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Research Article

# Effectiveness of cognitive behavioural psychotherapy alone and combined with pharmacotherapy in binge eating disorder: a differential research.

**Lanzarone Cristina, Cuzzocrea Francesca, Larcan Rosalba, Bongiorno Antonio and Minì Valentina**

**Abstract**

**Objective:** This research aimed to verify the differences between patients with Binge Eating Disorder (BED) treated with Cognitive Behavioural Therapy (CBT) alone and those treated with CBT in combination with medication.

**Method:** A selection of 30 subjects affected by BED was carried out on the basis of experimental outcomes that evidenced the efficacy of Selective Serotonin Reuptake Inhibitors (SSRIs) and Serotonin-Noradrenaline Reuptake Inhibitors (SNRIs) in the treatment of obesity associated with BED. Some participants underwent CBT alone while the others, in addition to CBT, were treated with bio-equivalent doses of either Paroxetine or Venlafaxine. Binge eating behaviour, impulse regulation, different features of eating behaviour (restrained eating, uncontrolled eating and emotional eating) and different psychotic conditions (Psychopathic Deviate, Depression and Hypomania) were studied during pre- and post-treatment phases. Both the psychological (CBT) and the pharmacological (CBT + SSRIs/SNRIs) therapies were assessed in keeping with the parameters examined.

**Results:** The data showed that CBT alone seems to favour a greater reduction in depression and hypomania as well as the subject's ability to control eating behaviour; whereas pharmacological treatment appears to control primarily the impulsiveness of food intake.

**Keywords**: Eating Behaviours, Bing Eating Disorder, Personality, Cognitive-Behavioural Therapy, Pharmacotherapy

## Introduction

Binge Eating Disorder (BED) was first defined by Stunkard in 1959[1]; he identified peculiar food intake features characterized by a loss of control in a subgroup of obese patients. Various efforts have been made, ever since, to provide a non-sociological approach to individuals with such a behaviour disorder, which has long been considered a variant of Bulimia Nervosa.

Unlike patients affected by Bulimia Nervosa, patients with BED appear to be overweight and mainly obese. Thus, the treatment aims not only at reducing BED and its related psychopathology, but also at assessing the weight gain experienced by these patients to prevent a further worsening of physical health.

Walsh & Devlin[2] evaluated the use of medication in the treatment of Bulimia Nervosa and BED, underlining the efficacy of antidepressant medication in the treatment of Bulimia Nervosa. The antidepressant efficacy led to consider its use in BED more accurately.

Williamson, Martin & Stewart[3] stated that pharmacotherapy was not an effective treatment for Anorexia Nervosa. However, it did prove to be successful in Bulimia Nervosa and BED, although subjects affected with eating disorders apparently respond better to psychotherapy approaches.

Systematic investigations have been conducted on the aetiology of BED. Biological and genetic factors, neurotransmitters and hormones have been involved in the onset of binge eating and play an important role in the regulation of hunger and mood.[4, 5] However, a definitive aetiological theory has not been developed and tested.[3]

BED is characterized by a relevant psychological component that in many cases is under-evaluated. Patients with BED have difficulty in interpreting the visceral sensations of hunger and satiety; they take large amounts of food even during regular meals and, moreover, their food contains more fat than protein.[6, 7]

In fact, Axis I and II disorders (DSM IV-TR) share common features with binge eating.[8] Axis I psychiatric disorders (including depression, anxiety, body dysmorphic disorder, or chemical addiction) characterize many BED patients, and research has evidenced the presence of panic, loss of control, impulsivity, compulsive behavior, obsessive thoughts about food and social phobia.[9] Axis II personality disorders (especially borderline personality disorder) are frequently related to patients suffering from eating disorder and comorbidity with Avoidant Personality Disorder and Obsessive-Compulsive Disorder was observed.[10]

BED is not associated with a restrained eating control, but probably with an increase of uncontrolled eating and emotional eating.[11, 12]

Pharmacological agents, compared to placebo, have been used in the treatment of BED. Appolinario, Bacaltuchuk, Sichieri et al[13] evaluated the efficacy of Sibutramine to reduce the frequency of binge eating, while McElroy, Arnold, Shapira et al[14]focused on Topiramate and evidenced a greater reduction in binge eating frequency but with side effects such as paraesthesia.

