A review of nighttime eating disorders

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Summary

Nighttime eating is categorized as either night eating syndrome (NES) or sleep-related eating disorder (SRED). These conditions represent an interruption in the overnight fast that characterizes human sleep. A critical review of the literature on NES and SRED will suggest that they are situated at opposite poles of a disordered eating spectrum. NES could be considered an abnormality in the circadian rhythm of meal timing with a normal circadian timing of sleep onset. Conversely, the feeding behavior in SRED is characterized by recurrent episodes of eating after an arousal from nighttime sleep with or without amnesia. Both conditions are often relentless and chronic. Multiple definitions of night eating have limited our ability to determine the exact prevalence of NES. Studies have suggested that central nervous system (CNS) serotonin modulation may lead to an effective treatment of NES. SRED is frequently associated with other sleep disorders, in particular parasomnias. Early studies have shown that the anti-seizure medication topiramate may be an effective treatment for SRED.

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Normal metabolic physiology during sleep: introduction

Nighttime in humans is typically characterized by a prolonged period of fasting associated with sleep.

With some cultural variation, sleep is initiated 1—4 h after the last meal. The length of sleep depends upon genetic and volitional factors but averages between 7 and 9 h. Thus, under normal physiologic conditions there is a consecutive absence of food intake for approximately half of every 24-h day. However, energy homeostasis is maintained through alterations in glucose regulation and appetite modulation.

Despite a lack of food intake, serum glucose levels are adequately maintained throughout the
sleep period, and both sleep and circadian phenomena operate to help maintain glucose homeostasis. This is in contrast to fasting during sedentary wakefulness which demonstrates a fall in glucose over 12 h.¹

Changes in systemic and cerebral glucose utilization during the sleep period helps maintain stable energy stores. The diminished motor activity that characterizes sleep contributes to decreased peripheral metabolism. But the majority of the decrease in glucose utilization is related to the decline in brain metabolism.² This effect is particularly noted during delta non–rapid eye movement (NREM) sleep which is concentrated in the first half of the sleep period. Positron emission tomography (PET) of NREM sleep demonstrates a 40% reduction in glucose metabolism compared to wakefulness.³ Conversely, PET has demonstrated that glucose utilization in the brain during REM sleep, primarily in the second half of the sleep period, is as high as and occasionally higher than during wakefulness.³ This correlates with glucose utilization data in the second half of the sleep period which is closer to wakefulness.¹

The sleep period is also characterized by impairment in glucose tolerance through sleep and circadian mechanisms. Under conditions of constant glucose infusion (which eliminates confounding meal timing factors), serum glucose levels high during both nighttime sleep and daytime naps. There is also a circadian mechanism to decrease glucose tolerance as serum glucose increases during a period of nocturnal sleep deprivation.⁴

Insulin disposal and growth hormone (GH) secretion help maintain stable glucose levels throughout the sleep period. Insulin disposal is increased during the sleep period, helping to maintain elevated glucose levels. GH is released at the onset of the sleep period and increases serum glucose levels by stimulating hepatic gluconeogenesis and inhibiting glucose uptake. Sleep deprivation suppresses GH secretion until sleep is initiated regardless of the circadian period.⁵ Sleep-onset GH secretion is also associated with increasing density of slow wave sleep.⁶ This relationship allows for anabolic processes to occur during periods of musculoskeletal and cerebral quiescence.

One major hormonal mediator of appetite is modified during the sleep period to promote fasting. Leptin, a peptide hormone secreted by adipocytes, mediates satiety by inhibiting hunger centers in the hypothalamus during states of energy surplus. Leptin has a nocturnal rise that correlates with sleep onset, and is noted under conditions of continuous enteral nutrition.⁷ Conversely, the hormone ghrelin, which is the initiation signal for feeding that is produced primarily in the stomach in response to fasting and circulates in the blood, has increased levels during sleep in humans.⁸ Presumably, therefore, during sleep there is a balance between increased ghrelin and increased leptin activity that does not promote eating.

In summary, energy homeostasis is maintained during sleep despite a prolonged fast through alterations in glucose metabolism and appetite regulation, as listed in Table 1.

### Introduction to night eating syndrome and sleep-related eating disorder

Abnormal eating during the main sleep period has been categorized as either night eating syndrome (NES) or sleep-related eating disorder (SRED). Some cases of nocturnal bulimia nervosa and dissociative disorders with night eating have been reported, but will not be discussed herein. Definitions of NES and SRED arose primarily from the obesity and sleep research fields, respectively, and are characterized to some extent by their comorbidites. For example, NES has been frequently associated with depression and SRED has been frequently associated with underlying sleep disorders such as sleepwalking. A critical review of the literature on NES and SRED will suggest that these conditions are situated at opposite poles of a spectrum of disordered eating during the main sleep period involving the inappropriate timing of food intake, inappropriate food choices and/or manner of eating (binging, etc.). Furthermore, there is a broad range in the level of consciousness during feeding, clinical consequences, and multiple sleep and psychiatric comorbidities in both NES and SRED. The use of published terms for these phenomena has been confusing, and Table 2 attempts to provide clarity to the extent that is currently possible. We now present a review of each disorder and follow with a discussion regarding their relationship to each other.

