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Review

No single electrophysiological marker for facilitation and inhibition of return: A review

Elisa Martín-Arévalo[a,b,*], Ana B. Chica[b,1], Juan Lupiáñez[b,1]

[a] ImpAct team, Lyon Neuroscience Research Center, INSERM U1028—CRNS-UMR5292, France
[b] Department of Experimental Psychology and Mind, Brain, and Behavior Research Center (CIMCYC), University of Granada, Spain

HIGHLIGHTS

- Different electrophysiological components have been related to facilitation and inhibition of return.
- We suggest that there is no single neural marker for facilitation and inhibition of return.
- Many variables (task set, cue-target interval, etc.) determine the electrophysiological modulation of cueing effects.

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ABSTRACT

Different electrophysiological components have been associated with behavioural facilitation and inhibition of return (IOR), although there is no consensus about which of these components are essential to the mechanism/s underlying the cueing effects. Different spatial attention hypotheses propound different roles for these components. In this review, we try and describe these inconsistencies by first presenting the electrophysiological component modulations of exogenous spatial attention as predicted by different attentional hypotheses. We then review and quantitatively analyze data from the existing electrophysiological studies trying to accommodate their findings. Variables such as the task at hand, the temporal properties and interactions between cues and targets, the presence/absence of intervening events, or stimuli arrangement in the visual field, might critically explain the discrepancies between the theoretical predictions and the electrophysiological modulations that both facilitation and IOR produce. We conclude that there is no single neural marker for facilitation and IOR because the behavioural effect that is observed depends on the contribution of several components: perceptual (P1), late-perceptual (N1, Nd), spatial selection (N2pc), and decision processes (P3). Many variables determine the electrophysiological modulations of different attentional orienting mechanisms, which jointly define the observed spatial cueing effects.

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* Corresponding author at: Integrative, Multisensory, Perception, Action and Cognition team (ImpAct team), Lyon Neuroscience Research Center, INSERM U1028—CRNS-UMR5292, 16 Ave. Doyen Lépine, 69676 Bron Cedex, France.
E-mail address: emartina@ugr.es (E. Martín-Arévalo).
[1] These two authors have contributed equally to this work.

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1. Introduction

Exogenous attentional mechanisms bias information processing in the brain, leading to a bottom-up selective response to salient or potentially relevant stimuli (see e.g., [28,31]). In the Posnerian cueing paradigm ([52]; see Ref. [8], for a review), spatially non-predictive peripheral cues, which trigger exogenous attentional capture [59], produce two distinct effects on target processing: (1) At short cue-target onset asynchronies (CTOAs; ~50–300 ms), reaction times (RTs) are usually faster for targets appearing at the same location as the peripheral cue (i.e., cued locations) than for targets presented at the opposite location (i.e., uncued locations), leading to a *facilitatory* effect. (2) At longer CTOAs (after ~300 ms), the opposite pattern of results emerges, with slower RTs for targets appearing at the cued location as compared to the uncued location. This latter effect, initially described by Posner and Cohen [53], is known as *Inhibition of Return* (IOR; [54]). Giving such an evocative name to the IOR effect has greatly contributed to the confusion between the *behavioural effect* that is measured (i.e., slower responses to targets appearing at cued locations as compared to uncued locations) and the *mechanism/s* underlying the effect (see Ref. [15]; for a review). Thus, the IOR terminology clearly reflects the theory initially proposed to explain the effect: IOR was theorized to be the consequence of an impaired ability to return attention to a previously attended location (see Ref. [34]; for a review). Although other explanations for the IOR effect have been currently considered, such as a *detection cost* [39] or *habituation* [14] of attentional capture to targets presented at a previously cued location (see also Refs. [2,21,41]), the mechanism/s underlying cueing effects still remains highly debated (see e.g. [40], for a review).

Several researchers have proposed that cueing effects reflect the modulation of multiples stages of processing (e.g. [2,29,32,39,64]), although no agreement has been reached about which of these stages of processing (and their associated electrophysiological components) is/are essential to the mechanism/s underlying both facilitation and IOR.

2. Electrophysiological component modulations predicted by different hypotheses

Many researchers have concentrated their efforts on finding the electrophysiological markers of facilitation and IOR, although results have been contradictory and no single electrophysiological marker can be unequivocally associated with behavioral facilitation or IOR. Table 1 presents the electrophysiological modulations hypothesized by the traditionally attentional reorienting hypothesis and two recent attentional–perceptual alternative hypotheses about facilitation and IOR. Note that according to Taylor and Klein [64], IOR can generate two mutually exclusive effects: (1) an attentional/perceptual effect, which occurs when saccadic eye movements are not permitted and the oculomotor system is suppressed; and (2) a motor effect, which occurs when saccadic responses are required and the oculomotor system is active (see also Refs. [25,35]; for reviews). Given that eye movements were strictly forbidden in most of the previous electrophysiological studies (although see Refs. [61–63]), we decided to focus this review on findings related to the attentional/perceptual effect, wherein the oculomotor system is actively suppressed. The interested reader is referred to Refs. [2,14,15,34,39,40], for different theoretical interpretations of the cueing effects, and to Luck et al. [37], for a review of the electrophysiological components of attention.

The traditional attentional reorienting hypothesis is assumed by most researchers in the field (see e.g. [34,55,57,62,63,65,68]), although not necessary by IOR experts, who greatly differ about their conception of IOR (see Ref. [15]). According to this hypothesis, three main processes occur during attentional orienting: (1) An initial attentional orienting to the cued location (most likely reflected in the P1 and/or N2pc component—posterior contralateral N2); (2) an attentional re-orienting to the fixation point, which occurs at long enough CTOAs (after ~300 ms). This attentional re-orienting might be enhanced or accelerated by the presence of intervening events between the cue and target[1] (see Ref. [44]; for a review of modulations produced by intervening events) and (3) an inhibited attentional orienting if the target is presented at the previously cued location as compared to the uncued location. This initial orienting and inhibited re-orienting to the target (proposed to produce both the facilitation and the IOR effect, respectively) might produce modulations at different stages of processing: perceptual (reflected in the P1 and N1 components), spatial selection (reflected in the N2pc component), and post-perceptual correlates of resource allocation and/or decisional processing (reflected in the Nd-negative difference, and P3 component, respectively; see e.g. [45]; see Section 3 for a discussion of these components).

Contrary to the traditional attentional reorienting hypothesis, the attentional–perceptual hypotheses only postulates two processes underlying the cueing effects (see e.g. [2,14,23,39]): (1) An attentional orienting to the cued location (most likely reflected in the P1 and/or N2pc component); and (2) a *habituated attentional capture* or *detection cost* (depending on the underlying hypothesis; see Table 1) when the target is presented at the cued location as compared to the uncued location (reflected in a reduced amplitude of the P1 and/or N1 component for cued as compared to uncued location trials). Importantly, both attentional–perceptual hypotheses do not consider attentional re-orienting as a necessary condition to observe the IOR effect (see e.g. [2] for a review; see also Refs. [6,14]). For example, Berlucchi [2] suggests an interpretation of the IOR effect in sensory terms, where the visual system response to the target would be reduced by the previous stimulation at the same spatial location, independently of attentional orienting. The *habituation hypothesis* ([14]; see also Ref. [2], for similar assumptions) postulates that the impaired early target perceptual processing (reflected in the P1 modulation) will only be measured when IOR is behaviourally observed (assuming an enhanced perceptual processing when facilitation is observed). Contrary, the *cue-target event integration-segregation hypothesis* ([39] for a review; see also Ref. [41]) postulates that perceptual processing should always be impaired by the very appearance of the cue when a long enough CTOA is used (after ~500 ms; [44]), no matter the behavioural result that is measured (facilitation

[1] The presence of intervening events favour the appearance of the IOR effect in some experimental situations in which no IOR would otherwise be observed (see e.g. [56,55,44,45]).

