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Conscious perception in patients with prefrontal damage

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ABSTRACT

We are conscious and verbally report some of the information reaching our senses, although a big amount of information is processed unconsciously. There is no agreement about the neural correlates of consciousness, with low-level theories proposing that neural processing on primary sensory brain regions is the most important neural correlate of consciousness, while high-level theories propose that activity within the fronto-parietal network is the key component of conscious processing (Block, 2009). Contrary to the proposal of high-level theories, patients with prefrontal lobe damage do not present clinical symptoms associated to consciousness deficits. In the present study, we explored the conscious perception of near-threshold stimuli in a group of patients with right prefrontal damage and a group of matched healthy controls. Results demonstrated that perceptual contrast to perceive the near-threshold targets was related to damage to the right dorsolateral prefrontal cortex, and with reduced integrity of the ventral branch of the right superior longitudinal fascicule (SLF III). These results suggest a causal role of the prefrontal lobe in conscious processing.

1. Introduction

Consciousness refers to our capacity to intentionally report a specific piece of information that has been processed. Not all the information that reaches our senses can access consciousness, as it has largely been demonstrated that unconscious information can affect several stages of processing (see e.g. Gaillard et al., 2009; Kentridge et al., 2004; King et al., 2016; van Gaal and Lamme, 2012; van Gaal et al., 2014) and modulate our behavior (see e.g. Jiménez et al., 2009; Lau and Passingham, 2007; Reuss et al., 2015). Nevertheless, how does information access consciousness, or which are the pre-requisites for information to be consciously processed, are still open questions in Cognitive Neuroscience (Aru et al., 2012).

Some models propose that attention is one of the pre-requisites for consciousness (Dehaene and Changeux, 2011; Dehaene et al., 2006; Mack and Rock, 1998; Marois and Ivanoff, 2005; Posner, 1994, 2012). Accordingly, without attention, important information can be unnoticed, as demonstrated in paradigms such as the attentional blink or change blindness (Raymond et al., 1992; Rensink, O'Regan and Clark, 1997). Attentional processes can boost perceptual information by increasing perceptual gain or biasing decision-making mechanisms (Carrasco, 2011). Contrary, other models claim that consciousness can be observed in the near absence of (some forms) of attention (Koch and Tsuchiya, 2007; Lamme, 2003), therefore proposing that attention and consciousness are dissociable (but see Chica et al., 2012; Chica et al., 2011).

Another open question in the field relates to the neural correlates of conscious processing. The so-called "lower-level" theories of consciousness propose that consciously and unconsciously processed information differ on the level of activation in primary sensory brain regions (Block, 2009). "High-level" theories of consciousness, on the other hand, propose that consciously and unconsciously processed information can similarly activate primary sensory brain regions, but consciously processed information activates a distributed set of fronto-parietal regions that enable the reverberation of information within the network (another key mechanism of consciousness, Lamme, 2006) (Lau and Rosenthal, 2011). The Global Neural Workspace model of consciousness (Dehaene and Changeux, 2011) highlights the role of long distance cortical networks, especially allowing the communication between fronto-parietal regions, which are important for information to ignite the system. In agreement with high-level theories, damage to the right parietal cortex, and its connections to the ipsilateral frontal lobe

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(Bartolomeo et al., 2007), can produce spatial hemineglect, a syndrome associated to severe impairments in both spatial attention and consciousness (Bartolomeo et al., 2012; Corbetta and Shulman, 2011). However, in apparent contradiction to high-level theories, patients suffering from frontal damage do not seem to present deficits in conscious perception. One study has demonstrated that prefrontal patients present a significantly elevated masking threshold for detecting visual stimuli (Del Cul, Dehaene, Reyes, Bravo and Slachevsky, 2009), in tight correlation with the degree of expansion of the lesions into the left anterior prefrontal cortex. However, because attentional processes were not controlled for in Del Cul et al. (2009) study, factors such as reduced attention or enhanced distractibility in frontal patients might explain their results.

The importance of long-distance cortical projections has also been tested in patients with multiple sclerosis (MS) (Reuter et al., 2009). MS patients needed a longer target-mask delay for consciously processing the stimulation. This effect correlated with structural damage in the dorsolateral prefrontal white matter and the right occipito-frontal fasciculus. The integrity of the Superior Longitudinal Fascicule (SLF), connecting the parietal and frontal cortex, has also been related to the attention and consciousness deficits observed in hemispatial neglect (Bourgeois et al., 2015; Bourgeois et al., 2012; Shinoura et al., 2009). The SLF is divided in three branches: dorsal, middle, and ventral (Thiebaut de Schotten et al., 2011). The latter is right lateralized and has been proposed (Thiebaut de Schotten et al., 2011) to overlap with the ventral attentional network described in models based on functional Magnetic Resonance Imaging data (Corbetta et al., 2008). This ventral network might be an interesting brain hub for conscious perception as demonstrated in studies with healthy participants (Todd et al., 2005), and patients suffering from hemispatial neglect (Golay et al., 2008; Riddoch et al., 2010).

The aim of the present work was to study conscious perception in a group of prefrontal patients, and to explore the correlation of patients' performance with the integrity of the right SLF III. We used a simple task in which participants were presented with a Gabor stimulus, which was titrated to be perceived at threshold. We explored if target contrast to perceive the Gabors was associated to the integrity of the right SLF III in our group of patients. Attentional orienting before the Gabor was manipulated with a peripheral cue, which made the Gabors spatially attended or non-attended. A neuropsychological battery was also administered to all participants, measuring attention, perception, memory, and executive functions.

2. Method

2.1. Participants

A total of 11 patients with right prefrontal brain damage were screened for inclusion in the present study. The inclusion criteria were: (1) damage to the right prefrontal cortex (either due to vascular lesions, traumatic brain injury, or tumor resection surgery); (2) no visual deficits or hemispatial neglect; (3) right-handedness; and (4) ability to maintain gaze and follow the instructions. Eight patients fulfilled the criteria and participated in the study (see Fig. 1 and Table 1). Nine healthy controls were also recruited, matched to the patients in age and years of education. The prefrontal patients did not show deficits in spatial orienting, sustained attention, alternating attention, verbal memory, inhibition, or planning. Their performance in verbal executive functions, which usually relay in left prefrontal areas (such as verbal working memory or verbal reasoning) was comparable to that of healthy controls. However, they showed deficits related to right prefrontal damage in visual working memory, as well as a specific impairment in selective attention, discrimination of objects from different perspectives (Minimal feature match test of the BORB), semantic fluency, and visual memory. Table 2 shows the demographical and clinical data for the included patients, including the statistical comparison between the groups.