#### Table 1: Energy homeostasis during sleep

<table>
<thead>
<tr>
<th>Decreased glucose utilization</th>
<th>Impaired glucose tolerance</th>
<th>Appetite suppression</th>
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</thead>
<tbody>
<tr>
<td>Decreased motor activity</td>
<td>GH secretion at sleep onset</td>
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<tr>
<td>Decreased cerebral glucose activity (NREM sleep)</td>
<td>Insulin disposal increased</td>
<td>Increased leptin (satiety hormone)</td>
</tr>
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² M.J. Howell et al.
Night eating syndrome

Historical descriptions

Over a 52-year period, the diagnostic profile of NES has expanded from evening hyperphagia (excessive eating between the evening meal and sleep onset) to evening hyperphagia and/or conscious night eating (eating after an awakening from sleep).

The night eating syndrome (NES) was first described in 1955 among patients with treatment-resistant obesity at a specialty center.9 The constellation of symptoms included: in 80% of the patients, night eating was noted at least 50% of days, with insomnia and morning anorexia. Night eating in the general obesity population was later noted to be low (10%) by comparison.10 Evening hyperphagia was described during established periods of wakefulness prior to sleep onset and consisted of high-caloric carbohydrate foods. Family conflicts and other social stresses were common and closely coincided with exacerbations of the night eating. Psychotherapy and environmental modification were ineffective in promoting weight loss. In fact, only 10% (2/20) of NES patients were able to lose more than one third of their initial excess weight.9

Clinical characteristics of night eating syndrome

Night eating is often a chronic phenomenon that has been described in normal weight, overweight and obese subjects. In a study of 106 subjects who described themselves as night eaters, 77% reported duration of greater than 5 years.11 The mean BMI was in the obese range (BMI = 31), although 20% of subjects had a BMI < 25.

NES patients have sleep and eating patterns that distinguish them from other eating disordered patients. For example, it has been reported that NES patients eat more frequently during the day than obese controls.12 Under laboratory conditions designed to determine maximum caloric intake per meal, NES patients consume more calories at night than obese patients with binge eating disorder (BED); and NES patients consume more calories at night than they do earlier in the day.13 Also, NES patients have more awakenings and more awakenings that involve eating compared to obese controls.12 These findings are important because they suggest that nocturnal eating (after an awakening from sleep) is common in NES.

NES has been characterized as a circadian delay of food intake with normal circadian timing of sleep onset. A similar phenomenon has been described in animal models where the gene expression for food intake is uncoupled from other circadian rhythms.14 A study of 46 overweight human NES subjects compared the circadian timing of feeding and sleeping to overweight controls without NES. The total caloric intake and sleep onset did not differ between groups but meal times were delayed in the NES group. The authors suggested that these findings indicate NES is a circadian delay in the timing of feeding relative to sleeping.15 Moreover, another study of 15 adult females with NES demonstrated that patients with NES did not differ from controls in the timing of nocturnal sleep onset or sleep termination.16

It is unclear whether neuroendocrine findings noted in NES are a cause or an effect of night eating. A clinical study of NES patients found alterations in glucose, insulin and ghrelin compared to controls. However, these findings were noted across the course of the night and were not correlated to individual episodes of night eating. Furthermore, there were no significant differences between NES and controls in other sleep and feeding related hormones such as melatonin and leptin. Moreover, eating (especially the consumption of so-called “comfort foods”) during these awakenings might delay the circadian signals for energy intake. This study did not report any data from hunger analog scales during awakenings and prior to any food ingestion. This is important data in regards to the phenomenology of night eating and any possible correlation with neuroendocrine findings.17 On the other hand, two clinical-PSG studies of NES are quite pertinent, since both studies identified patients without evening hyperphagia and without any reported hunger during recurrent nighttime awakenings with eating.18,19
Foods eaten in NES are often very similar to foods eaten during the daytime although high carbohydrate foods, such as breads and sugars are preferred.\textsuperscript{11} In a behavioral study of night eaters the carbohydrate content of evening food intake was higher (70\%) than daytime (47\%).\textsuperscript{12} NES patients do not typically consume unusual foods or inedible substances, an important distinction as these behaviors are often noted in SRED patients.

