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Inhibitory processing following damage to the parietal lobe

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Abstract

We investigated inhibitory properties of spatial attention in a group of four patients with lesions involving the posterior parietal lobe. In a first experiment, a double cue inhibition of return (IOR) procedure was employed. The parietal patients showed an IOR effect only when they had to detect targets that appeared on the contralesional side. In a second experiment, we combined an IOR procedure with a Stroop task [Psychon. Bull. Rev. 8 (2001) 315] to explore the neural basis of "inhibitory tagging" as described by Fuentes et al. [Q. J. Exp. Psychol. Hum. Exp. Psychol. 52 (1999) 149]. The results from the control participants replicated the findings of Vivas and Fuentes, Stroop interference was reduced at the cued location, relative to the uncued location. The parietal patients showed a similar result, but only for contralesional targets. These findings suggest that IOR is modulated by the parietal lobe, and that, through this process, the parietal cortex influences the application of inhibitory tagging to stimuli.

Keywords: IOR; Visuospatial attention; Contralesional; Extinction

1. Introduction

Posner and Cohen (1984) first demonstrated the existence of inhibitory processing in spatial attention. They found that RTs to a peripheral target were slowed by the earlier presentation of an irrelevant cue about 800 ms or so prior to the target. This phenomenon, known as "inhibition of return" (IOR) has since been studied extensively (see Klein, 2000, for a review). Initial studies emphasised the importance of ocular-motor programs in causing IOR and the possible dependence of this effect on midbrain structures such as the superior colliculus (Abrams & Dobkin, 1994; Posner, Rafal, Choate, & Vaughan, 1985; Rafal, Calabresi, Brennan, & Sciolto, 1989). However, more recent studies suggest that IOR can influence a wide range of cognitive processes, as evidenced by IOR effects in a range of discrimination tasks (Chasteen & Pratt, 1999; Fuentes, Vivas, & Humphreys, 1999a; Lupiañez, Milan, Tornay, Madrid, & Tudela, 1997; Pratt, 1995; Vivas & Fuentes, 2001). Such effects may be mediated by higher level neural areas, including parietal cortex (Bartolomeo, Sieroff, Decaix, & Chokron, 2001).

The early work on IOR assessed the questions of when IOR occurs, and on what tasks, but the question of *how* IOR influences performance has been relatively unexplored. In order to asses how IOR affects processing, we have combined IOR procedures with experimental paradigms sensitive to particular levels of stimulus processing, such as semantic priming and flanker interference (sensitive to response competition between stimuli; Fuentes, Vivas, & Humphreys, 1999b). When the prime or the flanker stimuli were presented at locations subject to IOR (i.e. the cued location, with a long interval between the cue and the stimuli) the standard effects of semantic priming and flanker interference reversed, compared with when stimuli were presented at neutral (uncued) locations. We proposed that the reversal of these effects was due to a process of inhibitory tagging brought about by IOR for previously attended locations. Inhibitory tagging temporarily sets an inhibitory link between the activated representations of stimuli at locations subject to IOR and their associated responses. For example, in a flanker interference paradigm requiring responses to the category of a target stimulus, this can mean that an inhibitory link is set between the category of the flanker (at a location subject to IOR) and its associated response. This slows performance when the target and the flanker belong to the same category and it can eliminate interference when the target and the flanker belong to different categories. Note that in these experiments we did not use a direct measure of IOR. Thus, for example in the flanker task, participants

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responded to central targets flanked by distractors presented at either cued or uncued locations. Under these circumstances a mechanism of inhibitory tagging would be effective at keeping separate the activation from the target and the distractor, by binding them to different spatial locations.

Since this tentative hypothesis was proposed (Fuentes et al., 1999b) we have replicated these striking results with different stimuli and tasks (Fuentes, Boucart, Vivas, Alvarez, & Zimmerman, 2000; Vivas & Fuentes, 2001; Vivas, Fuentes, Estevez, & Humphreys, submitted for publication-a). In one study, we combined flanker interference from multiple distractor features with an IOR procedure (Vivas et al., submitted for publication-a; Vivas, Fuentes, & Humphreys, submitted for publication-b). Participants had to respond to the colour of a central stimulus that could vary in colour or shape (a circle or a triangle in red or green). Along with the target, a distractor stimulus could appear at the previously cued or uncued location. The results replicated our previous findings, that flanker interference was reversed when the distractor appeared at a cued location. Importantly, flanker interference was inverted only for distractors that differed from the target in colour. This indicates that inhibitory tagging is mainly applied to task-relevant features of the stimulus (in this case colour but not shape). Furthermore, we have observed that IOR also modulated the interference effect produced by incongruent stimuli in a Stroop task (Vivas & Fuentes, 2001). There was a significant reduction in Stroop interference when the colour word fell at inhibited locations (Experiment 1; Vivas & Fuentes, 2001). In a second experiment, we separated the task-relevant and -irrelevant dimensions (the colour and the word) and found that Stroop interference was eliminated when the word fell at the cued (inhibited) location. Indeed, RTs were speeded on word–colour incongruent trials when the word appeared at the cued relative to the uncued location. To account for these results, we suggested that inhibitory tagging from IOR particularly affects stimulus-response codes that are normally derived rapidly (e.g. a naming response to a word rather than to a colour). By temporarily disconnecting stimuli from a response code, incongruent word responses are prevented from competing with the response to the (later derived) colour name, reducing Stroop interference. This argument enables us to extend the account of inhibitory tagging to situations in which response relevant and irrelevant information are part of the same object.

