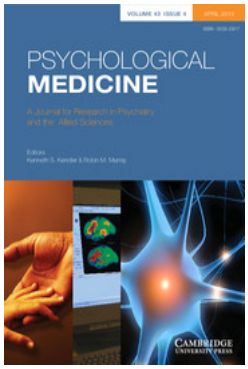


# Brain connectivity in body dysmorphic disorder compared with controls: a diffusion tensor imaging study

B. G. Buchanan, S. L. Rossell, J. J. Maller, W. L. Toh, S. Brennan and D. J. Castle

Psychological Medicine / *FirstView* Article / March 2013, pp 1 - 9
DOI: 10.1017/S0033291713000421, Published online: 11 March 2013

*Psychological Medicine*, Page 1 of 9. © Cambridge University Press 2013
doi:10.1017/S0033291713000421

ORIGINAL ARTICLE

# Brain connectivity in body dysmorphic disorder compared with controls: a diffusion tensor imaging study

**B. G. Buchanan[1]*, S. L. Rossell[1,2,3], J. J. Maller[1], W. L. Toh[1,3], S. Brennan[1,2] and D. J. Castle[3,4]**

[1] *Monash Alfred Psychiatry Research Centre, School of Psychology and Psychiatry, Faculty of Medicine, Nursing and Health Sciences, Monash University and The Alfred Hospital, Melbourne, Australia*
[2] *Brain and Psychological Sciences Research Centre (BPsyC), Faculty of Life and Social Sciences, Swinburne University of Technology, Melbourne, Australia*
[3] *Departments of Psychiatry and Behavioural Science, The University of Melbourne, Melbourne, Australia*
[4] *Psychiatry, St Vincent's Hospital, Melbourne Australia*

**Background.** Several neuroimaging studies have investigated brain grey matter in people with body dysmorphic disorder (BDD), showing possible abnormalities in the limbic system, orbitofrontal cortex, caudate nuclei and temporal lobes. This study takes these findings forward by investigating white matter properties in BDD compared with controls using diffusion tensor imaging. It was hypothesized that the BDD sample would have widespread significantly reduced white matter connectivity as characterized by fractional anisotropy (FA).

**Method.** A total of 20 participants with BDD and 20 healthy controls matched on age, gender and handedness underwent diffusion tensor imaging. FA, a measure of water diffusion within a voxel, was compared between groups on a voxel-by-voxel basis across the brain using tract-based spatial statistics within the FSL package.

**Results.** Results showed that, compared with healthy controls, BDD patients demonstrated significantly lower FA ($p<0.05$) in most major white matter tracts throughout the brain, including in the superior longitudinal fasciculus, inferior fronto-occipital fasciculus and corpus callosum. Lower FA levels could be accounted for by increased radial diffusivity as characterized by eigenvalues 2 and 3. No area of higher FA was found in BDD.

**Conclusions.** This study provided the first evidence of compromised white matter integrity within BDD patients. This suggests that there are inefficient connections between different brain areas, which may explain the cognitive and emotion regulation deficits within BDD patients.

*Received 20 April 2012; Revised 3 February 2013; Accepted 12 February 2013*

**Key words:** Body dysmorphic disorder, diffusion tensor imaging, neuroimaging, obsessive–compulsive spectrum, white matter.

## Introduction

Body dysmorphic disorder (BDD) is a mental disorder characterized by a preoccupation with an imagined defect in physical appearance, or if a slight abnormality is present, the concern about it is excessive. Individuals with BDD often engage in repetitive and ritualistic behaviours, including skin picking, camouflaging their supposed defect, and checking their appearance in the mirror. The prevalence of BDD is approximately 1.8% in community self-report samples (Buhlmann *et al.* 2010), with slightly higher prevalence among females. Individuals with BDD have impaired psychosocial functioning and quality of life (Phillips *et al.* 2005*b*), they may experience prolonged unemployment, severe social isolation and suicidal ideation, with approximately 25% of individuals with BDD attempting suicide (Phillips *et al.* 2005*a*; Buhlmann *et al.* 2010). Many individuals with primary BDD also fulfil the criteria for other mental disorders, including social phobia (Coles *et al.* 2006), major depression (Phillips *et al.* 2007*a*) and obsessive–compulsive disorder (OCD) (Stewart *et al.* 2008). The high level of co-morbidity may represent conceptual similarities between BDD and other disorders or aetiological links.

The severe body image distortions that individuals with BDD experience suggest that fundamental

* Address for correspondence: Mr B. G. Buchanan, School of Psychology and Psychiatry, General Office, Room 405, Building 17, Clayton Campus, Monash University, Victoria 3800, Australia.
(Email: Buchanan.ben@gmail.com)

cognitive and perceptual abnormalities are involved. Indeed, neuropsychological research has revealed a range of deficits in BDD, including in executive function (Hanes, 1998; Dunai *et al.* 2009; Labuschagne *et al.* 2011), selective attention (Buhlmann *et al.* 2002), information processing, verbal and non-verbal memory (Deckersbach *et al.* 2000; Dunai *et al.* 2009), recognition of emotion in others (Buhlmann *et al.* 2004) and visual processing (Feusner *et al.* 2007, 2010*a*). The combination of deficits in response inhibition, combined with heightened attention to threatening stimuli compared with controls, has suggested that frontal–striatal circuits may be important in BDD (Buchanan *et al.* 2011).