Recently investigations have considered whether genetic influences may contribute to the development of NES. In a study of 5–6-year-old children, it was reported that children of mothers with night eating behavior were more likely to demonstrate night eating compared to children of non-night eating mothers.\textsuperscript{20} Another study compared night eating in NES families to control probands. This investigation demonstrated that the odds of a patient with NES having an affected first-degree relative was significantly greater than that of a control (OR = 4.9).\textsuperscript{21}

**Prevalence**

Multiple definitions of NES have limited our ability to determine the exact prevalence of night eating.\textsuperscript{11,22,23} However, in general, studies have indicated a prevalence of approximately 1.5\% in adult populations.\textsuperscript{24,25} A pediatric study examining eating behavior in 5–6-year-old children revealed that 1.1\% had symptoms of night eating.\textsuperscript{20}

Investigations into the relationship between night eating and obesity have generally demonstrated a direct relationship between NES and BMI. A study of normal-weight individuals demonstrated a prevalence of 0.4\%,\textsuperscript{26} while 6–14\% of patients seeking weight loss treatment reported symptoms of NES.\textsuperscript{13,27,28} Furthermore, subjects with NES are significantly more likely to be obese than patients who do not meet criteria for NES (BMI: 33.0 vs. 27.4).\textsuperscript{29} Moreover, one survey of patients undergoing surgery for morbid obesity reported a high prevalence of NES (27\%).\textsuperscript{24} Conversely, a recent study reported a lower prevalence of NES (8.9\%) among bariatric surgery patients.\textsuperscript{30} Another study noted that among black women NES is not associated with obesity and in fact NES subjects were less likely to be overweight than non-NES subjects.\textsuperscript{25} Also, another comparison between obese and non-obese individuals with NES found similar characteristics.\textsuperscript{31} Further research is needed to explore ethnic variation in the relationship between NES and obesity.

In order to determine the clinical spectrum of night eating, 106 subjects who admitted to be nighttime eaters were interviewed\textsuperscript{11} using both the original 1955 criteria (>25\% of total calories after the last meal, morning anorexia, insomnia) and more stringent 1999 criteria (>50\% of daily energy intake consumed after the last meal, morning anorexia, nocturnal feeding and a duration of 3 months). Interestingly only 13\% of subjects who already claimed nighttime eating met the 1999 criteria while the less stringent 1955 criteria was met by 29\% of these subjects. These results suggest that night eating exists on a wide spectrum and the authors suggested that some mild cases of nocturnal eating may be considered “subsyndromal”.

Some studies have demonstrated that many night eaters may be of normal weight and perceive their behavior as nonproblematic. In a community survey of night eaters 25\% of the sample did not believe that the night eating was a problem unless they had coexisting eating disorders such as anorexia nervosa or bulimia nervosa.\textsuperscript{11} Subjects who were not obese or overweight were more likely to perceive their night eating as nonproblematic. Moreover, depending upon the definition, night eating is often noted in adolescent girls without psychological distress. A study of 9–19-year-old females noted that 50–70\% of subjects described eating more than 25\% of total daily calories after the evening meal. Furthermore, occasional episodes of night eating were more common (typically by a factor of 10) compared to multiple episodes of night eating a week.\textsuperscript{22}

**NES and obesity**

It is not yet clear whether NES predates and contributes to the development of obesity. In one report most subjects (60\%) claimed they did not develop NES until after they were obese. Furthermore, many NES patients were not overweight and did not consider nighttime eating problematic.\textsuperscript{12} Another investigation demonstrated that total energy intake did not differ between night eaters and controls.\textsuperscript{17} Conversely, another study reported that subjects were of normal weight prior to the development of NES and subsequent weight gain.\textsuperscript{31} Also, obese women with a history of night eating gained more weight in a 6-year follow-up study than other obese women; and, among 399 psychiatry patients, those who were obese were more likely to manifest NES than those who were not obese.\textsuperscript{29} Finally, a recent study demonstrated that the presence of NES correlates with increasing BMI.\textsuperscript{32} Further research is needed before we can conclude whether night eating contributes to weight gain, and also the extent to which overweight status or obesity promotes NES.
NES may lead to treatment resistant obesity. Originally, NES was described among treatment resistant patients at an obesity center.\(^9\) Furthermore, a study reported that obese NES patients lost less weight after 1 month than did obese patients without NES.\(^{13}\) However, further research is needed before we can conclude whether night eating leads to weight gain that is more difficult to treat than other forms of obesity.

