Behavioural and Metabolic Characterisation of Night Eating Syndrome

Thèse

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Résumé

Le syndrome de fringale nocturne (SFN) est caractérisé par un retard de l'apport énergétique et se manifeste soit comme une hyperphagie en soirée et/ou des ingestions nocturnes. D'autres caractéristiques cliniques de ce syndrome ont été identifiées : anorexie du matin, l'insomnie, un désire intense de manger le soir, une humeur dépressive et/ou une humeur qui se détériore en soirée, et la conviction que la personne doit manger pour dormir. Bien que la recherche sur NES a progressé de façon constante depuis l'apparition de ce syndrome dans la littérature scientifique en 1955, plusieurs questions n'ont pas été étudiées. En effet, une seule étude a examiné les symptômes du SFN chez les enfants et aucune n'a examiné les comportements de santé associés aux symptômes. De plus, bien que deux études aient démontré que les ingestions alimentaires nocturnes prédisent une plus grande prise de poids, il n'est pas clair si le poids ou certains marqueurs de la santé métabolique ont été associée à d'autres symptômes du syndrome. Enfin, aucune étude n'a exploré le changement des symptômes au fil du temps. Ainsi, l'objectif de cette étude était de mieux caractériser les comportements de santé et le profil métabolique associés à des symptômes du SFN dans une cohorte familiale. Un objectif secondaire visait à valider certains aspects du questionnaire qui mesure les symptômes du SFN de façon auto-déclarées, le « night eating questionnaire » (NEQ). L'échantillon de l'étude comprenait des participants de l'étude de cohorte longitudinale de QUALITY (parents biologiques et leur enfant âgé de 8 - 10 ans) évalués à deux reprises : entre 2005 - 2008 et 2008 – 2010. Dans l'ensemble, cette thèse montre que peu de personnes répondent aux critères du SFN, que ces symptômes sont liés à un délai dans certains comportements de santé (activité physique et nutrition) chez les enfants et que certains symptômes sont reliés au profil métabolique et à un gain de poids chez les parents.

Abstract

Night eating syndrome (NES) is characterised by a delayed pattern of energy intake and manifests as either evening hyperphagia and/or nocturnal ingestions of food. Other clinical features of this syndrome have been identified and include morning anorexia, insomnia, strong evening cravings for food, a depressed and/or evening-worsening mood and a belief that one must eat in order to sleep. Research on NES has progressed steadily since the disorder first appeared in the scientific literature in 1955. However, there is much more to be learned on the topic of NES. There is only one published study that examined night eating symptoms in children. There are no published studies that have examined health behaviours associated with these symptoms in children and none that have examined changes in NES symptoms across time. Furthermore, although two studies have demonstrated that nocturnal ingestions of food predicted greater weight gain, they did not explore if weight gain or markers of metabolic health were associated with other NES symptoms. Thus, the objective of this study was to examine the behavioural and metabolic characterisation of night eating symptoms in a family cohort. Furthermore, a secondary objective was to validate certain aspects of the Night Eating Questionnaire (NEQ), a measure of self-reported NES symptoms. The study sample consisted of participants of the longitudinal QUALITY cohort study and included two biological parents and one child aged 8 – 10 years upon recruitment and data were collected at two time points, from 2005 - 2008 and from 2008 – 2010. This thesis demonstrates that few individuals meet criteria for NES.NES symptoms are related to a behavioural delay in children and may be associated with a less healthful diet. Furthermore, certain night eating symptoms are related to weight gain and metabolic health in adults.

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List of Abbreviations

ADHD, attention deficit/hyperactivity disorder
AN, anorexia nervosa
BDI, Beck Depression Inventory
BED, binge eating disorder
BMI, body mass index
BN, bulimia nervosa
CBT, core body temperature
CI, confidence interval
DSM-VI, diagnostic and statistical manual of mental disorders
EH, evening hyperphagia
FAA, feeding anticipatory action
GI, glycemic index
HPA, hypothalamic-pituitary axis
ICC, intra-class correlation coefficient
Ins., insomnia
IQR, inter-quartile range
MA, morning anorexia
MDD, major depressive disorder
MetS, Metabolic syndrome
NEQ, night eating questionnaire
NES, night eating syndrome
NI, night eating or nocturnal ingestions
OR, odds ratios
SCN, suprachiasmatic nucleus
SD, standard deviation (DS déviation standard)
SE, standard error
SERT, serotonin reuptake transporter
SPA, spontaneous physical activity
SSRI, selective serotonin reuptake inhibitor
T2D/DT2, type 2 diabetes/diabète type 2
TEI, total energy intake
WC/CT, waist circumference/ circonférence de la taille

Dedication

This thesis is dedicated to my mother, Julia Cheverie (1949 – 1999)

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First, I would like to thank my PhD director, Dr. Vicky Drapeau for her continuous support, for her precious time that she has devoted to me and to my doctoral training and for her ongoing involvement in my development as a researcher. Dr. Drapeau provided me with every opportunity I needed to succeed. Furthermore, she always encouraged me and nurtured my scientific curiosity. In hindsight, I can see that she balanced her guidance and my independence perfectly. I learned from Dr. Drapeau not only about research and teaching, but also about life in general. She is truly an outstanding mentor.

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Foreword

This thesis is a compilation of studies that are part of a larger prospective study with an aim to examine the natural history and cardiometabolic consequences of obesity in youth – the QUALITY study. The primary investigator of the QUALITY study was the late Dr. Marie Lambert. Since, Dr. Melanie Hendersen has taken the primary investigator position. Dr. Vicky Drapeau initiated the Night Eating Syndrome sub-study. With the help of Dr. Albert Stunkard, Dr. Jennifer Lundgren and Dr. Angelo Tremblay, the team translated the Night Eating Questionnaire, which was thereafter implemented mid-way through the first cycle of data collection. The data, including the parent NEQ data, was collected at two testing sites, Hôpital Ste-Justine in Montreal and Hôpital Laval in Québec City.

My contributions to the studies presented herein are indicated in the following section. For both cycle one and two, I administered the NEQ to all children by telephone. I created and managed all data sets. In all studies except one, I proposed the original objectives, conducted all statistical analyses and drafted the manuscript. For the manuscript entitled “Night eating behaviour and metabolic health in mothers and fathers enrolled in the QUALITY cohort study”, I managed the data set and assisted Dr. Jennifer Lundgren with the statistical analyses and with the drafting of the manuscript. For all manuscripts, I was responsible for the submission process. Dr. Drapeau, Dr. Lundgren and Dr. O’Loughlin were very involved in the early stages of the manuscripts. They provided detailed feedback regarding the objectives, the writing and on the statistical analyses. In addition to playing a key role in the development of the study, the other co-authors were also very involved in the final editing process.

Introduction

The prevalence of obesity in developed nations has been increasing over the past half a century. Currently more than half of the Canadian population is either overweight or obese and the prevalence of obesity among youth has tripled in the past years (Statistics Canada accessed 2014). Obesity sub-types are accompanied by poor metabolic, physical and mental health. Obesity has a multifactorial etiology, which is expressed nicely in the following quote:

> "There are many routes to obesity and people who are gaining weight, carry with them a variable array of susceptibility factors." - Dr. John Blundell, 2005.

Because of the multifactorial etiology of obesity, many different mechanisms and pathways need to be investigated in order to understand this disease. Indeed, studies ranging from molecular biology to behaviours to how the external environmental is organized have been linked to the development of obesity. By studying these different mechanisms, the scientific community is slowly isolating obesity risk factors and identifying obesity sub-types which will aid in the future prevention and treatment of the disease.

The field of psychology has been continuously invested in the study of obesity. Specifically, this field has cultivated the scientific literature on how humans eat and their conscious and unconscious control over eating. The study of eating behaviours and disordered eating has clarified our understanding of obesity, particularly in an obesogenic environment with constant access to energy-dense foods. But this research only portrays one aspect of the problem.

Recently, the temporal organization of eating, that is when we eat, has been identified as a risk factor for obesity. Indeed, the field of chronobiology, that is the sub-discipline of biology that studies how organisms have adapted and respond to light and darkness, have become interested in how this biological system is implicated in energy balance and the development of obesity. Thus, in addition to what and how much we eat, when we eat may too play a role in the development of obesity.

In humans, our feeding cycle is ultimately controlled by the circadian system (*circa* = "about", *dies* = "day"). Indeed, the eating-related physiology of humans has developed to work

in concert with the 24-hour light/dark cycle via a central circadian clock. This central "clock" or the master clock as it is often referred to, is not akin to the analog clock face with which we are very familiar. Rather, the master clock is actually a group of neurons situated in the suprachiasmatic nuclei (SCN) of the anterior hypothalamus in the brain. The circadian clock is an intracellular molecular clock and functions via an approximate 24-h negative feedback loop consisting of clock genes and their protein products. The exact cascade of biological rhythms is explained in greater detail in several reviews (Schulz and Steimer 2009; Bass and Takahashi 2010; Froy 2011) but briefly, consequent to this cascade, these neural signals are eventually transferred to generate rhythmic physiological processes in the periphery (e.g. hormonal secretion, body temperature) and behaviours (e.g. sleep, locomotor activity, feeding) (Huang, Ramsey et al. 2011).

Although the master clock is endogenous (self-sustaining), the period of the cycle is not exactly 24 hours. Consequently, the circadian clock needs to be entrained (synchronized) to external cues to ensure the oscillations will not drift out of phase and to ensure that behaviours are synchronized to the external environment (Froy 2010). The main external cue or zeitgeber (synchronizer), of the circadian system is light. Other zeitgebers include activity, social cues and feeding. For humans, this means sleep during the dark phase and wakefulness and feeding during the light phase. However, these rhythms can be disrupted (chronodisruption)which has a negative impact on the physical and psychological health of the individual involved, such as the development of obesity (Froy 2010; Garaulet, Ordovas et al. 2010) and depression (Germain and Kupfer 2008; Soria and Urretavizcaya 2009).