Several neuroimaging studies have directly investigated structural and functional brain abnormalities in BDD. To date, these investigations have focused primarily on grey matter structures. The right amygdala has been shown to have increased activation in response to visual stimuli in a functional magnetic resonance imaging (fMRI) study (Feusner *et al.* 2007), while structural MRI techniques have shown that right amygdala volume correlates to symptom severity (Rauch *et al.* 2003; Feusner *et al.* 2009). Other abnormalities in areas related to emotion processing, such as the cingulate cortex (Atmaca *et al.* 2010), suggest that there are deficits in the limbic system. The orbitofrontal cortex has been shown to be reduced in volume compared with controls using structural MRI (Atmaca *et al.* 2010), and an fMRI study found hyperactive activation in the same area (Feusner *et al.* 2010*b*). Furthermore, neuropsychological (Dunai *et al.* 2009) research has shown deficits in cognitive functioning that may be related to the orbitofrontal cortex. Consistent with BDD symptomatology centred around visual stimuli, occipital lobe activation has been found to be different from that of controls (Feusner *et al.* 2010*b*). Less consistent abnormalities have been shown in the caudate nucleus (Atmaca *et al.* 2010), and temporal lobes (Carey *et al.* 2004), which could possibly be associated with memory dysfunction found in BDD samples (Deckersbach *et al.* 2000).

BDD and OCD are often compared as they share symptomatology (McKay *et al.* 1997; Stewart *et al.* 2008). There is a high co-morbidity rate (Stewart *et al.* 2008) between these disorders, they have comparable demographic characteristics (Frare *et al.* 2004), and have a genetic overlap (Monzani *et al.* 2012). In terms of neurobiology, abnormalities of the caudate nucleus (Rauch, 2000; Whiteside *et al.* 2004) and orbitofrontal cortices (Atmaca *et al.* 2010; Rotge *et al.* 2010) are common to both disorders. However, neuroimaging data are well replicated in OCD but scarce in BDD, and no study has directly compared the two disorders using neuroimaging techniques.

Diffusion tensor imaging (DTI) is a neuroimaging technique developed to examine the integrity of white matter tracts. Recent methodological advances in DTI have motivated a growing interest in disconnection models proposing that white matter structural connectivity modulates symptoms in various mental disorders. In OCD samples there are at least nine separate studies that have reported DTI data, whereas this technique has not yet been reported on in a BDD sample. Within OCD samples DTI has found abnormalities in the fronto-striatal neural pathway, the corpus callosum, superior longitudinal fasciculus as well as a generalized disorganization among neural tracts (Garibotto *et al.* 2010; Bora *et al.* 2011).

Within DTI analysis, fractional anisotropy (FA) is the most widely used measurement, and represents the normalized standard deviation of three tensor eigenvalues. In white matter the movements of water molecules are restricted by various tissue components (e.g. myelin sheath or membranes), so that diffusion is reported to be anisotropic (Basser *et al.* 1994; Basser & Pierpaoli, 1996). FA reflects the degree of directionality (anisotropy) within a voxel. High FA suggests that there is highly directional diffusion such as that seen in white matter fibre tracts, and low FA values are associated disorganized white matter (Mori *et al.* 2005). This technique, therefore, can allow investigators to examine the neural organization of white matter, which reflects the efficiency of how different parts of the brain communicate with each other.

This study has two main aims: (1) to investigate whether white matter abnormalities exist within a BDD cohort by comparing them with healthy controls; and (2) to characterize the biological abnormalities underlying the disorder, using FA. Given that OCD shares nosological links with BDD, our hypotheses were partly based on the reported OCD white matter abnormalities (although it is acknowledged that there is some inconsistency in the OCD literature). In addition the neuropsychological deficits and grey matter differences reported within BDD samples were used to inform our hypothesis that white matter integrity may be compromised in BDD. Specifically, it was hypothesized that the BDD sample would have widespread significantly reduced FA, including in the corpus callosum, superior longitudinal fasciculus, and inferior fronto-occipital fasciculus. Past neuroimaging studies (Feusner *et al.* 2009, 2010*b*) have suggested a relationship between structural and functional abnormalities and BDD symptom severity. Thus, the relationship between symptoms severity and FA was also investigated in this study. In addition, FA was correlated with current anxiety and depression scores to help investigate the influence of these co-occurring symptoms on white matter integrity.

**Table 1.** *Demographic and clinical variables*

| | BDD | Controls | Group comparison[a] |
|---|---|---|---|
| Demographic characteristics | | | |
| Mean age, years (S.D.) | 34.6 (11.5) | 31.9 (11.4) | $p=0.45$ |
| Mean length of education, years (S.D.) | 14.9 (2.4) | 16.3 (3.0) | $p=0.11$ |
| Mean WTAR IQ estimate (S.D.) | 106 (10.7) | 110 (6.5) | $p=0.13$ |
| Handedness, *n* | | | |
| Left | 3 | 3 | |
| Right | 17 | 17 | |
| Gender, *n* | | | |
| Male | 6 | 6 | |
| Female | 14 | 14 | |
| Clinical variables | | | |
| Mean BDD severity, BDD-YBOCS (S.D.) | 24.9 (9.6) | – | |
| Mean duration of illness, years (S.D.) | 10.8 (6.9) | – | |
| Mean depression, Zung (S.D.) | 46.0 (11.3) | 23.3 (8.7) | $p<0.05$ |
| Mean social anxiety, SIAS (S.D.) | 41.7 (18.0) | 17.1 (4.2) | $p<0.05$ |

BDD, Body dysmorphic disorder; S.D., standard deviation; WTAR, Wechsler Test of Adult Reading; IQ, intelligence quotient; BDD-YBOCS, Yale–Brown Obsessive Compulsive Scale Modified for Body Dysmorphic Disorder; Zung, Zung Self-rating Depression Scale; SIAS, Social Interaction Anxiety Scale.

