

Eating disorders related to obesity

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Obesity is a chronic disease with multifactorial cause, associated with significant mortality and morbidity. It impacts every aspect of the patient's life. This review discusses the eating disorders that are related to obesity. Binge eating disorder is more frequently seen in obese patients, but bulimia nervosa/disordered-eating behaviors have been included in the review as they can sometimes be associated with obesity. However, it is important to note that most patients with bulimia tend to be of normal weight or overweight. The two disorders are reviewed with diagnostic criteria, risk factors, medical complications, evaluation and treatment recommendations. There is also a comparison between the two disorders. The essential key point is that eating disorders are impulse-control disorders and are similar to addictive behaviors in some aspects. It is essential to treat a patient with obesity and eating disorders multimodally to ensure success.

The global explosion of obesity has resulted in increased awareness and research leading to innovative new diets, medications and surgeries. However, the problem continues to grow at an alarming rate, especially in adolescents. The Center for Disease Control and prevention (CDC) has reported that data from two National Health and Nutrition Examination Survey surveys show that among adults aged 20-74 years, the prevalence of obesity increased from 15.0% (in the 1976-1980 survey) to 32.9% (in the 2003-2004 survey). The two surveys also show, for children aged 2-5 years, the prevalence of overweight children increased from 5.0 to 13.9%; for those aged 6-11 years, prevalence increased from 6.5 to 18.8%; and for those aged 12-19 years, prevalence increased from 5.0 to 17.4%. Approximately two-thirds of the US population is overweight or obese; that is, 133.6 million or 66%. A third of the population is obese; 66.3 million or 31.4% [101].

According to a recent epidemiological review by Wang and Beydoun, 75% of adults will be overweight and 41% will be obese by 2015. Minority and low socioeconomic status groups such as "non-Hispanic black women and children, Mexican-American women and children, low socioeconomic status black men and white women and children, Native Americans and Pacific Islanders" are disproportionately affected. The meta-analysis found that 66% of US adults were overweight or obese in 2003–2004; with women 20–34 years having the fastest increase in rates of obesity and overweight. It also reported that 80% of black women aged 40 years or over is overweight; 50% are obese. Asians have lower obesity prevalence when compared with other ethnic groups. However, Asians born in the USA are four times more likely to be obese than their foreign-born counterparts. White children and adolescents had the lowest prevalence and risk of being overweight compared with their black and Mexican counterparts. Educated people seemed to have a lower prevalence of obesity, with the exception of black women [1].

Obesity is a chronic disease that leads to much medical morbidity and mortality. In 2004, a study reported that in the USA, obesityattributable medical expenditures were estimated at US\$75 billion, with US\$17 billion financed by Medicare and US\$21 billion financed by Medicaid [2].

Eating disorders that are normally related to obesity are binge eating disorder (BED) (classified under Eating Disorder NOS in the Diagnostic and Statistical Manual of Mental Disorders [DSM] IV) and bulimia nervosa (BN). While most patients with BN have normal weight or may be overweight, some can present with obesity. Patients with BN tend to be more preoccupied with their weight and a pursuit for thinness and tend to have severe restrictive dieting, interspersed with binge/purge episodes. Therefore, few patients with BN present with obesity; most tend to have normal weight or overweight. In the current review we will focus on eating disorders, diagnosis, risk factors evaluation and treatment.

Box 1. Diagnostic criteria for 307.51 bulimia nervosa.

Recurrent episodes of binge eating

An episode of binge eating is characterized by both of the following:

- Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
- A sense of lack-of-control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, enemas or other medications, fasting or excessive exercise.

The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.

Self-evaluation is unduly influenced by body shape and weight.

The disturbance does not occur exclusively during episodes of anorexia nervosa.

Specify type

Purging type

• During the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics or enemas.

Nonpurging type

• During the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics or enemas.

We will make note of differences/similarities between the two. BED is not an approved DSM IV diagnosis, perhaps because of its overlap with BN nonpurge type.