**NES and mental illness**

Investigators have explored the relationship between NES and mental illness. The original description of NES noted that psychosocial stressors were common and closely coincided with exacerbations of night eating.\(^9\) Among obesity patients, the subjects with NES have higher depression scores on the Zung Depression Inventory and lower self-esteem on the Rosenberg Self-Esteem Scale.\(^{13}\) Similarly, a behavioral study reported that subjects with NES had mood scores that were lower compared to obese controls. Interestingly, there was a circadian decline in mood scores after 4 p.m. for NES subjects,\(^{12}\) a finding that is the opposite the typical pattern of endogenous depression. Furthermore, NES is common among patients with mental illness. Investigators who surveyed and then interviewed outpatient psychiatry patients at two academic centers reported that 12.3% of patients met criteria for the NES. Finally, NES patients had higher rates of substance abuse than those without NES.\(^{29}\)

Researchers have investigated whether there is a relationship between NES and binge eating disorder (BED). We will not review the eating disorder literature here, however, the preponderance of data suggests that while there is some overlap between NES and BED, they are separate phenomena (as discussed in Ref.\(^{33}\)).

**Screening instruments**

The night eating questionnaire (NEQ) was developed to standardize screening for NES and thereby provide consistency for the diagnosis. The NEQ is composed of 14 questions pertaining to circadian variation of hunger, satiety, caloric intake, and sleep. Two more questions address how long the subject has been struggling with night eating and whether there is a familial trait to the behavior. As screening surveys will over-report the presence of night eating, it is imperative that a formalized interview confirm the diagnosis.\(^{34}\)

**Current classification and nomenclature**

Over the last 52 years, studies of NES have employed various criteria for distinguishing core clinical features: evening hyperphagia, presence or absence of eating after awakenings from sleep, and degree of morning anorexia. The diversity in definitions stems from the differences in clinical populations being studied, cultural differences in meal times, and changes in eating patterns over the last 50 years. This lack of uniform criteria has constrained clinical application of NES research and impedes therapy development. Moreover, many of the variations in diagnostic criteria for NES have common co-authors further confounding nosologic definitions.

The level of consciousness experienced during nocturnal eating episodes is often poorly characterized in NES literature. Typically nocturnal eating in NES articles is described as eating after an arousal. However, most studies do not explicitly state the level of awareness (none, partial, full) in NES subjects. This information is important for nosological distinctions between NES and SRED.

Over 20 criteria have been described, but historically two definitions have been most commonly employed in NES studies. The original NES definition from 1955 was derived from a group of patients with a constellation of symptoms that included greater than 25% of caloric intake after the evening meal (before sleep onset), insomnia, and morning anorexia.\(^9\) The NES definition was expanded in a study published in 1999 that utilized sleep and eating diaries, but without PSG monitoring, which found increased awakenings with conscious eating in the patients. However, neither level of consciousness nor degree of control over the eating was reported. Because of these findings, nocturnal feedings were included in revised criteria for NES and the % of total daily energy intake was increased (>50% of daily energy intake consumed after the last meal, morning anorexia, night eating and a duration of 3 months).\(^{12}\)

A recent report described another variation in the definition of NES. This study of 399 psychiatric outpatients defined NES as evening hyperphagia or night eating after awakenings. Unlike previous criteria there was no requirement for evening hyperphagia. This deviation represents another diagnostic variation in NES.\(^{29}\) However, this report did not include data on how many patients, if any, had eating only after awakenings from sleep without evening hyperphagia.
Cultural variation in meal timing limits attempts to define certain feeding as abnormal. For example, later Norwegian meal times compelled the investigators who reported the 1999 criteria to change the definition of abnormal evening eating from 6 p.m. to 8 p.m. Furthermore, Mediterranean feeding patterns are characterized by meal times that are later than those in the United States or Northern Europe. In one study, the majority of subjects in Genoa, Italy (70%) described typically having their final meal after 8:30 p.m.

Morning anorexia, despite being included in most diagnostic criteria, is inconsistently defined. In particular, the absence of hunger and food intake is often not quantified and thus open to interpretation. Different studies have used various terms such as: “skipping breakfast”, “negligible food intake”, “no appetite for breakfast”, or “not starting to eat until later in the day” (as reviewed in Ref. 23).

A more recent study suggested that the evening hyperphagia should be revised down to 25% of daily caloric intake. The authors noted that food intake after the evening meal and during nocturnal awakenings were 3-fold greater in obese NES patients compared to obese controls (34.5% vs. 10.0%). Based on this report, 25% of total daily energy intake was considered abnormal as it is two standard deviations above the mean. Furthermore, this study defines evening hyperphagia as occurring after the last meal, allowing for more cultural flexibility in meal timing.

Treatment

The obesity pandemic has underscored the importance of identifying reversible causes of weight gain. Regardless of variations in nomenclature, night eating is a consistently described phenomenon in obese populations, is more common with increasing BMI, may be associated with treatment resistance, and has comorbid psychological findings distinguishing it from other eating disorders. These features suggest that NES is a unique condition that may respond to specific therapeutic intervention.

