



Reduced cortical thickness in body dysmorphic disorder

Sally A. Grace^{a,*}, Ben G. Buchanan^b, Jerome J. Maller^c, Wei Lin Toh^c, David J. Castle^{d,e}, Susan L. Rossell^{c,e}



^a Brain and Psychological Sciences Research Centre, Swinburne University, Melbourne, Australia

^b Monash University School of Psychological Science, Monash University, Melbourne, Australia

^c Monash Alfred Psychiatry Research Centre, Monash University, Melbourne, Australia

^d Psychiatry, St Vincent's Hospital, Melbourne, Australia

^e Psychiatry, University of Melbourne, Melbourne, Australia

ARTICLE INFO

Keywords:

Cerebral cortex
Grey matter
Magnetic resonance imaging
Psychiatry

ABSTRACT

Recent neuroimaging studies in body dysmorphic disorder (BDD) have implicated abnormal structure and function of occipito-temporal and fronto-limbic regions in the potential pathophysiology of the disorder. To date, morphometric investigations have yielded inconsistent results, and have suggested that clinical symptoms may mediate structural brain abnormalities in BDD. We measured Grey Matter (GM) cortical thickness in 20 participants with BDD and 20 healthy control participants matched on age, gender, estimated IQ and handedness. We observed cortical thinning in BDD patients compared with healthy control participants within the left middle temporal and left inferior parietal gyrus. No significant relationships between cortical thickness and BDD symptom severity, insight, social anxiety and depression were observed within the BDD group. Thinning within left temporal and left inferior parietal regions supports the involvement of these regions in the pathophysiology of BDD.

1. Introduction

Body dysmorphic disorder (BDD) is a psychiatric disorder characterized by preoccupations with an objectively absent 'defect' in physical appearance (American Psychiatric Association, 2013). BDD is relatively common with reported prevalence rates between 1–2% in the general population (Mufaddel et al., 2013; Rief et al., 2006). Without treatment, BDD is associated with high levels of distress, poor insight manifesting as delusions, significant impairment in social and occupational functioning, and suicidal ideations (DeMarco et al., 1998; Phillips et al., 2006). In studies of cognition BDD has been associated with deficits in memory, executive functioning, and holistic visual processing, which have been linked to abnormalities in occipito-temporal and fronto-limbic regions (Feusner et al., 2011, 2007; Hanes, 1998; Li et al., 2015).

Morphometric MRI investigations have identified neuroanatomical abnormalities in BDD patients. Previous voxel-based morphometry (VBM), anatomical tracing, and volumetric segmentation data in BDD samples have demonstrated significant total whole-brain volume and grey matter (GM) volume reductions (Buchanan et al., 2014), as well as larger total white matter volumes (Atmaca et al., 2010; Rauch et al., 2003). In BDD samples, regional reductions in brain volume have also

been identified within the bilateral orbitofrontal cortex (OFC), bilateral anterior cingulate (Rauch et al., 2003), right orbitofrontal cortex, bilateral thalamus, left anterior cingulate cortex, left hippocampus and left amygdala, relative to controls (Buchanan et al., 2014), as well as larger left hemispheric caudate volumes (Rauch et al., 2003). A noticeable trend within these data is that morphometric abnormalities between BDD and healthy control groups appear to be mediated by clinical variables. For example, significant positive correlations of illness duration with bilateral orbito-frontal cortex (OFC) volumes (Atmaca et al., 2010), and right OFC volumes (Buchanan et al., 2014) have been observed in BDD samples. One study reported positive correlations of BDD symptom severity (as measured by the Yale-Brown Obsessive-Compulsive Disorder Scale; BDD-YBOCS; Phillips et al., 1997) with GM volumes in the left inferior frontal gyrus and right amygdala despite no significant overall group differences in brain morphometry (Feusner et al., 2009).

Further highlighting the sensitive influence of clinical variables on brain morphometry in BDD, a recent analysis of cortical thickness in BDD reported no group differences in cortical thickness in a sample of 49 BDD patients and 44 demographically matched healthy control participants (Madsen et al., 2015). They did, however, find significant associations between anxiety severity and GM cortical thinning in the

* Correspondence to: Brain and Psychological Sciences Research Centre, Advanced Technology Building, Burwood Rd, Hawthorn VIC 3122, Australia.
E-mail address: sgrace@swin.edu.au (S.A. Grace).

left superior temporal cortex and greater GM volume in the right caudate nucleus (Madsen et al., 2015). Altogether, this suggests that in general, studies of brain morphometry in BDD have yielded inconsistent results.

There are some methodological issues that are important when considering existing neuroimaging findings in BDD. Heterogeneous analysis techniques and sample characteristics have a significant impact on the generalisability of the findings. With the exception of two studies (Buchanan et al., 2014; Madsen et al., 2015) the sample sizes involved were small (i.e. <12 BDD patients compared to controls), and gender ratios, patient comorbidities and medication use are inconsistent.

To our knowledge, only one examination of cortical thickness has been performed in a sample of BDD participants. Thus, the aim of this study was to further extant morphometric data in BDD through an examination of cortical thickness and its relationship with clinical variables in participants with BDD compared with a sample of demographically matched healthy controls. We computed Pearson correlations between grey matter thickness and clinical variables within the BDD group. Specifically, we hypothesised that reported anxiety severity within the BDD sample would correlate with grey matter thinning within the temporal lobe.

