Disordered eating and obesity: associations between binge eating-disorder, night-eating syndrome, and weight-related co-morbidities

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Abstract

Binge-eating disorder (BED) and night-eating syndrome (NES) are two forms of disordered eating associated with overweight and obesity. While these disorders also occur in non-obese persons, they seem to be associated with weight gain over time and higher risk of diabetes and other metabolic dysfunction. BED and NES are also associated with higher risk of psychopathology, including mood, anxiety, and sleep problems, than those of similar weight status without disordered eating. Treatments are available, including cognitive behavior therapy (CBT), interpersonal psychotherapy, lisdexamfetamine, and selective serotonin reuptake inhibitors (SSRIs) for BED; and CBT, SSRIs, progressive muscle relaxation, and bright light therapy for NES.

Keywords

Introduction

Disordered eating spans the spectrum of over-controlled eating, resulting in anorexia nervosa, to a variety of forms of under-controlled eating such as bulimia nervosa and binge-eating disorder (BED), or night-eating syndrome (NES), which causes eating when humans are meant to be fasting. Two of these disorders (i.e., BED and NES) are often comorbid with overweight and obesity. We will review these disorders here, along with their relation to psychosocial and metabolic correlates.

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Binge Eating Disorder

Definition

According to the Diagnostic and Statistical Manual of Mental Disorders (DSM), 5th edition,[1] BED is defined by several criteria. Individuals must report consuming an unusually large amount of food in a short duration (compared to what others may consume in a similar situation) in addition to experiencing a loss of control over one's eating behavior during this time. In addition, at least three of the following characteristics must also be present: consuming food much more rapidly than normal, eating food until uncomfortably full, consuming large amounts of food when not hungry, consuming food alone to avoid embarrassment, or feeling disgusted, depressed, or guilty after the eating event. The diagnosis also requires that a significant amount of distress be associated with the binge episodes, which must occur at least once per week for 3 months or more (on average). Lastly, the disorder must not be accompanied by any regular compensatory behavior, nor should the binge eating occur solely during an episode of bulimia nervosa or anorexia nervosa.

Prevalence

BED affects an estimated 1–3% of the general population, making it the most common eating disorder.[2–3] Method of assessment is clinical interview or self-report questionnaire, with the latter generally identifying a higher prevalence.[4–5] Lifetime risk of BED diagnosis is higher among women than men,[2] however men who are diagnosed experience equal impairment.[6] Also at a higher risk are those under the age of 30 compared to older individuals.[2] Prevalence among varying ethnicities is somewhat unclear, with studies finding different racial/ethnic prevalence estimates,[7] and other studies suggesting no significant difference between whites and minorities.[8] Typical age of onset of the disorder is between 15.5–27.2 years old with the average duration lasting between 4–8 years.[2–3]

Comorbidities

Those with BED have a high prevalence of psychiatric and physical comorbidities. Nearly 80% of those with lifetime BED have suffered from another DSM disorder including mood, anxiety, substance use, and eating disorders.[2,9] In addition to psychiatric concerns, BED is independently associated with increased risk of physical comorbidities including chronic diabetes, hypertension, back/neck pain, chronic headaches, and other types of chronic pain.[2]

Obesity and weight-related factors

Those with BED are an estimated 3–6 times more likely to be obese than those without an eating disorder.[2–3] BED is also associated with an earlier onset of being overweight[8] and history of obesity, with about 30% of those with BED reporting childhood obesity.[10] Additionally, negative talk regarding shape, weight, and eating from family members,[11] body shape concerns, and cycling of weight[12] are all also correlated with a BED diagnosis. Binge eating is more prevalent among those seeking weight loss approaches compared to those who are not, with between 9–29% of those reporting binge episodes.[8, 13–15]. However, only 1–2% of those seeking weight loss treatment meet full DSM criteria for BED[13–15].

Among bariatric surgery patients the prevalence is much higher; prevalence rates of those seeking bariatric surgery who meet clinical diagnosis for BED range from 4.2%[5] to 47%[16], depending upon method of assessment.