In an attempt to explore inhibitory tagging in schizophrenia, Fuentes et al. (2000) carried out a similar experiment to Vivas and Fuentes (2001). The results from the control group of healthy adults replicated the main findings of the previous study, i.e. Stroop interference was eliminated when stimuli appeared at cued locations. However, although schizophrenic patients exhibited both normal IOR and normal Stroop effects, as compared to the control group, Stroop interference was not modulated at cued locations. Fuentes et al. (2000) explained the lack of interaction as a failure in the inhibitory tagging mechanism in the schizophrenic group. Cognitive deficits in schizophrenia have been associated with dysfunction of high-level attentional processes depending on anterior cortical structures (Posner & DiGirolamo, 1998) and specifically, with a dysregulation of the anterior cingulate cortex (DiGirolamo & Posner, 1996). The lack of inhibitory tagging in these patients then may reflect impaired operation of anterior cortical areas. Consistent with this, we have obtained similar results in a patient with an organic frontal lesion (Vivas et al., submitted for publication-a, submitted for publication-b). Thus, these findings suggest that IOR and inhibitory tagging are dissociable (IOR but not inhibitory tagging occurring in schizophrenics and a frontal lobe damaged patient), though the two processes normally interact to regulate stimulus processing.

2. Neural regions mediating inhibitory processing

Converging evidence from early studies with healthy adults and patients with neurological damage suggested a strong link between subcortical structures subserving oculomotor programs and IOR. For example, Posner et al. (1985) employed a procedure similar to Posner and Cohen (1984), to explore IOR in a group of five frontal lobe patients, four Parkinson, seven patients with parietal lesions and six patients with progressive supranuclear palsy (PSP; affecting the superior colliculus). They found that the parietal and frontal patients had a normal IOR effect for a cue-target interval of 1000 ms. In contrast, IOR was eliminated in the group of patients with PSP. Posner et al. (1985) concluded that high-level cortical processes are not involved in IOR, which is mediated by lower level structures such as the superior colliculus (Sapir, Soroker, Berger, & Henik, 1999). Also, IOR appeared to be more strongly generated by cues presented in the temporal hemifield than by cues presented in the nasal field, in healthy individuals (Rafal et al., 1989). This pattern of asymmetry suggests again that the generation of IOR may be mediated by the superior colliculus.

More recent work however, has reported fMRI activation in cortical areas associated with IOR. Rosen et al. (1999) reported significant cortical activation in the dorsal premotor area, frontal eye field, superior parietal cortex and subcortical activation in the thalamus associated with IOR in a double cue paradigm. They also found activation in other areas such as the anterior cingulate, the temporoparietal junction and the cerebellum. Lepsien and Pollman (2002) have also reported activation in the supramarginal gyrus under conditions of IOR in an fMRI study. This activation was additional to activation in brain areas involved in oculomotor programming (the supplementary motor area and the frontal eye fields). Surprisingly, both studies found no evidence of collicular activation associated with IOR. However, whether higher cortical areas are involved in producing IOR itself, or in modulating the effects of re-orienting attention when IOR

has been applied, remains unclear. Other studies have also shown that parietal damage can be associated with reduced IOR. For example, Bartolomeo and colleagues have reported various disturbances in IOR in patients with unilateral neglect associated with parietal damage. Bartolomeo, Chokron, and Sieroff (1999) used a paradigm in which patients made responses to successive targets appearing at the same or different locations; they found a positive advantage for repeated ipsilesional targets. Bartolomeo et al. (2001) had patients respond to a single target, which followed a previously presented spatial cue. They also found a non-significant positive advantage for ipsilesional targets at the cued location under temporal conditions that would normally generate IOR. This finding, of a failure to show IOR to an ipsilesional (rather than a contralesional) target, suggests that at least part of the deficit may be in suppressing a strong ipsilateral orienting response after parietal damage (e.g. Ladavas, Petronio, & Umiltà, 1990; Shalev & Humphreys, 2000) or in disengaging attention from the ipsilesional cue (Posner, Walker, Friedrich, & Rafal, 1984). The parietal lobe may operate as part of an orienting network concerned both with orienting attention to the locations of targets and with biasing attention away from old locations (IOR). A unilateral parietal lesion may bias this network so that a strong orienting response to the ipsilesional side over-rules any IOR applied there. The net effect is a failure to demonstrate IOR on the ipsilesional side. This is then of interest for understanding the impact of IOR on higher order cognitive processes—the focus of the present study.

We examined IOR in a group of patients who had suffered damage to the parietal lobe. In both experiments IOR was induced using the double cue procedure of Posner and Cohen (1984), where attention is initially cued to the periphery and then back to the centre by a central cue, with IOR usually then being observed for peripheral targets. In Experiment 1, the task was to make a simple reaction time response to a peripheral target. In Experiment 2, IOR was combined with a Stroop task. Patients had to name the hue of a colour word presented at a previously uncued or cued location (and so subject to IOR in the latter case). With our patients, impairment in IOR was demonstrated with ipsilesional targets (see also Bartolomeo et al., 1999, 2001). In Experiment 2, we test whether any impairment in IOR after parietal damage also leads to changes in inhibitory tagging in higher level tasks.

