What the Cognitive Deficits in Body Dysmorphic Disorder Tell Us about the Underlying Neurobiology: An Investigation of Three Cases

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Body Dysmorphic Disorder (BDD) has only recently received attention in current research. As such, little is understood with regards to the underlying cognitive impairments and neurobiological substrates implicated in this disorder. The current report provides first, a review of the background of BDD; second, a description of the clinical features of three BDD cases; and last, the outcomes from a cognitive assessment administered to three BDD cases. The cognitive assessment included (a) executive function, (b) facial affect perception, and (c) general social cognition. All three BDD cases illustrated many of the disorder’s clinical features such as excessive disproportionate concerns about their appearance, repetitive (checking) behaviours, and camouflaging. Further, they experience significant distress and impairment in social, occupational, and other important areas of functioning. They also demonstrated poor self-esteem, greater self-ambivalence, and more pronounced delusional thinking than a comparison group of ten healthy controls. The cognitive assessment demonstrated deficits in executive functioning and facial affect perception, but not in general social cognition. The findings implicate frontal-amygdala and temporal-parietal pathology in BDD although neuroimaging studies are needed to confirm this speculation. The implications of our findings for the treatment of BDD are discussed.
Body Dysmorphic Disorder (BDD) is a preoccupation with an imagined deficit or a slight physical anomaly in appearance; which causes significant distress or impairment in social, occupational, or other important areas of functioning (DSM-IV; American Psychiatric Association, 1994). The most common bodily related concerns involve facial features such as the nose, skin, hair, and even blemishes, pimples, veins or arteries; however, other features such as thighs, breasts, genitals, abdomen, hands and feet are often the focus of their preoccupation (see Phillips, 1998; Phillips, McElroy, Kec, Pope, & Hudson, 1993). Many BDD individuals carry out time-consuming and obsessive behaviors, such as frequent mirror checking, camouflageing or hiding their defect by wearing hats or wigs, and excessive grooming with make-up and/or hair products to make the defect seem less noticeable to others (Buhlmann & Wilhelm, 2004; Phillips et al., 1993; Veale, 2004; Veale & Riley, 2001; Wilson & Arpey, 2004). Furthermore, BDD individuals constantly fear negative evaluation and strongly believe that others take special notice of their appearance (Buhlmann, McNally, Wilhelm, & Florin, 2002; Hollander, Neville, Frenkel, Josephson, & Leibowitz, 1992). It has commonly been reported that the prevalence of BDD in the general population is around 1% (Faravelli et al., 1997; Otto, Wilhelm, Cohen, & Harlow, 2001). However, in more targeted settings such as dermatological and plastic surgery clinics, the rates are much higher, ranging for 6 to 15% (Altamura, Paluello, Mundo, Medda, & Mannu, 2001; Castle, Phillips, & Dufresne, 2004; Phillips, Dufresne, Wilkel, & Victorrio, 2000; Sarwer & Didie, 2002). BDD individuals are ashamed of their appearance and are more likely to seek help from cosmetic surgeons and dermatologists than from mental health professionals (Phillips, Hollander, Rasmussen, & Aronowitz, 1997; Veale & Lambrou, 2002). Not surprisingly, BDD is routinely underdiagnosed due to its secretive nature, embarrassment, and lack of awareness.

According to DSM-IV (APA, 1994), BDD is currently classified as a somatoform disorder. This is, however, an area of great dispute with a number of arguments for classifying it as a symptom of more pervasive disorders such as obsessive-compulsive disorder (Castle & Rossell, 2006; Phillips & Castle, 2002), social phobia (Phillips, 1991; Phillips et al., 1993), or eating disorders such as anorexia nervosa (Hollander, 1993). The high rates of similarity and/or comorbidity between BDD and a range of psychiatric disorders in terms of mood and anxiety levels, substance abuse, and eating and personality behaviors, add to the difficulty in defining and thus recognizing BDD individuals.

