

# The Neural Basis of Body Form and Body Action Agnosia

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## SUMMARY

Visual analysis of faces and nonfacial body stimuli brings about neural activity in different cortical areas. Moreover, processing body form and body action relies on distinct neural substrates. Although brain lesion studies show specific face processing deficits, neuropsychological evidence for defective recognition of nonfacial body parts is lacking. By combining psychophysics studies with lesion-mapping techniques, we found that lesions of ventromedial, occipitotemporal areas induce face and body recognition deficits while lesions involving extrastriate body area seem causatively associated with impaired recognition of body but not of face and object stimuli. We also found that body form and body action recognition deficits can be double dissociated and are causatively associated with lesions to extrastriate body area and ventral premotor cortex, respectively. Our study reports two category-specific visual deficits, called body form and body action agnosia, and highlights their neural underpinnings.

## INTRODUCTION

Brain lesions may disrupt visual object recognition in spite of relatively spared low-level visual perception, language, and general cognitive abilities (Biran and Coslett, 2003). This neuropsychological deficit, referred to as visual agnosia, may selectively affect the recognition of specific object categories (Caramazza and Shelton, 1998). A striking example of category-specific agnosia is the selective deficit in the visual processing and recognition of human faces referred to as prosopagnosia (Barton, 2003). This deficit seems to be associated with damage to the fusiform face area (FFA; Barton, 2003) and the occipital face area (OFA; Rossion et al., 2003; Sorger et al., 2007), two occipitotemporal regions selectively activated by visual presentation of human faces (Kanwisher et al., 1997; Gauthier et al., 2000; Haxby et al., 2000). Functional magnetic resonance imaging (fMRI) studies in healthy individuals have shown that visual processing

of nonfacial body parts selectively engenders bilateral activation of a lateral occipitotemporal region called extrastriate body area (EBA; Downing et al., 2001). EBA responds to viewing static and dynamic displays of the human body and its single parts, but not faces and objects (Peelen and Downing, 2007). More recent fMRI studies demonstrated the existence of another body selective area that is anatomically distinct from EBA. This area, located in the fusiform gyrus and known as fusiform body area (FBA), responds selectively to whole bodies and body parts and is adjacent to and partly overlaps the FFA (Peelen and Downing, 2005; Schwarzlose et al., 2005). FFA is more activated by the presentation of whole faces but also responds to face parts (Benuzzi et al., 2007; Rossion et al., 2000; Tong et al., 2000). In a similar vein, FBA responds more to whole bodies than to single body parts (Taylor et al., 2007). In contrast, EBA seems to be involved in processing the details of nonfacial body parts (Taylor et al., 2007; Urgesi et al., 2007b). This suggests that a network of areas is involved in extracting different information from face and body stimuli (Haxby et al., 2000; Peelen and Downing, 2007).

Viewing another person's acting body allows us to extract crucial social information related to the agent's identity and the meaning of the performed actions. Although intimately linked, the ability to perceive and to discriminate body forms and body actions relies on partially separated neural networks. Direct evidence for a double dissociation in processing body identity and body action in healthy subjects has been provided by a repetitive transcranial magnetic stimulation (rTMS) study in which the temporary inactivation of EBA impaired the visual discrimination of body forms but not of body actions; in contrast, the inactivation of ventral premotor cortex (vPMc) impaired the discrimination of body actions but not of body forms (Urgesi et al., 2007a). Although studies in healthy individuals hint at the existence of deficits in body processing similar to those reported for face processing, so far no neuropsychological evidence for these new types of visual agnosias has been provided. In two different studies, we explored the possible selective inability of brain damaged patients with lesions centered upon posterior or anterior areas in recognizing faces, body forms, and body actions. We used two psychophysics paradigms that tap the ability to (1) discriminate face parts, nonfacial body parts, and noncorporeal objects and (2) discriminate an actor's identity or the actions performed by him. The findings demonstrated the

**Table 1. Demographic and Clinical Information on the Patients' Groups with Anterior and Posterior Lesions**

Subj.	Les	Age	Days	Mot	Sens	VF	MMSE	Token	ExN	VE	PN
<b>Anterior Group</b>											
1	l F	50	115	<b>3</b>	–	–	27	65	–	–	0
2	l F	47	39	<b>3</b>	–	–	30	78	–	–	0.07
3	r F	73	52	0	–	–	24	55	–	–	0
4	l F	85	8	0	–	–	<b>22</b>	70	–	–	Np
5	l F-T	60	45	<b>3</b>	+	–	24	56	–	–	Np
6	l F	54	25	<b>1</b>	–	–	29	58	–	–	0
7	l F-T	51	47	<b>2</b>	–	–	28	58	–	–	0
8	l F-T	64	10	<b>1</b>	–	–	29	56	–	–	0
9	r F-T	72	44	<b>3</b>	–	–	29	78	–	–	0.07
10	r F-T	73	35	0	–	–	30	78	–	–	0
11	r F-T	41	8	<b>2</b>	–	–	29	72	–	–	Np
12	r F-T	60	18	0	–	–	28	70	–	–	–0.06
13	r F-T	62	68	<b>2</b>	–	–	24	70	–	–	–0.04
14	l F-T	68	21	<b>2</b>	–	–	26	70	–	–	–0.08
<b>Posterior Group</b>											
15	l T-O	79	6	0	–	–	30	78	–	–	0.04
16	l T-O	69	74	<b>2</b>	–	–	<b>21</b>	61	–	+	0
17	l T-O	64	98	<b>2</b>	+	–	24	67	–	–	0
18	l T-P	56	60	0	–	–	30	67	–	–	0
19	l P	66	90	0	–	–	30	70	–	–	Np
20	l T-O	61	57	0	–	–	24	56	–	–	Np
21	r T-O	49	103	<b>3</b>	–	–	29	78	+	+	<b>–0.37</b>
22	r T-O	57	11	0	–	–	30	78	–	–	0
23	r T-O	63	32	0	–	–	<b>22</b>	78	+	–	–0.08
24	r O	77	15	0	–	+	24	72	+	–	–0.04
25	r P-O	74	4	0	–	–	30	78	–	–	0.06
26	bil O	79	12	0	–	–	25	78	–	–	0.03
27	bil T-O	66	6	0	–	–	25	78	–	–	Np
28	bil O	76	6	0	–	–	30	78	–	–	0

Bold characters and the sign + indicate impaired performance. Les, cerebral areas affected by the lesion (r, right; l, left; bil, bilateral; F, frontal lobe; T, temporal lobe; O, occipital lobe; P, parietal lobe); Days, interval between stroke and examination; Mot, motor deficits (0-3, no deficit); Sens, sensorial disorders; VF, visual field deficits; MMSE, scores at Mini-Mental State Examination (cut-off = 24); ExN, Extrapersonal neglect (Albert test, drawing on memory and on copy); VE, visual extinction; PN, personal neglect (Comb and Razor test).

existence of two category-specific visual recognition deficits, hereafter called body form and body action agnosia. Furthermore, by using advanced brain lesion mapping procedures (Bates et al., 2003; Rorden et al., 2007), we determined the cortical areas causatively associated with these two types of body agnosia.