Studies have suggested that central nervous system (CNS) serotonin modulation may lead to an effective treatment of NES. In 1994 an investigation reported that all 6 patients treated with D-fenfluramine, over a 6–15 month follow-up period, had a pronounced reduction in the number of nighttime eating episodes and caloric intake. Another report described complete eradication of symptoms in 4 night eating subjects as early as 2 weeks after initiation of SSRI medication. Based on the relatively rapid effect in NES, it has been suggested that SSRIs act through a direct serotonergic effect in the hypothalamus which controls circadian rhythms and feeding behavior. Also, it has been suggested that the high carbohydrate food typically consumed in night eaters may be related to CNS serotonin modulation. In particular, a high carbohydrate-to-protein ratio facilitates the availability of tryptophan which is then converted into CNS serotonin that promotes the initiation of sleep and reversal of sleep disruption.

Clinical trials have considered whether SSRIs may lead to an effective NES treatment. One open-label, unblinded study investigated the effect of sertraline on NES. After 12 weeks the patients who completed the study reported less caloric intake at night, less nocturnal ingestions, and fewer awakenings. Another study assessed the effectiveness of sertraline in a group of 41 tele-medicine patients. After 8 weeks the subjects demonstrated decreased evening hyperphagia and the mean body weight of the overweight and obese subjects fell by 3 kg.

Recently, a double-blinded, placebo controlled randomized study assessed the efficacy of sertraline in the treatment of NES. Thirty-four patients were given a flexible-dose (50–200 mg/day) of the SSRI sertraline or placebo. Twelve of the 17 subjects (71%) in the sertraline group were classified as having responded compared to 3 of 17 (18%) in the placebo group. Furthermore, overweight and obese subjects in the sertraline group lost significantly more weight (–2.9 kg) after 8 weeks compared to the placebo group (–0.3 kg). The largest decrease in symptoms occurred by the second week, indicating an early effect. It is not known whether or not this is a specific sertraline effect or a more general SSRI effect.

Early studies have suggested that topiramate, an anti-seizure agent with anorexic effects, may be an effective treatment in relieving the abnormal feeding patterns of NES. In a case series of 4 treatment resistant patients (two with NES, two with SRED) topiramate was effective in both NES patients and in one SRED patient. Mean weight loss in all 4 subjects was 11.1 kg. In a case report of an obese NES patient with PTSD, 8 months of topiramate therapy was associated with a resolution of the night eating episodes and approximately 32 kg of weight loss. Controlled clinical trials are needed before determining the safety and efficacy of topiramate in NES.

Sedating agents have not been proven to be effective in treating NES and in many cases are associated with exacerbating night eating. In
a review of 23 patients enrolled in a clinical trial for NES, there were 16 reported exposures to sedating agents (primarily zolpidem). None of the exposures were considered effective.\(^{38}\) Importantly, zolpidem has been associated with triggering amnestic nocturnal eating in patients with and without NES.\(^{42,43}\) Non-pharmacologic interventions, such as phototherapy or relaxation training, have received scant mention in the literature. In conclusion, some NES therapies appear promising, but further research is needed. Sedating agents, in particular zolpidem, should be avoided as they are associated with unconscious nocturnal eating.

**SRED**

**Historical background**

Prior to the publication of the 1991 article that defined sleep-related eating disorder (SRED),\(^{44}\) there were several reported cases of nocturnal eating, including the first link between sleepwalking and nocturnal eating described in a 35-year-old woman with schizoaffective disorder, as reviewed in Ref.\(^{45}\). The latter was also the first case of unconscious nocturnal eating in association with psychotropic medications. A series of 3 sleep-related eating cases was published in 1990.\(^{46}\) All 3 patients described eating unpalatable foods such as raw bacon, often with subsequent amnesia, and PSG studies demonstrated disorders of arousal from delta NREM sleep.

In 1991, a case series was reported on 19 patients collected over 5 years who all described sleep related nocturnal eating.\(^{44}\) A follow-up report of 38 patients from the same series was published in 1993, (as reviewed in Ref.\(^{45}\)). Many patients had PSG evidence of a disorder of arousal, leading the authors to suggest that SRED is best classified as a parasomnia.

**Clinical characteristics**

The normal fast during sleep is lost in SRED. SRED is characterized by recurrent episodes of eating after an arousal from nighttime sleep with adverse consequences. The episodes are described as occurring in an involuntary or “out-of-control manner”. Often, patients cannot be easily awakened and in this regard SRED resembles somnambulism. Adverse consequences include: consumption of peculiar combinations of food or inedible substances, morning anorexia, dangerous food preparation behavior, sleep related injury, and weight gain.\(^{45,47,48}\) Patients often wake up with painful abdominal distention.