[a] Degrees of freedom=38.

## Method

### *Participants*

Two cohorts were recruited comprising 20 individuals with BDD and 20 (after one exclusion) healthy controls, aged between 19 and 64 years. The BDD participants were recruited from the St Vincent's Hospital Body Image Clinic, Melbourne, Australia. Recruitment was conducted via referrals from this clinic, where clients were identified as having BDD and introduced to the research project. Participants gave their informed consent and diagnosis was then confirmed using the Body Dysmorphic Disorder Diagnostic Module (BDD-DM) and symptom severity was recorded using the Yale–Brown Obsessive Compulsive Scale Modified for Body Dysmorphic Disorder (BDD-YBOCS; Phillips *et al.* 1997). BDD patients were excluded if they had a past or current psychotic disorder, OCD, bulimia nervosa, anorexia nervosa, alcohol or substance abuse history, intellectual/cognitive impairment, metal implants or neurological disturbance. Furthermore, BDD participants were excluded if they had a co-morbid mental disorder that was considered to be their primary diagnosis, ensuring that all individuals in the patient sample had BDD as their primary concern. Because primary diagnosis can be difficult to delineate when there is co-morbid OCD, individuals with OCD were excluded, whereas individuals who also fulfilled the criteria of social phobia or major depression were allowed if BDD was clearly assessed as their primary diagnosis. As BDD shows very high rates of psychiatric co-morbidity (Phillips *et al.* 2007*b*) our exclusion criterion was calibrated to obtain a highly representative sample.

Healthy control participants were sourced from a voluntary healthy research database, comprising members of the public. The control group had no personal or family history of a mental disorder and met the same exclusion criteria outlines for the BDD group. All participants had a Wechsler Test of Adult Reading pre-morbid intelligence quotient (IQ) score of >80, and all participants had English as their preferred language. Of the participants, one control was excluded after MRI acquisition due to incidental brain pathology and is not presented in any of the data tables.

Participants were assessed with the Mini-International Neuropsychiatric Interview (MINI; Sheehan *et al.* 1998) to evaluate the presence or absence of other mental disorders. Handedness was assessed with the Edinburgh Inventory, and social anxiety and depression were measured by the Social Interaction Anxiety Scale (SIAS) (Brown *et al.* 1997) and the Zung Self-rating Depression Scale (Zung, 1965), which has been shown to be sensitive to the clinical severity of depression in psychiatric samples (Biggs *et al.* 1978). Group comparisons were computed using independent-samples *t* tests at $p=0.05$. Demographic and clinical data are displayed in Table 1.

The two participant groups were well matched on age, gender, education, estimated IQ and handedness. The clinical data indicate that mean BDD severity was in the 'moderate' range which is defined as scores between 16 and 30 on the BDD-YBOCS (Phillips *et al.* 1997). Scores below 15 indicate 'mild' BDD symptoms and scores from 31 to 48 are defined as 'severe' symptoms. At the time of MRI acquisition our sample comprised two participants in the mild range, 13 with moderate symptoms and five in the severe range.

The depression scores as measured by the Zung Self-rating Depression Scale indicated that the BDD sample had a higher level of depressive symptoms compared with controls. On average the BDD group fell in the subclinical range of depressive symptoms, defined as a score below 50 (Zung, 1965). A score between 50 and 59 on the scale represents mild depression, between 60 and 69 indicates moderate symptoms and above 70 indicates severe symptoms (Zung, 1965). The SIAS scores showed that the BDD sample had a moderate level of social anxiety symptoms; less than people diagnosed with social phobia but higher than other anxiety disorders (Peters, 2000), which is typical of BDD samples (Coles *et al.* 2006). Assessment with the MINI showed that four BDD participants fulfilled the diagnostic criteria for current major depressive disorder or dysthymia, and five had current agoraphobia or social phobia, showing that our sample was representative of a typical BDD profile (Coles *et al.* 2006). Areas of aesthetic concern for our sample were generally the face, skin and hair. All but two members of the BDD sample were taking psychoactive medication: five where taking seroquel, four escitalopram, two duloxetine, two desvenlafaxine, two diazepam, and one each paroxetine, mirtazapine, lorazepam, methylphenidate, sodium valproate or clomipramine.

### *MRI acquisition*

Participants were scanned using a 3 T scanner (Siemens Magnetom TrioTim, Germany) at the Murdoch Childrens Research Institute (Royal Children's Hospital, Melbourne, Australia). The DTI scanning sequence was conducted as a component of a 1 h long scan. The scanner acquired an isotropic DTI sequence for FA estimations (number of directions = 60, *b* value = 2000s/m$^2$, slice thickness = 2.5 mm). Data were transferred to a Linux workstation for image processing and analyses.