Table 1
Predictions made by the different hypotheses concerning the cueing effects on the target-locked components. Reduction: smaller amplitude for cued than uncued location trials; enhancement: larger amplitude for cued than uncued location trials.

Hypotheses	Effect	P1	N1	Nd	P3
Attentional:	Facilitation	Enhancement	Enhancement	Inconsistent predictions*	Enhancement
reorienting hypothesis	IOR	Reduction	Reduction	Inconsistent predictions*	Reduction
Atentional–perceptual:	Facilitation	Enhancement	No predictions	No effects	No effects
habituation hypothesis	IOR	Reduction	No predictions	No effects	No effects
Atentional–perceptual: event	Facilitation	Reduction	Enhancement	Enhancement	Enhancement
integration–segregation hypothesis	IOR	Reduction	No effect or reduction	No effect or reduction	No effect or reduction

* No agreement among authors.

or IOR). According to this latter hypothesis, the final behavioural effect that is observed (facilitation or IOR) depends on other processes related to further discriminative processing (N1) or spatial selection (N2pc), and other post-perceptual correlates of resource allocation and/or decisional processes (reflected in the Nd and/or P3 component, respectively). It is proposed that when cue-target integration processes are disrupted (such as when using e.g., long CTOAs, intervening events, very short target durations, or any other manipulation that emphasizes the need for target detection) there is always a "detection cost" (measured as a P1 reduction for cued as compared to uncued location trials) that may or may not be behaviourally measured as IOR depending on the modulations of later-stage processes (N1, N2pc, Nd, and/or P3).

2.1. Task set predictions

The nature of the task is perhaps the clearest determinant of the time-course of spatial cueing effects [8,42]. However, despite the overall agreement about the fact that the temporal course of facilitation and IOR depends on task-demands (IOR appears at longer CTOAs in discrimination than in detection tasks; see e.g. [7,34,42]), no such agreement has been reached regarding to the mechanism/s underlying this task-based difference.

The traditional *attentional reorienting hypothesis* has explained the different time course of cueing effects on the basis of a larger attentional capture and, therefore, a later attentional disengagement from cued locations in discrimination tasks than in detection tasks [34]. More attentional resources are supposed to be assigned to cue processing in discrimination than in detection tasks. This entails a greater and longer facilitatory effect (and a later disengagement) in discrimination than in detection tasks, which would be reflected in P1 modulations. However, contrary to this hypothesis, attentional capture and attentional disengagement have been demonstrated to be different processes: IOR has been consistently reported with no evidence of previous facilitation, and attentional disengagement has been demonstrated to be neither necessary nor sufficient for IOR to be observed [9,41,43,49].

The *cue-target event integration–segregation hypothesis* makes an explicit prediction involving task set (see Ref. [39], for a review). Detection and localization tasks are mainly affected by the contribution of the "detection cost" to performance, which hinders onset-detection processes. Other tasks, such as discrimination or go-no go tasks, require the contribution of what is called "spatial selection benefit", in which the onset of peripheral cues benefit target discrimination by means of cue-target integration processes. The presence of the spatial selection benefit could behaviorally counteract the detection cost that is always present (and measured as a P1 reduction for cued as compared to uncued targets) at long enough CTOA (after ~500 ms). This hypothesis asumes that at long enough CTOAs, P1 reductions for cued as compared to uncued location trials can be related to behavioural facilitatory effects if other components (N1, Nd, or N2pc) reflect enhancements for cued as compared to uncued location trials (which the hypothesis interprets as spatial selection benefits). This hypothesis was tested in a previous electrophysiological study [45] using a discrimination task in which the presence/absence of an intervening event produced opposite effects on RTs: facilitation in the absence of an intervening event, and IOR when the intervening event was presented (see also experiment 2; [44]). Our results demonstrated that the P1 component was reduced for cued as compared to uncued targets both when the intervening event was present and when it was absent; therefore, perceptual processing was impaired independently of the behavioural result that was measured: facilitation or IOR (see Refs. [6,68] for similar results). Moreover, peripheral cues only modulated later components (namely N1, Nd, and P3) when no intervening event was presented and facilitation was behaviourally observed (see e.g. [27,48,68]; for similar results in some of these components).

3. Review of previous electrophysiological studies of cueing effects

A thorough review of the literature unmasks some important discrepancies between the predictions made by the previously reviewed hypotheses and the electrophysiological modulations that both behavioural facilitation and IOR produce. These modulations are shown to depend on many variables such as the task at hand, the temporal properties of cues and targets, the presence/absence of intervening events, and stimuli arrangement in the visual field. All these variables are reviewed below. Table 2 summarizes the results of previous studies showing the modulations of the most important electrophysiological components associated with peripheral cueing: P1, N1, Nd, and P3. We describe below these modulations for each component, and analyze whether the component is essential or not for the observation of the behavioral effect (facilitation or IOR).

3.1. P1 component

The facilitation effect has been traditionally associated with a general enhancement of the P1 component for cued as compared to uncued location trials; however, as it can be observed in Table 2, this enhancement of P1 amplitude is mainly observed at short CTOAs (<250 ms) [11,26,27]). After a long enough CTOA (after ~500 ms), the P1 component is either reduced for cued as compared to uncued locations or no modulations are observed, even though the behavioural facilitatory effect might still be observed [6,45]; see also [68], although facilitation was not significant). This observation is true for all tasks (see e.g. [6,11,17,26,45,68] although see Ref. [67]; for a significantly enhanced P1 for cued location trials as compared to uncued location trials at CTOAs longer than 500 ms). Note that Van der Lubbe et al. [67] reported an unusual behavioural facilitatory effect at a long CTOA (940 ms), associated with a significant enhancement of P1 amplitude. In their experiment, five different CTOAs (144, 188, 236, 588, and 940 ms), composed of mostly short CTOAs, were employed in a discrimination task. It is important to

Table 2
Summary of electrophysiological modulations of the cueing effects in previous electrophysiological studies.