The ethics committee from the local Government of Andalucía (CEI-Granada) approved the experiments. All participants gave their signed informed consent and were informed that they could discontinue participation in the study without penalty. They received a monetary compensation for their time and effort (10€/hour).

2.2. Apparatus and stimulih

A HP 550 computer (screen 13.1″ x 8.2”) running E-prime software (Schneider et al., 2002) controlled the presentation of stimuli, timing operations, and data collection. Stimuli were presented against a grey background (luminance = 52 cd/m^2) (see Fig. 2). Three black markers (4° high x 6° wide) were displayed, one in the center of the display, the other two placed 9° to the left or the right of the fixation point (as measured from the fixation point to the center of the lateral markers). The fixation point consisted of a black plus sign (0.4° × 0.4°), situated in the center of the central box. The spatial cue was a black circle (1° high x 1.5° wide) presented in the upper external corner of one of the markers. The target consisted of a Gabor stimulus (4 cycles per degree of visual angle, with a diameter of 3°, and SD of 0.3) with a maximum and minimum Michelson contrast of 0.92 and 0.02, respectively. The lines composing the Gabor could be tilted 10° to either the left or to the right.

2.3. Procedure

Fig. 2 displays the sequence and timing of a trial. Trials started with a fixation point presented for 304 ms. Then, the cue appeared for 304 ms, 50% of the trials in the left hemifield and the remaining 50% of the trials in the right hemifield. The inter-stimulus cue-target interval (ISI) lasted for 64 ms. After this interval, the Gabor was presented on 66.6% of the trials, while on the remaining 33.3% of the trials no Gabor was presented (catch trials). If the target was present, it appeared for 32 ms in one of the markers, with equal probability (50%). The cue indicated the Gabor location on 50% of the Gabor present trials. Participants were instructed to ignore the cue and verbally indicate if the Gabor was presented inside the left or right marker, or if no Gabor was perceived. They were instructed to respond accurately, with no time pressure.[1] The experimenter recorded the participant's responses using three keys of the keyboard.

Gabor contrast was manipulated before the experimental trials in order to adjust the percentage of consciously perceived stimuli. During titration, the timing was identical to the experimental trials, but no spatial cue was presented. All participants started with a supra-threshold stimulus (Michelson contrast = 0.184), presented at either the left or the right hemifield. Participants performed two separate titration blocks, one in which the Gabor appeared in the left marker and another one in which the Gabor appeared in the right marker. Perceptual sensitivity (A') to detect the Gabor (Abdy, 2007) was calculated (see below) after every 12 trials (8 Gabor present trials and 4 catch trials). If it was higher than 0.89, Gabors at the immediately following lower contrast level (present contrast minus 0.009) were used during the next 12 trials. Conversely, if perceptual sensitivity was lower than 0.78, Gabors at the immediately following higher contrast level (present contrast plus 0.009) were used during the next 12 trials. This

[1] Although reaction times (RTs) were not recorded in the experimental task, we did record RTs in some neuropsychological tests, such as the counting part of the 5 Digit Test. These RTs did not differ between prefrontal patients and healthy controls (Mann-Whitney *U* test, W = 49.50, p = 0.209, $BF_{incl.} = 0.895$), nor between the orbito-medial and dorsolateral patients (Mann-Whitney *U* test, W = 3.50, p = 0.476, BF $BF_{incl.}$ = 0.745). Therefore, we have no data to suggest inter-group variability regarding speed processing in our sample of participants.

