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# Sleep-Related Eating Disorders

## ABSTRACT

Although they share similar features, there are important differences between nocturnal eating syndrome and sleep-related eating disorder, which can be elucidated with a careful history and evaluation. While the former is best characterized as an eating disorder with associated insomnia, the latter is classified as a parasomnia, and is frequently affiliated with other primary sleep disorders. Nonsomatic therapies in isolation do not appear to be helpful for either condition, but effective pharmacotherapies have been described for both entities. Beyond the obvious discouragement and social embarrassment conferred by both conditions, there are also significant potential medical comorbidities, predominantly in the form of refractory obesity and related complications.

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## INTRODUCTION

When sleeping and eating behaviors are simultaneously affected, a fascinating spectrum of disease states may result. Nocturnal eating occurs in a variety of syndromes, but management can differ markedly, depending on the underlying etiology (Table 1). Nocturnal (night) eating syndrome (NES) and sleep-related eating disorder (SRED) will be the primary foci of this review. There is debate as to whether the two should be classified as independent entities or whether they should be considered as a continuum of a single condition involving eating urges and sleep disorders. This article will review the literature involving both topics and will strive to clarify nosologic and diagnostic ambiguities, with the ultimate goal of improving patient care.

**TABLE 1. Differential diagnosis of problematic nocturnal eating**

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| Nocturnal/night eating syndrome |
| Sleep-related eating disorder |
| Kleine-Levin syndrome |
| Dissociative disorder |
| Bulimia nervosa with nocturnal eating |
| Binge eating disorder |

## NIGHT (NOCTURNAL) EATING SYNDROME

**Historical background and clinical characteristics.** Stunkard and colleagues originally described NES ("night eating syndrome") in 1955 in 25 patients with refractory obesity.[1] Greater than 90 percent of the patients were female, and the median age was 35. Sixty-four percent of these individuals exhibited the triad of nocturnal hyperphagia, insomnia, and morning anorexia, with the conspicuous absence of daytime eating disorders. Although polysomnographic recording was not clinically available at this time, the behavior was reported as occurring during wakefulness, usually resulting in a complaint of sleep-onset insomnia due to hunger. The patients consumed high-calorie foods when symptomatic, and at least 25 percent of their daily caloric intake occurred after the evening meal. The frequency of episodes varied, but an association was noted with stressful life events and periods of weight gain. Psychopathology was quite prominent, particularly depression and anxiety.

Despite the distinctiveness of this clinical syndrome, only nine reports of nocturnal eating appeared in the literature during the 36 years following its original description, few of which referred to Stunkard's NES criteria.[1,25] In 1990, the entity of Nocturnal Eating (Drinking) Syndrome (also referred to as NES) was described in the first *International Classification of Sleep Disorders* (ICSD).[2] In contrast to Stunkard's original description,[1] the ICSD definition[2] primarily emphasized the awakenings of hungry infants in the context of a sleep-onset association disorder. Importantly, both definitions stipulate that other medical, psychiatric, or sleep disorders are not contributing to the sleep disturbance. Despite its description in the original ICSD,[2] studies frequently refer to NES in terms akin to Stunkard's criteria, resulting in nosologic confusion and a lack of diagnostic clarity.

**Demographics and associated features.** Based on survey studies involving community samples, the estimated frequency of NES is 1.5 percent in the general adult population.[26] Lundgren, et al., recently described an elevated frequency of 12 percent in a sample of outpatient psychiatric patients,[27] and the condition has been described in up to 27 percent of the severely obese adult population.[26] Increased frequency of the condition is also associated with substance abuse disorders and use of atypical antipsychotics.[27] With regard to the latter factor, the directionality of the relationship (and the confounding effect of medication-induced obesity) is unclear.