2. Methods

2.1. Participants

Participants comprised 20 individuals with BDD and 20 healthy control participants, aged between 19 and 64 years, all of whom provided informed consent (Table 1). Recruitment for the BDD group was conducted via referrals from St Vincent's Hospital Body Image Clinic in Melbourne, Australia, where clients were identified as having BDD and introduced to the research project (all had participated in a previous MRI study examining brain connectivity in BDD (Buchanan et al., 2013)). BDD diagnosis was confirmed by the research team 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). All but two of the BDD sample was

Table 1
Demographic and clinical variables for the BDD and control groups.

	BDD (n=20)	Healthy Controls (n=20)	Group Comparison (df =38)
Demographic characteristics			
Age (years)	34.6 (11.5)	31.9 (11.4)	$p = 0.45$
Years of education	14.9 (2.4)	16.3 (3.0)	$p = 0.11$
WTAR IQ estimate	106 (10.7)	110 (6.5)	$p = 0.13$
Handedness (L/R)	3/17	3/17	
Sex (M/F)	6/14	6/14	
Clinical variables			
BDD severity (BDD-YBOCS)	24.9 (9.6)		
Duration of Illness (years)	10.8 (6.9)		
BABS	15.71 (7.9)		
Zung	46 (11.26)		
SIAS	41.7 (17.58)		

Note: Data presented as mean (SD).

BABS=Brown Assessment of Beliefs Scale total score; BDD=Body dysmorphic disorder; WTAR=Wechsler Test of Adult Reading; Zung=Zung Self-Rating Depression Scale total score.

taking psychoactive medication: five quetiapine, four escitalopram, two duloxetine, two desvenlafaxine, two diazepam, and one each paroxetine, mirtazapine, lorazepam, methylphenidate, sodium valproate or clomipramine. Depressive symptoms were assessed using the Zung Self-Rating Depression Scale which is a brief self-administered survey to quantify the current depressive symptoms experienced by adult patients (Zung, 1965). Social anxiety symptoms were measured using the Social Interaction Anxiety Scale (SIAS), a self-report scale designed to measure social interaction anxiety (Mattick and Clarke, 1998). Degree of conviction and insight into beliefs (i.e. degree of delusionality) was measured using the Brown Assessment of Beliefs Scale (BABS; Eisen et al., 1998).

The control group comprised members of the public who had no personal or family history of a mental disorder. All participants had English as their preferred language and a Wechsler Test of Adult Reading (WTAR) pre-morbid intelligence quotient (IQ) score of >80. Participants were assessed with the Mini-International Neuropsychiatric Interview (MINI500) and BDD-DM. Handedness was assessed with the Edinburgh Handedness Inventory (EHI; Oldfield, 1971). A more detailed account of selection criteria and demographic characteristics is described elsewhere (Buchanan et al., 2013).

Exclusion criteria for all participants included: past or current psychotic disorder, alcohol or substance abuse history, intellectual or cognitive impairment, and metal implants or neurological disturbances (e.g. trauma brain injury). BDD participants were excluded if they had past or current diagnoses of OCD, bulimia nervosa, anorexia nervosa, or a comorbid mental disorder that was considered as their primary diagnosis, ensuring that all individuals in the clinical sample had BDD as their primary diagnosis.

2.2. Magnetic resonance imaging (MRI) acquisition

Participants were scanned using a 3T scanner (Siemens Magnetom Tim Trio, Germany) at the Murdoch Children's Research Institute (Royal Children's Hospital, Melbourne, Australia). An AC-PC aligned high-resolution structural T1-weighted MPRAGE sequence (512 slices; slice thickness =1 mm; TE =2.15 ms; TR =1900 ms; field of view =256 mm; in plane resolution 0.5×0.5 mm²) was acquired allowing high-quality data for structural brain image processing.

2.3. MRI scan processing and calculation of cortical thickness

2.3.1. Quantification of cortical thickness

Cortical reconstruction and segmentation was performed with the FreeSurfer 5.3 (FS) image analysis suite (<http://surfer.nmr.mgh.harvard.edu/>). FS output was checked on a slice-by-slice basis with the FreeSurfer viewer (freeview) for any segmentations that were not correct. They were then viewed as 3-dimensional renderings with tksurfer. Cortical thickness group analysis was undertaken within Qdec (Query, Design, Estimate, Contrast. A Graphical User Interface tool for FS; https://surfer.nmr.mgh.harvard.edu/fswiki/FsTutorial/QdecGroupAnalysis_freeview). BDD and control groups were compared for thickness difference, followed by correlational analyses between FS variables (whole cortex) and clinical scores in those with BDD. The subsequent parameters within Qdec were used for all of the analyses: Measure=Thickness, Smoothing=10, Monte Carlo simulation used to correct for multiple comparisons using a threshold of 1.3 ($p < 0.05$) and the appropriate two-tailed tests were applied.

2.3.2. Statistical analysis

FreeSurfer data were statistically analysed using SPSS for Windows version 19.0 (SPSS, Chicago, IL). Simple chi-square and analysis of variance (ANOVA) were employed to compare differences in GM thickness within- and between- the groups, covarying for the effects of age. We did not control for the effects of gender as there was not a

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