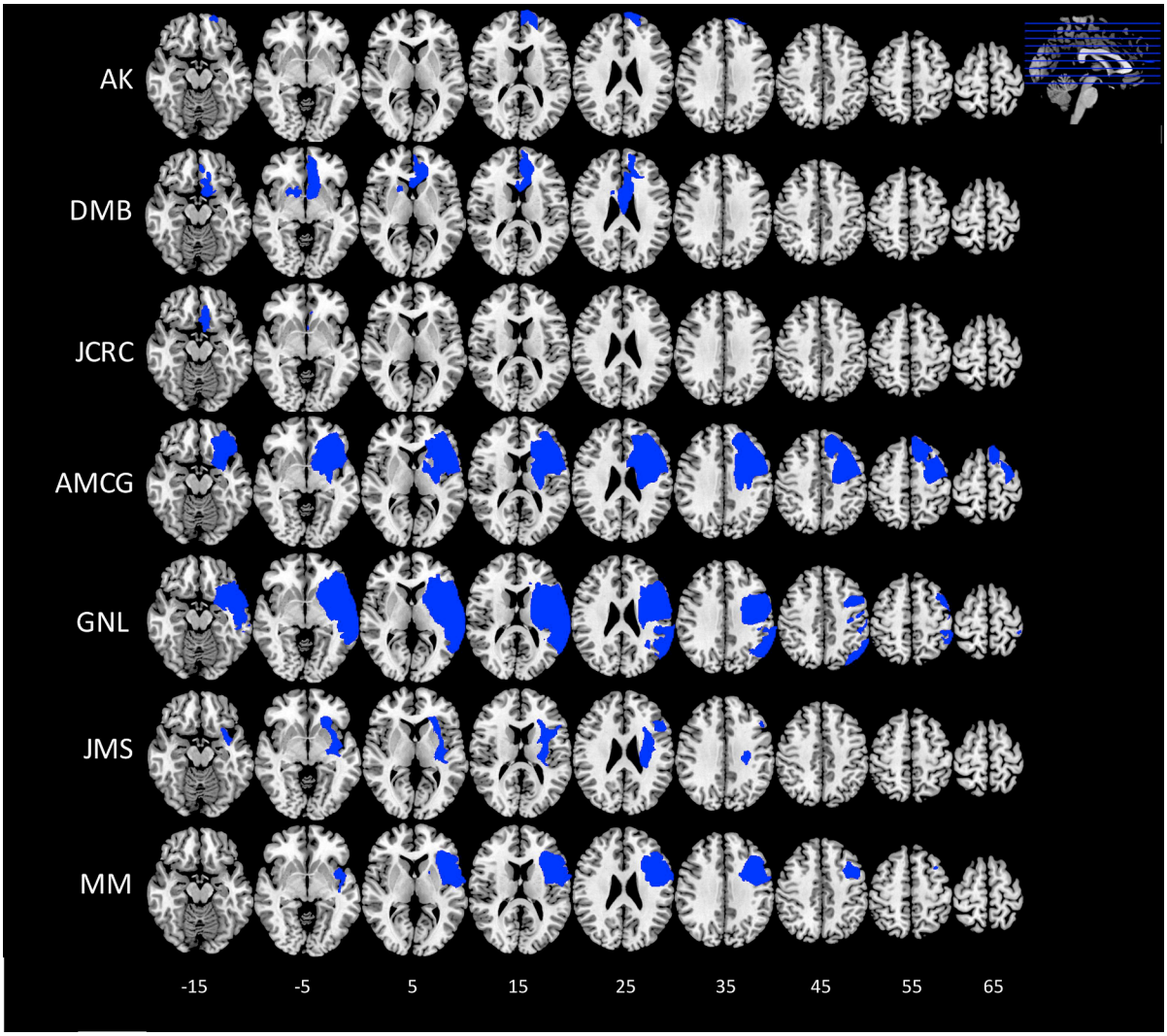

Fig. 1. Reconstruction of the brain lesions for each patient (one patient did not fulfill the criteria for magnetic resonance imaging). Results are superimposed on slices of the Montreal Neurological Institute standard brain (z MNI coordinates are shown on the bottom of the figure).

titration procedure continued until participants felt comfortable with the task, and perceptual sensitivity was > 0.78 and < 0.89 for two consecutive blocks of trials. Once Gabor contrast was calibrated for one hemifield, the same procedure was followed for the other hemifield (the order of calibration for the left and right hemifields was counterbalanced between participants).

The experimental blocks consisted of a total of 96 trials, of which 32 were catch trials. In the remaining 64 trials, the Gabor was present, either at the location of the cue (attentionally cued trial, 50%) or at the opposite location (attentionally uncued trial, 50%). A block of 24 practice trials preceded the experimental trials.

2.4. Structural imaging acquisition and lesion delimitation

Brain MRI 3D T1 scans were acquired on a Siemens 3 T TRIO TIM system with a 32- channel head coil for signal reception (echo time of 4.18 ms, repetition time of 2300 ms; acquisition matrix = 256 × 256; voxel resolution = 1 × 1 × 1 mm; slice thickness = 1 mm; 50% inter-slice gap; flip angle = 9°; 176 vol acquired). T2 images were also acquired for lesion delimitation purposes. Lesions were assessed by an expert radiologist (HB) and by a neuropsychologist (IC), trained to read brain scans. First, lesion extent was determined for each patient by manually drawing the lesion borders directly onto the original 3D T1 MRI, by using the MRIcro software (Rorden and Brett, 2000, www.mricro.com). Then, the 3D brain scans and lesion volumes were normalized to the standard Montreal Neurological Institute (MNI) brain template in Statistical Parametric Mapping-8 (http://www.fil.ion.ucl.ac.uk/spm) running under Matlab 8.1 (http://www.mathworks.com). In order to reduce lesion-induced registration errors, spatial normalization was performed excluding the voxels contained in the lesion mask, thereby preventing the damaged areas from biasing the transformation (Brett et al., 2001; Volle et al., 2008). After normalization, the brain lesion was manually segmented, and its borders were redefined in the normalized brain.

2.5. Diffusion-weighted imaging acquisition and preprocessing

We used a fully optimized acquisition sequence for the tractography of diffusion-weighted imaging (DWI), which provided isotropic (2 × 2 × 2 mm) resolution and coverage of the whole head with a posterior-anterior phase of acquisition. A total of 70 near-axial slices were acquired on a Siemens 3 T TRIO TIM system equipped with a 32-channel head coil. We used an echo time (TE) of 88msec and a repetition time (TR) of 8400 msec At each slice location, 6 images were acquired with no diffusion gradient applied. Additionally, 60 diffusion-weighted images were acquired, in which gradient directions were uniformly distributed on the hemisphere with electrostatic repulsion. The diffusion weighting was equal to a b-value of 1500 s/mm^2. Finally,

Table 1
Description of the lesion and lesion etiology for each patient.