### *FA analysis*

Tract-based spatial statistics (TBSS) developed by Smith *et al.* (2006) was used to create a mean FA skeleton that was representative of our 40 participants. TBSS uses non-linear registration to create a template for FA comparisons that allows voxelwise analysis of multi-subject diffusion data. The TBSS module in the FSL package (http://www.fmrib.ox.ac.uk/fsl/) was used to compute statistics. A two-sample *t* test was conducted using the randomize tool, which tests the *t* value at each voxel against a null distribution generated from 5000 random permutations of group membership. The output contained statistical maps corrected for multiple comparisons ($p<0.05$) using threshold-free cluster enhancement. This method has a high level of sensitivity to true differences while minimizing false positives by avoiding the specification of a subjective cluster-forming threshold (Smith & Nichols, 2009). Specific white matter areas were defined using integrated white matter atlases within FSL: the ICBM-DTI-81 White-Matter Labels Atlas and the JHU White-Matter Tractography Atlas, both developed in the Laboratory of Brain Anatomical MRI at Johns Hopkins University (USA).

In addition, a Pearson product-moment correlation analysis at $p<0.05$ was performed between voxelwise FA and symptom severity as measured by the BDD-YBOCS, Zung Self-rating Depression Scale and SIAS.

## Results

### *FA*

The BDD patients exhibited widespread significant reductions in corrected FA values compared with controls. Table 2 shows decreased FA values in BDD divided into anatomical regions, and shows areas of significant increase in eigenvalues 2 and 3. There were no significant differences between BDD and controls on eigenvalue 1.

Table 2 shows that there are widespread white matter differences in BDD. Since FA is a composite of eigenvalues 1, 2 and 3 and there was no differences on eigenvalue 1, the majority of FA reductions can be accounted for by an increase of radial diffusivity as represented by eigenvalues 2 and 3.

### *Correlation analysis*

The correlation analysis found that there was no significant correlation between BDD symptom severity scores and corrected FA in any voxel within the BDD sample. Likewise, there was no significant correlation between depression scores and FA in the BDD group. In the BDD group, social anxiety scores were found to correlate negatively with corrected FA values on the left superior longitudinal fasciculus (x = –38, y = 2, z = 22) at $p<0.05$. There was no correlation between depression and anxiety in any voxel in the control group.

## Discussion

As far as we are aware, this is the first study to examine white matter differences within a BDD sample using DTI. Additionally, this study had the largest BDD sample in a neuroimaging study to date, and our sample was thoroughly screened to ensure that BDD was the primary condition and that it was

**Table 2.** *Areas of decreased FA and increased eigenvalue 2 and 3 values in BDD*

| x | y | z | FA: *p* | Eigenvalue 2: *p* | Eigenvalue 3: *p* | Anatomical region |
|---|---|---|---|---|---|---|
| −39 | −47 | 22 | <0.05 | <0.05 | <0.05 | Superior longitudinal fasciculus (left)[a] |
| 40 | −44 | 19 | <0.05 | N.S. | <0.05 | Superior longitudinal fasciculus (right)[a,b] |
| −37 | −53 | 0 | <0.05 | <0.05 | <0.05 | Posterior thalamic radiation (left)[b] |
| 13 | 44 | −16 | <0.05 | <0.05 | <0.05 | Uncinate fasciculus (right)[b] |
| 31 | 42 | 3 | <0.05 | <0.05 | <0.05 | Inferior fronto-occipital fasciculus (right)[a] |
| −28 | 31 | 12 | <0.05 | <0.05 | N.S. | Inferior fronto-occipital fasciculus (left)[a] |
| −42 | −29 | −7 | <0.05 | <0.05 | N.S. | Inferior longitudinal fasciculus (left)[a] |
| −1 | −11 | 25 | <0.05 | <0.05 | <0.01 | Body of corpus callosum[b] |
| 18 | −37 | 30 | <0.05 | <0.05 | <0.05 | Splenium of corpus callosum (right)[b] |
| −9 | 29 | 11 | <0.05 | <0.05 | <0.05 | Genu of the corpus callosum[b], or forcepts minor left[a] |
| 9 | 29 | 10 | <0.05 | N.S. | N.S. | Genu of the corpus callosum[b], or forcepts minor (right)[a] |
| −8 | −5 | 11 | <0.05 | N.S. | N.S. | Anterior thalamic radiation (left)[a] |
| 9 | −31 | −23 | <0.05 | N.S. | N.S. | Corticospinal tract (right)[a] |
| −28 | −20 | −7 | <0.05 | N.S. | N.S. | Fornix or stria terminalis (cannot be resolved with resolution)[b] |

FA, Fractional anisotropy; BDD, body dysmorphic disorder; N.S., not significant.
[a] Area identified using the JHU White-Matter Tractography Atlas.
[b] Area identified using the ICBM-DTI-81 White-Matter Labels Atlas.

representative of the patient population (Phillips *et al.* 2007*a*). Importantly, this study provides preliminary evidence of neural abnormalities in BDD; specifically, evidence for a widespread loss of integrity in white matter connectivity.