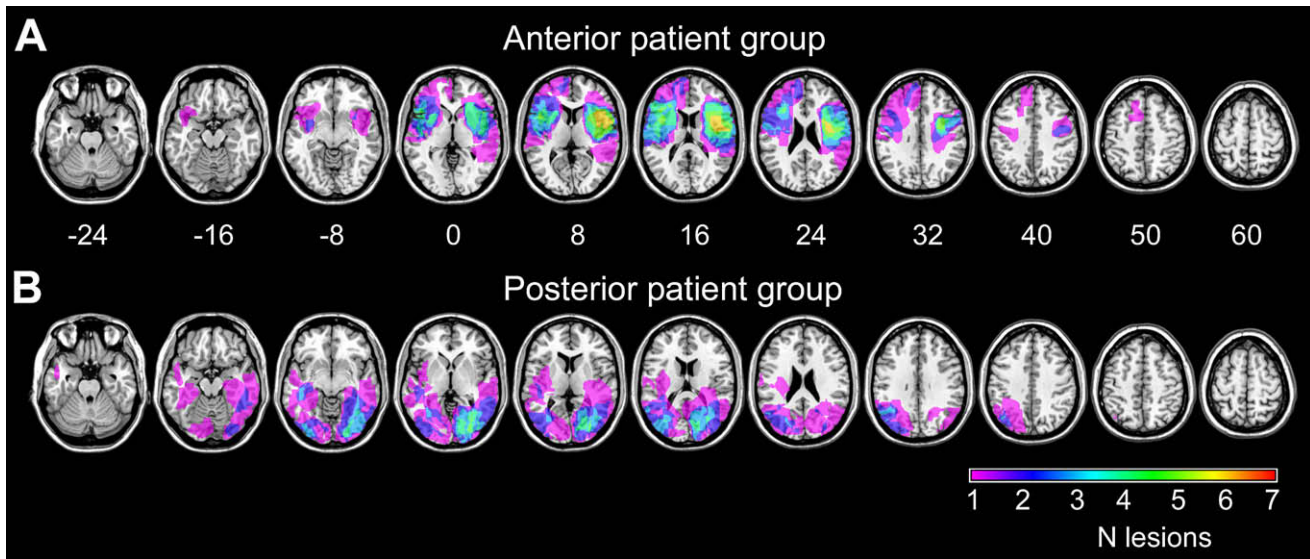
**RESULTS**

We tested the perceptual performance of 28 patients with lesions involving the anterior (n = 14) or the posterior areas (n = 14) of the left hemisphere (LH) and/or right hemisphere (RH). None of the patients presented with clinical signs of visual agnosia, apraxia, or noncontextual language comprehension deficits (see Table 1 and the Supplemental Material available online). Figure 1 shows the overlap between the lesions of patients

with anterior (Figure 1A) and posterior damage (Figure 1B). No significant difference was observed in the extent of the lesions of the patients with anterior (mean = 48.83 cc, SD = 26.86) and posterior damage (mean = 37.17 cc, SD = 22.37;  $t_{26} = 1.25$ ,  $p = 0.223$ ). Fourteen age- and education-matched healthy individuals served as control group.

**Study 1. Body, Face, and Object Part Discrimination**

Based on neuroimaging literature we used stimuli adept to activate cortical structures specifically dedicated to processing body (EBA and FBA; Peelen and Downing, 2007), face (OFA and FFA; Haxby et al., 2000), and object forms (lateral occipital complex area, LOC; Grill-Spector et al., 2001; Malach et al., 1995). Participants performed a two-choice matching-to-sample visual discrimination task in which they were required to decide which of two images matched a single sample seen previously.

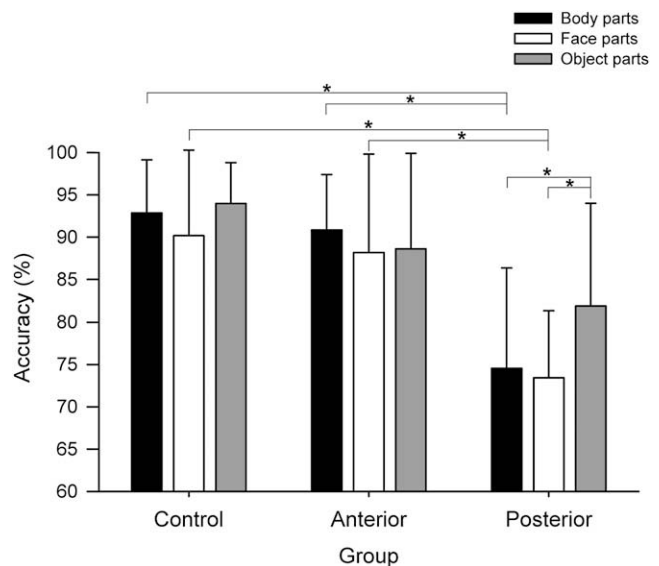


**Figure 1. Overlaps of the Patients' Lesions**

The lesions of each patient within each group was overlaid on the standard brain. The number of overlapping lesions in the anterior damage (A) and posterior damage group (B) is illustrated by different colors that code for increasing frequencies from violet (lesion in one patient) to red (lesion in seven patients).

Stimuli consisted of body parts, face parts, and noncorporeal objects (see Figure S1). To control for the type of processing required by the three stimulus categories, we tested the extent to which discrimination of the experimental stimuli (body, face, and object parts) was affected by inversion. In a separate experiment, we asked control participants to perform match-to-sample tasks with target and probe stimuli in upright or inverted position. We showed a significant, although small, inversion effect for face parts only, suggesting that face parts processing was based on configural analysis more than body and object parts processing (see Supplemental Material and Figure S2). Percent correct responses of patients and controls (Figures 2 and S3) were entered in a  $3 \times 3$  ANOVA with group (anterior damage patients, posterior damage patients, controls) as between-subject and stimulus category (body, face, and object) as within-subject variable. The significance of the main effect of group ( $F_{2,39} = 14.61$ ,  $p < 0.001$ ) was accounted for by the lower discrimination performance of patients with posterior damage (mean = 76.64%) as compared to patients with anterior damage (mean = 89.21%,  $p < 0.001$ ) and controls (mean = 92.34%,  $p < 0.001$ ). No difference was observed between patients with anterior damage and controls ( $p = 0.316$ ). The main effect of stimulus category was significant ( $F_{2,78} = 5.08$ ,  $p = 0.0008$ ), because discrimination accuracy for face parts (mean = 83.93%) was lower than for object parts (mean = 88.17%;  $p = 0.003$ ). Discrimination accuracy for body parts (mean = 86.09%) was not different from that for face ( $p = 0.109$ ) and object parts ( $p = 0.122$ ), thus showing that perceptual discrimination of body parts was not differently difficult per se. Crucially, a significant interaction between stimuli category and group ( $F_{4,78} = 2.56$ ,  $p = 0.045$ ) was found. Post hoc tests revealed that in the visual discrimination of body parts patients with posterior damage (mean = 74.55%, SD = 11.81) were more impaired than patients with anterior damage (mean = 90.85%, SD = 6.54;  $p = 0.009$ ) and controls (mean = 92.86%,

SD = 6.3;  $p = 0.004$ ). In a similar vein, in the visual discrimination of face parts patients with posterior damage (mean = 73.44%, SD = 7.92) were more impaired than patients with anterior damage (mean = 88.17%, SD = 11.64;  $p = 0.014$ ) and controls (mean = 90.18%, SD = 10.1;  $p = 0.007$ ). In contrast, for the visual discrimination of object parts the performance of posterior damage patients (mean = 81.92%, SD = 12.08) was similar to that of



**Figure 2. Performance in the Discrimination of Body, Face, and Object Parts**

Mean ( $\pm$ SD) accuracy of controls and of the patients with anterior and posterior lesions in the discrimination of body parts, face parts, and object parts. Patients with posterior brain damage were selectively impaired in the discrimination of body parts and face parts, but not of object parts. \* $p < 0.05$ .