Patient	Lesion etiology	Lesioned areas	Broadmann areas
AK (orbitofrontal-medial)	Surgical tumor resection (meningioma)	Superior Frontal (RH)	9
		Superior Frontal Medial (RH)	9/10
		Superior Frontal Orbital (RH)	11
		Middle Frontal Orbital (RH)	46
DMB (orbitofrontal-medial)	Ischemic stroke	Superior Frontal (RH)	9
		Superior Frontal Medial (RH)	9/10
		Medial Frontal Orbital (RH)	11
		Anterior Cingulate Cortex (B)	24/25/32
		Caudate (B)	
		Pallidum (B)	
		Putamen (B)	
JCRC (orbitofrontal-medial)	Aneurism of the Anterior Communicate Artery	Medial Frontal Orbital (RH)	11
		Anterior Cingulate Cortex (RH)	25
		Rectus (RH)	11
		Olfatory (RH)	25
		Caudate (RH)	
AMCG (dorsolateral)	Ischemic stroke	Superior Frontal (RH)	4/6/8
		Superior Frontal Medial (RH)	9/32
		Middle Frontal (RH)	9/46
		Middle Frontal Orbital (RH)	46
		Inferior Frontal Orbital (RH)	25/47
		Inferior Frontal (Pars Triangularis) (RH)	45
		Inferior Frontal (Pars Opercularis) (RH)	44
		Rolandic Operculum (RH)	43
		Insula (RH)	
		Amygdala (RH)	
		Caudate (RH)	
		Pallidum (RH)	
		Putamen (RH)	
GNL (dorsolateral)	Ischemic stroke	Middle Frontal (RH)	4/6
		Inferior Frontal Orbital (RH)	11/47
		Inferior Frontal (Pars Triangularis) (RH)	45
		Inferior Frontal (Pars Opercularis) (RH)	44
		Rolandic Operculum (RH)	43
		Precentral gyrus (RH)	4
		Postcentral gyrus (RH)	2/3
		Parietal Inferior (RH)	39/40
		Angular gyrus (RH)	39
		Supramarginal gyrus (RH)	40
		Superior Temporal (RH)	22
		Superior Temporal Pole (RH)	38
		Middle Temporal (RH)	21
		Insula (RH)	
		Amygdala (RH)	
		Pallidum (RH)	
		Putamen (RH)	
JMS (dorsolateral)	Hemorrhagic stroke	Middle Frontal (RH)	6
		Inferior Frontal Orbital (RH)	11/47
		Inferior Frontal (Pars Triangularis) (RH)	45
		Inferior Frontal (Pars Opercularis) (RH)	44
		Rolandic Operculum (RH)	44
		Superior Temporal (RH)	38
		Insula (RH)	
		Putamen (RH)	
MM (dorsolateral)	Ischemic stroke	Middle Frontal (RH)	6/46
		Inferior Frontal (Pars Triangularis) (RH)	45
		Inferior Frontal (Pars Opercularis) (RH)	44
		Rolandic Operculum (RH)	43
		Precentral gyrus (RH)	4
		Postcentral gyrus (RH)	43
		Superior Temporal (RH)	22
		Superior Temporal Pole (RH)	38
		Insula (RH)	
		Amygdala (RH)	
AFM (No RM)[a]	Traumatic Brain Injury	Fronto-basal regions (B)	
		Frontal convexity (RH)	

RH: Right Hemisphere. LH: Left Hemisphere. B: Bilateral.

[a] Anatomical information from this patient has been extracted from a computerized axial tomography scan that was taken at the moment of his hospital admission.

Table 2
Demographical and clinical characteristics of the participants of the study, for both brain-damaged patients and matched healthy controls, as well as the statistical comparison between the groups. Mean scores and standard deviations (in parenthesis) are presented. Asterisks represent statistically significant comparisons between the groups.

		Pre-frontal patients	Healthy controls	Comparison (Mann Whitney *U* Test)
Demographical information				
Gender		6 male 2 female	5 male 4 female	-
Age		53.38 (12.75)	57.11 (14.00)	W = 25.50 p = 0.335
Years of education		11.25 (4.83)	10.67 (3.84)	W = 41.00 p = 0.659
Months since lesion		7.75 (11.11)	-	-
Etiology		6 stroke 1 TBI 1 tumor resection	-	-
Neuropsychological assessment				
Cognitive functioning	Vocabulary WAIS-IV (scalar score)	12.38 (3.62)	13.78 (3.49)	W = 26.50 p = 0.383
Spatial orienting tests	Visual extinction (omissions)	0.25 (0.71)	0.22 (0.69)	W = 36.50 p = 1.000
	Line cancellation (omissions)	0.63 (1.68)	0.11 (0.33)	W = 37.00 p = 0.932
	Line Bisection (deviation in cm)	0.25 (0.70)	0.11 (0.22)	W = 33.50 p = 0.772
Visual perception	Item match BORB (hits)	31.63 (0.74)	31.75 (0.46)	W = 31.00 p = 0.954
	Minimal feature match BORB (hits)	24.00 (1.07)	24.87 (0.35)	**W = 15.00 p = 0.044***
Praxis	Copy of the Rey Figure (centile score)	25.31 (10.90)	32.83 (3.72)	W = 15.00 p = 0.089
Attention	Sustained attention. Auditory A Test (errors)	0.25 (0.46)	0.44 (0.72)	W = 32.00 p = 0.673
	Selective attention. Picture Completion of WAIS-IV (scalar score)	6.5 (3.66)	10.75 (1.91)	**W = 8.50 p = 0.013***
	Alternating attention. Flexibility of Five Digits Test (centile score)	39.63 (36.17)	51.22 (37.06)	W = 31.00 p = 0.664
	Inhibition. Five Digits Test (centile score)	48.5 (40.40)	40.66 (25.96)	W = 42.50 p = 0.561
Memory	Verbal working memory. Digits of WAIS-III (scalar score)	8 (4.14)	11.67 (1.58)	W = 16.50 p = 0.066
	Visual working memory. Corsi blocks, WMS-III (scalar score)	8.38 (2.97)	11.66 (2.34)	**W = 14.00 p = 0.035***
	Short-term, free recall. TAVEC (direct punctuation)	7.25 (5.34)	10.11 (3.95)	W = 24.00 p = 0.267
	Short-term, cued recall. TAVEC (direct punctuation)	8.38 (4.98)	12.00 (2.92)	W = 21.00 p = 0.161
	Long-term, free recall. TAVEC (direct punctuation)	7.5 (5.95)	11.33 (3.67)	W = 21.50 p = 0.174
	Long-term, cued recall. TAVEC (direct punctuation)	8.5 (5.63)	12.11 (3.62)	W = 21.00 p = 0.161
	Intrusions free recall. TAVEC (direct punctuation)	13.88 (23.99)	2.33 (2.83)	W = 46.00 p = 0.349
	Intrusions cued recall. TAVEC (direct punctuation)	2.33 (2.83)	1.89 (1.83)	W = 43.50 p = 0.492
	Visual memory. Recall of the Rey Figure (centile score)	8.94 (7.07)	17.78 (5.04)	**W = 6.50 p = 0.009***
Executive functions	Verbal reasoning. Similarities of WAIS-IV (scalar score)	11 (5.15)	13.11 (3.79)	W = 29.00 p = 0.529
	Semantic fluency. Animals (centile score)	16.5 (14.86)	43.33 (23.32)	**W = 11.50 p = 0.020***
	Phonetic fluency. "P" (centile score)	27.88 (37.72)	23.11 (33.81)	W = 33.00 p = 0.809
	Planning. Key search of BADS (profile from -2 to 4)	1.75 (1.58)	2.33 (1.41)	W = 27.50 p = 0.430

TBI: traumatic brain injury; WAIS: Wechsler Adult Intelligence Scale; BORB: Birmingham Object Recognition Battery; WMS: Wechsler Memory Scale; BADS: Behavioral Assessment of Disexecutive Syndrome. Asterisks indicate statistically significant effects.

at each slice, diffusion-weighted data were simultaneously registered and corrected for subject motion and geometrical distortion adjusting the gradient accordingly (ExploreDTI, http://www.exploredti.com) (Leemans and Jones, 2009).