The aetiology and neurobiology of BDD is unknown, although responses to medication with effects on serotonergic transmission, have suggested a possible serotonergic basis for this disorder (for reviews see Jefferys & Castle, 2003; Neziroglu & Khemlani-Patel, 2002; Phillips et al., 1993). Some authors have argued that this implicates a prefrontal pathophysiology (Neziroglu & Khemlani-Patel, 2002). Only a few neuroimaging studies have been conducted in BDD (Carey, Seedat, Warwick, Van Heerden, & Stein, 2004; Gabbay et al., 2003; Rauch et al., 2003), with findings pointing to a role for fronto-striatal and temporal-parietal regions. Rauch et al. (2003) used morphometric magnetic resonance imaging (MRI) to show, in comparison with a healthy control group, that BDD participants had a leftward shift in caudate nucleus asymmetry and increased white matter volume. Using a more advanced image technique (i.e., single photon emission computed tomography or SPECT imaging) Carey et al. (2004) demonstrated decreased perfusion in the left frontal and right striatal areas, the parieto-occipital and anterior cingulate cortices. More recently, in a case study, Gabbay et al. (2003) reported, using functional magnetic resonance imaging...
FROM COGNITION TO NEUROBIOLOGY IN BDD

(fMRI), fronto-temporal region atrophy and suggested this region, particularly the
temporal lobe, may be responsible for the obsessive-compulsive behaviors implicated
in BDD. These findings suggest that BDD may be mediated by widespread neuro-
circuitry. However, the small sample sizes, comorbidity, and the variety of imaging
techniques used, together with the absence of replications, limit the conclusions from
neuroimaging studies.

The current goal of cognitive neuroscience is to understand brain systems in-
volved in the psychopathology of mental disorders. To our knowledge, there have
been no previous cognitive neuroimaging studies completed in BDD. The majority
of the BDD literature thus far has focused on the phenomenology of the disorder
(Carey et al., 2004; Castle et al., 2004; Castle & Rossell, 2006; Gabbay et al., 2003;
Phillips et al., 1993). However, several behavioral studies have investigated cognitive
functioning in BDD (Buhlmann, McNally et al., 2002; Buhlmann, McNally, Etcoff,
Tüschen-Caffier, & Wilhelm, 2004; Buhlmann, Etcoff, & Wilhelm, 2006; Deckers-
bach et al., 2000; Hanes, 1998; Laniti, 2005; Osman, Cooper, Hackmann, & Veale,
2004; Yaryura-Tobias, Neziroglu, & Torres-Gallegos, 2002). These latter studies pro-
vide evidence of cognitive impairments in areas including executive function, memory,
facial affect perception, and attributional bias or self-discrepancy. Some authors have
made pathophysiological suggestions based on cognitive behavioral outcomes. For
example, Hanes (1998) reported poor performance in BDD on executive function
tasks (Stroop task and Tower of London task), similar to performance of OCD pa-
tients, and therefore suggested that the pathophysiology of BDD may implicate the
prefrontal region, a region well established to be implicated in OCD (Geus, Denys,
Sitskoom & Westenberg, 2007; Purcell, Maruff, Kyrios, & Pantelis, 1998; Saxena,
Brodly, Schwartz, & Baxter, 1998). Deckersbach et al. (2000) reported that BDD in-
dividuals differed significantly from controls on verbal and nonverbal learning and
memory indices (Rey Complex Figure Test and the California Verbal Learning Test).
The differences in free recall were mediated by deficits in the organizational strategies
of the BDD cohort and, therefore, it may be that BDD individuals recall details rather
than larger organizational design features (Sachs, Steger-Wuchse, Kryspin-Exner, Gur,
& Katschnig, 2004). This latter memory deficits observed, and which is also similar
in OCD (e.g., Savage et al., 1999), suggest the involvement of other brain regions
including medial temporal cortex in addition to the fronto-striatal implications pro-
posed earlier.