Other studies showed the efficacy of Selective Serotonin Reuptake Inhibitors (SSRIs) and Serotonin-Noradrenaline Reuptake Inhibitors (SNRIs) in the treatment of obesity associated with BED[15, 16, 17, 18] showing that SSRIs and Tricyclic Antidepressants (TCAs) reduced the frequency of BED compared to placebo.[19] To determine which patients are most likely to benefit from medications, and how to sequence the various therapeutic interventions available better, are questions still open to debate.

Moreover, results in many cases appear to be divergent. Ciao, Latner and Durso[20] underlined the influences of other factors that may influence treatment efficacy. They observed that many obese individuals who might benefit from weight loss treatment nevertheless do not plan or desire to seek treatment and perceive multiple barriers to treatments.

Pharmacotherapy may enhance weight loss,[19] although other results suggest that pharmacotherapy may be associated with a reduction in binge frequency in obese patients with BED, but it does not lead necessarily to weight reduction.[21] These medical treatments seem to be effective in reducing binge frequency over the short-term, and the subsequent discontinuation of the medication seems to be associated with a relapse of binge eating. Thus further studies of the role of pharmacotherapy in the treatment of BED need to be carried on.[22]

According to Williamson, Martin and Stewart[3] the additive effects of psychotherapy, e.g. Cognitive Behavioural Therapy (CBT), and pharmacotherapy have to be investigated. At present it seems that adding pharmacotherapy to psychotherapy does not help to reduce binge frequency compared to psychotherapy alone.

The efficacy of CBT has been substantiated by scientific literature[23, 24, 25, 26] and *Vaidya*[27] *stating that* CBT helps patients reduce eating disorder habits by making them aware of the cause of their self-sabotage, while affecting weight indirectly.

Psychotherapy treatment over a one-year period deals with binge symptoms and aims at reducing the possibility of relapse by gathering different techniques for the maintenance of long-term results through the use of specific individual intervention protocols. The main target of the intervention is to facilitate the management of no-control food intake episodes and of impulsivity through the alteration of behaviour, and of cognitive and emotional factors related to eating disorders.

Thus, the main objective of this research was to verify the possible differences between those subjects with BED who underwent psychotherapy combined with pharmacotherapy and those who underwent psychotherapy only. In particular, it aimed at verifying possible differences between the various therapeutic strategies on eating behaviour (restrained eating, uncontrolled eating and emotional eating) and the behavioural and psychopathological features (psychopathic deviate, depression and hypomania).

The main hypothesis was to determine if patients who underwent CBT and pharmacotherapy with bio-equivalent doses of the SSRI Paroxetine or SNRI Venlafaxine obtained a considerable benefit from the pharmacotherapy on impulse regulation, on eating behavior and on personality features compared to those who underwent CBT alone.

The second hypothesis was to verify if Paroxetine and Venlafaxine treatments were equally effective on impulse regulation, eating behavior and on personality characteristics.

**Methodology**

Participants

A group of 30 subjects with BED were selected. All these subjects applied for support to the Inter-Service-Psychology Clinic for Eating Disorders and they were assisted by a Cognitive Behavioural Therapist. They were all of Italian nationality, aged 22 to 52, with a Body Mass Index (BMI) range of 26 to 35. All participants belonged to a middle class socio-cultural level. They were informed of the objectives of the research and signed a consent form. Those subjects who were diagnosed with binge eating less than two years ago, those aged over 65, or those who were suffering from other debilitating or chronic diseases were preliminarily excluded from this research.