Type task	Study	Task characteristics	SOA (in ms)	Cue duration (in ms)	Intervening event	Target duration (in ms)	Cueing Effect (Uncued–Cued)	P1	N1	Nd*	P3
Detection	McDonald et al. [48]	Exp. 1	500–700	67	Absent	Until response	−13	Reduction (called Nd)	None	Yes	Enhancement
			900–1100	67	Absent	Until response	−17	n.s	n.s	None	Enhancement
		Exp. 2. Reduced sensory integration cue-target	100–300	67	Absent	Until response	6 (n.s)	n.s. (called Nd)	None	n.s	n.s
			500–700	67	Absent	Until response	−13	Reduction	n.s	None	n.s
	Hopfinger and Mangun [27]	Cue: four dots in each visual field (right/left)	34–234	34	Absent	50	8	Enhancement	IIN	Not reported	Enhancement
			566–766	34	Absent	50	−14	n.s	Not reported	Not reported	n.s
	Prime and Ward [57]	Stimuli above/below fixation	900–1200	200	Always present	1000	−36	n.s	Reduction	Yes	Not reported
	Wascher and Tipper [68]	Exp.1. Transient Cue	350	50	Absent	200	−20	Reduction	None	None	Not reported
			900	50	Absent	200	−31	Reduction	n.s	Yes	Not reported
		Exp.2. Sustained Cue	350	Until target offset	Absent	200	1 (n.s)	Reduction	None	None	Not reported
			900	Until target offset	Absent	200	12 (n.s)	Reduction	n.s	Yes	Not reported
	Van der Lubbe et al. [67]	Stimuli above/below fixation	588	50	Absent	200	−46	Reduction	n.s	Not reported	Not reported
			940	50	Absent	200	−43	n.s	n.s	Not reported	Not reported
	Chica and Lupiáñez [6]	Predictive Cue	1000	50	Absent	100	0	Reduction Ipsilateral	Reduction?	Yes?	Reduction Ipsilateral
		Counter-Predictive Cue	1000	50	Absent	100	−44	Reduction Ipsilateral	Reduction?	None	Enhancement
Localization	Eimer [17]	Exp.1. Predictive Cue	700	200	Absent	100	34	Reduction (Ipsi) and Enhancement (Contra)	n.s	n.s	None
		Exp.2. Nonpredictive Cue	700	200	Absent	100	0	n.s	n.s	Yes	None
	Prime and Ward [57]	Stimuli above/below fixation	900–1200	200	Always present	1000	−18	Reduction only over PO8	Reduction	Yes	Not reported
	Wascher and Tipper [68]		900	50	Absent	200	−24	Reduction	n.s	Yes	Not reported
			2000	50	Absent	200	−14	Reduction	n.s	Yes	Not reported
	Satel et al. [63]	Spatiotopic target - upper visual field. Four boxes above/below and left/right. Target response: eye movement	1500	300	Always present –eye movement	1500	−19	n.s	n.s	Yes Contralateral	Not reported
	Satel et al. [62]	One block: central fixation throught the trials (cue-ignored condition)	1200	300	Absent	3000	−21	Reduction Ipsilateral	Not reported	Not reported	Not reported
	Satel et al. [61]	Peripheral cues. Eye movement to cues and manual localization response to targets	900	300	Always present – eye movement	3000	−52	Reduction Ipsilateral	Reduction Ipsilateral	Yes Contralateral	Not reported
Discrimination	Hopfinger and Mangun [26]	Cue: Four dots in each visual field (right/left). Target: Short or tall vertical bars	34–234	34	Absent	50	16	Enhancement	None?	Not reported	Enhancement
			566–766	34	Absent	50	−2 (n.s)	Reduction	Enhancement?	Not reported	n.s
	Eimer [17]	Exp.1. Predictive Cue	700	200	Absent	100	13	n.s	Enhancement	Yes	Reduction
		Exp.2. Nonpredictive Cue	700	200	Absent	100	−2 (n.s)	Reduction (Ipsilateral)	Enhancement	Yes	Reduction
	Prime and Ward [56]	Stimuli above/below fixation	900–1200	200	Always present	1000	−21	Reduction	Reduction	Yes	Not reported
	Prime and Jolicoeur [55]	Stimuli above/below fixation	800–1100	200	Present	800	−14	Reduction	Reduction	n.s	Enhancement
			800–1100	200	Absent	800	−1 (n.s)	n.s	n.s	Yes	n.s
	Doallo et al. [11]	Uninformative peripheral cue	100	34	Absent	75	43	Enhancement at occipital electrodes	Not reported	Not reported	Not reported
			300	34	Absent	75	12 (n.s)	Reduction	Not reported	Not reported	Not reported
			500	34	Absent	75	4 (n.s)	Reduction	Not reported	Not reported	Not reported
			700	34	Absent	75	2 (n.s)	Reduction	Not reported	Not reported	Not reported
	Doallo et al. [12]	Uninformative peripheral cue	100	34	Absent	75	43	Not reported	Enhancement at one right temporal electrode	Not reported	Not reported
			300	34	Absent	75	12 (n.s)	Not reported	Enhancement Ipsilateral	Not reported	Not reported
			500	34	Absent	75	4 (n.s)	Not reported	n.s	Not reported	Not reported
			700	34	Absent	75	2 (n.s)	Not reported	n.s	Not reported	Not reported
	Van der Lubbe et al. [67]	Stimuli above/below fixation	588	50	Absent	200	8 (n.s)	n.s	n.s	Not reported	Not reported
			940	50	Absent	200	19	Enhancement	n.s	Not reported	Not reported
	Chica and Lupiáñez [6]	Predictive Cue	1000	50	Absent	100	18	Reduction Ipsilateral	Enhancement?	Not reported	Reduction Ipsilateral
		Counter-Predictive Cue	1000	50	Absent	100	−27	Reduction Ipsilateral	None?	Yes?	None
	Martín-Arévalo et al. [45]		500–700	50	Present	Until response	−11	Reduction	None	None	None
			500–700	50	Absent	Until response	10	Reduction	Enhancement	Yes	Enhancement
	Gutiérrez-Domínguez et al. [22]	Stimuli above/below fixation – spatial cueing effects averaged between visual fields	2000	100	Always present	Until response	−12	Not reported/None	Reduction UVF	Yes LVS	Not reported/None
Go-no go	Prime and Ward [57]	Stimuli above/below fixation	900–1200	200	Always present	1000	−33	Reduction	Reduction	Yes	Not reported
	Tian and Yao [66]		900–1200	50	Absent	200	−28	Reduction	Reduction	Not reported	Enhancement for no go trials

Reduction: smaller amplitude for cued than uncued location trials; enhancement: larger amplitude for cued than uncued location trials; none: no effect;?: effect observed but not subjected to statistical tests; n.s.: non-significant effect; IIN: Ipsilateral Invalid Negativity. Nd: Negative difference; Yes: Nd component present; LVF: lower visual field. UVF: upper visual field. Ipsi: Ipsilateral electrodes; Contra: Contralateral electrodes. Grey background indicates facilitation effects. The underlined studies are the only ones used for the subsequent correlation analyses (see Section 4).

note that the range of CTOAs strongly modulates cueing effects: at the same CTOA, IOR or facilitation can be behaviourally measured depending on whether a long or short range of CTOAs is employed (see e.g. [3,49]). Similarly, the task used in Van der Lubbe et al.'s study [67] consisted of discriminating the direction of an up/down pointing triangle, which could appear either above or below the fixation point, thus leading to a spatial Stroop interference. Interference tasks have been demonstrated to produce facilitation even at long CTOAs ([20]; see also in Ref. [38]). Therefore, in Van der Lubbe et al.'s study [67], the short range of CTOAs, and the use of a spatial Stroop task, might be responsible of the unusual facilitation observed at such a long CTOA.

As already mentioned, the IOR effect has been widely associated with a reduction in the amplitude of the P1 component for cued as compared to uncued locations independently of the task at hand (see e.g. [6,45,55,57,61,62,66–68]). Indeed, Satel et al. [62] performed a correlation analysis on the mean IOR scores, experiment-by-experiment, and the P1 modulation effects from 19 published experiments (see Ref. [62] Fig. 2). They reported a clear pattern of P1 reductions for cued as compared to uncued location trials associated with increased IOR scores ($r = -0.60$, $p < 0.05$, two-tailed), akin to what they reported in their own data ($r = -0.38$, $p < 0.05$, one-tailed). Thus, these authors claimed that the P1 modulation is closely associated with the IOR effect when the oculomotor system is suppressed. However, P1 reductions for cued as compared to uncued location trials have not been reported in other studies when IOR was behaviourally observed (see e.g. [22,27,48,57,63]). The P1 modulation has also been reported in the absence of significant IOR effects. For example, Hopfinger and Mangun [26] reported a significant P1 reduction for cued as compared to uncued location trials despite their failure to find a significant IOR effect using a discrimination task. In a detection task [27], they also showed a significant IOR effect with no modulation of the P1 component, concluding that the IOR effect is dissociable from the P1 modulation.

Differences in task parameters/characteristics could, nevertheless, explain such discrepancies in the previous results. Most studies showing the IOR effect without P1 reductions for cued as compared to uncued location trials have employed a vertical arrangement of stimuli in the visual field [22,57,63]. IOR has been associated with P1 modulation for cued as compared to uncued location trials on studies employing the most common horizontal arrangement of stimuli, where cues and targets appear on each side of the fixation point. Note that, as it has been pointed out by others (see e.g. [10,22]) different patterns of visual electrophysiological components could be recorded due to the orientation of the cerebral tissue involved in stimuli processing.

