Functional Neuroanatomy of Body Shape Perception in Healthy and Eating-Disordered Women

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**Background:** Abnormalities in perception and evaluation of body shape are a hallmark of eating disorders.

**Methods:** Brain responses to line drawings of underweight, normal weight, and overweight female bodies were measured with functional magnetic resonance imaging in 9 women with bulimia nervosa, 13 with anorexia nervosa, and 18 healthy women. Participants rated the stimuli for fear and disgust.

**Results:** In the three groups, the lateral fusiform gyrus, inferior parietal cortex, and lateral prefrontal cortex were activated in response to body shapes compared with the control condition (drawings of houses). The responses in the lateral fusiform gyrus and in the parietal cortex were less strong in patients with eating disorders compared with healthy control subjects. Patients with eating disorders rated the body shapes in all weight categories as more aversive than did healthy women. In the group with eating disorders, the aversion ratings correlated positively with activity in the right medial apical prefrontal cortex.

**Conclusions:** Processing of female body shapes engages a distributed neural network, parts of which are underactive in women with eating disorders. The considerable variability in subjective emotional reaction to body shapes in patients with eating disorders is associated with differential activity in the prefrontal cortex.

**Key Words:** Eating disorders, anorexia nervosa, bulimia nervosa, body image, functional neuroimaging.

Western society attributes central importance to body weight and shape. Women, in particular, are under pressure to pursue a slender body ideal, which is unattainable for the vast majority (Nichter and Nichter 1991; Thompson et al 1999). Consequently, dissatisfaction with one's own body weight and shape has become a rule rather than exception (Rodin et al 1985). Although most women are concerned about their weight and shape, the “undue influence of body shape and weight on self-evaluation” is a core symptom of the eating disorders and is common to anorexia (AN) and bulimia nervosa (BN) (American Psychiatric Association 1994).

Body weight and shape dissatisfaction, and discrepancy between actual and ideal body weight, are strongest in BN (Cash and Deagle 1997). In AN, body weight dissatisfaction is often masked by actually being underweight, but it resurfaces with weight gain and complicates the recovery process (Fairburn et al 1993). Weight and shape dissatisfaction precedes and predicts the onset of disordered eating behavior (Cattarin and Thompson 1994; Killen et al 1996; Smeets and Shaw 2002), influences the outcome of cognitive-behavioral treatment (Wilson et al 1999), and is modified in the course of effective therapy (Probst et al 1999). Even after successful management of eating behavior, the persistence of weight and shape concerns is a predictor of relapse (Carter et al 2004; Fairburn et al 1993; Halmi et al 2002).

Related to concerns about weight and shape in eating disorders is “a disturbance in the way in which one’s body weight or shape is experienced” (American Psychiatric Association 1994).

This manifests as systematic overestimation of one’s own body size, and this bias is stronger in AN than BN (Cash and Deagle 1997). Disturbance in body size estimation is likely to involve perceptual and affective components (Thompson et al 1999). The weight and shape concerns can bias estimates of body size in a concern-congruent manner, with the body perceived as being heavier and larger than it is. Individuals with eating disorders might also have functional abnormalities in brain systems concerned with processing body size or body image (Grunwald et al 2001; Smeets and Kosslyn 2001).

Given the importance of body image for mental health and the interest into the neurobiological underpinnings of mental disorders, there has been a dearth of investigations into the neural correlates of normal and pathological body image. Most have focused on two brain systems: one involved in visual perception of images of the body and involving the extrastriate body area (EBA) (Downing et al 2001) and the other denoting a mental map of one’s own body—the body schema. The EBA is located in the lateral occipitotemporal cortex and responds to visual images of human bodies and body parts (Downing et al 2001). The representation of one’s own body schema depends on neural circuits involving the right parietal cortex and its connections to the thalamus (McGlynn and Schacter 1989).