Damped Richardson Lucy Spherical Deconvolution (Dell'Acqua et al., 2010) was computed to estimate multiple orientations in voxels containing different populations of crossing fibers (Alexander, 2005; Anderson, 2005; Tournier et al., 2004). Algorithm parameters were chosen as previously described (Dell'Acqua, Simmons, Williams and Catani, 2013). A fixed-fiber response corresponding to a shape factor of $\alpha = 2 \times 10–3\ mm^2/s$ was chosen (Dell'Acqua et al., 2013). Whole brain tractography was performed selecting every brain voxel with at least one fiber orientation as a seed voxel. From these voxels, and for each fiber orientation, streamlines were propagated using Euler integration with a step size of 1 mm (Dell'Acqua et al., 2013). When entering a region with crossing white matter bundles, the algorithm followed the orientation vector of least curvature (as described in Schmahmann and Pandya, 2007). Streamlines were halted when a voxel without fiber orientation was reached or when the curvature between two steps exceeded a threshold of 45°. Spherical deconvolution, fiber orientation vector estimations, and tractography were performed using Startrack (http://www.natbrainlab.co.uk).

In order to facilitate the tractography dissection, regions of interest (ROI) for the right and left ventral branches of the SLF were defined on the CS-MNI template calculated above, based on the guidelines provided in previous reports (Catani and Thiebaut de Schotten, 2008; Rojkova et al., 2016). For each participant, the CS Map was registered to the CS-MNI152 template using ANTs.

Tract-specific measures of tract microstructural organization (i.e. mean Hindrance Modulated Orientational Anisotropy or HMOA for the whole tract) (Dell'Acqua et al., 2013) were extracted from each dissected tract. HMOA provides information about the microstructural diffusion properties of distinct fiber orientations and therefore specific to the orientation of the reconstructed tracts and more accurate than classical fractional anisotropy measures, which decreases when fibers cross due to local partial volume effect. White matter correlates were studied by using a track-wise statistical approach that takes into consideration how different voxels are associated along the same white matter pathway (Thiebaut de Schotten et al., 2014).

3. Results

We first analyzed the results of the titration task, i.e. Gabor contrast to perceive the Gabor in the left and right hemifield. Perceptual contrast to perceive the Gabors was similar when presented in the left or right hemifield for healthy participants (paired *t*-test, t(8) = 0.359, p = 0.729, Cohen's d = 0.120; $BF_{10} = 0.340$[2]), and for right prefrontal damage patients (paired *t*-test, t(7) = 1.670, p = 0.139; Cohen's

[2] In Bayesian statistics (Wagenmakers et al., 2018) a BF = 1 indicates no evidence favoring the alternative hypothesis (H_1) or the null hypothesis (H_0). BF > 1 indicate evidence in favor of H_1: 1–3 anecdotal evidence, 3–10 moderate evidence, 10–30 strong evidence. BF < 1 indicate evidence in favor of H_0: 0.33–1 anecdotal evidence, 0.10–0.33 moderate evidence, 0.03–0.10 strong evidence.

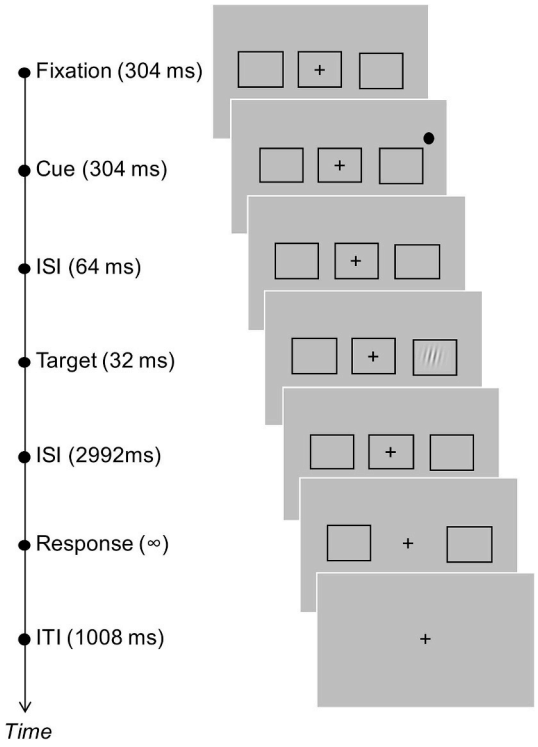

Fig. 2. Sequence and timing of a trial. Participants were instructed to ignore the cue and verbally report the location of the Gabor if detected or its absence if the Gabor was not perceived. The experimenter recorded the participants' responses. The figure shows an example of an attentionally cued trial, with the Gabor present.

close to 1 indicate a conservative criterion while values close to -1 indicate a non-conservative criterion (Stanislaw and Todorov, 1999). We analyzed mean A' for attentionally cued and uncued trials with a one-way ANOVA, in which group was introduced as a between-participants variable. This analysis demonstrated a main effect of validity, $F(1,15) = 7.25$, $MSE = 0.021$, $\eta^2 = 0.292$, $p = 0.017$; $BF_{Incl.} = 4.134$,[3] with increased perceptual sensitivity for attentionally cued as compared to uncued trials. The interaction between validity and group was not significant, $F = 2.60$, $MSE = 0.007$, $\eta^2 = 0.105$, $p = 0.128$; $BF_{Incl.} = 1.082$. When target location (left-right) was introduced in the ANOVA, no significant interaction with the validity effect was observed, $F < 1$; $BF_{Incl.} = 0.354$ (see Fig. 3).

