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Night eating behavior and metabolic heath in mothers and fathers enrolled in the QUALITY cohort study

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ABSTRACT

Background: Desynchrony between eating and sleeping patterns and poor sleep quality have been associated with obesity and metabolic abnormalities. This study examined the metabolic health correlates of night eating syndrome in adults enrolled in the QUALITY cohort study.
Methods: Night eating symptoms were assessed in 310 women (mean age = 40.3 ± 5.1 years, mean BMI = 28.8 ± 6.2 kg/m^2) and 305 men (mean age = 42.5 ± 5.9 years, mean BMI = 30.3 ± 5.0 kg/m^2). Anthropometric measures, fasting blood samples and blood pressure were used to diagnose metabolic syndrome (MetS) and type 2 diabetes (T2D) diagnosis was self-report. Correlational and case/control comparisons assessed night eating symptoms in persons with and without MetS and T2D.
Results: Night eating questionnaire (NEQ) scores were positively correlated with BMI. When controlling for BMI, NEQ scores were significantly negatively correlated with blood pressure in women and positively correlated with waist circumference and triglycerides in men. MetS diagnosis was associated with morning anorexia in both women and men and urges to eat at night in women only. T2D was associated with a depressed mood in women and with insomnia in men.
Conclusion: Symptoms of night eating syndrome are associated with higher BMI and poor metabolic health. Future research is needed to determine if night eating syndrome per se is a unique causal pathway in the development of obesity and metabolic disease.

1. Introduction

Night eating syndrome (NES) is a disorder of circadian delayed eating behavior with intact circadian sleep onset and offset; it has been recognized among obese individuals since the 1950s (Stunkard, Grace, & Wolff, 1955). Research diagnostic criteria include the core features of evening hyperphagia (i.e., consumption of ≥25% of total daily food intake after the evening meal) and/or nocturnal ingestions of food (≥two episodes/week), awareness of night eating behavior, and three of five associated appetite, mood, and sleep features (Allison et al., 2010). The etiology of NES is unknown, but shift work or other lifestyle/medical conditions that better explain the circadian disruption in eating behavior preclude the diagnosis (Allison et al., 2010).

Night eating behavior has been associated with weight gain over time (Andersen, Stunkard, Sorensen, Petersen, & Heitmann, 2004) and diabetic complications (Morse, Ciechanowski, Katon, & Hirsch, 2006), and individuals seeking weight loss have a higher prevalence of night eating behavior compared to general population samples (Gallant, Lundgren, & Drapeau, 2012). A circadian analysis of neuroendocrine hormones that regulate appetite in persons with NES compared to healthy controls documented disruption in the circadian rhythms of leptin (1.0 h phase delay), ghrelin (5.2 h phase advance), glucose (11.6 h phase advance/12.4 h phase delay), and insulin (2.8 h delay) (Goel et al., 2009). This cross-sectional study cannot clarify if night eating is the cause or the effect of circadian disruption in neuroendocrine hormones. Nonetheless, the circadian dysynchrony in these appetite regulating hormones could have a negative impact on metabolic health.

Abbreviations: NES, night eating syndrome; NEQ, night eating questionnaire; MetS, metabolic syndrome; T2D, type 2 diabetes.

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Circadian dysynchrony between eating and sleeping patterns has been associated with obesity and metabolic abnormalities in both rodents and humans. Animal models of circadian misalignment (e.g., genetic mutations, shift work) have demonstrated changes in physiological processes that can result in detrimental health effects. For example, Turek et al. (2005)) reported that *Clock* mutant, compared to wild-type mice, all fed a regular diet had significantly higher mean daily serum levels of triglycerides, cholesterol, glucose, and leptin. In addition, energy intake and body weight increased significantly over a 10-week period in the *Clock* mutant mice only. In an animal model of shift work, Salgado-Delgado, Angeles-Castellanos, Saderi, Buijs, and Escobar (2010) demonstrated that, compared to control rats, rats required to be active for 8 h during their normal resting phase (light phase) increased food intake during the resting phase with consequent weight gain and loss of glucose rhythmicity.

In humans, epidemiological studies of shift workers have documented an association between irregular work schedules and metabolic syndrome. In their review, Wang, Armstrong, Cairns, Key, and Travis (2011) reported that rotating and night shift work are associated with an increase in metabolic syndrome risk (odds ratios ranged from 1.46 to 3.50), but that the risk varied with the population under study and the definition of metabolic syndrome used.

Similar results have been observed after experimental circadian desynchronization in humans. Qin and colleagues (Qin et al., 2003) found that students required to eat the majority of their daily food intake in the evening and to sleep from 0130h to 0830h, experienced changes in their circadian rhythms of insulin and glucose, such that the response of insulin to glucose became impaired. Scheer, Hilton, Mantzoros, and Shea (2009) forced a 12-hour circadian misalignment in 10 adults. Compared to the baseline phase, the misalignment resulted in an increased mean arterial pressure, and increased postprandial glucose response despite an increase in insulin, along with a consistent 17% decrease in leptin throughout the "day", which was independent of the observed reduction in sleep efficiency.