**TABLE 2. ICSD diagnostic criteria for sleep-related eating disorder (SRED)[30]**

**A. Recurrent episodes of involuntary eating and drinking occur during the main sleep period.**

**B. One or more of the following must be present with the recurrent episodes of involuntary eating and drinking:**
**i) Consumption of peculiar forms or combinations of food or inedible or toxic substances.**
**ii) Insomnia related to sleep disruption from repeated episodes of eating, with a complaint of nonrestorative sleep, daytime fatigue, or somnolence**
**iii) Sleep-related injury**
**iv) Dangerous behaviors performed while in pursuit of food or while cooking food**
**v) Morning anorexia**
**vi) Adverse health consequences from recurrent binge eating of high-calorie foods**

**C. The disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.**

The condition should be in the differential diagnosis for all those complaining of sleep disturbances, as one study reported that approximately six percent of subjects presented to a sleep disorders center with a complaint of insomnia-related symptoms typical of NES, roughly 70 percent of whom were female.[3] The average duration from onset to clinical presentation is 10 years.[3] More than 50 percent of patients correlate onset of the condition with stressful life events, and nearly 28 percent have concurrent major psychiatric diagnoses.

According to a case control study of 10 NES patients (mean BMI 28.5, mean age 57.3, 80%

women), more than 50 percent of daily caloric intake is consumed between the hours of 8 PM and 6 AM, compared with 15 percent consumed during the same time period by controls, as recorded by actigraphy and sleep diaries.[15] Approximately 70 percent of evening caloric intake in NES patients consists of carbohydrates, as compared to roughly 47 percent during the day. Nighttime awakenings are also more common in the afflicted, with over 50 percent of awakenings occurring in association with eating episodes.

**Polysomnographic findings.** Two polysomnographic studies (one with a case-control design) of patients with NES showed no sleep abnormalities other than decreased sleep efficiency and total sleep time.[3,5] Seventy-nine percent of polysomnographically recorded awakenings with food consumption emanated from non-rapid-eye-movement (NREM) sleep (but none from slow-wave sleep) in the case-control study,[5] and full preservation of consciousness has been consistently described, with patients describing a compulsion to eat in the absence of hunger.[3,5] NES subjects were differentiated from a group of binge eaters and "nonspecific" nocturnal eaters by shorter periods of "eating latency" and "latency in recapturing sleep after eating episode," suggesting a close association between arousal and eating.[3]

**Pathophysiology.** Birketvedt and colleagues studied neuroendocrine parameters in a group of 12 female NES patients (mean age 53 years) and compared them to 21 controls.[15] Patients were admitted to a Clinical Research Center for 24 hours, given scheduled meals with a specified caloric content, and underwent serial blood samples. Comparisons were made between obese and nonobese subjects from each group (mean body mass index of obese NES and control subjects were 36.1 and 30.4, respectively). Both nocturnal melatonin levels and the nocturnal rise in leptin were lower in NES subjects compared to controls. The authors suggest that dysregulation of these hormones may result in inadequate suppression of appetite, impaired sleep consolidation, and could ultimately reflect underlying dysfunction of the hypothalamic-pituitary-adrenal axis. Although these findings were enticing explanations for observed behaviors, a more recent study failed to replicate them and found no significant differences in nocturnal melatonin or leptin levels when comparing NES patients to controls.[17]

A separate group analyzed 24-hour endocrine samples in seven subjects (1 female, mean age 21.7 years, mean BMI 22.9) in a randomized protocol with cross-over to either a diurnal or nocturnal lifestyle, the latter of which conformed to the eating pattern described in those with NES.[16] In the nocturnal group, melatonin and leptin analyses revealed the same patterns as the Birketvedt study, suggesting that the latter group's findings may have been due to the nocturnal lifestyle alone, with the confounding effects of light and its suppression of melatonin, in addition to the entrainment of leptin to meal timing. The cumulative data, therefore, suggest that deviations of endocrine parameters in NES patients, if present, are likely secondary to nocturnal eating behaviors rather than causative of them.

> Lundgren, et al., recently described an elevated frequency [of NES] of 12 percent in a sample of outpatient psychiatric patients,[27] and the condition has been described in up to 27 percent of the severely obese adult population.