The ANOVAs with response criterion as a dependent variable demonstrated no significant effects (all $Fs < 1$, and all $BF_{Incl.}$ between 0.247 and 0.482, for the ANOVAs with and without target side as independent variable).

When then used deterministic tractography to explore the contribution of the right SLF III to performance. As expected, patients presented an overall reduced HMOA for the right SLF III (mean HMOA = 0.069) as compared to healthy controls (mean HMOA = 0.095) (Mann Whitney *U* test, $W = 46.00$, $p = 0.029$; $BF_{10} = 7.002$), while no significant differences between patients and controls were observed for the left SLF III (Mann Whitney *U* test, $W = 38.00$, $p = 0.525$; $BF_{10} = 0.703$). We then explored if the mean HMOA of the right SLF III in the group of patients correlated with the behavioral effects observed. We found a significant correlation between the HMOA of the right SLF III and the perceptual contrast to perceive the Gabors during titration, $r = -0.93$, $p = 0.006$; $BF_{10} = 8.608$ (see Fig. 4A). This correlation was significant both for right and left sided targets, both $rs > -0.93$ ($BF_{10} = 7.384$ and $BF_{10} = 8.241$, respectively). Participants with lower contrast values to perceive the Gabors (i.e. with better perceptual abilities) presented increased HMOA of the

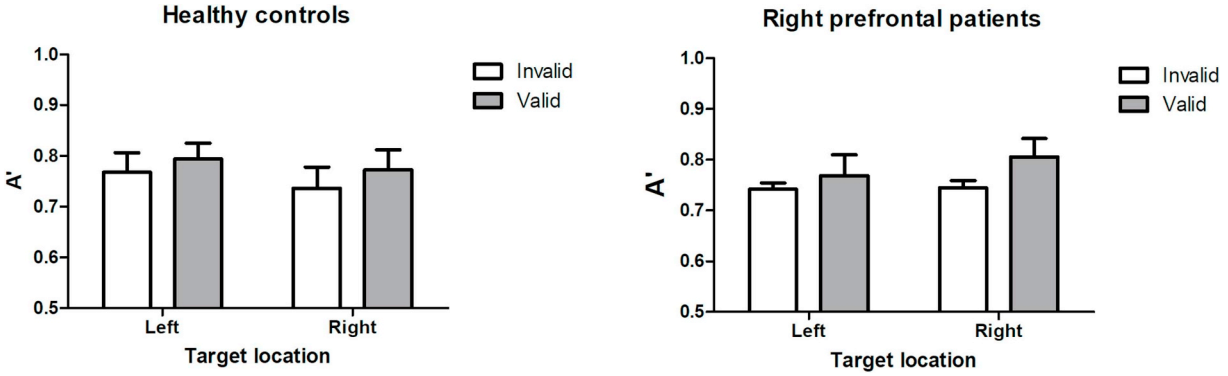

Fig. 3. Behavioral results. Perceptual sensitivity (A′) to detect the Gabor as a function of cue validity and target location for healthy controls and right prefrontal patients.

$d = 0.590$; $BF_{10} = 0.908$).

We then analyzed the results of the main task, with the aim of comparing Gabor perception at the attentionally cued and uncued location. We computed a non-parametrical index of perceptual sensitivity to detect the Gabor (A′), and response criterion (Beta"), based on the number of hits (correct detections when the Gabor was presented), and false alarms (FA; incorrect reports of the Gabor when it was not presented).

$$A' = 0.5 + \frac{(Hits - FAs)*(1 + Hits - FAs)}{4*Hits*(1 - FAs)};$$

$$Beta'' = \frac{Hits*(1 - Hits) - FAs*(1 - FAs)}{Hits*(1 - Hits) + FAs*(1 - FAs)}$$

A′ values usually range between 0.5 (the signal cannot be distinguished from the noise) to 1 (perfect performance). For Beta", values right SLF III as compared to participants with higher contrast values to perceive the Gabor. We also explored if the overall perceptual sensitivity (A′) and response criterion (Beta") correlated with the HMOA of the right SLF III. No significant correlations were observed for A' ($r = -0.21$, $p = 0.686$, $BF_{10} = 0.527$), while a marginal effect was observed for Beta" ($r = -0.77$, $p = 0.074$, $BF_{10} = 1.848$; see Fig. 4B). Decreased HMOA of the right SLF III was associated with a more conservative the criterion to report the Gabor. In healthy participants, none of the correlations reached statistical significance (all $rs < (ABS)0.49$), except for the correlation between the right SLF III and the overall response criterion ($r = -0.67$, $p = 0.047$, $BF_{10} = 2.261$) (see Fig. 4 B).

[3] Output effects for the main effects and interactions in the Bayesian ANOVAs are presented across matched models, following the recommendations of Wagenmakers et al. (2018) for Bayesian analyses in JASP.