Preliminary epidemiological data suggest that SRED is a common disorder, particularly among eating disorder groups. A self-administered questionnaire determined prevalence rates of 16.7% in an in-patient eating disorder group, 8.7% in an outpatient eating disorder group, and 4.6% in an unselected group of college students.\(^{49}\) The majority (60—83%) of reported cases of SRED are female.\(^{45,47,50}\)

SRED is a relentless, chronic condition. In the original description of SRED, over half (57.9%) described nocturnal eating at least once a night.\(^{44}\) In another study the majority of patients described a long history of involuntary nocturnal eating (mean duration 15.8 years) and nearly all reported eating on a nightly basis (1—6 times per night).\(^{47}\) Recently, a study reported that of 35 patients with nighttime eating, 25 ate more than once a night and 8 eat more than 5 times a night.\(^{50}\)

SRED is characterized by consumption of high-caloric foods and sometimes bizarre substances. Foods high in carbohydrates and fats are typically eaten, and binging is common. The majority of patients report that breads, pies and dairy products such as ice cream, and also chocolate, other sweets, and peanut butter, are most commonly consumed. Interestingly, hunger is notably absent in SRED and the food choices are often not consumed during the daytime.\(^{47,50}\)

Food preparation and consumption has resulted in safety concerns and adverse health consequences. Hazardous activity has included: drinking excessively hot liquids, choking on thick foods, and lacerations from careless food preparation. Furthermore, inedible and toxic substances have been consumed such as frozen food, uncooked spaghetti, cat food, egg shells, coffee grounds, sunflower shells, buttered cigarettes, glue, and cleaning solutions.\(^{45,47,51}\)

Various medical and dental consequences can occur from repeated nocturnal eating. Weight gain is commonly reported, and the increased BMI may then precipitate, or aggravate, preexisting diabetes mellitus (type I or II), hyperlipidemia, hypercholesterolemia, hypertension, and OSA. Furthermore, patients with SRED are at risk of ingesting poisonous substances or food to which they are allergic.\(^{45,47,51}\) Patients with SRED are at risk for poor dentition as the feeding behavior, usually high in carbohydrates, is not typically followed by dental hygiene practices.\(^{45}\) Furthermore, many patients will sleep with an oral bolus of food which combined with the circadian decline in salivary flow promotes the development of caries.
Finally, failure to exhibit control over nocturnal eating can lead to secondary depressive disorders related to excessive weight gain.  

Polysomnography (PSG) has been used to characterize SRED. Patients are usually encouraged to bring in commonly consumed nocturnal food in order to facilitate eating behavior. If a patient eats during the PSG study, the concomitant sleep-wake state is then identified and the sleep technologist can assess level of awareness at the time and subsequent recall in the morning. Most commonly, eating behavior arises from NREM sleep. A recent study documented that 44 of 45 nocturnal feeding episodes in 26 patients arose from NREM sleep. Also, 22 patients demonstrated excessive periodic limb movements (>5/h). Rhythmic masticatory muscle activity (RMMA) associated with arousal from NREM sleep has been described in SRED, but only recently has been carefully documented. In the original SRED case series prominent repetitive chewing movements were described during NREM sleep and after arousals from both NREM and REM sleep. Recently, RMMA was found in 29 of 35 patients diagnosed with SRED during their PSG evaluations. The mean number of masticatory movements per night was 116 and 48% of them were associated with EEG arousal. The pathophysiologic link of this sleep-related RMMA to SRED needs to be elucidated, although the authors proposed a dopaminergic mechanism.

SRED is frequently associated with other sleep disorders, in particular parasomnias, but also restless legs syndrome (RLS) and obstructive sleep apnea. In the original series of 38 patients, sleepwalking was noted in 23 and in another series of 23 patients, 11 were diagnosed with sleepwalking. Importantly, sleepwalking without eating often precedes SRED and then once nocturnal eating develops, it often becomes the predominant or even the exclusive sleepwalking behavior. This pattern has led many researchers to consider SRED a "sleepwalking variant disorder," but also a "final common pathway disorder that can be triggered by a variety of sleep disorders, clinical conditions, and pharmacotherapies.

SRED is associated with psychological and psychiatric comorbidities. In the original 1991 case series 47% of SRED patients had an Axis 1 disorder and many had daytime anxiety about potentially choking or starting fires while asleep. A follow-up study noted that 53% of patients described a past history of repeated abuse and scores on the Dissociative Experiences Scale approached those typically seen in post-traumatic stress disorder. Furthermore, the onset of nocturnal eating is often correlated with family/relationship problems or withdrawal from substance abuse. In a study of 35 patients with nocturnal eating, 14 met criteria for a depressed mood; and a separate study noted that patients with SRED endorsed more symptoms of depression and dissociation than those without SRED.