Consistent with the hypothesis that we would find similar reductions in FA in BDD as has been shown in OCD (Szeszko *et al.* 2005; Garibotto *et al.* 2010), we found reduced FA in the corpus callosum, superior longitudinal fasciculus, and inferior fronto-occipital fasciculus, bilaterally. The widespread FA reductions in our BDD sample can be accounted for through increases in radial diffusivity rather than reduced eigenvalue 1. Of the 12 areas identified in Table 2 as having significantly reduced FA, nine can be accounted for by eigenvalues 2 and 3. Indeed a comparison between Figs. 1 and 2 shows that mean diffusivity was more widespread than FA differences. This suggests loss of integrity in the white matter whereby attenuated white matter pathways allow water diffusion in directions that are not consistent with overall white matter directionality. Reduced FA has been shown in other work to be driven by myelin abnormalities which is largely under genetic control (Menzies *et al.* 2008). Therefore, neurodevelopment irregularities leading to abnormal myelination may be important to the predisposition to BDD. Reduced FA due to increased radial diffusivity has also been found in OCD (Bora *et al.* 2011).

While the loss of white matter integrity was widespread, it is worth considering the impact of specific neural pathways on BDD cognition. For example, reduced FA in the corpus callosum indicates attenuated interhemispheric communication. This may explain the neuropsychological research in BDD that has shown difficulties with integration of detailed and global information processing (Deckersbach *et al.* 2000; Feusner *et al.* 2010*a*). Moreover, white matter operating inefficiently may explain the Stroop task difficulties associated with BDD (Hanes, 1998), as reduced FA and other neurobiological correlates in frontostriatal–limbic regions have been shown to relate to performance on the Stroop and planning tasks in geriatric depression and OCD (van den Heuvel *et al.* 2005; Murphy *et al.* 2007). Furthermore, reduced FA in the bilateral superior longitudinal fasciculi suggests that connectivity between the prefrontal, parietal, occipital and temporal regions (Makris *et al.* 2005) may be compromised.

The reduced FA in the uncinate fasciculus is of particular interest because it is a major white matter fibre tract that connects the inferofrontal and anterotemporal cortices, and it travels over the lateral nuclei of the amygdala. Thus, lower FA within this tract suggests compromised fronto-amygdala structural connectivity in BDD. In the context of BDD, this is interesting when considering that frontal regions such as the orbitofrontal cortex serve important roles in top-down regulation of amygdala reactivity to control negative affect and mediate threat perception (Ochsner *et al.* 2002; Barrett *et al.* 2007).

It is clear from past research that there are grey matter differences in BDD patients compared with non-psychiatric controls, but how these interact with the white matter abnormalities reported here remains speculative. Volumes of the right amygdala and the

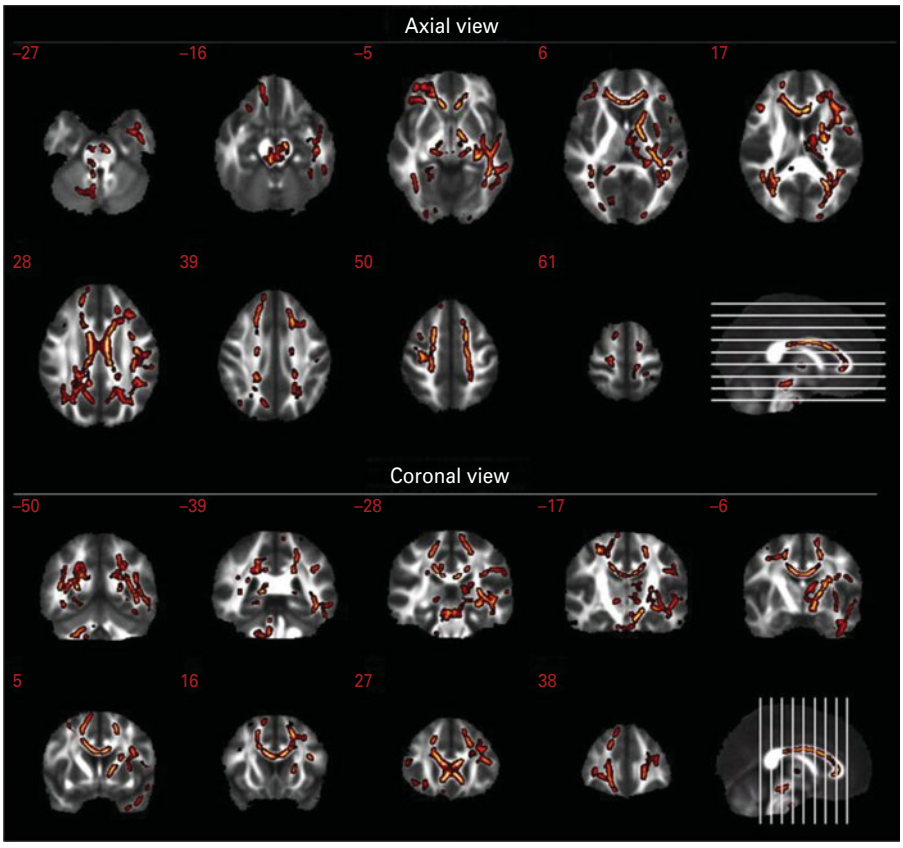


**Fig. 1.** Statistically significant fractional anisotropy reductions in body dysmorphic disorder sample compared with controls. Numbers in red represent distance (mm) from the anterior commissure.

