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Cognitive dysfunction in Body Dysmorphic Disorder: New implications for nosological systems & neurobiological models

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Abstract

Introduction. Body dysmorphic disorder (BDD) is a debilitating disorder, characterised by obsessions and compulsions relating specifically to perceived appearance, newly classified within the DSM-5 Obsessive-Compulsive and Related Disorders grouping. Until now, little research has been conducted into the cognitive profile of this disorder.

Methods. Participants with BDD (n=12) and participants without BDD (n=16) were tested using a computerised neurocognitive battery investigating attentional set-shifting (Intra/Extra Dimensional Set Shift Task), decision-making (Cambridge Gamble Task), motor response-inhibition (Stop-Signal Reaction Time Task) and affective processing (Affective Go-No Go Task). The groups were matched for age, IQ and education.

Results. In comparison to controls, patients with BDD showed significantly impaired attentional set shifting, abnormal decision-making, impaired response inhibition and greater omission and commission errors on the emotional processing task.

Conclusion. Despite the modest sample size, our results showed that individuals with BDD performed poorly compared to healthy controls on tests of cognitive flexibility, reward and motor impulsivity and affective processing. Results from separate studies in OCD patients suggest similar cognitive dysfunction. Therefore, these findings are consistent with the re-classification of BDD alongside OCD. These data also hint at additional areas of decision-making abnormalities that might contribute specifically to the psychopathology of BDD.

Key words

Body Dysmorphic Disorder; Neurocognitive, Affective, Cognitive flexibility

Introduction

Individuals with Body Dysmorphic Disorder (BDD) are troubled by intrusive thoughts that they have a bodily imperfection that is visibly unsightly [1]. In some cases, they have a minor physical flaw that would not be regarded as abnormal or noticeable by most people; in other cases, the defect is imaginary. They fear showing the 'imperfection' in public [2], leading to social avoidance and isolation. They spend considerable time ruminating about the perceived defect, and engage in time consuming checking, camouflaging and reassurance-seeking rituals [3].

BDD has been relatively neglected by research, perhaps in part due to the assumption that it is a rare condition. However, extant epidemiological data contradict this perspective. In a German sample of approximately 2500 individuals, selected to be representative of the general population, the point prevalence of BDD was estimated at 1.2-2.1% [4]. In a national household telephone survey conducted in approximately 2000 US citizens, the point prevalence was estimated at 2.4% [5]. Other studies, mostly conducted in college student samples, suggest a point prevalence rate of around 2.5% or greater [6-9]. In addition to being relatively common, BDD is associated with profound impairment in quality of life and everyday functioning [5]. Insight is frequently impaired and treatment-adherence is noted to be poor [10]. Furthermore, a prospective study conducted over four years in 185 subjects with BDD indicates that suicidality is a major concern. Each year, suicidal ideation occurred in more than 50% of individuals with BDD, 2.6% attempted suicide, and 0.3% completed suicide [11].

Anorexia nervosa (AN- restricting food) and bulimia nervosa (BN- binging and vomiting) are examples of eating disorders that are also associated with abnormal body image concerns and subjects with both these disorders have been shown to demonstrate a greater avoidance of their own image and negative self-evaluation than healthy controls [12]. Studies have demonstrated co-morbid and familial overlap between eating disorder, OCD and BDD [13,14]. In people with OCD, co-morbid

BDD has been reported in up to 37% of cases [15]. In patients with eating disorder (including both AN and BN), up to 45% have been found to show comorbid BDD [16,17]. Furthermore, in a seminal OCD family study, the first-degree relatives of OCD patients were at significantly elevated risk specifically for BDD, eating disorders grooming disorder and hypochondriasis, as compared to control relatives[14]. These findings are suggestive of a familial overlap between BDD and OCD on the one hand, a fact supported by previous reviews of age of onset, personality characteristics and course of illness[18] and similar cognitive deficits, such as set shifting[19] found in those with AN and BN.

In recognition of its nosological status as a compulsive disorder, the DSM-5 has moved BDD into the same category as obsessive compulsive disorder (OCD), under an expanded grouping of Obsessive Compulsive and Related Disorders American Psychiatric Association, [20]. Studies have demonstrated co-morbid and familial overlap between OCD and BDD [13]. In those with OCD, co-morbid BDD has been reported in up to 37% of cases [15]. Furthermore, in two seminal OCD family studies, the first-degree relatives of OCD probands were at significantly elevated risk for BDD, as well as trichotillomania, skin picking disorder and hypochondriasis, as compared to control relatives Bienvenu et al., [14,]Bienvenu et al., [21]. These findings are suggestive of a familial overlap between BDD and OCD on the one hand and between BDD and other putative obsessive compulsive and related disorders on the other, perhaps mediated by common genetic and/or cognitive predisposing factors.