In addition, the clinical presentation of BDD, such as the egosyntonic concerns
about physical appearance (Frare, Perugi, Ruffolo, & Toni, 2004), suggests that per-
ception and/or social processing abnormalities may be the key cognitive deficits of this
disorder. In fact, Veale, Kinderman, Riley, and Lambrou (2003) showed that BDD
individuals have an unrealistic idea as to how they should look, being more concerned
with a failure to achieve their own aesthetic standard than with the perceived ideals of
others. Only a few studies to date have directly examined general perceptual and social
cognitive abilities in BDD (Buhlmann, McNally, et al., 2002; Buhlmann, Wilhelm et
al., 2004, 2006; Yaryura-Tobias et al., 2002). Yaryura-Tobias et al. (2002) established that BDD and OCD patients were impaired at detecting body
distortions of their computerized facial image and were more likely to modify their
facial features (about 50% of cases) compared to the control group which showed no
modifications. Buhlmann et al. (2004; Buhlmann, McNally et al., 2002) examined
social processing in terms of facial emotion perception and found that BDD indi-
viduals have problems recognizing facial emotions. The BDD individuals in particular
misinterpreted “disgusted” as being “angry,” indicating a strong recognition bias for angry facial expressions. In another study, Buhlmann, Wilhelm et al. (2002) showed that BDD individuals exhibit a negative interpretive bias for body-related scenarios, social scenarios, and more general scenarios, whereas OCD individuals show such bias only for general scenarios. More recently, Buhlmann et al. (2006) showed an angry bias specific to self-referent situations (i.e., if they imagined themselves being in that situation) rather than to other-referent situations (i.e., if they imagined someone else being in that situation). These perceptual impairments evident in BDD individuals, and which particularly involve body- and face-related concepts as well as affect, may suggest additional brain areas are implicated in BDD. Current research investigating emotional recognition of facial stimuli in healthy controls has shown an activated network of subcortical and cortical regions including the amygdala (Iidaka et al., 2001) and the right limbic-frontal region including the insula cortex (Gorno-Tempini et al., 2001). Areas of the visual cortex including the fusiform (face) area (FFA; Halgren, Raji, Marinkovic, Jousmaki, & Hari, 2000; Kanwisher, McDermou, & Chun, 1997; Kanwisher, Stanley, & Harris, 1999; Puce, Allison, Gore, & McCarthy, 1995; Tong, Nakayama, Moscovitch, Weinrib, & Kanwisher, 2000), and the lateral occipital cortex also know as the extrastriate body area (EBA; Downing, Jiang, Shuman, & Kanwisher, 2001; Saxe, Jamal, & Powell, 2006), may also be implicated in BDD. In particular, Downing et al. (2001) suggested that the EBA is a specialized system involved in processing of visual appearance of the human body. Therefore, BDD may be associated with a wide-spread neurocircuitry, ranging for frontal to occipito-parietal cortical regions as well as subcortical (amygdala) regions.

The neurobiological and cognitive evidence reviewed, assist in determining a more accurate neuropsychiatric formulation of BDD. There remains, however, little understanding of the exact neurobiological and neuropsychological underpinnings of BDD, emphasizing the need for further research. To date, the cognitive evidence suggests executive functioning and facial perception deficits in BDD. More general social cognition, as an extension of face processing, has not been investigated and may be the key deficit underlying cognitive impairments in BDD.

In the present report, we describe three BDD cases to provide an overview of the disorder’s clinical features. Second, we examined cognitive functioning by targeting two known areas of impairment in BDD individuals such as (1) executive function and (2) facial affect recognition, and further extend face processing investigations by examining (3) more general social cognitive abilities. From this cognitive data, we discuss the putative neural substrates of BDD.

