Binge Eating Disorder and Body Uneasiness

Massimo Cuzzolaro  
University Sapienza, Roma  
Rehabilitation Clinical Institute "Villa delle Querce", Nemi, Roma

Lorenzo Donini  
University Sapienza, Roma  
Rehabilitation Clinical Institute "Villa delle Querce", Nemi, Roma

Maurizio Bellini  
University Alma Mater, Bologna

Chiara Santomassimo  
University Sapienza, Roma, Italy  
Rehabilitation Clinical Institute "Villa delle Querce", Nemi, Roma

Abstract

Debate continues regarding the nosological status of binge eating disorder (BED) and the specific diagnostic criteria, including whether, like anorexia nervosa and bulimia nervosa, it should be characterized by body image disturbances in addition to abnormal eating behaviour. The aims of this article are: a) to concisely review the main points of the literature that has developed on diagnosis and treatment (especially pharmacological) of BED and b) to present the results of an original research on body image in obese patients with BED. The study was aimed to verify the following hypothesis: in persons with obesity, BED is associated with greater body uneasiness independently of some possible modulating factors. We studied a clinical sample of 159 (89 females and 70 males) adult obese patients who fulfilled DSM-IV-TR diagnostic criteria for BED matched to 159 non-BED obese patients for gender, ethnicity, BMI class, age, weight, stature, onset age of obesity, education level, and marital status. We used the Body Uneasiness Test (BUT), a valuable multidimensional tool for the clinical assessment of body uneasiness in subjects suffering from eating disorders and/or obesity. Obese patients with BED reported higher scores than non-BED patients in the General Severity Index (BUT-A GSI) and in every BUT-A subscale. All differences were statistically significant in both sexes. As expected women obtained higher scores than men. According to some other studies, our findings suggest that a negative body image should be included among diagnostic criteria for BED. Consequently, treatment should be focused not simply on eating behaviour and outcome studies should evaluate changes of body image as well.

Keywords: binge eating disorder, obesity, body image, Body Uneasiness Test, drug treatment
INTRODUCTION

Binge eating - objective overeating accompanied by the subjective experience of loss of control over eating - is a symptom that crosses the entire field of eating disorders (ED) and the whole spectrum of body weights: skeleton-like individuals, normal-weight subjects, and obese people may describe a similar, conflictual, craving for food (Fairburn & Wilson, 1993; Russell, 1997). On the other hand, binge eating disorder (BED) is the name of a syndrome that was formally recognized as a possible new diagnostic category in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-IV (American Psychiatric Association, 1994).

The first description of recurrent compulsive overeating as a clinical phenomenon probably dates back to 1932 (Stunkard, 1990; Wulff, 1932, 2001). In the following six decades, a winding path led to the proposal that BED should be added to the current crowded list of mental disorders (Cuzzolaro & Vetrone, in press). We might point out four milestones:

i. Half a century ago, Albert Stunkard proposed the name of binge-eating syndrome for an eating pattern associated with obesity. We remind that an obese patient, Hyman Cohen, to name irresistible urges to overeat, coined the expression binge eating: "I didn’t enjoy it at all. It just happened. It is as a part of me just blacked out. And when that happened there was nothing there, except the food and me, all alone" (Stunkard, 1959).

ii. In 1979, Gerald Russell named bulimia nervosa (BN) a syndrome that he considered "an ominous variant" of anorexia nervosa (AN) (Russell, 1979). People with BN suffer from frequent binge eating attacks, regular compensatory behaviours (self-induced vomiting and/or laxative abuse, and/or dieting, and/or excessive physical exercise), and morbid fear of fatness. Most of them maintain their weight within the normal range.

iii. In the last decades of the past century clinicians increasingly reported on women and men who, like Stunkard’s patient, engaged in recurrent binge eating with marked distress but without the regular compensatory behaviours that characterize BN; most of these patients were obese. Based on these observations Spitzer and colleagues advanced the proposal of binge eating disorder (BED) as a new diagnostic category (Spitzer, 1991; Spitzer, Devlin, Walsh, Hasin, Wing, Marcus et al., 1992; Spitzer, Yanovski, Wadden, Wing, Marcus, Stunkard et al., 1993).

iv. In 1994, DSM-IV agreed with Spitzer et al’s suggestion and recognized BED as a possible new diagnostic category.

In the DSM-IV and in the DSM-IV-TR (American Psychiatric Association, 2000) BED is proposed as an example of the eating disorder not otherwise
specified (EDNOS) category. Besides, it is included in Appendix B (possible new diagnostic categories requiring further study). BED is defined as follows: "recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviours characteristic of bulimia nervosa". No equivalent diagnostic category exists in the ICD-10, the tenth edition of the WHO International Classification of Diseases (World Health Organization, 1992). The ICD-10 diagnosis for people who present the clinical picture of BED may be Atypical bulimia nervosa (code number F50.3) or Eating Disorder, unspecified (code number F50.9; WHO, 1993).

Overall, BED is not yet a diagnosis approved in either DSM or ICD but it is the most distinct subgroup in the diagnostic category of EDNOS and an increasing number of scientific articles have been devoted to it. Although the conceptual issues of BED haven’t yet met sufficient agreement, BED is already accepted as an eating disorder (ED) in actual practice (Basdevant & Guy-Grand, 2004; Fairburn & Brownell, 2002; Goldstein, 2005; Grilo, 2006; Mitchell, Devlin, de Zwaan, Crow, & Peterson, 2008; Wadden & Stunkard, 2002). What is more, the new diagnostic category of BED has connected the psychiatric field of ED with the medical area of obesity: "The bridge drew more attention to the psychological and psychiatric aspects of obesity and gave a contribution to the development of a multidimensional team approach to the assessment and treatment of eating and weight disorders" (Cuzzolaro & Vetrone, in press).

The purpose of this article is twofold: first, to concisely go over the main points of the literature that has developed on the nosological status of BED over the last fifteen years; and second, to present the results of an original research aimed to study body image uneasiness in a clinical sample of 159 (females 89 and males 70) adult obese patients who fulfilled DSM-IV-TR diagnostic criteria for BED.