In addition to the prevalence of obesity among those with BED, the disorder itself is associated with a variety of weight-related comorbidities. Certain evidence suggests that BED, perhaps due to eating behaviors adapted by those with the disorder, may pose as a risk factor for metabolic syndrome aside from obesity alone.[9] Metabolic syndrome labels a cluster of risk factors associated with increased risk for heart disease, dyslipidemia, diabetes, and other cardiovascular problems,[17] making it a public health concern. Prevalence of BED among those with type 2 diabetes is projected to range from 1.4%[15] to 25.6%,[18] with increasing prevalence among those with a higher body mass index (BMI).[15,19] Additionally, differences in prevalence of metabolic syndrome among those with BED exist among genders and ethnicities; men and African Americans with BED have a higher risk of developing components of metabolic syndrome such as increased systolic blood pressure, fasting glucose, hemoglobin A1c and triglycerides, and reduced high density lipoprotein levels compared to women and other ethnicities.[20] In a longitudinal study over the course of 5 years, those with BED who were obese had over two times the chance of developing a new diagnosis of dyslipidemia compared to a non-BED matched sample,[21] suggesting a link between the disorder and certain components of metabolic syndrome, aside from the risks presented solely from obesity alone.[21] However, rates of metabolic syndrome among those with obesity and BED versus those with obesity alone have been shown to be comparable, around 60%.[21–22] Recently, BED was found to be strongly associated with diabetes (five-fold increased risk) and components of the metabolic syndrome (two-fold increased risk) among participants of the Swedish National Registers.[23] Further, those with obesity and BED were at increased risk of respiratory and gastrointestinal diseases as compared to those without these disorders. More research is needed to further evaluate the relationship between metabolic syndrome and BED, apart from the effects of obesity alone.

Psychopathology

There are several explanatory models of binge eating pathology. One of the first theories of binge eating, the restraint model, explains binge eating as originating from shape and weight concerns, which lead to dietary restraint, then binge eating, followed by an ongoing cycle of restraint and binge eating.[24–25] Some studies have provided further support for this model, showing dietary restraint is associated with increased binge eating episodes among those with BED,[26–27] while other studies have shown dietary restraint precedes binge eating only about half of the time.[28] Those with BED tend to eat more often, eat higher calorie meals,[29] and have more frequent weight fluctuations compared to those with other eating disorders,[8] all behaviors which are consistent with the restraint model. This model could also help explain the higher prevalence of binge eating among people seeking behavioral weight loss, because those patients may be seeking methods to implement dietary restraint successfully, which is something they may not have been able to accomplish on their own without experiencing binge episodes. Two newer models are the affect regulation model and escape theory. The affect regulation model describes binge eating as a distracting coping mechanism used to reduce negative feelings. This behavior is then thought to be maintained

by the reinforcing reduction in negative affect through binge eating[30], despite evidence of no actual reduction in negative affect.[31–32] Escape theory explains binge eating as a strategy used to avoid self-awareness and redirect attention towards the binge eating instead of the stressor. This is thought to occur in an effort to avoid one's own feelings and thoughts and instead focus attention on the stimulus at hand,[33] i.e., the experience of eating.

Treatment

Treatment for BED largely targets the behavioral, psychological, and physical outcomes related to the disorder.[34–35] According to several meta-analyses and reviews, psychotherapies, specifically cognitive behavior therapy (CBT) based approaches, are widely considered the most effective intervention for BED. However, other methods of treatment, including weight loss and pharmacological treatments are proven effective in treating certain BED outcomes in specific populations.[34–36]

Psychotherapies—CBT based interventions are based on the restraint model which, as previously discussed, theorize that unhealthy eating, shape, and weight concerns give rise to food restriction. This initial restriction then leads to a cyclical pattern of restriction and binge eating.[25] CBT based interventions aim to stop this cycle by addressing abnormal thoughts and behaviors regarding eating, shape, and weight that trigger binge eating and by developing healthier responses to these triggers.[37] A meta-analysis by Vocks *et al.*[35] concluded that therapist-led CBT was more effective than untreated control groups in reducing binge eating frequency, decreasing days with binge eating, increasing binge eating abstinence, and also reducing depression, concern with weight, and concern with eating. CBT however, was not effective in reducing dietary restraint scores, shape concern, or weight. Self-help forms of CBT were effective in the same domains, except in reducing days with binge eating and depressive symptoms, but with the additional benefits of decreased dietary restraint scores and shape concern.[35] A meta-analysis by Brownley *et al.*[34] found therapist-led CBT to be more effective than a waitlist condition: increasing binge abstinence but not reducing binge-eating frequency, eating-related obsessions and compulsions, depressive symptoms, or weight-related outcomes.[35] The American Psychological Association and the National Institute for Health and Care Excellence recommend both therapist-led and self-help forms of CBT for treating the behavioral and psychological symptoms of the disorder as the first line approach, with interpersonal therapy (IPT) and dialectical behavior therapy as potential psychological alternatives.[36, 38]