In our experience, some patients present with symptoms related to BED, wherein episodes of eating are related to emotions and triggered by specific emotions such as sadness, depression, happiness, stress, anxiety and lastly, and perhaps most commonly, boredom. Currently, this would have to be diagnosed within the Eating Disorder not Otherwise Specified (EDNOS) group as it is an undefined 'emotional eating disorder'. The patients typically recognize the trigger but are unable to reduce the behavior. Treatment of the emotions by alternative methods, such as antidepressants and cognitive behavioral therapy (CBT), helps reduce the abnormal eating in response to the emotion.

Bulimia nervosa

Bulimia nervosa, as first described by Russell in 1979, is characterized by episodic binge eating followed by compensatory purging, both occurring at least twice a week for 3 months. The DSM IV further subdivides BN into purging (those who use behaviors such as laxative abuse and induce vomiting) and the nonpurging (that use excessive exercise and starvation).

Demographics

The peak prevalence of BN is 2–4% in white females aged 17–25 years in the Western nations. The age of onset is typically 18–19 years; BN is rare in patients of a younger age [3].

Diagnosis

The diagnostic criteria as per DSM IV text revision are listed in Box 1.

Risk factors

Family history of eating disorders increases the risk of developing eating disorders 7–12 times. It has been shown that up to 83% of the variance in BN can be accounted for by genetic factors. Twin studies have noted an increase in prevalence in monozygotic twins compared with dizygotic twins. Research in molecular genetics has focused on the 5HT 2A receptor gene, the estrogen receptor β gene and the *UCP2/UCP3* gene and have introduced new paths to understanding BN and anorexia nervosa (AN) [4].

Childhood sexual abuse, parental alcoholism or affective disorders, high levels of family conflict, low parental contact, lack of parental warmth and care, inappropriate parental control and high expectations in parents are all considered risk factors [5]. Premorbid negative self evaluation, impulsivity as well as stressful life events all predispose to the development of BN [6]. Body dissatisfaction has also been identified as a risk factor for developing BN [7]. Patients often report very strict dieting followed by episodes of binge/purge, associated with guilt, depression or anxiety that seem to engulf the patient. BN occurs across all ethnic and racial groups and is five times more common in urban than rural areas [8].

Medical complications

The medical complications with BN vary with the compensatory mechanisms used [9].

Induced vomiting

- Electrolyte abnormalities (hypokalemic, hypochloremic metabolic alkalosis) dehydration;
- Erosion of dental enamel;
- Calluses on the dorsum of the hand (Russell's sign);
- Parotid enlargement;
- Hyper amylasemia and acute pancreatitis;
- Mallory Wies tears, reflux disease, aspiration pneumonia and upper GI bleeds.

Laxative abuse

- Metabolic acidosis;
- Stimulant laxatives can cause diarrhea and dehydration;
- Chronic use of stimulant laxatives can cause hypofunctioning of the colon and result in constipation, blood loss, protein-losing enteropathy, osteomalacia, pseudo fractures, hypocalcaemia and hypomagnesaemia;
- If the laxatives are discontinued, dramatic water retention can result.

Ipecac abuse

- Cardiomyopathy;
- Hepatic toxicity;
- Peripheral neuropathy.

Medications

- Appetite suppressants can cause psychiatric symptoms such as anxiety;
- The use of diuretics can result in electrolyte abnormalities.

Binge eating

Binge eating can result in gastric dilatation.

Menstrual irregularities

Menstrual irregularities have been reported in approximately 45% of women. In a study of patients with BN, it was shown that 45% of patients who had BN but were of normal weight had menstrual irregularities. These patients had a higher frequency of vomiting, more cigarette smoking, and lower thyroxine T4 than those with BN and normal menses. Half of the patients who initially reported irregular menses resumed normal cycles after 12 months of treatment; a third of the patients still reported irregular menses. Higher rates of depressive symptoms, longer duration of eating disorder, current smoking, lower minimum body weight and greater difference between maximum and minimum weight were noted in patients with persistent menstrual irregularity after 12 months of treatment. Patients with BN who have menstrual irregularities are at an increased risk of developing osteopenia. Polycystic ovarian syndrome and increased free testosterone levels have been documented to be increased in frequency in BN patients. The frequent vomiting is thought to cause the insulin response that can cause hyperandrogenism and polycystic ovarian syndrome [10,11].