In patients with eating disorders, the limited extant research has focused on neural systems involved in body schema. Smeets and Kosslyn (2001) hypothesized that distorted perception of one’s own body in AN is mediated by a failure to integrate the general body schema in the left hemisphere with the concrete examples encoded in the right hemisphere. Results of a divided visual field experiment supported this theory, with more and quicker misattributions of enlarged own body images projected to the left hemisphere in AN patients. Grunwald et al (2001) hypothesized that misperception of own body size in eating disorder patients is one aspect of a more widespread disturbance in haptic (active tactile) perception and is associated with functional disturbance in the right parietal cortex. This was supported by the finding of decreased θ power in the right parietal region on the electroencephalographs of AN patients during a haptic perception task.
Recently, functional magnetic resonance imaging (fMRI) has been used to study cerebral reactions to distorted own or other female bodies in adolescent patients with AN and age- and gender-matched healthy control subjects (Seeger et al 2002; Wagner et al 2003). Whereas a pilot study in three patients reported specific responses in the right amygdala and in the brainstem to their own versus another woman’s body (Seeger et al 2002), a group analysis of 13 patients did not replicate these findings (Wagner et al 2003). In the latter investigation, most responses to own body images were common to healthy and AN subjects and included extensive dorsolateral prefrontal, supplementary motor, insular, inferior parietal, fusiform, and cingulate cortical areas. Only the response to own distorted bodies in the right inferior parietal lobule was higher in the AN group than in the healthy control subjects (Wagner et al 2003). Compared with those of Seeger et al (2002), the results of the latter study are more credible because they are based on a formal group comparison. Also, the functional localization of the disturbance in the inferior parietal cortex corresponds to previous research on body schema (Grunwald et al 2001; McGlynn and Schacter 1989; Smeets and Kosslyn 2001).

Some aspects of the methodology make the interpretation of the results of Wagner et al (2003) problematic, however. First, own and other body images were compared with scrambled, meaningless stimuli but not to meaningful non-body images. Subsequently, the extensive neural network detected as activated in this contrast reflects not only body-image-related but also general object-recognition processes. Second, because only distorted body images were used, it is unclear whether the obtained cerebral responses relate to the content per se (own or other body) or to the fact that the images were unnaturally distorted. Third, the subjects participated in a preparatory session, when the images were taken and distorted to “maximum unacceptability.” This means that there was a previous active exposure to the target images, and differences in brain responses to own and other images might be due to priming from the preparatory session.

The present study sought to investigate the neural correlates of body image in eating disorder patients and control subjects by examining cerebral response to body pictures. It complements the investigations by Seeger et al (2002) and Wagner et al (2003), because the three methodological issues raised above were addressed. Simple line drawings of female bodies of different sizes (underweight, normal, and overweight bodies) were contrasted to meaningful objects (line drawings of houses). The drawings were designed to focus the subjects’ attention on the body size and shape and to minimize additional information. The contrast between body and house conditions sought to identify the neural substrate of body perception. Contrasts between bodies of different sizes were designed to capture the processes related to body size perception and evaluation. Subjective ratings were used to identify the neural bases of individual differences in the affective appraisal of body shapes. These ratings were carried out after scanning, so there was no prescanning exposure to the stimuli. Because the degree of body image distortion varies across the spectrum of eating disorders (Cash and Deagle 1997), the present study included groups of patients with both AN and BN.

Six specific hypotheses were tested.

Hypothesis I: across all participants, there will be category-specific responses to bodies (as contrasted to houses) in the lateral occipitotemporal cortices (EBA) (Downing et al 2001) and in the right parietal cortex (body schema system) (Grunwald et al 2001; McGlynn and Schacter 1989; Smeets and Kosslyn 2001).

Hypothesis II: the overweight bodies would be perceived as aversive by all participants and would elicit greater responses in the amygdala and insula (Murphy et al 2003; Phan et al 2002).

Hypothesis III: body stimuli would lead to differential activation in the EBA and the body scheme parietal circuitry in patients compared with control subjects, reflecting dysfunction in body image processing. This would be independent of the size of the image; therefore, we expected no interaction of stimulus body size with activity in EBA and parietal regions.

Hypothesis IV: eating disorder patients would find body shapes more aversive than control subjects and would show greater responses in the medial prefrontal cortex, insula, and amygdala (Ellison et al 1998; Seeger et al 2002; Uher et al 2004; Wagner et al 2003).