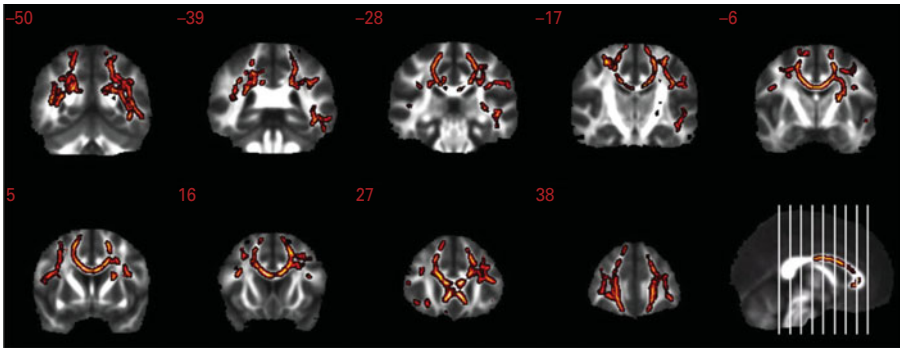


**Fig. 2.** Statistically significant mean diffusivity increases in body dysmorphic disorder sample compared with controls, coronal view. Numbers in red represent distance (mm) from the anterior commissure.

inferior frontal gyrus have been shown to correlate with current BDD symptom severity (Feusner *et al.* 2009), suggesting that differences in these grey matter structures are the most proximal contributor to symptoms, while white matter differences can be considered as a more distal, or perhaps predisposing, factor in BDD.

Our examination of whether BDD symptom severity correlated with FA was not significant, despite such a correlation being found in OCD samples (Garibotto *et al.* 2010). Given that we had a large range of symptom severity and a sample size that was able to provide robust correlation results, our non-significant results suggest that BDD has a distinct neurobiological makeup that is state-independent. Such a finding suggests important trait-related white matter abnormalities in BDD. This result would need to be replicated; further research could establish if these brain differences are present in an individual whether or not illness is active, and provide preliminary evidence

for a BDD phenotype that is perhaps related to the emerging phenotype considered important in OCD (Menzies *et al.* 2008).

The results showing a significant negative correlation between social anxiety scores and FA in the left superior longitudinal fasciculus are interesting, considering that the main measure of BDD symptom severity (the BDD-YBOCS) was not significantly correlated. It is possible that our social anxiety measure was sensitive to an important feature of BDD that our main symptom severity rating scale was not. Indeed, social anxiety can be seen as a important factor within BDD symptomatology and is best conceptualized as an expression of higher BDD symptoms rather than a separate problem of social phobia (Coles *et al.* 2006). The finding that there was no correlation between depression scores and FA in any voxel indicated that the FA differences among the two groups can be accounted for by the diagnosis of BDD independent from depression ratings. Indeed, this finding taken together with the non-significant contribution of BDD symptoms to FA suggests that state-based symptoms scores bear less importance to white matter than state-independent diagnosis.

The parallels that have been drawn between OCD and BDD in terms of clinical features (Stewart *et al.* 2008) are yet to be robustly supported using DTI imaging techniques. In fact, to date, the nine DTI studies in OCD have yielded inconsistent results, with both local FA increases and decreases reported (e.g. Menzies *et al.* 2008; Bora *et al.* 2011, Lochner *et al.* 2012). The inconsistent data in OCD and the preliminary evidence of widespread FA decreases provided by the current study in BDD should be supplemented by future studies to help determine biological similarities and differences between the two disorders.

A limitation of this study is that nearly all of our BDD participants were taking medication at the time of MRI acquisition. This may be problematic as treatment is likely to have reduced the severity of BDD symptoms, thereby possibly influencing FA. However, as white matter integrity is generally considered state independent, current BDD symptomatology is unlikely to have significantly influenced the results. At the time of MRI acquisition two of the BDD participants were rated as having mild BDD symptoms on the BDD-YBOCS, although an active diagnosis of BDD was still confirmed using the BDD-DM.

In conclusion, we believe this to be the first study to examine white matter integrity in a BDD sample. The main contribution of our data is that they provide evidence that individuals with BDD have compromised white matter fibres, reflected by changes in fibre directionality as indicated by eigenvalues 2 and 3. This reduced connectivity among different regions of the brain is widespread in nature and is not related to symptom severity.

## Acknowledgements

This study was funded by a Monash Strategic Grant.

## Declaration of Interest

None.

## References

**Atmaca M, Bingol I, Aydin A, Yildirim H, Okur I, Yildirim M, Mermi O, Gurok M** (2010). Brain morphology of patients with body dysmorphic disorder. *Journal of Affective Disorders* **123**, 258–263.

**Barrett LF, Bliss-Moreau E, Dunca SL, Rauch SL, Wright CI** (2007). The amygdala and the experience of affect. *Social Cognitive and Affective Neuroscience* **2**, 73–83.

**Basser PJ, Mattiello J, LeBihan D** (1994). MR diffusion tensor spectroscopy and imaging. *Biophysical Journal* **66**, 259–267.

**Basser PJ, Pierpaoli C** (1996). Microstructural and physiological features of tissues elucidated by quantitative-diffusion-tensor MRI. *Journal of Magnetic Resonance Series B* **111**, 209–219.

**Biggs JT, Wylie LT, Ziegler VE** (1978). Validity of the Zung Self-rating Depression Scale. *British Journal of Psychiatry* **132**, 381–385.