**BED: nosological status and diagnostic criteria**

The nosological status of BED has not yet met adequate accord. In 1999, Williamson and Martin reviewed the first five years of research on BED and concluded that questions about the definition of BED persisted: this cluster symptom presentation may be conceptualized as a separate psychiatric syndrome or it may be viewed as a frequent behavioural symptom associated with obesity (Williamson & Martin, 1999).

Stunkard, the researcher who originally described binge eating, in a thought-provoking article, wrote that the great variability of BED limits the implications that can be drawn from its diagnosis and, in particular, the presence or absence of BED is not a useful distinction in selecting treatment for obese individuals: BED may be more useful as "a marker of psychopathology" than as new distinct diagnostic entity (Stunkard & Allison, 2003).
Some years later, Walsh and Satir reviewed the literature published during the period 2002-2003 and concluded that "a consensus does not yet appear to have formed in the field regarding the wisdom of formally designating BED as an eating disorder" (Walsh & Satir, 2005). In contrast, Dingemans et al., discussing the empirical status of BED after a decade of research, concluded, "There is evidence to suggest that BED represents a distinct eating disorder category" (p. 76) (Dingemans, van Hanswijck de Jonge, & van Furth, 2005). In 2007, Wilfley et al. supported the same point of view: BED should be made an official diagnostic category in DSM-V (Wilfley, Bishop, Wilson, & Agras, 2007). In addition, some authors investigated the question of whether the current classifications of ED capture the natural clustering of eating-related pathology: should ED be conceptualized as discrete syndromes or as dimensions that differ in degree among individuals?

Williamson and colleagues explored the latent structure of ED symptoms, as defined by DSM-IV, and using taxometric analyses found empirical support for conceptualizing BN and BED as discrete syndromes (Williamson, Womble, Smeets, Netemeyer, Thaw, Kutlesic et al., 2002).

As regards diagnosis of BED, the Eating Disorders Work Group of the DSM-IV task force in conjunction with Spitzer and colleagues (1992) developed the provisional diagnostic criteria for BED along three lines:

- the person suffers from recurrent episodes of binge eating (the binge eating occurs, on average, at least two days a week for six months),
- the binge eating is not associated with the regular use of inappropriate compensatory behaviours,
- marked distress regarding binge eating is present.

In the last decade, several authors have raised doubts about the DSM-IV research criteria for BED (Cuzzolaro & Vetrone, in press). Firstly, difficulties with diagnosis may arise from the precise recognition of binge eating episodes in obese persons (Wilfley, Schwartz, Spurrell, & Fairburn, 2000). The distinction between binge eating (irresistible acute attack) and compulsive grazing (urge to eat without a complete loss of control that may last an entire day) may be arduous. In the second place, a number of researchers found that the boundary line between full-syndrome BED and partial or sub-threshold BED (STBED) is unclear (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Striegel-Moore, Dohm, Solomon, Fairburn, Pike, & Wilfley, 2000). Wilfley and colleagues included among their recommendations for DSM-V "unifying the frequency and duration cut-points for BN and BED to once per week for 3 months" (Wilfley et al., 2007). Next, distress as an emotional state should be distinguished from distress as impairment in social or occupational functioning (Dingemans et al., 2005). Then, obesity is not a current criterion for the diagnosis of BED but perhaps it should be included in the same way as underweight is a criterion for the diagnosis of AN. This problem is still
unresolved (Dingemans et al., 2005). Finally, unlike the two major eating disorders (ED) - anorexia nervosa (AN) and bulimia nervosa (BN) - BED does not include a body image disturbance among the DSM-IV provisional diagnostic criteria. Should body image disturbance be added to the diagnostic criteria for BED?

**BED and negative body image**

Schilder originally characterized body image as "the picture of our own body which we form in our mind, that is to say the way in which the body appears to ourselves" (Schilder, 1935). In fact, body image is a multidimensional psychological construct, which includes perceptual, cognitive, emotional, relational, and behavioural components (Pruzinsky & Cash, 2002). Rosen has coined the expression *negative body image* (Rosen, 1998, 2002) to include all cases of clinically significant uneasiness due to body image in subjects with objective anomalies concerning appearance (e.g. people with obesity) and in subjects with basically imaginary or exaggerated, or at times both, aesthetic problems (e.g. people with body dysmorphic disorder, AN or BN).

The first systematic studies on body image in subjects with obesity date back to the 1960’s (Stunkard & Burt, 1967; Stunkard & Mendelson, 1967) and their clinical relevance has continued to increase in the years. The obesity galaxy is enormous and heterogeneous: it is well known that such factors as gender, age, degree of obesity, onset age of overweight, ethnicity, social class, history of childhood teasing and parental criticism about weight, history of weight cycling, and presence of binge eating all show important modulating effects on body uneasiness (Sarwer & Thompson, 2002; Sarwer, Thompson, & Cash, 2005; Schwartz & Brownell, 2002).

It is well known that there are essential links between ED and body image disturbances (Habermas, 1989), at least in Western countries (Dorian & Garfinkel, 2002), from the beginning of the twentieth-century (Janet, 1903; Wulff, 2001). However, unlike AN and BN, current diagnostic criteria for BED focus completely on eating behaviour and feelings about binge eating and do not include body image distortion and/or distress. In the last fifteen years, several studies have remarked that obese individuals with BED present greater dissatisfaction and distress about their body appearance than obese people without BED.

