Eur Arch Psychiatry Clin Neurosci (2009) 259:46–54 DOI 10.1007/s00406-008-0833-8

ORIGINAL PAPER

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BOLD response during visual perception of biological motion in obsessive-compulsive disorder

An fMRI study using the dynamic point-light animation paradigm

Received: 14 February 2008 / Accepted: 3 June 2008 / Published online: 27 June 2008

Abstract *Objective* Although research has shown that deficits in various cognitive functions may underlie obsessive-compulsive disorder (OCD), studies have not yet clarified the specificity and etiology of perception processing, particularly the perception of biological motion that is correlated with social cognition. We used functional magnetic resonance imaging (fMRI) to investigate neural activity associated with the perception of biological motion in OCD patients. *Methods* The subjects were 15 patients with OCD and 15 age- and IQ-matched healthy volunteers. All subjects participated in a biological motion task in which they performed a one-back task signaling a repeated stimulus with a key press in each block condition to obligate attention to both types of stimuli. *Results* The biological motion versus scrambled motion contrast revealed that both OCD patients and healthy controls exhibited increased activation of the superior and middle temporal gyrus, the regions implicated in processing of biological motion, which is consistent with previous studies. However, direct comparison between OCD subjects and healthy controls indicated that patients with OCD exhibited increased activation in the right superior and middle temporal gyrus and the left inferior temporal and fusiform gyrus, and reduced activation in the right postcentral gyrus (BA 40) compared to healthy subjects. OCD patients exhibited increased activation in the ventral visual system, including the inferior temporal and fusiform gyrus. *Discussion* We observed a differential pattern of activity between OCD patients and healthy controls, indicating that OCD patients have functional differences related to the perception of biological motion. The differential activation between OCD patients and healthy subjects might contribute to the pathophysiological understanding of obsessive compulsive disorder.

Key words biological motion · OCD · social functioning · superior temporal gyrus

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Introduction

Current approaches to obsessive-compulsive disorder (OCD) emphasize the essential role of neurobiological abnormalities in its etiology and course. Specifically, structural and functional imaging studies have consistently suggested that dysfunction of

the fronto-subcortical circuits, including the orbitofrontal cortex (OFC), anterior cingulate cortex, and basal ganglia, could explain the pathophysiology of OCD [19, 34]. Along with brain imaging studies, neuropsychological studies of OCD have revealed a variety of cognitive impairments with regard to memory, executive functioning, and high-level perceptual processing as indicated by the Rey-Osterrieth Complex Figure Test [30, 36]. Overall, OCD can be conceptualized in terms of abnormalities in a distributed set of neural structures encompassing various cognitive impairments.

Among all available environmental stimuli such as objects, sounds, and movements, primates as social animals routinely observe dynamic motion information performed by others. Primates, including humans, constantly observe the behavior of others, particularly conspecific behavior, to integrate effectively within a social environment and to avoid predators [32]. In other words, the perception of actions performed by others is at the core of human social life and may be a key feature for survival. Therefore, the human ability to perceive motions that contain socially relevant information undoubtedly guides human social interactions. Humans are remarkably adept at perceiving and interpreting motion information that provides socially relevant information effortlessly and rapidly, even when the patterns of movement are impoverished by removing contours, an ability that is termed biological motion (BM) perception [6]. Researchers have recently used point-light biological motion stimuli to investigate higher level cognitive, affective, or social processes [22], as well as to investigate basic perception and identification of BM.

A number of neuroimaging studies have reported that some areas in the superior temporal sulcus (STS) and adjacent cortices along its straight segment on the surfaces of the superior temporal gyrus (STG), middle temporal gyrus (MTG), and angular gyrus [3] are particularly involved in the representation of biological and intentional motion [20, 33]. More recently, other imaging and psychophysical studies have indicated that the motor system, consisting of mirror neuron systems, plays a role in biological motion perception [9, 10, 27]. The STG is a specific region for the integration of motion and form information from the dorsal and ventral pathways [17]. The STS is also related to the amygdala, caudate, and OFC by the fronto-subcortical circuits [2, 4]; these regions are implicated in the processing of social and emotional information in human and non-human primates [1]. Studies of the STG in OCD patients have revealed metabolic and structural abnormalities of the lateral temporal cortex, including the STS [12, 13, 37]. Previous studies have suggested that social dysfunction may be associated with functional deficits of neural circuitry, including the STG and OFC [8], and that the perception of biological motion is impaired in individuals with social dysfunction such as children with autism [5, 8] and adults with schizophrenia [26]. Kim et al. [26] found a significant correlation between biological motion perception and social functioning.