**Table 2. Regions Associated with Impaired Performance in Body, Face, and Object Discriminations in Study 1 and with Relative Impairment in Body Form or in Body Action Discriminations in Study 2**

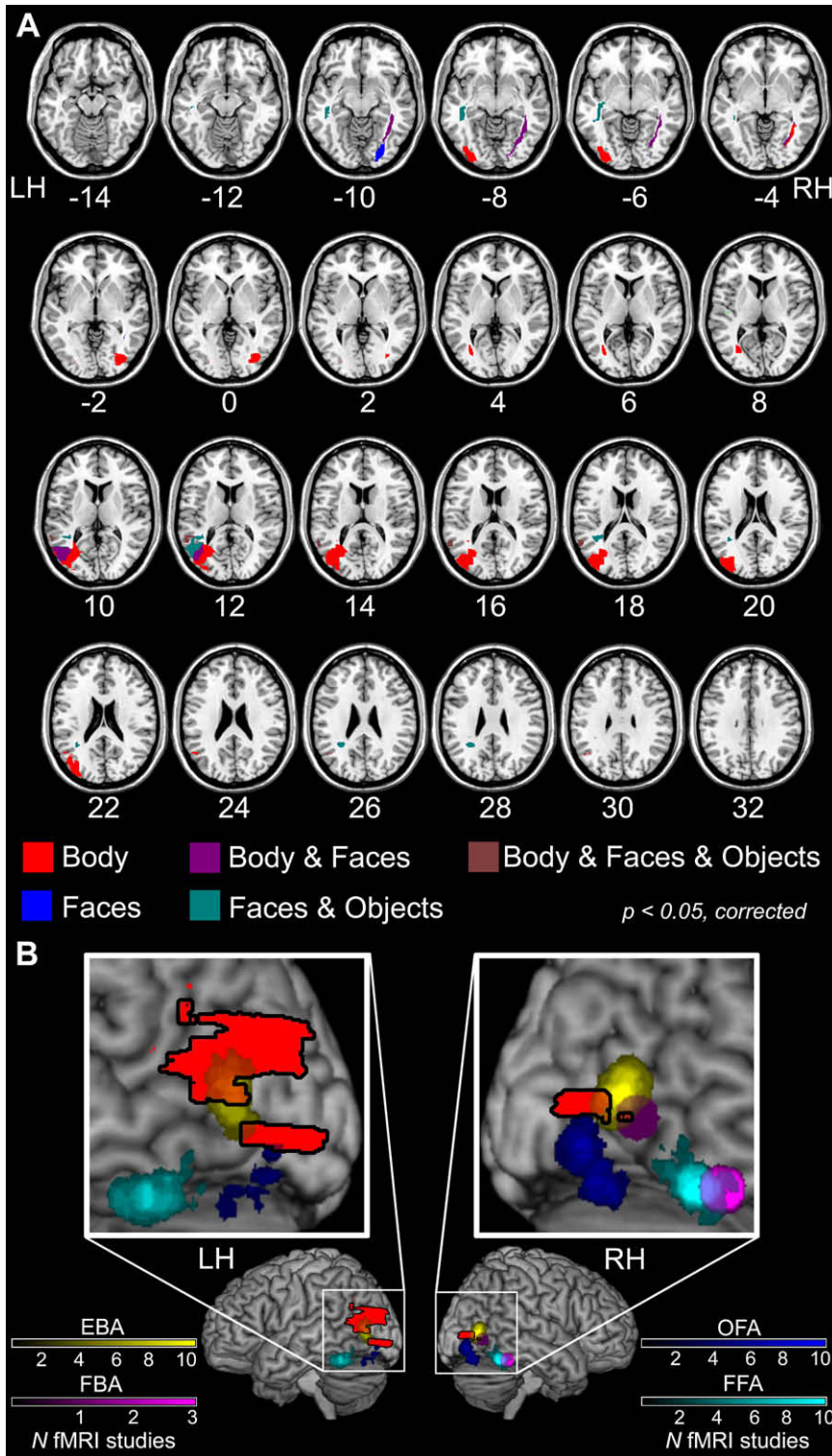
Region	x	y	z	BM Z Max	n Voxels
<b>Study 1</b>					
<b>Body Discrimination</b>					
Left inferior occipitotemporal cortex	-34	-86	-7	2.722	1,158
Left middle occipitotemporal cortex	-38	-76	13	5.03	6,817
Left superior temporal cortex	-58	-53	19	6.76	466
Right inferior occipitotemporal cortex	34	-55	-6	4.528	1,395
Right middle occipitotemporal cortex	34	-79	0	6.76	841
<b>Face Discrimination</b>					
Left inferior temporal gyrus, white matter	-43	-30	-8	3.886	669
Left superior temporal cortex	-42	-60	17	4.038	2,689
Right inferior occipitotemporal cortex	30	-64	-6	2.769	1,281
<b>Object Discrimination</b>					
Left inferior temporal gyrus, white matter	-43	-30	-8	7.275	669
Left superior temporal cortex	-54	-58	17	5.676	1,817
<b>Study 2</b>					
<b>Form versus Action Discrimination</b>					
Left inferior occipitotemporal cortex	-34	-86	-7	4.275	1,158
Left middle occipitotemporal cortex	-38	-77	14	9.232	4,481
Right middle occipitotemporal cortex	35	-81	6	4.455	6,577
<b>Action versus form discrimination</b>					
Left ventral premotor cortex	-41	7	13	51.43	38,682
Right ventral premotor cortex	34	20	19	3.296	5,682

For each region, the MNI coordinates of the center of mass are provided along with the maximum Brunner-Munzel (BM) z statistic obtained in each cluster and the number (n) of clustering voxels that survived the threshold of  $p < 0.05$ , false discovery rate corrected.

anterior damage patients (mean = 88.62%, SD = 11.27;  $p = 0.243$ ) and marginally significantly worse than that of controls (mean = 93.97%, SD = 4.82;  $p = 0.055$ ). No significant difference was observed between anterior damage patients and controls in the visual discrimination of body ( $p = 0.708$ ), face ( $p = 0.726$ ), and object parts ( $p = 0.379$ ). Furthermore, the visual discrimination of body parts was significantly more impaired than that of object parts in the posterior ( $p = 0.002$ ), but not in the anterior patient ( $p = 0.367$ ) and control group ( $p = 0.63$ ). In a similar vein, discrimination accuracy was lower for face parts than for object parts in the patients with posterior damage ( $p < 0.001$ ), but not in the patients with anterior damage ( $p = 0.847$ ) and in controls ( $p = 0.138$ ). Discrimination accuracy for body and face parts was comparable in both posterior ( $p = 0.63$ ) and anterior ( $p = 0.297$ ) patient groups as well as in controls ( $p = 0.278$ ). The analysis of the data of unilateral lesion patients showed no effect of the damaged hemisphere (see [Supplemental Material](#)), thus suggesting that patients with damage to posterior areas of the LH and RH were selectively impaired in the visual discrimination of the forms of body and face parts, but not of object parts.