Understanding of the neurobiology of BDD and related conditions may be informed by the use of cognitive tests that are dependent on the integrity of frontal lobe functioning. Various cognitive impairments have been identified in OCD using computerised paradigms from the Cambridge Neuropsychological Test Automated Battery (CANTAB www.cambridgecognition.com), including

in the domains of set-shifting (Extra-Dimensional Set-Shift (EDS)), inhibitory motor control (Stop-Signal Reaction Time (SSRT)), executive planning (Stockings of Cambridge (SOC) test), and affective bias toward negatively-valenced stimuli (for reviews see [22-24]).' These outcome measures can fractionate broad cognitive processes into constituent domains, and can be linked with different neural substrates[25]; they have been used in translational research across species[26,27], as well as in people with focal neurosurgical lesions, and in acute drug manipulations. This background validation is of value in interpreting new cognitive findings in conditions such as BDD. For some deficits (Extra-Dimensional Set-Shift, Stop-Signal Reaction Time, Stockings of Cambridge), similar cognitive dysfunction exists in unaffected first-degree relatives of patients with OCD and these therefore may represent predisposing or 'vulnerability' markers (e.g. [28-30]). The findings are broadly consistent with current neurobiological models of OCD, which implicate not only dysfunction within the classical orbitofrontal circuitry but also the dorsolateral prefrontal cortical circuitry, which incorporate these cortical regions but also subcortical nodes including the ventral and dorsal striatum [23,31].

There have been few published studies exploring neuropsychological function in BDD. Hanes and colleagues compared 14 subjects with BDD with 10 subjects with OCD and 24 controls, using a variety of non-computerised tests [32]. Both the BDD and OCD groups were similarly impaired, compared to controls, on tests of executive planning (Tower of London task) and colour-word interference (Stroop task), supporting the hypothesis that these two conditions are neurobiologically related. No significant deficits emerged in the BDD or OCD groups for category fluency and motor skill/speed on the Purdue Pegboard task, verbal learning on the Rey Auditory Verbal Learning task, or non-verbal learning/memory function on the Rey Complex Figures task (RCFT). In contrast, another study [33], again using non-computerised tests, identified impairment in non-verbal learning/mnemonic domains (Rey Complex Figures Task), along with verbal learning impairment

(California Verbal Learning Test), in 17 patients with BDD compared to 17 healthy controls. The authors postulated that the deficits were mediated by poor organisational strategy. Dunai and colleagues [34] additionally explored cognitive functioning in 14 patients with BDD versus 14 healthy controls, using selected computerised paradigms from the CANTAB. Patients with BDD were impaired on spatial working memory (Spatial Working Memory test) and executive planning (SOC test); findings similar to those reported separately for OCD [35]. In a more recent study, executive dysfunction was investigated in 14 BDD participants, 14 matched (age and gender) healthy controls, and 23 participants with OCD. Similarities were seen in the BDD and OCD groups in spatial span, spatial working memory, pattern recognition and spatial planning (SOC) tasks compared with healthy controls. However, those with BDD were found to have relatively greater deficits in executive functioning, on the accuracy measure of the SOC Task, than those with OCD and compared with healthy controls [36]. A recent study found similarities between BDD and OCD groups on measures of attention and memory, compared with controls [37]. Toh and colleagues used the Repeated Battery for the Assessment of Neuropsychological Status (RBANS), which assesses broad domains of memory, attention and visuospatial skills and benefits from the use of a healthy control group in the comparison of BDD and OCD samples. Their results showed some evidence of similarity in broad cognitive processing impairment that is unlikely to be disease specific. The study merits further exploration with tasks of greater neural specificity and robust evidence of impairment in OCD. The study offers further credibility to the idea that BDD falls within obsessive-compulsive spectrum disorders.