IPT, one of these psychotherapies, is based on the belief that binge eating is used as a coping mechanism to escape negative affect created by poor interpersonal social functioning.[39] In a randomized control trial ($n = 90$) comparing the long term efficacy of CBT compared to IPT, the two treatments showed similar efficacy with a total of 64.4% of participants achieving full recovery at the 4 year follow-up. CBT participants achieved a higher percentage of recovery post-treatment (81.8%) compared to IPT participants (64.4%); however, at 4 year follow-up CBT participants decreased in full-recovery (52.0%) and IPT participants improved in full-recovery (76.7%).[40] Overall, CBT is still recommended first, as it works most rapidly, but IPT seems to be a promising second line approach or perhaps may be a

good match for those with binge eating consistent with the escape theory of BED; but more research is needed regarding matching therapies with different phenotypes of the disorder.

Behavioral weight loss—While psychotherapies are often recommended as the first line of treatment, they are often not successful at achieving weight loss.[34–35] Behavioral weight loss (BWL) treats the disorder by targeting the excess weight that is often pervasive among the BED population.[2–3] Randomized control trials (RCT's) examining BWL are limited; however, the meta-analysis Vocks *et al.*[35] of uncontrolled studies showed a moderate effect size of BWL on binge eating frequency and weight pre-and post-treatment. In one RCT of 125 participants with obesity and BED, mean percent BMI loss was significantly greater among those who received BWL (–2.1) compared to CBT (–0.9) or CBT combined with BWL (1.5) post treatment.[41] However, binge frequency was significantly reduced through 6- and 12-month follow-up in the CBT group compared to BWL group,[41] suggesting the BWL intervention's effect on binge eating may be less durable.

Pharmacological treatments—Common forms of pharmacotherapy include antidepressants, antiepileptic medications, anti-obesity medications, and stimulant medications (typically used for attention deficit hyperactivity disorder),[42] with lisdexamfetamine being the only currently approved drug by the U.S. Food and Drug Administration for treating BED.[34] The meta-analysis by Vocks *et al.* found a significant effect of pharmacological treatments compared to control groups on reduction of days with binge eating, depressive symptoms, and a mild effect on binge eating abstinence.[35] The meta-analysis by Brownley *et al.* found compared to placebo, lisdexamfetamine and second-generation antidepressants (SGA's) (e.g. selective serotonin reuptake inhibitors (SSRIs)) resulted in greater abstinence from binge eating and less eating-related obsessions and compulsions.[34] Additionally, SGA's were significantly more effective in reducing depressive symptoms than placebo, while lisdexamfetamine and topiramate showed greater reductions in weight compared to placebo.[34] With pharmacological treatments, it is important to note the presence of potential side-effects associated with treatment. Brownley *et al.* found 80% of the studies included in their meta-analysis reported harms, compared to none from the psychological treatment studies.[34] From the studies included in this analysis, reported side effects for topiramate and SSRIs include insomnia, sleep disturbances, headaches, gastrointestinal upset, and sympathetic nervous system arousal, which are not uncommon.[34]

Summary

In sum, BED occurs in persons across the weight spectrum, but its prevalence increases with BMI. BED is comorbid with somatic disorders, including diabetes and the components of the metabolic syndrome. Binge-eating is often used to reduce distress related life stress or internal dysphoria, and it may also be related to chronic efforts of food restriction. Efficacious treatments for BED include CBT, BWL, and pharmacotherapy, such as lisdexamfetamine and SSRIs.

Night-eating syndrome

Definition

The other prominent form of disordered eating related to overweight and obesity is NES. NES was first described by Stunkard, Grace, and Wolff among a group of individuals with obesity seeking weight loss treatment.[43] They reported that those with the syndrome consumed a large majority of their caloric intake (25% or more) at a time when individuals without obesity would not be eating. In addition, the patients experienced insomnia and morning anorexia. Most of the patients with night-eating syndrome reported that they experienced symptoms during times of weight gain and life stress.[43] Since this time, the definition of NES has varied. For example, in later years, Stunkard's definition was expanded to include nocturnal ingestions.[44]

