
Screening, Diagnosis and Treatment of Patients with Binge Eating Disorder and Obesity: What the Endocrinologist Need to Know

[Simonetta Marucci](#)*, [Luca Busetto](#), Marco Chianelli, Alessandra Fusco, [Maria Carpentieri](#), Marina Armellini, Francesco Tassone, Marcello Sciaraffia, Maria Chantal Ponziani, Anna Nelva, [Carla Cuttica](#)

Posted Date: 11 December 2023

doi: 10.20944/preprints202312.0729.v1

Keywords: binge eating disorder; obesity; treatment; pharmacological therapy; surgery treatment



Preprints.org is a free multidiscipline platform providing preprint service that is dedicated to making early versions of research outputs permanently available and citable. Preprints posted at Preprints.org appear in Web of Science, Crossref, Google Scholar, Scilit, Europe PMC.

Copyright: This is an open access article distributed under the Creative Commons Attribution License which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Review

Screening, Diagnosis and Treatment of Patients with Binge Eating Disorder and Obesity: What the Endocrinologist Need to Know

Simonetta Marucci ^{*1}, Luca Busetto ², Marco Chianelli ³, Alessandra Fusco ⁴, Maria Carpentieri ⁵, Marina Armellini ⁵, Francesco Tassone ⁶, Marcello Sciaraffia ⁷, Maria Chantal Ponziani ⁸, Anna Nelva ⁹ and Carla Micaela Cuttica ¹⁰

¹ University Campus Biomedico Roma, simonetta.marucci@gmail.com

² Department of Medicine, University of Padova, Padova Italy, luca.busetto@unipd.it,

³ Unit of Endocrinology and Metabolism, Regina Apostolorum Hospital, Albano, Rome, marcochianelli@libero.it

⁴ Diabetology Centre, Villaricca, Azienda Sanitaria 2 Naples, Italy, alefusco@yahoo.it,

⁵ Endocrinology and Metabolism Unit, University Hospital S. Maria della Misericordia, Udine, Italy, carpentierimaria82@gmail.com, marina.armellini@asufc.sanita.fvg.it

⁶ Department of Endocrinology, Diabetes and Metabolism, Santa Croce e Carl Hospital, Cuneo, Italy, francesco.tassone@gmail.com,

⁷ Unit of Endocrinology, SS Annunziata Hospital, Taranto, Italy, m.sciara@libero.it

⁸ Unit of Diabetes and Metabolic Disease, SS Trinità Hospital, Borgomanero-Arona, Italy, mchantal.ponziani@gmail.com,

⁹ Unit of Endocrinology and Diabetology, Ospedale degli Infermi, Ponderano, Italy, nelvann@libero.it,

¹⁰ Unit of Endocrinology, Galliera Hospital, Genoa, Italy, carla.cuttica@galliera.it.

* Correspondence: Address correspondence to this author at University Campus Biomedico, Rome. simonetta.marucci@unicampus.it cell: +39, 338. 9577 328.

Abstract: Background Binge eating disorder (BED) is the most common eating disorder, categorized in DSM-V, but many patients are not diagnosed, because of the difficulty to consider BED distinct condition from obesity. Its identification is important for targeting individuals at high risk of obesity, adverse metabolic pattern, and cardiovascular disease. In this review we have highlighted the main aspects of screening, diagnosis and treatment of patients with BED and Obesity. **Methods** An electronic-based search using PubMed database was performed until 2022 and the pertinent articles were selected based on their relevance, English language and publication in peer-reviewed journals. **Results** Many BED patients are not obese, and most people with obesity do not have binge behaviour. In the diagnostic assessment it is important to evaluate not only clinical and nutritional status and medical comorbidity, but also psychological situation related to psychiatric comorbidities, to define the appropriate level of treatment. Well-tolerated drugs with action on both body weight and binges will be useful as a second-line complement to cognitive behavioural therapy (CBT). In addition, specific guidelines are needed to obtain consensus on appropriate recommendations in patients with obesity and BED approaching bariatric surgery, taking into account not only weight reduction and clinical data, but also eating behaviours. **Conclusions** Identification of BED is important for targeting individuals at high risk of obesity, adverse metabolic pattern, and cardiovascular disease. The challenge is also to achieve lasting weight loss in patients with BED and concomitant obesity.

Keywords: binge eating disorder; obesity; treatment; pharmacological therapy; surgery treatment

1. Introduction

Binge Eating Disorder (BED) was for the first time included and defined in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)* as the presence of "recurrent episodes of

binge eating", characterized by "eating in a discrete period of time, an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances", associated with "a sense of lack of control over eating during the episode." [1]. The binge eating episodes are also characterized by "eating much more rapidly than normal", "eating until feeling uncomfortably full", eating large amounts of food without hunger, feeling embarrassed and disgusted with oneself and eating alone, with a sense of guilty and depression. Other diagnostic criteria are the occurrence of binge eating "at least once a week for three months" and not associated to compensatory behaviours as in Bulimia Nervosa (BN). The changes of diagnostic criteria regarding BED, previously categorized in DSM IV as "eating disorder not otherwise specified", would help clinicians to better recognize this disorder in their patients.

BED is often associated with overweight and obesity, metabolic syndrome (MS) and diabetes mellitus (DM), but also occurs in people without obesity. BED occurs, together with Night Eating Syndrome (NES), with more psychological and mood disorders than patients with similar weight but without disordered eating [2]. BED is more common in women (3.5%) than in men (2%) and in obese individuals (5% to 30%) [2], particularly in those looking to lose weight. The onset of the disease is more frequent in early adulthood, but the prevalence among adolescents continues to increase.

Although BED is the most common eating disorder, many patients are not diagnosed, and some research report that clinicians can underdiagnose BED, as revealed in a survey [3] where among respondents who met diagnostic criteria for BED, only 3,2% had a diagnosis by health care providers. Although most people with obesity do not have binge episodes and many patients with BED are not affected by obesity, it is difficult for clinicians to consider BED as a distinct condition from obesity; this represents one of the largest obstacles to the recognition and treatment of this disorder.

2. Diagnosis

The diagnostic assessment is, perhaps, the most important and delicate moment of the entire treatment pathway since it guide the structuring of a therapeutic project inspired by criteria of appropriateness and effectiveness. In this phase, usually carried out on an outpatient basis, the patient should be evaluated at a clinical, nutritional and psychological level, in order to formulate a diagnosis of state regarding the eating disorder, to evaluate any associated clinical and psychiatric comorbidities and define, accordingly, the most appropriate level of treatment.

