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(Article begins on next page)
Hypomania across the binge eating spectrum. A study on hypomanic symptoms in full criteria and sub-threshold binge eating subjects

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Abstract

Backgrounds. Obese subjects affected by binge eating can be distinguished between those showing full criteria Binge Eating Disorder (BED) and those who show binge eating of insufficient frequency to satisfy DSM criteria, or sub-threshold BED (s-BED). The present paper aims to investigate whether subjects with BED full criteria show more hypomanic symptoms than those with s-BED, after controlling for personality variables as potential confounders.

Methods. The Hypomania Checklist (HCL-32), the Beck Depression Inventory (BDI) and the Temperament and Character Inventory (TCI) were administered to 103 obese patients with binge eating.

Results. Full criteria BED subjects were more likely to be female and showed higher HCL-32 scores and lower scores in character dimensions (Self-directedness and Cooperativeness) compared to s-BED subjects. A logistic regression with Eating Disorder Diagnosis as outcome measure (BED or s-BED) revealed that lower Cooperativeness, higher Hypomania scores and female sex predicted having BED full criteria.

Limitations. Further research is necessary to replicate these findings in a larger sample.

Conclusions. Patients with more severe binge eating might be more likely to have a comorbid bipolar spectrum disorder. Hypomanic symptoms should be assessed and mood stabilizing treatment should be considered in these patients.

Keywords: Binge eating; Obesity; Hypomania; Bipolar spectrum.
1. Introduction

The Diagnostic and Statistical Manual of Mental Disorders or DSM-IV-TR (American Psychiatric Association, 2000) defines Binge Eating Disorder (BED) as characterized by at least two binge episodes per week for a period of 6 months accompanied by a subjective sense of loss of control over eating. Distress about the binge eating and at least three of five additional criteria are also required. However, many authors argue that obese subjects with binge eating who do not meet the DSM-IV-TR frequency criterion (binge eating for at least two days per week) show impairments in general functioning similar to those experienced by patients with full syndromal BED (Crow et al., 2002 and Striegel-Moore et al., 2000). At the moment it is not clear if BED and s-BED subjects have different psychopathological features, and for example Latner and Clyne state that more research is necessary to further compare the psychopathology and functioning between individuals with full and partial BED (Latner and Clyne, 2008). An area of great clinical importance is that of mood disorder comorbidity.

The relationship of mood disorders with binge eating and obesity has been the subject of much research. Depression has been shown to be frequently associated with obesity (Simon et al., 2006), and it often presents with co-morbid binge eating (S. Pagoto et al., 2007); these conditions are both known to lead to poorer results in weight loss programs (Pagoto et al., 2007). Less attention has been devoted to the presence of symptoms of the bipolar spectrum in obese patients. Bipolar spectrum disorders are thought to be very frequent in subjects with eating disorders, and according to the work of McElroy et al. (2005), when exploring broad diagnostic criteria (“spectrum” conditions) of the two groups of disorders, intriguing analogies emerge with regards to phenomenology, course, family history, biology and treatment response. These authors have proposed that bipolar disorders and eating disorders might be separate but pathophysiologically related conditions, and suggested that certain eating disorders might be more likely to be associated with bipolar disorders than others. For example, hypomania has been found to co-occur specifically with binge eating behavior more often than expected by chance alone (Angst, 1998 and Gamma et al., 2008).

An internationally validated psychometric instrument of recent creation, the Hypomania Checklist (HCL-32) (Angst et al., 2005) has been used in a population of obese subjects seeking surgical treatment, the authors found a hypomanic condition characterized by overactivity to be very common in this patient population (Alciati et al., 2007). The study of the relation between mood disorders and eating behavior is further complicated by the fact that there might be some overlap between extreme personality traits and mood disorder symptoms. For example, Grucza et al. (2007) studied personality traits in BED obese subjects and found that BED symptoms were associated with high Novelty Seeking, high Harm Avoidance and low Self-directedness. Given that Novelty Seeking consists of lower order traits like Exploratory Excitability, Impulsiveness, Extravagance and Disorderliness (Svrakic et al., 1993), it is possible that the behavioral activation found in some BED subjects might be an expression of a stable personality trait like Novelty Seeking with a score in the higher range instead of being a sign of mood instability. Therefore, while assessing hypomania in patients with binge eating, it might be useful to integrate instruments that target the episodic nature of hypomania like the HCL-32 with others that assess stable personality traits like the TCI.