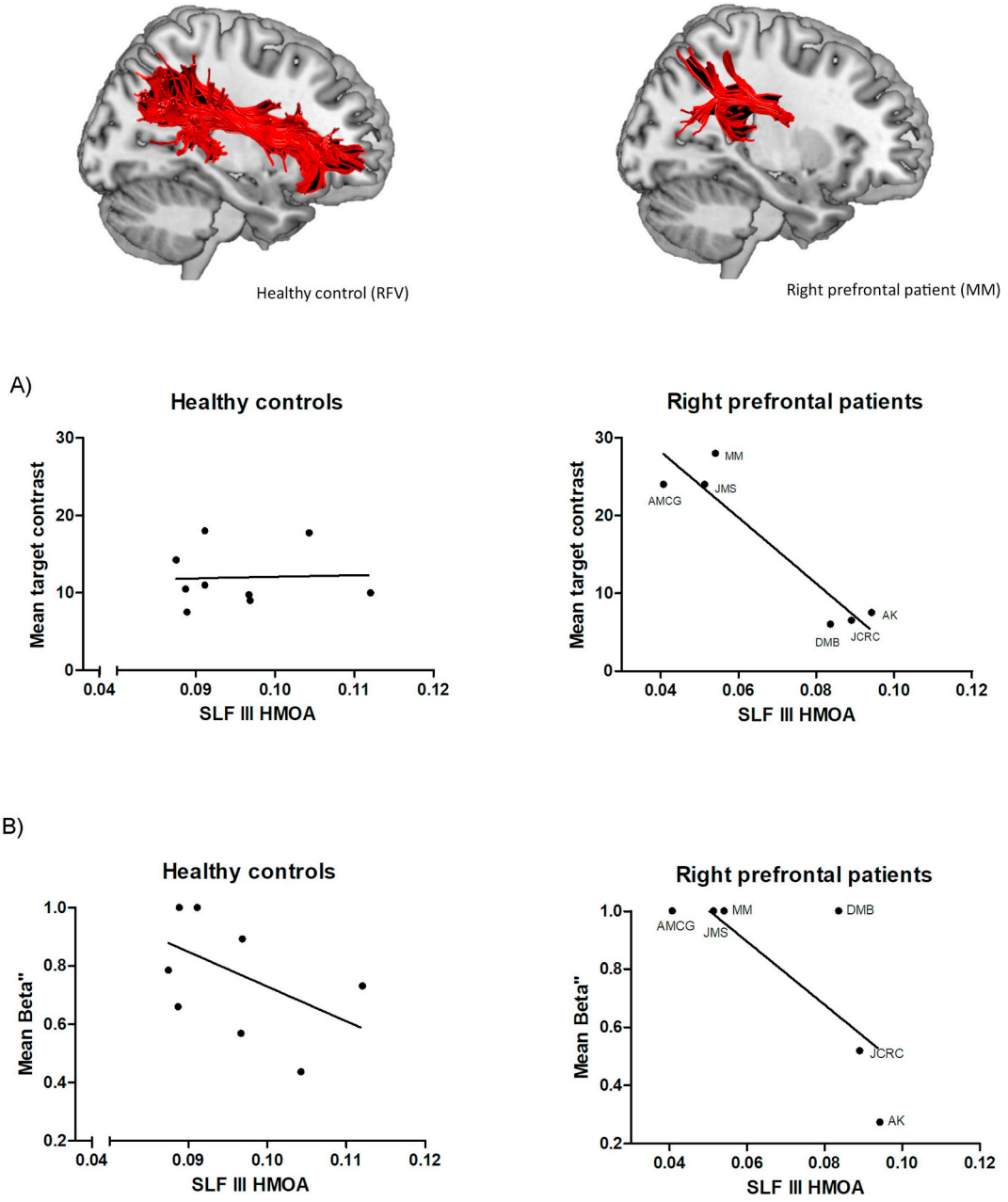

Fig. 4. Top: Representation of the right SLF III for a healthy control participant (left) and a right prefrontal patient presenting a disconnection of the fasciculus (right). Bottom: A) Correlation between the HMOA of the right SLF III and mean perceptual contrast to perceive the Gabor, for healthy controls (left) and right prefrontal patients (right). B) Correlation between the HMOA of the right SLF III and mean response criterion (Beta") to report the Gabor, for healthy controls (left) and right prefrontal patients (right). Two healthy participants presented exactly the same HMOA (0.091) and Beta" (1) values, and therefore their data appear overlapped in this figure. Patient GNL was not included in the correlation analyses because his lesion involved the right SLF III trajectory, and the tractography dissection revealed no right SLF III fibers (this patient had a mean target contrast of 13.5 and a mean Beta" of 1).

This result on Beta" for patients and controls should be interpreted with caution given that Bayesian statistics only demonstrated anecdotal evidence.

It is interesting to note that all patients with low HMOA values (which was associated to higher target contrast to perceive the Gabors and a more conservative response criterion) presented lesions in the dorsolateral prefrontal cortex, including the inferior frontal gyrus (patients AMCG, JMS, MM), while patients with higher HMOA values (and lower target contrast to perceive the Gabors) presented lesions in the orbitofrontal and medial frontal regions (patients AK, DMB, JCRC) (see Fig. 4 A). Therefore, our results indicate that damage to the prefrontal cortex or/and reduced integrity of the right SLF III is related to the conscious perception of near-threshold targets. Additional Mann-Whitney U tests were conducted to explore whether the validity effect on A′ and Beta" (mean scores for valid minus invalid trials) depended on lesion location (orbitofrontal-medial and dorsolateral), and whether these two groups of patients differed on their overall A′ and Beta" values. The results demonstrated a comparable validity effect for A′ in both sub-groups ($W = 9.00$, $p = 0.400$, $BF_{10} = 1.127$), as well as comparable overall A′ values ($W = 4.00$, $p = 0.629$, $BF_{10} = 0.550$) (see Table 3). Beta" analyses could not be conducted due to the lack of variance of the data in the dorsolateral patients group (all patients had a mean Beta" value of 1, indicating a conservative response criterion) (see Table 3).

Table 3
Participants' mean A′ and Beta" (with standard deviations in parenthesis) for valid and invalid trials in the spatial orienting task. Data from the orbitofrontal-medial and dorsolateral patients are presented separately.

	Validity	Right prefrontal patients				Healthy controls	
		Orbitofrontal-medial		Dorsolateral			
		Target Location		Target Location		Target Location	
		Left	Right	Left	Right	Left	Right
A′	Invalid	0.74 (0.05)	0.72 (0.05)	0.76 (0.01)	0.76 (0.02)	0.76 (0.15)	0.75 (0.16)
	Valid	0.83 (0.11)	0.84 (0.05)	0.78 (0.03)	0.84 (0.03)	0.82 (0.05)	0.82 (0.04)
Beta"	Invalid	0.56 (0.39)	0.52 (0.42)	1 (0.00)	1 (0.00)	0.78 (0.44)	0.77 (0.39)
	Valid	0.52 (0.42)	0.67 (0.57)	1 (0.00)	1 (0.00)	0.84 (0.26)	0.83 (0.28)

To summarize, our results demonstrate that right prefrontal damage patients did not present remarkable deficits in spatial orienting. They used the peripheral cues in a comparable manner to healthy controls. However, the contrast needed to perceive the Gabors at threshold was related to the damage of the dorsolateral prefrontal cortex and/or with the integrity of the right SLF III (the more reduced the integrity, the larger the contrast needed to perceive the Gabor).