Although a multidisciplinary team is recommended for diagnosis and management, this is not always possible, especially in peripheral hospital where patients with obesity are dealt with only by endocrinologists. Suggestions for the practicing endocrinologists for quick outpatient evaluation of presence of BED are therefore important. When patients should be referred to a second line specialist (psychologist or psychiatrist)? How a tentative rapid screening should be performed? What are the clinical clues raising suspicion for BED?

After the inclusion in DSM-V as a specific disease, with empirical and clinical consistency of diagnostic criteria, other core psychopathologic features, like body and weight overvaluation, may be of clinical and prognostic relevance, and they can be used as markers for diagnosis [4]. In patients with BED, we can found a polarization of thoughts on weight control, diet and binge-avoidance, worst eating control, higher fear of weight gain, and higher body-shape dissatisfaction than non-BED obese individuals, lower than in BN subjects [5]. Overvaluation of shape and weight can be considered in the severity rate and outcome, in treatment choice, and it would be a specific target for psychological therapies [6]. The food intake patterns are clinically important in BED: they are connected to negative emotions, working as a strategy for managing negative emotions and are sometimes consequent to excessive diet restrictions that can trigger bingeing [7]. Individuals with obesity and comorbid eating disorders, have higher BMIs, more severe level of depression and obsessive-compulsive symptoms, feeling of inadequacy, and they are at high risk of several medical and psychosocial complications [8]. Stigma and negative biases associated with psychiatric disorders, in patients with BED are also aggravated by negative weight based stereotypes and internalized, leading them to feel shame and believe that clinicians do not have time to discuss with them about eating habits and they perceive them judgmental about weight problems [9]. In a study on obese

patients with and without BED, negative attitude toward obesity significantly correlated with depression [10]. Other psychiatric comorbidities are mood disorders in 46%, impulse control disorders in 43%, substance use disorders in 23%, personality disorders in 29%, borderline, obsessive-compulsive personality disorders found in 10% [11]. Increasing evidence is emerging that an earlier onset of bingeing seems to predict worse outcome and to require more complex interventions. In addition, data showed that it tends to be a stable syndrome, with relative stability of binge eating patterns and lower crossover rates than other eating disorders [12].

In the assessment of BED, clinicians have many screening instruments like specific questionnaires, to recognize and treat the eating disorder and not only comorbidities. To diagnose BED, clinicians must consider that it is different from simply overeating. At the beginning of the diagnostic-therapeutic pathway, it is advisable to carry out: 1) a careful personal and familiar anamnesis (focused both on food behaviours, and on any psychiatric comorbidities); 2) an assessment of nutritional status and organic conditions, including the need for laboratory tests depending on the physical condition of the subject; 3) a psychiatric examination considering with special attention the possible risk of suicide and/or self-injurious behaviour; 4) an evaluation of the family and its possible involvement in the course of care (especially for adolescents) [13]. In the case of minors, the assessment must necessarily include the family; for adults it is highly desirable that the family and/or partner be included. There are currently no standardized tools to assess motivation for treatment.

The diagnosis is multidisciplinary and shared among the various professionals. The professional figures who must therefore participate in the assessment process and who carry out the diagnostic tests, necessary, at this stage, are the psychiatrist, the internist, endocrinologist, the clinical psychologist and the nutritionist. In consideration of the particular psychopathological characteristics and complexity of the disorder, the importance of the first contact with the patient to establish a good therapeutic relationship appears fundamental. It is also crucial, from the first contact, to guarantee the integration of the medical and psychological areas of evaluation [14].

The medical diagnostic protocol should include clinical-anamnestic evaluation, physical examination, fasting blood glucose, serum lipid profile (total, HDL and LDL cholesterol, triglycerides), uric acid, thyroid function, liver function (hepatic enzymes), serum creatinine, and cardiovascular assessment [15]. The assessment of nutritional status includes anthropometric evaluations (BMI and waist circumference), assessment of body composition if available (bio-electrical impedance analysis), and body weight history.

It is also necessary an evaluation of the eating behaviour and a careful qualitative-quantitative nutritional history through description of eating habits with particular regard to triggering events (emotional state, hunger score), quantitative and qualitative structuring of the meal, investigation of the use of dysfunctional behaviours such as strict dieting, excessive physical activity, binge eating, compensatory behaviours (self-induced vomiting, laxatives, diuretics, various drugs), obsession with food and body forms, insufficient fluid intake with semi-fasting, psychopathological and psychodiagnosics evaluation. [14]

The need to accurately investigate the psychological symptoms and eating habits of the patient who tends to deny own problem, may require the use of structured or semi-structured interviews. The most accredited questionnaires are: 1. EAT-40 –Eating Attitudes Test [16] of Garner (Italian version validated by M. Cuzzolaro and A. Petrilli in 1988); [17] 2. EDE 12.OD – Eating Disorders Examination [18]; (Italian version validated by V. Ricca) [19]. In the semi-structured interview, the meeting is managed by the therapist who marks the times, contents and developments, asking the patient specific questions about the history of weight, eating habits and attitudes regarding body experience, the meaning of the symptom in the psychological history of the patient and the structuring of the patient's personality. Since patients with ED often deny or underestimate their symptoms, a brief contact with family members, with the attending physician or in any case with a contextual figure is necessary in the collection of the first information.

The evaluation of the psychopathological characteristics of EDs in adolescence and pre-adolescence must be carried out with specific tools, among which the most recognized are: 1) EDE – Eating Disorder Examination, for the evaluation of behavioural and psychological traits related to

ED ([18]; 2) CBCL – Child Behaviour Checklist, for the evaluation of psychopathological symptoms and psychiatric comorbidity [20]. Since the family represents a resource and an integral element in the treatment of these pathologies, the quality of the relationships that children and adolescents have with their parents is observed, highlighting parental behaviours and attitudes such as the attitude to care, affections, sensitivity, cooperation, availability, indifference, rejection, control. It is very important the exploration of family dynamics, especially for minors and patients living with the family. This is not only in the case of young patients who still live within the family, but also in adult patients with BED (or other partial disorders). It is also necessary to investigate the family history with respect to psychiatric disorders, alcohol or substance abuse disorder, obesity, family interactions with respect to the disorder of the subject, family attitudes towards nutrition, exercise and physical fitness, as well as identify family stressors that can favour or hinder healing.