NES is characterized by recurrent episodes of night-eating, which is described as either excessive food consumption in the evening (after dinnertime, i.e., evening hyperphagia) or eating after awakening from sleep (i.e., nocturnal ingestions). NES is also characterized by at least three of the following symptoms: morning anorexia, the presence of a strong urge to eat between dinner and sleep and/or during the night, sleep onset and/or maintenance insomnia, frequently depressed mood or mood worsening in the evening, and a belief that one cannot get back to sleep without eating.[44–45] In order to be diagnosed with NES, individuals must be aware of and be able to recall the eating episodes. These symptoms must also cause significant distress and/or impairment in functioning and not be better explained by external factors or another disorder, such as a sleeping disorder or other disordered eating pattern.[1] NES is classified in the DSM 5 as an "other specified feeding or eating disorder."

Prevalence, demographics, and comorbidities

The prevalence of NES is estimated at 1.5% in the general population in the United States, which is similar to binge-eating disorder and higher than more traditionally recognized eating disorders, such as bulimia nervosa and anorexia nervosa.[46–48] Although not all persons with NES are overweight or obese, the syndrome is more frequently found in obese populations.[49–50] In a population-based twin study in Sweden, NES was 2.5 times more prevalent in males with obesity and 2.8 times more prevalent in females with obesity as compared to non-obese men and women.[51] In a 2003 review conducted by de Zwaan, Burgard, Schenck, and Mitchell,[52] NES prevalence was found to be between 6–64% among obese persons seeking weight loss surgery. Other studies have found up to 55% of individuals who seek bariatric surgery report some symptoms of NES.[53]

NES typically begins during early adulthood (late teens to late twenties) and appears to be long lasting with periods of remission and relapse, often tied to life stressors.[54–55] In one age and gender population survey, similar proportions of males and females reported waking at night to eat.[56] However, in an interview-based study of NES among Swedish Twin Registry participants, fewer men than women met full criteria for NES, possibly due to low levels of distress associated with their NES symptoms.[57] This may suggest that both men and women experience NES symptoms equally, but women are more negatively affected. The prevalence of NES and associated features in various racial groups is not well characterized, although a

wide number of studies have included Caucasian, Hispanic, African-American and Pima Indian ethic and racial group.[49]

NES is more common among people with insomnia, eating disorders, and other psychiatric disorders.[49] Individuals diagnosed with NES are more likely than those in the general population to have another eating disorder with prevalence estimates ranging from 5–44%.[49] Approximately 15–20% of patients with NES also have BED. Although there are some overlapping symptoms between NES and BED, it has been suggested that the disorders can be differentiated by the amount of food eaten per episode, as well as the motivation to eat (i.e., in NES, the motive is to achieve sleep) and level of concern regarding shape and weight.[45,58]

NES prevalence also increases with BMI in psychiatric populations. In a study conducted by Lundgren and colleagues, participants were recruited from psychiatric outpatient clinics.[59] The night eating questionnaire (NEQ)[60] was used to screen broadly for morning anorexia, food cravings in the evening, number of awakenings, and nocturnal ingestions, followed by a clinical interview. The Night Eating Syndrome History and Inventory was subsequently used to diagnose NES. The researchers found that 12.3% of the study population met criteria for NES. In addition, those with NES were more likely to have obesity.

NES can cause sleep disturbances, but sleep disturbances and insomnia may also precede NES. Studies have found that patients with NES have difficulties initiating and maintaining sleep.[50] Agents of sleep disturbances in individuals with NES may include nocturnal ingestions and insomnia. Although NES is largely characterized by nocturnal ingestions, these episodes also occur in sleep related eating disorder (SRED). This disorder is characterized by recurrent episodes of involuntary eating and drinking during sleep. It is considered a parasomnia rather than an eating disorder.[50] NES and SRED can be differentiated by a lower level of consciousness while eating, as well as the consumption of unusual subtances with SRED.[61]

Psychopathology

The cause of NES is unknown, but research suggests links to genetics and neurobiological and psychological processes. In a study conducted by Lamerz and colleagues, German children were more likely to report night eating if their mothers exhibited night eating behavior, as compared to children of mothers who did not engage in this behavior.[62] Also, in a study comparing night eating in families with NES to those without, the probability of an individual with NES having a first degree relative with the disorder was greater than those in the control group, suggesting heritability.[63] In fact, among participants in the Swedish Twin Registry, heritability of night eating symptoms was.[64]