CASE 1*

AM is a 58-year-old female who is married and is currently a housewife. She finished school at the age of 17. Over the years, she was employed on a full-time level for 6 years, but has been causal for the last 25 years. She was diagnosed with BDD due to her strong belief that she has a “very hairy neck.” For this reason, she has been to multiple electrolysis sessions in order to enhance her appearance. She constantly seeks reassurance from others by asking them about the amount of hair on her neck. At the

*Personal details have been changed to protect the privacy of the three individuals.
initial interview, AM denied having an excessive preoccupation with her appearance, however, she admitted to constantly checking herself in mirrors, with the time she spends in front of mirrors averaging around 3 hours per day. AM also has an associated psychopathology of depression. There are no signs of psychiatric illness in the family and her demographic record has suggested that substance or alcohol abuse has not been part of her life. Her treatment response to medication (300 mg sertraline [Zoloft]; 25 mg quetiapine fumarate [Seroquel]; 100 mg spironolactone [Aldactone]) at the time of testing was reported as excellent. In addition, she is currently also taking menopausal medication (i.e., 40 mg black cohosh extract [Remifemin]).

CASE 2

LH is a 27-year-old male who has been diagnosed with BDD since the age of 13. He is separated from his wife, and thus is living alone. He completed his secondary education and attempted 2 years of tertiary education in a course of Bachelor of Arts. His former employment involved being a chef and an education officer, however, he is currently unemployed and on a disability pension. His body-related concerns include a belief that his torso is too fat, his head too big and his stomach is misshapen. The associated behaviors include mirror checking of 15-minute duration, 20 times per day. He constantly rubs and pats the particular body parts and wears baggy clothes to make them less noticeable to others. On the day of interviewing, he was severely tense and socially anxious. He admitted that he prefers to avoid social situations and, therefore, feels isolated. He sees his preoccupations as “irrational” but is unable to resist the urges. Other symptoms include depression, and some obsessive-compulsive symptoms. His family history of psychiatric illness includes depression in one cousin and maternal grandmother. Alcohol does not seem to be part of LH’s life, however, he admitted to consuming a bottle of cognac in one session approximately three years ago. LH has not been involved in any substance use in the last 2½ years, however, he admitted using substances before then on different levels ranging from experimentally (amphetamine, morphine), occasionally (ecstasy, acid/mushies) to gross excess (sleeping pills, tranquilizers, and marijuana). His treatment response to medication (300 mg venlafaxine hydrochloride [Efexor]; 600 mg quetiapine [Seroquel]; and 20 mg escitalopram oxalate [Lexapro]) seems to be modest.

CASE 3

SD is a 16-year-old female who is currently finishing her secondary education. She is living at home with her parents and brother. SD was diagnosed with BDD at the age of 11 years and has a strong concern about the appearance of her skin. Ritualistic behaviors include picking at her skin and frequently checking herself in mirrors take up 1 to 3 hours of her day. She constantly tries to hide her skin by using cover-up techniques such as wearing excessive make-up and long-sleeved tops. She seems to have partial insight into her disorder. Other psychopathology includes symptoms of obsessive-compulsive disorder. There is a strong family history of psychiatric illness involving her parents, grandparents, aunts, uncle, and brother; however, the nature of these illnesses is unavailable. At the time of testing, SD’s psychiatrist reported her
treatment response to medication (80mg fluoxetine [Lovan]; and 50mg quetiapine fumarate [Seroquel]), to be moderate.

Method

The DSM diagnosis for BDD was confirmed in all three BDD cases using the Body Dysmorphic Disorder Diagnostic Module (BDD-DM; Phillips et al., 1997). Data from the BDD cases was compared to ten healthy controls. The exclusion criteria for all participants included: any neurological disorder, insufficient conversational English for meaningful participation, and current abuse of alcohol or drugs requiring specific clinical intervention. All participants were between the ages of 18 and 65 years and had an estimated premorbid IQ, as scored by the National Adult Reading Test (NART; Nelson, 1982) of >70. All participants performed the following clinical and cognitive assessments.