3. Metabolic Implications

Both binge eating and obesity are heritable conditions, and BED seems to be partially caused by genetic factors independent of obesity, as observed in a population-based sample of 2163 female twins evidencing only a modest overlap in the genetic risk factors that increase liability to each condition, considering genetic factors independent of obesity [21]. BED is independently associated with numerous medical comorbidity (including MS, DM, hypertension, dyslipidaemias, sleep problems/ disorders, pain conditions, asthma, gastrointestinal symptoms/ disorders, and among women, menstrual dysfunction, pregnancy complications, polycystic ovary syndrome) [22]. Abraham et al, in the large population-based cohort of the Framingham Heart Study, evidenced an association between BED and high risk of metabolic factors (higher odds of hypertension, hypertriglyceridemia, low HDL, insulin resistance and MS, higher fasting glucose levels, increased visceral subcutaneous and liver fat), attenuated after adjustment for BMI, except of fasting glucose, concluding that binge eaters are at high risk for cardiovascular disease [23]. Hudson et al, in a 5-year longitudinal study, evidenced in BED an increased risk (1.7 fold of any component and 2.4 fold of two or more components) of MS, regardless of the presence of obesity alone [24]. Already in childhood, the presence of BED predicts development of MS approximately 5 years later, making researching BED in children an important target for treatment [25]. Furthermore, a higher prevalence of BED was seen among NAFLD patients, suggesting a possible connection [26]. Rapid consumption of large amounts of food (often 2000-5000 kcal, ingested in a short time) increases inflammatory and oxidative stress, which is considered an important component for the development of the MS, for example producing rapid and sustained increases in glucose and insulin levels [27]. Research focused on the interactions between BED and gut microbiota: diet seems to be able to modify gut microbiota, facilitating dysbiosis that may produce inflammation, altered gut permeability, causing alterations in the hunger /satiety centre, making microbiota a possible target of therapy in BED [28]. Other possible mechanisms in BED seem to involve impaired adipocytokines secretion, leading to higher fasting glucose, such as an insufficient post-prandial leptin production and reduced levels of adiponectin [29,30]. Also incretin dysregulation, probably due to the eating behaviour, is involved in BED: both lower ghrelin pre-meal levels, that may be due to a downregulation by habitual overeating, and slightly-smaller post-meal decline in ghrelin levels, contribute to overeating providing a weaker satiety signal [31,32].

TREATMENT

Therapy of ED must necessarily be divided into different levels of treatment (APA, 2006;[1] NICE, 2004[33] which are:

- Outpatient regimen
- Rehabilitation regime (Residence and DH – the wording refers to semi-residentiality or day care center and not to hospital DH)
- Hospitalization regime (acute).

These levels are not overlapping or disarticulated, but each represent the most suitable and appropriate response to be used based on what the therapists have evaluated in the assessment phase. Depending on the progress or the peculiar problems that can occur during the therapeutic path, the

patient can pass from one level to another. The transition from one level of treatment to another, in the absence of a mandatory sequentiality, is the rule within a personalized care path based on an integrated and interdisciplinary therapeutic approach, which can last up to several years. It is necessary, anyhow, to consider that the transition from one level to another is a particularly delicate moment whether the intensity of the treatment is increased or vice versa, in which the patient is at high risk of destabilization and therefore it is very frequent that drop-out phenomena occur. Furthermore, patients usually don't seek treatment, because of the sense of shame for their eating behaviour, and they instead attempt to resolve by themselves their medical and psychological issues, leading also clinicians to focus on comorbidities, delaying treatment for BED. The treatment of comorbidities has little effect without recognition and treatment of BED, that could be exacerbated also by some medications, particularly those for mood and anxiety disorders that can stimulate appetite [34].

In BED patients, food intake is often related to mood fluctuations, which should be taken into account to manage dietary and behavioural strategies to achieve therapeutic goals [35]. Loss of control is a core feature associated with severe depression, greater body dissatisfaction and poorer related quality of life. Eating impulsiveness can be triggered by restriction of palatable foods and has been related to the hypothesis of a "hedonic deprivation", where eating impulsiveness can be triggered by restrictions on palatable foods and linked to neurobiological mechanisms similar to those of substance abuse. The "food dependence" is an important correlate of this finding, with dopamine, serotonin, and endogenous opioids systems playing a role in this regard [7]. Patients with BED show an increased reward sensitivity towards food and increased rash-spontaneous behaviour, thus shaping a phenotype of obesity with increased impulsiveness, which should be identified and confronted by specific psychological or pharmacological treatments [6].

BED should also be differentiated from other obesity-related eating patterns characterized by eating impulsiveness, not yet recognized as autonomous syndromes, like snacking (introduction of small amounts of food throughout the day), emotional overeating (eating in response to intense emotional states), and selective cravings (intense desire and consuming of specific foods, e.g. sweet eating). These dysfunctional eating behaviours could be considered as subtypes of binge-eating; however, their clinical implications have to be defined yet [9].

Individuals with obesity and comorbid EDs are at higher risk of medical and psychosocial complications than individuals with only one of these conditions, and the best long term treatment strategy has the primary goal to correct binge behaviour, alongside a sustainable weight loss [10], targeting also the increase and maintenance of motivation, the reduction of drop-out rates and the management of relapses [36]. The treatment combinations, need a rising intensity of intervention tailored on disease severity [37].

Psychotherapeutic approaches to BED, is based on cognitive behavioural therapy (CBT) models, recommendable as first-line treatments, effective on eating behaviour but with unclear results on weight loss [38]. Other simpler psychoeducational interventions and self-help treatments have showed significant efficacy in patients with lower disease severity and less comorbidity [39]. Among available treatments, are to be included CBT, interpersonal psychotherapy, selective serotonin reuptake inhibitors (SSRI), with the aim to reduce binge eating frequency, improve metabolic health and weight, and regulate mood [37]. The APA guidelines recommend a team approach including psychiatrists, psychologists, dietitians and social workers.

4. Drugs Overview

Despite psychological and behavioural therapy are considered the first-line treatment, pharmacotherapy could be one of the second-line possible treatments for BED. The matter is if drugs used for BED can also be effective for comorbid obesity, and, vice versa, if drugs used to treat obesity can also be effective in obese people with comorbid BED.

Numerous off-label medications have been studied over time, in the attempt to find an effective treatment for BED and its comorbidities, including antidepressants, anticonvulsants and anti-obesity agents, but to date the only on-label drug for the treatment of BED, approved by the FDA in 2015, is

Lisdexamfetamine dimesylate (LDX) [40]. In this review, drugs which are no longer available because of safety concerns are not discussed. Antidepressant drugs appear to have positive effects on body weight but they have not shown incontrovertible advantages in the long-term treatment of BED [41].