The present study aims to investigate the prevalence of hypomania in BED and s-BED patients as assessed with the Hypomania Checklist, controlling for personality traits assessed with the TCI as potential confounding variables. Based on previous findings linking binge eating and hypomania (Angst, 1998, Gamma et al., 2008 and Wittchen et al., 2003), we hypothesize that full criteria BED subjects, characterized by more frequent binge eating, might exhibit more hypompanic symptoms than subjects with sub-threshold BED. We will then discuss the implication of the results for the pharmacological treatment of these patients.
2. Methods

2.1. Subjects

The patients included in the study came to our Eating Disorders Centre during the year 2009 seeking treatment for obesity and binge eating. The following inclusion criteria were used: obesity (Body Mass Index > 30) with binge eating (defined as a score higher than or equal to 17 at the Binge Eating Scale), age 18 or older, and willingness to take part to the study. The study procedure was carefully explained and informed consent was obtained from all the subjects. We excluded from the study the subjects who had a history of Axis I psychiatric disorders other than depression, like patients with a diagnosed, full-blown bipolar disorder, in order to achieve a higher degree of sample homogeneity and avoid confounding effects. Of 125 patients initially considered, 103 obese patients were enrolled in the study. 16 patients (12.8%) were excluded because they did not satisfy the criteria for BED nor for s-BED diagnosis; 4 were excluded (3.2%) because they had been diagnosed with a bipolar spectrum disorder before coming to our Centre; 2 (1.6%) did not complete all the questionnaires.

2.2. Assessment

All patients were assessed by a psychiatrist, who diagnosed BED in patients who fulfilled all the criteria according to the DSM-IV-TR (American Psychiatric Association, 2000), and who diagnosed s-BED when all the criteria were satisfied except the frequency criterion. Self-rating assessment instruments were also used: all patients completed the Binge Eating Scale (Gormally et al., 1982) assessing binge eating, the Temperament and Character Inventory (Svrakic et al., 1993) assessing personality and the Beck Depression Inventory (Beck and Steer, 1987) providing a measure of depressive symptoms. Informed consent was obtained from all the subjects after explaining the objectives of the study.

2.3. Statistical analysis

A comparison between BED and s-BED subjects was performed to test for differences in socio-demographic and clinical features. For between-groups comparisons of categorical variables, Chi-square was used. ANCOVA was used to compare continuous psychopathological variables controlling for sex. In order to rule out confounding effects between mood and personality variables, a logistic regression model was computed with eating disorder diagnosis (BED and s-BED) as outcome and clinical and psychopathological variables as predictors. Finally, the presence of a direct correlation between binge eating scores (BES) and Hypomania scores (HCL-32) was tested with Pearson's method. A significance level of p < 0.05 was used; all analyses were performed with PASW Statistics.

3. Results

3.1. Diagnosis

Patients with BED were 44 (43%), while 59 patients (57%) were classified as s-BED. Demographic characteristics of the two groups are reported in Table 1. Only sex was found to significantly differ in the two groups (p = 0.01), with more males in the s-BED category.
Table 1. Demographic and clinical characteristics of BED and s-BED patients.

<table>
<thead>
<tr>
<th></th>
<th>BED</th>
<th>s-BED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>41 ± 13</td>
<td>44 ± 12</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>1/43 (2.3%)*</td>
<td>11/48 (18.6%)*</td>
</tr>
<tr>
<td>Years of education (mean ± SD)</td>
<td>11 ± 3</td>
<td>11 ± 3</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>36.6 ± 4.9</td>
<td>38.4 ± 5.9</td>
</tr>
<tr>
<td>Years since onset of Weight gain</td>
<td>15 ± 11</td>
<td>18 ± 10</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>59</td>
</tr>
<tr>
<td>BES</td>
<td>28.7 ± 7.6**</td>
<td>23.0 ± 10.3**</td>
</tr>
</tbody>
</table>

BED = Binge Eating Disorder; BMI = Body Mass Index.
*χ² = 6.5; p = 0.01.
**ANCOVA results: F = 7.46; p < 0.007. Sex was used as a covariate but did not show significant interactions (F = 1.25; p = 0.26).
Comparisons between other variables were not significant.

3.2. Comparing BED and s-BED subjects for eating and general psychopathology

Patients with BED and s-BED diagnoses were compared regarding HCL-32, TCI, BES and BDI scores, the results are shown in Table 2. Patients with BED full criteria have lower scores in Self-directedness and Cooperativeness, and higher HCL-32 scores. Sex was used as a covariate but statistically significant interactions were not noted.