To determine the lesion correlates of body, face, and object discrimination performances and to explore the possible active association of specific lesions with deficits in discriminating body and face parts, we performed a voxel-based lesion-symptom mapping (VLSM) analysis. We entered as predictors in the

VLSM analysis the percent correct responses in body, face, and object part discrimination of patients with lesions in posterior and anterior areas of the LH and RH. The regions associated with impaired performance in body, face, and object part discrimination, along with the coordinates of the center of mass based on the Montreal Neurological Institute (MNI) probabilistic brain atlas, are listed in [Table 2](#). The VLSM analysis revealed that impaired performance in body part discrimination was associated with lesions of bilateral inferior and middle occipitotemporal cortex and of a left hemisphere region located in the superior temporal sulcus ([Figure 3A](#) and [Table 2](#)). Lesioned voxels in the left inferior occipitotemporal cortex clustered in the inferior occipital gyrus (BA 19). While this cluster was in close proximity to the left fusiform gyrus, it was more posterior than the location of fMRI activations to bodies and faces in the fusiform gyrus (see [Supplemental Material](#)). In contrast, the RH ventral cluster was in a more ventromedial location and involved the right fusiform gyrus and the underlying white matter (BA 19 and 37), in a location corresponding to FBA ([Peelen and Downing, 2005; Schwarzlose et al., 2005; see Supplemental Material and Table S1](#)). The left and right middle occipitotemporal clusters involved the left and right middle occipital and temporal gyri (BA 19 and 37) and correspond to the location of EBA as shown in fMRI studies ([Downing et al., 2001; see Figure 3B, Table S1, and Supplemental Material](#)). While lesions of left and right middle occipitotemporal



**Figure 3. Voxel-Based Lesion-Symptom Mapping for Body, Face, and Object Discrimination**

(A) The maps show the voxels selectively associated with impaired performance in the discrimination of body parts (red) and face parts (blue). Furthermore, additional colors indicate (as shown in the figure) voxels associated to deficits in the discrimination of body and face parts, face and object parts, and body, face, and object parts. No area was selectively associated with deficits in object part discrimination. The behavioral measures refer to the patients' accuracy in body, face, and object parts discrimination. In all colored voxels, *p* values reached the false discovery rate (FDR) corrected significance threshold of *p* < 0.05. (B) Cortical renderings of the voxel clusters selectively associated with deficits in processing body parts, but not face and object parts (red colors) with the areas of body and face selective activations in fMRI. We created 6 mm radius ROIs around the coordinates reported in the fMRI studies that localized the extrastriate body area (EBA), the fusiform body area (FBA), the occipital face area (OFA), and the fusiform face area (FFA). The map for each functional area represents the number of fMRI studies that localized the category-selective activations in each voxel (see Supplemental Material and Table S1). Left hemisphere (LH) is on the left, and right hemisphere (RH) is on the right.

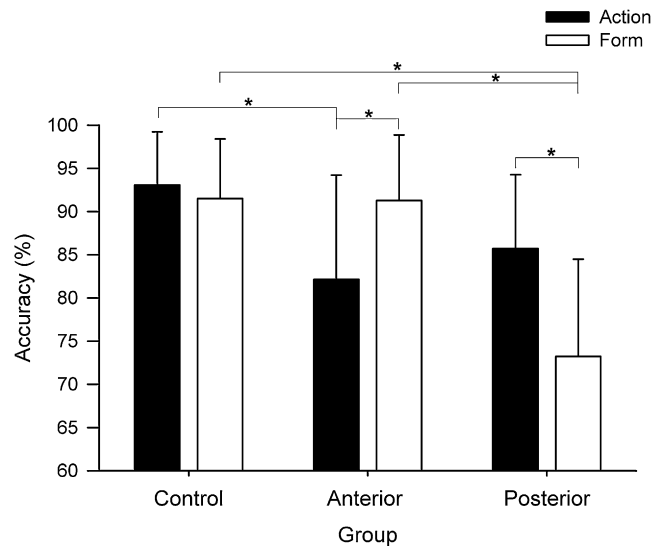
cortex were selectively associated with impaired discrimination of body parts, but not of face and object parts, the body related cluster in the right ventromedial occipitotemporal cortex partially overlapped with the RH cluster associated with impaired face perception abilities (Figure 3A). Indeed, we found in the right in-

ferior occipitotemporal cortex voxels selectively associated with body perception deficits, voxels selectively associated with face perception deficit and voxels associated with deficits in the processing of body and face parts, but not of object parts. Face and body clusters partially overlapped in the most ventral and medial voxels corresponding to the right fusiform areas that are selectively activated when observing bodies (FBA; Peelen and Downing, 2005; Schwarzlose et al., 2005) and faces (FFA; Kanwisher et al., 1997). Voxels selectively associated with deficits in the discrimination of face parts but not of body parts were in a more lateral and posterior location, corresponding to OFA (Rossion et al., 2003; Sorger et al., 2007; see Table S2 and Supplemental Material). A further cluster in the LH involved the white matter at the border between the inferior and middle temporal gyri and the fusiform gyrus (BA 37 and 20). Damage to this LH region, however, affected the processing of both faces and objects, thus suggesting that this effect may be due to disconnection of early visual areas from anterior temporal areas involved in high-order processing of objects (Simons et al., 2003). In a similar vein, the cluster located

in the left superior temporal sulcus, involving the middle and superior temporal gyri (BA 21 and 22) and the underlying white matter, was associated with impaired performance in discrimination of body, face, and object parts. The location and functional properties of this LH cluster suggest its possible link with the semantic categorization of objects (Borowsky et al., 2007). In keeping with fMRI studies showing that category-selective activations in the posterior occipitotemporal cortex are consistent only for bodies and faces (Downing et al., 2006), no area was selectively associated with object parts discrimination deficit. Furthermore, since we did not include patients with heavy visual field deficits or clinical signs of visual agnosia, the VLSM analysis did not reveal any early visual cortex cluster associated to impairments in any of the stimuli categories.

### Study 2. Body Form and Body Action Discrimination

Patients performed a two-choice matching-to-sample visual discrimination task in which they were required to decide which of two images matched a single sample seen previously. Stimuli consisted of pictures depicting body parts (see Figure S4) that are likely to activate EBA (Downing et al., 2001). However, all the pictures implied action and are likely to activate vPMc (Urgesi et al., 2006). The matching and nonmatching stimuli in each pair depicted either the same action performed by two different models (body form discrimination) or the same model performing two different actions (body action discrimination). Percent correct responses of patients and controls (Figures 4 and S5) were entered in a  $3 \times 2$  ANOVA, with group as between-subject and task (action and form discrimination) as within-subject variable. In keeping with results of study 1, we found a significant main effect of group ( $F_{2,39} = 8.94$ ,  $p < 0.001$ ), showing that patients with posterior lesions (mean = 79.46%) performed worse than patients with anterior lesions (mean = 86.72%,  $p = 0.022$ ) and controls (mean = 92.3%,  $p < 0.001$ ). Interestingly, the difference between the performance of patients with anterior lesion and controls across the two tasks was only marginally significant ( $p = 0.075$ ). The main effect of task was nonsignificant ( $F_{1,39} = 1.69$ ,  $p = 0.202$ ), suggesting a comparable task difficulty. Crucially, we found a highly significant interaction between task and group ( $F_{2,39} = 24.6$ ,  $p < 0.001$ ). Post hoc tests revealed that, while performance of controls in the action (mean = 93.08%, SD = 6.15) and form tasks (mean = 91.52%, SD = 6.87) was comparable, patients with anterior lesions had lower scores in the action (mean = 82.14%, SD = 12.1) than in the form discrimination task (mean = 91.3%, SD = 7.57;  $p < 0.001$ ). In contrast, patients with posterior damage performed with significantly less accuracy in the form (mean = 73.21%, SD = 11.28) than in the action discrimination task (mean = 85.71%, SD = 8.56;  $p < 0.001$ ). Furthermore, in the action discrimination task patients with anterior damage were impaired with respect to controls ( $p = 0.027$ ), while the performance of posterior damage patients was comparable to that of controls ( $p = 0.126$ ). In contrast, in the form discrimination task patients with posterior damage were impaired with respect to controls ( $p < 0.001$ ), while the performance of anterior damage patients was comparable to that of controls ( $p = 0.959$ ). Finally, while patients with anterior versus posterior damage performed at comparable level in the action discrimination task ( $p = 0.412$ ), the latter patients per-