Evaluation

Patients with BN tend to hide their illness and very often have normal weight or are overweight. A complete physical examination is essential, with a history of BN with or without compensatory purging behaviors. Laboratory tests to include urinalysis (elevated pH suggestive of vomiting, ketones indicative of starvation and elevated specific gravity in dehydration); a complete blood count and electrolyte, lipid studies and serum chemistries. If the patient also has menstrual irregularities a hormonal study is indicated to include thyroid function tests, lutenizing hormone, follicle-stimulating hormone, estradiol and prolactin. An electrocardiogram is essential when there is a history of ipecac use or electrolyte abnormalities. It is prudent to check bone density for osteopenia, especially in patients with menstrual irregularities.

Treatment course & outcome

The goal of treatment is to stop abnormal eating behaviors and provide patients with support to normalize eating behaviors. It is essential that the patient receives medical treatment to reduce risks; in-patient medical treatment may occassionally be required for medical complications to keep the patient safe. Outpatient treatment is indicated when the medical risks are eliminated. An intensive outpatient program such as a day program helps to monitor eating habits intensively, as well as provide support and CBT.

Psychological therapies

Cognitive behavioral therapy has been documented to be most effective in reducing binge eating and vomiting [12]. Interpersonal therapy has also been shown to be effective in long-term studies [13,14]. The goals of therapy are to normalize eating, find alternate coping strategies for triggers that cause binges or purging and to address concern about body image.

Pharmacotherapy

Most antidepressants tend to reduce bulimic behaviors. Fluvoxetine has been studied extensively and a dose of 60 mg optimally reduces bulimic behaviours, improves mood and anxiety and reduces concerns regarding body image. Other medications, such as mood stabilizers like lithium, have shown little change in abnormal eating behaviors [15]. The use of CBT in combination with pharmacotherapy have been shown to produce the best results [16,17]. Adolescents with BN show a reduction in binge–purge behaviors with family therapy. Families with high 'expressed emotions' or critical comments and punishment-oriented families seemed to have more severe illness.

Keel *et al.* followed patients with BN for 11 years and 70% showed full or partial remission only, whilst 11% still met the criteria for BN. It was shown that patients with BN who were treated with CBT and/or antidepressants had improvements in psychosocial functioning compared with patients who received no treatment [18]. Personality disorder, premorbid obesity, longer duration of illness at presentation, history of substance abuse, family history of alcoholism and paternal obesity have been shown to be poor prognostic factors for BN [12,18].

Binge eating disorder

Binge eating disorder is characterized by periods of eating where the patient eats more than their normal intake, eats more rapidly, even when not hungry, feels a loss of control over eating and feels guilt over the episodes. The episodes occur at least twice a week for 6 months. It is the most common eating disorder. Most patients with BED are either overweight or obese, but some patients can also have normal weight. It was first described by Stunkard in 1959 [19]. It is considered as EDNOS in the DSM IV [20] (Box 2).

Demographics

Approximately 2% of all adults in the USA (as many as 4 million Americans) have BED. Approximately 10–15% of people who are mildly obese and who try to lose weight on their own or through commercial weight-loss programs have BED. The disorder is even more common in people who are severely obese. Prevalence of BED in treatment-seeking obese patients has been reported to be approximately 30%. However, those seeking bariatric surgery tend to have higher rates closer to 60–70% [21].

BED is a little more common in women than in men; three women for every two men have it. However, BED when compared with AN or BN has a larger percentage of men affected. Males had less body dissatisfaction and heavier ideal body sizes [22]. The disorder affects Hispanic women more than black women, followed by white women. All three groups show a greater severity of binge eating associated with a higher body mass index and more depression [23].

People who are obese and have BED often became overweight at a younger age than those without the disorder. They might also lose and regain weight ('yo-yo diet' or weight cycling) more often. Those patients with earlier onset BEDs showed longer and more frequent binge-free periods than those with later onset. They also reported a history of mood disorders and BN more often than the patients with late-onset BED [24].

Of note, it was shown by most studies that, typically, onset of BED occurred in teenagers, thus making it especially important to screen teenagers

Box 2. Binge-eating disorder.