Hypothesis V: the systematic distortion of body ideal in favor of a thinner physique would be reflected by greater response in the emotion-processing regions (amygdala, insula, medial prefrontal cortex) to pictures of overweight and normal-weight bodies in patients versus control subjects.

Hypothesis VI: there would be significant correlations between subjective ratings of negative affect to the body shapes and neural activity in the brain regions underlying emotion processing, such as the amygdala and the medial prefrontal cortex.

Methods and Materials

Participants

Twenty-two patients with eating disorders were recruited from the inpatient (n = 9) and outpatient (n = 13) services of the South London and Maudsley Trust. Eighteen healthy women (CO) were recruited by advertisement and screened for abnormal eating habits (underweight, binge-eating, self-induced vomiting, purging) and neurological or psychiatric disease. Lifetime diagnosis and inclusion criteria were ascertained with the structured EATATE interview, based on the Eating Disorder Examination (Fairburn and Cooper 1993) and customized for use in genetic studies (Ribases et al 2004). Nine patients fulfilled DSM-IV criteria for BN; all of them reported bingeing and self-induced vomiting at least once per day; two of them had a history of AN. Thirteen patients fulfilled DSM-IV criteria for AN; 7 of restrictive (RAN) and 6 of binge-purge (BPAN) subtype (American Psychiatric Association 1994). Exclusion criteria for all groups were metallic implants, psychotropic medication other than antidepressants, claustrophobia, psychosis, and alcohol or drug dependence.

The mean ± SD age was 29.6 ± 9.3 years in the BN group, 25.4 ± 10.2 years in the AN group, and 26.6 ± 8.6 years in control subjects; between-group differences in age were nonsignificant [R²(2.37) = 5, p > .1]. Current body mass index (BMI) was lower in AN (16.2 ± 1.6) than in BN (22.6 ± 2.5) and control subjects (22.4 ± 3.0). One control and one AN patient were left-handed. The mean disease duration was 14.2 ± 8.6 years in BN and 11.8 ± 10.2 years in AN patients. Nine participants (five BN and four AN) were taking antidepressant medication (selective serotonin reuptake inhibitors).

After description of the study, written consent was obtained as approved by the South London and Maudsley Ethics Committee. All participants also completed experiments with food and emotional images, which are reported separately (Uher et al 2003, 2004).
Stimuli

Three sets of black-and-white line drawings of female bodies in swimming costumes were custom-created to represent underweight (BMI < 17.5), normal-weight (20 < BMI < 25), and overweight (BMI > 27.5) female bodies in similar positions. The drawings were matched to silhouette scales (Fallon and Rozin 1985) and photographs of women with known BMI to fit the selected weight categories. The control stimuli were line drawings of houses of varied sizes and styles. Examples are given in Figure 1.

Procedure

Body and house images were presented on a rear-projection screen and viewed through a double-mirror periscope fitted to the headcoil. Participants were given the verbal instruction, “You will be shown drawings of houses and bodies. Look at each of them and think how acceptable such a house/body-shape would be for you.” Each image was shown for 2.5 sec, followed by a blank screen for 5 sec. Ten body pictures in a 30-sec block (“on” condition) were followed by 10 control pictures (“off” condition). This sequence was repeated five times for each type of stimuli. There were 17 images per category, so that each image was presented three times (with the exception of one image per category being presented only twice). The order of underweight, normal-weight, and overweight bodies was counterbalanced between participants. After leaving the scanner, participants rated individual images on numeric analogue scales of 1–7 for disgust and fear, following the written instruction, “What do you feel when looking at this image?” (Scale I: “Fear”; Scale II: “Disgust”; 1 = not at all, 7 = very much).

Image Acquisition

Gradient echo echoplanar images were acquired on a 1.5-Tesla neuro-optimized MR system (GE Medical Systems, Milwaukeee, Wisconsin). Fifty T2*-weighted whole-brain volumes were acquired in each condition (repetition time 3 sec, echo time 40 msec) in 16 near-axial 7-mm-thick slices (7-mm interslice gap) depicting blood oxygen level–dependent (BOLD) contrast with an in-plane resolution of 3 mm.