Based on the above limited evidence, the current study sought to explore specific aspects of cognitive functioning in BDD and healthy volunteers using relevant tests from the CANTAB. We focused on motor response inhibition (using the SSRT), cognitive flexibility (using the Intra-Extra Dimensional (IED) Set Shifting Task), and affective processing using the Affective Go/NoGo task

(AGN). These three cognitive domains are linked to behavioural inhibition and have not previously been investigated in BDD, but have been found to be impaired in non-comorbid OCD. For example, Kerwin et al., [38] found deficits in global and local processing, visual processing and cognitive flexibility in un-medicated individuals with BDD compared with non-clinical controls. These deficits were greater in those with more severe illness with poor insight and the finding merit further research to ascertain whether these areas serve to maintain BDD symptoms.

We also included a test of decision-making (Cambridge Gambling Task- CGT), which tests aspects of reward-based impulse control, and which has previously been observed to be intact in OCD [35], but which is impaired in patients with behavioural and substance addiction [24,39]. It was hypothesised that BDD would be associated with a similar cognitive profile to that previously reported in OCD: namely, significantly impaired response inhibition and set-shifting, evidence of affective bias with increased sensitivity to negatively-valenced cues, but intact decision-making.

Materials and Methods

Participants

BDD patients, aged between 18 and 65 years of age, were recruited from the specialist OCD/BDD outpatient clinic of one of the authors (NAF). All had a DSM-IV diagnosis of BDD, ascertained by a semi-structured diagnostic interview by a consultant psychiatrist and a detailed clinical assessment amplified by the Yale Brown Obsessive Compulsive Checklist and Scale for Body Dysmorphic Disorder (BDD-YBOCS) [40] to determine the degree of illness severity. In order to meet the inclusion criteria for the study, BDD was required to constitute the primary illness. All psychiatric

comorbidity (such as OCD) as documented in the case notes was recorded. Healthy controls were recruited from a non-treatment seeking population via the University of Hertfordshire email recruitment system. In order to meet the inclusion criteria for the study, healthy controls must not have a diagnosis of BDD or any other other primary illness. Illness symptomatology was measured using the BDD-YBOCS, Montgomery-Asberg Depression Rating Scale (MADRS) and the Hamilton Anxiety Scale (HAM-A).

Demographic analysis

Twelve individuals with BDD (mean duration of illness 133.5 months [11.13 years]) and 16 healthy individuals without BDD (controls) completed cognitive tasks and clinical questionnaires (Table 1). Importantly, the two groups did not differ significantly with regard to age, education and estimated IQ using the National Adult Reading Test [41] see Table 1).

Table one about here

Age, IQ and education-matched control participants without a diagnosis of BDD were recruited from the University of Hertfordshire. Control participants were approached via speaking to individuals on the University premises or via the university's SONA system (an online computerised system by which students can indicate their interest in participating in research studies). Participants were requested to be without Body Dysmorphic Disorder and were screened to exclude the presence of the disorder symptoms using the BDD-YBOCS using a cut off of >10. None of the participants in the control group scored more than 10 on this clinical rating scale.

Clinical measures

Severities of depression and anxiety symptoms were quantified in all participants using the Montgomery-Åsberg Depression Rating Scale [42] and the Hamilton Anxiety Scale [43].

Neuropsychological Measures

Participants completed the following paradigms from the CANTAB - see below. The tasks were administered in a fixed order (as below), in a quiet testing environment, supervised by a trained test administrator.

The intra/extra dimensional set-shift task (IED- http://www.cambridgecognition.com/tests/intra-extra-dimensional-set-shift-ied)

This is a nine-stage visual discrimination task and measures cognitive flexibility [44] in which two stimuli are presented at a time and participants ascertain, by computerised feedback, which of the stimuli is correct, and thus, the 'rule' of the game. Following six consecutive indications of the correct stimulus the 'rule' alters. The extra dimensional shift (EDS) stage of the task is crucial for determining divergent thinking deficits as the participant is required to shift their attentional focus from the previously relevant stimulus dimension to a previously irrelevant stimulus dimension. Such set-shifting depends on the ventrolateral prefrontal cortex [45]. The outcome measures of interest on this task include the total number of errors and total number of stages successfully completed.