**Bora E, Harrison B, Fornito A, Cocchi L, Pujol J, Fontenelle L, Velakoulis D, Pantelis C, Ycel M** (2011). White matter microstructure in patients with obsessive–compulsive disorder. *Journal of Psychiatry and Neuroscience* **36**, 42–46.

**Brown EJ, Turovsky J, Heimberg RG, Juster HR, Brown TA, Barlow DH** (1997). Validation of the Social Interaction Anxiety Scale and the Social Phobia Scale across the anxiety disorders. *Psychological Assessment* **9**, 21.

**Buchanan BG, Rossell SL, Castle DJ** (2011). Body dysmorphic disorder: a review of nosology, cognition and neurobiology. *Neuropsychiatry* **1**, 71–80.

**Buhlmann U, Glaesmer H, Mewes R, Fama JM, Wilhelm S, Brähler E, Rief W** (2010). Updates on the prevalence of body dysmorphic disorder: a population-based survey. *Psychiatry Research* **178**, 171–175.

**Buhlmann U, McNally RJ, Etcoff NL, Tuschen-Caffier B, Wilhelm S** (2004). Emotion recognition deficits in body dysmorphic disorder. *Journal of Psychiatric Research* **38**, 201–206.

**Buhlmann U, McNally RJ, Wilhelm S, Florin I** (2002). Selective processing of emotional information in body dysmorphic disorder. *Journal of Anxiety Disorders* **16**, 289–298.

**Carey P, Seedat S, Warwick J, van Heerden B, Stein DJ** (2004). SPECT imaging of body dysmorphic disorder.

*Journal of Neuropsychiatry and Clinical Neurosciences* **16**, 357–359.

**Coles ME, Phillips K, Menard W, Pagano ME, Fay C, Weisberg RB, Stout RL** (2006). Body dysmorphic disorder and social phobia: cross-sectional and prospective data. *Depression and Anxiety* **23**, 26–33.

**Deckersbach T, Savage CR, Phillips K, Wilhelm S, Buhlmann U, Rauch SL, Baer L, Jenike MA** (2000). Characteristics of memory dysfunction in body dysmorphic disorder. *Journal of the International Neuropsychological Society* **6**, 673–681.

**Dunai J, Labuschagne I, Castle D, Kyrios M, Rossell S** (2009). Executive function in body dysmorphic disorder. *Psychological Medicine* **40**, 1541–1548.

**Feusner J, Moller H, Altstein L, Suger C, Bookheimer S, Woon J, Hembacher E** (2010*a*). Inverted face processing in body dysmorphic disorder. *Journal of Psychiatric Research* **44**, 1088–1094.

**Feusner J, Moody T, Hembacher E, Townsend J, McKinley M, Moller H, Bookheimer S** (2010*b*). Abnormalities of visual processing and frontostriatal systems in body dysmorphic disorder. *Archives of General Psychiatry* **67**, 197–205.

**Feusner J, Townsend J, Bystritsky A, Bookheimer S** (2007). Visual information processing of faces in body dysmorphic disorder. *Archives of General Psychiatry* **64**, 1417–1426.

**Feusner J, Townsend J, Bystritsky A, McKinley M, Moller H, Bookheimer S, Feusner JD, Townsend J, Bystritsky A, McKinley M, Moller H, Bookheimer S** (2009). Regional brain volumes and symptom severity in body dysmorphic disorder. *Psychiatry Research* **172**, 161–167.

**Frare F, Perugi G, Ruffolo G, Toni C** (2004). Obsessive–compulsive disorder and body dysmorphic disorder: a comparison of clinical features. *European Psychiatry* **19**, 292–298.

**Garibotto V, Scifo P, Gorini A, Alonso C, Brambati S, Bellodi L, Perani D** (2010). Disorganization of anatomical connectivity in obsessive compulsive disorder: a multi-parameter diffusion tensor imaging study in a subpopulation of patients. *Neurobiology of Disease* **37**, 468–476.

**Hanes KR** (1998). Neuropsychological performance in body dysmorphic disorder. *Journal of the International Neuropsychological Society* **4**, 167–171.

**Labuschagne I, Castle DJ, Rossell SL** (2011). What the cognitive deficits in body dysmorphic disorder tell us about the underlying neurobiology: an investigation of three cases. *International Journal of Cognitive Therapy* **4**, 21–33.

**Lochner C, Fouché JP, du Plessis S, Fineberg N, Chamberlain SR, Stein DJ** (2012). Evidence for fractional anisotropy and mean diffusivity white matter abnormalities in the internal capsule and cingulum in patients with obsessive–compulsive disorder. *Journal of Psychiatry and Neuroscience* **37**, 110059.

**Makris N, Kennedy DN, McInerney S, Sorensen AG, Wang R, Caviness VS Jr, Pandya DN** (2005). Segmentation of subcomponents within the superior longitudinal fascicle in humans: a quantitative, *in vivo*, DT-MRI study. *Cerebral Cortex* **15**, 854–869.

**McKay D, Neziroglu F, Yaryura-Tobias JA** (1997). Comparison of clinical characteristics in obsessive–compulsive disorder and body dysmorphic disorder. *Journal of Anxiety Disorders* **11**, 447–454.

**Menzies L, Williams GB, Chamberlain SR, Ooi C, Fineberg N, Suckling J, Sahakian BJ, Robbins TW, Bullmore ET** (2008). White matter abnormalities in patients with obsessive–compulsive disorder and their first-degree relatives. *American Journal of Psychiatry* **165**, 1308–1315.