Many specialists consider the off-label topiramate, an anticonvulsant approved for the treatment of epilepsy since the 1990s, as a promising second-line therapy for BED, since it is able to reduce both binges and weight, although limited tolerability may arise due to adverse drug reactions [42]. A recent meta-analysis of available randomized clinical trials, observed that topiramate was able to reduce the number of binge episodes per week, the number of binge days per week and the body weight respect to placebo. However, topiramate users discontinued treatment for safety reasons more frequently than placebo participants [43].

LDX, is the first on-label treatment for BED in adults (≥ 18 years), but is not indicated for weight loss. It is a pro-drug of d-amphetamine first approved in 2007 by the FDA for the treatment of attention-deficit/hyperactivity disorder (ADHD). After oral administration, LDX is absorbed from the small intestine by active transport via the oligopeptide transporter peptide 1. In erythrocyte cytosol of the blood cells, LDX undergoes enzymatic hydrolysis of the peptide bond between L-lysine and LDX, thus producing the pharmacologically active d-amphetamine and L-lysine as a by-product. D-amphetamine is metabolised by CYP2D6 and excreted primarily via the kidney. Plasma d-amphetamine has a more advantageous pharmacokinetic profile than an equivalent dose of immediate-release d-amphetamine, due to a lower C_{max} , an extended T_{max} and a lower inter and intra-individual variability. D-amphetamine is able to cross the blood brain barrier to increase central noradrenergic, dopaminergic and serotonergic neurotransmission, which could produce a combination of effects on appetite/satiety, reward, cognitive processes, including attention and impulsivity/inhibition [44,45]. The efficacy of LDX in the treatment of moderate to severe BED has been demonstrated in many studies, indicating that LDX is able to reduce overall binge eating episodes and symptoms such as severity and obsessive-compulsive and impulsive characteristics of the BED [45,46]. Evaluating the maintenance of efficacy of LDX therapy, a multicentre, double-blind, placebo-controlled, randomized clinical trial evidenced that continued LDX treatment is associated with a significantly lower risk of relapse for binge eating at 6 months, compared to placebo (3.7% vs 32.1%). Participants BMI was of 18 to 45 kg/m² at baseline, but it should be noted that they were predominantly female, white, and affected by obesity. LDX adverse events reported by more than 5% of participants, were most mild or moderate (dry mouth, headache, insomnia, nasopharyngitis, upper respiratory tract infection), and only two serious: breast cancer and nerve root. Furthermore, although the study did not include pregnancies, an unexpected pregnancy and the death of the infant born with exomphalos, limb malformation, and congenital diaphragmatic hernia, were reported. Increase in blood pressure and heart rate was noted, such as a decrease in body weight (mean \pm SD: - 8.29 \pm 7.62 kg in LDX; - 4.25 \pm 5.29 kg, in placebo group) [47]. Currently LDX has no indications for weight loss, mainly for the greater cardiovascular risk associated with obesity. Cardiovascular safety information from BED clinical trials is limited, given the exclusion of patients at higher risk (e.g. persons with diabetes, moderate to severe hypertension and cardiovascular disease), so LDX should not be used in patients with symptomatic cardiovascular disease, nor in patients with moderate to severe hypertension who use other sympathomimetic drugs or who have a family history of sudden/cardiac death and should be used with caution in patients who are involved in strenuous physical activity. In another systematic review and meta-analysis was observed greater efficacy of LDX than placebo in reducing binge days per week, BED-related obsessive-compulsive symptoms, weight and remission rates, although discontinuation rates were higher for LDX than for placebo [48]. Further information is awaited from an ongoing registered clinical trial (ClinicalTrials.gov identifier: NCT03924193) testing the usefulness of combining LDX with CBT, to evaluate the efficacy of combined approach for BED patients with comorbid obesity.

Numerous drugs are currently approved as an adjunct to a reduced-calorie diet and increased physical activity in adults with obesity (BMI ≥ 30 kg/m²), or overweight with BMI ≥ 27 kg/m² in the presence of at least one weight-related comorbidity (such as hypertension, Type 2 diabetes, or dyslipidemia); but they are not approved for the treatment of BED [49,50]. Many studies have

explored the effects of Orlistat, a lipase inhibitor that produces a dose-dependent reduction in the absorption of dietary fat, as an adjunct to CBT, with several conflicting results. Some authors have observed greater weight loss than adding placebo to CBT, in the presence of comparable improvements in eating disorder, considering Orlistat an opportunity to treat patients with obesity and BED [51]. Others observed that adding orlistat to behavioural weight loss produced greater weight-loss than placebo among obese patients without BED, but not among those with BED [52]. Overall, orlistat, even in the long term, did not appear to adversely affect eating behaviour, compared to placebo, during a behavioural program [53].

The combination of Naltrexone and Bupropion sustained release (NB), which works by stimulating POMC neurons (bupropion) as well as blocking endogenous feedback that inhibits POMC activity (naltrexone), acting on hypothalamic and reward circuits, has been proven to be an effective therapy for weight loss in patients with obesity [54–56]. Carbone et al., in an open-label trial, compared patients with BED and obesity with a control group of patients with obesity without BED, treated with NB for 16 weeks in addition to a low-calories diet and increased physical activity. They observed an improvement in pathological eating behaviour and a significant and similar weight loss ($\Delta\text{BMI}\% \approx 8\%$) in both groups, concluding that NB appear to be an effective and well-tolerated option for weight loss in patients with obesity and BED [57]. In a recent placebo-controlled double-blind pilot randomised control trial, Grilo et al evaluated the effects of 12 weeks of NB or placebo, in persons with BED and obesity ($\text{BMI } 30\text{--}50 \text{ kg/m}^2$), evaluating also long-term effects through 6-month follow-up after therapy discontinuation. They observed significant reductions in binge-eating, eating-disorder psychopathology, depression, and weight during treatment, without significant differences between NB and placebo, although the proportion of patients achieving 3% weight loss was significantly greater with NB than placebo (45.5% vs 0%) and weight loss and reductions in bingeing were significantly correlated in the NB group. At 6-month follow-up, results remained improved from baseline, with no significant differences between NB and placebo and no significant differences were reported in adverse events [58]. A large-scale RCT is currently investigating the use of NB in the treatment of BED compared to placebo (ClinicalTrials.gov identifier: NCT03539900). The results of the randomized double-blind placebo-controlled study NCT03045341 were recently published. This study tested NB and BWL therapy, alone and in combination, in 136 patients with BED and obesity (81.6% women, mean age 46.5 years, mean BMI 37.1 kg/m^2). Binge-eating remission rates were 17.7% in the placebo group, 31.3% in the NB group, 37.1% in the BWL+placebo group, and 57.1% in the BWL+NB group. BWL was significantly superior to no BWL, and NB was significantly superior to placebo, but no significant interaction was found between BWL and medication. The 5% weight loss rates were 11.8% in the placebo group, 18.8% in the NB group, 31.4% in the BWL+placebo group, and 38.2% in the BWL+NB group. The results support the potential efficacy of NB for BED treatment, especially when associated with BWL which was able to provide superior improvement on secondary measures (eating disorder psychopathology, depression, eating behaviors, and cholesterol and HbA1c levels) compared with no BWL [59].