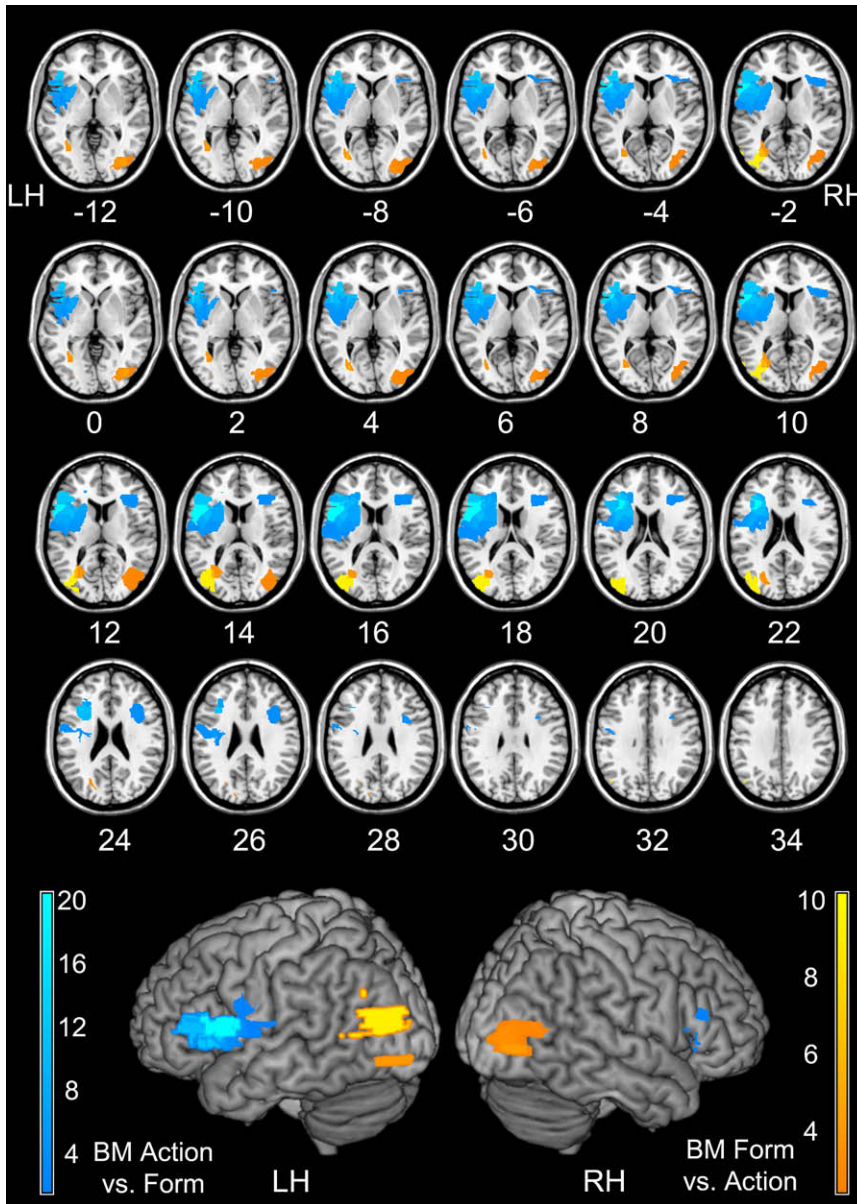


**Figure 4. Performance in the Discrimination of Body Action and Body Forms**

Mean ( $\pm$ SD) accuracy of controls and of the patients with anterior and posterior lesions in the body action and body form discrimination tasks. Results indicate a double dissociation between processing of body actions and body forms and lesions to anterior and posterior brain areas. \* $p < 0.05$ .

formed significantly worse than the former in the form discrimination task ( $p < 0.001$ ). Supplementary analyses conducted only on the data of the patients with unilateral lesions showed that the double dissociation between the action and form tasks and anterior and posterior lesions was independent from the side of the damaged hemisphere (see Supplemental Material). The reported pattern of results shows a clear double dissociation where patients with anterior lesions were more impaired in the body action than in the body form discrimination task and patients with posterior lesions were more impaired in the body form than in the body action discrimination task. This rules out that different task difficulties or the general perceptual deficit exhibited by patients with posterior damage may explain the results. Furthermore, despite the interindividual differences in the absolute level of performance, the behavioral pattern was consistent within each group thus hinting at the robustness of the experimental effects (see Figure S5). Therefore, our findings suggest that lesions involving the anterior areas, either on the LH or on the RH, selectively impair the ability to match the action performed by other individuals. By contrast, lesions involving the posterior areas, either on the right or on the left hemisphere, impair performance in matching the identity of the acting body.

To determine the areas associated with higher impairment in the visual discrimination of body form than body action and vice versa, we entered LH and RH patients' behavioral performance for the two tasks in a VLSM analysis. The ratios between percent correct response in the action discrimination task and percent correct responses in the form discrimination task were entered as predictors in the VLSM analysis. Figure 5 and Table 2 show the areas that were significantly associated with impaired performance in the body form discrimination task. Bilaterally symmetric clusters centered over the lateral occipitotemporal



**Figure 5. Voxel-Based Lesion-Symptom Mapping for Body Action and Body Form Discrimination**

The maps show the z-statistics corresponding to Brunner and Munzel (BM) test comparing the behavioral performance of lesioned and intact patients on a voxel-by-voxel basis. The behavioral measures were the ratio between the patients' accuracy in the body action and body form discrimination task and its reciprocal. In all colored voxels p values reached the false discovery rate (FDR) corrected significance threshold of  $p < 0.05$ . Impaired performances on the action discrimination task were significantly associated with lesions of left and right ventral premotor cortex (blue color scale). Impaired performances in the body form discrimination task were associated with lesions of left and right middle occipitotemporal cortex and of left inferior occipital cortex (yellow color scale). Left hemisphere (LH) is on the left, and right hemisphere (RH) is on the right.

A complementary pattern of results was obtained from the VLSM analysis for the relative discrimination performance in the body action versus the body form task (Figure 5 and Table 2). A large cluster of voxels centered on the pars opercularis and extending into the pars triangularis of the left inferior frontal gyrus as well as into the insula and the rolandic operculum and the underlying white matter was significantly associated with a more impaired performance in the action than the form discrimination task. The RH cluster associated with impaired performance in the action discrimination task was smaller than but correspondent in location to the LH cluster. Interestingly, it involved the pars opercularis and triangularis of the inferior frontal gyrus. The location of the action-related clusters in the LH and RH corresponds to the

cortex resulted significantly associated with more impaired performance in the visual discrimination of body forms as compared to visual discrimination of body actions. The clusters in both the LH and RH were located at the border between the middle occipital gyrus and the middle temporal gyrus (BA 19 and 37). The location of these bilateral clusters associated with impaired form discrimination performance corresponds to the location of left and right EBA (Downing et al., 2001). Interestingly, the LH and RH clusters corresponded for size and location to the LH and RH middle occipitotemporal clusters that were associated to body part discrimination deficits in study 1. A further LH cluster was located in the left inferior occipital gyrus (BA 19) nearby the fusiform gyrus and corresponded for size and location to the inferior occipital cluster associated with body part discrimination deficits in study 1.

vPMc. In keeping with the finding that magnetic stimulation of vPMc impairs visual discrimination of body actions but not of body forms (Urgesi et al., 2007a), the present results indicate that brain lesions involving left vPMc induce a specific impairment in the action discrimination task.