Researchers have begun to focus more on social cognition, which is the ability to conceptualize other people's thoughts, feelings, and behavior, as a new perspective on treatment outcomes of various psychiatric disorders [41]. Social functioning is the most important dimension of prognosis in the treatment of schizophrenia. A deficit in social cognition can therefore be considered an early detectable marker for the illness [23, 31]. Despite the critical role played by social cognition in daily social interaction, researchers know very little about how it relates to OCD. A recent psychophysical study [25] reported that OCD patients exhibited significantly poorer performance on biological motion tasks compared to healthy controls when subjects were permitted to perceive global form and motion; these results suggest possible functional deficits within the brain regions described above and impaired social functioning associated with this perceptual deficit.

Based on the above findings, we thought that the next step should be the clarification of abnormal brain activity related to biological motion perception in OCD patients using functional magnetic resonance imaging (fMRI) and biological and scrambled motion stimuli defined by point-lights. Based on past results, we hypothesized that differential activation between OCD patients and healthy controls while observing biological motion would contribute to the pathophysiological understanding of OCD.

Methods

Subjects

We recruited 15 patients with OCD from the OCD outpatient clinic at Seoul National University Hospital, Korea. All patients met the criteria set out by the Diagnostic and Statistical Manual of Mental Disorders-II (DSM-II) for a diagnosis of OCD, based on the Structured Clinical Interview for DSM-II (SCID). Fifteen healthy controls age- and IQ-matched to the patient group were recruited from the community. Exclusion criteria included a lifetime history of any axis I disorder and a history of any significant head injury, seizure disorder, or mental retardation. To estimate IQ, the Korean version of the Wechsler Adult Intelligence Scale (K-WAIS) with Vocabulary, Arithmetic, Block Design, Picture Arrangement, and Digit Span subscales was administered to all subjects except one patient for personal reasons.

Of the 15 patients, 7 were drug naïve and 8 were being treated using medication at the time of testing; 2 were being treated with MAOI, 3 were being treated with SSRI with anti-anxiety medication, and 3 patients were being treated with SSRI, anti-anxiety medication, and anti-psychotics. One patient had been diagnosed with co-morbid panic disorder, one with obsessive-compulsive personality disorder, and one with schizotypal personality disorder. The remaining 12 patients had not been diagnosed with any co-morbid Axis I or II disorders (SCID II) [14]. Clinical assessments were conducted using the Yale-Brown

Obsessive Compulsive Scale (Y-BOCS) for OCD symptom severity. The Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI) were used to measure the severity of depression and of anxiety, respectively. Six patients scored >16 on BDI self-reports, but no patients were diagnosed with major depressive disorder according to the SCID I. We assessed OCD patients based on five symptom dimensions [29] and classified them into the following subgroups: contamination/cleaning ($N = 6$), aggressive/checking ($N = 6$), and sexual/religious ($N = 2$). One patient could not be classified. All subjects had normal or corrected-to-normal vision and provided written informed consent after receiving a complete description of the scope of the study; healthy control subjects were paid for their participation. This study was approved by the institutional review board of the Seoul National University Hospital.

Stimuli

We used the same stimuli used by Grossman et al. [20]. Stimuli were presented in counterbalanced blocks, beginning with a rest period for MR saturation. Stimuli consisted of biological motion, scrambled point-light animations, and a resting period baseline. A total of 24 biological motion sequences were created by videotaping the appropriate placement of 12 dots on body joints and the head of an actor engaged in various activities, including running, kicking, climbing, jumping, and throwing. The segments were digitized, and the joint positions in each frame were encoded as motion vectors with initial starting positions. All visual stimuli were displayed using Matlab (Mathworks, Inc.) together with routines from the Psychophysics Toolbox (http://psychtoolbox.org/wikka.php?wakka=HomePage). All dots were white against a black background, and subtended approximately 12 arc min of visual angle. Each biological motion sequence consisted of 20 frames displayed over a 1-s period, followed by a 1.34-s blank inter-stimulus interval (ISI). At this appropriate frame rate, the biological sequences produced the perception of smooth apparent motion, depicting natural body movement.