On the other hand, some discrepancies might also come from another important variable, CTOA duration. The longest CTOA usually employed in most of the previous studies has been 1200 ms (see Table 2). However, Satel et al. [63] and Gutiérrez-Domínguez et al. [22] used a CTOA of 1500 ms and 2000 ms, respectively. Both studies reported no significant modulations of P1 for cued as compared to uncued location trials associated with IOR. In fact, Gutiérrez-Domínguez et al. [22] argued that P1 effects could disappear at such long CTOAs. Yet, it is still unknown whether P1 modulations associated with IOR are no longer observed at CTOAs longer than 1200–1500 ms. Target duration might also be an important variable to consider (see Ref. [44]; for a review of modulations produced by the target duration). With the exception of Hopfinger and Mangun [27], all previous studies showing no modulations of P1 for cued as compared to uncued location trials associated with IOR used a target duration longer than 1000 ms or until response [22,48,57,63].

Therefore, stimuli arrangement in the visual field, as well as cue–target interactions, target duration, and cue–target interval, might be responsible of the discrepancies observed among studies about the P1 modulation for cued as compared to uncued location trials associated with the IOR effect.

In sum, there is not a definitive correspondence between the P1 modulation for cued as compared to uncued location trials and the cueing effect that is measured. Although with some above-reviewed exceptions ([22,27,57,63]), after a long enough CTOA (after > 500 ms), the P1 component is reduced for cued as compared to uncued locations no matter the behavioural effect that is measured (facilitation or IOR; see Table 2).

3.2. N1 component

The N1 component might be enhanced for cued as compared to uncued location trials in association with facilitatory effects [12,17,45]. However, this effect has also been reported in the absence of significant behavioural facilitation [12]; and several studies reporting significant behavioural facilitation effects have not subjected this component to statistical tests (see e.g. [6,26], see Table 2).

Other studies have associated the IOR effect with a reduced amplitude of the N1 component for cued as compared to uncued location trials (see e.g. [22,55–57,61,66]), although no modulations of the N1 component have been observed elsewhere (see e.g. [48,45,63,67,68]). While in the former case (reductions of the N1 component for cued as compared to uncued location trials associated with IOR) most studies used either discrimination or go-no go tasks (although see Refs. [57,61] using detection or localization tasks), in the latter case (absence of N1 modulations associated with IOR), detection or localization tasks were used [63,67,68]; although see Ref. [45], using a discrimination task). Thus, as it can be observed in Table 2, the N1 component modulation associated with either facilitation or IOR are not as usual as one could think, and if any, its observation seems to depend on some variables such as the task at hand.

More concretely, N1 modulations in detection and localization tasks reporting IOR effects have been mainly non-significant, not shown, or not subjected to statistical tests ([68,6,27,48,62,63] although see Ref. [57], reporting a significant N1 modulation and significant IOR effect). In contrast, significant N1 enhancements for cued as compared to uncued location trials associated with facilitation effects have been mainly observed in discrimination tasks [12,17,45]. Indeed, Eimer [17] reported significant N1 modulations when a discrimination task was performed (and either significant facilitation or non-significant IOR were behaviourally observed) as compared to a localization task, in which the modulations were not significant. This result is consistent with our previous work [45], in which significant N1 modulations were only observed when we found significant behavioural facilitation in a discrimination task. As shown in Table 2, this modulation of the N1 component was abolished, however, with the same task and temporal parameters, by presenting an intervening event between cue and target, producing a significant IOR effect.

The exception to the above-reviewed studies concerning task set is mainly found in some of the Prime and colleagues' studies ([55–57] although see also Ref. [66]). They demonstrate significant N1 reductions for cued as compared to uncued location trials associated with significant IOR effects in detection, localization (see Ref. [61] for a similar finding in a localization task), discrimination, and go-no go tasks. It is noteworthy that the modulations associated with the N1 component are more likely to be contralateral or bilateral to the target position (see e.g. [6,26,45,48,67,68]). However, in most of the Prime et al.'s experiments, N1 modulations were associated with either the left or right hemisphere, but unrelated to the target position. Moreover, most of these studies [55–57,61] have used a long CTOA (>800 ms), a long cue duration (>200 ms), or a long target duration (between 800 and 3000 ms). Thus, although it

is still unclear whether different manipulations can give rise to a different lateralization of the effect, and/or different effects themselves, differences in the experimental paradigm could be crucial for understanding these discrepant results.

In short, it is clear that the existing studies do not provide a consistent association between modulations of the N1component and the observed behavioural cueing effects, and if any, it seems highly dependent of some variables such as the task at hand.

3.3. Nd component

The Nd component is defined as a negative difference between cued and uncued location trials in the time period between ~220–300 ms post-stimulus (see e.g. [17,57]). Despite being present in most experiments, no matter the task at hand or the behavioural effect that is measured (facilitation or IOR), there is no agreement between studies about its topography, interpretation, and/or underlying mechanisms (see Table 2).

Some authors have reported a modulation of this component associated with facilitation effects at midline electrodes [16]. It has been related with selection or enhanced sensory processing for cued as compared to uncued location trials (see e.g. [16,17] see also Ref. [45], for similar finding and interpretations). Wascher and Tipper [68] also associated this component, observed at parietal electrodes, with facilitatory process that might be compensating for earlier perceptual inhibition at cued as compared to uncued location trials.

In contrast, other studies have related the Nd component with inhibitory effects at occipital electrodes (see e.g. [22,48,55–57,61,63]), potentially reflecting an association with the IOR effect [63]. In fact, in recent studies, Satel and colleagues have tried to determinate whether this component might be the more reliable electrophysiological correlate/marker of manual IOR after saccadic responses to the cue ([61]; see also Ref. [63]).

Inspection of Table 2 clearly reveals that with the available data, it is not safe to draw firm conclusions about this component, requiring more consistent additional research. For example, Prime and Jolicoeur [55] reported modulations of the Nd component but, in contrast to previous results [57], the Nd modulation was only significant when no intervening event was presented between the cue and target, and the IOR effect was not observed. Based on their own data, as well as in the results reported by Wascher and Tipper [68], wherein the Nd effect varied inversely with the magnitude of the IOR effect, these authors argued that it is likely that the processes underlying the Nd component are not directly involved in generating the IOR effect. Likewise, Prime and Ward [57] did not directly relate the processes underlying the Nd component with the IOR effect.

McDonald et al. ([48]; experiment 2) also reported significant behavioural IOR associated with an absence of modulations of the Nd component. They suggested that peripheral cues sometimes appear to reduce the amplitude of the P1 component for cued as compared to uncued location trials and some other times appear to lead to a cued-uncued difference (which they called Nd) that lasts longer than the P1 component (see also Ref. [57]). Moreover, Gutiérrez-Domínguez et al. [22] also showed a Nd modulation that depended on the visual field (only observed in the lower visual field). These authors interpreted this component as an N2-like effect, reflecting the re-focusing of spatial attention to previously inhibited locations.

Thus, taken all this evidence together, and given the variability of results and the different interpretations attributed to this component, we reckon it might be risky to consider the Nd component as the only electrophysiological correlate of the behaviourally observed facilitation or IOR effects.

3.4. P3 component

Although the P3 component is thought to reflect processes involved in stimulus evaluation, being usually considered as an index of the neural processing associated with task-relevant information [50,51], most previous studies have not tested modulations of this component neither associated with significant facilitation [17,67] nor IOR ([22,56,57,61,63,68]; see Table 2). Moreover, while a few studies have reported a facilitation effect associated with a significant P3 enhancement for cued as compared to uncued location trials [26,27,45], others have reported facilitation associated with significant P3 reductions for cued as compared to uncued location trials [6,17]. Significant IOR has been also reported in association with P3 enhancements for cued as compared to uncued location trials ([48], experiment 1; [55], although very often no modulations have been reported [6,27,45]; see Table 2).

Therefore, although some studies have reported P3 modulations for cued as compared to uncued location trials associated with either facilitation or IOR, this finding has not been consistently reported in the previous literature, and further research should be carried out before assuming strong conclusions about the P3 component and its link with cueing effects.