Wilfley et al. (2000) found that patients with BED had weight and shape concerns comparable to BN patients and higher than AN patients and concluded that their results "supported the status of BED as an eating disorder and suggest that the elevated Eating Disorder Examination scores reflect the combined impact of being objectively overweight and having disordered cognitions and behaviours about eating, shape, and weight". The Eating Disorder Examination (EDE) is a semistructured interview useful in assessment and diagnosis of eating disorders (Fairburn & Cooper, 1993).
Reas et al. (2005) studied repetitive body checking (e.g. pinching some body areas to check for fatness) and avoidance behaviours (e.g. avoiding close-fitting clothes) in 377 (80 men and 297 women) overweight (BMI ≥ 25) treatment-seeking BED patients. Significant associations emerged between checking and restraint, and conversely, between avoidance and binge eating (Reas, Grilo, Masheb, & Wilson, 2005). Hrabosky et al. (2007) found that in 399 patients with BED, shape/weight overvaluation was unrelated to BMI but was strongly associated with psychometric measures of eating-related psychopathology and psychological functioning (higher depression and lower self-esteem). They suggested that shape/weight overvaluation "warrants consideration as a diagnostic feature for BED" (Hrabosky, Masheb, White, & Grilo, 2007). One year later Grilo et al. found that overweight BED patients who overvalue their shape/weight reported greater eating-related psychopathology and depression levels than overweight BED patients do with subclinical levels of overvaluation. Nevertheless, both BED groups reported greater overall eating pathology and depression levels than the overweight non-BED comparison group (Grilo, Hrabosky, White, Allison, Stunkard, & Masheb, 2008). There are gender differences. Reas and colleagues remarked that obese women with BED reported significantly greater levels of body checking than obese men with BED (Reas et al., 2005). Guerdjikova et al. compared 44 obese males with BED with 44 age- and race-matched obese females with BED seeking weight loss treatment. They found that males had fewer previous attempts at weight loss or less help-seeking behaviour, possibly related to their less pronounced body dissatisfaction (Guerdjikova, McElroy, Kotwal, & Keck, 2007).

As regards general psychiatric comorbidity, it is linked with increased severity of ED symptoms; in general, there are associations between specific ED symptoms and specific forms of comorbidity (Spindler & Milos, 2007). Fernandez-Aranda et al. recently studied a sample of women with ED and found that impulse control disorders (e.g. compulsive buying disorder, kleptomania) occurred more in individuals with binge eating subtypes, and were associated, in particular, with greater body image disturbance (Fernandez-Aranda, Pinheiro, Thornton, Berrettini, Crow, Fichter et al., 2008).

**Is BED an indication or a contraindication for specific treatments?**

**Can drugs relieve BED and weight/shape concerns?**

The validity of the diagnosis of BED as a discrete syndrome depends largely on the answers to these questions. For clinicians treating obese patients in a weight control program, a crucial unresolved question is whether individuals with BED benefit from specialized interventions (surgical, pharmacological, or psychosocial) in addition to standard behavioural weight control treatment. As regards obesity surgery, more than a few studies suggest that a diagnosis of BED or a history of binge eating prior to treatment may be associated with unsatisfactory weight loss in
obese patients submitted to bariatric procedures (Mitchell & de Zwaan, 2005; Sallet, Sallet, Dixon, Collis, Pisani, Levy et al., 2007).

Patients presenting with both obesity and BED face multiple challenges: promoting and maintaining weight loss, normalizing their eating patterns, improving their physical and psychological health, and working to enhance their own acceptance of their body image. Several psychological and pharmacologic treatment approaches have been used in this population. Most suppress binge eating in the short term, and some seem promising in the long term as well. However, sustained weight loss remains a largely unrealized goal. A particular question is whether the diagnosis of BED is an indication for specific pharmacological treatments.

In the last two decades, BED has been studied intensely as target for pharmacotherapy. Table 1 makes a list of 24 double-blind placebo-controlled trials.

**Table 1.** Drug treatment of binge eating disorder: double-blind placebo-controlled trials from 1990 to 2008

<table>
<thead>
<tr>
<th>Reference</th>
<th>Drug</th>
<th>Number of subjects</th>
<th>Number of weeks</th>
<th>mg/day</th>
<th>Reduction in the frequency of binges</th>
<th>Weight loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>(McCann &amp; Agras, 1990)</td>
<td>desipramine</td>
<td>23</td>
<td>12</td>
<td>≤ 300</td>
<td>d &gt; p</td>
<td>ns</td>
</tr>
<tr>
<td>(Marcus et al., 1990)</td>
<td>fluoxetine</td>
<td>21</td>
<td>52</td>
<td>60</td>
<td>ns</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Alger et al., 1991)</td>
<td>imipramine or naltrexone</td>
<td>33</td>
<td>8</td>
<td>50-200</td>
<td>ns</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Stunkard et al., 1996)</td>
<td>d-fenfluramine</td>
<td>28</td>
<td>8</td>
<td>30</td>
<td>d &gt; p</td>
<td>ns</td>
</tr>
<tr>
<td>(Hudson et al., 1998)</td>
<td>fluvoxamine</td>
<td>85</td>
<td>9</td>
<td>50-300</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Laederach-Hofmann et al., 1999)</td>
<td>imipramine</td>
<td>31</td>
<td>8</td>
<td>75</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(McCleary et al., 2000)</td>
<td>sertraline</td>
<td>34</td>
<td>6</td>
<td>50-200</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Arnold et al., 2002)</td>
<td>fluoxetine</td>
<td>60</td>
<td>6</td>
<td>20-80</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(McCleary et al., 2003a)</td>
<td>topiramate</td>
<td>61</td>
<td>14</td>
<td>25-600</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Pearlstein et al., 2003)</td>
<td>fluvoxamine</td>
<td>20</td>
<td>12</td>
<td>239</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>(Appolinario et al., 2003)</td>
<td>sibutramine</td>
<td>60</td>
<td>12</td>
<td>15</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(McCleary et al., 2003b)</td>
<td>citalopram</td>
<td>38</td>
<td>6</td>
<td>20-60</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Girard et al., 2005b)</td>
<td>fluoxetine</td>
<td>108</td>
<td>16</td>
<td>up to 60</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>(Devlin et al., 2005)</td>
<td>fluoxetine</td>
<td>116</td>
<td>20</td>
<td>up to 60</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>(Milano et al., 2005)</td>
<td>sibutramine</td>
<td>20</td>
<td>12</td>
<td>10</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Golay et al., 2005)</td>
<td>orlistat</td>
<td>89</td>
<td>24</td>
<td>360</td>
<td>ns</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Girard et al., 2005a)</td>
<td>orlistat</td>
<td>50</td>
<td>12</td>
<td>360</td>
<td>ns</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(McCleary et al., 2005)</td>
<td>zonisamide</td>
<td>60</td>
<td>16</td>
<td>100-600</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Bauer et al., 2006)</td>
<td>sibutramine</td>
<td>73</td>
<td>16</td>
<td>15</td>
<td>ns</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(McCleary et al., 2007b)</td>
<td>topiramate</td>
<td>407</td>
<td>16</td>
<td>25-400</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(McCleary et al., 2007a)</td>
<td>atomoxetine</td>
<td>40</td>
<td>10</td>
<td>40-120</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Claudino et al., 2007)</td>
<td>topiramate</td>
<td>73</td>
<td>21</td>
<td>200</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Guerrdikova et al., 2008)</td>
<td>escitalopram</td>
<td>44</td>
<td>12</td>
<td>26.5</td>
<td>?</td>
<td>d &gt; p</td>
</tr>
<tr>
<td>(Wilfley et al., 2008)</td>
<td>sibutramine</td>
<td>304</td>
<td>24</td>
<td>15</td>
<td>d &gt; p</td>
<td>d &gt; p</td>
</tr>
</tbody>
</table>