Glucagon Like Peptide-1 receptors have been found in central nervous system areas involved in appetite regulation, and GLP-1 receptor agonists (GLP1ra) are able to modulate appetite and reward-related brain areas in humans. Therefore, some authors hypothesized that GLP1ra used for weight loss could have positive effects in BED [60]. The glucagon-like peptide-1 analogue liraglutide (LIR) is approved for obesity in adults and in adolescent from the age of 12 years. In a pilot study, LIR showed a significant improvement in binge eating and a reduction in body weight, BMI, waist circumference, systolic blood pressure, fasting glycaemia and total cholesterol in obese ($\text{BMI } 35.9 \pm 4.2 \text{ kg/m}^2$) non-diabetic subjects, after 3 months of treatment. However, the authors also reported a significant increase in ghrelin levels, highlighting the risk of the drug losing efficacy over time [61]. In combination with intensive behavioural therapy (IBT) LIR was able to produce greater short-term improvements in body weight, dietary disinhibition, global eating disorder psychopathology, and shape concern than IBT alone [62].

Phentermine/topiramate extended release (PHEN/TPM-ER) is another drug used for the treatment of obesity; it is an oral combination of phentermine hydrochloride, an appetite suppressant

sympathomimetic amine, and topiramate, an anti-epileptic medication [63]. PHEN/TPM-ER was shown to be safe and well-tolerated with low discontinuation rates in adults with obesity, and recently FDA approved it for chronic weight management in patients with obesity aged ≥ 12 years [64]. PHEN/TPM-ER in a 12-week, open-label, prospective study was found to be well tolerated and effective in reducing body weight (-4.9 ± 1.2 kg), BMI and binge eating symptoms in individuals with BED and obesity or overweight [65]. In a randomized, placebo-controlled crossover trial, in 22 adults with obesity (BMI 31.1 ± 6.2 kg/m², female 96%, aged 42.9 ± 10.1 years) predominantly with BED and four with BN, 12-weeks PHEN/TPM-ER was shown to be well tolerated and significantly more effective than placebo in reducing binge eating and weight (-5.8 kg; placebo $+0.4$ kg) [66].

5. Bariatric Surgery Overview

Higher rates of EDs are reported in bariatric surgery candidates, and BED is the most common in pre-operative prevalence rates, with a wide range due to different assessment methods and study designs, including variation in DSM-5 criteria, ranging 17% in a recent meta-analysis [67,68]. At present BED is no longer considered a contraindication for the access to bariatric surgery, as long as a targeted specialist framework and a multidisciplinary assessment are made, with possible psychotherapeutic treatment and intervention on proper nutrition education, focused on normalization of eating pattern and improvement in mood, providing strict monitoring before and after surgery by an interdisciplinary team [69–72]. Numerous studies revealed conflicting results about the relationship between the presence of BED and post bariatric surgery weight loss, revealing poorer effects [73,74], or no relationship also in the long-term [93=dawes=nota 7472 68], probably due to the different study methodology. A recent study suggested that bariatric surgery improves food addiction symptomatology, but subjects in whom food addiction persisted after bariatric surgery likely have a more severe form that is characterized by greater binge eating characteristics and psychosocial distress that may improve with cognitive behavioural therapy [75]. The post-operative reduced gastric capacity objectively limits the amount of food ingested at one time, so is certainly difficult to determine which is the definition of “large” amount of food eaten. Existing diagnostic and standard criteria may be insufficient to reveal the atypical presentations in post-operative patients, making necessary to develop appropriated screenings and internationally validated measures [76]. Furthermore, after bariatric surgery shape concerns are frequent, as after weight loss there is loose skin and impairment caused by excessive skin that may produce in these patients greater body dissatisfaction and depressive mood [77]. Kruseman et al evidenced that 8 years after gastric bypass more than half of the patients obtained a successful weight loss (59%), but disordered eating behaviour (BED or NES) was also frequent (51%) [78]. Kofman et al. after gastric bypass also evidenced greater weight regain, lesser excess weight loss, poorer health-related quality of life correlating with frequency of binge eating as risk factors for diminished weight outcomes [79]. All this data evidenced the importance of close monitoring after surgery to reveal postoperative re-emerging disorders, as well as new eating disorders, in a long-term follow-up.

6. Conclusions

In conclusion, data show that BED diagnoses have important implications for treatment outcomes in patients with obesity. Identification of BED is important for targeting individuals at high risk for weight gain, adverse metabolic pattern and cardiovascular disease. Producing weight loss in patients with BED and comorbid obesity is particularly challenging. Well-tolerated treatments able to cure both bingeing behaviour and obesity are also still needed. Randomized trials are necessary to evaluate the safety and efficacy of obesity or BED drugs when used to treat both conditions, many authors stressing the importance of long-term follow-up studies. Indeed, limitations in the existing data, due to the lack of specific and uniform guidelines do not allow to obtain a consensus about the appropriate treatment recommendations in obese BED patients both pre-operatively and post-operatively. Periodic follow-up screenings and interdisciplinary care are advised. The definition of successful outcome after bariatric surgery should take into account not only weight reduction and clinical data, but also problematic eating behaviours.

Author Contributions: For research articles with several authors, a short paragraph specifying their individual contributions must be provided. The following statements should be used “Conceptualization, X.X. and Y.Y.; methodology, X.X.; software, X.X.; validation, X.X., Y.Y. and Z.Z.; formal analysis, X.X.; investigation, X.X.; resources, X.X.; data curation, X.X.; writing—original draft preparation, X.X.; writing—review and editing, X.X.; visualization, X.X.; supervision, X.X.; project administration, X.X.; funding acquisition, Y.Y. All authors have read and agreed to the published version of the manuscript.”, please turn to the [CRediT taxonomy](#) for the term explanation. Authorship must be limited to those who have contributed substantially to the work reported.

Funding: Please add: “This research received no external funding” or “This research was funded by NAME OF FUNDER, grant number XXX” and “The APC was funded by XXX”. Check carefully that the details given are accurate and use the standard spelling of funding agency names at <https://search.crossref.org/funding>, any errors may affect your future funding.