## DISCUSSION

Damage to human cerebral cortex can lead to a selective breakdown of visual recognition processes, a class of neuropsychological defects referred to as visual agnosias. Specific deficits in recognizing peoples' identity from their faces (Barton, 2003) or in recognizing entities belonging to certain conceptual categories, such as natural or man-made items, have been reported (for review on category-selective deficits, see Caramazza and

Shelton, 1998; Biran and Coslett, 2003). Our neuropsychological paradigm and lesion mapping procedures demonstrate the existence of body-specific recognition disorders that are distinct from face-selective recognition deficits both at the behavioral and at the neural level. Most importantly, we demonstrate two classes of brain damage-induced category-specific visual recognition disorders that we call body form and body action agnosias.

### Neural Correlates of Face Discrimination Deficits

The first main result of study 1 is that patients with posterior brain lesions were selectively impaired in matching body parts and face parts, but showed no difficulty in matching noncorporeal objects.

Neuropsychological studies have described patients with prosopagnosia, i.e., the conspicuous difficulty or inability to identify a familiar face in the presence of intact person recognition on the basis of gait, voice, clothes, jewelry, and of spared non-person-related object recognition (Barton, 2003). Evidence of brain alterations around the fusiform gyrus has been reported in both congenital (Behrmann et al., 2007) and acquired (Barton, 2003; Schiltz et al., 2006) prosopagnosia. However, neuroimaging studies in patients (Rossion et al., 2003; Sorger et al., 2007) as well as rTMS studies in healthy individuals (Pitcher et al., 2007) have emphasized that OFA may also play an important role in face processing. While FFA may be more involved in configural processing of faces, OFA may be involved in processing the details of face parts (Yovel and Kanwisher, 2005). As configural processing is reduced for isolated face features (Rhodes et al., 1993; Tanaka and Farah, 1993), one may wonder whether processing of our face part stimuli involved FFA activations. In our control experiment, however, we showed that inversion disrupted processing of face parts but not of body and object parts. This result, together with the lesion analysis, would suggest that configural processing and FFA are, at least partially, involved during our face parts discrimination task (see [Supplemental Material](#)).

One important result of our study is the association between impairment in face parts discrimination and lesions of the right ventromedial temporal cortex, in close proximity to FFA (Kanwisher et al., 1997), and of the right inferior occipital cortex, in a location corresponding to right OFA. This suggests that intact face perception abilities require the functional interconnection between the different areas belonging to the distributed system for face perception (Haxby et al., 2000; Rossion et al., 2003; Schiltz et al., 2006). In keeping with neuropsychological (Barton, 2003), neuroimaging (Kanwisher et al., 1997), and rTMS studies (Pitcher et al., 2007) showing a RH dominance of FFA and OFA, the ventromedial and lateral temporal lobe clusters associated with face parts discrimination deficits were lateralized to the RH. Note that the VLSM clusters involved not only gray matter areas but largely extended into the underlying white matter. Therefore, in contrast to fMRI studies which allow us to investigate only the functional specialization of gray matter, lesion mapping analysis can highlight the behavioral consequences of damage and thus provide an important advance toward a functional neuroanatomy of white matter.

### Neural Correlates of Body Perception Deficits

Along with deficits in processing face part stimuli, patients with posterior damage presented with selective deficits in the visual

discrimination of body parts. Therefore, we provide brain-damage related evidences of selective deficits in the visual discrimination of bodies. While the issue of face recognition deficits has attracted much interest, so far no neuropsychological study has explored the possible existence of body-specific visual agnosia. Neuropsychological evidence that non facial body parts and full bodies may represent a specific knowledge category comes from single case studies of patients with selectively impaired (Sacchett and Humphreys, 1992; Suzuki et al., 1997) or spared (Shelton et al., 1998) ability to name body parts or to understand terms related to them (see also Kemmerer and Tranel, 2008). Moreover, neuropsychological studies have shown that while frontoparietal lesions are associated with disorders of body schema (on-line coding of body postures), left temporal lesions are associated with deficits in the semantic (knowledge of names and functions of body parts) and structural (location of body parts) representations of the body (Schwoebel and Coslett, 2005). Specific body-related disturbances concerning out-of-body perceptions (Blanke et al., 2004), disownership of body parts (Aglioti et al., 1996; Moro et al., 2004), deficits in the representation of the spatial relationships between body segments (Guariglia et al., 2002), and the general semantics of body structures (Coslett et al., 2002) have also been reported. While the body-related disorders reported in previous studies regard the disruption of body knowledge at a representational level, no study has so far reported selective deficits in processing human bodies at the perceptual level.

Body perception abilities may be spared in patients with face perception deficits (Duchaine et al., 2006) and impaired in patients with objects agnosia and intact face perception (Moscovitch et al., 1997), thus suggesting that the neural correlates of face and body processing are dissociated. On the other hand, the coexistence of body and face processing deficits in the absence of object processing deficits has been reported in patients with congenital prosopagnosia (Righart and de Gelder, 2007). In keeping with this study, posterior damage patients in the present study were impaired in processing face part as well as body part stimuli. However, lesion mapping analysis revealed that deficits in body and face parts discrimination were associated with damage in partially different areas. Lesions involving the right ventromedial temporal cortex were associated with deficits in the visual discrimination of both body and face parts. This suggests that, due to their anatomical proximity, damage of the neural connections from occipital to the fusiform areas that respond selectively to bodies (FBA; Peelen and Downing, 2005; Schwarzlose et al., 2005) and faces (FFA; Kanwisher et al., 1997) may induce deficits in the visual processing of body and face parts. The lesion correlates of body processing were segregated from that of face processing in the lateral temporal cortex, where a bilateral occipitotemporal region corresponding to EBA was associated with deficits in body but not face processing. The role of lesions involving EBA in the observed body processing deficits is in keeping with a previous rTMS study demonstrating that interference with EBA impairs the visual discrimination of body forms, but not of face and object forms (Urgesi et al., 2004). Thus, another main result of study 1 concerns the neural correlates of body part discrimination deficits. Patients with lesions involving EBA and FBA presented specific deficits in processing the

human bodies but were almost intact in noncorporeal objects recognition. This expands previous neuroimaging studies reporting that the representation of the human body engages neural structures partially distinct from those subserving the representation of noncorporeal objects (Downing et al., 2001; Peelen and Downing, 2007).