*Note. d > p = drug superior to placebo; ns = not significant difference*
Research is still in its preliminary stages but a number of drugs have shown evidence of some therapeutic value in BED and according to several researchers pharmacotherapy may be a useful component of a multidimensional treatment approach. The medication dosage is usually at the high end of the recommended range. Some studies have examined the added benefit of drugs and psychosocial interventions.

Fluoxetine, an antidepressant of the selective serotonin reuptake inhibitor (SSRI) class was the first drug to receive an indication for treatment of bulimia nervosa and was tested in several trials for BED, often associated with cognitive behavioural therapy (CBT). In a double-blind placebo-controlled trial of fluoxetine plus behaviour modification in the treatment of obese binge-eaters and non-binge-eaters, Marcus et al. found that patients treated with fluoxetine (60 mg/day) plus behaviour modification lost significantly more weight than those treated with placebo plus behaviour modification (Marcus, Wing, Ewing, Kern, McDermott, & Gooding, 1990). However, the drug did not appear to have a differential benefit for binge-eaters.

Grilo et al. (2005b) obtained partly dissimilar results when they compared fluoxetine (60 mg/day), placebo, CBT+fluoxetine and CBT+placebo in a sample of 108 patients with BED (Grilo, Masheb, & Wilson, 2005b). Remission rates (zero binges for 28 days) for completers were 29% (fluoxetine), 30% (placebo), 55% (CBT+fluoxetine), and 73% (CBT+placebo). Weight loss was modest and did not differ across treatments. The authors concluded that CBT, but not fluoxetine, demonstrated efficacy for the behavioural and psychological features of BED, but not for obesity.

Molinari et al. (2005) compared CBT, fluoxetine and CBT+fluoxetine: the two groups which underwent psychotherapy resulted in a better outcome after 12 months - in terms of number of bingeing episodes, weight loss and psychological well being - than the group treated with pharmacological therapy alone; furthermore, the drug did not appear to add a significant benefit to CBT (Molinari, Baruffi, Croci, Marchi, & Petroni, 2005). This investigation was not placebo-controlled.

Devlin et al. (2005) studied 116 overweight/obese women and men with BED receiving a 16-session standard group behavioural weight control treatment over 20 weeks (Devlin, Goldfein, Petkova, Jiang, Raizman, Wolk et al., 2005). Simultaneously, subjects were randomly assigned to receive CBT+fluoxetine, CBT+placebo, fluoxetine, or placebo in a two-by-two factorial design. This interesting study was designed to examine the added benefit of two adjunctive interventions, individual CBT, and fluoxetine, offered in the context of standard group treatment. In general, patients showed substantial improvement in binge eating but little weight loss. Subjects who received also individual CBT improved more in binge frequency while fluoxetine treatment was associated with greater reduction in depressive symptoms. Two years later Devlin et al. (2007) followed up
the same patients and found that CBT was still associated with lower binge frequency and fluoxetine with a significant advantage on depressive symptoms: short-term (5-month) treatment may provide long-term (24-month) benefit (Devlin, Goldfein, Petkova, Liu, & Walsh, 2007).

In other studies, on the contrary, success seemed to be very short-lived. Stunkard et al. (1996) conducted an 8-week controlled clinical trial of the appetite suppressant d-fenfluramine with 28 severely obese female patients meeting full criteria for BED (Stunkard, Berkowitz, Tanrikut, Reiss, & Young, 1996). They found that the rate of binge eating in the d-fenfluramine group fell three times more rapidly than that in the placebo group but at 4-month follow-up, the binge frequency had increased to pre-treatment levels and no longer differed from that of the placebo group. Furthermore, surprisingly, in this trial the appetite suppressant d-fenfluramine was not associated with significant weight loss. Some years ago, this drug has been withdrawn from the market due to adverse cardiovascular effects, specifically valvular heart disease.

In Hudson and colleagues’ study fluvoxamine, a SSRI antidepressant drug, was found effective as well in the acute treatment of BED (reduction in the frequency of binges) (Hudson, McElroy, Raymond, Crow, Keck, Carter et al., 1998). We highlight three other results of this trial. First, there was no significant difference between placebo and fluvoxamine groups in the rate of decrease in Hamilton depression scale scores. Next, the placebo response rate was very high: 44% of the patients given placebo displayed a greater than 50% reduction in binges/week; in passing, even higher placebo response rates have been observed in other studies of BED and these figures prove that this disorder may often improve even with placebo. Finally, compared with placebo, fluvoxamine was associated with a significantly greater rate of reduction in BMI.

