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Short communication

A comparison of executive function in Body Dysmorphic Disorder (BDD) and Obsessive-Compulsive Disorder (OCD)

I. Labuschagne [a,*], S.L. Rossell [b,c,d,e], J. Dunai [b,c], D.J. Castle [b,f], M. Kyrios [d]

[a] School of Psychology and Psychiatry, Monash University, Clayton campus, Clayton, Victoria 3800, Australia
[b] School of Behavioral Science, School of Psychology, University of Melbourne, Melbourne, Victoria 3010, Australia
[c] Department of Psychiatry, University of Melbourne, Melbourne, Victoria 3010, Australia
[d] Brain and Psychological Sciences Research Centre, Swinburne University, 427 Burwood Rd, Hawthorn, Victoria 3122, Australia
[e] Cognitive Neuropsychology Lab, Monash-Alfred Psychiatry Research Centre (MAPrc), Monash University, 1st Floor, Old Baker Building, The Alfred, Commercial Road, Melbourne, VIC 3004, Australia
[f] Department of Psychiatry, St Vincent's Hospital, PO Box 2900, Fitzroy, Victoria 3065, Australia

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ABSTRACT

Evidence now suggests executive dysfunction in Body Dysmorphic Disorder (BDD) which may be related to Obsessive-Compulsive Disorder (OCD). However, neurocognitive performance in the two disorders has rarely been compared. This study compared 14 BDD participants on neurocognitive tasks taken from the Cambridge Automated Neuropsychological Test Automated Battery (CANTAB) with previously published data from 23 OCD participants (Purcell, Maruff, Kyrios, & Pantelis, 1998). Effect sizes from three executive function tests (Spatial Span, Spatial Working Memory and Stockings of Cambridge), and one visual memory task (Pattern Recognition) were compared for group differences using difference and equivalence testing. Equivalence testing was used to determine whether BDD and OCD effects sizes were equivalent, non-equivalent or equivocal. Results indicated an equivocal pattern for Spatial Span, Spatial Working Memory, Pattern Recognition and most Stockings of Cambridge measures. However, results for Stockings of Cambridge accuracy measure indicated a non-equivalent pattern, with BDD but not OCD participants performing significantly worse than controls. Results suggest a number of similarities in neurocognitive function in BDD and OCD, although it was not possible to establish statistical equivalence on most study measures. The findings raise the possibility of more severe planning deficits in BDD compared to OCD.

1. Introduction

Body Dysmorphic Disorder (BDD) is characterized by a preoccupation with imagined or minor defects in appearance as well as repetitive behaviors that are designed to hide or improve the supposed defects. These symptoms ultimately lead to significant distress and impairment in social and/or occupational functioning (DSM-IV-TR; American Psychiatric Association (APA), 2000). Individuals with BDD frequently present with comorbid Obsessive-Compulsive Disorder (OCD) (Altamura, Paluello, Mundo, Medda, & Mannu, 2001; Frare, Perugi, Ruffolo, & Toni, 2004; Hollander, Cohen, & Simeon, 1993), and the two disorders share many features (Phillips et al., 2010). For example, individuals with BDD experience recurrent, persistent and intrusive preoccupations about their perceived physical defects (Buhlmann et al., 2002; Perugi et al., 1997; Phillips, McElroy, Keck, Pope, & Hudson, 1993) which are similar to the obsessions seen in OCD. Ritualistic behaviors in BDD such as mirror checking and hair grooming (Phillips et al., 1993; Veale and Riley, 2001) are similar to the compulsions of OCD. Both BDD and OCD also have a similar age at onset and course (Phillips et al., 2007), and may both respond preferentially to selective-serotonin reuptake-inhibitors (Hollander, Liebowitz, Winchel, Klumker, & Klein, 1989; Hollander et al., 1999; Phillips, 1998; Soomro, Altman, Rajagopal, & Oakley-Browne, 2008), suggesting a common neurochemical dysfunction. Based on this evidence, the APA has proposed that BDD should be classified under Obsessive-Compulsive and Related Disorders in the new DSM-V (American Psychiatric Association (APA), 2013; also see Phillips et al., 2010) rather than under Somatoform Disorders (DSM-IV-TR; APA, 2000). There are, however, some significant and important clinical differences between the two conditions. For instance, BDD patients are reported to have poorer insight with greater delusional endorsement (Labuschagne, Dunai, Castle, Kyrios, & Rossell, 2010) than those with OCD (Frare et al., 2004; Phillips, McElroy, Hudson, & Pope, 1995). In addition, BDD patients are more likely than OCD patients to demonstrate lifetime suicidal ideation,