The Night Eating Syndrome (NES) (Stunkard, Grace et al. 1955c) is a clinical manifestation of what is believed to be a disorder of internal circadian disorganization. Indeed, NES is predominantly characterized by a circadian delay in food intake (excessive evening eating and nocturnal ingestions) coupled with an intact sleep phase. Night eating syndrome is associated with comorbid psychopathology (Lundgren, Allison et al. 2008a; Vinai, Allison et al. 2008b; Vander Wal 2012) and the delayed eating behaviours that characterize this disorder may be an obstacle for body weight control (Andersen, Stunkard et al. 2004a; Gluck, Venti et al. 2008a) and diabetic management (Morse, Ciechanowski et al. 2006). The following chapter will focus on the evolution of the diagnostic criteria, the prevalence of the disorder and at-risk populations, and the physiological and behavioural characterization of this disorder. Moreover, circadian underpinnings and possible treatments will be discussed.

Chapter 1: Literature Review

1.1: Sixty years of research: the night eating syndrome (NES)

NES was first reported by Albert J. Stunkard in 1955 (Stunkard, Grace et al. 1955b) after recognizing specific symptoms associated with one patient's resistance to weight loss. The first clinical definition was described as a triad of morning anorexia, evening hyperphagia (25% of total daily caloric intake after 7pm) and insomnia (at least half of the time). Although the definition of NES has achieved some temporal stability in the components relating to a delayed eating pattern and insomnia, the precise definition and research criteria have varied, yet nonetheless evolved, across time (section 1.2, Figure1.1). This original definition remained mostly unchanged until the late 1990s.

Seven years later, Stunkard *et al*. (1996) changed the extent of evening hyperphagia to > 50% total calories after 7pm to compensate for the under-reporting of self-reported energy intake. Thereafter, an important article published in 1999 by Birketvedt *et al.* (1999a), which aimed to identify the neuroendocrine and behavioural characteristics of NES, added nocturnal ingestions of food (at least once per night) and a minimum symptom duration of at least 3 months to the diagnostic criteria. However, most of the research did not stay within these guidelines and the definition continued to vary.

The change in extent of evening hyperphagia from > 25% to > 50% of total energy intake created confusion and, until the end of 2002, the general definition of > 50% after 7pm remained the norm, regardless of how well the energy intake of the participants was monitored. Several review articles were published during this time - all stating the need for a standardized definition, specifically regarding an agreed-upon definition of evening hyperphagia. This was emphasize in the study by Striegel-Moore *et al*. (2004b) which demonstrated the variability in the prevalence of the disorder as a function of different definitions of evening hyperphagia.

After 2004, more studies began requiring a 3 month minimum duration of symptoms (Yanovski 2004; Allison, Wadden et al. 2006; Friedman, Even et al. 2006; Lundgren, Allison et al. 2006a). This criterion was increasingly important if NES was to be included in the new addition of the Diagnostic and Statistical Manual of Mental Disorders, which was at this time was the DSM-IV (Striegel-Moore, Franko et al. 2006a). Also during this time, the frequency of

nocturnal ingestions was reduced to 1 - 3 times per week (Allison and Stunkard 2005; Striegel-Moore, Dohm et al. 2005).

Even in 2006, the precise definition of evening hyperphagia criterion remained inconsistent across studies. In an attempt to standardize this variable, Allison *et al.* (2006) reported that consuming 25% of the daily total energy intake after the evening meal was atypical and thus, should be used to define evening hyperphagia. Indeed, they demonstrated that obese and overweight individuals with NES reported consuming 34 ± 10% of their total energy intake after the evening meal whereas BMI-matched controls reported consuming 10 ± 6.9%. To reach the 25% value, they added two standard deviations to the mean evening intake of the controls and reached a value of 24%. Again, these recommendations were not followed and the definition of evening hyperphagia continued to vary.

After 2008, NES research gained forward momentum, a large part due to the work of Dr. Stunkard's team. Item Response Theory Analysis identified the most discriminating NES criteria to be onset/maintenance insomnias, nocturnal ingestions and/or evening hyperphagia and demonstrated that morning anorexia was the least discriminating factor (Allison, Engel et al. 2008). The Night Eating Questionnaire (NEQ), a close-ended questionnaire designed to screen for NES, was developed and validated (Allison, Lundgren et al. 2008a). In 2010, NES research diagnostic criteria were standardized after an international scientific meeting (section 1.2, Table 1.1) (Allison, Lundgren et al. 2010b). The Night Eating Syndrome History and Inventory (NESHI), a semi-structured clinical interview used to diagnose NES, was published in the book "Night Eating Syndrome" (Lundgren, Allison et al. 2012). As a result of these research efforts over the past 60 years, NES was included in the Diagnostic and Statistical Manual of Mental Disorders (V) as an Other Specified Feeding or Eating Disorder (American Psychiatric Association 2013a).

This chapter will be separated in two parts: 1) is a review of the literature with a focus on obesity that was published in *Obesity Reviews* in 2012 and 2) a compliment to the published review that includes additional sub-topics and studies published after 2012.

1.2: Night eating syndrome and obesity

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Abstract

The rising prevalence of obesity is a global concern. Eating behaviour and circadian rhythm are proving to be important factors in the etiology of obesity. Increased late-night eating, insomnia, a depressed mood and distress characterize night eating syndrome. It is evident that prevalence is higher among weight-related populations than the general community. The exact relationship between this syndrome and obesity remains unclear. Reasons for the discrepancies found in the literature likely include varying diagnostic criteria and a wide range of study population characteristics. NES does not always lead to weight gain in thus certain individuals may be susceptible to night eating-related weight gain. Weight loss through surgical and behavioural treatments has shown success in diminishing symptoms. The increasing literature associating obesity with circadian imbalances strengthens the link between the Night Eating Syndrome and obesity. Circadian genes may play a role in this syndrome. This review will examine different aspects of obesity in the context of the Night Eating Syndrome.

Résumé

La prévalence croissante de l'obésité est un problème mondial. Les comportements alimentaires et le rythme circadien se révèlent être des facteurs importants dans l'étiologie de l'obésité. Le syndrome de fringale nocturne (SFN) est caractérisé par un délai du cycle alimentaire, l'insomnie, une humeur dépressive et de la détresse psychologique. Bien que la prévalence du SFN est plus élevée chez les populations ayant des problèmes de poids que la communauté en général, la relation exacte entre ce syndrome et l'obésité reste incertaine. Plusieurs raisons peuvent expliquer cette controverse dans la littérature scientifique notamment la variabilité des critères diagnostiques et les différences entre les différentes populations étudiées. On remarque que même si ce syndrome ne conduit pas toujours à un gain de poids, certaines personnes semblent être plus sensibles au gain de poids liés à ce profil d'alimentation. La perte de poids grâce à des traitements chirurgicaux et comportementaux a connu un certain succès dans la diminution des symptômes. La littérature associant l'obésité à un déséquilibre du rythme circadien appuie l'hypothèse du lien entre ce syndrome et l'obésité. En effet, des gènes impliqués dans le rythme circadien pourraient jouer un rôle dans ce syndrome. Cette revue de la littérature portera sur les différents aspects de l'obésité dans le cadre du syndrome de fringale nocturne.

The Night Eating Syndrome

The Night Eating Syndrome (NES) is a clinical representation of a circadian imbalance first reported among obese individuals resistant to weight loss. The first clinical definition was described as a triad of morning anorexia, evening hyperphagia (25% of total energy intake after 7pm) and insomnia (Stunkard, Grace et al. 1955c). Since, the definition of NES has varied over the years. Recently, however, standardized diagnostic criteria were proposed (Table 1.1) (Allison, Lundgren et al. 2010b). The prevalence of NES varies considerably depending on the study population and diagnostic criteria. Nonetheless, trends in the literature emerge revealing certain populations are at greater risk for developing NES. Among these are individuals with weight problems. The purpose of this article is to review the literature regarding NES and obesity and to examine the different links between NES, obesity and the circadian rhythm.

Table 1.1. Proposed diagnostic criteria for NES

NES Diagnostic Criteria
Daily eating pattern of evening/nighttime hyperphagia of one or both of the following:
At least 25% caloric intake after the evening meal
At least two episodes of nocturnal eating per week
Awareness and recall of evening and nocturnal eating episodes
At least three of the following must be present:
Morning anorexia and/or skipped breakfast four or more mornings per week
Presence of a strong urge to eat between dinner and sleep onset and/or during the night
Sleep onset and/or sleep maintenance insomnia four or more nights per week
Presence of a belief that one must eat in order to return to sleep
Mood is frequently depressed and/or mood worsens in the evening
The disorder is associated with significant distress and/or impairment in functioning.
The disordered pattern of eating is maintained for at least 3 months
The disorder is not secondary to substance abuse or dependence, medical disorder, medication or another psychiatric disorder.