One may wonder why, albeit rare, prosopagnosia stands as a conspicuous clinical deficit whereas body agnosia can be documented only by using sensitive tests like those used in our studies. Two non-mutually-exclusive explanations can be offered. The first is that under standard daily life interaction conditions, the most fundamental cues to identity are provided by faces more than by bodies, thus limiting the importance given by patients and clinicians to body perception abilities. It is relevant that no posterior damage patient in the present study reported spontaneously or after explicit request the subjective experience of having difficulties in recognizing human bodies and body parts in daily life. Thus, disclosing body perception deficits may require the use of accurate and sensitive testing procedures. The second possible explanation is that body selective areas are small and partially overlap with other occipitotemporal areas involved in object recognition (LOC; Grill-Spector et al., 2001; Malach et al., 1995), face recognition (FFA and OFA; Kanwisher et al., 1997), or motion processing (middle temporal areas; Tootell et al., 1995). It is thus plausible that the deficits in body perception may be masked by the coexistence of non category-selective form or motion agnosia. Furthermore, deficits of visual body processing following unilateral or bilateral lesions may be partially compensated for by the remaining intact body-selective areas in the ipsilateral or in the contralateral hemisphere.

### Phenomenology and Neural Basis of Body Form and Body Action Agnosia

Neuroimaging studies have demonstrated that body selective occipitotemporal areas are activated by presentation of both moving and static images of human bodies and body parts, independently of whether they imply motion or not (Downing et al., 2001). On the other hand, observation of actual (e.g., Costantini et al., 2005; Rizzolatti and Craighero, 2004) or implied (Urgesi et al., 2006) body actions activates a frontoparietal mirror-neuron system that matches action observation and execution and that is not responsive to static bodies. This scenario would suggest that while body-selective areas in the occipitotemporal cortex may be involved in extrapolating information about identity from body forms, the frontoparietal mirror neuron system may be involved in perceiving and understanding body actions. However, the type of correlational studies discussed above cannot establish whether activation of a given area is crucial rather than epiphenomenal to task performance. Brain-damaged patients may constitute an ideal model for testing the active role of specific cortical areas in body form and body action perception. Interestingly, a few studies in apraxic patients suggest that neuropsychological deficits in action understanding may follow lesions of the frontal (Pazzaglia et al., 2008) or the parietal cortex (Buxbaum, 2001; Heilman et al., 1982). Saygin et al. (2004) showed that aphasic patients with lesions of the left premotor and sensorimotor cortex were impaired in an action com-

prehension test that required matching pictures or names of actions to a picture of the corresponding object. Furthermore, a recent study using VLSM methods show that lesions of superior temporal and ventral and dorsal premotor cortex of the LH are associated with reduced sensitivity to biological motion perception (Saygin, 2007). Although patients with LH and RH damage exhibited impaired perception of biological motion, a VLSM analysis was performed only in the former group. Therefore, our study expands previous knowledge by demonstrating that (1) both left and right ventral premotor cortices are causatively associated with action perception and (2) specific neural substrates underpin body form and body action agnosia.

Importantly, so far no neuropsychological study has documented a double dissociation between the visual discriminations of body forms and body actions following brain lesions involving occipitotemporal and premotor cortex. Therefore, the main result of study 2 is that body form and body action recognition are double dissociated. Indeed, while patients with posterior lesions were impaired in the body form discrimination task, the opposite pattern of results was found in body action discrimination tasks. Note that action discrimination deficits were observed in the absence of evident apraxic and/or aphasic deficits, thus ruling out the possible influence of disorders in gesture production and language comprehension and hinting at the gnostic component of the deficits. Crucially, the same match-to-sample operation was required in the two tasks. Moreover, the experimental stimuli were the same in the two tasks, thus ruling out any effect of low-level visual differences. Thus, any dissociation between the tasks was likely to emerge from the implicit discrimination of differences in the morphological details of the model's body part or in the type of action implied by the model's posture. In accordance with the results of study 1, lesion mapping analysis revealed that deficits in the visual discrimination of body form were associated with lesions of the left and right middle occipital and temporal gyri in the LH and RH, in close proximity to the location of EBA (Downing et al., 2001). The inferior occipitotemporal clusters selectively associated with body form perception deficits reflect damage to the body selective areas in the fusiform cortex, which may correspond to FBA (Peelen and Downing, 2007). By contrast, deficits in body action discrimination were associated with lesions of the left and right vPMc. Interestingly, these regions are activated during action observation in neuroimaging studies with healthy individuals (reviewed in Rizzolatti and Craighero, 2004). Furthermore, the ventral premotor clusters correspond to the region that, according to interferential rTMS studies, is actively involved in visual discrimination of static (Urgesi et al., 2007a; Candidi et al., 2007) and moving displays of body actions (Pobric and Hamilton, 2006) as well as in the ability to imitate observed actions (Heiser et al., 2003).

### Conclusions and Future Directions

Our findings demonstrate specific deficits in body processing in brain-damaged patients, in addition to the well-known face-processing deficits. Moreover, the causative evidence coming from this brain lesion study supports the hypothesis that visual analysis of human body stimuli is based on the division of labor into two cortical systems, with EBA and FBA representing the actors' identity and vPMc mapping the observed action in a neutral

format with respect to the identity of the acting bodies. While anterior, premotor areas may represent the observed actions without taking into account the actor's identity, posterior, occipitotemporal areas may be involved in mapping morphological features of human bodies. This function may be fundamental for keeping constant the identity of others even when body configurations change enormously and at very fast rates during action. Nevertheless, occipitotemporal areas may receive modulatory signals from sensorimotor systems and thus may be involved in the multimodal representation of the actors' body identity (Astafiev et al., 2004). Our report of two specific forms of visual agnosia not only may help to deepen our knowledge about category-based processing of the world but also may have relevant clinical implications. Indeed, the sensitive tests used in our study may represent an additional diagnostic tool for the clinical assessment of gnostic functions. Finally, considering that most rehabilitation techniques make use of visual demonstrations of movements and postures the study may also have an impact on rehabilitation practice.

## EXPERIMENTAL PROCEDURES

### Participants

Twenty-eight patients suffering from ischemic or hemorrhagic stroke were recruited from the Neurology and Rehabilitation Units of the Sacro Cuore Hospital (Negrar, Verona) and at the Hospital of the Fondazione Santa Lucia (Rome) over a 30 month period. The main inclusion criterion was the presence of lesions, documented by CT (26 patients) or T1-weighted MRI (2 patients) scans, involving the prerolandic (anterior group) or the temporo-parietal-occipital structures (posterior group). Therefore, patients with large, anterior and posterior hemispheric lesions and with involvement of subcortical structures were excluded from the study. Furthermore, to avoid primary visual deficits possibly masking any category-specific perceptual impairment, we excluded patients with heavy visual field deficits. Moreover, no patient with clinical signs of visual agnosia or apraxia was included in the study (Supplemental Material). Each group was comprised of 14 patients (5 females in both groups). In the anterior group, there were eight and six patients with left- and right-sided lesions, respectively. In the posterior group, there were six, five, and three patients with left-sided, right-sided, and bilateral lesions, respectively. Bilateral lesions were due to a single cerebrovascular accident. All the patients were native Italian speakers and were right-handed according to Briggs and Nebes (1975) laterality inventory. None had a history of psychiatric diseases or previous neurological disorders. A group of 14 healthy individuals (7 females) with no neurological, psychiatric, or other medical problem served as control group. All the control individuals were native Italian speakers and were right-handed. The three groups were matched for age (control, mean = 66.1 years, range = 54–78 years; anterior, mean = 61.4 years, range = 41–85 years; posterior, mean = 66.9 years, range = 49–79 years;  $F_{2,39} = 1.29$ ,  $p = 0.287$ ) and education (control, mean = 9.9 years of school, SD = 2.7; anterior, mean = 8.7 years, SD = 4.5; posterior, mean = 8.6 years, SD = 5.9;  $F_{2,39} < 1$ ). Furthermore, the two patient groups were matched for the interval between stroke and testing (anterior, mean = 38.2 days, range = 8–115 days; posterior, mean = 41 days, range = 4–103 days;  $t_{26} = -0.22$ ,  $p = 0.828$ ) and general cognitive abilities (Mini Mental State Examination; Folstein et al., 1975; anterior, mean = 27.07, SD = 2.62; posterior, mean = 26.71, SD = 3.43;  $t_{26} = 0.31$ ,  $p = 0.759$ ). Participants provided written informed consent and the procedures were approved by the local ethics committee. The study was carried out in accordance with the guidelines of the Declaration of Helsinki.