The Cambridge Gambling Task (CGT- http://www.cambridgecognition.com/tests/cambridge-gambling-task-cgt)

This task assesses dissociable aspects of decision-making. Participants are asked to accumulate as many points as they can by making gambles across a range of different winning probabilities. Each trial has differing proportions of red and blue boxes from which participants are asked to place a bet

on the location of a yellow token based on their confidence in their choice. The bet amount either increases incrementally (5%, 25%, 50%, 75%, 95% of total collected points) or decreases (reverse order) over time. Outcome measures include mean percentage of points gambled (total proportion of overall bets), quality of decision-making (this measures rational decision making and is measured by the calculating the proportion of trials where the participant chose the more likely outcome (box colour), risk taking (the mean proportion of points bet on trials where the most likely outcome was chosen), deliberation time (how long it took to decide on which bet to choose) and delay aversion; this is measured as the tendency for participants to bet larger amounts due to an unwillingness, or inability, to wait for bets to decrease on trials where bet amounts are presented in descending order compared with when bets are presented in ascending order.

The Stop Signal Task (SST- http://www.cambridgecognition.com/tests/stop-signal-task-sst)

This is a measure of pre-potent motor inhibition. Participants are required to respond rapidly to left or right oriented arrows, presented on a blank screen. When an audible sound emits (the 'stop-signal') from the task screen, participants are required to inhibit their response for that arrow and their degree of success is measured. Over the course of the test, the time between the presentation of the 'go' stimulus and the 'stop-signal' varies using a tracking algorithm. The main outcome measure on the task is the Stop-Signal Reaction Time (SSRT), which is an estimate of the time taken by the given individual to stop or suppress a response that would ordinarily be undertaken; longer SSRTs equate to poorer motor response inhibition, or greater 'motor impulsivity'. The other outcome measure of interest is the median reaction time for 'go' trials; a generic measure of response speed not relating to inhibitory control.

The Affective Go/NoGo (AGN- http://www.cambridgecognition.com/tests/affective-go-no-go-agn)

This task assesses mood processing bias. A series of positive and negative words are presented on screen. The participant is required to respond to predetermined 'target' words by pressing a key pad when they see a target word. This target word will be 'positive' or 'negative' in valence. Other non-affective words are considered 'distractor' words and participants are required to avoid responding to these words and to only respond to the 'target' word. The outcome variables of interest include the mean correct latency representing the length of time each participant takes to respond to target words, as well as the total number of commission errors (pressing for a positive target word when it is a negative one or vice-versa) and omission errors (failing to respond when one should have done so).

Statistical analysis

Between-group differences were investigated by conducting a multivariate Analysis of Variance (MANOVA) using IBM SPSS. Further exploratory analysis in SPSS included a test of covariance using anxiety (Ham-A) and depression (MADRS) scores as covariates. This being an exploratory study, statistical significance was defined as $p<0.05$ uncorrected.

Results

Clinical analysis

The BDD group showed a range of symptom-severity ranging from mild to moderately severe (BDD-YBOCS total range 7-24). The mean BDD Y-BOCS was 13.25 (SD 4.88), representing mild BDD. Control BDD-YBOCS scores ranged from 0-10 with an average score of 2.38 (SD= 3.40). None of the 16 control participants were taking prescribed medication, while all 12 of the BDD participants were taking prescribed medication (2 citalopram, 6 escitalopram, 3 fluvoxamine and 1 sertraline). Nine of the twelve BDD patients expressed symptoms of comorbid OCD. Two of the

nine were also diagnosed with comorbid social anxiety disorder and one patient was diagnosed with gender dysphoria (DSM-5), previously Gender Identity Disorder American Psychiatric Association, [20]. These diagnoses were based on clinical assessments by a consultant psychiatrist. These details were not measured at the time of testing, but were taken from patient case notes and discussions with the treating psychiatrist. Although both groups showed low levels of anxiety and depressive symptomatology, the BDD group showed significantly greater severity of symptoms of depression (MADRS, p=.01), and anxiety (Ham A p=.03) – see Table 2. Fifty per cent of the participants with BDD scored very low on the MADRS ('normal or symptom absent' with a MADRS score of less than 7 [52]. The majority of the remainder (n=5) scored within the 'mild depression' with scores between 7 and 19, and 1 participant scored 24 representing 'moderate depression' [52]

Table 2 about here

Neurocognitive analysis.