The literature suggests that NES is exacerbated during times of major life stress. This finding has led researchers to examine the role of cortisol and other stress hormones in night eating. Cortisol is a hormone released by the adrenal cortex in response to stress that has been associated with overeating and weight gain.[65–66] In a study conducted by Birketvedt and colleagues, 12 participants with NES and 21 control participants were fed fixed meals at regular intervals over a 24 hour period with no food after 8:00 PM.[67] Blood drawn every 2 h

over 24 h showed that cortisol levels were highest from 8:00 AM until 2:00 AM in night eaters, as compared to individuals in the control group. This finding led researchers to hypothesize that NES may be associated with a dysregulation of the hypothalamic–pituitary–adrenal axis, which controls the cortisol stress response.[68]

In 1955, Stunkard and colleagues suggested that major life stress worsened symptoms of night eating and removing that person from a stressful environment would alleviate symptoms.[43] More recent studies have found that stress, as well as psychiatric disorders, such as depression, are often associated with the initial onset of night eating and maintenance of the disorder.[47] Additionally, researchers are now suggesting that emotions and cognitions may play a major role in the onset and maintenance of NES. Nocturnal episodes may be maintained by possessing the belief that one cannot sleep without eating, as well as the desire to control the anxiety associated with that belief.[69–70]

Association with obesity, disordered feeding, and metabolism

NES appears to be associated with obesity. As mentioned earlier, previous studies suggest that the syndrome is more common among weight loss treatment seeking individuals.[49] NES may also be associated with weight gain. Marshall and colleagues compared the characteristics of people with obesity and NES and those of individuals without obesity or the disorder. Twenty-one patients with obesity were included in the study which included a clinical interview, while 80 patients completed the NEQ on the Internet.[54] The participants were separated into three groups: 40 participants with a BMI less than 25 kg/m^2 completing the NEQ on the internet, 40 participants with a BMI greater than 30 kg/m^2 completing the NEQ on the internet, and 21 participants with a BMI greater than 30 kg/m^2 who completed a clinical interview. The study found that there were remarkable similarities between the internet-based participants without obesity and those with obesity on their total and individual item NEQ scores. The most important difference between the two groups was that the non-obese individuals with NES were 8.9 years younger than the obese group, suggesting that NES may contribute to the development of obesity.[54] These results are also in line with the finding that night eating severity is correlated with a higher BMI in middle-aged, but not young adults.[71]

Other studies suggest that obesity and weight gain are not necessarily associated with NES. In a cross-sectional observational study examining 266 participants with class II-III obesity either with or without NES, the researchers found that NES was non-significantly associated with higher BMI. There were also no differences in metabolic complications or psychological features, with the exception of depression.[72] However, given that all patients were obese, there could be a ceiling effect. In a nationally representative population study in Denmark, NES, as indicated by endorsement of "getting up at night to eat", was not associated with weight gain during the previous or following 5 years. However, women with obesity and NES gained more weight than women with obesity but without the disorder, suggesting that getting up at night to eat may contribute to additional weight gain in individuals with obesity.[56]

Delays in usual daytime eating patterns increase the risk of metabolic syndrome.[73–74] Research suggests that NES has a prevalence of 9.7% in those with diabetes, and those with

symptoms of night eating have substandard metabolic control (elevated A1C) and two or more diabetes related complications.[75] In another study conducted by Hood and colleagues, 194 participants with type 2 diabetes showed that 7% of the sample met criteria for NES. Night eating was also correlated with poorer glycemic control, which could be a result of the food choices made by individuals with night eating, including high carbohydrate and high fat foods.[76]

Treatment

Several treatment approaches have been tested for NES. Psychotherapy has shown efficacy in reducing night eating symptoms. Allison and colleagues developed a CBT approach that was administered in a pilot study to 25 patients over 12 weeks.[77] The intervention included building rapport, providing education about CBT, self-monitoring of sleep and eating disturbances and negative thoughts, regulation of eating and sleep patterns, development of coping skills, and, for those with overweight or obesity, behavioral weight management. At the conclusion of the study, the participants showed significant reductions in caloric intake in the evening (after dinner), nocturnal ingestions and awakenings at night and improvements in mood and quality of life. They also lost a modest but statistically significant amount of weight (3.1 kg).[77]