Measures and procedure

Participants were recruited according to the nature of the assessments. More specifically, assessments had to address the effect of psychotherapy and pharmacotherapy in subjects with BED on their impulse regulation, their eating behavior (restrained eating, uncontrolled eating and emotional eating), and their personality features (Psychopathic Deviate, Depression and Hypomania). The 30 subjects selected were randomly assigned to three different treatments. Ten subjects had only CBT, ten subjects underwent psychotherapy with Paroxetine, and ten subjects underwent psychotherapy with Venlafaxine.

Each participant answered questionnaires during the assessment phase and in the post-training phase (after one year of psychotherapy). More specifically:

*Binge Eating Scale (BES)*[28] is a 16-item questionnaire which assesses the presence of binge eating behaviour indicative of an eating disorder. The score ranges from 0 to 46 (non-binging <17; moderate binging = 18-26; severe binging = 27 and higher), which in this research had an adequate internal consistency (a = 0.84).

*Eating Disorder Inventory-2 (EDI-2)*[29] aims at quantifying some psychological and behavioural features. It consists of 64

questions grouped into 11 scales. For each item, the participants are asked to answer by using the following frequency adverbs: "always", "usually", "often", "sometimes", "rarely", and "never". The rating is measured with a score between 0 and 3: the maximum score of 3 corresponds to the intensity of the symptom ("always" or "never" depending on whether the direction of the item is positive or negative), score 2 corresponds to a degree intensity immediately below ("usually" or "rarely"), score 1 to an even lower level of intensity ("often" or "sometimes"), while a score of 0 is assigned to the three "asymptomatic" answers. So, those items with a positive direction are assigned the following scores: always = 3, usually = 2, often = 1, sometimes = 0, rarely = 0, never = 0; items with a negative direction are evaluated in the opposite way: never = 3, rarely = 2, occasionally = 1, and often, usually, always = 0. The sub-scale scores are calculated by simply adding all the scores of items of each specific sub-scale.

This research availed the *Impulse Regulation Scale* with an adequate internal consistency (a = 0.82). This scale shows the ability to regulate impulsive behaviour, especially binge behaviour.

*Three Factor Eating Questionnaire (TFEQ)*[30] is a self-report questionnaire consisting of 51 items. The questionnaire refers to daily dietary practice and measures three different aspects of eating behaviour: (1) restrained eating (conscious restriction of food intake in order to control body weight or to promote weight loss – cut-off: ≤11; a = 0.79); (2) uncontrolled eating (tendency to eat more than usual due to a loss of control over intake accompanied by subjective feelings of hunger – cut-off: ≤8; a = 0.81): (3) emotional eating (inability to resist emotional cues – cut-off: ≤7; a = 0.83).

*Minnesota Multiphasic Personality Inventory-2 (MMPI-2)*[31] consisting of 567 items with dichotomy answers (true/false) is most commonly used by mental health professionals to assess and diagnose mental illness. The MMPI is based on ten clinical scales that are used to indicate different psychotic conditions. In this research the scoring of the three following scales were taken into consideration: (1) Psychopathic Deviate Scale (Pd) (50 items) which measures social deviation, lack of acceptance of authority and amorality. This scale can be thought of as a measure of disobedience. High scorers tend to be more rebellious, while low scorers are more likely to accept authority. An adequate internal consistency was obtained in this research (a = 0.83); (2) Depression Scale (D) (57 items). The highest scores may indicate depression, while moderate scores tend to reveal a general dissatisfaction with one's own life. A sound internal consistency was obtained through this research (a = 0.81); (3) Hypomania Scale (H), with 46 items, identifies such characteristics of hypomania as elevated mood, accelerated speech, locomotive activity, irritability, flight of ideas, and short periods of depression. In this research the internal consistency was a = 0.79.