McElroy et al.’s investigations showed that two other SSRI antidepressant drugs, sertraline (McElroy, Casuto, Nelson, Lake, Soutullo, Keck et al., 2000) and citalopram (McElroy, Hudson, Malhotra, Welge, Nelson, & Keck et al., 2003b) were also associated with significantly greater rates of reduction in binges/week and BMI than placebo. Leombruni et al. (2007) did not find significant differences between sertraline (dose range: 100-200mg/day) and fluoxetine (dose range: 40-80 mg/day) in a randomized, double-blind, 24-weeks trial in obese patients with BED: a significant improvement in the Binge Eating Scale score and a significant weight loss emerged (Leombruni, Piero, Dosio, Novelli, Abbate-Daga, Morino et al., 2007). Both reboxetine (Silveira, Zanatto, Appolinario, & Kapczinski, 2005), a selective noradrenalin reuptake inhibitor, and venlafaxine (Malhotra, King, Welge, Brusman-Lovins, & McElroy, 2002), antidepressant of the serotonin-norepinephrine reuptake inhibitor (SNRI) class, appeared to be effective and well-tolerated agents for the treatment of BED in non-controlled studies. Reboxetine was also associated with a statistically significant decrease in BMI.
Evidence suggests (Tata & Kockler, 2006) that topiramate, an anticonvulsant drug, may have mood-stabilizing properties, and cause decreased appetite and weight. Two studies described the short- and long-term efficacy of topiramate for BED associated with obesity. The short-term study (McElroy, Arnold, Shapira, Keck, Rosenthal, Karim et al., 2003a) was a 14-week, single-center, randomized, double blind, placebo-controlled trial; compared with placebo, topiramate was associated with a significantly greater rate of reduction in binge frequency and BMI. Subsequently, the long-term study (McElroy, Shapira, Arnold, Keck, Rosenthal, Wu et al., 2004) - a 42-week, open-label extension trial - showed enduring improvement in a number of patients with BED and obesity but was also associated with a high discontinuation rate. Topiramate was titrated from 25 mg/day to a maximum of 600 mg/day (median final dose of 250 mg/day). Adverse reactions included paresthesias, cognitive impairment, somnolence, headache, nausea, and gastrointestinal distress. These adverse effects are transient but they may interfere with patients' tolerability of topiramate therapy.

In 2007, a 16-week, multicenter, study (McElroy, Hudson, Capece, Beyers, Fisher, & Rosenthal, 2007b) in a large sample of 407 obese patients with BED (age 18–65) confirmed that topiramate was well tolerated and efficacious in improving the features of BED and in reducing body weight. In particular, topiramate induced binge eating remission in 58% of patients (placebo in 29%; p < .001). A flexible-dose investigation started at 25 mg/day and was titrated weekly over an 8-week period to 400 mg/day or the maximum tolerated dose. Discontinuation rates were high (about 30%) in each group. According to Guerdjikova et al, anecdotal observations, topiramate may be an effective treatment also for patients with BED and obesity who experience recurrent binge eating and weight gain after initially successful bariatric surgery (Guerdjikova, Kotwal, & McElroy, 2005).

Zonisamide is another antiepileptic drug associated with weight loss that appears to be a promising candidate for selective use in the treatment of overweight/obesity (Appolinario, Bueno, & Coutinho, 2004). McElroy et al. (2006) evaluated this anticonvulsant drug in 60 obese outpatients with BED (McElroy, Kotwal, Guerdjikova, Welge, Nelson, Lake et al., 2006). Compared with placebo, zonisamide was associated with a significantly greater rate of reduction in binge eating episode frequency, body weight, and Three Factor Eating Questionnaire Disinhibition Scale score (Stunkard & Messick, 1985). Eight patients receiving zonisamide discontinued because of adverse events. The authors concluded that zonisamide was efficacious, but not well tolerated, in the short-term treatment of BED. At this time, only three products have been approved for treatment of obesity: sibutramine, orlistat and rimonabant (in Europe and Canada), a cannabinoid type 1-receptor blocker.

Sibutramine and orlistat have been studied in patients with BED: both sibutramine and orlistat promote weight loss in obese patients with BED but only sibutramine may reduce the frequency of binge eating and the intensity of related
depressive symptoms. Sibutramine is a serotonin and norepinephrine reuptake inhibitor. An important double-blind study by Appolinario et al. (2003) evaluated its efficacy and tolerability in 60 obese patients with BED (Appolinario, Bacaltchuk, Sichieri, Claudino, Godoy-Matos, Morgan et al., 2003). The researchers found a significant reduction in the number of days with binge episodes in the sibutramine group compared with the placebo group; this was associated with a significant weight loss (-7.4 kg) compared with a small weight gain in the placebo group (1.4 kg). Sibutramine was also associated with a significantly greater rate of reduction in Binge Eating Scale (BES; Gormally, Black, Daston, & Rardin, 1982) and Beck Depression Inventory scores (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

There was a significant reduction in the number of days with binge episodes in the sibutramine group compared with the placebo group, this was associated with an important and significant weight loss (-7.4 kg) compared with a small weight gain in the placebo group (1.4 kg). Milano et al. (2005) used a smaller dose of sibutramine (10 mg/day) and, after 12 weeks, found that the binge frequency among patients who received sibutramine was significantly lower than that among those who received placebo (Milano, Petrella, Casella, Capasso, Carrino, & Milano, 2005). In 2008 (Wilfley et al., 2008), the first large-scale, multisite, placebo-controlled trial confirmed the efficacy of sibutramine (15 mg) in reducing binge eating, weight (sibutramine group mean = -4.3kg [SD = 4.8], placebo group mean = -0.8kg [SD = 3.5]), and associated psychopathology (Yager, 2008).

Bauer et al. (2006) recently assessed the effect of sibutramine and cognitive-behavioural weight loss (CBWL) treatment in obese subjects with and without subthreshold binge eating disorder (sBED). Treatment with CBWL programs and sibutramine leads to a higher weight loss than CBWL + placebo. Subjects with sBED significantly reduced their binge episodes during treatment, but with no augmenting effect of sibutramine (Bauer, Fischer, & Keller, 2006).

Orlistat is a gastrointestinal lipase inhibitor that exerts its activity in the lumen of the stomach and small intestine and promotes weight loss through the partial (30%) inhibition of dietary fat absorption. A number of investigators demonstrated the efficacy and safety of orlistat but they excluded patients with BED. Golay et al. (2005) hypothesized that orlistat may be considered as part of the management of overweight/obesity also for patients with BED. They randomized in double-blind fashion 89 obese patients with BED to treatment with orlistat or placebo in combination with a mildly reduced-calorie diet (Golay, Laurent-Jaccard, Habicht, Gachoud, Chabloz, Kammer, et al. 2005). After 24 weeks, the orlistat-group showed a mean weight loss from baseline significantly greater than the placebo group. However, patients in both treatment groups showed a comparable decrease in the mean number of reported binge eating episodes (and duration of episodes) per week. At the end of the study, orlistat-treated patients were less likely to have a
diagnosis of BED than placebo-treated patients were but this difference did not reach statistical significance.