Scrambled animations were derived from the normal biological motion sequences by reshuffling the temporal phases and spatial locations of the dots defined by the original animations. Although identical individual dot trajectories were maintained, and the overall orientation of the animation was retained, this reshuffling resulted in a perceptually unorganized and meaningless incoherent pattern of moving dots. We hypothesized that we would observe different activation patterns in the biological motion condition, for example, patterns with specific factors responsible for interpreting characteristics of biological motion such as complex coherent motion, a human body, and intentional action.

Procedure

Imaging was performed using a 1.5 Tesla scanner (Siemens AVANTO, Germany). During the scanning session, we acquired a set of whole-brain high-resolution T1 anatomical images of each participant's head (176 slices, $0.45 \times 0.45 \times 0.9$ mm), and interleaved multislice BOLD T2*-weighted gradient echoplanar imaging produced 25 contiguous 5 mm thick axial slices (FOV 21 cm; TR = 2.34 s; TE = 52 ms, FA = 90°, $3.28 \times 3.28 \times 5$ mm, no interslice gap) approximately parallel to the anterior-posterior commissure plane, covering the whole brain. Functional images were performed in two runs; each run lasted for 311 s, and a total of 264 volumes were acquired for each subject. The animations were blocked into 16.38-s phases, alternating between different stimulus conditions. Within each phase, animations were presented every 2.34 s (1 s each, 1.34-s interstimulus interval). Throughout each block, the subject performed a one-back task by pressing a button whenever the same stimulus appeared on two successive presentations, given that attention can modulate fMRI signals in visual areas [39]. Each block contained seven animations that had a 50% chance of being repeated in successive trials [20]. Although behavioral responses were not recorded during the actual scan periods, each observer practiced the task before going into the scanner. During practice trials, the task was described to each subject and examples of stimuli sequences were presented until subjects indicated that they understood by making oral responses to the examples. The animation sequences used during the practice session were identical to those displayed in the experimental conditions. Stimuli were back-projected using an LCD projector onto a translucent screen located at the subject's feet and viewed through a periscope mirror attached to the birdcage head coil.

fMRI analysis

Functional MRI data were analyzed using SPM2 (http://www.fil.ion.ucl.ac.uk/spm). We used a two-stage analysis procedure in which any contrasts reflecting activations in each subject were entered into a second-level analysis to emulate a random-effect analysis. We first assessed the main effects of differences between the biological and scrambled condition in both groups and then analyzed interactions or differences in activation. We discarded the first five volumes of each functional times series to eliminate non-equilibrium effects of magnetization. We used sinc interpolation to realign all volumes to the middle image in the first session to correct for between-scan movements and to remove signals correlated with head motion. We performed spatial normalization into a standard template provided by the Montreal Neurological Institute (MNI template) using a statistical parametric mapping echoplanar imaging template. This transformation was subsequently applied to the T2* data, and we performed downsampling to a resolution of $2 \times 2 \times 2$ mm voxel size. The normalized images were smoothed using a Gaussian kernel (full width at half maximum = 9 mm). For each subject, three regressors were created by modeling the BOLD response to each stimulus condition such as biological motion, scrambled motion, and resting baseline period as a box car with an onset cycle equal to the length of each block, convolved with a hemodynamic response function in the context of a general liner model. Low-frequency signal drifts in the MR signal were removed using a 150-s high-pass filter.

For the first-level analysis, we generated activation maps for each subject by applying t statistics in SPM2. At this step, we computed the contrast for each of the two motion conditions against the rest baseline period and the contrast of biological motion minus scrambled motion, resulting in three contrast images for each subject. We used a statistical threshold of $P < 0.001$, uncorrected for multiple comparisons. We also removed any activation clusters smaller than 60 voxels. Next, these first-level contrast images were used in second-level random effects analysis using two-sample t tests ($P < 0.001$ uncorrected, minimum clusters size of five voxels) to identify main effects in OCD patients compared with control subjects. Coordinates of the location peak of activation were converted from MNI template coordinates to Talairach coordinates using the mni2tal.m function developed by M. Brett (http://www.mre-ebu.eam.ac.uk/Imaging). The Talairach Daemon (http://ric.uthscsa.edu/projects/talairachdaemon.html) was used to identify the anatomical locations of functional clusters of activation.