Recurrent episodes of binge eating (at least twice per week for 6 months)

Marked distress with at least three of the following:

- Eating very rapidly
- Eating until uncomfortably full
- Eating when not hungry
- Eating alone
- Feeling guilty or disgusted after a binge

No recurrent compensatory purging, exercising, or fasting

Absence of anorexia nervosa

appropriately [25,26]. It was noted that those who started binge eating before dieting had a younger age of onset of binge eating (12 years); and a younger mean age for meeting criteria for BED (19 years) [27]. Several studies have reported that those who began binge eating first had a younger age of onset at 11.6 years, being overweight by 12.4 years and onset of dieting at 17.1 years.

Obese patients with BED show a greater degree of psychiatric comorbidity and lower self esteem compared with obese patients who do not binge [28]. The prevalence of depression has been reported to be as high as 50%.

The DSM IV lists BED in the EDNOS category. Its main symptoms are listed in Box 2; however, in clinical practice, of significance is the feeling of loss of control. The disorder clearly seems to have an association with poor impulse control. The definition states 'large amounts'; this is relative and often at times varies greatly from person to person. Obese patients often eat large portions and these have to be differentiated from other abnormal eating behaviors such as grazing. The trigger for eating is also important in these patients, as hunger is not always the trigger. Many patients have never felt hungry and therefore cannot clearly recognize it. Diagnosis can be made clinically using the criteria listed or rated using questionnaires such as the Binge eating scale, first developed by Gormally et al. in 1982 and published in Addictive Behavior. Sublinical BED patients resemble patients with BED rather than those without BED; therefore, it can be concluded that there exists a continuum of BED based on severity [22].

Risk factors

There have been many suggested causes of BED. From the inheritance aspect, BED does tend to have an increased prevalence in those with a family history of eating disorders. This could be combination genetics, but also environmental learning. Sadly this is based on what we learn early on, as eating habits dominate most of our lives. Patients struggle to break life-long patterns of learned behavior through CBT. Growing up with parents who have problems with abnormal eating can clearly affect patient's eating habits early on. Needless to say that children model parents, and therefore healthy eating habits at home early on would go far in preventing obesity.

BED can be thought of as a 'spectrum disorder,' blending between impulse control disorder to an eating disorder, and also bears many similarities to substance abuse and addiction. The patients paint a clinical picture of 'food addiction' craving food, primacy of thought and preoccupation feelings of anxiety or depression when withholding food, guilt after a binge and hiding the habit. They tend to relapse to food after several attempts at dieting. They also demonstrate the same cognitive dissonance seen in smokers who continue to smoke in spite of being aware of the risks.

From the life story perspective, childhood sexual abuse is very common in patients with BED who also have obesity. It has been shown to affect a third of obese women and one-eighth of obese men. Childhood abuse is associated with abnormal eating behaviors as well as depression, anxiety, substance abuse and somatization. Patients report comfort from eating as children. They seem to feel 'insulated' against sexual activity, which they may find anxiety-provoking. Being obese allows a 'barrier' of fat between them and the abuser [29].

Teasing about general appearance or about weight and size seem to have an effect on body image and psychological functioning in patients with BED. In a study of 115 females who had BED, it was found that general appearance teasing (GAT), but not weight and size teasing (WST), was associated with current weight concerns and body dissatisfaction. Both GAT and WST were significantly associated with current psychological functioning. Patients with earlier onset of obesity reported more WST than patients with later onset obesity. Higher frequency of GAT was associated with greater binge eating frequency in obese women and with greater restraint in non-obese women [30].

Risk factors for developing BED include negative self evaluation, low self esteem, parental depression, adverse childhood experiences and exposure to critical comments from other family members about shape, weight or eating prior to the onset of BED [31].

Evaluation

All patients with obesity should be screened for BED. This includes measuring body mass index and asking about criteria for BED, particularly impulse-control issues with food. Given that most patients with BED are obese, they can have multiple medical comorbidities, and the checking vitals, fasting lipid and sugar levels, thyroid function of insulin levels is indicated [32]. A thorough psychological evaluation is helpful given the increased prevalence of psychopathology in these patients.