A Multivariate Analysis of Variance (MANOVA) was conducted in order to ascertain differences between the BDD and control groups. The MANOVA revealed significant differences between groups overall $F(1,26)= 6.89$, $p= .01$)

Table 3 about here

Intra/Extra Dimensional set shift task (IED)

The BDD group made significantly more total errors (adjusted) on the task versus controls. These errors were specifically seen at the extra-dimensional shift (EDS) stage (stage 8). All participants in both groups passed stages 1-7; however, only 50% (n=6) of the BDD group passed the EDS stage

while all control participants (n=16) passed the EDS stage (see Figure 1). No notable changes to the significance of each variable were seen when results were co-varied for anxiety and depression.

Figure 1 about here

Stop Signal Task (SST)

The BDD group showed significantly longer stop-signal reaction times (SSRTs) than the controls. General psychomotor speed (measured as median 'go' reaction times) did not differ significantly between the groups. No notable changes to the significance of each variable were seen when results were co-varied for anxiety and depression.

Cambridge Gambling Task (CGT)

The BDD group showed significantly more delay aversion than controls. However, the BDD group gambled a significantly smaller proportion of total points overall. Between-group differences were also found in risk taking (measured by the proportion of total points bet over all trials), with the BDD group showing a significantly lower incidence of risk taking than controls. Groups did not differ significantly in terms of the proportion of rational decisions made overall. No significant differences were found with regard to the deliberation time when making bets. No notable changes to the significance of each variable were seen when results were co-varied for anxiety and depression.

Affective Go/ No-Go (AGN)

<u>Reaction time</u>

Analysis of variance showed that the BDD group were slower to respond correctly to presented words than the controls. Sub-analysis indicated that individuals with BDD took significantly longer to respond to positive words when compared to controls. The groups did not differ significantly for negative words.

Commissions

ANOVA showed that the number of commission errors differed significantly between the groups, due to higher errors in patients than controls overall. Sub-analysis indicated that there were significantly more commission errors in those with BDD than controls for positively and negatively valenced words but not neutral words.

Omissions

More non-responses (omissions) were seen in the BDD group compared with controls overall. When exploring emotional valence, the BDD group made statistically more omissions for positively valenced words, and for negatively valenced words; but not neutral words. No notable changes to the significance of each variable were seen when any of the AGN results were co-varied for anxiety and depression.

Discussion

This study contributes to the body of research documenting impaired neurocognitive performance in BDD. Differences were seen between individuals with and without BDD and cognitive results generally appeared to be unaffected by severity of mood and anxiety symptoms.

Cognitive Inflexibility: The BDD group made significantly more errors on the IED task, with a significantly higher error rate at stage 8 of the task (the extra-dimensional shift stage- EDS). Only 50% of the BDD group progressed to stage 8 (EDS). Results from the IED task indicates significant attentional (or cognitive) inflexibility within the BDD group. A number of studies have found

deficits in cognitive flexibility in OCD patients [28,35,53,54], with the deficits appearing exclusively at the extra-dimensional stage (EDS), as was the case in the current study. The neurobiology of attentional shift flexibility has been the subject of translational study. Research into rodents [55], primates [56-58] and humans [45,48,59] implicate the ventro-lateral prefrontal cortex (or functionally homologous regions) as being required for intact cognitive flexibility.

The finding of cognitive inflexibility in the BDD group converges with published findings for OCD[35] and with the clinical presentation of the disorder – specifically with the performance of compulsive (repetitive, urge-driven) behaviour. Individuals with BDD engage compulsively in thoughts or behaviours related to appearance and find difficulty diverting attention to non-image related thoughts or 'purposeful' forms of activity. However, we cannot exclude the possibility that the cognitive inflexibility found in the BDD group in this study is attributable to the presence of comorbid OCD, which was present in 9 of the participants. Indeed, significant differences were seen for completed stage errors, when comparing the participants in the BDD group who had a diagnosis of OCD with those who did not, suggesting that the presence of OCD may have had an influence upon cognitive flexibility. This may be clinically relevant, in that people with BDD comorbid with OCD may have a more rigid response style, which could impede ability to adjust behaviors in day-to-day life, and to engage with psychological treatments.

Decision Making: The Cambridge Gambling Task (CGT) is a measure of decision-making abilities with the advantage of assessing different aspects of decision-making separately [60-62]. Individuals with OCD have been found to be unimpaired on the CGT [28], though abnormal performance on the task versus controls can be elicited in OCD with acute serotonergic challenge (Lochner et al., submitted). However, our results showed abnormal decision-making in a BDD sample. A higher incidence of delay aversion was seen in BDD patients (i.e. participants were unwilling to wait for bets to increase/decrease) suggesting an increased degree of impatience (decision-making

impulsivity). Hollander and Wong [63], in their investigation of gambling disorder and its associations with BDD, found that individuals with BDD showed an increased tendency for gambling.