Data Analysis

After motion correction (Bullmore et al 1999a), the BOLD effect was modeled by two Poisson functions with hemodynamic delays of 4 sec and 8 sec. The least-squares model of the weighted sum of these two functions was compared with the signal in each voxel to obtain a goodness-of-fit statistic. The distribution of this statistic under the null hypothesis was calculated by wavelet-based resampling of the time series and refitting the models to the resampled data. Generic group activation maps, depicting regions where the BOLD signal is significantly stronger in response to the active images (body) than to the control images (houses), were constructed by mapping the observed and randomized test statistics into standard space and computing median activation maps. Main effects of group (BN, AN, control subjects) and stimulus (underweight, normal-weight, overweight) and interaction between the two factors were established by cluster-level analysis with data randomization between groups to determine the sampling distribution of group differences under the null hypothesis. Similarly, correlations of subjective ratings of images with voxel- and cluster-wise values of the goodness of fit were obtained by calculating the Pearson product–moment correlation at each voxel. The probability of occurrence of any cluster in the observed data was computed by reference to the null distribution (Bullmore et al 1999b). In group analyses, all activations above the cluster-wise significance threshold of \( p < .01 \) are reported. This provides a reasonable balance between type I and type II errors (overall probability of the occurrence of a false-positive cluster is \(<1.0\) but \(>\)0.5); however, a cluster-wise threshold of \( p \leq .001 \) is required for whole-brain correction (overall probability of occurrence of any false-positive cluster is \(<.05\). Subjective ratings were averaged for each category of stimuli and then analyzed with analysis of variance with planned orthogonal contrasts (Contrast I: all patients vs. control subjects; Contrast II: BN vs. AN).

Results

Subjective Ratings of Stimuli

The ratings of fear and disgust were highly correlated for all categories of stimuli (Pearson’s \( r = .71 \) \(< r < .94 \)), and hence an average of these two is reported as a measure of “aversiveness” (Figure 1). Images of bodies were rated as more aversive by patients than by control subjects \([F(2,117) = 25.9, p < .001;\] Contrast I: \( t(1,117) = 6.6, p < .001 \)]. This was true for all three categories of body shapes [underweight: \( t(1,37) = 3.1, p < .01 \); normal: \( t(1,37) = 4.1, p < .001 \); overweight: \( t(1,37) = 6.7, p < .001 \)]. This effect was more marked in AN than in BN \([F(2,117) = 2.8, p < .05 \]). Furthermore, there was significant group \( \times \) stimulus interaction \([F(4,111) = 3.1, p < .05 \]): whereas healthy participants rated the underweight bodies as most aversive, the eating-disordered patients reported more aversion to the overweight bodies. The AN patients reported more aversion to the normal-weight bodies, compared with both the BN and control participants.

As can be seen from the error bars and scatter plots (Figure 1), there was considerable variability in subjective ratings within the patient groups. Some patients scored in the range of healthy control subjects, whereas others gave much more negative ratings. Specifically, we have identified a group of five subjects (one BN, four AN) who rated images of normal bodies as more aversive than images of underweight bodies, reflecting a severe disturbance in body image. All control subjects rated the under-
To capture the relative preference for an underweight body ideal, an index of body-image disturbance was calculated as 
\[
\frac{\text{aversion to normal-weight bodies} - \text{aversion to thin bodies}}{\text{aversion to normal-weight bodies} + \text{aversion to thin bodies}}
\] for correlational analyses involving the imaging data.

There were no between-groups differences in the ratings of the images of houses.

**Generic Group Activation Maps**

Details of group activation maps are given in Table 1 and Figure 2. In response to body shapes (compared with houses),

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**Table 1. Group Activation Maps in Response to Body Shapes**