†Allison *et al. (Allison, Lundgren et al. 2010b)*; NEQ, night eating questionnaire

Table 1.2. Prevalence of NES in the general population and in obese samples

Population	*n*	BMI (kg/m^2)	Age (yrs)	Prevalence (%)	NES criteria				
					EH (%)	NI	MA	Ins.	NEQ
General Community(Colles, Dixon et al. 2007)	158	24.8 ± 5.1	41.3 ± 13.5	5.7	50		X	X	
General Community(Striegel-Moore, Dohm et al. 2005)	1341	--	20 - 23	1.6	50	X	X		
General Community(Rand, Macgregor et al. 1997)	2097	24.9 ± 4.9	52.8 ± 19.8	1.5	Unspecified		X	X	
General Community(Rand and Kuldau 1986)	232	NW*	35.3 ± 10.0	0.5	Unspecified		X	X	
Weight loss (Calugi, Dalle Grave et al. 2009b)	266	50.7	42.9	10.1	25	X			
Weight loss (Colles, Dixon et al. 2007)	93	32.7 ± 7.3	55.1 ± 12.4	4.3	50		X	X	
Weight loss (Jarosz, Dobal et al. 2007)	88	40.9 ± 7.1	40.6 ± 10.8	18.2	--				X
Weight loss (Adami, Campostano et al. 2002)	166	43.5 ± 9.9	--	15.7	25		X	X	
Weight loss (Gluck, Geliebter et al. 2001)	76	36.7 ± 6.5	43.5 ± 9.5	14.0	50		X	X	
Weight loss (Napolitano, Head et al. 2001)	83	41.1 ± 10.7	47.5 ± 15.6	43.4	50		X		
Weight loss (Stunkard, Berkowitz et al. 1996)	79	35.3 ± 4.0	39.2 ± 8.6	8.9	50		X	X	
Obesity-related clinic (Ceru-Bjork, Andersson et al. 2001)	194	40 ± 5	44 ± 12	6	50		X	X	
Obesity-related clinic (Aronoff, Geliebter et al. 2001)	110	55 ± 14	48 ± 13	50.9	50		X	X	
Obesity-related clinic (Stunkard 1959)	100	--	--	12	25		X	X	
Obesity-related clinic (Stunkard, Grace et al. 1955c)	25	35.6 ± 7.1	35 (18-56)	64	25		X	X	
Bariatric surgery candidates (Colles, Dixon et al. 2008)	129	44.3 ± 6.8	45.2 ± 11.5	17.1	50		X	X	
Bariatric surgery candidates (Colles, Dixon et al. 2007)	180	44.5 ± 6.8	44.8 ± 11.2	19.4	50		X	X	
Bariatric surgery candidates (Allison, Wadden et al. 2006)	215	50.4 ± 8.1	44.4 ± 10.7	1.9	25	X			
Bariatric surgery candidates (Adami, Meneghelli et al. 1999)	63	46.9	37.6 (19 – 61)	7.9	50		X	X	
Bariatric surgery candidates (Powers, Perez et al. 1999)	116	53.4 ± 10.9	39.6 ± 9.3	10	25		X	X	
Bariatric surgery candidates (Kuldau and Rand 1986)	174	--	36 (18 – 59)	15	Unspecified				

Note: EH: evening hyperphagia, % total energy intake after 7pm or evening meal; NI: nocturnal ingestion; MA: morning anorexia; Ins.: insomnia; NEQ: Night Eating Questionnaire; -- Not reported

*NW: normal weight defined by authors as ± 5kg of acceptable weight for height

Year	Author	Definition changes/additions
1955	Stunkard *et al*.	Morning anorexia, evening hyperphagia (≥ 25% of total energy intake after 7pm), insomnia (at least half of the time)
1986	Rand and Kuldau	A distress item (tense/upset) was added
1996	Stunkard *et al.*	Evening hyperphagia: ≥ 50% to compensate for under-reporting of self-reported energy intake
1999	Birketvedt *et al.*	Nocturnal ingestions was formally added, 3 month minimum duration of symptoms required
2005	Allison *et al.*	Frequency of nocturnal ingestions was reduced to 1 – 3 times per week
2006	Allison *et al.*	Lowered evening hyperphagia to ≥ 25% of total energy intake (non-normative eating behaviour)
2008	Allison *et al.*	The Night Eating Questionnaire was validated and a score of ≥ 25 was identified to screen for NES
2008	Allison *et al.*	Item Response Theory Analysis revealed morning anorexia and/or delayed morning meal was a less important diagnostic criterion
2010	Allison *et al.*	Proposed diagnostic criteria

Figure 1.1. Timeline of the night-eating syndrome (NES) definition

NES and Obesity

Compared to the general community, it is evident that NES is more prevalent in populations with weight-related issues (Table 1.2). Although many studies observed a positive relationship between NES and body weight (Kuldau and Rand 1986; Rand and Kuldau 1986; Aronoff, Geliebter et al. 2001; Grilo and Masheb 2004; Lundgren, Allison et al. 2006a; Morse, Ciechanowski et al. 2006; Colles, Dixon et al. 2007; Tholin, Lindroos et al. 2009; Lundgren, Smith et al. 2010), many others have not (Ceru-Bjork, Andersson et al. 2001; Gluck, Geliebter et al. 2001; Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Andersen, Stunkard et al. 2004b; Ruth H. Striegel-Moore 2004; Allison, Wadden et al. 2006; Friedman, Even et al. 2006; Jarosz, Dobal et al. 2007; Gluck, Venti et al. 2008b; Striegel-Moore, Franko et al. 2008; Calugi, Dalle Grave et al. 2009a; Olbrich, Muhlhans et al. 2009).

Possible reasons for this discrepancy include inter-study differences in NES diagnostic criteria, i.e. extent of evening hyperphagia, in methods of expressing NES, i.e. dichotomous

(present/absent) *vs*. continuous (NEQ score), in study population characteristics, i.e. low body weight variability, and the possibility that NES is not related to body weight in every individual. In non-clinical populations with large variations in body size, NES symptom severity measured by the continuous Night Eating Questionnaire (NEQ) score, has been positively associated to BMI in dental patients (Lundgren, Smith et al. 2010) and in a cohort of families with children at risk for obesity (Lundgren, Drapeau et al. 2010). Moreover, several studies examined the prevalence of NES across groups of varying body weight (Kuldau and Rand 1986; Rand, Macgregor et al. 1997; Aronoff, Geliebter et al. 2001; Colles, Dixon et al. 2007; Tholin, Lindroos et al. 2009). Among these studies, all have reported a higher prevalence of NES among overweight/obese groups. In one study, rates seemingly rise exponentially in groups classified as obese and above (Colles, Dixon et al. 2007). An example of this is the high prevalence of NES among bariatric surgery candidates (Table 1.2). There is sufficient evidence to suggest NES is related to obesity, however the nature of this relationship remains unclear and the cross-sectional study designs cannot suggest cause and effect.

NES and weight change

NES and weight gain

NES could theoretically lead to weight gain as a result of excess calories consumed at night. Indeed, studies have shown night eating leads to weight gain (4.3 – 4.5 kg over 3 – 6 years) (Andersen, Stunkard et al. 2004a; Gluck, Venti et al. 2008a) and that obesity onset occurs earlier in life (6.0 - 7.5 yrs) in those with NES (Napolitano, Head et al. 2001; Allison, Crow et al. 2007). Additionally, de Zwaan *et al.* (2006) found NES preceded obesity in 40% of obese night-eaters. Certain individuals or populations may be susceptible to NES-related weight gain. In one population study, obese female night-eaters gained significantly more weight than obese women who did not eat at night (5.2 kg *vs.* 0.9 kg over 6 years) (Andersen, Stunkard et al. 2004a). There was no significant difference in weight gain in male night-eaters and non night-eaters, regardless of body weight. Further evidence supporting the hypothesis that NES contributes to obesity in certain individuals is found in the aforementioned study by de Zwaan *et al*. (2006) where only some normal weight individuals gained weight after NES onset. Reasons for this selective weight gain are mostly unknown and the retrospective nature of this data is a main limitation of this study. Behavioural factors may explain in part the lack of weight gain among some individuals with NES. In a study among normal weight individuals, those with NES were reported to have more days of intense exercise, greater cognitive restraint and a greater

fear of weight gain than those without NES (Lundgren, Allison et al. 2008a). This type of behaviour profile may have successfully controlled their body weight.

Further evidence supporting the implication of NES in elevated body weight is seen in clinical trials of the pharmacological treatment of NES (O'Reardon, Stunkard et al. 2004; O'Reardon, Allison et al. 2006). In one study, NES symptoms significantly improved and were accompanied by a significant reduction in body weight (2.9 kg) in overweight individuals treated with Sertraline for 18 weeks. This reduction in body weight was in the absence of weight loss advice or guidance (O'Reardon, Allison et al. 2006). Taken together, not every individual with NES exhibits weight problems, yet the syndrome may aggravate or maintain obesity, specifically in those susceptible to weight gain

NES and Weight loss

NES may present a challenge to weight loss in some individuals. Accordingly, NES was first described among individuals characterized by unsuccessful weight management, specifically resistance to weight loss (Stunkard, Grace et al. 1955b). Since this initial report, few have focused on NES as a possible obstacle to weight loss. Gluck *et al.* (2001) observed less weight loss (4.4 ± 3.1 kg *vs.* 7.3 ± 3.2 kg, $p = 0.003$) whereas Dalle Grave *et al.*(Dalle Grave, Calugi et al. 2011) reported no difference in weight loss among NES and controls (BMI units: -1.9 ± 2.2 kg/m^2 *vs*. -1.5 ± 1.3, $p = 0.318$) (Dalle Grave, Calugi et al. 2011). Both studies diagnosed full NES criteria by the NEQ; therefore, the discrepancy is likely a result of the type of intervention. The former consisted of a daily 900 kcal liquid diet and a weekly session with a nutritionist where the latter employed mild caloric restriction, a daily exercise regime and group psycho-educational sessions. As stated by the authors of the liquid diet intervention, those with NES did not comply as well with the diet as did the controls, albeit there were no differences in dropout rates between groups. Their night eating became an obstacle to caloric restriction in a weight loss context (Gluck, Geliebter et al. 2001). In this respect, weight loss through behaviour modification may be more appropriate for those with NES. More studies are needed to confirm this.