Clinical Assessment. Current psychopathology was rated using four measures. The Rosenberg Self-Esteem Scale (RSE; Rosenberg, 1965) is a 10-item self-report measure used to evaluate global self-esteem. The percentage of items endorsed is reported with higher scores reflecting higher self-esteem. The Self-Ambivalence Measure (SAM-19; Bhar & Kyrios, 2007) is a 19-item questionnaire that measures the extent to which beliefs about self-worth are uncertain, conflicted, and a source of anxious introspection or a chronic preoccupation. Individuals scoring higher are ambivalent about self-worth, and thus, have difficulty synthesizing self-views, perceive their self-concept in terms of dichotomies, and thus, continuously redefine self, and focus on excessive personal preoccupation. Delusional ideas were assessed using two measures: the Peter’s Delusional Inventory (PDI; Peters, Joseph, & Garety, 1999) and the Creative Experience Questionnaire (CEQ; Merckelbach, Horselenberg, & Muris, 2001). The 21-item PDI measures the number and quality of unusual or delusional beliefs. The overall percentage of delusional ideas endorsed is reported as well as the three mean ratings on the PDI subscales including “distress,” “preoccupation,” and “conviction.” The CEQ, an instrument measuring fantasy proneness, comprises of 25-items that relate to daydreaming, intense fantasies, and imagination. The percentage of items endorsed is reported with higher scores indicating higher levels of fantasy proneness.

Cognitive Assessment. Three tasks were used to investigate executive function, face perception, and social cognition. Executive functioning (1) was assessed using the Controlled Oral Word Association Test (COWAT; Benton & Hamsher, 1976), which measures both phonological fluency (using the letters F, A, and S) and semantic fluency (using the categories: animals, food, and body parts). Participants were required to respond orally in 60 seconds with as many words as possible that began with the target letter or that falls within the particular category. Facial affect perception (2) was examined using the Name Affect subtest of the Comprehensive Affecting Testing System (CATS; Ekman & Friesen, 1975, 1976). The test measures the participants’ ability to recognize and name facial affect within a single face presented. This task involves facial expressions of six basic emotions including Happy, Sad, Angry, Fear, Disgusted, and Neutral. Social processing (3) was further evaluated using the Picture Sequencing Task (PST; Langdon & Coltheart, 1999). The task consists of 18 stories, each depicted in four-card (21 cm X 15 cm) picture sequences. There were two practice examples and four examples of each of the four experimental story types: social-script stories (ability
to reason logically), mechanical stories (ability to infer causal relations), capture stories (ability to disengage cognitively salient and misleading information), and false-belief stories (ability to infer false beliefs and to correctly predict that others can act on the basis of beliefs that misrepresent reality). Sequences were presented in a random order and cards were placed face down in front of subjects. Subjects were asked to turn the cards over and to place them in the correct order to show a logical sequence of events. The order of the cards and the time taken were recorded.

**Results**

The data obtained from the measures used during the clinical and cognitive assessments were compared between each individual BDD case and the healthy control group. Deficits were defined in terms of standard deviations (SDs) away from the norm. Table 1 displays the demographic information and psychopathological data obtained during the clinical assessment for the three BDD cases and the group of healthy controls (n = 10).