Acknowledgments: In this section you can acknowledge any support given which is not covered by the author contribution or funding sections. This may include administrative and technical support, or donations in kind (e.g., materials used for experiments).

Conflicts of Interest: Declare conflicts of interest or state “The authors declare no conflict of interest.” Authors must identify and declare any personal circumstances or interest that may be perceived as inappropriately influencing the representation or interpretation of reported research results. Any role of the funders in the design of the study; in the collection, analyses or interpretation of data; in the writing of the manuscript, or in the decision to publish the results must be declared in this section. If there is no role, please state “The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results”.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Fifth Edition. Washington, DC: American Psychiatric Publishing; May 18, 2013.
2. McCuen-Wurst C, Ruggieri M, et al. Disordered eating and obesity: associations between binge-eating disorder, night-eating syndrome, and weight-related comorbidities., *Ann N Y Acad Sci* **2018** *1411*(1):96-105. <https://doi.org/10.1111/nyas.13467>
3. Cossrow N, Pawaskar M, et al. Estimating the prevalence of binge eating disorder in a community sample from the United States: comparing DSM-IV-TR and DSM-5 criteria. *J Clin Psychiatry*.**2016** *77*(8):e968-e974. PubMed doi:10.4088/JCP.15m10059.
4. Amianto F., Ottone L., et al. Binge-eating disorder diagnosis and treatment: a recap in front of DSM-5. *BMC Psychiatry* **2015** *15*:70 DOI 10.1186/s12888-015-0445-6
5. Schag K, Schönleber J, et al. Food-related impulsivity in obesity and binge eating disorder - a systematic review. *Obes Rev* **2013***14*(6):477–95. doi:10.1111/obr.12017
6. Grilo CM, Crosby RD, et al. Overvaluation of shape and weight in binge eating disorder, bulimia nervosa, and sub-threshold bulimia nervosa. *Behav Res Ther*. **2009**, *47*(8):692–6
7. Gianini LM, White MA, et al.. Eating pathology, emotion regulation, and emotional overeating in obese adults with Binge Eating Disorder. *Eat Behav*. **2013**;*14*(3):309–13
8. da Luz FQ, Hay P, Touyz S, et al, Obesity with Comorbid Eating Disorders: Associated Health Risks and Treatment Approaches, *Nutrients*. **2018** Jun 27;*10*(7):829. doi: 10.3390/nu10070829.
9. Herman BK, Safikhani S, et al.,The patient experience with DSM-5-defined binge eating disorder: characteristics, barriers to treatment, and implications for primary care physicians. *Postgrad Med*. **2014** Sep;*126*(5):52-. doi: 10.3810/pgm.2014.09.2800.
10. Barnes RD, Ivezaj V, et al. An examination of weight bias among treatment-seeking obese patients with and without binge eating disorder. *Gen Hosp Psychiatry*. **2014**;*36*(2):177-80. PubMed doi:10.1016/j.genhosppsych.2013.10.011.
11. Friborg O, Martinussen M, et al. Personality disorders in eating disorder not otherwise specified and binge eating disorder: a meta-analysis of comorbidity studies. *J Nerv Ment Dis*. **2014** Feb;*202*(2):119-25. doi: 10.1097/NMD.0000000000000080
12. Peterson CB, Swanson SA, et al. Longitudinal stability of binge-eating type in eating disorders. *Int J Eat Disord*. **2012**;*45*(5):664–9. doi:10.1002/eat.22008
13. Quaderni del Ministero della salute. Linee di indirizzo nazionali per la riabilitazione nutrizionale nei disturbi della alimentazione *ISSN 2038-5293. N 29, sett 2017*
14. American Psychiatric Association (APA, 2006). Practice Guideline for the Treatment of Patients with Eating Disorders – Third Edition
15. Volkan Yumuk, Constantine Tsigos, et al. Obesity Management Task Force of the European Association for the Study of Obesity. *Obes Facts* **2015**;*8*:402–424)

