribute by the task instructions. In the compatible condition both routes activate the same, correct response, while in the incompatible condition the direct route first activates the incorrect response and the controlled route activates the correct response somewhat later. This response conflict in the incompatible trials is assumed to cause the Simon effect.

According to the activation-suppression hypothesis (Ridderinkhof, 2002) the initial behavioral response activated by task-irrelevant location information is actively and selectively inhibited, and this selective inhibition becomes effective only after a given amount of time because it takes some time for the inhibition to build up (Burle, Possamaï, Vidal, Bonnet, & Hasbroucq, 2002; Burle, van den Wildenberg, & Ridderinkhof, 2005). Such selective response inhibition is reflected in the shape of the $\delta h(t)$ plots: initially the positive compatibility effect for condition NC increases with RT but for slower responses it reduces (and can become negative) due to active and selective response inhibition. Burle et al. (2002) suggested that this implies an online executive control system that detects, stops, and corrects premature response tendencies within a trial, that is, at a very short time scale. Burle et al. (2005) concluded that Simon tasks and spatial cueing tasks (see Figure 1B) both involve a common mechanism of response activation followed by the selective suppression of that activation.

Thus, as illustrated in Figure 15, in condition NC, the task-irrelevant arrow location is quickly and automatically coded in gaze- and body-centered reference frames (Gaze, Body). If the spatial codes that the arrow location generates in these frames are similar enough to the current task set, then the BG will gate the compatible response via the direct pathway and this can trigger overt responses around 210 ms after target onset. However, for incompatible targets, as soon as the arrow direction is available via the object-loop (IT), a conflict emerges causing the BG (a) to temporarily raise the global response threshold via the hyperdirect pathway, (b) to selectively inhibit the prematurely activated incorrect response channel via the indirect pathway, and (c) to start selectively gating the correct response, so that all responses emitted after 300 ms are correct (Wiecki & Frank, 2013).

A valid informative cue triggers response activation and suppression, but not attention?

The fact that an informative cue increases hazard of response occurrence in bins before 210 ms (Figures 7, 8, 9, 10 and 11; parameter 33 in Table 2) shows that the cue activates the spatially compatible response channel, which then sometimes leads to overt responses. Given that the cue is presented 150 ms before the target, we can expect premature cue location-triggered responses around 60 ms (210 - 150) after target onset. As soon as the cue is identified as a non-target, the BG can start to selectively inhibit the prematurely activated (correct or incorrect) response channel, as reflected by the temporary dips and up-rises in the $ca(t)$ functions for compatible and incompatible VA conditions, respectively, that emerge around 90 ms after target onset.

We have seen that participants differ in whether they more strongly inhibit cue-triggered responses for the horizontal or vertical rectangle conditions, and how all but one display a hazard advantage around 150-200 ms for horizontal rectangles when the cue is presented. The latter finding suggests the involvement of an allocentric reference frame centered on the cued rectangle (Figure 15). As shown in Figure 12, for both rectangle orientations target onset generates an egocentric code, but only for horizontally oriented rectangles will the target generate an additional task-interfering allocentric code. This early effect of rectangle orientation on hazard around 150-200 ms is a robust one as it is present in 9 out of 10 participants. Recent studies have shown that a Simon effect can be observed simultaneously for both egocentric and allocentric reference frames (Li, et al., 2019; Wang, et al., 2016). People thus seem to differ in how easily they can – or are willing to – suppress response channels activated by various task-irrelevant spatial codes in different reference frames.

But why is it not the case that the Simon effect emerges systematically earlier for VA compared to NC in the hazard functions (Figure 7A)? This is surprising if one assumes that the space-based effect is due to a facilitation of target processing in terms of efficiency due to the fact that the valid informative cue has summoned attention to the location of the upcoming target. If the valid cue would speed up target processing then the spatial compatibility effects should also emerge earlier compared to NC. Instead, the valid cue triggers early responses before 200 ms that are unaffected by target properties.

Interestingly, Yaron and Lamy (2020) concluded that the brain waits for clues that the appropriate moment has arrived to deploy attention. This would mean that attention is only deployed after target onset. Consistent with this idea is the conclusion of Alilović, Timmermans, Reteig, van Gaal, and Slagter (2019) that initial visual afferent activity may be impenetrable to top-down influences by spatial prediction and attention. This would imply that we should also revisit the early informative cueing studies using response history and conditional accuracy analyses (Posner, 1980).

Together with Chen (2012) and Chen, Cave, Basu, Suresh, and Wiltshire (2020) I conclude that the two-rectangle paradigm is not ideal for studying *pure* object-based attention effects. For example, condition IS-x-H is not systematically faster than ID-x-V (see estimates of the median RT in the survival plots), and there are no consistent signs that compatibility effects emerge later for ID than IS as predicted by the attentional disengagement hypothesis – they do appear later for IS-x-V and ID-x-H than for IS-x-H and ID-x-V in many participants. Interestingly, all participants show a higher (or equal) hazard estimate for IS-x-V compared to ID-x-H for bins after ~300 ms, which seems consistent with the time-consuming spreading of attention along an object surface in perceptual areas. This attentional spreading effect might be masked by the large NCE in conditions IS-x-H and ID-x-V.

A functional distinction between exogenous and endogenous attention

In the less frequent invalid-cue conditions, the target appears unexpectedly at an uncued location and there are typically less premature responses in the invalid-cue conditions compared to the valid-cue conditions. This can be explained by assuming that the hyperdirect pathway of the BG temporarily increases the global response threshold in case of a “surprise” or “location conflict” signal, in order to allow more information (stimulus or memory) to influence which of multiple response channels is ultimately selected (Frank, 2006).