<table>
<thead>
<tr>
<th>Brodmann's Area</th>
<th>L Laterality</th>
<th>Size (Voxels)</th>
<th>Talairach's coordinates</th>
<th>Effect Size</th>
<th>Significance Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>19, 37 Right</td>
<td>287</td>
<td>-43 -63 -13</td>
<td>.59</td>
<td>.0003</td>
</tr>
<tr>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>19, 37 Left</td>
<td>231</td>
<td>-43 -67 -8</td>
<td>.37</td>
<td>.0003</td>
</tr>
<tr>
<td>Parietal</td>
<td>40, 7 Right</td>
<td>244</td>
<td>29 -63 48</td>
<td>.41</td>
<td>.0003</td>
</tr>
<tr>
<td>Parietal (inferior parietal lobule), occipital</td>
<td>40, 19 Left</td>
<td>84</td>
<td>-29 -63 42</td>
<td>.18</td>
<td>.0005</td>
</tr>
<tr>
<td>Lateral prefrontal</td>
<td>44, 9, 46, 47 Right</td>
<td>192</td>
<td>51 15 26</td>
<td>.27</td>
<td>.0003</td>
</tr>
<tr>
<td>Lateral prefrontal/prefrontal</td>
<td>6, 44 Left</td>
<td>35</td>
<td>-47 4 31</td>
<td>.16</td>
<td>.0034</td>
</tr>
<tr>
<td>Healthy Women: Underweight Body Shapes</td>
<td>Occipitotemporal (medial frontal gyrus)</td>
<td>10, 47 Left</td>
<td>10</td>
<td>-36 52 -7</td>
<td>.10</td>
</tr>
<tr>
<td>Healthy Women: Overweight Body Shapes</td>
<td>Anterior cingulate gyrus</td>
<td>32 Right</td>
<td>28</td>
<td>7 15 42</td>
<td>.12</td>
</tr>
<tr>
<td>Eating Disorders: Normal Body Shapes</td>
<td>Putamen</td>
<td>Right</td>
<td>37</td>
<td>11 4 9</td>
<td>.11</td>
</tr>
<tr>
<td>Thalamus (dorsomedial)</td>
<td>Right</td>
<td>22</td>
<td>7 -15 4</td>
<td>.09</td>
<td>.0084</td>
</tr>
</tbody>
</table>

First, activations are given, which are common to all three conditions (underweight, normal, overweight body shapes) and all groups (healthy women, anorexia nervosa, bulimia nervosa); then, other activations are listed under the specific combination of condition and group. All activations that have been positively associated with the presentation of body shapes at a cluster-wise significance threshold of $p \leq .01$ are included. The “Size” of each cluster is given as number of voxels (each voxel is 60 mm$^3$). The “Location” is specified as Talairach’s x, y, and z coordinates of the center of mass for each cluster (Talairach and Tournoux 1988). The strength of activation is given as “Effect Size”, and the “Probability” of occurrence of an activated cluster by chance in the specific activation map is given as a $p$ value.

**Figure 2.** Generic group activation maps for body shape images. The depicted activations were associated with the active stimuli at the clusterwise level of significance, $p \leq .01$. Four representative axial slices are shown in radiological convention (right hemisphere is on the left-hand side of the image). The vertical position of each slice is determined by the z coordinate, which is given at the bottom of the figure.
both patients and healthy women activated the lateral fusiform gyrus (corresponds to EBA, Brodmann’s areas [BA] 19 and 37), the inferior parietal cortex (maximum response in BA 40), and the lateral prefrontal cortex (maximum response in the inferior frontal gyrus, BA 44). For all three regions, the body-shape-related activations were stronger and/or more extensive in the right hemisphere (Table 1). The medial aspects of the fusiform and lingual gyri were consistently more active in the control condition with images of houses (Figure 2).

Other activations reached the cluster-wise \( p < .01 \) thresholds only in some of the conditions. There were no brain regions that showed greater activation across all participants to overweight, relative to normal-weight or underweight bodies; however, in the group of healthy women, the right dorsal anterior cingulate cortex (BA 32) was activated in response to overweight bodies, and an area in the anterior ventrolateral prefrontal cortex (medial frontal gyrus, BA 47 and 10) was responsive to the underweight body-shapes. In the eating-disordered patients, there were activations in subcortical structures (thalamus and putamen) in response to normal- and overweight but not underweight body shapes.

**Group Comparisons**

Patients with eating disorders, as a group, showed a weaker reaction to body shapes in the occipitotemporal cortex (including the EBA) and in the parietal cortex than did healthy women (Table 2). These effects were more marked in the AN patients, who showed weaker activation, relative to both BN patients and control subjects, in the right and left lateral fusiform gyrus and in the right parietal cortex in response to all three categories of body shapes. Patients with BN showed activation in the right lateral fusiform gyrus intermediate between the healthy women and those with AN but did not differ from control subjects in terms of parietal activation. There were no regions of significantly increased activations in any patient group compared with the healthy control subjects. There was an interaction between group (patients vs. control subjects) and condition (overweight vs. underweight body shapes) in the right fusiform gyrus (\( p < .01 \)), which was due to a relatively stronger response in healthy control subjects to overweight body shapes and in the patients to underweight body shapes.