To clarify whether the SPM results were caused by symptom severity, medication, depression, or anxiety level, we conducted further analyses using a region of interest (ROI) approach. We identified the brain areas that were significantly activated in the between-group analysis and defined the functional ROIs, which included the postcentral gyrus, inferior temporal gyrus, fusiform gyrus, and superior temporal gyrus. We then calculated the percentage of BOLD signal change for each subject using MarsBaR software (http://marsbar.sourceforge.net/) to extract beta values for each condition from a 5-mm sphere around the peak-activated voxel for each cluster of the functional ROIs in the normalized images. We also correlated Y-BOCS, BDI, and BAI values with brain activations in the OCD patients.

Table 1 Demographic and clinical characteristics

Variable	Control ($N = 15$) Mean (SD)	OCD ($N = 15$) Mean (SD)	Group comparisons[a]
Demographic			
Gender (male/female)	9/6	12/3	NS
Age (years)	25.67 (3.46)	23.40 (4.69)	NS
Education (years)	15.20 (1.32)	14.27 (1.86)	NS
IQ score	114.87 (10.27)	110.93 (11.80)**	NS
Clinical			
Y-BOCS	–	17.80 (7.60) 17.25 (5.57) medicated 21.57 (6.24) unmedicated	– $T = 1.417$
BDI	5.00 (4.91)	14.80 (10.45)	$T = -3.287$*
BAI	7.53 (6.09)	18.13 (10.74)	$T = -3.324$*
Age of onset (years)		17.33 (5.67)	
Duration of illness (years)		6.13 (3.76)	

Y-BOCS Yale-Brown Obsessive Compulsive Scale; *BDI* Beck Depression Inventory; *BAI* Beck Anxiety Inventory

[a]Independent sample t-test was used

*$P < 0.01$; ** IQ test was not administered to 1 patient due to personal reason

Results

Demographics and clinical and neuropsychological ratings

There were no significant differences between the patient and control groups with regard to any demographic feature, including gender, age, years of education, and IQ (Table 1). The OCD group, however, scored significantly higher on the BDI ($t(28) = -3.29$, $P = 0.003$) and the BAI ($t(28) = -3.32$, $P = 0.002$) than did the control group (Table 1).

fMRI results

To identify the cortical structures associated with the perception of biological motion, we assessed group maps to compare biological motion blocks with scrambled motion blocks. These revealed significant activation in distributed areas, including the superior and middle temporal gyrus adjacent to the STS, the inferior frontal gyrus, and the inferior occipital gyrus in both groups (Table 2). However, healthy controls also exhibited greater activation in the inferior parietal lobule, premotor cortex, OFC, MPFC, insula, and cingulate cortex (Fig. 1), all of which are cortical areas that form the dorsal visual pathway and are associated with biological motion perception. In contrast, OCD patients exhibited greater activation in the fusiform gyrus, the inferior temporal gyrus, the region implicated in the ventral visual pathway, and the middle frontal gyrus (Fig. 1). Between-group comparison indicated that patients with OCD had significantly increased activation in the right STG, right MTG, left fusiform gyrus, left inferior temporal gyrus, and right cerebellum and markedly reduced activation in the right postcentral gyrus corresponding to Brodmann area 40, compared to controls (Table 3). We subdivided the OCD group into medicated and non-medicated groups and calculated ROI values for each group. Both the medicated and non-medicated OCD groups exhibited a similar pattern of activation compared with the control group (Fig. 2). Scores of OCD symptom severity (Y-BOCS), depression level (BDI), and anxiety level (BAI) were not significantly correlated with the brain activation of the previously defined ROIs.

Discussion

To the best of our knowledge, this is the first neuroimaging study to investigate the differences between healthy controls and patients with OCD using biological motion display. We designed this study based on this approach to clarify whether OCD patients have impaired perception of motions with rich social information and thus fail to consider the goals and intentions of other beings. We found that while observing biological motion, patients with OCD exhibited abnormally increased activation in the regions of the STG, MTG, inferiotemporal cortex, fusiform gyrus, and cerebellum, along with reduced activation in the postcentral region (BA 40) compared to healthy controls (Fig. 2). All of this aberrant activation in OCD patients seems to contribute to deficient perception of biological motion (e.g., 25).