Table 2. Differences between BED and s-BED subjects in personality and psychopathology controlling for sex as a covariate (ANCOVA).

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD</th>
<th>F</th>
<th>Significance (2-tailed)</th>
<th>Sex as covariate</th>
</tr>
</thead>
<tbody>
<tr>
<td>TCI-NS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BED</td>
<td>20.6 ± 5.8</td>
<td>F = 0.82</td>
<td>p = 0.36</td>
<td>p = 0.22</td>
</tr>
<tr>
<td>s-BED</td>
<td>19.8 ± 5.5</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>TCI-HA</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>BED</td>
<td>24.9 ± 6.0</td>
<td>F = 0.88</td>
<td>p = 0.35</td>
<td>p = 0.068</td>
</tr>
<tr>
<td>s-BED</td>
<td>23.2 ± 5.4</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>TCI-RD</td>
<td></td>
<td></td>
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<tr>
<td>BED</td>
<td>16.4 ± 3.5</td>
<td>F = 1.64</td>
<td>p = 0.20</td>
<td>p = 0.10</td>
</tr>
<tr>
<td>s-BED</td>
<td>15.1 ± 3.5</td>
<td></td>
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<tr>
<td>TCI-P</td>
<td></td>
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<tr>
<td>BED</td>
<td>4.3 ± 2.3</td>
<td>F = 0.04</td>
<td>p = 0.95</td>
<td>p = 0.96</td>
</tr>
<tr>
<td>s-BED</td>
<td>4.3 ± 1.8</td>
<td></td>
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<tr>
<td>TCI-SD</td>
<td></td>
<td></td>
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<tr>
<td>BED</td>
<td>19.0 ± 7.5</td>
<td>F = 4.85</td>
<td><strong>p = 0.030</strong></td>
<td>p = 0.44</td>
</tr>
<tr>
<td>s-BED</td>
<td>22.3 ± 6.3</td>
<td></td>
<td></td>
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<tr>
<td>TCI-CO</td>
<td></td>
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<tr>
<td>BED</td>
<td>28.2 ± 7.2</td>
<td>F = 5.38</td>
<td><strong>p = 0.022</strong></td>
<td>p = 0.22</td>
</tr>
<tr>
<td>s-BED</td>
<td>30.9 ± 6.0</td>
<td></td>
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<tr>
<td>TCI-ST</td>
<td></td>
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</tr>
<tr>
<td>BED</td>
<td>13.5 ± 6.6</td>
<td>F = 0.01</td>
<td>p = 0.89</td>
<td>p = 0.26</td>
</tr>
<tr>
<td>s-BED</td>
<td>13.3 ± 6.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BED</td>
<td>15.7 ± 7.4</td>
<td>F = 0.50</td>
<td>p = 0.48</td>
<td>p = 0.23</td>
</tr>
<tr>
<td>s-BED</td>
<td>14.3 ± 6.5</td>
<td></td>
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<tr>
<td>HCL-32</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>BED</td>
<td>17.1 ± 3.9</td>
<td>F = 5.59</td>
<td><strong>p = 0.020</strong></td>
<td>p = 0.22</td>
</tr>
<tr>
<td>s-BED</td>
<td>15.3 ± 4.2</td>
<td></td>
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</tbody>
</table>

TCI = Temperament and Character Inventory; NS = Novelty Seeking; HA = Harm Avoidance; RD = Reward Dependence; P = Persistence; SD = Self-directedness; C = Cooperativeness; ST = Self-transcendence; BDI = Beck Depression Inventory; HCL-32 = Hypomania Checklist.
3.3. Correlations

A logistic regression model was then calculated, entering personality variables, depression and Hypomania scores and sex as predicting variables and eating disorder diagnosis (BED and s-BED) as outcome, in order to control for confounding between personality traits and hypomania. In the regression model, Female sex (p = 0.046), Cooperativeness (p = 0.044) and HLC-32 (0.023) scores were significantly associated with a diagnosis of BED full criteria (R2 = 0.20). Self-directedness lost significance in the logistic regression model (p = 0.3).

A significant correlation (r = 0.23; p = 0.016) was also found between HCL-32 and BES.

4. Discussion

To our knowledge, this is the first study evaluating differences in hypomanic symptoms between BED and s-BED patients.