3. Experiment 1: inhibition of return (1000 ms SOA)

3.1. Method

3.1.1. Participants

Sixth healthy adults participated in this experiment. They ranged in age from 41 to 62, with a mean of 45.5 years. They all had normal or corrected-to-normal vision. We examined four patients, all with lesions affecting the inferior parietal lobe. Three had unilateral lesions (DB, MB and MH), and one had bilateral lesions that were more severe in the left relative to the right hemisphere (FL). This last patient showed a lateralised pattern of performance, and so will be presented with right stimuli labelled as appearing on the contralesional side. Clinical details of the patients are presented in Table 1, and transcriptions of their lesions are shown in Fig. 1. At the time of testing, no patient presented with a clinical pattern of unilateral spatial neglect, though all manifested extinction under bilateral double simultaneous stimulation with brief presentations.

3.1.2. Stimuli

Stimuli were presented on a colour monitor (VGA) of an IBM/PC compatible computer, and responses were recorded through the computer keyboard. The stimuli consisted of a string of four Xs. At a viewing distance of 60 cm each character was 0.48 high and 0.38 wide degrees of visual angle. The target stimulus appeared inside of the peripheral boxes. Each box subtended 2.58 × 4.49 degrees of visual angle. The distance between the peripheral box and the fixation point was 4.30 degree of visual angle. The software used for creating and running the experiment was Micro Experimental Laboratory (MEL; Schneider, 1988). Participants had to press the space bar as soon as they saw the target stimulus.

3.1.3. Procedure

On each trial, a central fixation plus appeared for 500 ms, followed by three white boxes for 1000 ms (see Fig. 2). Then, one of the lateral boxes thickened (the peripheral cue) for 300 ms. After an interval of 200 ms with three white boxes, the central box thickened (the central cue) for 300 ms, followed by a further inter-stimulus interval (ISI) of 200 ms before the target was presented. The target appeared on 86% of the trials and was absent in the remaining 14% of the trials. The target consisted of a string of Xs and participants were

Table 1
Age, sex, aetiology, location of the lesion, and neurological signs of the four patients who served as participants in Experiments 1 and 2

	Age/sex	Aetiology	Location	Neurological signs
FL	64/M	Carbon monoxide poisoning	Bilateral globus pallidus damage, lateral occipital damage (bilateral), left inferior parietal lobe	Amnesia, attentional dyslexia, initially agnosic (recovered), extinction
MH	48/M	Anoxia	Left inferior parietal, angular gyrus	Extinction, mislocalization
DB	62/M	Stroke	Left middle and superior temporal, sylvian fissure, angular gyrus, inferior parietal	Anomia, extinction
MB	59/F	Stroke	Right inferior frontal, superior temporal gyri, inferior parietal (including the supramarginal gyrus)	Initially neglect (recovered), extinction