In addition to the shift/night work research demonstrating a relationship between circadian misalignment and metabolic health, there is evidence that poor sleep duration and quality contribute to obesity and metabolic health problems (Crispim et al., 2007; Knutson, Spiegel, Penev, & Van Cauter, 2007; Spiegel, Tasali, Leproult, & Van Cauter, 2009). This occurs through both homeostatic (e.g., appetite stimulating hormone up-regulation) and non-homeostatic (e.g., increased sensitivity to the reward properties of food) eating behaviors (Chaput & Tremblay, 2012). This literature is particularly relevant to NES, because although sleep timing is not circadian advanced or delayed (Rogers et al., 2006), people with NES often have sleep onset and/or sleep maintenance insomnia (Allison et al., 2010) and reduced sleep efficiency (Rogers et al., 2006).

Given the growing literature on the negative health consequences of circadian misaligned eating and sleep behavior, as well as the impact of poor sleep quality on eating behavior on weight gain and metabolic health, it is important to examine the health correlates of NES. This is especially true in populations which have an increased prevalence of night eating behavior and are already at risk for diabetes and metabolic syndrome, such as obese individuals (Gallant et al., 2012) and persons taking antipsychotic medications (Lundgren et al., 2006). Notably, despite the provisional status of NES in the psychiatric nomenclature as an Other Specified Feeding or Eating Disorder (American Psychiatric Association, 2013) as well as its potential diagnostic overlap with sleep, mood, and other eating disorders, night eating behaviors can still have a negative impact on health. In fact, the study of specific behaviors in relation to health in contrast to full syndromes is beneficial in that the clinical health significance of particular behaviors can be evaluated. The current study, therefore, tested the hypothesis that night eating and associated behaviors which constitute the night eating syndrome research diagnostic criteria (Allison et al., 2010) would be associated with obesity, metabolic syndrome and type 2 diabetes in men and women.

2. Materials and methods

2.1. Participants

Participants included 310 women and 305 men enrolled in the QUALITY (Quebec Adiposity and Lifestyle Investigation in Youth) cohort study, a longitudinal study of 630 families initiated in 2005 to assess risk factors for cardiovascular disease and type 2 diabetes in children at risk for obesity (for details see Lambert et al., 2012). Children and their parents were recruited in elementary schools located in the Montreal and Québec City areas. Inclusion criteria for the QUALITY study specified that eligible children be 8–10 years old at the time of recruitment, and that at least one biological parent was obese (BMI >30 kg/m^2) or abdominally obese (waist circumference >88 cm for women, or 102 cm for men). Only families of Caucasian origin were included to reduce genetic admixture. All parents provided signed informed consent. Children were not included in the present study because night eating behavior is rare in young persons and a baseline diagnosis of type 2 diabetes in children was an exclusion criterion for the QUALITY study. The study was approved by the ethics review boards of Laval University and CHU Ste. Justine.

2.2. Assessment

2.2.1. Night eating behavior

The Night Eating Questionnaire (NEQ; Allison et al., 2008) was added to the QUALITY study assessment procedures in 2006, after the study initiation. As such, only a subset (310 women and 305 men) of the 630 parental couples who completed the baseline QUALITY assessment had NEQ data available for analyses. The NEQ is a 14-item, self-report instrument that assesses the behavioral and psychological symptoms of NES. The NEQ assesses morning hunger and timing of first food consumption (2 items), food cravings and control over eating behavior both before bedtime (2 items) and during nighttime awakenings (2 items), percent of food consumed after dinner (1 item), initial insomnia (1 item), frequency of nocturnal awakenings and ingestion of food (3 items), mood disturbance (2 items), and awareness of nocturnal eating episodes (1 item, included to differentiate a diagnosis of Sleep-Related Eating Disorder from NES, but not included in the total score). Each item is rated on a five point Likert-type scale (0–4 points); total scores can range from 0 to 52 points. For the QUALITY study, the NEQ was translated from English to French and then back-translated to ensure translation accuracy. The NEQ was self-administered by participants on their testing day.

The NEQ can be used as a continuous measure, describing the severity of the NES symptoms (Allison et al., 2008), or it can be used to assess symptoms of NES based on research diagnostic criteria (Allison et al., 2010; Lundgren et al., 2012). The prevalence of night eating behavior in the QUALITY cohort parents and children, as well as its familial co-occurrence, has been previously reported (Lundgren et al., 2012). Full threshold NES was reported in 0.5% of women and 0.3% of men, but the prevalence of evening hyperphagia and nocturnal ingestions of food was slightly higher, ranging from 0.5% to 3.3%. For the current analyses, the NEQ was used as a continuous measure of night eating syndrome symptomatology and specific symptoms were dichotomized (present/not present) using the following cut-points: a) evening hyperphagia: ≥25% total energy intake after the evening meal; b) nocturnal ingestions of food: at least half the time with sleep maintenance troubles >once per week; c) morning anorexia: no appetite at all in the morning and/or eat for the first time after 12h00; d) urge to eat at night: very much so/extremely so for cravings for food in the evening after supper and/or during night awakenings; e) insomnia: sleep onset troubles usually or always and/or sleep maintenance troubles >once a week; f) belief that one must eat in order to return to sleep: ≥somewhat of a belief; and g) depressed mood: currently feeling somewhat/very much so/extremely blue and/or when