**Correlation of Brain Activity with Subjective Ratings**

Because the variability in the ratings of body shapes within the control group was relatively small, the correlation analysis focused on patients with eating disorders. Composite activity maps to all three categories of body shapes were used in the correlations. Reactivity to body shapes in the right apical medial prefrontal region (superior frontal gyrus, BA 10; \( x = 14, y = 60, z = 4 \)) correlated positively with the mean aversion rating of body shapes (\( r = .54, p < .01 \)). On the other hand, reactivity in the left lateral fusiform gyrus (BA 19 and 37; \( x = -47, y = -67, z = -7 \)) tended to be lower in those who reported greater subjective aversion to body shapes (\( r = -.45, p < .05 \)). The index of body image disturbance (aversion in response to normal relative to underweight bodies) was positively correlated (\( r = .51, p < .01 \)) with reactivity in the inferior medial temporal region (including BA 34 and the amygdala; \( x = 14, y = 0, z = -18 \)). This latter correlation was largely due to three patients with severe body image disturbance and strong reactivity to body shapes in this region (Figure 3).

**Discussion**

Previous research has indicated that the processing of body image is underpinned by neural systems involved in analyzing perceptual aspects of the human body (EBA; Downing et al 2001) and in representing a person’s own body schema (right parietal cortex; e.g., McGlynn and Schacter 1989). In the present study, the pattern of brain response to female body shapes is consistent across different categories of stimuli (underweight, normal, overweight) and across groups of participating women (with or without an eating disorder) and includes bilateral activations in the lateral fusiform gyrus, inferior parietal cortex, and dorsolateral prefrontal cortex, supporting our Hypothesis I. These findings are consistent with those of Wagner et al (2003), who found activity in these three regions in response to distorted body images in both healthy women and women with anorexia nervosa. The present body-selective activation in the lateral occipitotemporal cortex subsumes the previously identified EBA (Downing et al 2001) but is larger and also includes more ventral areas previously identified as responsive to faces or animals more than to inanimate objects (Chao et al 1999; Ishai et al 1999).

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**Table 2. Group Comparisons of Activation Maps in Response to Body Shapes**

<table>
<thead>
<tr>
<th>Condition Comparison</th>
<th>Brodmann’s Area</th>
<th>Laterality</th>
<th>Size</th>
<th>Talairach’s Coordinates</th>
<th>Significance Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating Disorders &lt; Healthy Women</td>
<td>Parietal (inferior lobule)</td>
<td>Right</td>
<td>53</td>
<td>x = 33, y = -56, z = 48</td>
<td>.0006</td>
</tr>
<tr>
<td></td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Right</td>
<td>107</td>
<td>x = 51, y = -59, z = -8</td>
<td>.007</td>
</tr>
<tr>
<td></td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Left</td>
<td>36</td>
<td>x = -36, y = -70, z = -13</td>
<td>.008</td>
</tr>
<tr>
<td></td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Right</td>
<td>67</td>
<td>x = 29, y = -67, z = 31</td>
<td>.006</td>
</tr>
<tr>
<td>Anorexia Nervosa &lt; Healthy Women</td>
<td>Parietal (superior parietal lobule)</td>
<td>Right</td>
<td>120</td>
<td>x = 40, y = -60, z = -13</td>
<td>.0002</td>
</tr>
<tr>
<td></td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Left</td>
<td>33</td>
<td>x = -40, y = -67, z = -2</td>
<td>.008</td>
</tr>
<tr>
<td>Bulimia Nervosa &lt; Healthy Women</td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Right</td>
<td>78</td>
<td>x = 51, y = -59, z = -8</td>
<td>.002</td>
</tr>
<tr>
<td>Anorexia Nervosa &lt; Bulimia Nervosa</td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Right</td>
<td>92</td>
<td>x = 51, y = 7, z = 26</td>
<td>.006</td>
</tr>
<tr>
<td></td>
<td>Parietal (superior parietal lobule)</td>
<td>Right</td>
<td>132</td>
<td>x = 29, y = -70, z = 37</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Occipitotemporal (lateral fusiform gyrus)</td>
<td>Right</td>
<td>95</td>
<td>x = 51, y = -59, z = -13</td>
<td>.0002</td>
</tr>
</tbody>
</table>