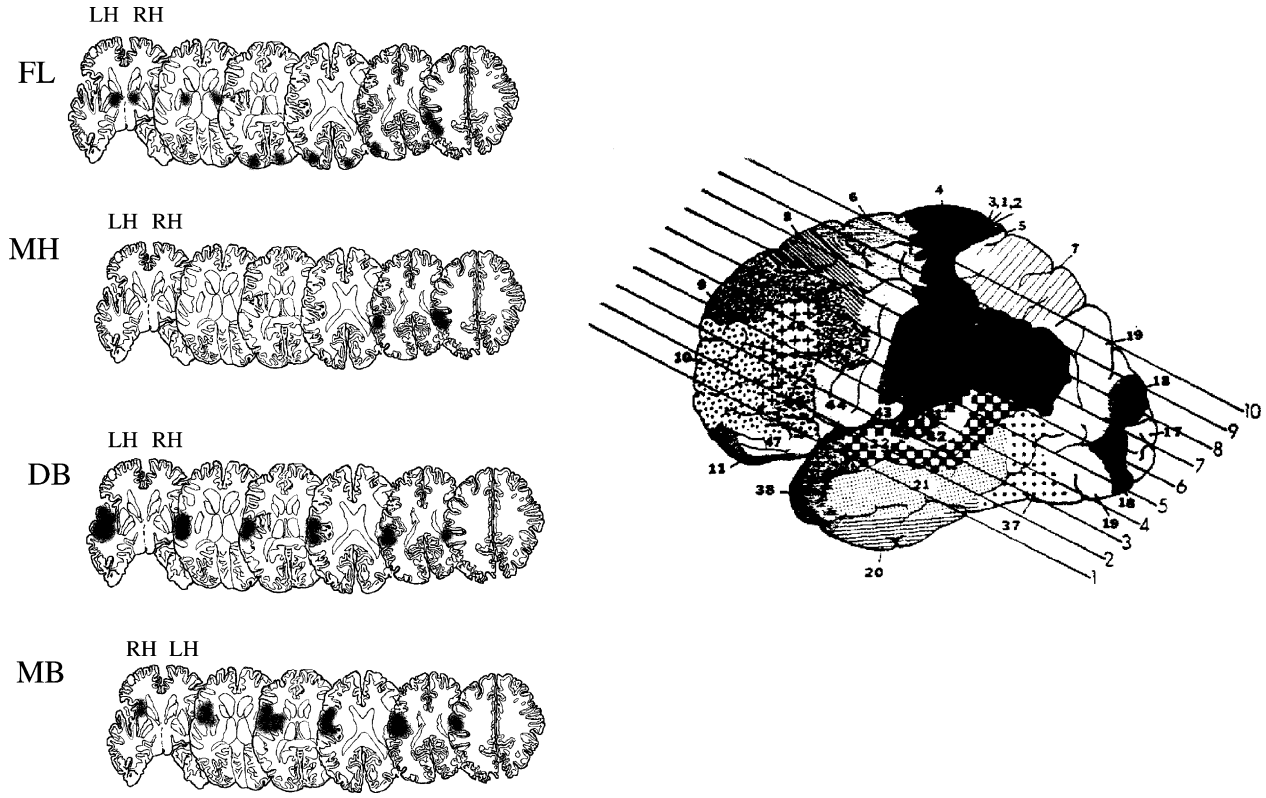

Fig. 1. MRI scans plotted onto standardised slices of the four patients (FL, MH, DB, MB) who served as participants in Experiments 1 and 2. The standardised plates are taken from Gado, Hanaway, and Frank (1979). Only slices three to eight are depicted here. For MH, FL and DB, the left side of each slice represents the left hemisphere (LH). For MB, the left side represents the right hemisphere (RH).

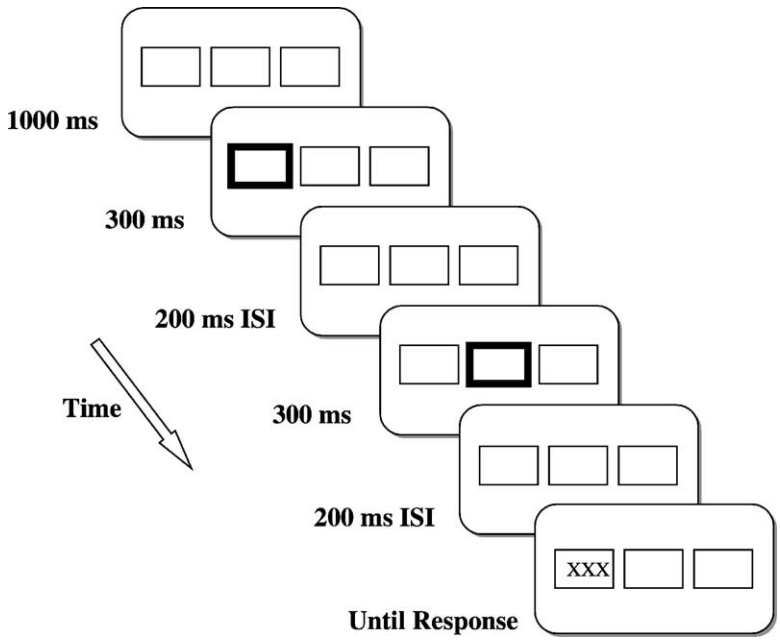

Fig. 2. Sequence of events and exposure duration of stimuli for a cued trial in Experiments 1 and 2.

Table 2
The mean of median correct RTs as a function of group, field and location in Experiment 1

Location	Control participants		Parietal patients	
	Left field	Right field	Ipsilesional field	Contralesional field
Cued	466	457	427	488
Uncued	436	423	434	440

Table 3
Individual means for correct RTs as a function of field and location, for the group of patients, in Experiment 1

	Ipsilesional field			Contralesional field		
	Cued	Uncued	IOR effect	Cued	Uncued	IOR effect
MH	372	367	5	386	346	40
FL	504	562	−58	575	560	15
DB	418	398	20	511	428	83
MB	416	410	6	481	426	55

asked to press the space bar as soon as they saw the stimulus. On trials without a target participants were instructed not to respond.

The patients ran one practice block of 24 trials followed by one experimental block of 102 trials twice in two different sessions. In the experimental block the target was presented on 88 trials (86%). On half of these trials (44), the target appeared in the left hemifield, and on the remaining trials it fell in the right hemifield. Also, for each hemifield, the target was presented at a validly cued location on half the trials (22), and at invalidly cued location on the other trials.

3.2. Results and discussion

The mean of the median correct RTs for the control group are shown in Table 2. Correct RTs were submitted to a repeated measures ANOVA with field (left and right) and location (cued and uncued) as within subject factors. The results showed a main effect of location, $F(1, 5) = 9.53$, $P < 0.05$. RTs were higher for the cued location (461 ms) than for the uncued location (429 ms). No other effects reached statistical significance, $P > 0.05$.

For the patients the means of the median correct RTs are shown in Table 2. Correct RTs were submitted to a repeated measures ANOVA with field (ipsilesional and contralesional), and location (cued and uncued) as within subject factors. None of the main effects of field or location were significant, Ps > 0.05. There was, however, a significant field × location interaction, $F(1, 3) = 42.18$, $P < 0.05$. The analysis of the simple effects showed a significant effect of IOR (48 ms) in the contralesional hemifield, $F(1, 3) = 11.84$, $P < 0.05$, but this effect did not emerge in the ipsilesional hemifield (−7 ms), $F < 1$.

There were no errors, by either the patients or the controls.