Binge eaters should be regarded as a distinct subgroup of obese patients who have specific personality characteristics and issues. A study by de Swan *et al.* revealed significant positive associations between binge eating and eating/weightrelated characteristics such as a history of frequent weight fluctuations, the amount of time spent dieting, drive for thinness and tendency for disinhibition of eating. The subjects exhibited more feelings of ineffectiveness, stronger perfectionist attitudes, more impulsivity, less self esteem and less introceptive awareness the more problems with binge eating they reported [33].

Treatments

Obesity as well as binge eating has to be addressed as goals of treatment. The treatment goals have to be realistic and aimed more at reducing abnormal eating behaviors, stopping very strict diets and developing healthier eating habits and eliminating binge eating. Identifying triggers for a binge, emotions or situations can lead to alternative coping styles and reducing binge eating. The primary focus should be on reducing abnormal eating behaviors and not weight loss.

Selective serotonin reuptake inhibitors, such as fluvoxamine, and serotonin-norepinephrine reuptake inhibitors like Venlafaxine have been reported to reduce the frequency of binge eating [34,35]. Drugs like D-fenfluramine, although successfull in reducing binges, had dangerous side effects and were discontinued. Other medications such as topiramate and inositol have been used with varying success. Atomoxetine was reported to reduce binge eating behaviors in one study [36]. In a recent study, topiramate added to CBT has been shown to cause weight loss [37].

CBT has shown to be highly effective in reducing abnormal eating behaviors, as well as improving general psychological well-being and reducing weight and shape concerns [38]. Patients should be encouraged to combine exercise with CBT. Weight loss, while a positive outcome, should not be regarded as a goal.

Several studies have been performed looking at self help versus guided self-help. Most patients did better on guided self-help as opposed to either self-help or no help.

Fairburn *et al.* showed that 5 years after initial evaluation, only 9% of patients met the criteria for BED [12]. Most patients had not been treated for BED; however, no significant changes were noted with weight. In their study, Agras *et al.* found that when a weight loss program followed CBT, weight loss was seen in those patients that had successfully stopped binging by the end of CBT [39]. A recent review found that BED is a risk factor for poor response to traditional obesity treatments.

Therefore, in treating obese patients with BED, successful outcome depends on addressing the abnormal eating behaviors as well [40].

Comparison between BN & BED

When compared, BN and BED are two separate entities. There are more males affected by BED than BN. An earlier mean age of onset was noted in BN (15.7 vs 17.2 years) [41]. Patients with BED began binging prior to dieting, while those with BN started dieting before binge eating. Patients with BN had a higher level on drive for thinness on the eating disorder inventory, whereas those with BED scored highest on the body dissatisfaction scale. While patients with BN had higher rates of psychopathology compared with patients with BED, patients with BED had higher rates of psychopathology compared with obese patients without BED [42]. BN patients had higher levels of depression and obsessive symptomatology [12].

Patients with BN tended to binge on high carbohydrates and sugars, whereas patients with BED binged on foods they would generally eat as part of a meal. Patients with BED tended to eat in response to emotions of depression, anxiety or boredom [43]. Nonpurging bulimics also tended to differ from BED in the order of onset of binging versus dieting. Both disorders are linked by their pathology; lower self-esteem predicts concerns about eating weight and shape. These in turn are predictive of binge eating.

The two eating disorders that are related to obesity are distinct in their clinical characteristics but have some similarities. They are both associated with significant psychopathology and need to be addressed multimodally to ensure success.

Conclusion

Given the alarming predictions of increasing rates of overweight and obesity all around the world, it will become the leading cause of death. It is a preventable disease and can be controlled. Education of the masses, incentives for weight loss and prevention programs in schools have been on the increase. However, it does seem like too little too late. This review is focused on the two disorders that result in obesity. There is much need for research and development of medications and new innovative treatments to address this growing problem. Research will shed new light on pathways in the brain, which once revealed as related to the abnormal patterns of eating behavior, can be modified and changed to successfully help these patients.

Executive summary

- Obesity has doubled in adults since the 1980s and tripled in children and adolescents.
- Eating disorders related to obesity can result in thwarting the innovative treatments. Persistent weight loss is difficult to achieve as patients go back to abnormal eating behaviors that are key in causing obesity.
- Patients with abnormal eating behaviors have a unique relationship with food. Their eating is triggered by a variety of emotions; typically, hunger is not one of them.