Recent neuroimaging studies have consistently identified the posterior STS spreading into the adjacent STG, the intraparietal cortex, and the lateral occipital complex [20, 21] as the regions that manage the ability to integrate form and motion, as well as the ability to define form from motion. In addition, the STS is a critical region for the processing of several kinds of biological information, including facial recognition [3]. Therefore, dysfunction of this region would cause a deficit in biological or socially relevant information processes. Our findings of increased STG activity in patients with OCD suggest that OCD patients may recruit aberrant neural networks in the perception of BM, compared to healthy controls. Consistent with our findings, previous studies have reported higher regional cerebral blood flow and partial volumetric reduction of the STG in patients with OCD [12, 13, 37]. Some researchers have also found abnormal STG activity and impaired biological motion perception in patients with autism and schizophrenia [8, 26]. Our results provide neural correlates to recent psychophysical findings of impaired BM perception in patients with OCD [25]. The

Table 2 Brain regions showing significant activation associated with biological motion versus scrambled motion contrast in patients with OCD and control subjects (all results at $P < 0.001$ uncorrected, $k = 60$ voxels)

Area of activation	Control ($n = 15$)						OCD ($n = 15$)					
	L/R	Coordinates			*Z* value	BA	L/R	Coordinates			*Z* value	BA
		X	*Y*	*Z*				*X*	*Y*	*Z*		
Superior temporal sulcus	R	55	−52	12	4.38	39	R	54	−36	11	4.79	22
	L	−56	−60	24	4.34	39	L	−55	−41	0	5.54*	22
	L	−57	−48	19	4.25	40						
Superior temporal gyrus	R	63	−29	5	4.95	22	R	50	−50	8	6.36*	22
	L	−55	−28	14	4.85	42						
	L	−40	−57	23	4.24	39						
Middle temporal gyrus	R	55	−50	1	4.85	37	R	57	−41	0	6.47*	21
							L	−50	−46	4	4.90	21
hMT/V5							R	59	−62	9	5.23*	39
Inferior occipital gyrus	R	38	−78	4	3.90	19	R	32	−95	−2	4.39	18
Inferior parietal lobule	R	53	−30	27	4.87	40						
	R	48	−69	42	4.38	39						
	L	−59	−34	22	4.08	40						
	L	−38	−62	40	3.53	39						
Premotor cortex	L	−38	10	49	4.33	6						
Medial prefrontal cortex	L	−6	45	47	3.89	8						
Orbitofrontal cortex	R	4	44	−19	3.38	11						
	L	−6	48	−16	3.78	11						
	L	−14	40	−12	3.31	10						
Insula	L	−36	−26	16	3.58	13						
	L	−44	−11	12	3.38	13						
Anterior cingulate cortex	R	12	−6	44	5.29*	24						
	R	4	−20	36	3.92	24						
Posterior cingulate cortex	L	−2	−31	37	3.95	31						
Inferior frontal gyrus (VLPFC)	L	−53	31	2	4.24	45	L	−51	30	10	3.58	46
	L	−48	24	8	4.10	45	L	−51	22	15	3.45	45
							R	55	27	−1	4.79	47
							R	53	29	6	4.25	45
Middle frontal gyrus (DLPFC)							R	46	15	34	4.24	9
							R	44	21	28	3.92	9
							R	50	27	26	3.80	46
							L	−46	19	23	4.20	46
Fusiform gyrus							L	−40	−53	−12	4.61	37
							R	42	−44	−16	4.50	37
Inferior temporal gyrus							L	−48	−32	−14	3.80	20

L left; *R* right; *BA* Brodmann's area; *VLPFC* ventrolateral prefrontal cortex; *DLPFC* dorsolateral prefrontal cortex
* $P < 0.05$ FWE corrected

inferior temporal cortex has been implicated in object recognition, visual imagery of objects, and retrieval of visual objects from memory [18, 40]. Therefore, increased activity of the inferior temporal area and the lateral temporal area in OCD patients might occur either as compensation for the deficit in the neural circuit associated with the interpretation of action by nurturing (somewhat inefficiently) additional strategies and efforts, or as a by-product of the visual analysis of information associated with the processing of moving dots.

The inferior parietal and lower postcentral cortex (BA 40) play an important role in linking sensation and action [18]. Most studies of how visual perception is related to the process from intention to action have reported activation in both regions, but we found no activation in either region in patients with OCD. This lack of activation supports previous studies that have reported decreased functional activity in the parietal cortex in subjects with OCD [24, 28].