All between-groups differences in brain reactions to body shapes at a cluster-wise significance threshold of \( p \leq .01 \) are included. The “Size” of each cluster is given as number of voxels (each voxel is 60 mm3). The “Location” is specified as Talairach’s x, y, and z coordinates of the center of mass for each cluster. The strength of difference is indicated as the “Probability” of occurrence of an activated cluster by chance in the specific comparison (\( p \) value).
It has been suggested that the characteristic disturbances in body image found in patients with eating disorders (e.g., Cash and Deagle 1997) might reflect functional abnormalities in these regions (Grunwald et al 2001; Smeets and Kosslyn 2001). In the present study, there was less activity in the occipitotemporal and parietal regions in eating-disordered patients compared with control subjects, with this relative inactivity being more marked in patients with AN. This lower responsiveness in the present patient group is unlikely to be due to nonspecific decreases in brain reactivity, because these same patients demonstrated increased or equivalent levels of activation relative to control subjects in response to food-related or nonspecific emotional stimuli (Uher et al 2004). The present findings provide support for Hypothesis III, that body-image-processing brain circuits are dysfunctional in patients with eating disorders. The low functionality of this network might facilitate the development of body image disturbance in some individuals and might be related to deficits in spatial and tactile perception (Grunwald et al 2001; Tchanturia et al 2002). Further research is needed to address the issue of whether this abnormality is a consequence or an antecedent of the eating disorder.

It is important to note that the present findings stand in contrast to the increased reactivity in the inferior parietal lobule in response to own distorted body images in adolescent patients with AN reported by Wagner et al (2003); however, direct comparison across studies is difficult because no negative differences (areas less activated in the AN group) are reported in the latter article. Furthermore, the relative increase in activity in the study by Wagner et al was specific to distorted pictures of one’s own body and not present to pictures of other bodies. This suggests that this finding reflects the self-relatedness of stimuli or previous exposure, rather than body processing per se.

We anticipated that functional abnormalities in the neural circuits underlying body image processing in patients with eating disorders would be independent of body image stimulus size; however, we found an interaction of body image size with group in the lateral fusiform gyrus. Specifically, relative underactivity in this region in patients, compared with control subjects, was evident to images of overweight bodies, but this pattern was reversed to images of underweight bodies. In addition, in the patient group, overall subjective ratings of the aversiveness of the body shapes correlated negatively with activity in the fusiform gyrus, with lower activations being associated with greater subjective aversion. This suggests that dysfunction in the EBA in eating-disordered patients is related to the subjectively aversive nature of the stimuli; however, because these findings were weak and unpredicted, further research is clearly warranted.

As expected, patients rated body images as more aversive than control subjects. Furthermore, AN patients were more averse to normal-weight bodies than BN patients and control subjects; however, these trends in subjective ratings were generally not reflected in the fMRI data. We found no support for Hypothesis II that overweight body shapes would lead to greater activation in brain regions implicated in emotion processing. There was also no evidence supporting Hypothesis IV, because reactivity in emotion-processing regions of the brain did not differentiate between eating disorder patients and control subjects. Neither were there any reliable interactions between body image stimulus size and activation in these regions either within or between groups (Hypothesis V not supported). In the group of patients with eating disorders, we did find activation in the basal ganglia and the thalamus in response to normal- and overweight bodies. The differences from normal-weight condition were not significant, however, and did not generalize to the control group. In the light of these data, it is notable that, although the previous study by Wagner et al (2003) found responses in some emotion-processing brain regions (anterior cingulate, insula cortex), that study also did not find any group difference in these regions between AN patients and control subjects.
This lack of emotion system activation in response to body images in the present data, and in particular the lack of group differences in the present data and in the previous study (Wagner et al 2003), might be due to a number of factors. For example, the relatively low salience of body shape stimuli for healthy women combined with high variability in subjective perception of different body shapes in patients might have contributed. In addition, metacognitive processes might have confounded the findings; it is possible that women are implicitly performing a self-comparison with the presented body shapes (Stice et al 2003; Tiggemann and Slater 2004). In that case, the aversiveness of an overweight body shape would be balanced by the relatively positive self-comparison, and vice versa. Hence, future investigations of neural correlates of body image–related processes might profit from more specific paradigms, which would include explicit judgment or comparison of body shape and weight.