16. Garner D.M. & Garfinkel P.E.). The Eating Attitudes Test: an index of the symptoms of Anorexia Nervosa. *Psicol Med*, **1979** 9, 273–279
17. Cuzzolaro M., Petrilli A., Validazione della versione italiana di EAT-40, *Psychology*, **1988**
18. Fairburn C.G. & Cooper Z., EDE 12.OD, in: *Binge Eating: Nature, Assessment and Treatment*. **1993**.
19. Calugi S, Ricca V, et al. The Eating Disorder Examination: Reliability and validity of the Italian version. *Eating and Weight Disorders*. **2015**. doi:DOI 10.1007/s40519-015-0191-2.
20. Achenbach T. Manual for the Child Behavior Checklist/2–3 and 1992 Profile. Burlington, VT: *University of Vermont Department of Psychiatry* **1992**.
21. Bulik CM, Sullivan PF, et al. Genetic and environmental contributions to obesity and binge eating. *Int J Eat Disord*. **2003** 33:293–8. <https://doi.org/10.1002/eat.10140>
22. Olguin P, Fuentes M, et al. Medical comorbidity of binge eating disorder. *Eat Weight Disord*. **2017**; 22(1):13–26
23. Abraham TM, Massaro JM, et al., Metabolic characterization of adults with binge eating in the general population: the Framingham heart study. *Obesity*. **2014** Nov;22(11): 2441–49.
24. Hudson JI, Lalonde JK, et al. Longitudinal study of the diagnosis of components of the metabolic syndrome in individuals with binge-eating disorder. *Am J Clin Nutr*. **2010**; 91:1568–73.
25. Tanofsky-Kraff M, Shomaker LB, et al. Children’s binge eating and development of metabolic syndrome. *Int Journ Obesity*. **2012**; 36: 956–62
26. Zhang J, Abbasi O, et al. Pilot study of the prevalence of binge eating disorder in non-alcoholic fatty liver disease patients *Ann Gastroenterol*. **2017**;30(6):664–69.
27. Patel C, Ghanim H, et al. Prolonged reactive oxygen species generation and nuclear factor-kappaB activation after a high-fat, high-carbohydrate meal in the obese. *J Clin Endocrinol Metab*. **2007**; 92(11):4476–79.
28. Carbone EA, D’Amato P, et al., A systematic review on the role of microbiota in the pathogenesis and treatment of eating disorders. *Europ Psychiatry* **2021**;64(1)e2: 1–14.
29. Taylor AE, Hubbard J, et al. Impact of binge eating on metabolic and leptin dynamics in normal young women. *J Clin Endocrinol Metab*. **1999**; 84:428–34.
30. Monteleone P, Fabrazzo M, et al. Opposite changes in circulating adiponectin in women with bulimia nervosa or binge eating disorder. *J Clin Endocrinol Metab*. **2003**; 88:5387–539
31. Geliepter A, Gluck ME, et al. Plasma ghrelin concentrations are lower in binge-eating disorder. *J Nutr*. **2005**; 135(5):1326–30
32. Hernandez D, Metha N, et al. Meal-related acyl and des-acyl ghrelin and other appetite-related hormones in people with obesity and binge eating. *Obesity* **2019**; 27: 629–35.
33. National Institute for Clinical Excellence (NICE, 2004). Eating Disorder – Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders
34. Flament MF, Bissada H, et al. Evidence-based pharmacotherapy of eating disorders. *Int J Neuropsychopharmacol*. **2012**;15(2):189–207. PubMed doi:10.1017/S1461145711000381
35. Masheb RM, Grilo CM, et al. An examination of eating patterns in community women with bulimia nervosa and binge eating disorder. *Int J Eat Disord*. **2011**;44(7):618–24. doi:10.1002/eat.20853
36. Palavras, M.A.; Hay, P. et al., Comparing cognitive behavioural therapy for eating disorders integrated with behavioural weight loss therapy to cognitive behavioural therapy-enhanced alone in overweight or obese people with bulimia nervosa or binge eating disorder: Study protocol for a randomised controlled trial. *Trials* **2015**, 16, 578
37. Palavras, M.A.; Hay, P. et al, The efficacy of psychological therapies in reducing weight and binge eating in people with bulimia nervosa and binge eating disorder who are overweight or obese-a critical synthesis and meta-analyses. *Nutrients* **2017**, 9, 299. [CrossRef] [PubMed]
38. Devlin, M.J.; Goldfein, J.A. et al. Cognitive behavioral therapy and fluoxetine as adjuncts to group behavioral therapy for binge eating disorder. *Obesity* **2005**, 13, 1077–1088. [CrossRef] [PubMed]
39. Brambilla, F.; Samek, L, et al. Multivariate therapeutic approach to binge-eating disorder: Combined nutritional, psychological and pharmacological treatment. *Int. Clin. Psychopharmacol*. **2009**, 24, 312–317. [CrossRef] [PubMed]
40. Hilbert A, Petroff D, et al., Meta-analysis on the long-term effectiveness of psychological and medical treatments for binge-eating disorder. *Int J Eat Disord*. **2020** 53:1353–1376. <https://doi.org/10.1002/eat.23297>
41. Reas DL & Grilo CM Pharmacological treatment of binge eating disorder: update review and synthesis. *Expert Opin Pharmacother* **2015** 16 (10): 1463–1478. <https://doi.org/10.1517/14656566.2015.1053465>
42. Nourredine M, Jurek L, et al., Use of Topiramate in the Spectrum of Addictive and Eating Disorders: A Systematic Review Comparing Treatment Schemes, Efficacy, and Safety Features. *CNS Drugs* **2021** 35:177–213. <https://doi.org/10.1007/s40263-020-00780-y>
43. Nourredine M, Jurek L, et al., . Efficacy and safety of topiramate in binge eating disorder: a systematic review and meta-analysis. *CNS Spectr* **2020** 26(5):459–467. <https://doi.org/10.1017/s1092852920001613>

44. Ward K & Citrome L Lisdexamfetamine: chemistry, pharmacodynamics, pharmacokinetics, and clinical efficacy, safety, and tolerability in the treatment of binge eating disorder. *Expert Opinion on Drug Metabolism & Toxicology* **2018**, 14:229-238. <https://doi.org/10.1080/17425255.2018.1420163>
45. Schneider E, Higgs S, et al. Lisdexamfetamine and binge-eating disorder: A systematic review and meta-analysis of the preclinical and clinical data with a focus on mechanism of drug action in treating the disorder *Eur Neuropsychopharmacol* **2021** 53:49-78. <https://doi.org/10.1016/j.euroneuro.2021.08.001>
46. McElroy SL, Hudson JI, et al., Efficacy and safety of lisdexamfetamine for treatment of adults with moderate to severe binge-eating disorder: a randomized clinical trial. *JAMA Psychiatry* **2015** 72(3):235-46. <https://doi.org/10.1001/jamapsychiatry.2014.2162>
47. Hudson JI, McElroy SL, et al. Efficacy of Lisdexamfetamine in Adults With Moderate to Severe Binge-Eating Disorder. A Randomized Clinical Trial, *JAMA Psychiatry*. **2017** 74(9):903–910. <https://doi.org/10.1001/jamapsychiatry.2017.1889>
48. Fornaro M, Solmi M, et al., Lisdexamfetamine in the treatment of moderate-to-severe binge eating disorder in adults: systematic review and exploratory meta-analysis of publicly available placebo-controlled, randomized clinical trials. *Neuropsychiatr Dis and Treat* **2016** 12:1827–1836 <https://doi.org/10.2147/ndt.s109637>
49. Shi Q, Wang Y, et al. Pharmacotherapy for adults with overweight and obesity: a systematic review and network meta-analysis of randomised controlled trials. *Lancet* **2022**, 399:259-69. [https://doi.org/10.1016/s0140-6736\(21\)01640-8](https://doi.org/10.1016/s0140-6736(21)01640-8)
50. Cignarella A, Busetto L, et al. Pharmacotherapy of obesity: An update. *Rev Pharmacol Res* **2021** 169:105649 <https://doi.org/10.1016/j.phrs.2021.105649>
51. Grilo CM, Masheb RM, et al. Cognitive behavioral therapy guided self-help and orlistat for the treatment of binge eating disorder: a randomized, double-blind, placebo-controlled trial. *Biol Psychiatry* **2005** 57(10):1193-201. <https://doi.org/10.1016/j.biopsych.2005.03.001>
52. Grilo CM, White MA. Orlistat with behavioral weight loss for obesity with versus without binge eating disorder: Randomized placebo-controlled trial at a community mental health center serving educationally and economically disadvantaged Latino/as. *Behav Res Ther*. **2013** 51:167–175. <https://doi.org/10.1016/j.brat.2013.01.002>
53. Svendsen M, Rissanen A, et al., Effect of Orlistat on Eating Behavior Among Participants in a 3-year Weight Maintenance Trial. *Obesity* **2008** 16:327–333. <https://doi.org/10.1038/oby.2007.82>
54. Greenway FL, Fujioka K, et al., Effect of naltrexone plus bupropion on weight loss in overweight and obese adults (COR-I): a multicentre, randomised, double-blind, placebo-controlled, phase 3 trial. *Lancet* **2010** 376:595–605. [https://doi.org/10.1016/s0140-6736\(10\)60888-4](https://doi.org/10.1016/s0140-6736(10)60888-4)
55. Wadden TA, Foreyt JP, et al. Weight loss with naltrexone SR/bupropion SR combination therapy as an adjunct to behavior modification: the COR-BMOD trial. *Obesity* **2011**. 19:110–120. <https://doi.org/10.1038/oby.2010.147>
56. Apovian CM, Aronne L, et al., A randomized, phase 3 trial of naltrexone SR/bupropion SR on weight and obesity-related risk factors (COR-II). *Obesity (Silver Spring)*. **2013** 21:935–943. <https://doi.org/10.1002/oby.20309>
57. Carbone EA, Caroleo M, et al., An open-label trial on the efficacy and tolerability of naltrexone/bupropion SR for treating altered eating behaviours and weight loss in binge eating disorder. *Eat Weight Disord* **2020** 26(3):779-788. <https://doi.org/10.1007/s40519-020-00910-x>
58. Grilo CM, Lydecker JA, et al., Naltrexone + Bupropion Combination for the Treatment of Binge-eating Disorder with Obesity: A Randomized, Controlled Pilot Study. *Clin Ther*. **2021** 43(1): 112–122. <https://doi.org/10.1016/j.clinthera.2020.10.010>
59. Grilo CM, Lydecker JA, et al., Naltrexone-Bupropion and Behavior Therapy, Alone and Combined, for Binge-Eating Disorder: Randomized Double-Blind Placebo-Controlled Trial. *Am J Psychiatry* **2022**; 179:927–937 <https://doi.org/10.1176/appi.ajp.20220267>
60. McElroy SL, Mori N, et al., Would glucagon-like peptide-1 receptor agonists have efficacy in binge eating disorder and bulimia nervosa? A review of the current literature. *Med Hypotheses* **2018** 111:90-93. <https://doi.org/10.1016/j.mehy.2017.12.029>
61. Robert SA, Ghani Rohana A, et al., Improvement in binge eating in non-diabetic obese individuals after 3 months of treatment with liraglutide - A pilot study. *Obes Res Clin Pract* **2015** 9(3):301-304. <https://doi.org/10.1016/j.orcp.2015.03.005>
62. Chao AM, Wadden TA, et al., Effects of Liraglutide and Behavioral Weight Loss on Food Cravings, Eating Behaviors, and Eating Disorder Psychopathology *Obesity (Silver Spring)* **2019** 27(12): 2005–2010. <https://doi.org/10.1002/oby.22653>
63. Garvey WT Phentermine and topiramate extended-release: a new treatment for obesity and its role in a complications-centric approach to obesity medical Management *Expert Opin. Drug Saf*. **2013** 12(5):741-756. <https://doi.org/10.1517/14740338.2013.806481>