In another randomized, placebo-controlled study, Grilo et al. (2005a) added orlistat (120 mg three times a day) to guided self-help CBT (CBTgsh) to treat 50 obese patients with BED (Grilo, Masheb, & Salant, 2005a). The addition of orlistat was associated with greater weight loss than the addition of placebo. Like in Golay et al.’s study, the authors found in both groups significant but similar improvements in eating disorder psychopathology and psychological distress. Both sibutramine and orlistat promote weight loss in obese patients with BED but only sibutramine reduces the frequency of binge eating and the intensity of related depressive symptoms (Appolinario et al., 2003).

Novel pharmacological interventions are needed and many compounds may be promising and/or are in early phases of clinical testing (Steffen, Roerig, Mitchell, & Uppala, 2006). For example, Broft et al. (2007) recently studied, in a small open-label trial, baclofen, a GABA-B agonist that may be useful the treatment of substance use disorders, and also reduces binge-like eating in rodents (Broft, Spanos, Corwin, Mayer, Steinglass, Devlin et al., 2007). Baclofen (60 mg/day) was associated with decreased binge eating frequency in patients with BED and BN. Atomoxetine is a highly selective norepinephrine reuptake inhibitor associated with weight loss. It was originally intended to be a new antidepressant drug and was the first non-stimulant drug approved for the treatment of attention-deficit hyperactivity disorder (ADHD). McElroy et al evaluated atomoxetine in the treatment of BED in a 10-week, randomized, double-blind, placebo-controlled, flexible dose (40-120 mg/day) trial. Compared with placebo, atomoxetine (N = 20) was associated with a significantly greater rate of reduction in binge-eating frequency, BMI, and Three Factor Eating Questionnaire hunger subscale (Stunkard & Messick, 1985). The drug was fairly well tolerated.

It is hoped that in a near future the development of genetic researches in the field of ED will probably "help the clinician to choose the most appropriate treatment, using genetic polymorphisms of vulnerability genes, those linked to endophenotypes, or genes implicated in the metabolism of the drug treatment" (Ramoz, Versini, & Gorwood, 2007). To sum up, at present, several medications may contain binge eating, at least in the short term, but weight loss is small or absent. Furthermore, most trials were very brief and, in particular, did not study body image and weight and shape concerns at baseline, endpoint, and follow-up.

**An observational study**

In this section we present the results of an observational (empirical comparison of groups) cross-sectional (data were collected from subjects at a single time) study on body image in obese patients with and without BED.
METHOD

Participants

We studied a sample of 159 (males 70 and females 89) adult (mean age of males: 35.77±10.17; mean age of females: 36.93±11.62) subjects with obesity (Body Mass Index, BMI ≥ 30 kg/m²) and BED (DSM-IV-TR), drawn from 3,581 outpatients who came to lose weight to three medical centres, accredited by the Italian Health Service for the treatment of obesity, from 2005 to 2007.

We recruited all the male patients (70) who fulfilled the DSM-IV-TR provisional criteria for BED and 89 consecutive female patients. Then we matched them to 159 non-BED obese patients (males 70 and females 89) drawn from the same population. Each O-BED subject was matched exactly to one control for gender, ethnicity (all Caucasian), and BMI class (Class I = 30-34.9; Class II = 35-39.9; Class III ≥ 40) looking for the best compromise for six other matching variables: age, weight, stature, onset age of obesity, education level, and marital status. All patients agreed to complete self-administered questionnaires.

Measures

Weight and Height

A physician measured each patient’s weight on a medical-balance and height by a stadiometer while patients were in their underwear without shoes.

Psychometric measures

We used the Body Uneasiness Test (BUT), a valuable multidimensional tool for the clinical assessment of body uneasiness in subjects suffering from eating disorders (Cuzzolaro, Vetrone, Marano, & Battacchi, 2000; Cuzzolaro, Vetrone, Marano, & Garfinkel, 2006; Ravaldi, Vannacci, Bolognesi, Mancini, Faravelli, & Ricca, 2006) and/or obesity (Carano, De Berardis, Gambi, Di Paolo, Campanella, Pelusi et al., 2006; Dalle Grave, Cuzzolaro, Calugi, Tomasi, Temperilli, & Marchesini, 2007; Marano, Cuzzolaro, Vetrone, Garfinkel, Temperilli, Spera et al., 2007). The BUT is a self-administered questionnaire that consists of two parts: BUT-A (34 items) and BUT-B (37 items). Higher scores indicate greater body uneasiness. The BUT was specifically designed to explore several areas in clinical and non-clinical populations:

- BUT-A: body shape/weight dissatisfaction, avoidance, compulsive control behaviours, detachment and estrangement feelings towards one’s own body (examples of items: I avoid mirrors; If I begin to look at myself, I find it difficult to stop; I have the sensation that my body does not belong to me)
- BUT-B: specific worries about particular body parts, shapes, or functions (examples of items: the shape of my face; buttocks; odour; blushing).

In keeping with the previous validation studies the BUT-A scores were combined in a Global Severity Index (GSI, 34 items) and in 5 subscales resulting from factorial analysis: Weight Phobia (WP - fear of being or becoming fat, 8 items), Body Image Concerns (BIC - worries related to physical appearance, 9 items), Avoidance (A - body image-related avoidance behaviour, 6 items), Compulsive Self-Monitoring (CSM - compulsive checking of physical appearance, 5 items), and Depersonalization (D - detachment and estrangement feelings toward the body, 6 items); BUT-B scores were combined in two global measures (Positive Symptom Total – PST and Positive Symptom Distress Index – PSDI) and in eight factors that examine specific worries about particular sets of body parts or functions.