Motor Impulsivity: Significant differences in motor impulsivity were found between BDD patients and controls on the Stop Signal Reaction Time (SSRT) task. Impaired motor response inhibition has been proposed to represent an endophenotype of OCD, as studies have found that unaffected relatives are also impaired on the SSRT [28]. Performance on the SSRT is dependent on an intact right inferior frontal gyrus [64,65]. A number of further brain areas have been implicated in impaired response inhibition in OCD [66] including the orbitofrontal cortex, anterior cingulate, parietal cortex, caudate-putamen and cerebellum, suggesting involvement of circuits within and outside the orbitofrontal –striatal -thalamic loop.

Overall Impulse Control: Our data suggest that participants with BDD exhibit signs of both decision-making impulsivity and motor impulsivity. These findings align with the clinical phenomenology; many of the characteristic behavioural symptoms of BDD, e.g. being unable to resist the urge to undertake cosmetic, and even 'do it yourself (DIY)' surgery to 'correct' perceived flaws, may be construed as poor impulse control. Indeed Veale [67] reported that of 25 patients he interviewed, nine (36%) had carried out their own DIY surgery in an attempt to dramatically alter their appearance. In addition, suicidal acts are common in patients with BDD. A large prospective study of suicide showed that in 185 BDD participants followed up over 4 years, for each year spent in the study an average of 57.8% reported suicidal urges, 2.6% attempted suicide and 0.3% (2 people) completed suicide.

While 'impulsivity' implies a predisposition toward performing rapid and unplanned reactions to stimuli and 'compulsivity' relates to the urge-driven performance of repetitive unwanted acts, both domains can be considered to represent a dysfunction in impulse control [68] and both may be represented in BDD. Separate cortico-striatal circuits are thought to sub-serve impulsivity (ventral)

and compulsivity (dorsal)[23]. Hyperactivity of the striatal circuit (generation of activity) and hypoactivity of the prefrontal circuit (inhibition) may represent a common mechanism underpinning impulse control deficits in a range of obsessive-compulsive disorders such as OCD and BDD[23].

Affective Processing: On the AGN task, the BDD group showed a longer reaction time between the presentation of a target word and a correct response i.e. they took longer to respond to the target word, when a correct answer was given. In addition, individuals with BDD showed a higher instance of errors characterised by responding to distracter stimuli (non-target words) and also a higher instance of non-response on target stimuli compared with controls. These data mirror previous findings for OCD, in which disorder inappropriate motor responses to non-target stimuli were observed in comparison to those seen in healthy controls [69,70]. Findings in OCD studies have been specific for word valance, with negative words being more difficult to forget in OCD groups a potential suggestion of incorrect processing of negative words [71] but additional findings suggest that the type of word most difficult to forget in OCD groups is the type associated with their current OCD presentation- positive or negative [72]. In the current study, individuals with BDD showed a longer reaction time, more errors and non-responses for positive and negative target words, but not neutral target words. Previous OCD research revealed elevated commission errors for neutral words, compared with happy and sad words, in patients in one study [73]; while another study found more omission errors for sad target words in OCD[46]. One interpretation for the current results in BDD patients is that the disorder is associated with more generalized dysregulation of emotional processing circuitry, with a global untoward impact of emotional information on attentional processing. Thus, the presentation of emotionally valenced stimuli (whether positive or negative) results in performance decrements that generalize across both commission and omission errors, with neutral stimuli not having such a pronounced effect.

Also, increased errors in the BDD group to positive and negative target and distractor words could result from individuals with BDD being unusually sensitive to emotional cues, i.e. stimuli that have some meaning to the BDD disorder. These could be negative words such as 'ugly' or even positive words such as 'attractive'. Our findings revealed differences based on word valence, and not on neutral word trials, suggesting that the symptoms of BDD may rely on an inherent focus on *both* negatives and positives about appearance. Additionally, this bias within the BDD condition may result from cognitive inflexibility, in that individuals with BDD may become 'stuck' in a routine of thinking about positive and negative aspects of themselves.