Previous studies have suggested that a cortical network consisting of the superior temporal, inferior frontal, and inferior parietal cortices is the region of the so-called human mirror neuron system (MNS), and it has been implicated as a core neural network in understanding actions and intentions in primate brains [9, 32]. To understand the actions of others, information about the actions must be visually characterized in the STS and then relayed via the inferior parietal cortex to the inferior frontal cortex, which is implicated in the motor planning system [15]. According to this theory, humans understand actions when they are able to map a visual representation of the observed action onto the motor representation of the same action [32]. This reflexive process in understanding action automatically establishes an implicit direct link between agent and observer [16]; the visual features of an observed action are thus not sufficient to understand the action. Some recent findings have contradicted the need for a visual

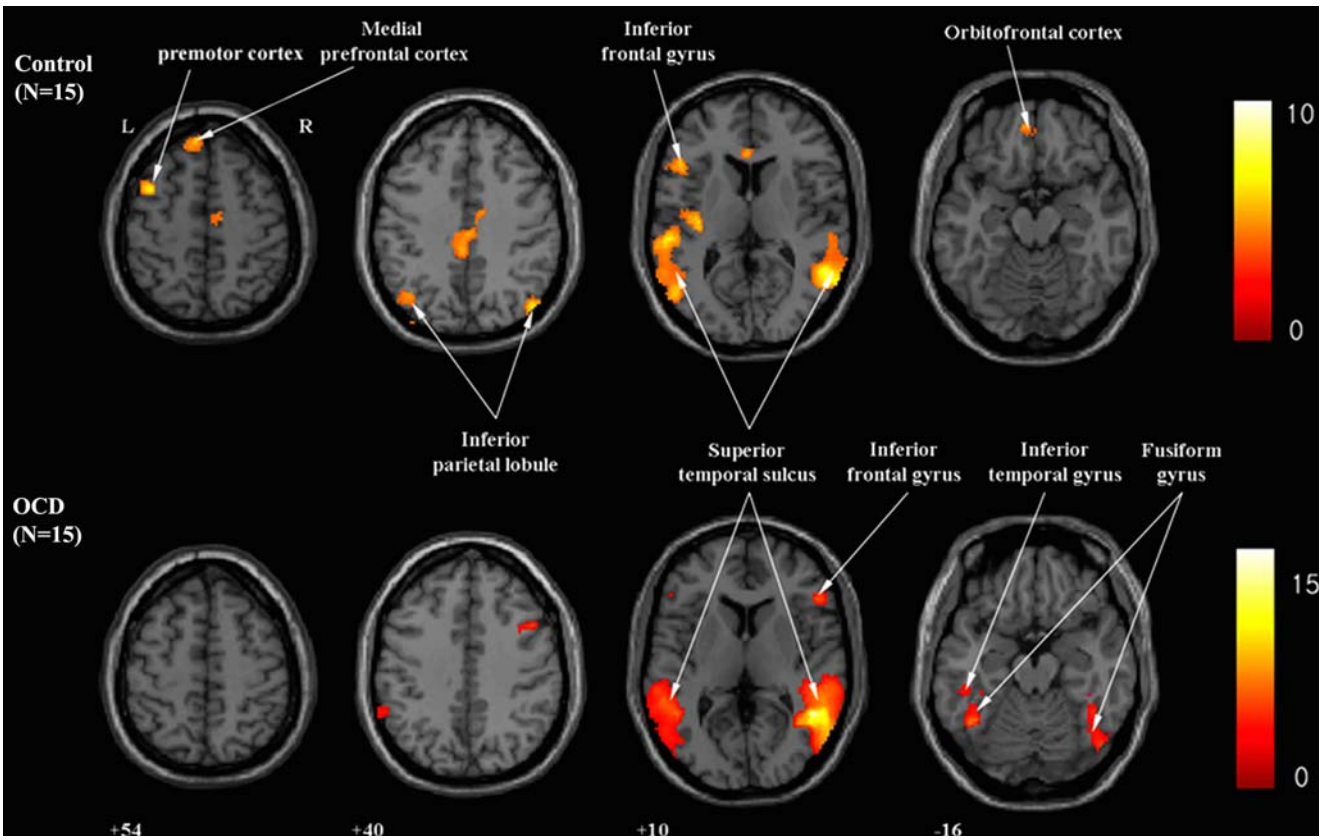

Fig. 1 Brain regions that show significant activation selective to biological motion in healthy control subjects and OCD patients. The activation is mapped on the MNI template ($P < 0.001$, uncorrected, $k > 60$ voxels). *Color bars* represent the *T*-value. Increased blood oxygenation level-dependent responses in healthy subjects were observed in the regions, which implicate in social brain areas, such as the inferior frontal cortex, the medial prefrontal cortex, the orbitofrontal cortex, the premotor cortex, the superior temporal sulcus, and the inferior parietal cortex. In OCD increased activations were detected in the superior temporal sulcus and the fusiform gyrus