### Experimental Stimuli and Tasks

#### Study 1. Discrimination of Faces, Body, and Noncorporeal Objects

In this study, we investigated the ability of patients with anterior and posterior damage in the visual discrimination of body parts, face parts, and object parts.

Stimuli and task were modified versions of those previously used in a rTMS study in healthy individuals (Urgesi et al., 2004). We modified the color pictures by means of the Adobe Photoshop software (Adobe Systems Incorporated, San Jose, CA) to stress the morphological differences between the stimuli in each pair (Figure S1A). Sixteen pairs of stimuli were used for each category and each stimulus was presented twice, for a total of 32 stimuli for category. Before the experimental session, patients observed a printed example of stimuli and completed a six-trial practice block. The experiment consisted of two different 48-trial blocks. In each block, each stimulus set was presented separately with a block design; a Latin square balancing of the category order was used. A short rest was allowed before proceeding to a different stimulus category.

#### Study 2. Discrimination of Body Form and Body Actions

We used a modified version of the task tested in a rTMS study of healthy individuals (Urgesi et al., 2007a). The stimuli were static snapshots depicting the middle phase of specific actions performed by two models. Preliminary investigations showed that the original form discrimination task (Urgesi et al., 2007a) was too difficult for elderly healthy people. Thus, we modified the color pictures to emphasize differences in the morphology of the two models' body parts (Figure S4A). In different blocks, participants were given two delayed matching-to-sample tasks. In both tasks, participants had to decide which one of two different probe images matched a previously presented sample stimulus. In the *action discrimination task*, the matching and the nonmatching stimuli depicted two different actions executed by the same model with the same body part. In the *form discrimination task*, the matching and nonmatching stimuli differed only for the morphology of body parts and depicted the same action performed by two different models. As in the original experiment (Urgesi et al., 2007a), the same set of stimuli was used in the two tasks. The action and form discrimination tasks were presented in two 32-trial blocks; the order of task administration was counterbalanced across participants. A short rest was allowed between the two tasks. Before the experimental session, patients inspected printed examples of stimuli and completed a six-trial practice block.

### Procedure

Each patient was tested in two experimental sessions each of which lasted approximately 1 hr. The two experiments were carried out in the same or in separate sessions along with the standard neuropsychological examination. Control participants were tested in a single session lasting approximately 1 hr. Except for the stimuli, the experimental procedure was similar in the two experiments. Stimulus presentation timing and randomization were controlled by using E-prime V1.1 software (Psychology Software Tools Inc., Pittsburgh, PA) running on a PC. Participants sat 57 cm away from a 15 inch LCD monitor (resolution, 1024 × 768 pixels; refresh frequency, 60 Hz) where stimuli appeared on a white background and subtended a 10.6° × 10.6° square region around the fovea. A trial started with the presentation of a central fixation point lasting 1000 ms. A sample stimulus was presented for 1500 ms at the center of the monitor. Image persistence was limited by presenting a random-dot mask (10.6° × 10.6° in size; duration, 1000 ms) obtained by scrambling the corresponding sample stimulus by means of a custom-made image segmentation software. Immediately after the disappearance of the mask, the two probe stimuli appeared and remained on the screen until a response was made. They were presented vertically at the center of the screen and the upper or lower position of the matching stimulus was randomized. Patients were asked to indicate verbally or by pointing which of the two probe stimuli matched the sample stimulus. The trial event timelines for study 1 and study 2 are provided in Figures S1 and S4, respectively. The examiner recorded the subjects' responses, pressing one of the two mouse keys that corresponded to one of the two positions on the screen on which the probe stimuli were presented. When the patients were fixating the center of the screen, the examiner pressed a key on the keyboard to proceed to the next trial. Accuracy of responses was automatically recorded and stored for analysis.

### Behavioral Data Handling

Individual percentages of correct responses were calculated for each task of the two experiments (32 trials per cell). Analyses were performed by means of the Statistica 7 software (StatSoft, Inc., Tulsa, OK). Patients' performances

in the two experiments were analyzed by means of different mixed-model ANOVAs. All post hoc pairwise comparisons were carried out by means of the Duncan test. For between-group comparisons, across or within each level of the repeated-measure variable, the mean square error for the between-group portion of the effect was used as estimate of the error variance. For comparisons between levels of the within-subject variable, across or within each group, the mean squares error for the respective effect was used as estimate of the error variance.

### Lesion Mapping

For each patient, lesions were drawn on the T1-weighted template MRI scan from the Montreal Neurological Institute provided with the MRIcron software (Rorden and Brett, 2000; available at <http://www.microware.com/mricron>). Lesion drawing was performed by an examiner who was blinded as to the clinical features and the behavioral results. Superimposing each patient's lesion onto the standard brain allowed us to estimate the total brain lesion volume (in cc). Furthermore, the location of the lesions was identified by overlaying the lesion area onto the Automated Anatomical Labeling template provided with MRIcron. We determined the neural correlates of the impaired performance in facial and nonfacial body parts and noncorporeal objects perception (study 1) and of the double dissociation between posterior and anterior lesions and body form and body action discrimination (study 2) by using VLSM methods implemented in MRIcron and nonparametric mapping (NPM) software (Rorden et al., 2007). In study 1, we entered the individual percentages of correct responses in body, face, and object parts discrimination in three VLSM analyses to identify the voxels whose damage was associated to specific deficits. In study 2, we entered the ratio between accuracy in body form and body action discrimination ( $[\text{action discrimination accuracy}/\text{form discrimination accuracy}] * 100$ ) and its reciprocal as predictors in two VLSM analyses. The nonparametric permuted Brunner-Munzel rank-order statistic analysis for each voxel of the brain was used (Rorden et al., 2007). Colored VLSM maps were then produced that represent the z statistics of the voxelwise comparisons between lesioned and nonlesioned patients. The maps indicate the voxels at which patients with a lesion in a given voxel performed worse than patients without lesion to that voxel on specific behavioral measures concerning body-, face-, and object-parts discrimination. The alpha level of significance was set at  $p < 0.05$  and was corrected for multiple comparisons by using the false discovery rate (FDR) threshold (Nichols and Hayasaka, 2003). Only voxels that survived the above threshold were overlaid to the standard brain. Moreover, only voxels lesioned in more than three patients were tested. We used this criterion to balance between the need to increase the statistical power by testing only voxels that were injured in a significant number of individuals and to detect the effect of regions that are reliable predictors of deficits but were lesioned in just a few patients (see Rorden et al., 2007).

### SUPPLEMENTAL DATA

The Supplemental Data include five figures, one table, and supplemental text and can be found with this article online at [http://www.neuron.org/supplemental/S0896-6273\(08\)00804-0](http://www.neuron.org/supplemental/S0896-6273(08)00804-0).

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