Bulimia nervosa

- Although most frequently patients with bulimia nervosa have normal weight or are overweight, it is included here as some
 patients can become obese. It is characterized by episodic binge eating followed by compensatory purging, both occurring at
 least twice a week for 3 months. The Diagnostic and Statistical Manual of Mental Disorders IV further subdivides bulimia nervosa
 into purging (those who use behaviors such as laxative abuse and induce vomiting); and the nonpurging that use excessive
 exercise and starvation.
- Peak prevalence is in white females aged 17–25 years.
- Family history, childhood sexual abuse, parental alcoholism or affective disorders, high levels of family conflict, low parental contact, lack of parental warmth and care, inappropriate parental control and high expectations in parents and body dissatisfaction are all considered risk factors.
- Medical complications result in induced vomiting, ipecac abuse, laxative abuse and binge eating.
- Evaluation includes a complete physical examination, labs, hormonal studies and electrocardiograms.
- The goal of treatment is to stop abnormal eating behaviors and provide the patient with support to normalize eating behaviors. It is essential that the patient receives medical treatment to reduce risks; inpatient medical treatment may occassionally be required for medical complications to keep the patient safe.
- Medications such as antidepressants and appetite suppressants work well with psychological therapies like cognitive behavior therapies, interpersonal therapy and family therapy.

Binge eating disorder

- Binge eating disorder is characterized by periods of eating where the patient eats more than their normal intake, eats more rapidly, even when not hungry, feels a loss of control over eating and feels guilt over the episodes. The episodes occur at least twice a week for 6 months. It is the most common eating disorder. Most patients with binge eating disorder are either overweight or obese, but some patients can also have normal weight.
- Prevalence of binge eating disorder in treatment-seeking obese patients has been reported to be approximately 30%. However, those seeking bariatric surgery tend to have higher rates closer to 60–70%.
- Binge eating disorder is a little more common in women than in men; three women for every two men have it.
- Obese patients with binge eating disorder show a greater degree of psychiatric comorbidity and lower self esteem compared with obese patients that do not binge. The prevalence of depression has been reported to be as high as 50%.
- Diagnosis can be made clinically using the criteria listed or rated using questionnaires such as the Binge eating scale.
- Genetics, environment, childhood sexual abuse, negative self evaluation, low self esteem, parental depression, adverse childhood experiences and exposure to critical comments from other family members about shape, weight or eating prior to the onset of binge eating disorder are all considered as risk factors.
- Selective serotonin reuptake inhibitors, such as fluvoxamine, and serotonin-norepinephrine reuptake inhibitors like venlafaxine have been reported to reduce the frequency of binge eating. Topiramate when used in conjunction with cognitive behavior therapy has good results.
- Psychological treatments such as cognitive behaviour therapy and interpersonal therapy are used to treat binge eating disorder in conjunction with treating associated psychopathology.

Future perspective

The ever-increasing tide of obesity has caused much alarm and a search for new and innovative treatments is on the rise. Surgical treatments have been effective for weight loss. However, patients tend to return to disordered behaviors after 2–3 years postoperatively. The disease model of eating disorders and obesity are being studied with an effort to uncover neuron regulatory keys to unlock the energy–nutrition dysregulation. The easy solution would be the discovery of medication acting centrally with persistent aversive effects to abnormal eating behavior, which is safe, with minimal side effects. That would be too simplistic – behavioral treatments to change abnormal eating patterns seem to have better results and cannot be bypassed. Multimodal treatments, with life-long support are the key to treating abnormal eating behaviors, while prevention of eating disorders by early interventions are the future of managing a growing epidemic. While one can hope that future research will bring in new treatments to reduce abnormal eating behaviors and thus affect obesity and its comorbidities, it is important to recognize the core psychological issues and address them.