It is also possible that the presentation of body shapes as line drawings is not sufficiently evocative; however, line drawings of body shapes proved to be effective in provoking subjective aversion in the present study, and silhouette tests with similar drawings are widely used for evaluation of body image disturbance (Bulik et al 2001; Fallon and Rozin 1985). Nonetheless, more naturalistic stimuli, such as color photographs, might be more effective in a symptom-provocation paradigm. For example, slender models presented in visual media have been shown to provoke negative feelings and impair the body image in susceptible young women (Groesz et al 2002); application of such stimuli is warranted in future neuroimaging research on eating disorders.

Given the possibility that line drawings might not have been particularly evocative, it might be that differential reactivity to the current body shape images in emotion-processing brain regions was only present in patients who found these stimuli strongly aversive. In the present group of patients with eating disorders, there was indeed large between-subject variability in the subjective ratings of the drawings. Whereas many patients rated the images similarly to healthy control subjects, others gave much more negative ratings. In support of Hypothesis VI, correlation analyses demonstrated that the mean aversion ratings in the patient group were associated positively with activity in the apical medial prefrontal cortex. Furthermore, the index of body image disturbance (coding a relative preference for underweight body shapes) correlated positively with a cluster of activity in the inferior medial temporal lobe, including the amygdala.

This medial prefrontal cortex activation is comparable to reactions to subjectively aversive food stimuli (Uher et al 2003, 2004). It seems that whereas the reactivity in this region to food is present in most eating disordered individuals, the reactivity to body shape stimuli is restricted to a smaller subgroup, for whom such stimuli are subjectively strongly disturbing. Although these two clusters overlap in the same anatomically defined area (Brodmann’s area 10), however, the activation correlated with body shape ratings is located anterior to the food-responsive cluster. Hence, further investigation will be needed to explore the role of the medial prefrontal cortices in the genesis of symptoms in eating disorders.

In terms of the medial temporal and amygdala activation, on closer inspection it was apparent that this correlation was largely due to the three patients who showed the highest levels of body image disturbance and strongest reactivity to body shapes in this region. It should be noted that this was a post hoc analysis intended to explain the within-group variability in the present data. The data must therefore be interpreted with caution. This same region was previously found to be reactive to derogatory body-related words in young women (Shirato et al 2003), to distorted images of own bodies (Seeger et al 2002), and to high-caloric drinks (Ellison et al 1998) in patients with AN; other studies did not find such associations (Uher et al 2004; Wagner et al 2003). The present data on relatively strong amygdala reactivity in a small subgroup of patients might explain why findings concerning activity in this region were inconsistent in previous reports.

Although the present study is the largest in functional neuroimaging research on eating disorders to date, the nature of our sample limits the generalizability of our findings. Most of the patients included have had a chronic eating disorder and had been ill for many years. It is likely that the role of body image disturbance varies over the course of the illness, and factors determining the onset of disorder might no longer be salient at the chronic stage. The factor of illness duration might explain some differences between studies because the study by Wagner et al (2003) was based on a substantially younger and less chronic sample. Finally, although current and lifetime diagnoses of eating disorder have been carefully established, the assessment of comorbidity relied on several screening questions, and a comprehensive diagnostic interview covering all psychiatric disorders was not used. The issue of comorbid disorders needs to be formally addressed in future investigations.

In conclusion, in the present study we found a highly consistent pattern of brain activity related to body shape processing in healthy women and in women with eating disorders. Patients with eating disorders exhibited a relative underactivity of these networks, and this might underlie the failure to represent and evaluate one’s own body in a realistic way.

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