**Genetic factors.** Although data regarding the heritability of NES is limited, a recent study demonstrated an aggregation of the condition among family members.[27]

> Increased frequency of [NES] is associated with substance abuse disorders and use of atypical antipsychotics.[27]

## SLEEP-RELATED EATING DISORDER

**Historical background and clinical characteristics.** Schenck and colleagues first described SRED in 19 adult patients in 1991.[6] This was extended to a series of 38 patients, over 90 percent of whom were evaluated with polysomnography.[11] While the condition observed shared many overlapping features with NES, it was distinguished by a preponderance of partial or complete amnesia for eating episodes, a strong association with other sleep disorders, and a high frequency of arousals from slow-wave sleep, characterizing it as a variant of a NREM arousal parasomnia. Furthermore, none of the patients had eating disturbances prior to sleep onset or complained of the initial insomnia typical of Stunkard's description of NES.

**Demographics and associated features.** One study estimated the prevalence of SRED at nearly five percent in the general population, and at roughly 9 to 17 percent in

those with eating disorders.[28] According to studies by Schenck, et al., and Winkelman, the average age of onset of SRED is approximately 22 to 27 years, with a mean of approximately 12 to 16 years before clinical presentation.

Prior sleep diagnoses are reported in nearly 80 percent of subjects with SRED, including restless legs syndrome, periodic limb movements of sleep, and somnambulism (either exclusively or in combination with another parasomnia).

Greater than 65 percent of the patients are female, and more than 40 percent are overweight. More than 80 percent of patients describe a diminished level of consciousness during eating episodes, with a varying degree of amnesia for the events.[7,11] As there is significant variability in level of alertness during eating episodes, both within and between nighttime periods, this characteristic is not required for the diagnosis.[7,14] Criteria from the recently published *International Classification of Sleep Disorders, Second Edition* (ICSD-2), are shown in Table 2.

Nightly eating is reported in approximately 70 percent of SRED patients, usually in the absence of hunger or thirst. More than 65 percent of these patients ingest unpalatable substances, such as frozen foods and buttered cigarettes. Injuries from careless food preparation occur in approximately one-third of patients.[11] Forty-four percent attribute their overweight status exclusively to nocturnal eating.[11] Winkelman's series described a 35-percent prevalence of associated eating disorder diagnoses, whereas only five percent of Schenck's series had a lifetime diagnosis, a discrepancy perhaps affected by selection bias.

Prior sleep diagnoses are reported in nearly 80 percent of subjects, with most describing restless legs syndrome (RLS), periodic limb movements of sleep (PLMS), or somnambulism, either exclusively or in combination with another parasomnia.[7] Stressful events are associated with onset of symptoms in approximately 16 percent of cases.[7,11] Associated psychiatric disease is commonly present, including affective disorders in 37 percent, anxiety disorders in 18 percent, and prior substance abuse in 24 percent.[11]

Many drugs have been implicated in the initiation of SRED, including triazolam, amitriptyline, olanzapine, and risperidone.[8,10,11] Two series have described associations with zolpidem.[9,12] In Winkelman's series of 37 patients, 70 percent of the patients were taking psychotropic drugs at the time of the study. In Schenck's series, psychotropic drug use was also reported, but it is unclear at what frequency they were used.[11] The presence of psychotropic medications may bear relevance to the varying levels of alertness described in association with eating episodes.[14]

Associated psychiatric disease is commonly present in cases of SRED, including affective disorders, anxiety disorders, or prior substance abuse.

**Polysomnographic findings.** In the two series initially published, polysomnography demonstrated findings consistent with somnambulism in roughly 50 to 70 percent of patients, PLMS in approximately 25 percent, and obstructive sleep apnea (OSA) in 10 to 14 percent.[7,11] In 5 of 6 patients in whom nocturnal eating was observed, all arousals emanated during NREM sleep and exclusively during slow-wave sleep in three individuals.