* Corresponding author. Tel.: +61 3 9902 0481; fax: +61 3 9905 3948.
E-mail address: Izelle.labuschagne@monash.edu (I. Labuschagne).

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lifetime major depressive disorder and lifetime substance use disorder (Phillips et al., 2007).

Despite the distress and burdensome nature associated with BDD, and its relatively high prevalence (e.g. 1.7%) (Rief, Buhlmann, Wilhelm, Borkenhagen, & Brahler, 2006), BDD remains poorly understood and under-researched (Castle & Rossell, 2006; Phillips et al., 1993; Veale, 2004). Critically, little is known about cognitive impairments associated with BDD. This is of concern because greater understanding of the cognitive deficits characteristic of BDD could assist in the development of more effective treatment strategies. In addition, the possibility of a relationship between BDD and OCD, suggests that these disorders may share similar neurocognitive characteristics. This in turn may indicate similar etiology and underlying neurobiological dysfunction and could inform both nosological discussions and the design of specific neurocognitive remediation packages for each disorder.

To date, there have been few published neurocognitive studies of BDD, but results of studies have generally suggested executive dysfunction. For example, Hanes (1998) compared patients with BDD ($n=14$), OCD ($n=10$), and schizophrenia ($n=14$), to a group of control participants ($n=24$) on tests of executive function, memory and motor function. This study reported similarly impaired performance on the New Tower of London and Stroop in BDD and OCD patient groups compared to controls, suggesting poor executive function in both BDD and OCD and providing support for the idea of BDD being classified under Obsessive-Compulsive and Related Disorders. Deckersbach and colleagues (2000) compared BDD patients ($n=17$) with an equal number of controls on the Rey Complex Figure Test (RCFT) and the California Verbal Learning Test (CVLT), and found deficits on both tests, suggesting both non-verbal and verbal memory impairments respectively. The authors argued that these memory deficits were mediated by poor organizational strategies, possibly resulting from executive dysfunction (Deckersbach et al., 2000). Recently, we compared BDD patients ($n=14$) with matched controls and again established executive function deficits, including problems with on-line manipulation, planning and organization of information. However, patients in this study also demonstrated intact spatial memory capacity, motor speed and visual memory (Dunai, Labuschagne, Castle, Kyrios, & Rossell, 2010).

Although these studies report executive dysfunction in BDD, comparison between studies is difficult because many of the cognitive tasks used differed between studies, and conflicting results were obtained where similar tasks were used. For example, Hanes (1998) found no differences between controls and BDD patients on the RCFT and the Rey Auditory Verbal Learning Task, whilst Deckersbach and colleagues (2000) observed deficits in immediate recall for the RCFT and CVLT in their BDD cohort. In our study (Dunai et al., 2010), tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB) were selected to facilitate comparison between BDD patients and an OCD sample who had completed the same tasks in a study conducted by Purcell et al. (1998); see below.