SRED and amnesia

The presence of impaired consciousness has generally been a feature distinguishing NES from SRED. However, as mentioned level of consciousness (LOC) in NES has not been adequately characterized and the extent of impaired awareness and amnesia in SRED awaits further delineation. Also, level of awareness during eating episodes in
SRED may vary between episodes within the same night and from night-to-night over the longitudinal course of the disorder. In the original series of SRED cases, 84% of 38 patients described at least partial impairment in awareness. In another case series, 91% reported incomplete consciousness and/or amnesia for the eating behavior. Additionally, in a recent study all 26 patients diagnosed with SRED claimed full alertness after engaging in nocturnal eating the previous night in the sleep laboratory, and none had evening hyperphagia.

The spectrum of amnesia noted in previous studies may be explained by comorbid sleepwalking and the use of hypnotic and other psychotropic medications, (as discussed in Ref. 50). There is a strong association between a diminished level of awareness and a history of sleepwalking. In a community survey of 92 subjects who admitted to nocturnal eating only 17.5% of participants reported that they were at least partially unaware of the nocturnal eating. However, participants with a sleepwalking disorder were far more likely 73%, to be at least partially unaware of the behavior. Importantly, none of the participants who were fully aware of nocturnal feeding had a history of sleepwalking. In fact, prior to the original SRED case series the two reports of nocturnal eating with amnesia were associated with sleepwalking or psychotropic medication. Furthermore, the majority of the original 38 cases of SRED had a previous history of sleepwalking or were taking sedating medications. Moreover, amnesia is a common side effect of zolpidem, the agent most commonly associated with SRED. Finally, all 26 patients in a recent PSG study that reported full awareness were drug free and only one had a history of sleepwalking. However, the authors did not report whether the subjects had an altered level of consciousness during nocturnal eating episodes at home prior to enrollment in the study. Level of consciousness during eating may be different in a sleep laboratory compared to a more familiar sleeping environment (viz. at home).

Based on these reports, in which impaired consciousness was not a universal finding, albeit a common finding, the American Academy of Sleep Medicine chose not to include reduced awareness and subsequent amnesia as required diagnostic criterion for SRED in the International Classification of Sleep Disorders — 2nd version (ICSD-2). Given the published data currently available, we now propose a modification of the ICSD-2 criteria for SRED that would include a ‘‘level of consciousness’’ criterion that can be reconciled with abnormal eating behaviors, along with other changes listed in Table 3. Overlap disorders are listed in Table 3 in recognition of the considerable diversity that exists among patients with abnormal eating immediately preceding, and during, the main sleep period.

### Treatment

The abnormal eating in SRED can usually be controlled by treating any comorbid OSA or RLS/periodic limb movement disorder (PLMD), but treating SRED that is idiopathic or comorbid with sleepwalking remains a problematic issue. In the original 1991 case series, 8 sleepwalking patients were effectively treated with clonazepam and/or bromocriptine treatment. However, cumulative published experience indicates that benzodiazepine monotherapy is ineffective when SRED is not associated with sleepwalking and that either bedtime topiramate or dopaminergic therapy (levodopa, pramipexole, bupropion) are the preferred therapies. Furthermore, SRED patients with RLS/PLMD likewise respond to treatment with combinations of carbidopa/L-dopa, codeine and clonazepam. Additionally, dopamine dysfunction has been linked to both PLMD and rhythmic masticatory muscle activity, both of which are found in patients with SRED.

<table>
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<tr>
<th>Table 3 Clinical characteristics</th>
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<tr>
<td><strong>Timing of food intake</strong></td>
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<tr>
<td><strong>Level of consciousness during feeding</strong></td>
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<tr>
<td><strong>Unusual food intake (at times inedible substances)</strong></td>
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<tr>
<td><strong>Associated disorders</strong></td>
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<td><strong>Medication associations</strong></td>
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<td><strong>Reported treatments</strong></td>
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In SRED patients with OSA, continuous positive airway pressure has controlled sleep-related eating in both reported cases.\textsuperscript{45} Drug-induced SRED is usually fully-reversed by discontinuing the offending drug.\textsuperscript{42,43,45}