**Genetic factors.** Consistent with other arousal disorders, a familial relationship has been described in 5 to 26 percent of SRED subjects in two case series.[7,11] De Ocampo and colleagues described a woman with SRED whose fraternal twin sister and father were also affected.[29]

**Pathophysiology.** Physiologic studies have not been performed in patients with SRED. In those with concomitant sleep disorders, it has been suggested that arousals may provoke varying levels of consciousness, prompting somnambulism and sleep-related eating in predisposed individuals.[7] A recent videopolysomnographic study described recurrent swallowing and chewing movements during NREM sleep in 29 of 35 patients (83%).[14] Bearing in mind treatment reports describing efficacy with dopamine agonists,[13] the authors proposed that dopaminergic mechanisms may be involved in the pathophysiology of the condition.

**Differential diagnosis of syndromes of nocturnal eating.** SRED and NES should be differentiated from other conditions associated with nocturnal eating (Table 1). Kleine-Levin syndrome may be associated with compulsive eating during waking periods, but occurs predominantly in adolescent males,

classically in concert with hypersexuality, behavioral abnormalities, and periodic hypersomnolence. Nocturnal eating associated with dissociative disorders occurs with varying levels of awareness during electroencephalogram (EEG)-defined wakefulness, but individuals commonly exhibit other psychopathology associated with the primary psychiatric illness.

> Stressful events are also associated with onset of symptoms.

Controversy exists as to whether NES in particular represents an extension of a daytime eating disorder. In the case of binge eating disorder and bulimia nervosa (BN) with nocturnal eating, the eating disorder continues to manifest itself throughout the day. In further support of its distinction from daytime eating disorders, one study compared NES patients with four subjects with BN. The BN group did not report nighttime awakenings, had less eating episodes per day, and consumed, on average, approximately seven times more than the night-eating subjects.[15]

**Evaluation.** A thorough sleep history is essential to recognition and diagnosis of sleep-related eating disorders. Most patients will not describe the complaint spontaneously, and therefore direct questioning may be required. The timing, frequency, and description of food ingested during eating episodes should be elicited, and a history of concurrent psychiatric, medical, and sleep disorders must also be sought and evaluated. A complete medication history, including when pharmaceuticals were started, is required.

In some patients (e.g., those with associated RLS), the history alone may be sufficient, and an empiric therapeutic trial may be appropriate without additional studies. However, if other sleep disorders are suspected (e.g., OSA) or if the episodes are stereotypic or have resulted in injury, polysomnography should be performed. Ideally, food should be placed at the bedside, and a synchronized video should be recorded. When seizures are a consideration, a full EEG montage should be employed.

## TREATMENT OF NES AND SRED

**NES.** Treatment reports of both NES and SRED are primarily anecdotal at this juncture, but two small controlled trials have been published. An eight-week, double-blind, randomized, controlled trial utilizing sertraline in NES patients showed improvement in 12 of 17 (71%), 58 percent of whom experienced remission or complete resolution of symptoms, as determined by a variety of assessment scales.[23]

In a case series of four patients with nocturnal eating ($n$=2 SRED, 2 NES) administered topiramate, one patient with NES had complete remission of nocturnal eating, and the remainder had either a moderate or marked response to treatment. Notable weight loss was observed in all patients, and benefits were maintained at a mean of 8.5 months.[20] In a series of seven NES patients, d-fenfluramine induced complete recovery in one patient, and ≥50-percent reduction in the number of nocturnal eating episodes in an additional five subjects.[4] As this medication was removed from the market, it no longer represents a viable treatment option.

Nonpharmaceutical options for treatment of NES are limited. One case report described efficacy with morning light therapy.[31] Acknowledging the role of stress in the condition, a placebo-controlled study of 19 women with NES examined the role of relaxation training and failed to demonstrate statistically significant differences in the number of nocturnal eating events between groups.[24]

**SRED.** In a small, double-blind, placebo-controlled trial utilizing pramipexole in patients with SRED, nocturnal median motor activity was decreased (as assessed by actigraphy) and subjective improvement of sleep quality was reported.[13] Nevertheless, 27 percent of subjects had RLS (a condition known to respond to this medication), and number and duration of waking episodes related to eating behaviors were unchanged.