| TABLE 1. Demographics and Clinical Data of the Three BDD Cases and Healthy Control Group |
|-----------------|-----------------|-----------------|-----------------|
|                  | Controls        | BDD             |                 |
|                  | n = 10          | n = 3           |                 |
|                  | Male/Females    | Case 1          | Case 2          | Case 3          |
|                  | 3:7             | F               | M               | F               |
| Age              | 34.9 (12.8)     | 58*             | 25              | 16*             |
| Education (no. of years) | 21.1 (12.2) | 12              | 12              | 10              |
| NART             | 115.2 (11.2)    | 95*             | 118             | 110             |
| Rosenberg self-esteem | 78.3 (16.3) | 60*             | 33.3**          | 30**            |
| PDI (% yes)      | 16.9 (9.6)      | 33.3*           | 47.6***         | 85.7****        |
| Distress         | 1.4 (0.7)       | 2.7*            | 3.1**           | 3.0***          |
| Preoccupation    | 1.7 (0.5)       | 2.7**           | 3.5***          | 2.1             |
| Conviction       | 3.2 (0.8)       | 2.4             | 4.2*            | 3.9             |
| SAM-19           | 14.0 (5.6)      | 29**            | 50***           | 33***           |
| CEQ (% yes)      | 29.8 (14.4)     | 16              | 40              | 83.3***         |

Note. Mean (SD). NART (National Adult Reading Test); PDI (Peter’s Delusional Inventory); SAM (Self-Ambivalence Measure); CEQ (Creative Experience Questionnaire). *< or > 1 SD, **< or > 2 SD, ***< or > 3 SD.

The demographic data showed that Case 1 and Case 3 differed from the healthy control group by one SD on Age (Case 1 older and Case 3 younger) and Case 1 has lower IQ. There was no difference in Education. Compared to the healthy control group, the clinical assessment showed that all three BDD cases had significantly lower self-esteem (at least one or two SDs lower) as measured by the RSE, and greater self-ambivalence (two or three SDs higher) as measured by the SAM. The three BDD cases also endorsed more delusional items than controls on the PDI which causes a greater amount of distress and preoccupation. Case 3 demonstrated severely high levels of creative experiences or fantasy proneness on the CEQ (three SDs higher).

Table 2 displays the data obtained during the cognitive assessment. The BDD cases showed impaired phonological fluency compared to the healthy control group.
(one to three SDs lower), but semantic fluency was intact on the COWAT. On the facial affect task of the CATS, two of the three BDD cases were impaired on overall recognition of facial expressions (i.e., % correct) compared to the healthy control group (three SDs lower). Out of the six basic emotions (Happy, Sad, Angry, Fear, Disgust, and Neutral), the BDD cases were particularly poor at detecting Neutral facial expressions, frequently labelling them as Angry (i.e., negative). Surprisingly, the three BDD cases performed similar to the healthy control group on a measure of general social processing (i.e., PST).

**DISCUSSION**

The three BDD cases illustrated many of the disorder’s clinical features described earlier. For example, all three cases were excessively preoccupied with defects in their appearance that were either minor or purely imaginable. These preoccupied concerns caused significant distress in their everyday lives and commonly involved associated and ritualistic behaviors such as prolonged and/or frequent mirror checking.

Psychopathological data showed that BDD is strongly associated with low self-esteem, high self-ambivalence, and greater delusional thinking, as was evident in all three BDD cases. Guidano and Liotti (1983) described ambivalent individuals as having an incompatible self-image or a “non-articulate view” of self, which makes it difficult for the individual to be certain about his or her self, and thus, have difficulty synthesizing self-views into a coherent picture of self. This leads to overcompensations in decisions about self-worth, as individuals tend to engage in all-or-nothing thinking. Self-ambivalence can be distinguished from self-esteem in that it does not necessarily refer to whether the self is regarded positively or negatively, instead one’s description and evaluation of self fluctuate form one extreme to another and are felt to be contradictory. Veale et al. (2003) have reported that BDD individuals have an unrealistic ideal or demand as to how they should look (more like depressed patients), being more concerned with a failure to achieve their own (internal) aesthetic standard. It can
be suggested that BDD individuals achieve clarification about their self-concept, first
by being vigilant for signs and conduct aligned with their ideas of positive self, and
second by trying to protect against becoming aware of negative self-representations
by engaging in inappropriate behavior (Bhar & Kyrios, 2007). It can be suggested
that the high levels of self-ambivalence may be a compensatory response to restore a
low self-esteem. This may involve perfectionistic and obsessive-compulsive behaviors
including ruminating or worrying about one’s sense of self, and looking for signs or
clues in the outside or internal world about one’s true worth. Thus, self-esteem and
ambivalence levels may be the key features leading toward self-discrepancy. This in
turn would explain the high levels of unusual or delusional thinking related to their
“ideas” about their appearance as measured by the PDI.

