

Familial Aggregation in the Night Eating Syndrome

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ABSTRACT

Objective: This study examined the extent to which the night eating syndrome (NES) affects first-degree relatives of NES and control probands.

Method: NES participants and controls were assessed with the Night Eating Questionnaire (NEQ), the Night Eating Syndrome History and Inventory (NESHI), 10 day sleep and food records, the Eating Disorder Examination (EDE), the Structured Clinical Interview for DSM IV Axis I Disorders (SCID I), and a Family History Questionnaire (FHQ) to assess the presence of NES among first-degree relatives. A proband predictive model, using logistic regression analyses and the generalized estimating equation to control for correlation among observations within families was used to assess familial aggregation.

Results: The odds of an NES proband having an affected first-degree relative were significantly greater than that of a control proband (odds ratio = 4.9, $p < .001$). A number of covariates were included in the model: proband body mass index (BMI) (kg/m^2), proband gender, proband age, proband ethnicity, first-degree relative gender, relationship to proband (i.e., mother, father, or sibling), and the interaction between relationship to proband and proband status (night eater or control); none was statistically significant ($p > .05$).

Conclusion: The study showed a strong aggregation of NES in families. © 2006 by Wiley Periodicals, Inc.

Keywords: night eating syndrome; familial aggregation; first degree relatives

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Introduction

The night eating syndrome (NES) is characterized as a delay in the circadian pattern of food intake manifested by evening hyperphagia and/or nocturnal awakenings with ingestion of food.^{1,2} Night eaters frequently report morning anorexia, insomnia, and negative mood that worsens during the evening; the onset of NES is often associated with life stress.³

The etiology of NES is unknown, but genetic influences have been suspected. The response of NES to sertraline⁴ suggests that genes regulating serotonin gene may be involved. Additionally, a recent study found that mice with a mutation in the *Clock* gene consume significantly more food than wild-type mice during the light period (when mice usually sleep more and eat less) and become obese.⁵

A first step in understanding potential genetic and environmental influences is a study of familial aggregation. Accordingly, the current study examined the extent to which the NES phenotype affects first-degree relatives of both night eaters and control participants.

Methods

Participants

Night eaters were recruited from printed advertisement, radio talk shows, and television commercials describing NES. Controls, recruited from printed and radio advertisements, were sought to approximate the age, gender, body mass, and ethnicity of the night eaters. Advertisements for both night eaters and controls included the following eligibility requirements: age 18 or older, not currently involved in an occupation requiring nightshift work, not currently enrolled in a weight reduction program, and not currently taking any psychotropic medications. Advertisements to recruitment night eaters specified that they must have difficulties with overeating after dinner and/or with waking up at night to eat; advertisements for controls stated that they could not experience these difficulties.

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Participants included 103 adults diagnosed with NES (mean age = 43.0 ± 11.3 years; mean BMI = 33.3 ± 8.1 kg/m²; percentage white = 62.1; percentage female = 71.8) and 42 control participants (mean age = 39.3 ± 11.3 years; mean BMI = 36.0 ± 6.2 kg/m²; percentage white = 59.5; percentage female = 66.7). Night eater and control participants differed significantly only on BMI ($p = .05$); accordingly, proband BMI was included as a covariate in the analyses.

Measures

The Night Eating Questionnaire (NEQ⁶) was used to assess the presence of night eating behavior. It is comprised of 14 questions assessing hunger and craving patterns throughout the day, percentage of calories ingested after suppertime, feelings of depression, insomnia and awakenings, and nocturnal food cravings and ingestions.

The Night Eating Syndrome History and Inventory (NESH; unpublished semi-structured interview) was used in conjunction with food records to establish a diagnosis of NES. The NESH included questions about the schedule and amount of food intake throughout the 24-hour day, history of NES symptoms, sleeping routine, mood symptoms and life stressors, weight and diet history, and previous treatment strategies for NES.

The Eating Disorder Examination (EDE⁷) was used to assess the presence of any concurrent eating disorder. It is a semi-structured clinical interview that assesses dietary restraint, eating concern, and weight and shape concern, as well as bingeing and purging behaviors.

The Structured Clinical Interview for DSM IV Axis I Disorders (SCID I⁸) was used to formally assess the presence of Axis I psychiatric diagnoses.

Night eaters and controls completed detailed 24-hour food records for 10 consecutive days. The first two days were practice days and day 10 did not include nighttime data. Accordingly, days 3–9 were used to confirm a diagnosis of NES. Food intake was analyzed using ESHA Food Processor version 8.0.