A sequence of a maximal PCE (here around 150 ms) followed by a strong NCE (here around 240 ms) as observed here in the $ca(t)$ functions for conditions IS-x-H and ID-x-V (for only those participants that emit responses in these bins), has also been observed in a masked response priming experiment with centrally presented arrows (Burle, et al., 2005; Panis & Schmidt, 2016) and is highly suggestive of active and selective response inhibition. Indeed, as shown in Figure 12 there is no information or spatial code present that can *activate* the incompatible response channel. Note that the incompatibility between the egocentric location of the *cue* and the direction of the target arrow (i.e., the location of the correct response button) is an unlikely explanation because it cannot explain the presence of the earlier PCE, and the timing of the NCE (~390 ms after cue onset) is rather late to be caused directly by the cue.

But why then does the NCE in $ca(t)$ emerge between 150 and 240 ms? As shown in Figure 12, the target location relative to the invalid focus of attention before target onset generates an additional task-relevant spatial code signal only for conditions IS-x-H and ID-x-V. Together with Panis and Schmidt (2020) I assume that the cue attracts attention and that the locus of attention forms the center of a spatial reference frame. If we then also assume that arrow direction identification requires attentional focusing (Yoo, Tsotsos, & Fallah, 2019) – involving a target-centered reference frame where the tip of the arrow is located to the left or right of its center (see Figure 15) – then the automatic response activation that its location generates in the attention-centered reference frame in conditions IS-x-H and ID-x-V around 150 ms – displaying a PCE – must be quickly inhibited in order to allow a response guided by the direction information. Selective and active inhibition of the premature response activation – and concurrent disinhibition of the opposite response – can thus also explain why suddenly only error responses appear in conditions IS-C-H and ID-C-V around 150-240 ms for each participant, before arrow direction signals take over.

Therefore, does this proposal contradict the idea that attention is only deployed after target onset (Yaron & Lamy, 2020)? Perhaps Yaron and Lamy (2020) are dealing with *endogenous* attention operating in an allocentric reference frame (Endo in Figure 15), while the locus of *exogenous* attention forms the center of an egocentric spatial reference frame (Exo in Figure 15; see Schöner, et al., 2016, for similar ideas). These speculative ideas should be tested in future confirmatory studies.

Finally, once arrow direction signals become available via the object loop, spatial compatibility effects will emerge in the hazard functions with a sign that depends on how strongly and selectively the premature response activation caused by some loop(s) has already been suppressed. In other words, in each participant a positive or negative spatial compatibility effect in hazard will emerge depending on the relative activation level of both response channels at the time when target signals become available.

Future studies of attentional selection processes

The three dimensions of attention discovered so far (exogenous/endogenous/memory, content, activation/suppression) can be linked to the components in Figure 15. For example, attention can select or suppress locations, objects, etc., because selection processes can also occur within a neural population when more than one stimulus is present in the scene, due to long-range inhibitory connections between neurons in the same population (Schöner, et al., 2016). However, because synaptic connections within and between populations only change on a relatively long time scale (minutes), the BG are necessary to be able to select information on the fast time scale of a single trial (milliseconds).

As always, the conclusions that can be drawn based on a single study are rather limited. Also, without an explicit computational model it is of course impossible to effectively characterize the complex time-varying balance between response activation and suppression processes that give rise to the observed hazard and conditional accuracy estimates. However, the current exploratory study shows (a) that differences between means conceal the highly dynamic behavior that participants display within a trial, and (b) that the data are consistent with a multiple-route model of response activation kept in check by inhibitory control processes.

An important challenge for future confirmatory studies will now be to characterize and temporally disentangle the contribution of each of these BG loops to response activation and suppression, using statistical control for the passage of time on multiple time scales during data analysis using event history and conditional accuracy analyses, next to clever experimental control. For example, future distributional studies with a small-*N* design could change the event-of-interest (e.g., saccade onset), manipulate the rectangle-cue and cue-target SOAs, decouple the body-centered and gaze-centered reference frames, compare detection and discrimination tasks, present the task-irrelevant and task-relevant information at different points in time, check whether an informative cue simply draws exogenous attention or also lowers the global response threshold due to an expectation signal, test whether VA, ID, and IS interact with BLOCK, etc., in order to better test various process models of the underlying cognitive machinery.

Conclusions

More and more distributional results point to an important role for task-irrelevant activation processes and inhibitory control processes in shaping RT and accuracy distributions in various research paradigms (Panis, Moran, Woltersdorfer, & Schmidt, 2020; Panis & Schmidt, 2016, 2020). As discussed by Van Gelder (1995), it is very likely that cognition is the behavior of a dynamical system. To understand the behavior of a dynamical system it is crucial to track its output over different time scales (Schöner, et al., 2016) using a distributional analysis (VanRullen, 2011), such as discrete-time hazard analysis (Allison, 2010; Townsend, 1990). I hope to have convinced the reader that the use of an established, more advanced data-analysis method can maximize the return from the collected data.

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Footnotes

- 1 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 (Singer & Willett, 2003).
- 2 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.
- 3 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 to get 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 the current data set took at least 15 hours as the model never converged. For these reasons, I opted for a simple random effects structure with three parameters (intercept, TIME, and their correlation; Tables 2 and 3).