3.5. N2pc component

The N2pc component refers to an enhanced negativity, between 180 and 350 ms after stimulus onset, contralateral to the visual field where the target is presented as compared to the ipsilateral visual field (see Ref. [18], for a review). Although the N2pc is becoming an increasingly popular component for the investigation of attentional processes (see e.g. [18,19,24,33,36,46,69,70]), to the best of our knowledge, only a few electrophysiological studies have been published to date reporting N2pc modulations in relation to cueing effects (see Table 3). The N2pc has not traditionally been analyzed in Posner-like tasks because the component was thought to be dependent on the presence of distractors [18,36]. Thus, the component has been generally interpreted as the electrophysiological marker of the distractor-suppression (see Ref. [18] for a review; see also [36,69]).

However, beyond the long-lasting debate regarding the distractor-suppression vs. target-enhancement interpretation of the N2pc (see Ref. [18] for a review), the component has been recently related to the selection of task-relevant stimuli rather than to the inhibition of surrounding distracters (see e.g. [30,33,46]). As it can be observed in Table 1, the analysis of this component might be helpful to prove or refute attentional vs. perceptual hypotheses about IOR. While the attentional reorienting hypothesis presumes that spatial attentional selection will always be impaired at the cued location as compared to the uncued location (reflected in N2pc modulations), the attentional–perceptual hypotheses do not make such prediction. According to the later hypothesis, the most relevant process underlying the IOR effect is the perceptual impairment at cued locations as compared to uncued locations (reflected in the P1 component modulation for cued as compared to uncued location trials, and associated with a detection cost – according to the *cue-target event integration-segregation hypothesis* – [39]; or to an habituated response – according to the *habituation hypothesis* – [14]; see also Ref. [2], for a review).

Inspection of Table 3 reveals that the facilitation effect has been related to a N2pc enhancement for cued as compared to uncued location trials [45], while IOR has been related to a N2pc reduction or delay for cued as compared to uncued location trials [45,47,71]. This finding suggests that the inhibitory process/es underlying the IOR effect might impair the probability of target selection at recently attended locations. Indeed, to the extent of our knowledge, the component has been significantly reported in localization

Table 3
Summary of the N2pc modulations in previous electrophysiological studies.

Type task	Study	Task characteristics	SOA (in ms)	Cue duration (in ms)	Intervening event	Target duration (in ms)	Cueing effect (uncued–cued)	N2pc
Visual search	McDonald et al. [47]	Target-target IOR	1000–1400	(Target 1) 100	Always present	100	−18	Reduction (no change in latency)
Discrimination	Martín-Arévalo et al. [45]	Cue-target paradigm	500–700	50	Present	Until response	−11	Reduction
			500–700	50	Absent	Until response	10	Enhancement
Localization	Yang et al. [71]	Cue-target paradigm	500–1000	100	Absent	2000	−11	Delayed (no change in amplitude)

Reduction: smaller amplitude for cued than uncued location trials; Enhancement: larger amplitude for cued than uncued location trials. Grey background indicates facilitation effects.

[71], visual search [47], and/or discrimination tasks [45]. In all these tasks, target spatial selection is a necessary process for correct performance. Thus, this might be consistent with Eimer and Kiss' study [19] in which participants reported either the orientation of a uniquely colored target bar among other bars (a condition in which spatial selection is needed) or reported the orientation of target bars presented without other bars (a condition in which spatial selection is not needed). In the former case, they observed a cue-induced N2pc component while in the latter case they did not. Nevertheless, it is worth considering that the N2pc component, although unrelated to cueing effects, has also been observed in detection tasks (see e.g. [33,46]).

In short, the N2pc is the only component reflecting opposite and significant modulations in clear correlation with the behavioural effect that is measured (facilitation or IOR). Nevertheless, based on the small amount of data associating the N2pc component with cueing effects, strong conclusions thereon should be made with caution and further research should be carried out.

4. Correlation analyses

In order to quantitatively assess the relationship between the behavioural cueing effect and its associated electrophysiological modulation, we conducted a Pearson correlation analysis on the mean experiment-by-experiment behavioural cueing effect scores and the electrophysiological modulation of each target-locked component. We analyzed all the studies that have reported these values (i.e., P1, N1, Nd, and P3—we found no available data for N2pc). It is important to note that the correlation analyses of this section were only performed from studies that reported numerical values rather than from all existing studies in the literature (for example, absence of numerical values from [6,17,45,66,68]).

As can be observed in Fig. 1A, the analysis of the P1 modulation from the 23 published experiments from 10 published papers [11,26,27,55–57,61–63,67] revealed a significant negative correlation ($r = -0.75$; $p < 0.05$; two-tailed) between the cueing effect and the P1 modulation, suggesting that the larger the IOR effect, the larger the P1 reduction for cued as compared to uncued location trials (see Satel et al. [62], for a similar analysis and results). When we performed this correlation analysis by task, we only observed a significant negative correlation for discrimination tasks ($r = -0.71$; $p < 0.05$; two-tailed; see also Fig. 1A; for detection and localization tasks, both ps > 0.05—no available data for go-no go tasks).

As can be observed in Fig. 1A, the correlation seems to be driven by the opposition between experiments showing facilitation and a larger P1 for cued as compared to uncued locations trials, and those showing IOR and a reduced P1 for cued as compared to uncued locations trials. Given that all but one experiment [27] with detection or localization tasks show behavioural IOR (and a reduced P1 for cued as compared to uncued locations trials) no correlation might be observed for these tasks. In fact, for discrimination tasks, no significant correlation is observed either if only the experiments showing IOR are considered. Note that the different number of available studies for each task (namely, 11 vs. 7 and 4, for discrimination, detection and localization tasks, respectively) could also have an influence on this correlation analysis by task.

Fig. 1A demonstrates that reduced P1 for cued as compared to uncued locations trials seems to be a *necessary* condition to observe IOR, as all experiments reporting significant behavioural IOR showed a decrease in P1 amplitude (there is only one exception, [55] but demonstrating statistically null effects: a non-significant −1 ms cueing effect and a non-significant larger P1 for cued as compared to uncued locations trials). Nevertheless, P1 reduction might *not be sufficient*, as other factors might also have an influence, as some studies – unfortunately some of those not included in this correlation analysis – have shown facilitation instead of IOR even

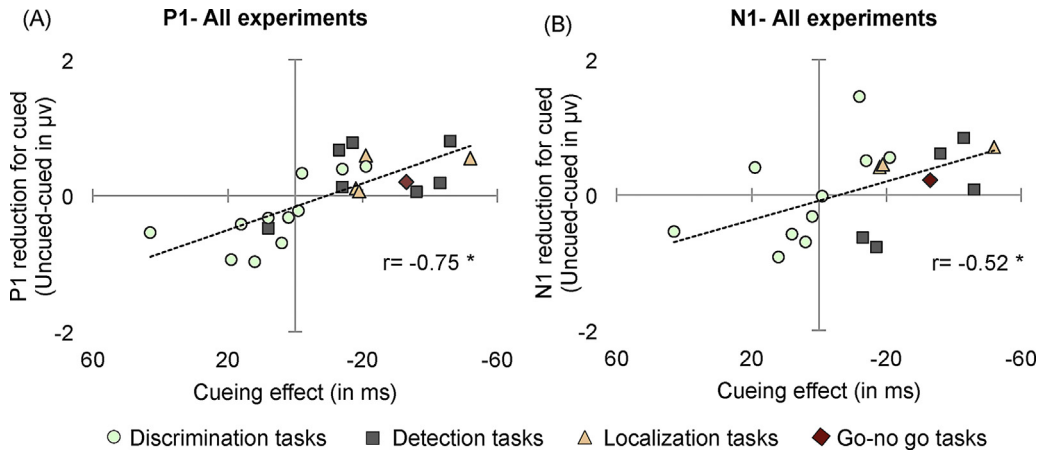

Fig. 1. Correlation analyses between behavioural cueing effect scores and (A) P1 cueing effects of 23 published experiments from 10 published papers [11,26,27,55–57,61–63,67]; (B) N1 cueing effects of 19 published experiments from 8 published papers [12,48,55–57,61,63,67]. Cueing effect: uncued-cued location trials. * represents significant effects ($p < 0.05$; two-tailed). Dashed lines in each graph indicate the trendline.

when a reduction in P1 for cued as compared to uncued location trials is observed (see e.g. [6,45,68]; see Table 2).