As previously mentioned, pharmacotherapy treatment of NES, and concurrent reduction in NES symptoms, has lead to weight loss in two clinical trials (O'Reardon, Stunkard et al. 2004; O'Reardon, Allison et al. 2006). In line with this, weight loss has resulted in improvements in NES prevalence and symptoms. In bariatric surgery candidates, many reported a post-surgical reduction in NES prevalence (Hsu, Betancourt et al. 1996; Hsu, Sullivan et al. 1997; Latner,

Wetzler et al. 2004; Colles, Dixon et al. 2008) or NES symptoms (Hsu, Betancourt et al. 1996; Latner, Wetzler et al. 2004; Colles, Dixon et al. 2008). Bariatric surgery is an extreme form of weight modification with results usually unattainable by lifestyle modification alone. Nonetheless, weight loss by traditional methods, such as exercise and caloric restriction, demonstrated similar trends: slight reductions in post-treatment NEQ scores (22.6 ± 5.9 *vs.* 19.8 ± 6.5, $p < 0.001$), a reduction in prevalence of evening hyperphagia and/or nocturnal ingestions (n = 8/35, post/pre) and a reduction in nocturnal eating frequency in 51.4% of the pre-treatment individuals diagnosed with NES (Dalle Grave, Calugi et al. 2011). The reasons for NES symptom improvement in a weight-loss context are unclear. It is possible that improvements in psychological factors as a result of weight loss, e.g. stress, depression, binge eating, could have affected NES symptoms. Nonetheless, weight loss, per say, may affect NES symptoms. In the aforementioned behavioural study (Dalle Grave, Calugi et al. 2011), reductions in NES symptoms persisted despite no improvements in depression and binge eating scores after the intervention. Behaviour modifications, i.e. exercise, are not usually present in surgery-related weight loss and could have also played a role in NES symptom changes. Accordingly, cognitive behavioural therapy coupled with mild caloric restriction has shown promising results in NES symptom improvement with moderate body weight losses (mean loss 3.10 kg) (Allison, Lundgren et al. 2010a). These results were not compared to a control group and are slightly lower than mean weight losses reported by obese individuals for cognitive behavioural treatment of obesity (approximately 10% of initial body weight) (Cooper, Doll et al. 2010), More research is needed in the area of obesity treatment among individuals with NES and the relationship between weight-loss and NES symptom improvement.

Obstacles to weight control in NES

1. Energy intake

NES is inconsistently related to increased energy intake. Among individuals diagnosed with NES, two studies reported increased energy intake (400 – 600 kcal per day) compared to controls (Birketvedt, Florholmen et al. 1999b; Lundgren, Allison et al. 2008b) whereas no difference was observed in others (O'Reardon, Ringel et al. 2004; Allison, Ahima et al. 2005) (Table 1.3). The sample sizes in these studies were small and energy reporting errors and study sample characteristics could explain the inconsistencies, In larger studies, individuals presenting only late-night or nocturnal eating, reported 300 – 500 kcal more per day than those not expressing this eating behaviour (Gluck, Venti et al. 2008a; Striegel-Moore, Franko et al.

2008). Variation in energy intake may, in part, explain why some individuals seem more susceptible to NES-related weight gain. Under these circumstances, increased energy intake would be an obstacle to weight management.

Table 1.3. Total energy intake in NES and controls

	n		Age		BMI		Energy Intake	
	NES	Control	NES	Control	NES	Control	NES	Control
Birketvedt *et al.* (1999a)	10	10	57.3 (12.2)	47.1 (10.7)	28.5 (3.9)	28.2 (4.9)	2928 (1988)*	2322 (920)
Lundgren *et al.* (2008a)	19	22	42.0 (15.5)	36.5 (12.1)	22.5 (1.7)	21.7 (1.9)	2284.5 (620.3)*	1856.2 (545.2)
O'Reardon *et al.* (2004)	46	43	43.3 (9.8)	39.0 (11.0)	34.9 (7.1)	36.7 (6.2)	2314.4 (748)	2420 (748)
Allison *et al.* (2005)	15	14	40.8 (8.7)	38.6 (9.5)	36.1 (7)	38.7 (7)	2958.9	2892.8

All studies measured full NES criteria; * significantly different from controls

2. Sleep disturbances

Individuals with NES usually experience insomnia(Allison, Engel et al. 2008) with reported 1.5 – 4.5 awakenings per night(Birketvedt, Florholmen et al. 1999a; O'Reardon, Ringel et al. 2004; Rogers, Dinges et al. 2006). Regardless of body weight, perceived and measured (polysomnography) sleep quality is impaired in those with NES. For example, studies have found greater sleep latency, reduced sleep duration, and reduced sleep efficiency in persons with NES (Rogers, Dinges et al. 2006; Lundgren, Allison et al. 2008a) without differences in onset/offset sleep times (O'Reardon, Ringel et al. 2004; Rogers, Dinges et al. 2006). Different degrees of NES symptoms may have an additive effect on sleep quality. For example, depressed individuals with NES have presented more sleep troubles than depressed individuals without NES and non-depressed individuals with NES (Striegel-Moore, Franko et al. 2008). Lack of sleep and reduced sleep quality is related to body weight (Cappuccio, Taggart et al. 2008) and may lead to weight gain (Gangwisch, Malaspina et al. 2005; Patel, Malhotra et al. 2006; Chaput, Despres et al. 2008), although the latter has been criticized(Cappuccio, Taggart et al. 2008; Marshall, Glozier et al. 2008; Horne 2011; Magee and Hale 2011; Nielsen, Danielsen et al. 2011). Possible mechanisms of action are an increased opportunity to eat

(Sivak 2006), elevated hunger and appetite sensations (Spiegel, Tasali et al. 2004), reduced daytime physical activity (Schmid, Hallschmid et al. 2009) and altered metabolic control(Spiegel, Tasali et al. 2009). Short sleep and reduced sleep quality in NES may interfere with behaviours and physiology required for weight control. More research is required regarding the relationship between sleep patterns, NES symptoms and body weight.

3. Psychological vulnerability

NES is characterized by a depressed mood with an evening-worsening diurnal pattern(Birketvedt, Florholmen et al. 1999a) (for a complete review on mood and NES, please see Vinai et al.(2008a)). Self-reported depressive symptoms measured by the Beck Depression Inventory (BDI)(Beck, Steer et al. 1988) are more severe in those with NES(Powers, Perez et al. 1999; Pawlow, O'Neil et al. 2003; O'Reardon, Ringel et al. 2004; Allison, Ahima et al. 2005; Allison, Grilo et al. 2005; Allison, Wadden et al. 2006; Allison, Crow et al. 2007; Allison, Grilo et al. 2007; Colles, Dixon et al. 2007; Lundgren, Allison et al. 2008a; Calugi, Dalle Grave et al. 2009b; Striegel-Moore, Rosselli et al. 2009; Dalle Grave, Calugi et al. 2011). The collective mean BDI score of individuals with NES from these studies is 16.2, which equates moderate depression. The true relationship between NES and depression could potentially be masked as severely depressed individuals are frequently excluded from NES studies (O'Reardon, Ringel et al. 2004; Allison, Ahima et al. 2005; Allison, Grilo et al. 2005; Lundgren, Allison et al. 2008a; Allison, Lundgren et al. 2010a; Dalle Grave, Calugi et al. 2011). Major depressive disorder and NES, however, have been shown to have neurophysiological differences suggesting separate syndromes(Lundgren, Amsterdam et al. 2009).

Other psychiatric conditions are also a concern in NES. The prevalence of NES is high among those with psychiatric disorders(Lundgren, Allison et al. 2006a; Lundgren, Rempfer et al. 2010), particularly in those with schizophrenia (Palmese, Degeorge et al. 2011) and bulimia nervosa(Lundgren, Shapiro et al. 2008; Lundgren, McCune et al. 2011). Accordingly, an increased presence of lifetime mood and anxiety disorders have been found in those with NES (de Zwaan, Roerig et al. 2006; Boseck, Engel et al. 2007). This psychological vulnerability could present a challenge to weight control. A recent meta-analysis confirmed depression and obesity are involved in a bi-directional relationship(Luppino, de Wit et al. 2010) and individuals with other psychiatric disorders, such as bipolar disorder(Keck and McElroy 2003) and anxiety disorders (Gariepy, Nitka et al. 2010), are at greater risk for overweight and obesity. Depressive symptoms and other obesity-related correlates of mood and psychiatric disorders, e.g.

medication, reduced physical and social activities, eating behaviour, may interfere with behaviour needed for weight control. Future studies need to identify the extent of the implication of depressive symptoms and behavioural consequences in the development or maintenance of obesity in NES.

NES, circadian rhythm and obesity

Biological rhythm ensures that human behaviour is appropriately synchronized to the external environment. Individuals may experience negative physiological (Ko and Takahashi 2006) and psychological (Barnard and Nolan 2008) effects in a circumstance of circadian disruption. Neuroendocrine and behavioural studies have demonstrated a potential circadian arrhythmicity among those with NES (Birketvedt, Florholmen et al. 1999a; O'Reardon, Ringel et al. 2004; Allison, Ahima et al. 2005; Goel, Stunkard et al. 2009) which manifests in late night eating with a phase delay of 1.5 hours (Goel, Stunkard et al. 2009). The cause for this disruption is unknown. Several physiological systems have been hypothesized to be involved in the mechanistic drive in NES, such as the glucocorticoid and serotonergic systems (Stunkard, Allison et al. 2009b). This circadian arrhythmicity could be one of the links between NES and obesity since emerging findings have linked chronodisruption with increased body weight (Garaulet, Ordovas et al. 2010). The nature of this relationship is complex and the directionality remains unclear (Figure 1.2).