The levels of Cronbach’s alpha coefficients range between .64 and .89. All the subscales but one (BUT-B VII, a factor that contains only two items) showed Cronbach’s alpha coefficients greater than .70 (Cuzzolaro et al., 2006; Marano et al., 2007). Therefore, internal consistency of BUT appears good (Nunnally, 1978; Thompson, Penner, & Altabe, 1990). For the present study, we considered only the BUT-A scores.

The English version of the BUT and scoring instructions are reported in the validation studies (Cuzzolaro et al., 2006; Marano et al., 2007). The test was administered in a self-reported fashion (i.e., the investigators did not assist the subjects in the compilation of the questionnaire). The diagnosis of BED was formulated based on the semi-structured diagnostic interview, EDE 12.0D (Fairburn & Cooper, 1993). The ethical committees of the individual centres approved the protocol of this multicenter study. All participants gave written informed consent for participation.

Data Analyses

We used chi-square statistics for categorical dependent variables and t-test for quantitative dependent variables. The analyses were performed using JMP 6.0.3 (SAS Institute, Cary, NC).

RESULTS

Table 2 shows means and standard deviations of age, weight, stature, and BMI of the different subgroups. Comparing the BED patients with the non-BED patients of the same sex (t-test) no statistically significant difference was found at the .05 alpha level.

---

1 The Italian and the French versions of the test are available from the authors on request.
Table 2. Matching variables: age, weight, height and Body Mass Index

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BED N = 70</td>
<td>NON-BED N = 70</td>
<td>t</td>
<td>BED N = 89</td>
<td>NON-BED N = 89</td>
<td>t</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>M 35.77</td>
<td>36.24</td>
<td>0.26</td>
<td>36.93</td>
<td>36.90</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>10.17</td>
<td>9.67</td>
<td></td>
<td>11.62</td>
<td>11.52</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>M 141.39</td>
<td>141.78</td>
<td>0.09</td>
<td>109.73</td>
<td>110.24</td>
<td>0.20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>27.41</td>
<td>23.52</td>
<td></td>
<td>17.10</td>
<td>17.35</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>M 0.76</td>
<td>0.78</td>
<td>1.37</td>
<td>1.63</td>
<td>1.63</td>
<td>0.72</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>0.07</td>
<td>0.07</td>
<td></td>
<td>0.06</td>
<td>0.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>M 45.40</td>
<td>44.73</td>
<td>0.51</td>
<td>41.49</td>
<td>41.35</td>
<td>0.16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>7.63</td>
<td>6.92</td>
<td></td>
<td>5.69</td>
<td>6.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BED – Binge Eating Disorder

Figure 1 illustrates the onset age of obesity (percentages) of the subgroups. Comparing the BED patients with the non-BED patients of the same sex (chi-square test) no statistically significant difference was found at the .05 alpha level (chi-square: 0.43 for males and 0.00 for females).

Figure 1. Differences between onset age of obesity

Table 3 shows the education level and the marital status of the subgroups. Comparing the BED patients with the non-BED patients of the same sex (chi-square test) no statistically significant difference was found at the .05 alpha level.
Table 3. Matching variables: education level and marital status

<table>
<thead>
<tr>
<th>Educational level</th>
<th>Males</th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BED</td>
<td>NON–BED</td>
<td><strong>X^2</strong></td>
<td>BED</td>
<td>NON–BED</td>
<td><strong>X^2</strong></td>
</tr>
<tr>
<td>Elementary school (≤ 5 years)</td>
<td>4%</td>
<td>6%</td>
<td>0.71</td>
<td>10%</td>
<td>8%</td>
<td>0.28</td>
</tr>
<tr>
<td>Middle school (8 years)</td>
<td>37%</td>
<td>44%</td>
<td>0.54</td>
<td>33%</td>
<td>36%</td>
<td>0.23</td>
</tr>
<tr>
<td>Diploma (13 years)</td>
<td>49%</td>
<td>40%</td>
<td>1.17</td>
<td>49%</td>
<td>52%</td>
<td>0.09</td>
</tr>
<tr>
<td>University degree</td>
<td>10%</td>
<td>10%</td>
<td>0</td>
<td>8%</td>
<td>4%</td>
<td>0.88</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Marital status</th>
<th>Males</th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BED</td>
<td>NON–BED</td>
<td><strong>X^2</strong></td>
<td>BED</td>
<td>NON–BED</td>
<td><strong>X^2</strong></td>
</tr>
<tr>
<td>Single</td>
<td>57%</td>
<td>49%</td>
<td>0.81</td>
<td>40%</td>
<td>40%</td>
<td>0.00</td>
</tr>
<tr>
<td>Married</td>
<td>33%</td>
<td>37%</td>
<td>0.32</td>
<td>54%</td>
<td>51%</td>
<td>0.20</td>
</tr>
<tr>
<td>Divorced</td>
<td>10%</td>
<td>14%</td>
<td>0.69</td>
<td>3%</td>
<td>8%</td>
<td>1.74</td>
</tr>
<tr>
<td>Widowed</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2%</td>
<td>1%</td>
<td>0.35</td>
</tr>
</tbody>
</table>

BED – Binge Eating Disorder

Table 4 shows the BUT-A scores (means and standard deviations) of the different subgroups. Patients with BED reported significantly (t-test) higher scores than non-BED patients in the General Severity Index (BUT-A-GSI) and in every subscale of the BUT-A.