The development of self - image and the role of appearance is thought to be influenced by environmental factors, including significant life events and shaped by memory [74-76]. Individuals with BDD commonly report instances of bullying and teasing, potentially increasing their propensity for negative perception of themselves and of specific body parts [76,77]. The finding of attentional bias toward affectively valenced words is consistent with this literature and may help explain how such experiences become overvalued and may result in an obsessive preoccupation with body image. Few studies have tested attention in BDD. Our findings suggest future research investigating the effect of BDD on attention to environmental cues, and the consequent impact on psychosocial function, is desirable.

Limitations: Our modest BDD sample may have had reduced statistical power to detect other potential differences of relevance. Other BDD studies of this type have also reported a small sample size and it may be that recruitment to BDD studies is particularly challenging (anecdotally, our perception was that BDD patients seemed reluctant to engage in research that focused attention on themselves). Nonetheless, replication in larger samples is required. OCD and affective comorbidity could have had a confounding influence on the findings, considering 75% of our BDD group had

comorbid OCD and 50% comorbid depressive symptomatology. On the other hand, the BDD cases were drawn from a well-defined clinical cohort, BDD was recognised by the patients and their clinicians as the primary disorder and constituted the focus for clinical treatment. BDD in clinical cohorts is almost always comorbid with disorders such as OCD and depression [10,78] and by including patients with relevant comorbidity, the results may be generalised to BDD patients seen in the clinical setting. While the relatively low magnitude of BDD-YBOCS scores in some of the BDD group participants may pose a limitation in clearly differentiating groups, this finding may be attributed to the effect of clinical treatment. This, in itself, does not invalidate our findings and may relate to trait, rather than state, illness. A clinical control was not used as a comparison to the BDD and healthy control groups. In future studies, it would be beneficial to compare those with a diagnosis of BDD with an OCD control group in order to further investigate cognitive correlates between these two clinical presentations and potentially further support the presence of BDD on the obsessive compulsive spectrum. Additional benefit would be gained from performing a full structured diagnostic screen on participants in order to gain a better picture of overall illness profile, or absence of illness.

Recognition of the influence that medication may have had on potentially changing the neurocognitive performance of BDD participants should be noted as all 12 of the BDD participants were taking medication (2 citalopram, 6 escitalopram, 3 fluvoxamine and 1 sertraline) at the time of testing. Certainly serotonin is known to play an important role in decision-making and emotional processing. Future research could be extended to investigate unaffected relatives, so as to avoid potential medication-related confounds. Research should also explore the functional impact of specific aspects of cognitive impairment on daily life, treatment-adherence, and suicidal activity.

Conclusion

Patients with BDD were impaired compared to healthy controls on tests of cognitive flexibility, reward and motor impulsivity and affective processing. Results from previous studies in the OCD population show similar deficits in cognitive flexibility and motor impulsivity; therefore our findings are consistent with the re-classification of BDD with OCD. However, the current study suggests that BDD may be characterized by additional abnormalities in domains of decision-making and emotional processing that differ from previous findings in OCD. While the study used a modest sample size and comorbid OCD may confound results, future work should explore the impact of these abnormalities on everyday functioning, ability to engage successfully with treatment and suicidality.

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Tables & Figures

Table 1. Demographic analysis; BDD and control groups

	BDD (n=12)		Control (n=16)			
Variable	Mean	SD	Mean	SD	F	p
Age (years)	30.08	(8.92)	35.80	(12.10)	1.87	.18
Education (years)	14.08	(1.88)	14.41	(1.99)	0.23	.64
NART (IQ)	113.80	(2.95)	115.00	(3.34)	0.22	.64

Table 2. Clinical measures

	BDD (n=12)		Control (n=16)			
	Mean	**SD**	**Mean**	**SD**	**F**	**p**
HAM-A	8.08	(6.75)	3.94	(3.04)	4.76	.03*
MADRS	7.50	(5.98)	2.50	(4.29)	6.66	.01*
BDD-YBOCS	13.25	(4.88)	2.38	(3.40)	48.40	<.001*

Note: HAM-A: Hamilton Anxiety Scale, MADRS: Montgomery-Åsberg Depression Rating Scale, BDD-YBOCS: Yale Brown Obsessive Compulsive Scale for Body Dysmorphic Disorder