Forcing circadian-regulated behaviour, such as sleeping and eating, to be uncoupled from regular biological rhythm disrupts lipid and glucose metabolism, insulin regulation and may lead to obesity (Garaulet, Ordovas et al. 2010), such as seen in shift workers (Szosland 2010). In one experimental study, insulin sensitivity decreased, leptin secretion was suppressed and the rhythm of cortisol was completely inverted after 10 adults were subjected to a protocol in which their behavioural circadian rhythm (i.e. eating, sleeping) was uncoupled from their biological rhythm (Scheer, Hilton et al. 2009). Similarly, a cross-over study (Qin, Li et al. 2003) revealed late night eating (50% of their total calories in the evening) and delayed sleep (01h30 – 08h30 vs. 22h30 – 06h30) blunted the nocturnal peaks of melatonin and leptin and impaired the glucose and insulin relationship. The hormonal milieu created by behavioural and circadian misalignment and late night eating could lead to reduced energy expenditure (reduced levels of leptin), increased appetite sensations and weight gain.

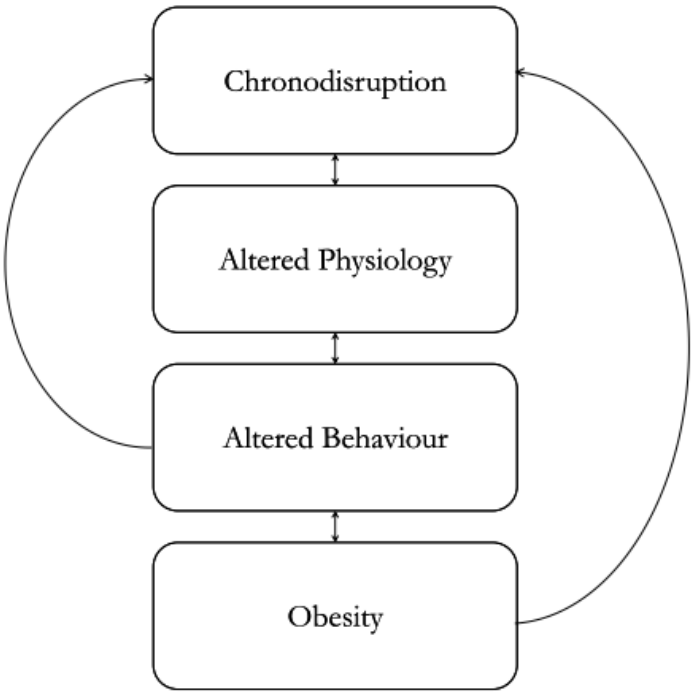

Figure 1.2. A schematic diagram of the relationship between chorondisruption, eating behaviour and the circadian rhythm.

Epidemiological evidence of this is seen in a Swedish cohort where obesity was related to skipping breakfast and lunch and eating at night (OR 1.41, 1.31 and 1.62, respectively) (Berg, Lappas et al. 2009)and in the high prevalence of obesity among shift workers (Antunes, Levandovski et al. 2010). Animal models confirm the association between late-night eating, metabolic arrhythmicity and obesity. Mice fed during the light phase (i.e. during their biological night) had metabolic arrhythmicities (Salgado-Delgado, Angeles-Castellanos et al. 2010) and gained more weight than mice fed the same diet during the dark phase (Arble, Bass et al. 2009; Salgado-Delgado, Angeles-Castellanos et al. 2010). The fact that various appetite-regulating hormones follow an exogenous circadian rhythm may indicate that timing of meals is important for proper metabolic and body weight regulation. In “late sleepers”, calories consumed after 8pm was an independent predictor of BMI after controlling for age, sleep duration, and timing of sleep onset and late night eating was a stronger predictor of obesity than total energy intake(Baron, Reid et al. 2011). More human research is needed to verify if the timing of food

intake promotes circadian arrhythmicity and weight gain independent from energy expenditure and absolute energy intake.

Obesity in itself may alter the circadian system. High fat diet-induced obesity in mice has been shown to alter circadian rhythm gene expression both in the periphery, i.e. liver and kidney (Hsieh, Yang et al. 2010) and centrally, in the nucleus of the solitary track (Kaneko, Yamada et al. 2009). Additionally, mice fed a high-fat diet were less able to readjust after simulated jet-lag, suggesting altered circadian synchronization to light (Mendoza, Pévet et al. 2008). An obese state may actually exacerbate circadian imbalances in humans. Along these lines, individuals suffering from obesity and diabetes have dampened amplitudes of rhythms (Huang, Ramsey et al. 2011). Taken together, circadian behaviour misalignment and obesity may be involved in a cyclical relationship driven by the behaviour and circadian genetic variations (Figure 1.2).

Genetics – the missing link between NES and obesity?

In addition to behavioural chronodisruption being related to altered metabolism (Garaulet, Ordovas et al. 2010), circadian gene variations have been associated with negative health outcomes. Behaviour associated with circadian and serotonergic gene variations are similar in nature to the behaviour profile observed with NES in that they involve meal timing and eating behaviour, sleep, mood and obesity (Caspi, Sugden et al. 2003; Turek, Joshu et al. 2005; Mendlewicz 2009; Yang, Liu et al. 2009; Garaulet, Corbalan-Tutau et al. 2010; Garaulet, Sanchez-Moreno et al. 2011; Huang, Ramsey et al. 2011).

The Clock gene may be of high importance as it synchronizes internal metabolism with the environment and plays a role in energy balance (Garaulet and Madrid 2010). In humans, Clock gene mutation carriers (the minor C allele) were resistant to weight loss after a 12-14 week intervention, had shorter sleep durations, had higher ghrelin concentrations, reported a delayed breakfast time with a preference for the evening (Garaulet, Sanchez-Moreno et al. 2011). Likewise, individuals with polymorphisms in PERIOD2, a clock-related gene, had higher abdominal obesity, had more extreme snacking, increased stress with dieting, ate more when bored and skipped breakfast more than those without the polymorphisms (Garaulet, Corbalan-Tutau et al. 2010). Also, in addition to the aforementioned Clock gene, other circadian genes, such as BMAL1, and TIMELESS, have been associated with mood disorders, including mood worsening in the evening and insomnia (Mendlewicz 2009). Animal studies have found similar

results. Mice with Clock and *mPer2* gene mutations were obese, hyperphagic with increased feeding in the light phase (Turek, Joshu et al. 2005; Yang, Liu et al. 2009; Huang, Ramsey et al. 2011), had metabolic abnormalities, and were more susceptible to weight gain on a high-fat diet (Turek, Joshu et al. 2005; Yang, Liu et al. 2009; Huang, Ramsey et al. 2011).

Genes implicated in the serotonergic system may also be of interest. Serotonin is implicated in the circadian rhythm and is a precursor to melatonin. In the absence of serotonin, one case study reported abnormal sleep-wake rhythm, mild hypersomnia, and conscious nocturnal awakenings with ingestion (Leu-Semenescu, Arnulf et al. 2010). Increased serotonin reuptake receptor (SERT) binding was found in the mid-brain and temporal lobe of individuals with NES (Lundgren, Newberg et al. 2008). The short allele of the 5-HTTLPR gene has been associated with depression following a stressful life event (Caspi, Sugden et al. 2003) and being more "biologically reactive to stress" (Gotlib, Joormann et al. 2008). There is evidence that NES may involve a genetic component (Lundgren, Allison et al. 2006b; Tholin, Lindroos et al. 2009; Root, Thornton et al. 2010) but to date no studies have focused on specific genes involved in this syndrome. Genetic studies would greatly enhance our understanding of NES.

Future Directions

To date, the literature on NES and body weight is limited and the majority of studies are small in sample size and cross-sectional in nature, often with retrospective data. Prospective studies are lacking and longitudinal findings would greatly clarify the relationship between NES and weight change. Moreover, risk factors for NES-related weight gain need to be identified. Future studies should also focus on child NES symptomology. Studying NES in this population would give insight on the onset, the progression and possible risk factors for developing NES. Additionally, non-traditional treatments of NES need to be considered. Feeding (Froy, Chapnik et al. 2008; Froy 2010), and to a lesser extent, exercise(Atkinson, Edwards et al. 2007) are known zeitgebers (synchronizers) and thus could be a focus for circadian-resetting, if indeed NES is a problem of the circadian rhythm, and research should focus on identifying factors implicated in behavioural weight loss treatment which may improve night-eating symptoms. Finally, genetic studies will greatly enhance our understanding of night eating, the systems involved and future directions for treatment.

Conclusions

The prevalence of NES is higher in weight-related populations than in the general community. NES may lead to weight gain in vulnerable individuals, specifically among those susceptible to obesity. NES and its associated symptoms, such as nocturnal eating, depression and sleep-related problems, likely present a challenge to weight control and, in this sense, may exacerbate weight problems. The fact that weight-reduction interventions improve NES symptomatology is perplexing and implies increased weight may be involved in night eating symptoms. Thus, NES and obesity may be involved in a cyclic relationship. Behavioural weight loss interventions may be more successful in controlling weight among those with NES. The scientific literature is establishing concrete evidence connecting obesity and chronodisruption but questions remain regarding NES and obesity. Circadian genes may be involved in the biological drive in NES but, to date, no studies have examined the implication of gene variations among those with NES. More research is needed regarding the onset and symptomology progression of NES, NES-related weight gain and the extent of circadian disruption in individuals suffering from this syndrome.