**Results**

The Statistical Package for Social Science (SPSS 10.1) was implemented to verify the hypothesis. The limited number of subjects enabled analysis of data through non-parametric statistics. In order to verify statistical differences between simple comparisons on paired data the Mann-Whitney (U) test[32] was applied. In order to verify statistical differences within phases (pre- Vs post-training), Wilcoxon Signed Ranks Tests[33] were calculated separately on paired data.

Table 1 synthesizes the means and standard deviations of *eating behaviour* and of *impulse regulation* obtained by the three groups: CBT alone; psychotherapy with Paroxetine (CBT+P); and psychotherapy with Venlafaxine (CBT+V) in pre- and post-treatments.

By comparing the total scoring in *BES* during the pre-treatments phase, no statistical differences between groups were noticed. Subjects who only underwent CBT had the same result than those who had addition of Paroxetine (CBT+P) [U = 64; Z = 0.35; p = 0.75] and Venlafaxine (CBT+V) [U = 59; Z = 0.62; p = 0.55]. There were no initial statistical differences between the two groups that received pharmacotherapy [U = 50; Z = 0.1; p = 0.99].

In the post-treatment phases, the presence of binge eating behaviour appeared to be the same in all groups. Subjects belonging to the CBT group obtained the same results as those belonging to the CBT+P [U = 58; Z = 0.68; p = 0.5] and the CBT+V groups [U = 47; Z = 1.36; p = 0.19]. No statistical differences between medication use were noticed [U = 41; Z = 0.69; p = 0.53].

All groups in post-treatment phases seem to equally benefit from the treatment. Comparing scores obtained by participants in the pre- and post-treatments, statistically significant differences were found in subjects undergoing CBT [Z=3.38, p < 0.001] and those with the addition of Paroxetine [Z = 2.848; p < 0.004] and Venlafaxine [Z = 2.859, p < 0.004].

For this research, *Impulse Regulation Scale* scores were taken into consideration. In the pre-treatment phase relative to each group, all groups showed the same difficulties to regulate impulsive behaviour. The CBT group showed the same impulse regulation as those belonging to the CBT+P [U = 68; Z = 0.12; p = 0.93] and CBT+V group [U = 64; Z = 0.33; p = 0.75]. No initial statistical differences between pharmacotherapy groups were found [U = 45; Z = 0.39; p = 0.74].

In post-treatment, no statistical differences between groups were observed. The CBT group achieved the same results as the CBT+P [U = 60; Z = 0.56; p = 0.58] and CBT+V group [U = 64; Z = 0.32; p = 0.75]. No statistical differences between the use of Paroxetine and Venlafaxine were found either [U = 39; Z = 0.84; p = 0.44].

| Groups | Phases | *Binge Eating Disorder* | | | | *Impulse Regulation Scale* | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | MIN | MAX | M | SD | MIN | MAX | M | SD |
| CBT (N=10) | Pre | 28 | 35 | 31.43 | 2.41 | 74 | 94 | 85.93 | 6.84 |
| | Post | 25 | 33 | 28.71 | 2.46 | 71 | 91 | 83.07 | 6.67 |
| CBT+P (N=10) | Pre | 26 | 35 | 30.90 | 3.54 | 77 | 94 | 86.80 | 5.29 |
| | Post | 24 | 31 | 27.90 | 2.85 | 74 | 91 | 82.10 | 5.72 |
| CBT+V (N=10) | Pre | 27 | 35 | 30.80 | 2.78 | 83 | 94 | 87.80 | 3.91 |
| | Post | 24 | 31 | 27.20 | 2.57 | 79 | 90 | 83.80 | 3.71 |

**Table 1** – Minimum and maximum scores, Means and Standard Deviations of eating behaviour and of impulse regulation obtained by three differential groups.