Control participants showed a standard IOR effect that did not interact with visual field. In contrast, for the group of parietal patients, IOR did not emerge for targets in the ipisilesional field. Three of the patients showed a reduced non-significant advantage of invalid over valid trials in the ipsilesional relative to the contralesionl field, and one patient showed a tendency for a facilitation effect in the ipsilesional field (patient FL, see Table 3). The difference in the magnitude of IOR in the ipsilesional and contralesional fields is unlikely to reflect the contrast in overall RTs (since RTs to ipsilesional targets are overall faster, any IOR effects could be lessened). On average, RTs were slowed to contralesional targets on invalid trials by 1.4% (taking RTs to ipsilesional targets on invalid trials as a baseline). In contrast, the difference in IOR effects across the two fields, relative to the same baseline, was 12.4%. This held for each patient (for MH there was a 6.3% difference in RTs across the fields on invalid trials, whilst the change in the magnitude of IOR was 9.5% relative to the invalid, ipsilesional baseline; for FL the comparative figures were −0.3 and 13%; for DB the figures were 7.5 and 15.8%, and for MB: 3.9 and 12%).

Our result differs from the original report on IOR in parietal patients (Posner et al., 1985), but matches the data of Bartolomeo et al. (1999, 2001) who also failed to find IOR for ipsilesional targets. However, there are two important differences between the results found in the present study and the findings of Bartolomeo et al. First, Bartolomeo et al. found that repeated events showed facilitation instead of inhibition for ipsilesional targets. Second, this pattern of results held only for those patients who showed clinical signs of neglect, whereas patients with lesions in the temporal, parietal and frontal lobe but without neglect exhibited IOR for both fields (Bartolomeo et al., 1999). The authors attributed the lack of IOR in the ipsilesional field to two possible deficits in patients with neglect; a strong facilitatory process that would mask inhibition and a difficulty in disengaging their attention from ipsilesional cues in order to respond to contralesional targets (Bartolomeo et al., 1999, 2001). That is, as previous studies have reported, parietal patients show a selective bias towards ipsilesional objects (Posner et al., 1984). Importantly, our data suggest that impaired IOR for ipsilesional target can be observed also in parietal patients without clinical signs of neglect. One possibility is that the presence of neglect in the parietal patients in Bartolomeo et al. study may explain why they found facilitation while we found lack of inhibition (only one patient showed a tendency for facilitation) in the ipsilesional field. Thus, parietal patients with neglect may have a stronger bias towards the ipsilesional side, producing stronger facilitation for targets presented in this side. However, a problem with this account is why then the group of patients without neglect, in Bartolomeo et al. (1999) study, did show normal IOR in both fields. One explanation for this apparent contradiction is that any tendency for reduced IOR in the ipsilesional field after parietal lesions could have been masked by the presence of lesions in other brain areas (unfortunately

Bartolomeo et al., 1999, did not report individual data in their study).

A second alternative explanation for the present data may depend on the lack of eye movement monitoring in our study. It is possible that patients could have made saccades toward the ipsilesional field, so that targets would have appeared at fixation on valid trials. However, rather than just producing a lack of IOR, such a strategy should generate a positive facilitation effect for validly cued, ipsilesional targets (since an eye movement should be made to these items on valid but not invalid trials). Our results did not confirm this. Three patients showed a reduction of IOR instead of facilitation, and only one patient showed a tendency for facilitation. Furthermore, the standard deviation for the ipsilesional valid condition (S.D. = 55) was not higher than for the contralateral valid condition (S.D. = 79), as one might expect if the patients made saccades towards the ipsilesional cues on some trials.

Consequently, we propose that the failure to observe IOR for ipsilesional targets in the group of parietal patients can be explained in terms of ipsilesional covert orienting and/or slowed disengagement of the attention from ipsilesional cues in order to respond to contralateral cues. Studies with Alzheimer's disease (Faust & Balota, 1997) have suggested that central cues might help participants to reorient their attention towards the centre, in a double cue paradigm (see also, Abrams & Dobkin, 1994; Posner & Cohen, 1984). However, in the present experiment patients with parietal damage did not benefit from the use of a central cue. Indeed, the second cue could have added a second difficulty in disengaging their attention from a prior stimulus (in this case the central cue) towards the contralesional target (Bartolomeo et al., 2001). Thus, the attentional orienting processes needed to observe IOR in onset detection tasks seem to be affected in patients with parietal damage. This then enables us to examine the effect of IOR on other processes, in Experiment 2. Here we evaluated how parietal damage affected inhibitory tagging limited to IOR (Fuentes et al., 1999b).

4. Experiment 2: inhibitory tagging

In this study we employed the procedure used by Vivas and Fuentes (2001) to generate inhibitory tagging. The participants were required to name the colour of a target that could appear in a previously cued or uncued location. The target could be a string of Xs or an incongruent colour word. According to our previous studies (Fuentes et al., 2000; Vivas & Fuentes, 2001) we should find a reduction in Stroop interference at cued locations for control participants. In contrast, given the evidence for IOR being affected by parietal damage (Experiment 1), we should again find an interaction between IOR and visual field for the parietal patients. There should be inhibitory tagging to targets in the contralesional field (e.g. reduced Stroop interference), but inhibitory tagging may be less effective in the ipsilesional field (i.e. the net effect should be increased Stroop interference for targets in the ipsilesional relative to the contralesional field). This would provide evidence for IOR, modulated by the parietal lobe, being necessary to generate inhibitory tagging. Importantly, though, it would indicate that parietal lobe is not the site of inhibitory tagging itself, if there is reduced Stroop interference for contralesional targets. Inhibitory tagging may remain possible but fails to be triggered by the lack of IOR to ipsilesional targets.