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1.3: The prevalence of NES – a more comprehensive view

As in the literature specific to NES and obesity, the prevalence of NES varies considerably in the overall literature, partly because of the heterogeneity of the study populations. Indeed, since 1990 NES has been examined in target samples of the general population and in populations characterized by obesity, by different psychopathologies, by sleep-related and metabolic diseases (Table 1.4).

The heterogeneity in study populations poses a challenge when synthesizing the prevalence of NES but has also permitted trends in NES prevalence to emerge from the literature. Indeed, these date demonstrate that certain populations are at greater risk for developing NES than others. As depicted in Tables 1.2 and 1.4, the prevalence of NES in the general community ranges from 0.5% to 5.7% (Rand and Kuldau 1986; Rand, Macgregor et al. 1997; Striegel-Moore, Dohm et al. 2005; Colles, Dixon et al. 2007; Runfola, Allison et al. 2014) and increases in populations with weight or psychological problems as well as in populations with eating disorders. Populations characterized by other health problems related to obesity or to sleep, do not seem to be exceedingly affected by NES.

The variability of the prevalence of NES is also attributable to the inconsistency in the definition of NES. Studies have illustrated this by comparing the NES prevalence across different NES definitions. As expected, the broader the NES definition, the higher the prevalence. This is illustrated in two studies (Figure 1.3). Epidemiological data revealed that 32% of the general population reported consuming > 25% of total energy intake after 7pm based on 24-hour food recalls compared to 11% who reported consuming > 50% (Striegel-Moore, Franko et al. 2006b). Similarly, using the NEQ, 7.2% of 1636 young adults (18 – 26 yrs) reported consuming 25% of their total energy intake after the evening meal whereas this decreased to 1.8% when the extent of evening hyperphagia was increased to 50% (Runfola, Allison et al. 2014). From the same study, 2.4% reported nocturnal ingestions, 0.5% reported a NEQ score of > 30 and 2% reported a NEQ score of > 25. These studies not only show how sensitive the NES prevalence is to small changes in the diagnostic criteria, but they also illustrate how widespread isolated night eating behaviours can be.

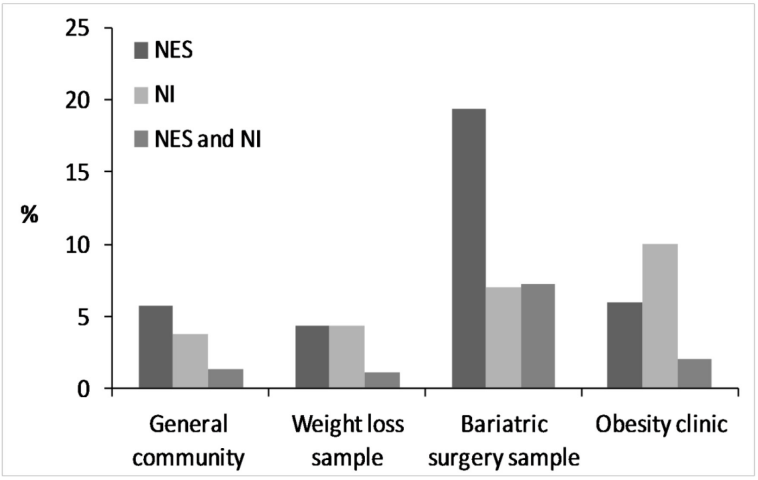

Figure 1.3. The prevalence of Night Eating Syndrome (NES) and night eating behaviour. Colles et al. (2007) investigated NES in a sample of the general community, a weight loss sample and a bariatric surgery sample. Ceru-Bjork et al.(2001) investigated different definitions in a sample from an obesity clinic. For both studies, NES was defined as > 50% calories after 7pm, morning anorexia and insomnia and nocturnal ingestions (NI) was defined as awakening from sleep to eat.

These studies demonstrate that asymptomatic evening hyperphagia is prevalent in otherwise healthy young adults whereas nocturnal ingestions is prevalent in treatment-seeking obese individuals, suggesting that isolated nocturnal ingestions may be more indicative of problematic eating than evening hyperphagia. Accordingly, nocturnal ingestions with and without NES was associated with worse health than NES alone (Ceru-Bjork, Andersson et al. 2001; Colles, Dixon et al. 2007). Furthermore, in a validation study the authors concluded that evening hyperphagia occurred frequently and in isolation from other NES criteria whereas nocturnal ingestions occurred with evening hyperphagia more than half of the time (Allison, Lundgren et al. 2014). The authors stated that “the presence of nocturnal ingestions was more coherent with other symptoms” and symptom coherence is an important aspect of psychiatric disorders (Allison, Lundgren et al. 2014).

These studies demonstrate the importance of standardizing the NES criteria in the literature. Also, these studies also demonstrate the necessity of investigating this syndrome in different populations. Indeed, the recent literature has explored NES in a variety of populations at risk for physical and mental health problems.

Table 1.4. Prevalence of NES in studies measuring at least 3 criteria (eating, sleep, mood, stress) or using a cut-off score of the NEQ

Population	*n*	BMI (kg/m^2)	Age (yrs)	Prevalence (%)
General Community				
Colles et al. (2007)	158	24.8 ± 5.1	41.3 ± 13.5	5.7
Striegel-Moore et al. (2005)	1341	--	20 - 23	1.6
Rand et al. (1997)	2097	24.9 ± 4.9	52.8 ± 19.8	1.5
Rand et al. (1986)	232	NW*	35.3 ± 10.0	0.5
Nolan et al., (2012)	246	23.3 ± 4.1	18.8 ± 1.4	5.7
Fischer et al. (2012)	1514		21.7 ± 2.3	1.3
Runfola et al., (2014)	1636	23.3 ± 3.4	20.9 ± 1.7	4.2
Lundgren et al., (2012) (women)	395	29.2 ± 6.4	40.7	0.5
Lundgren et al., (2012) (men)	395	30.5 ± 5.3	42.8	0.3
Weight loss				
Calugi et al. (2009b)	266	50.7	42.9	10.1
Colles et al. (2007)	93	32.7 ± 7.3	55.1 ± 12.4	4.3
Jaroz et al. (2007)	88	40.9 ± 7.1	40.6 ± 10.8	18.2
Adami et al. (2002)	166	43.5 ± 9.9	--	15.7
Gluck et al. (2001)	76	36.7 ± 6.5	43.5 ± 9.5	14.0
Napolitano et al. (2001)	83	41.1 ± 10.7	47.5 ± 15.6	43.4
Stunkard et al. (1996)	79	35.3 ± 4.0	39.2 ± 8.6	8.9
Obesity Clinic				
Ceru-Bjork et al. (2001)	194	40 ± 5	44 ± 12	6
Aronoff et al. (2001)	110	55 ± 14	48 ± 13	50.9
Stunkard (1959)	100	--	--	12
Stunkard et al. (1955c)	25	35.6 ± 7.1	35 (18-56)	64
Cleator et al. (2013)	144	47 ± 9.5	44.6 ± 12.1	2.8**
Dalle Grave et al. (2013)	586	47.7 ± 9.8	38.2 ± 6.5	3.1**
Bariatric Candidates				
Colles et al. (2008)	129	44.3 ± 6.8	45.2 ± 11.5	17.1
Colles et al. (2007)	180	44.5 ± 6.8	44.8 ± 11.2	19.4
(only night eating) Allison et al. (2006)	215	50.4 ± 8.1	44.4 ± 10.7	1.9
Adami et al. (1999)	63	46.9	37.6 (19 – 61)	7.9
Powers et al. (1999)	116	53.4 ± 10.9	39.6 ± 9.3	10
Kuldau and Rand (1986)	174	--	36 (18 – 59)	15

Table 1.4. continued

Population	*n*	BMI (kg/m^2)	Age (yrs)	Prevalence (%)
Psychiatric populations/eating disorders				
Lundgren et al. (2009)	68	37.2 ± 8.1	43.9 ± 10.4	25
Lundgren et al. (2006a)	205	29.1 ± 7.5	40.8 ± 12.7	12.3
Lundgren et al. (2011)	68	21.1 ± 8.5	29.8 ± 11.2	25
Kucukgoncu et al. (2014b)	155	26.8	35.8	21.3
Other				
Obstructive Sleep Apnea(Olbrich, Muhlhans et al. 2009)	81	34.4 ± 6.6	53.7 ± 10.8	8.6*
Type II diabetes (Allison, Crow et al. 2007)	845	36.4 ± 6.2	59.9 ± 6.9	3.8
Restless Leg Syndrome (Provini, Antelmi et al. 2009)	100	26.3 ± 5.0	63.0 ± 13.0	0.03
Restless Leg Syndrome (Antelmi, Vinai et al. 2014)	120	26.1 ± 4.1	63.8± 11.5	17
Obese insomniacs (Vinai, Cardetti et al. 2014)	98	39.5	48.4	8.2
Sleep clinic (Spaggiari, Granella et al. 1994)	200	-	-	5

NEQ: Night Eating Questionnaire, * screened positive NEQ > 25, **NEQ > 30; †night eating only

1.4: Eating patterns and behaviours in NES

NES is a disorder characterized mainly by atypical eating patterns. Specifically, NES patients show a temporal delay in energy intake that manifests as two main eating behaviours: evening hyperphagia (excessive evening eating) and nocturnal ingestions of food (awakenings from sleep to eat). Often, NES patients also report morning anorexia (reduced morning appetite and/or reduced morning food consumption), but this is not a core criterion of the disorder.