Early studies have shown that the anti-seizure medication topiramate may be an effective treatment for nocturnal eating and SRED. Anti-seizure treatment trials have demonstrated that topiramate is commonly associated with weight loss secondary to loss of appetite. A chart review of 30 patients treated with topiramate for SRED reported that 68\% of patients were considered treatment responsive over a mean follow-up period of 11.6 months, and 28\% lost more than 10\% of their body weight. Unfortunately adverse events were high and 41\% discontinued topiramate after 12 months.\textsuperscript{59} Paresthesias, visual symptoms, and rarely, renal calculus are side-effects of topiramate. In another series, published as an abstract, of SRED patients treated with topiramate approximately two-thirds of patients were treatment responsive, using weight loss as the primary outcome measure, and at a mean nightly dose of 102 mg there was a mean 9.2 kg weight loss over a mean follow-up interval of 1.8 years. In this series, topiramate was better tolerated.\textsuperscript{60}

Similar to NES, serotonin modulation may be effective in treating SRED. In the original case series, fluoxetine was effective in 2 of 3 SRED patients.\textsuperscript{44}

In conclusion, treatment should be initially aimed at controlling comorbid sleep disorders and eliminating provocative agents. At this time two types of pharmacotherapies appear to be potentially effective in controlling SRED, topiramate and dopaminergics. More research, in particular randomized controlled trials, are indicated in order to determine the efficacy and safety of SRED therapies.

SRED and NES conclusions

SRED and NES share similarities, as they both have a chronic course, familial associations, comorbid psychiatric disorders, and are frequently associated with weight gain and obesity. The more carefully NES and SRED are studied the more likely that overlap between groups will be identified. PSG at an accredited sleep center is appropriate to identify treatable comorbid sleep disorders, and also PSG findings that may be particularly associated with SRED, such as sleep related RMMA, PLMs, etc., which may contribute to further understanding of the pathophysiology (e.g., CNS dopaminergic dysfunction) of SRED. Food diaries should be systematically administered to SRED patients. Also, future research may identify clinical subtypes of NES associated with various sleep disorders besides insomnia, as already suggested.\textsuperscript{18,50} Thus, a uniform screening questionnaire encompassing both SRED and NES should be developed for clinical and research purposes, and there should be greater coordination among the sleep, eating disorders and obesity research fields to help standardize diagnostic criteria and facilitate further therapeutic trials. The relationships among serotonergic, dopaminergic, and other neurotransmitter dysfunction in NES and SRED can be explored within the context of therapeutic outcome trials.

If these conditions are to be considered distinct phenomena situated at opposite poles of pathology, then there may be certain nosological implications. NES could be considered an abnormality in the circadian rhythm of meal timing without interruption of the normal sleep state fast. Conversely, the feeding behavior in SRED could be considered a disruption in the normal fasting state of sleep regardless of recall ability. Furthermore, without a psycho-biological functional consequence, a clinical disorder cannot be diagnosed. Further research is needed to consider whether “asymptomatic” night eating is a prodromal condition or a normal variant found in the general population.

Practice points

1. Abnormal night eating has typically been categorized as either night eating syndrome (NES) or sleep-related eating disorder (SRED).
2. NES is described as evening hyperphagia, nocturnal eating and morning anorexia while SRED is characterized by recurrent episodes of eating after an arousal from nighttime sleep with adverse consequences.
3. NES is frequently familial and often associated with underlying mood disorders such as depression.
4. Most studies have demonstrated a relationship between nocturnal eating and obesity, however, it is not clear whether the disordered eating predates and contributes to the development of obesity.
5. Nocturnal eating is noted in normal weighted individuals who perceive the phenomena as nonproblematic.
6. Foods eaten in the NES are similar to daytime food intake while SRED patients describe eating in an "out-of-control manner" with foods that are atypical of daytime intake and occasionally include inedible substances such as raw meat or soap.
7. SRED is associated with psychotropic medications in particular zolpidem but other sedative hypnotics as well.
8. Food and sleep diaries should be administered to NES and SRED patients to characterize their condition and to monitor treatment response.
9. The night eating questionnaire (NEQ) followed by a formal interview is a reliable method to confirm the diagnosis of NES.
10. Polysomnography with a seizure montage is needed to identify reversible sleep disorders such as sleep apnea.
11. Studies suggest that sertraline, a selective serotonin reuptake inhibitor is an effective treatment for NES while the anti-seizure medication topiramate, may be effective for SRED.

**Research agenda**

1. Define the overlap and distinguishing features of NES and SRED.
2. Develop a uniform screening questionnaire for NES and SRED.
3. Determine if nocturnal eating predates and contributes to the development of obesity.
4. Consider whether asymptomatic night eating is a prodromal condition or a normal variant found in the general population.
5. Attempt to understand the serotonergic, dopaminergic, and other neurotransmitter dysfunction in NES and SRED in the context of clinical trials.
6. Greater coordination among the sleep, eating disorders, and obesity research fields.

**References**


* The most important references are denoted by an asterisk.