**Monzani B, Rijsdijk F, Iervolino AC, Anson M, Cherkas L, Mataix-Cols D** (2012). Evidence for a genetic overlap between body dysmorphic concerns and obsessive–compulsive symptoms in an adult female community twin sample. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* **159B**, 376–382.

**Mori S, Wakana S, Nagae-Poetscher L, Van Zijl PCM** (2005). *MRI Atlas of Human White Matter*. Elsevier: Amsterdam.

**Murphy C, Gunning Dixon F, Hoptman M, Lim K, Ardekani B, Shields J, Hrabe J, Kanellopoulos D, Shanmugham B, Alexopoulos G** (2007). White-matter integrity predicts Stroop performance in patients with geriatric depression. *Biological Psychiatry* **61**, 1007–1010.

**Ochsner K, Bunge S, Gross J, Gabrieli J** (2002). Rethinking feelings: an fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience* **14**, 1215–1229.

**Peters L** (2000). Discriminant validity of the Social Phobia and Anxiety Inventory (SPAI), the Social Phobia Scale (SPS) and the Social Interaction Anxiety Scale (SIAS). *Behaviour Research and Therapy* **38**, 943–950.

**Phillips K, Coles ME, Menard W, Yen S, Fay C, Weisberg RB** (2005*a*). Suicidal ideation and suicide attempts in body dysmorphic disorder. *Journal of Clinical Psychiatry* **66**, 717–722.

**Phillips K, Didie ER, Menard W** (2007*a*). Clinical features and correlates of major depressive disorder in individuals with body dysmorphic disorder. *Journal of Affective Disorders* **97**, 129–135.

**Phillips K, Hollander E, Rasmussen S, Aronowitz B, DeCaria C, Goodman W** (1997). A severity rating scale for body dysmorphic disorder: development, reliability, and validity of a modified version of the Yale–Brown Obsessive Compulsive Scale. *Psychopharmacology Bulletin* **33**, 17–22.

**Phillips K, Menard W, Fay C, Weisberg R** (2005*b*). Demographic characteristics, phenomenology, comorbidity, and family history in 200 individuals with body dysmorphic disorder. *Psychosomatics* **46**, 317–325.

**Phillips K, Pinto A, Menard W, Eisen JL, Mancebo M, Rasmussen SA** (2007*b*). Obsessive–compulsive disorder *versus* body dysmorphic disorder: a comparison study of two possibly related disorders. *Depression and Anxiety* **24**, 399–409.

**Rauch SL** (2000). Neuroimaging research and the neurobiology of obsessive–compulsive disorder: where do we go from here? *Biological Psychiatry* **47**, 168–170.

**Rauch SL, Phillips K, Segal E, Makris N, Shin LM, Whalen PJ, Jenike MA, Caviness VS Jr, Kennedy DN**

(2003). A preliminary morphometric magnetic resonance imaging study of regional brain volumes in body dysmorphic disorder. *Psychiatry Research: Neuroimaging* **122**, 13–19.

**Rotge JY, Langbour N, Guehl D, Bioulac B, Jaafari N, Allard M, Aouizerate B, Burbaud P** (2010). Gray matter alterations in obsessive–compulsive disorder: an anatomic likelihood estimation meta-analysis. *Biological Psychiatry* **35**, 686–691.

**Sheehan D, Lecrubier Y, Sheehan K, Amorim P, Janavs J, Weiller E, Hergueta T, Baker R, Dunbar G** (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry* **59** (Suppl 20), 22–57.

**Smith SM, Jenkinson M, Johansen-Berg H, Rueckert D, Nichols TE, Mackay CE, Watkins KE, Ciccarelli O, Cader MZ, Matthews PM, Behrens TEJ** (2006). Tract-based spatial statistics: voxelwise analysis of multi-subject diffusion data. *Neuroimage* **31**, 1487–1505.

**Smith SM, Nichols TE** (2009). Threshold-free cluster enhancement: addressing problems of smoothing, threshold dependence and localisation in cluster inference. *Neuroimage* **44**, 83–98.

**Stewart S, Stack DE, Wilhelm S** (2008). Severe obsessive–compulsive disorder with and without body dysmorphic disorder: clinical correlates and implications. *Annals of Clinical Psychiatry* **20**, 33–38.

**Szeszko P, Ardekani B, Ashtari M, Malhotra A, Robinson D, Bilder R, Lim K** (2005). White matter abnormalities in obsessive–compulsive disorder: a diffusion tensor imaging study. *Archives of General Psychiatry* **62**, 782–790.

**van den Heuvel O, Veltman D, Groenewegen H, Witter M, Merkelbach J, Cath D, van Balkom A, van Oppen P, van Dyck R** (2005). Disorder-specific neuroanatomical correlates of attentional bias in obsessive–compulsive disorder, panic disorder, and hypochondriasis. *Archives of General Psychiatry* **62**, 922–933.

**Whiteside S, Port J, Abramowitz J** (2004). A meta-analysis of functional neuroimaging in obsessive–compulsive disorder. *Psychiatry Research* **132**, 69–79.

**Zung WW** (1965). A self-rating depression scale. *Archives of General Psychiatry* **12**, 63–70.