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Bibliography

- Wang Y, Beydoun MA: The obesity epidemic in the United States – gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and metaregression analysis. *Epidemiology Rev.* 29, 6–28 (2007).
- Finkelstein EA, Fiebelkorn IC, Wang G: State-level estimates of annual medical expenditures attributable to obesity. *Obes. Res.* 12, 18–24 (2004).
- Flament M, Ledoux S, Jeamet P et al.: A Population Study of Bulimia Nervosa and Subclinical Eating Disorder in Adolescence. In: *Eating disorders in Adolescence: Anorexia* and Bulimia Nervosa. Steinhausen HC (Ed.), Walter De Gruyter Inc., NY, USA, 21–36 (1995).
- Klump KL, Kaye WH, Strober M: The evolving genetic foundations of eating. *Psychiatr. Clin. North Am.* 24(2), 215–225 (2001).
- Schmidt U, Tiller J, Treasure J: Psychosocial factors in the origins of bulimia nervosa. *Int. Rev. Psychatry* 5, 51–60 (1993).
- Fairburn CG, Cooper PJ: The clinical features of bulimia nervosa. *Br. J. Psychiatry* 144, 238–246 (1984).
- Shaw HE, Stice E, Springer DW: Perfectionism, body dissatisfaction, and self-esteem in predicting bulimic symptomatology: lack of replication. *Int. J. Eat. Disord.* 36(1), 41–47 (2004).
- Hoek HW, Bartelds AIM, Bosveld JJF *et al.*: Impact of urbanization on detection rates of eating disorders. *Am. J. Psychiatry* 152, 1272–1278 (1995).
- Mitchell JE, Pomeroy C, Huber M: A clinician's guide to the eating disorders medicine cabinet. *Int. J. Eat. Disord.* 7, 211–223 (1988).
- Raphael FJ, Rodin DA, Peattie A *et al.*: Ovarian morphology and insulin sensitivity in women with bulimia nervosa. *Clin. Endocrinol.* 43, 451–455 (1995).

- Glendale KA, Bulk CM, Joyce PR et al.: Menstrual cycle irregularity in bulimia nervosa. Associated factors and changes with treatment. J. Psychosom. Res. 49, 409–415 (2000).
- Wilson GT: Cognitive behavior therapy for eating disorders: progress and problems. *Behav. Res. Ther.* 37, S79–S95 (1999).
- Fairburn CG, Norman PA, Welch SL: A prospective study of outcome in bulimia nervosa and the long-term effects of three psychological treatments. *Arch. Gen. Psychiatry* 52, 304–312 (1995).
- Kotler LA, Walsh BT: Eating disorders in children and adolescents: pharmacological therapies. *Eur. Child Adolesc. Psychiatry* 9(Suppl. 1), 1108–1116 (2000).
- Agras WS: Pharmacotherapy of bulimia nervosa and binge eating disorder: longer term outcomes. *Psychopharmacol. Bull.* 33, 433–436 (1997).
- Walsh BT, Agras WS, Devlin MJ *et al.*: Fluoxetine for bulimia nervosa following poor response to psychotherapy. *Am. J. Psychiatry* 157, 1332–1334 (2000).
- Van Furth EF, Van Strien DC, Martina LM *et al.*: Expressed emotion and prediction of outcome in adolescent eating disorders. *Int. J. Eat. Disord.* 20, 19–31 (1996).
- Keel PK, Mitchell JE, Miller KB *et al.*: Long-term out come in bulimia nervosa. *Arch. Gen. Psychiatry* 56, 63–69 (1999).
- Stunkard AJ: Eating patterns and obesity. Psychiatr. Q. 33, 284–292 (1959).
- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. APA Press, Washington, DC, USA (1994).
- 21. Vaidya V: Prevalence of binge eating disorder in patients seeking bariatric surgery, and its correlation with personality characteristics. (2007) (In Press).