| Groups | Scales | Pre-Treatment | | | | Post-Treatment | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | MIN | MAX | M | SD | MIN | MAX | M | SD |
| CBT | restrained eating | 5 | 9 | 6.64 | 1.28 | 5 | 8 | 5.86 | .95 |
| | uncontrolled eating | 13 | 16 | 14.71 | 1.07 | 11 | 15 | 12.93 | 1.21 |
| | emotional eating | 8 | 13 | 9.93 | 1.21 | 7 | 11 | 8.57 | 1.22 |
| CBT+P | restrained eating | 5 | 9 | 6.50 | 1.43 | 5 | 7 | 6.00 | .82 |
| | uncontrolled eating | 13 | 16 | 14.10 | 1.11 | 10 | 13 | 11.60 | 1.07 |
| | emotional eating | 8 | 13 | 10.70 | 1.64 | 8 | 11 | 9.80 | 1.03 |
| CBT+V | restrained eating | 5 | 8 | 6.10 | 1.11 | 4 | 7 | 5.50 | .85 |
| | uncontrolled eating | 12 | 16 | 13.90 | 1.37 | 10 | 13 | 11.40 | 1.17 |
| | emotional eating | 9 | 13 | 10.60 | 1.43 | 7 | 11 | 9.10 | 1.19 |

**Table 2** – Minimum and maximum scores, Means and Standard Deviations of different aspects of eating behavior (restrained eating, uncontrolled eating and emotional eating) obtained by three differential groups

| Groups | Scale | Pre-Treatment | | | | Post- Treatment | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | MIN | MAX | M | SD | MIN | MAX | M | SD |
| CBT | Psychopathic Deviate (Pd) | 68 | 85 | 74.14 | 5.02 | 60 | 80 | 66 | 5.38 |
| | Depression (D) | 63 | 81 | 72.50 | 5.58 | 61 | 78 | 69.86 | 5.39 |
| | Hypomania (H) | 39 | 75 | 62 | 8.15 | 41 | 72 | 59.50 | 7.29 |
| CBT+P | Psychopathic Deviate (Pd) | 70 | 84 | 74.80 | 3.97 | 67 | 80 | 71.40 | 3.72 |
| | Depression (D) | 66 | 76 | 70.80 | 3.91 | 64 | 74 | 67.80 | 3.58 |
| | Hypomania (H) | 46 | 70 | 60.30 | 7.94 | 40 | 63 | 54.10 | 8.08 |
| CBT+V | Psychopathic Deviate (Pd) | 70 | 85 | 76.20 | 5.41 | 66 | 80 | 72.90 | 5.06 |
| | Depression (D) | 65 | 80 | 70.80 | 4.76 | 61 | 75 | 66.60 | 4.69 |
| | Hypomania (H) | 42 | 72 | 60.10 | 10.67 | 41 | 69 | 54 | 10.27 |

**Table 3** – Minimum and maximum scores, Means and Standard Deviations of different aspects of Psychopathic Deviate obtained by three differential groups

All groups seemed to benefit from the treatments. In fact, comparing the scores obtained by participants in the pre- and post-treatments, statistically significant differences were observed in subjects who underwent CBT [$Z = 3.38$, $p < 0.001$] as well as in subjects supported by Paroxetine [$Z = 2.84$; $p < 0.005$] and Venlafaxine [$Z = 2.97$, $p < 0.003$].

Table 2 synthesizes the means and standard deviations of different features of eating behavior (restrained eating, uncontrolled eating and emotional eating)showed by the three groups (CBT, CBT+P, and CBT+V) in pre- and post-treatment.

In eating behaviour as well, all groups in pre-treatment phases appeared to be equivalent. The CBT group had the same mean result in restrained eating than those subjects who also underwent pharmacotherapy [CBT+P: $U = 65$; $Z = 0.33$; $p = 0.74$; and CBT+ V: $U = 53$; $Z = 1.03$; $p = 0.55$]. In the pre-

training no statistical differences between groups with pharmacotherapy were noticed [U = 42; Z = 0.59; p = 0.58].