The cognitive results from the three BDD cases confirmed previous suggestions
that BDD is associated with executive function and facial affect perception impair-
ments when compared to healthy controls (Deckersbach et al., 2000; Hanes, 1998).
As an extension of face processing, we decided to also look at more general social
cognitive abilities in BDD. Surprisingly, the BDD cases showed no impairment in
social cognitive functioning. The most striking cognitive deficit was on the facial affect
perception. The current findings showed a strong recognition bias for angry facial ex-
pressions when our cases were presented with neutral faces. That is, they consistently
labelled neutral faces as angry. This emotion recognition bias for angry expressions
is consistent with previous work (Buhlmann et al., 2004, 2006). The latter authors
showed that BDD cases misinterpret other facial expressions, particularly disgusted
ones, more often as angry, and this seems to be especially the case for self-referent situ-
ations. Here, the bias was not simply caused by angry-disgust confusion. It is possible
that this bias fosters the “imagined” beliefs prominent in individuals with BDD, such
that they are more likely to interpret other individuals’ facial expressions as a repelling
response due to their appearance or “ugliness.” This is based on the notion that angry
faces signal personal rejection (Gilboa-Schechtman, Ben-Artzi, Jeczemien, Maron, &
Hermesh, 2004) and it seems reasonable to suggest that this attentional bias may lead
to improved memory for social rejection and overly negative interpretation of social
events. Similar to BDD, individuals with OCD (e.g., Buhlmann et al., 2004) as well
as individuals with depression (e.g., Leyman et al., 2007), showed evidence of an
emotion recognition bias for angry expressions, suggesting this bias to be involved in
a broader spectrum of disorders, and therefore questioning the current classification
of BDD as a somatoform disorder. The impaired facial recognition in BDD may be
explained by findings reported by Deckersbach et al. (2000) whom suggested that
BDD individuals recall and focus on specific isolated details, rather than on larger and
more global organizational features. Therefore, when looking at a face, BDD individu-
als cannot recognize the whole emotion. This impaired perceptual ability may also be
explained by the high self-discrepancy found in BDD (Veale et al., 2003). In turn, this
would explain their fear of negative evaluations and ideas of reference (or exaggerated
beliefs) about their appearance. It can be suggested that further group-based research
examining whether BDD individuals consistently mislabel all other emotions as angry,
as well as questionnaire research examining what being “angry” means to BDD indi-
viduals, might be revealing here.

The BDD cases also showed deficits in executive function, specifically on phono-
logical (i.e., letter) fluency but not on semantic (i.e., categorical) fluency. It is specu-
lated that the poor performance obtained on the fluency task would be even greater
with group data, and thus provides evidence of an executive functioning impairment.
It is of importance to note that the reduction in scores on phonological fluency cannot be accounted for by perseverations made during the assessment (i.e., total error scores matched that of the controls), unlike other disorders like schizophrenia where perseverations are prevalent (e.g., Crider, 1997). The executive functioning deficits are consistent with Hanes’s (1998) findings which showed that both BDD and OCD respondents were impaired on measures of executive functioning such as the Tower of London (planning abilities) and Stroop task (response inhibition). In addition, the observed memory and learning deficits found in a BDD group by Deckersbach et al. (2000) was mediated by executive function deficits and was also similar to that observed in OCD patients. The simple fluency task used in the current study is more easily administrated than previous tasks, thus has greater clinical utility.