Table 4. Means and standard deviations of body uneasiness scores in binge eating disordered and non-binge eating disordered obese patients

<table>
<thead>
<tr>
<th>BUT-A scores</th>
<th>Males</th>
<th></th>
<th></th>
<th>Females</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BED</td>
<td>NON-BED</td>
<td><strong>t</strong></td>
<td>BED</td>
<td>NON–BED</td>
<td><strong>t</strong></td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td></td>
<td>M</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Global Severity Index</td>
<td>2.11</td>
<td>1.33</td>
<td>4.32***</td>
<td>2.83</td>
<td>1.97</td>
<td>5.22***</td>
</tr>
<tr>
<td></td>
<td>1.07</td>
<td>0.93</td>
<td></td>
<td>0.98</td>
<td>1.19</td>
<td></td>
</tr>
<tr>
<td>Weight Phobia</td>
<td>2.57</td>
<td>1.73</td>
<td>4.04***</td>
<td>3.17</td>
<td>2.45</td>
<td>3.84**</td>
</tr>
<tr>
<td></td>
<td>1.22</td>
<td>1.08</td>
<td></td>
<td>1.13</td>
<td>1.36</td>
<td></td>
</tr>
<tr>
<td>Body Image Concerns</td>
<td>3.02</td>
<td>2.06</td>
<td>3.72**</td>
<td>3.71</td>
<td>2.79</td>
<td>4.78***</td>
</tr>
<tr>
<td></td>
<td>1.43</td>
<td>1.44</td>
<td></td>
<td>1.10</td>
<td>1.44</td>
<td></td>
</tr>
<tr>
<td>Avoidance</td>
<td>1.68</td>
<td>0.76</td>
<td>4.63***</td>
<td>2.66</td>
<td>1.47</td>
<td>6.12***</td>
</tr>
<tr>
<td></td>
<td>1.32</td>
<td>0.85</td>
<td></td>
<td>1.28</td>
<td>1.33</td>
<td></td>
</tr>
<tr>
<td>Compulsive Self Monitoring</td>
<td>0.95</td>
<td>0.67</td>
<td>2.03*</td>
<td>1.47</td>
<td>1.11</td>
<td>2.29*</td>
</tr>
<tr>
<td></td>
<td>0.87</td>
<td>0.64</td>
<td></td>
<td>1.06</td>
<td>1.04</td>
<td></td>
</tr>
<tr>
<td>Depersonalization</td>
<td>1.48</td>
<td>0.79</td>
<td>3.65**</td>
<td>2.32</td>
<td>1.32</td>
<td>5.23***</td>
</tr>
<tr>
<td></td>
<td>1.19</td>
<td>0.89</td>
<td></td>
<td>1.26</td>
<td>1.30</td>
<td></td>
</tr>
</tbody>
</table>

*p < .05     **p < .01     ***p < .001

BUT-A – Body Uneasiness Test; BED – Binge Eating Disorder
DISCUSSION

Several investigations found that subjects with BED appear very similar to those with AN or BN in terms of their overvalued ideas regarding weight and shape. In clinical as well as community samples, persons suffering from BED reported significantly greater levels of body dissatisfaction and weight/shape concerns compared to non-BED subjects; this correlation is not explained by the degree of depression (Barry, Grilo, & Masheb, 2003; Masheb & Grilo, 2000; Mussell, Mitchell, de Zwaan, Crosby, Seim, & Crow, 1996a; Mussell, Peterson, Weller, Crosby, de Zwaan, & Mitchell, 1996b; Striegel-Moore, Cachelin, Dohm, Pike, Wilfley, & Fairburn, 2001).

It is well known that such factors as gender, age, onset age of overweight, degree of obesity, ethnicity, social class, all show important modulating effects on body image disturbances in obese persons (Marano et al., 2007; Sarwer & Thompson, 2002; Sarwer et al., 2005; Schwartz & Brownell, 2002).

The empirical investigation we have described in this article was an observational study aimed to verify the following hypothesis: in persons with obesity, BED is associated with greater body uneasiness independently of some possible modulating factors.

In planning our research, we have tried to remove some biases by matching, that is the most direct and intuitive method of adjustment for overt (those that have been accurately measured) biases: matching compares each individual to one control who appears comparable in terms of observed covariates (Rosenbaum, 2005).

In our study, each BED subject was matched exactly to one non-BED control for gender, ethnicity (all Caucasian), and BMI class looking for the best compromise for some other variables: when the recruitment was completed, we did not find significant differences for these variables between BED and non-BED subgroups.

To assess body image disturbance we used the self-report questionnaire BUT. We have chosen this test because it is a valuable multidimensional instrument that was developed with the specific aim of investigating not only body dissatisfaction, but also other dimensions of so-called negative body image: body shape and/or weight dissatisfaction, body image-related avoidance behaviours, compulsive body checking behaviours, detachment and estrangement feelings towards one’s own body, specific worries about particular body parts, shapes or functions (Cuzzolaro et al., 2006). In addition, the BUT was recently validated in Italy in a very large sample of subjects with obesity (Marano et al., 2007).

Obese patients with BED reported higher scores than non-BED patients in the General Severity Index (BUT-GSI) and in every subscale of the BUT-A. All differences were highly significant except one: in the Compulsive Self-Monitoring subscale (CSM) BED patients obtained scores higher than non-BED controls but p
was only < .05 in both sexes. Finally and unsurprisingly, obese women with BED reported higher scores than obese men with BED in the BUT-A-GSI and in every subscale of the BUT-A. We can find a parallel result in Reas et al.’s study (2005): obese women with BED reported significantly greater levels of body checking than obese men with BED.

**Limitations**

The first limitation of this study is the absence of a general population sample of obese subjects with BED and without BED. Next, the absence of clinical and general population samples of non-obese (BMI < 30) subjects with BED. Finally, it could be useful to compare the BUT scores with those obtained by using other body image assessment tools or a diagnostic semi-structured interview, such as Body Dysmorphic Disorder Evaluation, BDDE (Cuzzolaro & Aveni, 2000; Rosen & Reiter, 1996).

**Clinical implications and future research directions**

In accordance with a number of other studies (Hrabosky & Grilo, 2007; Masheb & Grilo, 2000) our findings suggest that a negative body image should be included among diagnostic criteria for BED. A negative body image could also drive obese binge eaters to seek bariatric surgery more often than non-BED obese subjects, other conditions being equal. Colles, Dixon, & O'Brien (2008) wrote that loss of control is central to psychological disturbance associated with BED and suggested that "feelings of loss of control could drive binge eaters to seek bariatric surgery in an attempt to gain control over body weight and psychologically disturbing eating behaviour".

In conclusion, assessment and treatment of persons with obesity and BED should consider cognitive as well as behavioural dysfunctional attitudes and outcome studies should be focused not only on eating behaviour but on body image as well.

**REFERENCES**


Received: 23/07/2008