The analysis of the N1 modulation from the 19 published experiments from 8 published papers [12,48,55–57,61,63,67] also revealed a significant negative correlation ($r = -0.52$; $p < 0.05$; two-tailed) between the cueing effect and the N1 modulation, showing that the larger the N1 reduction for cued as compared to uncued location trials, the larger the IOR effect (see Fig. 1B). None of the correlations separately performed for each task reached significance (all ps > 0.05). However, it is noteworthy that, as previously mentioned, N1 modulations in detection and localization tasks reporting IOR effects have been mainly not reported or non-significant [6,27,48,62,63,68], which could be skewing this correlation analysis by task.

Finally, neither the analysis of the Nd modulation from the 11 published experiments from 7 published papers [22,48,55–57,61,63], nor the analysis of the P3 modulation from the 10 published experiments from 4 published papers [26,27,48,55] revealed significant correlations (all ps > 0.05).

Thus, replicating previous findings (see Ref. [62]), P1 modulations were found to be associated with cueing effects, but this relationship was only present in discrimination tasks, and disappeared when only experiments reporting IOR effects were considered. It is well known that, especially in discrimination task, the nature and size of the observed cueing effects highly depend on the CTOA, and other variables such as the temporal properties of cues and targets, or the presence/absence of intervening events. Therefore, the P1 modulation might index the process that is highly dependent on the task at hand, CTOA, and other factors, with positive modulations of P1 (increased amplitude for cued as compared to uncued location trials) when facilitation is observed (i.e., discrimination tasks, short CTOAs, long target durations, etc.) and negative modulations of P1 (reduced amplitude for cued as compared to uncued location trials) when IOR is observed (i.e., detection tasks, long CTOAs, short target durations, etc.). However, it is important to highlight that although the P1 reduction for cued as compared to uncued location trials seems to be a necessary condition to observe IOR, it is not sufficient, as P1 was not the only component associated with IOR: N1 also showed a (smaller) negative correlation with the behavioural effect that is measured. These results strengthen our conclusion that there is no single neural marker for facilitation and IOR because the net behavioural effect that is observed depends on the contribution of perceptual (P1), but also late-perceptual (N1) components. Although the existing studies do not allow for a significant quantitative analysis, many of the reviewed variables such as the task at hand, or the properties of the cues and targets, might determine the contribution of other components such as the Nd, N2pc, or P3.

5. Concluding remarks

Inspection of the Table 1, which summarizes the electrophysiological modulations hypothesized by both the traditionally dominant attentional reorienting and attentional-perceptual hypotheses, seems to indicate that the P1 component might be the crucial component to prove or refute the different hypotheses proposed to explain cueing effects. However, as previously noted (see also Ref. Martín-Arévalo et al. [45]), it is safe to conclude that, even if there is a significant correlation between P1 modulation and cueing effects, this component cannot be considered as the only neural marker underlying the cueing effect (facilitation or IOR).

On the one hand, this correlation might support the traditional idea that the reduction of P1 for cued as compared to uncued location trials is a direct electrophysiological measure of IOR, as IOR is observed when P1 is reduced for cued as compared to uncued location trials, and facilitation is observed when P1 is enhanced for cued as compared to uncued location trials. Thus, in detection and localization tasks, P1 is reduced for cued as compared to uncued location trials in most if not all cases, and IOR is usually observed. In discrimination tasks, however, facilitation is observed in some conditions together with an enhanced P1 for cued as compared to uncued location trials (such as short CTOAs, long cue/target durations, etc.), and IOR is observed in other conditions together with a reduced P1 for cued as compared to uncued location trials (such as long CTOAs, short cue/target durations, or by presenting an intervening event). Nevertheless, as stated above, this correlation might also be interpreted as showing that a P1 reduction for cued as compared to uncued location trials is a necessary but not sufficient condition to observe IOR.

It is worth noting that this correlation analysis was not performed from all existing studies in the literature (namely, absence of numerical values of the P1 modulation from Refs. [6,12,17,45,66,68]), which could skew the conclusions if we only rely on this type of analysis. Unfortunately, some of these studies might be critical to test whether the P1 reduction for cued as compared to uncued location trials might be not only necessary but also sufficient to behaviourally observe IOR.

Thus, with only one exception (Van der Lubbe et al. [67]), a key qualitative observation is that after a long enough CTOA (>500 ms), the P1 component is either reduced for cued as compared to

uncued locations or no significant modulations are reported (see e.g. [6,11,17,22,26,27,48,57,63]). The critical point here is that those P1 reductions for cued as compared to uncued location trials can be present no matter whether the observed behavioural result is positive (i.e., facilitation) or negative (i.e., IOR) (see e.g. [6,45]; see also Ref. [68], although facilitation was not significant; see Table 2). This finding is difficult to accommodate by the traditional attentional reorienting hypothesis (see Ref. [34] for a review) and the habituation hypothesis [14].

Nevertheless, it could be consistent with the cue-target event integration-segregation hypothesis (see Ref. [39] for a review), which postulates that target perceptual processing (as reflected by P1 modulations) does not reflect the behavioural IOR effect, but one of the mechanisms underlying the effect, the detection cost. According to this view, the detection cost (which could be likely indexed by P1 reductions for cued as compared to uncued location trials) would be negatively contributing to the cueing effect that is observed. Other mechanisms, however, triggered by the peripheral cue, might positively contribute to cueing effects. Thus, the net behavioural effect that is observed (either facilitation or IOR) should also depend on the contribution of other late-perceptual (N1, Nd), spatial selection (N2pc), and decision processes components (P3). The fact that both P1 and N1 component modulations correlated with IOR scores supports this claim.

Our review highlights the N2pc component as an important marker to understand the mechanism/s underlying the cueing effects even though there is little evidence to date. Further research is strongly encouraged in order to shed light on this long-lasting debate.

To recapitulate, the present paper provides a thorough qualitative and quantitative review of the electrophysiological modulations predicted by the main hypotheses about cueing effects (facilitation and IOR) and the most important electrophysiological studies. We have discussed important discrepancies between the theoretical predictions and the electrophysiological modulations that both facilitation and IOR produce by considering their boundary conditions, and we have analyzed the contribution of different components (P1, N1, Nd, P3, and N2pc) to the behavioral effect (facilitation or IOR). We conclude that, although P1 reductions for cued as compared to uncued location trials seem to be a necessary condition to observe IOR, there is no single neural marker for facilitation and IOR because many variables determine the electrophysiological modulations of the different mechanisms of attentional orienting (P1, N1, Nd, N2pc, and P3), which jointly define the spatial cueing effect that is observed. The mechanisms underlying spatial attentional effects – still debated by different spatial attention hypotheses – may be refined by incorporating these new findings.

6. Considerations and future directions

Most of the previous electrophysiological studies have reported measures of brain activity related to target processing, while electrophysiological modulations associated with cue processing have not been considered. To the best of our knowledge, only a few published papers have recently considered cue-related activations during the time interval between cue and target onset in the context of exogenous attention [4,5,65]. Tian et al. [65] is the only one specifically exploring facilitation and IOR in a cue-target paradigm. They report a complex pattern of cue-locked modulations (namely, C1, early P1, and early Nc2 modulations associated with facilitation, and Nc, P3, PL1, and PL2 modulations associated with IOR). In order to properly address the electrophysiological correlates of spatial attentional effects, electrophysiological activity locked to the appearance of the cue should also be explored in further studies to advance knowledge.