Pharmacological treatments have also been examined, as NES research has suggested that the serotonin system plays a role in regulating eating, sleep, and mood, all components of NES. The hypothesized decrease in serotonin availability in NES would lead to disturbances in circadian rhythms and a decrease in satiety, thereby increasing risk of evening hyperphagia and nocturnal ingestions.[78] Therefore, increasing postsynaptic serotonergic activity by blocking the serotonin reuptake transporter by use of SSRIs may be effective in treatment NES. The first clinical trial examining the efficacy of pharmacotherapy was a 12-week, open label study of 17 participants treated with the SSRI sertraline.[79] Two additional trials with sertraline followed, including an RCT. The three studies showed significant reductions in awakenings, nocturnal ingestions, and caloric intake after the evening meal, [79–81] including a response rate of 67% and a remission rate of 29% with sertraline.[79] Those who achieved remission also lost a significant amount of weight (−4.8 kg). In the RCT, those with overweight and obesity lost an average of −2.9 kg compared to those receiving the placebo.[76] The researchers noted that the effects on NES were independent of its antidepressant effects, with no significant correlation found between NES symptoms and mood changes.[79] Escitalopram has also been tested with reductions in the core NES symptoms, number of nocturnal ingestions and percent of energy intake consumed after dinner, in an open label trial.[82] However, no significant differences in symptom reduction were found when escitalopram was compared to a placebo in an RCT.[83]

Progressive muscle relaxation (PMR) has also been studied in an effort to find alternative treatments for NES. PMR has been shown to reduce stress, which is often associated with NES. In a recent study, Vander Wal and colleagues randomized 44 men and women to one of three groups: education, education plus PMR, or PMR plus exercise.[84] The three groups reported reduction in NES symptoms, as well as symptoms of depression, anxiety, and perceived stress. The only significant difference between groups was the percentage of food

eaten after the evening meal, with the PMR group showing the greatest reduction, followed by the PMR plus exercise group and the educational group, respectively.[84]

Phototherapy—or bright light therapy—another alternative treatment for NES, has been studied to examine its possible efficacy in treatment of symptoms. Phototherapy has been shown to increase the availability of postsynaptic serotonin.[85] In two case studies, bright light therapy was used to treat both seasonal depression and symptoms of NES. In a study conducted by Friedman and colleagues, a 51-year-old woman diagnosed with NES and comorbid depression was treated with paroxetine; light therapy was added. After 14 daily sessions of white light for 30 min, the patient no longer met the DSM-IV criteria for depression or the core symptoms of NES. However, once the light treatment was discontinued, her symptoms of NES returned, although her depression remained in remission. After being treated for another 12 days with light therapy, her night eating symptoms remitted once more.[86] Additionally, in a pilot study conducted on the effects of bright light therapy among 15 individuals with NES, significant reductions were found in NES symptoms and mood and sleep disturbances post treatment.[87]

Summary

NES is a disorder of a delayed pattern of eating that is associated with increased weight and metabolic dysfunction. NES is often associated with other forms of psychopathology, including mood, anxiety and insomnia. Treatments are available, including CBT, PMR, bright light, and pharmacotherapies (mainly SSRIs). Further research is needed to understand the etiology of NES and to test these treatments more rigorously and in larger samples.

Conclusion

Overall, BED and NES occur in 5–15% of persons with obesity. Both disorders likely contribute to weight gain and are linked to medical comorbidities, or the worsening of their symptoms, such as diabetes and metabolic syndrome (Table 1). Identification of these disorders through simple screening questions and referral to proper treatment is recommended, because patients often do not bring up these issues on their own due to the guilt and shame related to these disorders. However, absence of treatment likely exacerbates their medical co-morbidities and impedes treatment efforts overall. As such, asking about the presence of loss of control eating involving large amounts of food and eating habits in the evening and during the nighttime could yield important information for healthcare providers wanting to optimize treatment of the medical issues their patients are presenting, as well as improve psychological functioning and quality of life. Further research on treatment options and dissemination of treatments for these disorders is also warranted.

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Table 1

Comparing and contrasting BED and NES.

	Binge-eating disorder	Night-eating syndrome
Consume objectively large amounts of food in a discrete period	X	
Consume at least 25% of energy after dinner and/or wake to eat at least twice per week		X
Feel a loss of control over the episodes	X	X
Belief that one must eat to sleep is present		X
Associated with increased risk of diabetes and metabolic syndrome	X	
Associated with more poorly controlled metabolic functioning, including glucose control	X	X
Associated with increased co-morbidity of mood and anxiety symptoms	X	X