4.1. Method

4.1.1. Participants

Five healthy adults participated in this experiment. They ranged in age from 31 to 54, with a mean of 41 years. They had normal or corrected-to-normal vision. The patients were the same as those in Experiment 1.

4.1.2. Stimuli

The stimuli were presented on a colour monitor (VGA) of an IBM/PC compatible computer, and responses were recorded through the computer keyboard. The stimuli consisted of a string of four Xs (coloured red, green or blue) and the colour words RED, GREEN and BLUE presented in (respectively) blue or green, red or blue, and red or green. At a viewing distance of 60 cm each character was 0.48 high and 0.38 wide degrees of visual angle. The software used for creating and running the experiment was MEL (Schneider, 1988). Participants were asked to name the colour of target stimulus. Reaction time responses were recorded through a voice key attached to the computer. Errors were registered by the investigator through a serial response box, also attached to the computer (MEL; Schneider, 1988).

4.1.3. Procedure

On each trial, a central fixation plus appeared for 500 ms, followed by three white boxes for 1000 ms (see Fig. 2). Subsequently, one of the lateral boxes thickened (the peripheral cue) for 300 ms. After an interval of 200 ms with three white boxes, the central box thickened (the central cue) for 300 ms, followed by a further ISI of 200 ms before the target was presented. The target stimulus was a row of Xs (neutral) or a coloured word (RED, GREEN or BLUE). The colour word was always incongruent with the hue. This appeared until a response was made. Participants were asked to name the stimulus colour and to ignore the word.

Participants ran one practice block of 24 trials followed by three experimental blocks of 96 trials. In the experimental block, the target appeared in the left hemifield on half of the trials (48), and in the right hemifield on the other half. Also, for each hemifield, the target was presented at a valid (cued) location on half the trials (24) and at an invalid (uncued) location on the remaining trials. Finally, for each set of 24 trials, there were 12 trials for each Stroop condition, neutral and incongruent.

Table 4
The mean of the mean correct RTs, and percentage of errors as a function of field, location and congruence for the group of control participants in Experiment 2

Location	Left field		Right field	
	Incongruent	Neutral	Incongruent	Neutral
Cued	799 (3.7)	739 (0.2)	799 (0.4)	725 (0.4)
Uncued	790 (0.2)	671 (0.4)	781 (0.2)	676 (0.2)

4.2. Results and discussion

The means of the mean correct RTs, and the percentage errors (in parentheses), for the control group are shown in Table 4. Correct RTs were submitted to a repeated measures ANOVA with field (left and right), location (cued and uncued) and congruence (incongruent and neutral) as within subject factors. There were significant main effects of location and congruence, $F(1, 4) = 7.97$, $P < 0.05$, and $F(1, 4) = 32.63$, $P < 0.05$, respectively. That is, we found IOR (RTs slower to targets at cued relative to uncued locations by 35 ms) and Stroop effects (incongruent trials slower than neutral trials, by 90 ms). Furthermore, there was a significant location × congruence interaction, $F(1, 4) = 16.18$, $P < 0.05$. The analysis of the simple effects of the interaction showed a significant main effect of congruence at both the cued location, $F(1, 4) = 18.00$, $P < 0.05$, and the uncued location, $F(1, 4) = 37.62$, $P < 0.01$. However, the interaction was due to a reduction of Stroop interference at the cued location (a 67 ms effect), when compared to the uncued location (a 112 ms effect). There was no effect of the visual field, Fs < 1. The analysis of errors did not show any significant effect, Ps > 0.05.

The mean of the mean correct RTs and percentage of errors (in parentheses) for the group of patients are shown in Table 5. Correct RTs were submitted to a repeated measures ANOVA with field (ipsilesional and contralesional), location (cued and uncued) and congruence (incongruent and neutral) as within subject factors. There was a marginally significant effect of congruence, $F(1, 3) = 6.14$, $P = 0.089$. RTs were slower for incongruent stimuli (1017 ms) than for neutral stimuli (870 ms). Neither the main effects of field or location, nor the first order interactions reached statistical significance, Ps > 0.05. However, there was a significant three-way interaction between field, location and congruence, $F(1, 3) = 10.22$, $P < 0.05$. To analyse the interaction, we ran separate ANOVAs for each visual field with location (uncued and cued) and congruence (incongruent and neutral) as within subject factors. In the ipsilesional field, neither the main effects of location and congruence, nor their interaction, were significant, Ps > 0.05. In the contralesional field, there was a marginally significant main effect of congruence (1004 ms versus 910 ms), $F(1, 3) = 6.88$, $P = 0.079$, but most important there was a significant location × congruence interaction, $F(1, 3) = 8.77$, $P < 0.05$. The analysis of the simple effects showed a significant Stroop effect at the uncued location (182 ms), $F(1, 3) = 14.03$, $P < 0.05$, whereas there were no reliable differences between the incongruent (1046 ms) and neutral (960 ms) conditions at the cued location, $P > 0.05$. In addition, to confirm the interaction between field, and location found in Experiment 1, we conducted an ANOVA with field (ipsilesional and contralesional) and location (cued and uncued) as within subject factors only for neutral trials. None of the main effects of field and location were significant, Ps > 0.05. However, there was a marginally significant interaction between those effects, $F(1, 3) = 6.45$, $P = 0.08$. The interaction was due to an effect of IOR for contralesional targets (99 ms) that, as before, did not emerge in the ipsilesional field (−24 ms).