By analyzing the groups in pre-treatment phases no statistical differences in uncontrolled eating behaviour and emotional eating behaviour were found. In pre-treatment phase, the CBT subjects had the same statistical mean in uncontrolled eating [U = 48; Z = 1.33; p = 0.21] and in emotional eating [U = 48; Z = 1.32; p = 0.21] as those who took Paroxetine. No statistical differences were found when comparing CBT subjects with those that were taking Venlafaxine [uncontrolled eating: U = 48; Z = 1.33; p = 0.21; emotional eating: U = 48; Z = 1.32; p = 0.21]. In the pre-training phase there were no statistical differences between groups with pharmacotherapy [uncontrolled eating: U = 46; Z = 1.44; p = 0.17; emotional eating: U = 51; Z = 1.12; p = 0.28] were found.

All groups showed indistinctively less difficulty on restrained eating habits. In fact, by comparing post-training scores, the participants of CBT obtained the same results as those treated with Paroxetine [U = 61; Z = 0.56; p = 0.62] and Venlafaxine [U = 58; Z = 0.75; p = 0.51]. Therefore CBT alone appeared to be less effective on reducing uncontrolled eating than those with the addition of Paroxetine [U = 30; Z = 2.4; p < 0.02] and Venlafaxine [U = 26; Z = 2.6; p < 0.009]. Participants who underwent only CBT presented with less difficulties on emotional eating control than those with Paroxetine [U = 31; Z = 2.31; p < 0.02], but they achieved the same post-treatment score as those supported by Venlafaxine [U = 52; Z = 1.08; p = 0.31].

Comparing post-treatment outcomes, the effectiveness of Paroxetine and Venlafaxine appeared to be the same on restrained eating behaviour [U = 34; Z = 1.2; p = 0.25], on a better controlled eating behaviour [U = 45; Z = 0.39; p = 0.69] and on a higher emotional eating control behavior [U = 33; Z = 1.29; p = 0.2].

Comparing pre- and post-treatment results helped to observe a significant improvement in all groups. Participants who followed only CBT showed less difficulty to restrained eating behaviour [Z = 2.6; p < 0.009] in post-treatment. The same results were observed in those supported by Venlafaxine [Z = 2.12; p < 0.03], while no statistical differences were detected in a post-treatment phase in those groups supported by Paroxetine [Z = 1.89; p = 0.06].

Moreover it was possible to observe a considerable decrease in uncontrolled eating behaviour in all groups [CBT: Z = 3.49; p < 0.0001; CBT+P: Z = 2.84; p < 0.005; CBT+V: Z = 2.88; p < 0.004]. The same results were observed in the way emotional eating was handled. All groups benefited from treatments [CBT: Z = 3.27; p < 0.001; CBT+P: Z = 2.46; p < 0.01; CBT+V: Z = 2.87; p < 0.004].

Table 3 synthesizes means and standard deviations of Psychopathic Deviate (Pd), Depression (D) and Hypomania scales (H) obtained by the three groups (CBT, CBT+P, and CBT+V) in pre- and post-treatments.

As evidence of the homogeneity of the groups, the comparisons revealed no statistically significant differences in the pre-treatment phase. The CBT group subjects and those who received Paroxetine [Pd: U = 58; Z = 0.67; p = 0.51; D: U = 55; Z = 0.85; p = 0.39; H: U = 63; Z = 0.41; p = 0.71] showed similar scores. Likewise, the CBT group subjects and those treated with Venlafaxine [Pd: U = 53; Z = 0.99; p = 0.34; D: U = 58; Z = 0.71; p = 0.51; H: U = 65; Z = 0.26; p = 0.79] showed similar scores. The two groups treated subsequently with pharmacological support showed similar initial scores as well [Pd: U = 44; Z = 0.46; p = 0.68; D: U = 50; Z = 0.1; p = 0.99; H: U = 44; Z = 0.45; p = 0.68].

Comparing the results obtained in the post-treatment phase instead, those participants exposed to CBT alone showed a greater reduction of Pd compared to those who had taken Paroxetine [U = 23; Z = 2.76; p < 0.005] and Venlafaxine [U = 23; Z = 2.77; p < 0.005], whereas no differences were found comparing the scores obtained post-treatment in both groups of subjects with pharmacological treatments [U = 41; Z = 0.65; p = 0.53].

In post-treatment, the CBT group participants showed similar scores when compared to those taking Paroxetine [D: U = 54; Z = 0.91; p= 0.37; H: U = 41; Z = 1.67; p = 0.09] and Venlafaxine [D: U = 44; Z = 1.49; p = 0.14; H: U = 49; Z = 1.2; p = 0.23]. There have been no further significant differences in scores obtained post-treatment by the two pharmacotherapy groups [D: U = 39; Z= 0.84; p = 0.44; H: U = 0.47; Z = 0.23; p = 0.85].

All participants seem to have benefited from the proposed treatment. The CBT group had a significant reduction of Pd [Z = 3.3; p < 0.001], D [Z = 3.37; p < 0.001] and H [Z = 3.19; p < 0.001].

A similar result was found by comparing the pre- and post-treatment scores of the subjects supported by Paroxetine [Pd: Z = 2.7; p < 0.007; D: Z = 2.82; p < 0.005; H: Z = 2.82; p < 0.005].

Even in the group treated with Venlafaxine, a significant reduction of Pd [Z=2.87; p< 0.004], D [Z = 2.84; p < 0.004] and H [Z = 2.81; p < 0.005] was confirmed.

**Discussion**

The use of pharmacological therapy for overweight patients with BED has been less thoroughly studied. SSRIs (Citalopram, Sertraline, Fluoxetine, and Fluvoxamine) have mainly been used as the active compound in the pharmacological trials of patients with BED in order to improve mood symptoms and weight

loss.[34] Likewise, in many cases, promising results have been obtained with Venlafaxine in BED.[35]

Most of the research has focused on specific aspects of binge eating disorder, such as reduction in binge frequency and weight reduction. In general the results are associated with higher discontinuation rates.[36]

In this research, we did not only focus on the binge eating behaviour and impulse regulation in patients with BED. The main objective of this research was to analyze some aspects of eating behaviour (restrained eating, uncontrolled eating and emotional eating) and, more specifically, different psychotic conditions (psychopathic deviate, depression and hypomania).

The first hypothesis of this research was to verify differences between patients with binge eating disorder that followed CBT either with or without a pharmacotherapy support. The results confirmed that CBT and pharmacotherapy are equally effective in the treatment of BED and equally modified patients' impulse regulation. Paroxetine and Venlafaxine medications did not enhance the control of binge eating or guarantee management of impulse regulation better than CBT alone.

This research also aimed at evaluating the efficacy of CBT with or without pharmacotherapy on some factors related to eating behaviour, such as the tendency to consciously monitor and reduce the caloric intake (restriction), the tendency to lose control on food intake (uncontrolled eating) and the conscious perception of the sensation of craving for food (emotional eating). The results suggest that CBT offers the same results regarding the reduction of caloric intake (restriction) as pharmacological treatment. It is less efficient in reducing the lack of control in food intake (uncontrolled eating), although it helps to reduce the sensation of craving for food (emotional eating) compared to pharmacotherapy.

In this research the effects of standardized treatments of CBT with or without the use of pharmacotherapy with bio-equivalent doses of Paroxetine and Venlafaxine were analyzed on psychopathic deviation, depression, and hypomania. The results confirmed that CBT showed a greater reduction of psychopathic deviation compared to those groups who underwent pharmacotherapy. Moreover, pharmacotherapy led to a higher reduction of depression and hypomania than CBT alone.

The second hypothesis was to verify if the SSRI Paroxetine and SNRI Venlafaxine were equally effective on impulse regulation, eating behaviour and personality features. The analysis showed that Paroxetine and Venlafaxine were equally effective on binge eating control and impulse regulation, but some differences in reducing dysfunctional eating behavior were found. Venlafaxine, compared to Paroxetine, seems to offer a greater improvement in emotional eating and restriction eating behavior. In fact CBT could be efficient to assess the tendency to reduce caloric intake (restriction) and to reduce the sensation of craving for food (emotional eating) more than Paroxetine alone. In order to reduce the tendency to lose control on food intake (uncontrolled eating) it could be helpful to administer Paroxetine or Venlafaxine.

Limitations

While the clinical groups were equivalent in all the parameters taken into consideration in the pre-treatment phase, the absence of a control group (no treatment) significantly reduced the possibility to accurately verify the conclusion. Due to ethical reasons we were not allowed to select a group of patients without any specific treatment. In order to correct this weakness in the research, it might be helpful to extend the sample and analyze the changes over a longer period of time.

It is relevant to analyze appropriately these aspects through controlled trials in order to test the efficacy and long-term outcome of psychotherapy, pharmacotherapy, and psychotherapy in combination with pharmacotherapy for treating BED.

Conclusion

In conclusion, patients with eating disorders usually suffer from other psychiatric disorders besides their eating disorder. Many results also confirm substantial comorbidity among obesity, BED, mood and anxiety disorders and metabolic syndrome in weight loss seeking populations.[37]

In such cases, it is important to understand the characteristics of the additional psychiatric disorders and the impact these ones have throughout the treatment.

As underlined by American Dietetic Association (ADA) Reports,[38] understanding the complexities of eating disorders, such as influencing factors, comorbid illness, medical and psychological complications, is critical in the effectiveness of the treatment of eating disorders.

Eating disorders are complex medical illnesses since they have psychological, behavioural, and physiological components. Previous researchers underlined the importance to investigate gender differences in binge eating and associated behavioural correlates[39] and, in order to prevent eating disorders, it is important to carry out individual treatment even on personality traits if the individual disorders have already occurred.[40] Of course, a multidisciplinary approach involving a collaborative team of psychological, nutritional, and medical specialists as underlined in this research must be pursued in order to obtain important and at least short-term results.[41]

The results of this research confirm the need to analyze BED from an integrative perspective and to suggest treatments based on an interdisciplinary approach. The psychological (CBT) and pharmacological (Venlafaxine and Paroxetine) therapies were

both efficient in different ways on the reduction of all the negative variables related to eating disorder. However any treatment could be inadequate in the absence of an accurate diagnosis that takes into consideration biological, genetic, psychological and nutritional components.

The assessment phase still plays an important role in determining which treatment is best for each patient. Accuracy in the medical examination when dealing with medical issues, as well as during the assessment examination and the psychological functioning evaluation is recommended.

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**Author Details**
CRISTINA LANZARONE, PSYCHOTHERAPEUTIC, SISDCA, Via Narbone,48, Palermo, Italy. FRANCESCA CUZZOCREA, PHD PROFESSOR, Department of Human and Social Sciences,University of Messina, Via Tommaso Cannizzaro, 278 Messina, Italy. ROSALBA LARCAN, FULL PROFESSOR, Department of Human and Social Sciences,University of Messina, Via Tommaso Cannizzaro, 278 Messina, Italy. ANTONIO BUONGIORNO, FULL PROFESSOR, Department of Psychology, University of Palermo, Italy. VALENTINA MIMì,PSYCHOTHERAPEUTIC, SISDCA, Via Narbone,48, Palermo, Italy.
CORRESSPONDENCE: FRANCESCA CUZZOCREA, PHD PROFESSOR, Department of Human and Social Sciences,University of Messina, Via Tommaso Cannizzaro, 278 – Messina - Italy.
Email: fcuzzocrea@unime.it

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