Capitalizing on the positive response demonstrated in the aforementioned case series,[20] a more recent study, presently available only in abstract form, described efficacy of topiramate in 17 consecutive patients with SRED.[21] In the 65 percent of patients who remained on the medication, all reported either substantial improvement or complete remission of night-eating, and significant weight loss was achieved (mean 9.4kg). Finally, remission of nocturnal eating has been described in two SRED patients treated with fluoxetine[11] and, although there is some confusion as to the diagnostic category described, a recent case series appears to describe successful treatment of SRED with paroxetine or fluvoxamine.[32]

Treatment targeted to associated sleep disorders is also essential. In Schenck's series, five patients with RLS/PLMS and one patient with somnambulism and PLMS had remission of their SRED with combinations of carbidopa/L-dopa, codeine, and clonazepam.[11] Two patients with OSA had resolution of their symptoms with nasal continuous positive airway pressure (nCPAP) therapy. Clonazepam monotherapy was effective in 50 percent of patients with coexisting somnambulism.[11]

Interestingly, dopaminergic agents as monotherapy were effective in 25 percent of the SRED-sleepwalking subgroup, and

in combination with benzodiazepines (mainly clonazepam), opiates, or both in approximately 87 percent of subjects.[11] Success with combinations of dopaminergic and opioid drugs, with the occasional addition of sedatives, has also been described in a series of seven patients without associated sleep disorders.[19] In those for whom opioids and sedatives are relatively contraindicated (e.g., in those with histories of substance abuse), two case reports have described success with a combination of bupropion, levodopa, and trazodone.[18]

Notably, hypnotherapy, psychotherapy, and various behavioral techniques, including environmental manipulation, have not been demonstrated to be effective.[11]

Nevertheless, behavioral strategies should complement the overall treatment plan and should include deliberate placement of food to avoid indiscriminate wandering, maintenance of a safe sleep environment, and education regarding proper sleep hygiene and stress management. Offending medications must also be eliminated.

## CONCLUSION

Nocturnal eating is an element of a variety of syndromes. The distinction between NES and SRED can at times be challenging, and there is debate as to whether the two conditions represent separate entities or simply two poles of a pathophysiologic continuum. Features common to both conditions include heritability, an increased prevalence of psychopathology, a female predominance, and a temporal connection between onset and stressful events. Moreover, both may result in social embarrassment, pronounced sleep disruption and ultimately health problems secondary to obesity. The pathophysiology of both processes is unclear.

In an attempt to eliminate nosologic confusion, NES is no longer recognized in the sleep lexicon, as it is viewed as an eating disorder with associated initial insomnia. In contrast, SRED requires a disturbance of sleep maintenance and is classified as a parasomnia. In many respects, publication of the ICSD-2 has served to blur the distinction between the two disorders, and many cases that were formerly characterized as NES would now be characterized as SRED, due to the lack of a criterion for altered consciousness and the inclusion of associated sleep disorders. Whether this more inclusive definition will advance the understanding of nocturnal eating is unclear, but standardized criteria should enhance the reliability of diagnoses at different centers.

Evaluation of these conditions consists of a detailed sleep history, in conjunction with a complete medical and psychiatric history. Workup frequently requires polysomnography with synchronized video and a full EEG montage. Food placed at the bedside is recommended.

Non-somatic therapies in isolation appear unhelpful in treating either condition. Treatment of underlying sleep disorders is essential. Two small controlled trials report efficacy of sertraline and pramipexole in the treatments of NES and SRED, respectively, and anecdotal reports describe success with topiramate and other dopaminergic agents, either alone or in combination with opioids and sedatives. Success with other selective serotonin reuptake inhibitors has also been reported in SRED. A sole case report describes the benefit of morning light therapy in a patient with NES. Behavioral interventions comprise a comprehensive treatment plan.

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