4. Discussion

The aim of the present study was to test a clear prediction of high-level theories of consciousness, the causal implication of the frontal lobe in conscious perception. We also explored the contribution of white matter pathways connecting the parietal and frontal cortex to conscious perception, as these long-range connections have been proposed to be important for the reverberation of information necessary to access consciousness (Lamme, 2006). Results demonstrated that patients with right dorsolateral prefrontal damage and/or reduced integrity of the right SLF III presented an impaired conscious perception, needing a larger contrast to perceive the targets at threshold.

Consistent with previous observations, the right lateralized ventral network seems to be important for conscious perception. This ventral network, and in particular the temporo-parietal junction (TPJ), has been demonstrated to play an important role for the selection of information in cluttered scenes. TPJ deactivates when filtering distracting events, and activates when the target is presented (Doricchi et al., 2010; Shulman, et al., 2007). TPJ lesions also produce hemispatial neglect (Golay et al., 2008; Riddoch et al., 2010), a syndrome characterized by re-orienting deficits and impairments of conscious perception.

Damage to the right prefrontal lobe does not produce attention and/or consciousness deficits comparable to those observed in hemispatial neglect. In our study, validity effects on perceptual sensitivity and response criterion were comparable for patients and controls. As expected, frontal patients did not show any disengagement deficit, as the one observed after right parietal damage (Bartolomeo and Chokron, 2001). However, the neuropsychological tests demonstrated a specific impairment in selective attention that could be related to specific perception difficulties when the perceptual load was high in some of the neuropsychological tasks (such as the Minimal Feature Match and the Picture Completion test) (Murphy et al., 2016), and when detecting a near-threshold stimulus. Results demonstrated that, the more reduced the HMOA of the right SLF III, the larger the contrast needed to perceive the Gabors. This result suggests the importance of the communication between the parietal and frontal cortices for conscious perception, as proposed by some influential models (Dehaene and Changeux, 2011). The correlation was observed for right prefrontal damage patients, but not in the healthy control group. Given the distributed nature of conscious processing in the brain, the integrity of the HMOA of the right SLF III might not be so important in healthy controls, in which other brain regions or networks (thalamic: Bachmann, 2011; Seth et al., 2005) (fronto-parietal: Dehaene and Changeux, 2011) can also influence conscious perception.

Interestingly, all patients suffering from dorsolateral prefrontal damage in this study presented lesions to the inferior frontal gyrus. Previous literature on attention networks has pointed out that the inferior frontal gyrus has an important role in novelty detection (Kiehl et al., 2001; Corbetta and Shulman, 2002). As part of the attention network for goal-directed (top-down) selection for stimuli and responses, the temporoparietal cortex and the inferior frontal cortex are proposed to be involved in the detection of behaviorally relevant stimuli, particularly when they are salient or unexpected (Corbetta and Shulman, 2002). Damage to the inferior frontal gyrus might have affected conscious perception, raising the threshold to detect the targets (i.e. making them less sensible to change detection).

Dorsolateral prefrontal patients in our study also presented a more conservative response criterion than orbitofrontal-medial patients (note that this data could not be statistically analyzed due to the lack of variance of the score in the dorsolateral group). This result might suggest that the prefrontal lobe is related to the accumulation of evidence for perceptual decision-making, which is consistent with the proposed role of frontal (frontal eye field and dorsolateral prefrontal cortex) and parietal regions (lateral intraparietal area) in accumulating sensory evidence, by computing the difference between the activities of populations of neurons of other sensory regions, such as e.g. MT, that code for different directions of motion (see Heekeren et al., 2008; for a review). This observation is also consistent with previous studies in healthy participants in which non-invasive techniques, such as Transcranial Magnetic Stimulation (TMS) have been used to demonstrate the causal role of the right dorsolateral prefrontal cortex in conscious perception (Turatto et al., 2004) and metacognitive aspects of awareness (such as confidence ratings) (Rahnev, Nee, Riddle, Larson, & D'Esposito, 2016; Rounis et al., 2010).

Our results demonstrate the role of prefrontal damage in conscious perception in the absence of general attentional problems. When exploring frontal lobe contributions to conscious perception it is important to understand if the possible deficits observed after frontal lobe damage are or not associated to deficits in attentional processes such as deficits in selective attention, sustained attention, or working memory. Our group of patients used the peripheral cue in a comparable manner to the healthy control group. They did not demonstrate any deficit in sustained attention, as measured with the Auditory A Test. However, they did present deficits in selective attention (measured with the Picture Completion test), visual memory (measured with the Rey Figure test), and visual working memory (measured with the Corsi blocks test). Further analyses demonstrated that mean scores in each of these tests did not differ between patients with dorsolateral and orbitofrontal-medial prefrontal damage (selective attention: $W = 7.00$, $p = 0.857$, $BF_{10} = 0.558$, visual memory: $W = 5.50$, $p = 0.634$, $BF_{10} = 0.637$, visual working memory: $W = 3.00$, $p = 0.359$, $BF_{10} = 0.868$, and semantic fluency: $W = 7.00$, $p = 0.858$, $BF_{10} = 0.722$). Therefore,

although our group of patients did present general impairments in selective attention, visual memory, visual working memory, and semantic fluency, we cannot conclude that these deficits were related to conscious perception. We also reckon that this conclusion should be taken with caution given the sample size of the present study.

To conclude, our results demonstrate that lesions in the dorsolateral prefrontal cortex affect conscious perception of near-threshold information, which is associated to the integrity of the ventral branch of the SLF. These results support higher-level theories of consciousness, demonstrating the contribution of frontal regions to conscious perception.

Conflict of interest

The authors declare no conflict of interest.

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