Temporal delay in food intake

The magnitude of the temporal delay has been quantified in several studies. Using cosinor analysis, NES patients had a 1.5 hour delay in their feeding rhythm compared to controls (Goel, Stunkard et al. 2009). Descriptive analyses demonstrated that non-obese NES patients attained 75% of their total energy intake 4 hours later than matched controls (Lundgren, Allison et al. 2008b). The cumulative intake in energy intake in the NES patients steadied off approximately 4 - 5 hours after the plateau in energy intake observed in the controls (O'Reardon, Ringel et al. 2004).

Extent of evening hyperphagia

The cut-off for the presence of evening hyperphagia is currently no lower than 25% of the day's energy intake; however, NES patients can consume much more than this amount after the evening meal. Non-obese NES patients consumed 32.2 ± 18.6 % of their total energy intake after supper but before bedtime (Lundgren, Allison et al. 2008a). In another, NES patients consumed 34.6% ± 10 (O'Reardon, Ringel et al. 2004). For comparison, controls from these studies reported consuming approximately 10% of total energy intake during this time. These studies clearly demonstrate that consuming at least 25% of the total daily energy intake after the evening meal is an atypical eating pattern.

Frequency and content of nocturnal ingestions

Investigators have also examined the frequency and energy content of nocturnal ingestions in NES patients. NES patients experienced 1.2 – 8 nocturnal ingestions per night (Spaggiari, Granella et al. 1994; Birketvedt, Florholmen et al. 1999a; O'Reardon, Ringel et al. 2004; Lundgren, Allison et al. 2008a). Moreover, it was very uncommon for individuals without NES to report this behaviour (1 control in all studies). The energy content per nocturnal ingestion ranged from 270 to 290 kcal which equated to 500 – 2000 kcal per night (Spaggiari, Granella et al. 1994; Greeno, Wing et al. 1995; Birketvedt, Florholmen et al. 1999a; Lundgren, Allison et al. 2008a). NES patients frequently chose high carbohydrate (CHO) foods as their nocturnal meals (Spaggiari, Granella et al. 1994; Birketvedt, Florholmen et al. 1999a)(Chapter 6); in one study, the nocturnal meal was 70.3% CHO whereas their daytime meals were 46.6% CHO (Birketvedt, Florholmen et al. 1999a). Indeed, Spaggiari et al., reported that one woman's nocturnal meal consisted of six slices of cake, two fruits, a piece of cheese and two cups of milk (Spaggiari, Granella et al. 1994). However, few studies have examined the nutritional qualities of foods eaten at night or the habitual diet of night eaters.

NES patients – evening grazers?

In addition to a delayed pattern of intake, NES patients were described to be "evening grazers" (Stunkard, Allison et al. 2009a). Grazing has been defined as "eating a considerable amount of food over a long period of time" (Striegel-Moore, Franko et al. 2006b). Boston et al., used Gaussian models to describe the temporal eating patterns of NES patients. They found that NES patients' meals are less discrete and more energetic as the evening progresses (Boston, Moate et al. 2008) (Figure 1.4). Specifically, they demonstrated that NES patients have reduced peak rates of consumption for breakfast and lunch but much greater rates for supper.

Moreover, the timing of the peak rate occurred much later for supper in NES compared to controls, which supports the delayed pattern of intake. Furthermore, the meal spread was generally longer for NES, particularly for supper, and meals tended to overlap. This is illustrated in Figure 1.4. This eating style is supported the results of Goel et al., where the amplitude of the feeding rhythms of NES patients was attenuated by 30% (Goel, Stunkard et al. 2009). In addition, two studies reported NES participants having more daily eating episodes than controls (Birketvedt, Florholmen et al. 1999b; Lundgren, Allison et al. 2008b) and NES patients were also characterized by irregular and chaotic daytime eating patterns (Rand and Kuldau 1986; Cleator, Judd et al. 2013).

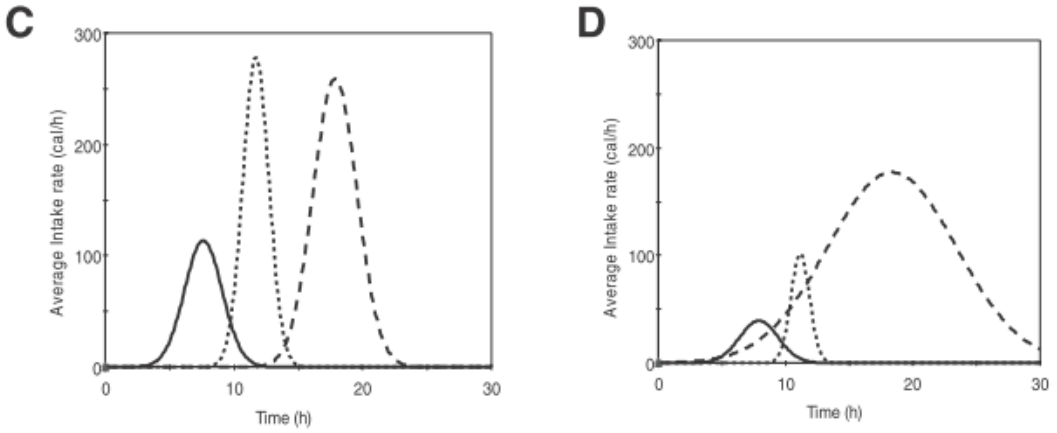

Figure 1.4. The Boston Model that describes the temporal eating patterns between controls (C) and individuals with NES (D). *Taken from (Boston, Moate et al. 2008).*

Total energy intake in NES patients and controls

As described in the manuscript in section 1.2, despite the atypical eating patterns associated with this disorder, NES patients do not consistently report greater daily energy intake (Table 1.3, Figure 1.5). In two studies, NES patients consumed 400 – 600 kcal more per day than controls (Birketvedt, Florholmen et al. 1999b; Lundgren, Allison et al. 2008b). In contrast, there were no differences in total energy intake between NES patients and controls in others (O'Reardon, Ringel et al. 2004; Allison, Ahima et al. 2005). The inconsistencies could be attributed to study populations. For example, NES patients consumed more energy than controls in the non-obese samples only. It is possible that differences in energy intake are no longer evident when compared to obese controls. The inconsistencies could also be attributed

to the presence of morning anorexia. Although two studies did not require this criterion to diagnose NES (Allison, Ahima et al. 2005; Lundgren, Allison et al. 2008a), it is common in NES and variability in morning energy intake would influence total daily energy intake. Accordingly, obese in-patients who ate at night (night-eaters) and who consumed similar amounts of energy in the morning as non night-eaters, ended up consuming 300 – 500 kcal more during the day than non night-eaters (Gluck, Venti et al. 2008a).

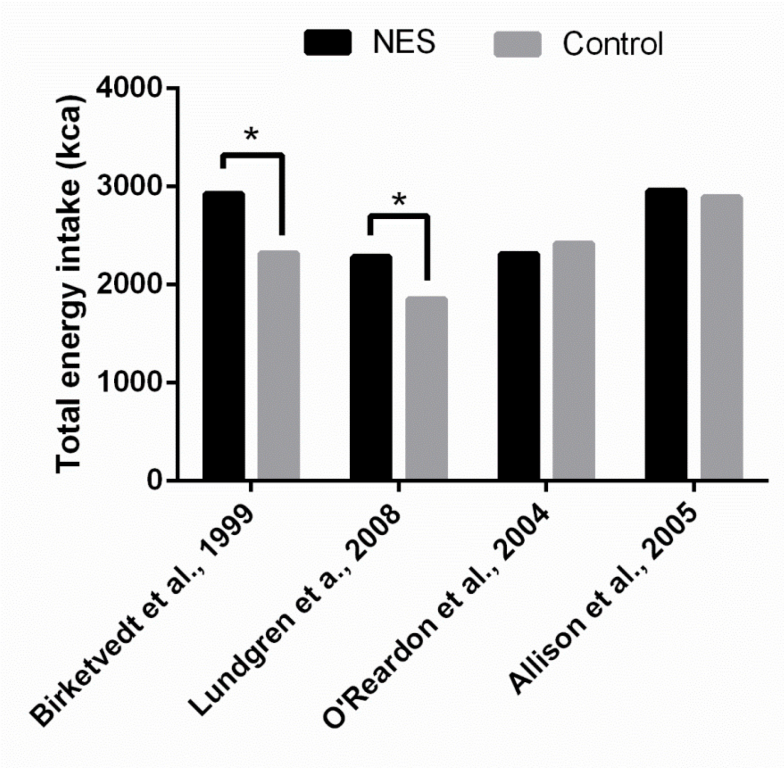

Figure 1.5. Total energy intake between NES and controls. Birketvedt et al., (Birketvedt, Florholmen et al. 1999a) overweight sample (10 NES, 10 controls); Lundgren et al., non-obese NES sample (19 NES, 22 controls); O'Reardon et al., obese sample (46 NES, 43 controls) and Allison et al., obese sample (15 NES, 14 controls).

NES and Eating behaviour traits

Because of the altered eating patterns in NES, researchers are interested in investigating the eating behaviour traits in patients with the disorder. In the NES literature, the Three Factor Eating Questionnaire (TFEQ) (Stunkard and Messick 1985) was the most common

questionnaires to assess eating behaviour traits. This questionnaire measures cognitive restraint, disinhibition and susceptibility to hunger. The summary of eating behaviour traits in NES and controls are illustrated in Figures 1.6, 1.7 and 1.8.