Table 3. Neurocognitive test performance

		BDD		Control				
		Mean	**SD**	**Mean**	**SD**	**F**	**p**	**Cohen's d**
IED	Stages completed	8.00	1.04	8.94	.25	8.87	.007*	1.21
	Total Errors	26.75	10.95	13.18	4.98	13.00	.001*	1.47
	EDS Errors	17.25	12.10	4.75	3.92	10.56	.003*	1.32
CGT	Delay Aversion	.47	.17	.28	.19	5.22	.03*	0.94
	Deliberation Time (msec)	1827.05	741.28	2133.09	533.32	1.84	.20	0.55
	Overall proportion of bet	.50	.05	.76	.09	63.16	<.001*	3.24
	Risk taking	.56	.06	.69	.14	1.68	.04*	1.25
	Quality of DM	.89	.15	.87	.24	.45	.51	0.27
SST	Mean Reaction Time	477.19	130.99	465.57	76.36	.02	.90	0.00
	Stop Signal Reaction Time	182.64	74.84	137.81	52.62	4.66	.04*	0.87
AGN	Mean Correct Latency	535.00	82.31	473.22	60.78	4.85	.03*	0.90
	POSITIVE	545.46	90.80	418.74	49.73	19.76	<.001*	1.81
	Total Omissions	6.92	3.40	1.19	1.64	24.44	<.001*	2.00
	POSITIVE	1.33	1.37	.13	.34	7.11	.01*	1.09
	NEGATIVE	1.66	1.49	.19	.40	10.90	.003*	1.34
	NEUTRAL	.00	.00	.06	.25	.26	.61	0.20
	Total Commissions	10.17	7.66	4.62	2.09	5.86	.02*	0.96
	POSITIVE	3.75	3.13	.75	.85	10.78	.003*	1.34
	NEGATIVE	2.16	1.69	1.12	.72	3.74	.06	0.79
	NEUTRAL	.19	.54	.10	.42	.50	.49	0.29

*Note: * denotes a statistically significant result. IED (Intra/Extra Dimensional shift task), CGT (Cambridge Gambling Task), SST (Stop Signal Task), AGN (Affective Go/NoGo task)*

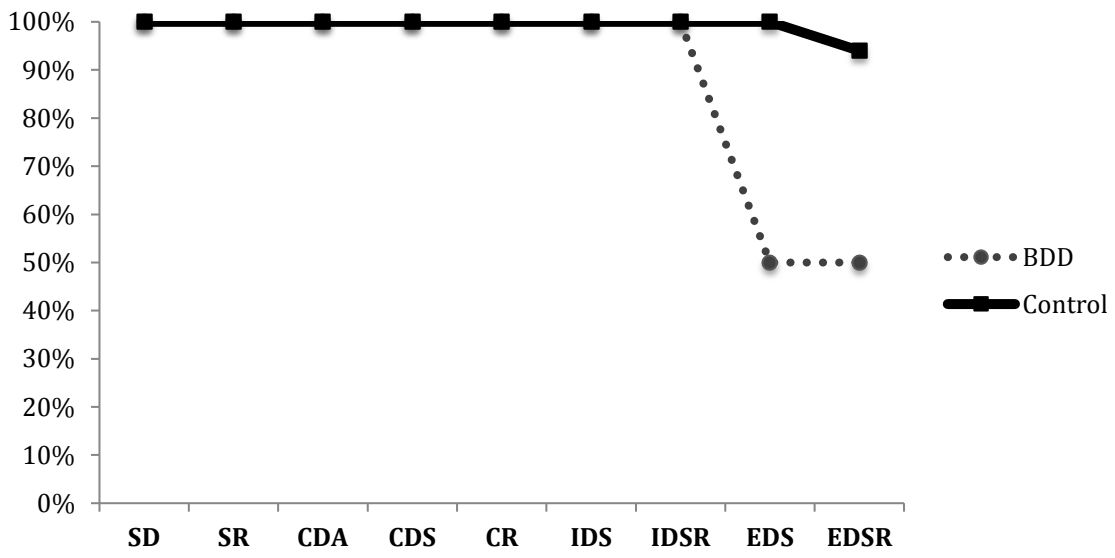

Figure 1. Percentage of BDD and control participants passing each stage on the IDED task

Note: SD = simple discrimination; SR = simple reversal; CDA = compound discrimination adjacent; CDS = compound discrimination superimposed; CR = compound reversal; ids = intra-dimensional shift; IDSR = intra-dimensional shift reversal; EDS = extra-dimensional shift; EDSR = extra-dimensional shift reversal