Despite impaired performance on the facial affect task and executive function, social cognitive processing was generally intact for the BDD cases, with the exception of one case showing significant impairment on a subtask of the Pictures Sequencing Task (i.e., PST) associated with “false-beliefs” (i.e., impaired mentalizing ability). This case was significantly older than the other three cases, but there were no other demographic differences. The intact social cognitive functioning observed in the other two BDD cases came as a surprise due to an angry-bias observed earlier in the current report and previous studies (e.g., Hanes, 1998), together with previous suggestions that angry faces reflect social rejection (e.g., Gilboa-Schechtman et al., 2004). Because this is case study data, collection of group data using this task is still advised to confirm these results.

Finally, there are a number of limitations in the current study. We examined a small number of individual BDD cases rather than a group of BDD participants. This preliminary case study data does, however, provide useful information with regards to cognitive abilities useful to follow up with group-based research. Further research could also employ measures of delusional thinking that capture the beliefs held in BDD more accurately, rather than general questionnaires designed for used in psychosis. The Brown Assessment of Beliefs Scale (BABS; Eisen et al., 1998) would, therefore, be useful. A more detailed examination of general perceptual abilities is also recommended. We postulate that Gestalt perception may be particularly impaired in BDD. Gestalt tasks measure our ability to process parts and amalgamate these parts into a whole image. Last, the current study focused on the visual perception of faces. It can be recommended that future studies include tasks associated with other body-related parts as it is evident that BDD individuals show preoccupied concerns not only of facial features, but also some part(s) of the body other than the face. Future research should further investigate the emotion-processing bias evident in BDD individuals in order to establish whether it is an angry-specific bias and if so, whether it is an attentional bias toward or away (i.e., protective) from such emotional stimuli. In a recent study, Kurosaki, Shirao, Yamashita, Okamoto, and Yamawaki (2006) showed gender differences in healthy controls in brain activation when confronted with their own distorted body image; thus, gender is another potentially important factor to consider for future research in BDD studies.

In conclusion, the current report provided a description of the clinical features involved in BDD and confirmed the importance of affect processing and executive function in these individuals. Furthermore, for the first time, the current findings showed that general social cognitive processing may be intact in BDD. The misinterpretation of other people’s facial expressions may be responsible for fostering beliefs that others are repelled by their perceived ugliness, thus contributing to the maintenance of the disorder. Based on the current cognitive evidence, we suggest that abnormal amygdala
activity and prefrontal regulation might be present in BDD. Interestingly, a number of other studies have demonstrated impaired amygdala function (both hyper- and hypo-activity) during emotional face processing in a number of different diagnostic conditions such as OCD (hypo-activity; see Phillips & Mataix-Cols, 2004), social-phobia (hyperactivity; see Stein, Goldin, Sareen, Zorrilla, & Brown, 2002), and schizophrenia (hypo-activity; see Streit et al., 2001). We might predict a mixed response in BDD; that is, a hyperactivity to nonemotional or neutral faces implicating heightened activity to nonthreatening events, and a hypo-response to faces that would normally be considered as emotive. The difficulties reported here and previously (e.g., Deckersbach et al., 2000; Hanes, 1998) on executive functioning, demonstrated similarity to OCD, thus, show support for frontostriatal-amygdala system dysfunction as well as possible temporal and parietal cortical abnormalities in BDD.

The data presented here has a number of implications with regards to the treatment of BDD. First, our finding of high self-ambivalence provides a basis to include self-ambivalent perceptions as a cognitive style in BDD within cognitive-behavioral models. Second, confirming impaired executive function and affect face processing suggests that cognitive remediation techniques used to improve deficits in these neuropsychological domains could have some utility in BDD. Executive function remediation packages aim to improve planning, error detection, and organization, while facial affect training helps individuals extract the correct information from faces to allow accurate identification of emotions. Last, accurate identification of the neurobiological substrates will inform the development of neurobiological treatments, for example, brain stimulation techniques. This will be particularly important for patients that show treatment resistance.

REFERENCES