Moreover, given that the main limitation of electrophysiological measures such as ERPs relates to its low spatial resolution, null results (absence of modulations) should not be taken as an unequivocal indication of its absence, hence the importance of combining both quantitative and qualitative analyses. Thus, it might be possible that the behavioural IOR that is measured in detection tasks (especially when using very short target durations) comes from an earlier activity in subcortical structures such as the superior colliculus (SC), which critically contribute to IOR [1,13,58,60] in concert with other cortical structures [13].

Finally, we strongly emphasize that cross-study comparisons should be done with caution because previous electrophysiological studies significantly differed in their experimental parameters. The modulations of most electrophysiological components have been shown to depend on many variables such as the task at hand, the temporal properties of cues and targets, the presence/absence of intervening events, and stimuli arrangement in the visual field. Future systematic research will have to clarify how the combination of these variables modulates spatial attentional effects, while establishing their neural correlates. More research is also strongly encouraged concerning the N2pc component, which seems to be the only one reflecting opposite and significant modulations in clear correlation with facilitation and IOR.

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References

[1] E.J. Anderson, G. Rees, Neural correlates of spatial orienting in the human superior colliculus, J. Neurophysiol. 106 (5) (2011) 2273–2284.
[2] G. Berlucchi, Inhibition of return: a phenomenon in search of a mechanism and a better name, Cogn. Neuropsychol. 23 (7) (2006) 1065–1074.
[3] M. Cheal, G. Chastain, Efficiency of visual selective attention is related to the type of target, Psychol. Res. 66 (2) (2002) 110–115.
[4] A.B. Chica, F. Botta, J. Lupiáñez, P. Bartolomeo, Spatial attention and conscious perception: interactions and dissociations between and within endogenous and exogenous processes, Neuropsychologia 50 (5) (2012) 621–629.
[5] A.B. Chica, S. Lasaponara, J. Lupiáñez, F. Doricchi, P. Bartolomeo, Exogenous attention can capture perceptual consciousness: ERP and behavioural evidence, Neuroimage 51 (3) (2010) 1205–1212.
[6] A.B. Chica, J. Lupiáñez, Effects of endogenous and exogenous attention on visual processing: an inhibition of return study, Brain Res. 1278 (2009) 75–85.
[7] A.B. Chica, J. Lupiáñez, P. Bartolomeo, Dissociating inhibition of return from the endogenous orienting of spatial attention: evidence from detection and discrimination tasks, Cogn. Neuropsychol. 23 (7) (2006) 1015–1034.
[8] A.B. Chica, E. Martín-Arévalo, F. Botta, J. Lupiáñez, The Posner paradigm: how to design and interpret spatial attention experiments, Neurosci. Bio-Behav. Rev. 40 (2014) 35–51.
[9] S. Danziger, A. Kingstone, Unmasking the inhibition of return phenomenon, Percept. Psychophys. 61 (6) (1999) 1024–1037.
[10] F. Di Russo, A. Martinez, M.I. Sereno, S. Pitzalis, S.A. Hillyard, Cortical sources of the early components of the visual evoked potential, Hum. Brain Mapp. 15 (2) (2001) 95–111.
[11] S. Doallo, L. Lorenzo-Lopez, C. Vizoso, S.R. Holguin, E. Amenedo, S. Bara, et al., The time course of the effects of central and peripheral cues on visual processing: an event-related potentials study, Clin. Neurophysiol. 115 (1) (2004) 199–210.
[12] S. Doallo, L. Lorenzo-Lopez, C. Vizoso, S.R. Holguin, E. Amenedo, S. Bara, et al., Modulations of the visual N1 component of event-related potentials by central and peripheral cueing, Clin. Neuropsychol. 116 (4) (2005) 807–820.
[13] M.C. Dorris, R.M. Klein, S. Everling, D.P. Muñoz, Contribution of the primate superior colliculus to inhibition of return, J. Cogn. Neurosci. 14 (8) (2002) 1256–1263.
[14] K.R. Dukewich, Reconceptualizing inhibition of return as habituation of the orienting response, Psychonomic Bull. Rev. 16 (2) (2009) 238–251.
[15] K.R. Dukewich, R. Klein, Inhibition of return: a phenomenon in search of a definition and a theoretical framework, Atten. Percept. Psychophys. 77 (4) (2015) 1647–1658.