Patients with full criteria BED showed significantly higher HCL-32 scores compared to patients with sub-threshold binge eating, while depressive symptoms do not seem to differ significantly between the two groups. Hypomanic symptoms also show a direct correlation with binge eating severity. These results are consistent with the work of several authors. Perugi and Akiskal (2002) proposed that affective instability, which characterizes bipolar spectrum disorders, might be a particularly potent predictor of binge eating. Greenberg and Harvey in a previous study found that the interaction between dietary restraint and biphasic mood shifts was a better predictor of the severity of binge eating than the interaction between dietary restraint and depression, thus suggesting a central role of affective instability in determining the degree of binge eating behavior (Greenberg and Harvey, 1987).

Some differences in character could also be found between the two groups, with binge eating subjects showing lower Cooperativeness. These values indicate that BED subjects appear to be less empathetic, tolerant, compassionate and supportive compared to obese subjects with less frequent binge eating. The results of the logistic regression analysis suggest that personality dimensions do not account for the differences in hypomania rates. Thus, the higher hypomania rates in BED subjects does not appear to map upon similar stable psychological traits (e.g. Novelty Seeking) but suggests a dimension of mood instability independent of trait personality features assessed by the TCI.

Subjects with s-BED are known to suffer from a degree of functional impairment resulting from the eating disorder comparable to that of those diagnosed with BED full criteria and they also share with BED subjects personality traits and psychopathological features that differ from those of obese non eating-disordered subjects (Fassino et al., 2002, Fassino et al., 2003 and Grucza et al., 2007). However, their profile of mood dysregulation seems to differ from that of subjects with more severe binge eating. This finding could have relevant clinical implications regarding pharmacological treatment.

Three classes of drugs have been studied in randomized controlled trials for Binge Eating Disorder: antidepressants, antiepileptic drugs and obesity drugs (Reas and Grilo, 2008). The literature on the subject is still relatively small, and it is still unclear which patient is most likely to benefit the most from which kind of drug. Ideally, the most parsimonious treatment for patients suffering from co-morbid eating and bipolar spectrum disorders would entail using a drug which is effective in treating both syndromes (McElroy et al., 2006). Our preliminary results suggest that future clinical studies should investigate whether treatment of BED obese subjects with a severe degree of binge eating and a higher prevalence of hypomanic symptoms with a mood stabilizing agent can lead to a better control of binge eating through mood stabilization and the amelioration of affective instability compared to
antidepressant treatment. Side effects like mood elevation, dysphoria and restlessness should also be assessed during treatment with antidepressants in order to determine if they are more frequent in patients with BED than in s-BED subjects, because the higher frequency of bipolar spectrum symptoms in BED subjects might entail a greater risk of mood elevation as a result of antidepressant treatment.

Prospective studies are also needed to determine whether higher levels of hypomania can lead in time to a higher risk of binge eating in the general population.

Other implications and possibilities for research regard the metabolic comorbidities of obese binge eating patients. HPA axis hyperactivity has been proposed as a biological correlate of bipolar disorder, and it also appears to be associated with binge eating (Kendler et al., 1996). Obese patients with BED and bipolar spectrum symptoms might be more at risk of metabolic comorbidities of obesity compared to s-BED subjects. Bipolar patients in general seem at a particularly high risk of developing obesity (Maina et al., 2008) and of developing the metabolic syndrome, as many studies from a number of different countries demonstrate (McIntyre et al., 2010 and Salvi et al., 2008).

Some limitations need to be considered when interpreting these results. Grouping patients in BED and s-BED categories on the basis of the frequency of binge eating is a choice that reflects current DSM-IV-TR diagnostic criteria that have been challenged by the research literature: broader criteria are probably needed in order to include in treatment programs patient who suffer from a significant degree of psychopathology and functional impairment (Latner and Clyne, 2008). A more precise description of the psychopathology of BED and s-BED patients seems nonetheless useful to establish psychopathological comorbidities as targets for pharmacological treatments. Furthermore, the sample size of the study renders necessary confirmation in other patient populations to assess if the present results can be generalized.

In conclusion, the present findings highlight the presence of more severe hypomaniac symptoms in obese BED patients compared to s-BED patients. Longitudinal interventional trials investigating the impact of currently available antidepressant and mood stabilizing drugs on the two groups of patients are needed to provide more specific indications for the pharmacological treatment of Binge Eating Disorder.

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This study did not receive specific funding.

Conflict of interest
The authors declare no conflict of interest.

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