A family history questionnaire (FHQ) was completed by NES and control probands to assess the presence of NES among parents, siblings, and offspring. Proband responses were compared with their self-report during the NESH interview. When discrepancies were encountered and when the probands were unsure about the presence of NES, the first-degree relative was coded “unaffected.”

BMI was calculated from height and weight measured at their interview visits. Normal weight was defined as 18.5–24.9 kg/m², overweight was 25–29.9 kg/m², and obese was 30+ kg/m².

Procedure

As part of a comprehensive study of the characterization of NES, NES and control participants provided informed consent, completed the NEQ and the FHQ, and heights and weights were measured. NES participants were interviewed using the NESH to assess their NES symptoms. Both NES and control participants were interviewed with the EDE and kept detailed 24-hour food records for 10 consecutive days to confirm their diagnostic status.

Participants were diagnosed as “NES positive” if they met the following criteria based on food records and NESH interview: (1) consuming ≥25% of total daily caloric intake after dinner, and/or (2) nocturnal awakenings with ingestion of food occurring ≥3 times/week. Participants must have experienced symptoms for no less than 3 months based on self-report. This operational definition was modified from previously described criteria¹ because recent data with a larger sample of night eaters and controls found that night eaters consumed 34.6% ± 10.1% of total daily caloric intake after dinner compared with controls who consumed 10.0% ± 6.9% of total daily caloric intake after dinner.² Based on a figure of 2 *SD* above the mean for controls, 25% of calories consumed after the evening meal was considered abnormally large.

Participants who also met criteria for binge eating disorder (BED; NES, $n = 20$; control, $n = 0$) were retained in the analyses because the literature suggests that BED and NES are two distinct conditions, although they may co-occur.^{9,10}

Statistics

The proband predictive model described by Hudson et al.¹¹ and Laird and Cuenco¹² was used to predict the binary outcome (affected or not affected) of first-degree relatives. One logistic regression analysis was conducted, using the generalized estimating equation (GEE) with exchangeable correlation structure to control for correlation among the family members. Status as NES or control proband was used as the predictor; covariates in the model were proband BMI, proband age, proband gender, proband ethnicity (white vs. non-white), first-degree relative gender, first-degree relative relationship to the proband (mother, father, or sibling), and the interaction between proband status (night eater or control) and first-degree relative relationship to the proband.

Results

The first-degree relatives of NES probands had significantly greater odds of being affected with NES than the first-degree relatives of control probands (odds ratio [OR] = 4.9; standard error [SE] = 2.9; 95% confidence interval [CI] = 2.0, 12.3; $p = .0006$).

None of the covariates was statistically significant (all p values $> .05$) including proband BMI, proband age, proband gender, proband ethnicity, first-degree relative gender, relationship to proband (mother, father, or sibling), and the interaction between relationship to proband and proband status (night eater or control).

Conclusion

This is the first study to examine the familial aggregation of NES. It found that NES was more likely to aggregate among family members of night eaters than of controls, suggesting that NES is familial. Congruent with our findings, a recent study of German children (5–6 years of age) and their mothers by Lamerz et al.¹³ found that the children of mothers who engaged in night eating behavior were 7.8 times more likely than children of non-night-eating mothers to show signs of the syndrome.

The strengths of the current study, in contrast to the study carried out by Lamerz et al.,¹³ included the use of an adult sample of probands, parents, and sibling, diagnosis of probands using food and sleep records in conjunction with clinical interview, and robust statistical methods using the generalized estimating equation to control for correlated family data.

Although 20 of the NES probands also met criteria for BED, only seven of those concurrently diagnosed reported a family history of NES. It is unlikely that familial aggregation can be attributed to BED aggregation. Several NES probands, however, also met criteria for an Axis I disorder; co-aggregation of NES and Axis I disorders (e.g., Major Depressive Disorder) should be examined in future studies.

Although a first step in understanding the etiology of NES, this study was limited in the following ways. First, the family history method, rather than the family interview method, was used to assess night eating among first-degree relatives. Because the limitations of the family history method are well documented,¹⁴ we sought, but we were unsuccessful, in recruiting family members to participate in verification interviews. Second, some probands were unsure if family members were affected with NES, and were, therefore, considered unaffected. Third, control probands may have been less likely to recognize NES among relatives compared with NES probands, for which the disorder is more salient. Fourth, the study was nonblinded in that the interviewer knew the diagnosis of the proband at the time the family history was obtained.

Despite these limitations, the study was able to show strong familial aggregation of NES; these results highlight the need for a classic twin study of NES.

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