Restraint

Dietary restraint is the voluntary restriction of energy intake with the aim of either weight loss or weight control. Unlike more classical eating disorders (anorexia and bulimia nervosa), NES patients generally report similar levels of dietary restraint as controls (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Colles, Dixon et al. 2007; Gluck, Venti et al. 2008b; Striegel-Moore, Franko et al. 2009). One exception using the TFEQ was in normal weight NES patients who reported higher cognitive restraint scores than the normal weight controls. Similarly, in a large sample of mostly healthy weight individuals (74%), those with NES reported higher restraint scores measured by the EDE than those without (2.2 vs. 1.4) (Runfola, Allison et al. 2014). Dietary restraint does not seem to be characteristic of NES patients but it may play a role in weight control in healthy weight individuals with NES (Figure 1.6).

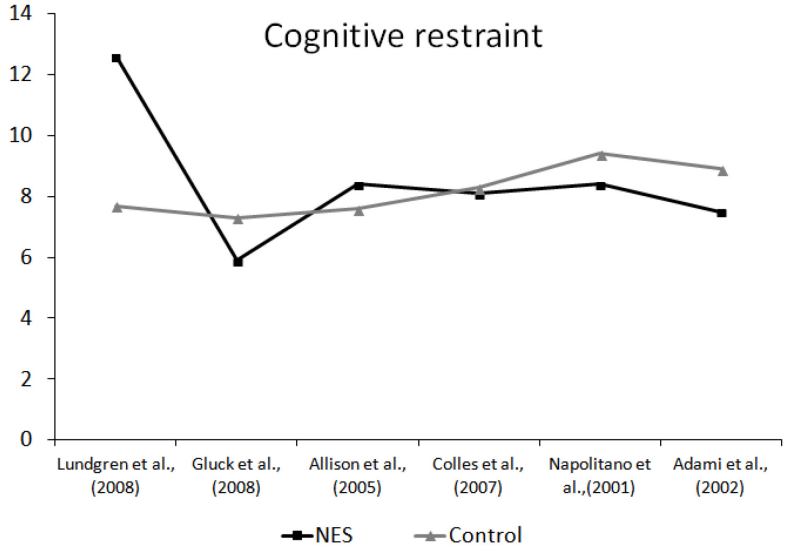

Figure 1.6. Mean cognitive restraint scores for NES patients and controls. Studies are ordered by mean BMI of the sample (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Allison, Ahima et al. 2005; Colles, Dixon et al. 2007; Gluck, Venti et al. 2008a; Lundgren, Allison et al. 2008a).

Disinhibition

Disinhibition has been examined in NES patients. Disinhibited eating is the inhibition of dietary restraint although it can be present in the absence of dietary restraint and measures overeating tendencies related to emotions, situations and habits. As such, it has also been described as opportunistic eating (Bryant, King et al. 2008). NES patients reported higher disinhibition scores compared to controls in some studies (Allison, Grilo et al. 2005; Lundgren, Allison et al. 2008b) but not others (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Colles, Dixon et al. 2007; Gluck, Venti et al. 2008b). Differences in disinhibition scores between NES patients and controls seem to be more apparent in samples of lower mean BMI, as illustrated in Figure 1.7.

It is possible that the TFEQ disinhibition scale may not capture the temporal nature of overeating in NES patients. Perhaps exploring the disinhibition subscales of situational, emotional and habitual overeating would be more informative (Bond, McDowell et al. 2001); however, no studies have examined these subscales in NES patients. Emotional eating has been examined using other tools of assessment (see the following section on emotional eating).

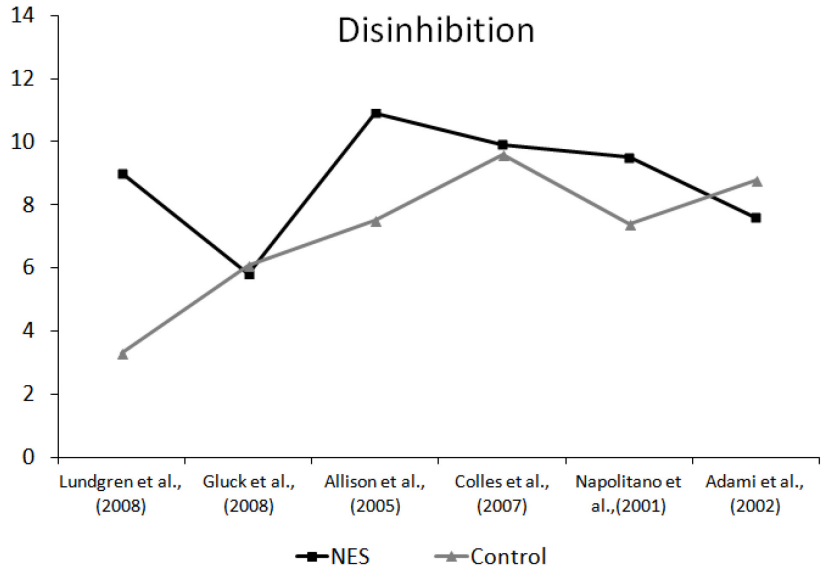

Figure 1.7. Mean disinhibition scores for NES and controls. Studies are ordered by mean BMI of the sample (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Allison, Ahima et al. 2005; Colles, Dixon et al. 2007; Gluck, Venti et al. 2008a; Lundgren, Allison et al. 2008a).

Hunger

The TFEQ also measures susceptibility to hunger. The items of this scale reflect the individuals' perception of hunger and a high score indicates the individual always feels hungry (and consequently does not feel full) and that hunger is driven more by hedonic hunger, i.e., "frequent thoughts, feelings and urges about food in the absence of any short- or long-term energy deficit" (Lowe and Butryn 2007) than homeostatic hunger, i.e., hunger consequent to prolonged energy deprivation. Although several items relate to temporal hunger sensations ("I sometimes get very hungry late in the evening or at night" and "At certain times of the day, I get hungry because I have gotten used to eating then"), again, there were no obvious consistencies in the literature regarding the relationship between NES and hunger susceptibility (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Allison, Grilo et al. 2005; Jarosz, Dobal et al. 2007; Gluck, Venti et al. 2008b; Lundgren, Allison et al. 2008b). Similar to disinhibition, the graph illustrates that body weight may also moderate the relationship; the hunger scores of NES patients seem to differ more in samples with lower mean body weights (Figure 1.8).

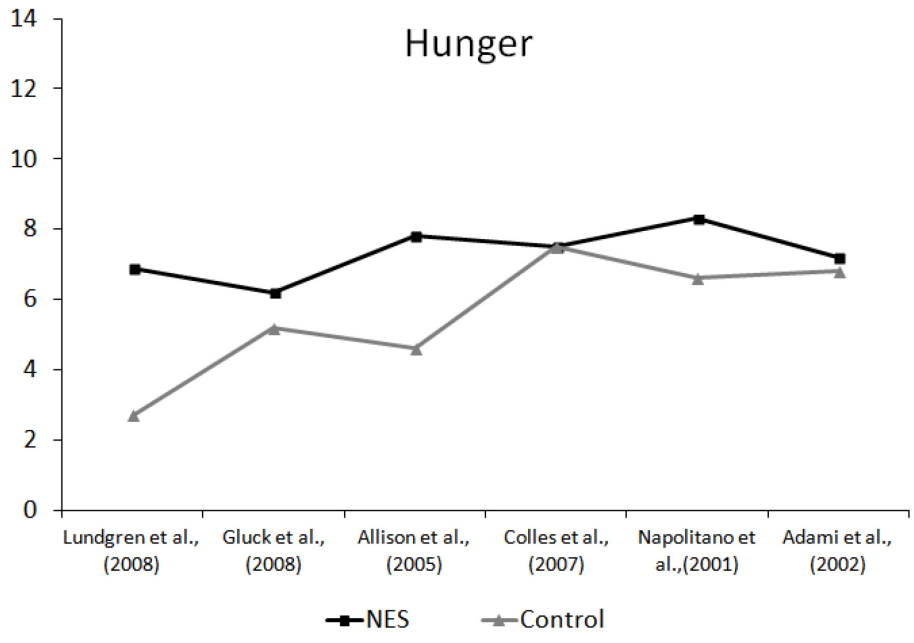

Figure 1.8. Mean sample hunger scores for NES patients and controls. Studies are ordered by the mean BMI of the sample (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Allison, Ahima et al. 2005; Colles, Dixon et al. 2007; Gluck, Venti et al. 2008a; Lundgren, Allison et al. 2008a).

Emotional eating

Emotional eating is eating in response to negative affect and is measured by tools such as the Dutch Eating Behaviour Questionnaire (DEBQ), the Mood Eating Scale (Jackson and Hawkins 1980) and the Emotional Eating Scale (ESS) (van Strien 1986; Arnow, Kenardy et al. 1995). In university students, those who met NES criteria had higher emotional eating scores on the DEBQ than those who met some and no criteria (Nolan and Geliebter 2012) and NES symptom severity was positively correlated with emotional eating (stress-related eating) (Meule, Allison et al. 2014). Furthermore, Diabetics with NES were characterized by eating in relation to emotional triggers, specifically eating in relation to loneliness and worry (Morse, Ciechanowski et al. 2006). NES patients report perceived stress and depression and these emotions could trigger emotional eating in NES.

NES and appetite control

NES patients experience a unique temporal appetite pattern. Consequently, several researchers examined perceived appetite, hunger and fullness, at different times of the day in NES patients (as opposed to more general eating behaviour traits, such as susceptibility to hunger, as discussed above). In one study, prior to a daytime *ad libitum* liquid test meal, NES patients reported less hunger and greater fullness compared to controls (Gluck, Geliebter et al. 2001). