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Comparison between Naltrexone-Bupropion and Psychological Treatment in Binge Eating Disorder

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ABSTRACT

Binge eating disorder (BED) belongs to an eating disorder that occurs in both adult and adolescent populations. The disorder is associated with physical disorders such as obesity. It also touches the psychological sphere including impulse control disorder. Recent years have been associated with the development of new treatment strategies for this disorder. Safety-appropriate pharmacological treatments are still being sought. Substances such as lisdexamfetamine or topiramate initially combined to treat obesity-related BED have proved ineffective or unsafe in this indication. Recent studies report the possible efficacy of combining naltrexone with bupropion in the treatment of BED. The combination of these substances affects the genetic mechanisms that promote the production of leptin, the satiety hormone. Psychological treatments such as cognitive behavioral therapy (CBT), its variant dialectical behavior therapy (DBT), interpersonal therapy (IPT) or behavioral weight loss therapy (BWL) still have a well-established and fundamental role in the treatment of eating disorders, including BED. This systematic review included meta-analyses on the treatment of BED between 2004 and 2025. Only double-blind, placebo-controlled RCTs were included in the study. Databases such as PubMed, PsycINFO, and Google Scholar were searched. Most studies showed no advantage of NB over placebo in reducing the frequency of binge eating episodes and BED symptoms. The main benefit of NB in the treatment of BED is weight loss. Preliminary results showed that combining a pharmacological approach (NB) with a psychological approach (CBT) is most effective in reducing BED symptoms.

Key Words: Binge eating disorder, Naltrexone, Bupropion, Cognitive behavioral therapy, Psychological treatment, Obesity, Behavioral weight loss

INTRODUCTION

Binge eating disorder (BED) is defined, according to ICD-11 classification, as "Frequent, recurrent episodes of binge eating (e.g., once a week or more over a period of 3 months). Binge eating is defined as a distinct period of time during which the individual experiences a loss of control over his or her eating behavior. A binge eating episode is present when an individual eats notably more or differently than usual and feels unable to stop eating or limit the type or amount of food eaten. Other characteristics of binge eating episodes may include eating alone because of embarrassment, or eating foods that are not part of the individual's regular diet". Conditions such as Prader- Willi Syndrome or depressive disorder should exclude the diagnosis of BED.[6] BED is currently estimated to affect 1.5% of women and 0.3% of men worldwide. In adolescence, BED is even more prevalent, but often transient. Many adults with BED report longstanding symptoms; less than half are recognized in Healthcare. The disorder is often linked to the presence of obesity, impulse control problems and emotional dysregulation.[14] Research shows that there is neurobiological evidence of BED linked to abnormalities in neuronal connections located in the prefrontal cortex.[15] This is the site responsible for cognitive

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function, thinking, reasoning, language and impulsive behavior, among other things. Those affected by BED experience stronger stimulation of the reward system in response to stimulation - food.

There is a correlation between the severity of binge eating symptoms in people with eating disorders, including BED, and the frequency of subsequent BED diagnoses during the COVID-19 pandemic. This indicates a possible influence of increased stress levels on the development of eating dysregulation in predisposed, stress-prone individuals. This reduces adaptive capacity and abolishes regulatory factors helpful in controlling BED-related impulses.[9]

Two main therapeutic approaches are currently used in BED treatment strategies. One is psychological and psychotherapeutic treatment - mainly CBT and its modifications, such as Behavioral weight loss therapy. The second option is pharmacotherapy, whose effects in the treatment of BED are still the subject of scientific research. One of the most promising therapies is the combination of naltrexone with bupropion.[18] These drugs in monotherapy are widely known for the treatment of alcohol and other psychoactive substance dependence and depressive disorders. [19]

METHODOLOGY

A systematic literature search was performed using databases such as PubMed, PsycINFO, Google Scholar. The search terms included " Binge eating disorder", "Cognitive–behavioral therapy", "Naltrexone-bupropion". Studies published in English from 2004 to 2025 that examined CBT and other psychological methods and naltrexone-bupropion effects on BED were included. The inclusion criteria were adult patients suffering from binge eating disorder in combination with obesity, defined as a BMI > 30, or without obesity. Exclusion criteria were studies that assessed the effect of therapy (psychological or pharmacological) on 'eating disorders' or 'disordered eating' without specifying binge eating disorder. After searching the databases, 22 articles on naltrexone-bupropion therapy in the treatment of BED were found. Ultimately, after removing duplicates (9) and articles that did not meet the inclusion criteria (7), 6 articles examining the effect of naltrexone-bupropion drug therapy in BED were included in the study. Articles on CBT and BED treatment were included in the study in the same way. Seventy-nine articles were found, and after removing 34 duplicates and excluding 33 studies that did not meet the criteria, 12 studies were included in the study. Risk of bias in the included studies was assessed using the Cochrane Risk of Bias Tool, which enabled a systematic evaluation of methodological quality and potential sources of bias.. Only double-blind, placebo-controlled RCTs were included in the study. Randomisation was performed according to obesity status and gender.

RESULTS

Naltrexone- buproipion (NB) and Binge eating disorder (BED)

Analysis of the impact of NB on the effectiveness of BED remains exploratory. Research on this topic is not exhaustive and the topic should be explored further. However, the available literature suggests that there may be positive effects of using a combination of naltrexone and bupropion in certain BED treatment situations. For example, Grilo et al. conducted a randomised double-blind placebo-controlled trial, which found a significant improvement in the remission rate of binge eating in BED in the group of patients taking fixed doses of NB compared to the placebo group. The superiority of naltrexone-bupropion over placebo in the frequency of binge eating attacks was not confirmed.[2,3,4]

Other studies did not confirm the above-mentioned relationships of NB on symptoms and binge eating in BED. Analyses confirm that there was a significant reduction in binge eating, depression, and body weight in patients included in the study, but these results were not the effect of an advantage of NB use over placebo. [2,3,4]

There is evidence that the use of fixed doses of NB might be associated with weight loss in BED patients. Percentage weight loss, i.e. proportional weight reduction from baseline in most studies, confirmed the superiority of NB over placebo. The longer the treatment of the chronic phase of BED with NB lasted, the greater the superiority of NB efficacy over placebo. The proportion of patients who achieved a weight loss between 3 percent and 5 percent (depending on the study) was significantly higher in the NB group than in the placebo group. [4,2,3]

In another study, Girlo et al. showed an increase in the probability of BED symptom remission in the group that did not use NB during the acute phase of the attack. The use of NB in this phase results in a decrease in the probability of remission of BED symptoms if placebo was continued in the chronic phase, or the probability remains unchanged if the intervention with NB was continued in the chronic phase. [3]

Genetic variability in the Nucleobindin 2 (NUCB2) gene, which encoded the peptide nesfatin-1, appeared to play an important role in determining the efficacy of naltrexone–bupropion (NB) treatment in individuals with obesity-associated binge eating disorder (BED). The rs757081 polymorphism was one of the most widely studied SNPs (single nucleotide polymorphisms) in the NUCB2 gene. This SNP had two alleles, C and G. The C allele was considered a strong predictor of weight gain, while the G allele had a protective function in this regard. Carbone et al. indicated that the type of genotype in the range of these two alleles influences the treatment response with NB. NB might be more

effective in individuals with the CG heterozygous genotype compared to those with the CC/GG genotypes. [20]

Cognitive- behavioral therapy (CBT) with other psychological methods and Binge Eating Disorder (BED)

Cognitive behavioral therapy is a form of psychotherapy that makes visible the relationship between beliefs, thoughts and feelings and the behaviors that accompany them. It shows how perceptions influence the choice of how to respond. It is used as a primary treatment for post-traumatic stress disorder, personality disorders, anxiety disorders, among others. CBT has also been adapted to treat eating disorders such as bulimia nervosa and binge eating disorder (BED). In these disorders, CBT teaches how to control emotions and impulses so that they do not influence binge eating.[7]

Several variations of CBT are known, with the aim of further improving the treatment of BED.

Lammers et al. compared a variation of CBT, Dialectical behavior therapy (DBT) to a classical form of CBT. Both forms of treatment were responsible for the reduction of overeating attacks. The global level of eating disorder psychopathology also decreased in both. This effect was greater and was achieved faster with CBT than DBT. CBT was also more responsible for the decrease in depressive symptoms assessed with the Beck scale.

Eating disorders such as Bulimia nervosa (BN), BED, OSFED (type BN or BED) or unspecified feeding or eating disorders (UFED) are characterised by food, weight and body shape concerns resulting from raised Body Mass Index. [13]

A study by Hay et al. assessed the existence of a prevalence of CBT and Healthy APproach to weIght management and Food in Eating Disorders (HAPIFED) in people with eating disorders, including BED, with BMI≥27 and<40kg/m^2. Analyses showed a loss of > 5% of body weight sustained at 12-month follow up. This outcome was achieved by more than 20% of participants from both groups (HAPIFED and CBT) with no advantage for either intervention. However, HAPIFED was favoured over CBT-E in terms of a reduction in purging behavior, as well as improved rates of overeating seizure remission (i.e. more participants abstaining from overeating in the past 3 months), particularly at 12-month follow-up. The remaining analysis did not show an advantage for any of the treatment strategies.[11,16]

An important aspect of the well-established position of CBT in the treatment of eating disorders, including BED, is its effect on impulsivity. Techniques used during therapy such as self-control training and regular eating have been shown to have an effect on reducing episodes of impulsive overeating.[10]

Boswell et al. investigated whether this effect is enhanced by combining CBT with pharmacotherapy that affects the serotonergic system. Fluoxetine, a selective serotonin reuptake inhibitor, modulates the serotonergic system, which has been linked to impulsive responding, a connection to eating disorders including BED.[8]

Findings from the study showed that in each of the treatment groups (CBT alone, Fluoxetine alone, CBT with Fluoxetine) impulsivity decreased significantly during treatment and also during follow up. A rapid decrease in impulsivity during the initial phase was associated with subsequent reductions in eating-disorder psychopathology, depression scores, and BMI during treatment, but not with reductions in binge eating. During the 12-month follow up, the association between reductions in impulsivity and depression scores continued, in contrast to other outcomes.[8]

Table 1: Comparative Effectiveness of Psychotherapeutic and Combination Methods

Study	Interventions Compared	Main Findings	Key Outcomes
Lammers et al.	CBT vs. Dialectical Behavior Therapy (DBT)	Both reduced binge episodes and eating-disorder psychopathology.	CBT achieved faster and greater improvements, including reductions in depressive symptoms (Beck scale).
Hay et al.	CBT vs. HAPIFED (Healthy Approach to Weight Management and Food in Eating Disorders)	Both produced >5% sustained weight loss at 12 months.	HAPIFED showed greater reductions in purging behaviors and higher remission of binge eating; no overall superiority in weight outcomes.
Fairburn et al.	CBT for Eating Disorders (CBT-ED) vs. Interpersonal Therapy (IPT)	Both reduced BED symptoms.	CBT-ED achieved higher remission at treatment end (65.5% vs. 33.3%); long-term follow-up showed similar rates (≈40%), indicating IPT's delayed but sustained effect.
Boswell et al.	CBT alone, Fluoxetine alone, CBT + Fluoxetine	All groups showed significant reductions in impulsivity.	Early decreases in impulsivity predicted improvements in psychopathology, depression, and BMI; mood improvements persisted at 12 months, but binge frequency did not differ across groups.

The conclusions of the studies are promising but insufficient, so there is a need for further investigation in the future.

Other studies have determined the effectiveness of IPT (interpersonal therapy) in a wide range of eating disorder patients, including those with BED. A comparison of this method to the classic form of cognitive behavioral therapy for eating disorders (CBT -ED) showed that the remission rates of symptoms at the end of treatment were 65.5% in the CBT-ED group and 33.3% in the IPT group, respectively. At 60-week follow-up, remission rates were 69.4% (CBT-ED) and 49% (IPT). In contrast, at further follow up, remission rates in the CBT- ED and IPT groups were similar to each other, at 40% for CBT-ED and 39% for IPT, respectively. This result suggests that the effects of IPT for eating disorders occur later than those of CBT, and after this time, the efficacy of both forms is similar.[12]

Comparison beetween naltrexone/bupropion (NB) and psychological therapy

The available literature and previous studies look for additional benefits of combining both BED treatment strategies. Grillo et al. in the Randomised Double-Blind Placebo-Controlled Trial divided the study group into those taking placebo, naltrexone-bupropion, BWL (behavioral weight loss therapy) + placebo or BWL+ naltrexone-bupropion. Remission rates were 17.7% for the placebo group, 31.3% for the naltrexone-bupropion group, 37.1% for the BWL + placebo group, and 57.1% for the BWL + naltrexone-bupropion group. The use of BWL in the therapeutic regimen was associated with a 3-fold higher chance of BED remission compared with no use of BWL. With NB pharmacotherapy, the odds of achieving remission were 2-fold higher than with placebo. Rates of participants attaining >5% weight loss were 11.8% for the placebo group,18.8% for the naltrexone-bupropion group, 31.4% for the BWL+placebo group, and 37.1% for the BWL+ naltrexone-bupropion group. BWL was significantly better than no treatment for binge eating remission, binge eating frequency and achieving > 5% weight loss. NB was superior to placebo only for binge eating remission. No interaction between NB and BWL was demonstrated, confirming the results of other studies on this topic.[1]

Table 2: Outcomes of NB, BWL, and Combination Treatment in Grilo et al. RCT

Treatment Group	BED Remission (%)	>5% Weight Loss (%)	Key Effects
Placebo	17.7%	11.8%	Baseline comparison group
Naltrexone–Bupropion (NB)	31.3%	18.8%	Higher remission than placebo; limited effect on weight loss
BWL + Placebo	37.1%	31.4%	Strong effect on remission, binge frequency, and weight loss
BWL + NB	57.1%	37.1%	Highest remission and weight-loss outcomes; no NB×BWL interaction observed

DISCUSSION

CBT is the primary form of psychotherapy used to treat disorders such as post-traumatic stress disorder, social and specific phobias and anxiety. In the eating disorder spectrum its role is to understand the chain of the reaction leading to symptoms, train impulse control and develop methods, as well as improve perception of one's body image.

Psychotherapeutic methods such as cognitive behavioral therapy (CBT) or interpersonal therapy (IPT) have been prevalent in the treatment of binge eating disorder for many years. Studies suggest that during the follow up of patients with resolution or reduction of BED symptoms, the remission rate and other lasting therapeutic effects oscillate around 50 %. [1] Factors limiting the use of the above methods are the lack of or difficult access to them. In addition, the use of these techniques is not associated with clinically significant weight loss. An increasing number of studies suggest that the behavioral weight loss (BWL) method is comparable to CBT and IPT in reducing overeating attacks. It also has an additional beneficial effect on weight reduction, which is an advantage of this method over CBT and IPT. BWL trials have reported binge-eating remission rates ranging from 38% to 74% and percent weight loss ranging from 2.6% to 5.1%.[1]

Achieving weight loss in obese people with BED is significantly more difficult than treatment therapy for non-BED-related obesity. One of the first drugs to appear in the treatment of obesity in people with BED was topiramate, which belongs to the group of antiepileptic drugs. Its use in BED-related obesity resulted in significantly better results in achieving weight loss compared to placebo. Side effects of the drug and poor tolerability and high discontinuation rates led to the discontinuation of the drug for this indication. [1]

Lisdexamfetamine is another pharmacological treatment for binge-eating disorder approved by the U.S. Food and Drug Administration (FDA) which results in abstinence from overeating attacks of 32%-40% .[1] However, the indications

for its use are limited. It is contraindicated in people with a history of substance abuse, and the safety of its use in the treatment of obesity is uncertain and requires further study.

Several gene variants such as the melanocortin-4 receptor gene (MC4R), the proopiomelanocortin gene (POMC), and the leptin receptor gene (LEPR) are so far known to be genetically linked to eating disorders including BED. The latter two variants are more prevalent and better understood, but studies suggest that the MC4R mutation may be much more important than the other variants. [17] The cerebral regulation of nutrition is based on the satiety and hunger centre. Leptin is its major component. This hormone is responsible for the feeling of satiety by inhibiting the appetite-stimulating system. POMC (proopiomelanocortin) neurons located in the hypothalamus promote the anorectic effect of leptin. Naltrexone with bupropion stimulates POMC neurons, which potentiates the leptin effect and inhibits the physiological feedback that shuts down this response.

Naltrexone/Bupropion is an FDA-approved drug for weight loss in obesity. There are reports which prove that this combination may also be effective in treating obesity associated with BED, as well as BED itself.

Bupropion is a selective neuronal reuptake inhibitor of catecholamines (norepinephrine and dopamine). Naltrexone is a long-acting, specific opioid antagonist with a minor agonist component. It binds competitively to receptors located in the central and peripheral nervous system, blocking the access of exogenous opioids to these receptors.

Studies report that combining these two substances into a single preparation may have a beneficial effect on BED therapy. However, most studies do not support these reports. Naltrexone- bupropion therapy for BED does not show an advantage over placebo in binge eating remission and binge eating frequency.

There are papers that have confirmed the effect of NB in achieving weight loss in obese individuals with BED. Grilo et. al indicated that Naltrexone/bupropion had a significantly higher percentage of weight loss ≥5% than placebo (27.9% vs. 6.5%).[2,4]

Other reports suggested that naltrexone/bupropion was not significantly different from placebo in terms of weight loss, with an observed weight loss of 2.1% lower than the 6.1% observed in obesity without overeating disorders [4]. There is a need for further research in this area. Effective pharmacological approaches for the treatment of BED as well as obesity associated with BED need to be confirmed . It is also crucial to investigate how the combination of pharmacological treatments can potentiate the therapeutic effect achieved with psychological approaches.

There is a need for further research with respect to both pharmacological and psychological treatment combinations.

CONCLUSION

There are now a range of treatment options for BED. Well-established psychological therapies are based on reducing impulsivity and reinforcing positive eating patterns. Most studies do not show an advantage of NB use over placebo in reducing the frequency of seizures and symptoms of BED. The benefit of NB use in obesity-associated BED is mainly due to a weight reduction of 3-5%. This effect is less pronounced in the group of patients with psychological treatment. Promising are the results of a study by Grilo et al. showing that combining NB therapy with a form of psychological treatment (behavioural weight loss therapy) results in a higher remission rate of BED symptoms, as well as a higher probability of losing > 5% of body weight. Further research using both pharmacological and psychological approaches is needed. Combining these two modalities may lead to a potentialised therapeutic effect in patients suffering from eating disorders, including BED.

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Author's contribution

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