Table 3 Significant increases in BOLD response associated with biological motion versus scrambled motion contrast in group comparisons (all results at $P < 0.001$ uncorrected, $k = 5$ voxels)

Comparison	Area (BA)	Coordinates			*Z* value
		X	*Y*	*Z*	
Control > OCD	R Postcentral gyrus (40)	51	−24	22	4.11
OCD > Control	R Middle temporal gyrus (21)	55	−43	−1	4.35
	R Superior temporal gyrus (22)	51	−48	8	3.18
	L Fusiform gyrus (37)	−44	−53	−11	3.74
		−42	−45	−11	3.71
	L Inferior temporal gyrus (20)	−48	−32	−14	3.56
	R Cerebellum	14	−61	−24	3.40

L left; *R* right

description of action to understand action; they have suggested that if an individual has experienced sufficient similar actions to understand and recognize the action, this would enable him or her to understand the action using MNS, even in the absence of a visual description of the action [32]. Based on these findings, recent studies have reported that not only the STS, but also the premotor and inferior frontal regions that consist of human MNS, are necessary even for the perception of point-light biological motion stimuli representing action and that these regions are causally related to deficits in biological motion perception [35]. Therefore, healthy individuals would process biological motion information without full awareness as a result of repeated experience of biological motion perception and normal development of MNS [16]. Our findings of increased activation within the premotor cortex and the inferior prefrontal and inferior parietal cortices in healthy subjects that are observing biological motion (Table 2) directly support this hypothesis. In contrast, patients with OCD exhibited no significant activation in those areas, but instead exhibited hyperactivation of the visual ventral system. The aberrant activation in the network consisting of the STG, inferior parietal cortex, and inferior frontal cortex in patients with OCD suggests that their perception action might be mediated by activity in the inferotemporal lobe, extrastriate visual areas, and STG [9] and that patients with OCD may understand actions based on a visual analysis of the various elements that form an action, rather than a reflexive process using the network consisting of the STG,