[16] M. Eimer, Spatial cueing, sensory gating and selective response preparation: an Erp study on visuospatial orienting, Electroencephalogr. Clin. Neurophysiol. 88 (5) (1993) 408–420.
[17] M. Eimer, An ERP study on visual spatial priming with peripheral onsets, Psychophysiology 31 (2) (1994) 154–163.
[18] M. Eimer, The N2pc component as an indicator of attentional selectivity, Electroencephalogr. Clin. Neurophysiol. 99 (3) (1996) 225–234.
[19] M. Eimer, M. Kiss, Involuntary attentional capture is determined by task set: evidence from event-related brain potentials, J. Cogn. Neurosci. 20 (8) (2008) 1423–1433.
[20] M.J. Funes, J. Lupiáñez, B. Milliken, Separate mechanisms recruited by exogenous and endogenous spatial cues: evidence from a spatial Stroop paradigm, J. Exp. Psychol. 33 (2) (2007) 348–362.
[21] S. Gabay, A.B. Chica, P. Charras, M.J. Funes, A. Henik, Cue and target processing modulate the onset of inhibition of return, J. Exp. Psychol. 38 (1) (2012) 42–52.
[22] F.J. Gutiérrez-Domínguez, P. Pazo-Álvarez, S. Doallo, L.J. Fuentes, L. Lorenzo-López, E. Amenedo, Vertical asymmetries and inhibition of return: effects of spatial and non-spatial cueing on behavior and visual ERPs, Int. J. Psychophysiol. 91 (2014) 121–131.
[23] T.C. Handy, A.P. Jha, G.R. Mangun, Promoting novelty in vision: inhibition of return modulates perceptual-level processing, Psychol. Sci. 10 (2) (1999) 157–161.
[24] C. Hickey, V. Di Lollo, J.J. McDonald, Electrophysiological indices of target and distractor processing in visual search, J. Cogn. Neurosci. 21 (2009) 760–775.
[25] M.D. Hilchey, R.M. Klein, J. Satel, Returning to inhibition of return by dissociating long-term oculomotor IOR from short-term sensory adaptation and other nonoculomotor inhibitory cueing effects, J. Exp. Psychol. Hum. Percept. Perform. 40 (4) (2014) 1603–1616.
[26] J.B. Hopfinger, G.R. Mangun, Reflexive attention modulates processing of visual stimuli in human extrastriate cortex, Psychol. Sci. 9 (6) (1998) 441–447.
[27] J.B. Hopfinger, G.R. Mangun, Tracking the influence of reflexive attention on sensory and cognitive processing, Cogn. Affective Behav. Neurosci. 1 (1) (2001) 56–65.
[28] J.B. Hopfinger, V.M. West, Interactions between endogenous and exogenous attention on cortical visual processing, Neuroimage 31 (2) (2006) 774–789.
[29] A.R. Hunt, A. Kingstone, Inhibition of return: dissociating attentional and oculomotor components, J. Exp. Psychol. Hum. Percept. Perform. 29 (5) (2003) 1068–1074.
[30] P. Jolicœur, P. Sessa, R. Dell'Acqua, N. Robitaille, On the control of visual spatial attention: evidence from human electrophysiology, Psychol. Res. 70 (6) (2006) 414–424.
[31] J. Jonides, Voluntary versus automatic control over the mind's eye's movement, in: J. Long, A. Baddeley (Eds.), Attention and Performance XI, Lawrence Erlbaum, Hillsdale, NJ, 1981, pp. pp. 187–283.
[32] A. Kingstone, J. Pratt, Inhibition of return is composed of attentional and oculomotor processes, Percept. Psychophys. 61 (6) (1999) 1046–1054.
[33] M. Kiss, J. Van Velzen, M. Eimer, The N2pc component and its links to attention shifts and spatially selective visual processing, Psychophysiology 45 (2008) 240–249.
[34] R.M. Klein, Inhibition of return, Trends Cogn. Sci. 4 (4) (2000) 138–147.
[35] R.M. Klein, M.D. Hilchey, Oculomotor inhibition of return, in: I.D.G.S. Liversedge, S. Everling (Eds.), The Oxford Handbook of Eye Movements, Oxford University Press, Oxford, 2011, pp. 471–492.
[36] S.J. Luck, S.A. Hillyard, Electrophysiological correlates of feature analysis during visual search, Psychophysiology 31 (1994) 291–308.
[37] S.J. Luck, G.E. Woodman, E.K. Vogel, Event-related potential studies of attention, Trends Cogn. Sci. 4 (2000) 432–440.
[38] C. Luo, J. Lupiáñez, M.J. Funes, X. Fu, Reduction of the spatial stroop effect by peripheral cueing as a function of the presence/absence of placeholders, PloS One 8 (7) (2013) e69456.
[39] J. Lupiáñez, Inhibition of return, in: A.C. Nobre, J.T. Coull (Eds.), Attention and Time, Oxford University Press, Oxford, UK, 2010, pp. 17–34.
[40] J. Lupiáñez, R.M. Klein, P. Bartolomeo, Inhibition of return: twenty years after, Cogn. Neuropsychol. 23 (7) (2006) 1003–1014.
[41] J. Lupiáñez, E. Martín-Arévalo, A.B. Chica, Inhibition of return: attentional disengagement or detection cost? Psicológica 34 (2013) 221–252.
[42] J. Lupiáñez, E.G. Milán, F.J. Tornay, E. Madrid, P. Tudela, Does IOR occur in discrimination tasks? Yes, it does, but later, Percept. Psychophys. 59 (8) (1997) 1241–1254.
[43] J. Lupiáñez, B. Milliken, C. Solano, B. Weaver, S.P. Tipper, On the strategic modulation of the time course of facilitation and inhibition of return, Q. J. Exp. Psychol. A 54 (3) (2001) 753–773.
[44] E. Martín-Arévalo, A.B. Chica, J. Lupiáñez, Task dependent modulation of exogenous attention: effects of target duration and intervening events, Attent. Percept. Psychophys. 75 (6) (2013) 1148–1160.
[45] E. Martín-Arévalo, A.B. Chica, J. Lupiáñez, Electrophysiological modulations of exogenous attention by intervening events, Brain Cogn. 85 (2014) 239–250.
[46] V. Mazza, M. Turatto, A. Caramazza, Attention selection, distractor suppression and N2pc, Cortex 45 (7) (2009) 879–890.
[47] J.J. McDonald, C. Hickey, J.J. Green, J.C. Whitman, Inhibition of return in the covert deployment of attention: evidence from human electrophysiology, J. Cogn. Neurosci. 21 (4) (2009) 725–733.
[48] J.J. McDonald, L.M. Ward, K.A. Kiehl, An event-related brain potential study of inhibition of return, Percept. Psychophys. 61 (7) (1999) 1411–1423.
[49] B. Milliken, T. Lupiáñez, M. Roberts, B. Stevanovski, Orienting in space and time: joint contributions to exogenous spatial cueing effects, Psychonomic Bull. Rev. 10 (4) (2003) 877–883.
[50] J. Polich, Updating P300: an integrative theory of P3a and P3b, Clin. Neurophysiol. 118 (2007) 2128–2148.
[51] J. Polich, P.C. Ellerson, J. Cohen, P300, stimulus intensity, modality, and probability, Int. J. Psychophysiol. 23 (1996) 55–62.
[52] M.I. Posner, Orienting of attention, Q. J. Exp. Psychol. 32 (1980) 3–25.
[53] M.I. Posner, Y. Cohen, Components of visual orienting, in: H. Bouma, D. Bouwhuis (Eds.), Attention and Performance X, Lawrence Erlbaum, London, 1984, pp. 531–556.
[54] M.I. Posner, R.D. Rafal, L.S. Choate, J. Vaughan, Inhibition of return: neural basis and function, Cogn. Neuropsychol. 2 (1985) 211–228.
[55] D.J. Prime, P. Jolicoeur, On the relationship between occipital cortex activity and inhibition of return, Psychophysiology 46 (6) (2009) 1278–1287.
[56] D.J. Prime, L.M. Ward, Inhibition of return from stimulus to response, Psychol. Sci. 15 (4) (2004) 272–276.
[57] D.J. Prime, L.M. Ward, Cortical expressions of inhibition of return, Brain Res. 1072 (1) (2006) 161–174.
[58] R.D. Rafal, M.I. Posner, J.H. Friedman, A.W. Inhoff, E. Bernstein, Orienting of visual attention in progressive supranuclear palsy, Brain 111 (Pt 2) (1988) 267–280.
[59] M. Ruz, J. Lupiáñez, A review of attentional capture: on its automaticity and sensitivity to endogenous control, Psicológica 23 (2002) 283–309.
[60] A. Sapir, N. Soroker, A. Berger, A. Henik, Inhibition of return in spatial attention: direct evidence for collicular generation, Nat. Neurosci. 2 (12) (1999) 1053–1054.
[61] J. Satel, M.D. Hilchey, Z. Wang, C.S. Reiss, R.M. Klein, In search of a reliable electrophysiological marker of oculomotor inhibition of return, Psychophysiology 51 (10) (2014) 1037–1045.
[62] J. Satel, M.D. Hilchey, Z. Wang, R. Story, R. Klein, The effects of ignored versus foveated cues upon inhibition of return: an event-related potential study, Attent. Percept. Psychophys. 75 (1) (2013) 29–40.
[63] J. Satel, Z. Wang, M.D. Hilchey, R.M. Klein, Examining the dissociation of retinotopic and spatiotopic inhibition of return with event-related potentials, Neurosci. Lett. 524 (2012) 40–44.
[64] T.L. Taylor, R. Klein, Visual and motor effects in inhibition of return, J. Exp. Psychol. Hum. Percept. Perform. 26 (5) (2000) 1639–1656.
[65] Y. Tian, R.M. Klein, J. Satel, P. Xu, D. Yao, Electrophysiological explorations of the cause and effect of inhibition of return in a cue-target paradigm, Brain Topogr. 24 (2) (2011) 164–182.
[66] Y. Tian, D. Yao, A study on the neural mechanism of inhibition of return by the event-related potential in the Go/Nogo task, Biol. Psychol. 79 (2) (2008) 171–178.
[67] R.H. Van der Lubbe, R.O. Vogel, A. Postma, Different effects of exogenous cues in a visual detection and discrimination task: delayed attention withdrawal and/or speeded motor inhibition? J. Cogn. Neurosci. 17 (12) (2005) 1829–1840.
[68] E. Wascher, S.P. Tipper, Revealing effects of noninformative spatial cues: an EEG study of inhibition of return, Psychophysiology 41 (5) (2004) 716–728.
[69] G.F. Woodman, S.J. Luck, Electrophysiological measurement of rapid shifts of attention during visual search, Nature 400 (1999) 867–869.
[70] G.F. Woodman, S.J. Luck, Dissociations among attention, perception, and awareness during object-substitution masking, Psychol. Sci. 14 (6) (2003) 605–611.
[71] D. Yang, S. Yao, C. Ding, S. Qi, Y. Lei, Electrophysiological evidence for inhibition of return effect in exogenous orienting, Exp. Brain Res. 221 (2012) 279–285.