The analysis of errors did not show any significant effects, Ps > 0.05.

When comparing the magnitude of the IOR effect in Experiments 1 and 2, for the group of patients, it appears that IOR tends to be greater in magnitude in the discrimination task (99 ms of effect, Experiment 2) as compared to the detection task (48 ms, Experiment 1). This difference could be an artefact resulting from a difference in their baseline reactions times (RTs were higher in the discrimination task, Experiment 2, than in the detection task, Experiment 1). To account for the large difference in reaction times for the detection (Experiment 1) and discrimination task (Experiment 2) we conducted new analyses on transformed individual mean RTs. Therefore, for each field condition in each task we calculated IOR scores using the following formula: (cued RT − uncued RT)/uncued RT × 100. These scores represent the percentage change in RT in the cued location relative to the uncued location (Chasteen & Pratt, 1999; Faust & Balota, 1997; Langley, Fuentes, Hochhalter, Brandt, & Overmier, 2001).

Table 5
The mean of the mean correct RTs, and percentage of errors as a function of field, location and congruence for the group of patients in Experiment 2

Location	Ipsilesional field		Contralesional field	
	Incongruent	Neutral	Incongruent	Neutral
Cued	983 (0.75)	818 (0.13)	1046 (0.63)	960 (0)
Uncued	997 (0.38)	842 (0.13)	1043 (0.13)	861 (0)

5. Transformed IOR scores

IOR effects as represented by percentage change scores are presented in Fig. 3. Individuals' transformed scores were submitted to a 2 × 2 mixed ANOVA with task (detection and colour discrimination) as the between subject factor and field (ipsilesional and contralesional) as the within subject factor. The main effect of field approached significance, $F(1, 6) = 4.88$, $P = 0.069$. IOR effects were greater on the contralesional field ($M = 11.90\%$) than in the

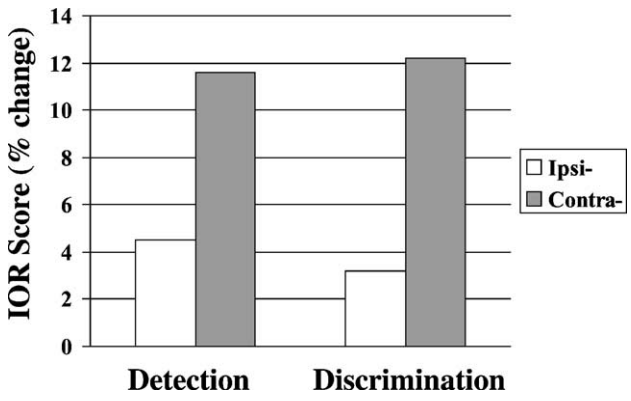

Fig. 3. IOR scores as a function of task (detection and discrimination) and field (ipsilesional and contralesional).

ipsilesional field ($M = 3.89\%$). No other effects were significant, Fs < 1.

The results from the control participants replicated the reduction of the Stroop interference at the cued location reported by Vivas and Fuentes (2001). The fact that we have replicated this effect in several laboratories with different procedures, with manual (Fuentes et al., 2000) and with vocal responses (Experiment 2) indicates its robustness. According to our hypothesis of inhibitory tagging, the reduction in Stroop interference would be an indirect consequence of colour naming responses being facilitated because the more rapidly processed colour name is temporarily disconnected from its response (when compared with the more slowly derived name for the hue; Vivas & Fuentes, 2001).

In the group of parietal patients there was again evidence for a reduced IOR for ipsilesional relative to contralesional stimuli, in the neutral condition (see Fig. 3), replicating Experiment 1 (but in this case with a vocal-key response). Thus, IOR can be observed in the contralesional field for both detection and discrimination tasks, in patients with damage to the parietal lobe. Furthermore, analysis with IOR transformed scores revealed no differences in the magnitude of IOR between the two tasks (see Fig. 3). This finding confirms previous studies that have found IOR effects of similar magnitude in detection and discrimination tasks, in healthy adults, when the cue-target SOA is long enough (Lupiañez et al., 1997).

There was, in addition, an interaction between visual field, IOR and the Stroop effect. There was a reduction in Stroop interference at the cued location (as with the controls) but only for contralesional targets. This finding confirms our hypothesis that the interaction between Stroop interference and stimulus location would be modulated by the visual field. In the absence of IOR, there was no evidence for inhibitory tagging (for the ipsilesional targets). The mechanism for inhibitory tagging itself seems intact, however, since a reliable reduction in Stroop interference occurred to previously cued targets in the contralesional field.

6. General discussion

The results from the present study showed a normal IOR for control participants in a double cue procedure similar to the one employed by Posner and Cohen (1984). Also, the control results in Experiment 2 replicated those reported by Vivas and Fuentes (2001), with in this case both IOR effects and the IOR × Stroop interaction occurring when vocal RTs were recorded.

The data from the parietal patients differed from the controls in several respects. First, the patients did show an IOR effect in both detection and discrimination tasks, but only when targets appeared in the contralesional field (Experiments 1, and 2, neutral condition). Second, in Experiment 2, there was an interaction between the IOR and Stroop effects, found in the group of control participants, but this interaction was now modulated by the visual field. That is, there was a reduction of the Stroop interference at the previously cued location, but only when the target was presented in the contralesional field. There was no evidence for either IOR or modulation of Stroop interference with ipsilesional targets.