64. Dhillon S Phentermine/Topiramate: Pediatric First Approval. *Paediatr Drugs* **2022** Published 05 September 2022 <https://doi.org/10.1007/s40272-022-00532-z>
65. Guerdjikova AI, Williams S, et al., Combination Phentermine– Topiramate Extended Release for the Treatment of Binge Eating Disorder: An Open-Label, Prospective Study. *Innov Clin Neurosci*. **2018** 15:17–21. <http://www.ncbi.nlm.nih.gov/pmc/articles/pmc6040720/>
66. Safer DL, Adler S, et al., A randomized, placebo-controlled crossover trial of phentermine-topiramate ER in patients with binge-eating disorder and bulimia nervosa *Int J Eat Disord* **2020** 53(2):266-277. <https://doi.org/10.1002/eat.23192>
67. Sarwer DB, Allison KC, et al. Psychopathology, disordered eating, and impulsivity as predictors of outcomes of bariatric surgery. *Surg Obes Relat Dis*. **2019** April; 15(4): 650–55.
68. Dawes AJ, Maggard-Gibbons M, et al. Mental health conditions among patients seeking and undergoing bariatric surgery: a meta-analysis. *JAMA* **2016**;315(2):150–63.
69. Mechanick JL, Apovian C, et al. Clinical Practice Guidelines for the Perioperative Nutrition, Metabolic, and Nonsurgical Support of Patients Undergoing Bariatric Procedures – 2019 Update: Cosponsored by AACE/TOS/ASMBS/OMA/ASA. *Obesity* **2020**; 28: 1-58.
70. Standard Italiani per la Cura dell’Obesità SIO-ADI 2016-2017 <https://www.sio-obesita.org/wp-content/uploads/2017/09/STANDARD-OBESITA-SIO-ADI.pdf>
71. Linee Guida di Chirurgia dell’Obesità SICOB 2016 https://www.sicob.org/00_materiali/linee_guida_2016.pdf
72. Brode CS, Mitchell JE. Problematic eating behaviors and eating disorders associated with bariatric surgery. *Psychiatr Clin North Am*. **2019**;42 (2): 287-97. <https://doi.org/10.1016/j.psc.2019.01.014>
73. Chao AM, Wadden TA, et al. Binge-eating disorder and the outcome of bariatric surgery in a prospective, observational study: two-year results. *Obesity*. **2016**; 24(11):2327-33.
74. Conceição EM, Mitchell JE, et al., Stability of problematic eating behaviours and weight loss trajectories after bariatric surgery: a longitudinal observational study. *Surg Obes Relat Dis*. **2017**;13(6):1063–70.
75. Cassin S, Leung S, et al., Food addiction is associated with binge eating and psychiatric distress among post-operative bariatric surgery patients and may improve in response to cognitive behavioural therapy. *Nutrients* **2020**;12: 2905-17.
76. Conceição EM, Utzinger LM, et al. Eating disorders and problematic eating behaviours before and after bariatric surgery: characterization, assessment and association with treatment outcomes. *Eur. Eat. Disord Rev*. **2015**; 23: 417–25. doi: 10.1002/erv.2397
77. Ramalho S, Bastos AP, et al. Excessive skin and sexual function: relationship with psychological variables and weight regain in women after bariatric surgery. *Obesity Surgery*. **2014**;18–23.
78. Kruseman M, Leimgruber A, et al., Dietary, weight, and psychological changes among patients with obesity, 8 years after gastric bypass. *J Am Diet Assoc*. **2010** Apr;110(4):527-34.
79. Kofman MD, Lent MR, et al. Maladaptive eating patterns, quality of life, and weight outcomes following gastric bypass: results of an Internet survey. *Obesity*. **2010** Oct;18(10):1938-43.