In contrast to BDD, neurocognitive performance in OCD patients has been well-documented (Fontenelle, Mendlowicz, Mattos, & Versiani, 2006; Kuelz, Hohagen, & Voderholzer, 2004), with executive dysfunction a common finding. Examples of findings suggesting executive dysfunction in OCD include decreased cognitive flexibility and set-shifting (Henry, 2006; Lawrence, 2006) and reduced engagement in decision-making and planning activities (Shin et al., 2004; van den Heuvel et al., 2005). In addition, there is a general consensus that impaired use of implicit organizational strategies may underlie the memory impairments observed in OCD (Greisberg & McKay, 2003; Maruff, Purcell, & Pantelis, 2002, Savage et al., 1999, 2000). For example, Purcell and colleagues (1998) conducted a detailed investigation of neuropsychological function in OCD using a battery of tests from the CANTAB with known sensitivity to frontal and subcortical systems. They reported deficits in spatial working memory and decreased motor speed on a computerized version of the Tower of London planning task, with the performance of OCD patients being similar to that of patients with frontal excisions (Purcell et al., 1998).

Evidence from the above studies strongly suggests executive dysfunction and frontal-striatal involvement in OCD (Saxena & Rauch, 2000), and raises the possibility of similar deficits with dysfunction in similar neuroanatomical pathways in BDD. However, to date there has been only one direct comparison of neurocognitive performance in BDD and OCD (Hanes, 1998) leaving open the possibility of important differences which may have significant implications for both etiology and treatment. In our study, although not a direct comparison, we aim to statistically compare the performance of BDD and OCD patients on cognitive tasks selected from the CANTAB. This was achieved by comparing data from the current BDD sample[1] with previously published data from an OCD sample (Purcell et al., 1998). Effect sizes for the performance of BDD ($n=14$) and OCD ($n=23$) participants compared to their respective controls were calculated and compared using both difference and equivalence testing. Only those measures common to both studies were available for comparison in this way. Based on results of previous studies of both BDD and OCD, we hypothesized that BDD and OCD participants would show an equivalent pattern of performance compared to their respective controls such that effect sizes between BDD and OCD will not be significantly different and the effect sizes will be equivalent (i.e., contained within the predetermined equivalence range).

2. Method

2.1. Participants

The BDD sample consisted of 14 DSM-IV BDD patients compared with 14 age-, education- and gender-matched healthy controls. The DSM-IV diagnosis for BDD was confirmed using the Body Dysmorphic Disorder Module (Phillips, 1994). The OCD sample comprised 23 DSM-IV OCD patients compared with 23 age-, education-, estimated IQ- and gender-matched healthy controls. Diagnosis of OCD was confirmed using the Anxiety Disorders Interview Schedule for DSM-IV (Brown, DiNardo, & Barlow, 1994). Both patient and control samples were screened for history of psychiatric illness, family history of mental illness, alcohol and/or substance abuse and head injury. Exclusion criteria for patients and controls were major medical or neurological illness and head injury. Additional exclusion criteria for control participants were current or past psychiatric illness and/or alcohol or substance abuse. For BDD patients, who have a high incidence of comorbidity, the presence of other Axis I disorders was not an exclusion criteria. For OCD participants from the Purcell et al. study (1998) the presence of other Axis I disorders (but not symptoms) was an exclusion criteria. For both studies, written voluntary informed consent was gained from participants; and the studies were approved by relevant ethics committees. More details of the participant groups and study designs for BDD and OCD samples can be found in the original studies (Dunai et al., 2010; Purcell et al., 1998), respectively.

2.2. CANTAB tasks

In both studies, all participants completed three executive function tasks and one visual memory test, selected from the CANTAB (Morris, Evendon, Sahakian, & Robbins 1987). More procedural details for each task can be found in Dunai et al. (2010). Briefly, *Spatial Span* (*SS*) measures the ability to remember a sequence of squares presented on a screen in short-term memory. Possible scores range from 0 to 9, with higher scores being superior. *Spatial Working Memory* (*SWM*) involves searching 3–8 boxes for tokens hidden inside, and generates three scores. A between-search error involves returning to a box in which a token has previously been found. A within-search error involves searching any box more than once during a search sequence. Finally, strategy scores reflect how often search sequences are initiated from the same box within a trial, with higher scores

[1] Some of the data collected using this sample of BDD participants has already been published in Dunai et al. (2010).