In general, these results suggest that IOR plays a role in inhibitory tagging. When IOR is reduced in one field, then any modulation of Stroop interference was altered. However, given the previous findings with schizophrenic (Fuentes et al., 2000) and a frontal lobe patient with an organic lesion (Vivas et al., submitted for publication-a, submitted for publication-b), a preserved IOR mechanism is not sufficient for inhibitory tagging to take place. We discuss both IOR and inhibitory tagging below.

6.1. Neural basis of IOR

IOR is observed when attention is oriented towards a peripheral location and then subsequently removed from that location (Posner & Cohen, 1984). Accordingly, IOR can be considered as an attentional bias towards novel locations. Since the original study by Posner and Cohen (1984), extensive research has been conducted into the causes of IOR. On one account, IOR is related to a bias in oculomotor programming. Evidence for this comes from studies with normal participants (Posner & Cohen, 1984; Rafal et al., 1989), with patients suffering from PSP (Posner et al., 1985; Sapir et al., 1999), and with children (Clohessy, Posner, Rothbart, & Vecera, 1991; Valenza, Simion, & Umilta, 1994). For example, Posner and Cohen (1984) did not observe IOR with central cues, which need not produce an oculomotor bias. Nevertheless, central cues can generate IOR as long as a saccade is prepared or executed (Rafal et al., 1989). Furthermore, IOR is more robustly produced by signals in the temporal hemifield of normal participants, diagnostic of the involvement of the superior colliculus (Rafal et al., 1989). In line with these findings, patients with PSP, a degenerative disease that affects midbrain areas (including the superior colliculus) and that reduces the ability to perform saccades, show a reduced IOR effect (Faust & Balota, 1997; Posner

et al., 1985). Finally, it has been shown that IOR occurs in infancy prior to complete cortical development (Clohessy et al., 1991; Valenza et al., 1994).

Although there is strong evidence that support the role of midbrain areas (and in particular the superior colliculus) in generating IOR, there also converging data from neuropsychological and functional imaging studies indicating that IOR effects are also linked to activity in the posterior parietal cortex (Bartolomeo et al., 1999, 2001; Lepsien & Pollman, 2002; Rosen et al., 1999). Our data are clearly compatible with this. Dazinger, Fendrich, and Rafal (1997) have suggested that 'the inhibitory tag generated in the midbrain may need to be transmitted to the parietal cortex through the pulvinar to be encoded in spatiotopic co-ordinates' (p. 306). The implication of this assertion is that an intact superior colliculus may not be a sufficient condition to observe IOR in detection and discrimination tasks, and that biases in spatial attention need to be implemented by posterior parietal cortex. For example, the posterior parietal cortex may contain a spatial map that signals the relative salience of locations for attention. Locations subject to IOR may be less salient in this map. In addition, unilateral parietal damage might heighten the salience of ipsilesional signals within the map, since such may receive less attentional competition from contralesional signals in the map. This imbalance in saliency may be sufficient to over-ride an IOR applied to ipsilateral locations, so that IOR is at least reduced in such patients.

At present it is not clear why some studies have reported null effects on IOR in parietal patients (Posner et al., 1985), though in other case effects have been observed (Bartolomeo et al., 1999; here). The varying results may reflect the degree of damage or the sensitivity of the task to factors other than oculomotor bias. Nevertheless, the present positive evidence indicates that damage to parietal cortex modulates IOR and it can at least reduce IOR in patients with unilateral parietal damage.

6.2. Inhibitory tagging in patients with parietal damage

Two different inhibitory effects seem to take place in the IOR procedure when discrimination tasks are employed. One, IOR prevents attention from returning to an already explored location (Posner & Cohen, 1984). The second effect seems to modulate the processing of the stimuli that appear in the inhibited (cued location). We have called this latter effect 'inhibitory tagging' (Fuentes et al., 1999b). More specifically, we propose that inhibitory tagging acts to disconnect the activated representation of the stimulus at a cued location from its associated response. We believe that this mechanism can account for the reduction of Stroop interference at the cued location observed in control participants in the present experiment (see Vivas & Fuentes, 2001, for a detailed explanation). Now, since the parietal patients only showed IOR, and modulation of Stroop interference, to contralesional targets, we conclude that IOR is needed to produce the inhibitory tagging effect. As we have noted, though, IOR is not sufficient. The data from the present study, when combined with those obtained from schizophrenic and a frontal lobe patient with an organic lesion (Fuentes et al., 2000; Vivas et al., submitted for publication-a, submitted for publication-b), indicate that the process of providing an inhibitory link between a stimulus and its associated response takes place with more anterior cortical structures. We suggest that IOR, mediated by the posterior parietal cortex, provides a signal to anterior frontal areas that sets a temporary inhibitory link between representations activated by stimuli at inhibited locations and their response. In this way, the parietal cortex may serve as a useful way station in the neural network controlling spatial attention. In particular, the parietal cortex may be involved in translating an oculomotor bias in subcortical structures, into a signal that modulates processing in more anterior cortical structures concerned with response selection. The parietal cortex, then, would play a role in generating higher level properties of IOR.

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