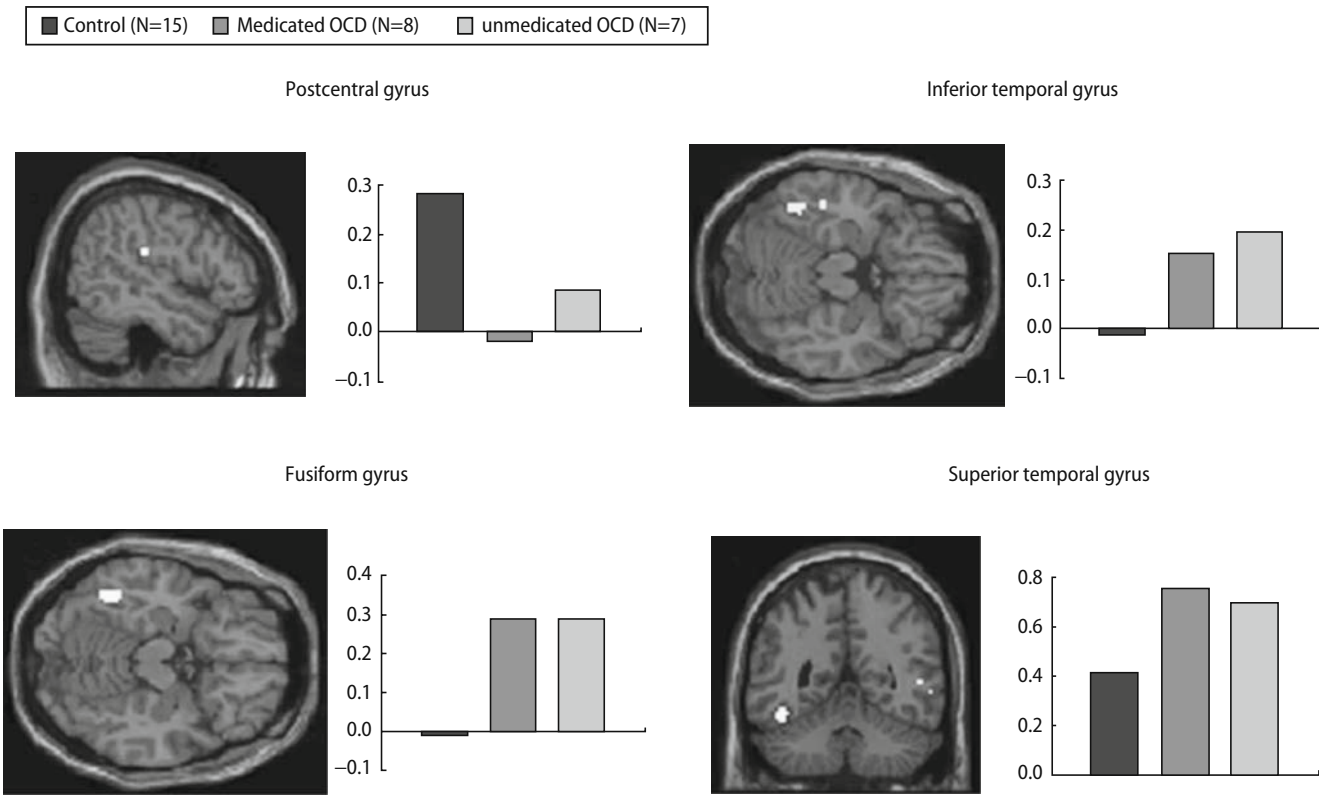

Fig. 2 Brain regions showing significant activation during viewing biological motion in group comparisons. Significantly different regions between group are mapped on the MNI template ($P < 0.001$, uncorrected, $k > 20$ voxels). *Bar graphs* depict comparison of activation in functional ROIs in control, medicated OCD patients, and unmedicated OCD patients, in the four regions that were brain areas significantly activated in between-group analysis. The *Y*-axis in the ROI graph is arbitrary unit of beta value

inferior parietal cortex, and inferior frontal cortex. Szeszko et al. [38] reported that compared to healthy subjects, patients with OCD had less white matter of the supramarginal gyri within the parietal lobes and suggested a disruption of cortical-cortical or cortical-subcortical connectivity with other brain regions.

Overall, our results suggest that the perception of biological motion is an automatic and effortless process for healthy controls, but not for OCD patients. Therefore, it is likely that patients with OCD have functional differences within the brain regions described above, which could lead to a difficulty in the automatic understanding of action because of impaired learning and representing visual information [7, 26], thereby resulting in a difficulty in biological motion perception. If efficient social functioning is associated with accurate and rapid perception of subtle socially relevant stimuli, functional differences in biological motion perception may compromise social functioning in OCD patients [25]. However, this interpretation is highly speculative because fMRI cannot show causality but only association. Therefore, additional experiments are needed to verify the association between biological motion perception and social function.

This study had several limitations. First, depression and anxiety levels were significantly higher in OCD patients than in healthy controls, which could have confounding effects. However, we examined the extent to which these levels were correlated with brain activation and found no significant correlation. Second, approximately half of the patients were taking medication at the time of scanning. Although the specific pharmacological effects of medication on biological motion perception in patients with OCD are unknown, it seems unlikely that the observed differences in activation between the two groups were caused by medication, based on previous studies of patients with schizophrenia [11, 26]. Third, we did not record behavioral responses during scanning while the subjects were engaged in a one-back task to maintain a fixed level of attention. Although behavioral data are not available, all subjects practiced the task before entering the scanner and were asked to reply orally to presented stimuli until they were sure that they understood the task.

Conclusion

Patients with OCD had functional differences compared to controls in the relay station from the STG via the parietal cortex to the inferior frontal cortex, indicating abnormalities in biological motion per-

ception. To further elucidate the pathophysiology of OCD, future research should test the correlation between biological motion perception and social function and clarify whether patients with OCD have a deficit in functional connectivity between the mirror neuron system and social brain circuitry.

Acknowledgments This research was supported by a grant (M103KV010012-08K2201-01210) from the Brain Research Center of the 21st Century Frontier Research Program funded by the Ministry of Science and Technology, Republic of Korea. The authors thank Randolph Blake, Sohee Park and Jejoong Kim for developing the task of biological motion and their comments on this article. The authors also thank Yong-Sik Jung for MR imaging technical assistance and the staff of the Clinical Cognitive Neuroscience Center for help with recruitment.

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