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Information resources

- The 'Practice Guidelines for Eating Disorders' supplement, American Journal of Psychiatry (1993), and 'Eating Disorders guidelines' from NICE, Lancet (2005) are both useful tools to help the clinician. However, it is important to base the choice of treatment on the subjective judgment of the treating physician. Treatment should be tailored to the patient needs.
 - Strieber-Moore RH, Wilson GT Wifely DE et al.: Binge eating in an obese community sample. Int. J. Eat. Disord. 23, 27–37 (1998).
 - Fitzgibbon ML, Spring B, Avalon ME, Blackman LR: Correlates of binge eating in Hispanic black and white women. *Int. J. Eat. Disord.* 24, 43–52 (1998).
 - Raymond NC, Mussell MP, Mitchell JE et al.: An age matched comparison of subjects with binge eating disorder and bulimia nervosa. Int. J. Eat. Disord. 18, 135–143 (1995).
 - 25. Grillo CM, Masheb RM: Onset of dieting vs binge eating. *Int. J. Obes.* 24, 404–409 (2000).
 - Marcus MD, Moulton MM, Greeno CG: Binge eating onset in obese patients with binge eating disorder. *Addict. Behav.* 20, 747–755 (1995).
 - Spurrell EB, Wilfley DE, Tanofsky MB, Brownell KD: Age of onset for binge eating. Are there different pathways to binge eating? *Int. J. Eat. Disord.* 21, 55–65 (1997).
 - Mitchell JE, Mussell MP: Comorbidity and binge eating disorder. *Addict. Behav.* 20, W725–732 (1992).
 - Gustafson TB, Sarwer DB: Childhood sexual abuse and obesity. *Obes. Rev.* 5(3), 129–135 (2004).
 - Jackson TD, Grilo CM, Masheb RM: Teasing history, onset of obesity, current eating disorder psychopathology, and psychological functioning in binge eating disorder. *Obes. Res.* 8(6), 108–113 (2000).
 - Fairburn CG, Doll HA, Welch SL *et al.*: Risk factors for binge eating disorders – a community-based case control study. *Arch. Gen. Psychiatry* 55, 425–432 (1998).
 - Barlow SE, Dietz WH: Obesity evaluation and treatment: expert committee recommendations. *Pediatrics* 102, E29 (1998).
 - de Zwaan M, Mitchell JE, Seim HC *et al.*: Eating related and general psychopathology in obese females with binge eating disorder. *Int. J. Eat. Disord.* 15(1), 43–52 (1994).

- Hudson JI, McElroy SL, Raymond NC et al.: Fluvoxamine in the treatment of binge eating disorder. A multi-center, double blind placebo controlled trial. Am. J. Psychiatry 155, 1756–1762 (1998).
- Malhotra S, King KH, Welge JA, Brusman-Lovins L, McElroy SL: Venlafaxine treatment of binge eating disorder associated with obesity. *J. Clin. Psychiatry* 63, 802–806 (2002).
- McElroy SL, Guerdjikova A, Kotwal R et al.: Atomoxetine in the treatment of binge eating disorder: a randomized placebo controlled trial. *J. Clin. Psychiatry* 68, 390–398 (2007).
- Claudino AM, de Oliveira IR, Appolinario JC *et al.*: Double-blind, randomized, placebo-controlled trial of

topiramate plus cognitive-behavior therapy in binge-eating disorder. *J. Clin. Psychiatry* 68(9), 1324–1332 (2007).

- Loeb KL, Wilson GT, Gilbert JS, Labouvie E: Guided and unguided self help in binge eating. *Behav. Res. Ther.* 38, 259–272 (2000).
- Agras WS, Telch CF, Arnow B *et al.*: Weight loss, cognitive behavior therapy, and desimipramine treatments in binge eating disorder. An additive design. *Behav. Ther.* 25, 225–238 (1994).
- Blaine B, Rodman J: Responses to weight loss treatment among obese individuals with and without BED: a matched-study metaanalysis. *Eat. Weight Disord.* 12(2), 54–60 (2007).

- Fairburn CG, Cooper Z, Doll HA *et al.*: The natural course of bulimia nervosa and binge eating disorder in young women. *Arch. Gen. Psych.* 57, 659–665 (2000).
- Mitchell JE, Mussell MP, Peterson CB *et al.*: Hedonics of binge eating in women with bulimia nervosa and binge eating disorder. *Int. J. Eat. Disord.* 26, 165–170 (1999).
- Decaluwe V, Braet C: The cognitive behavioral model for eating disorders: a direct evaluation in children and adolescents with obesity. *Eat. Behav.* 6, 211–220 (2005).

Website

101. Centers for disease control and prevention (2007)

www.cdc.gov/nccdphp/dnpa/obesity/trend/ maps/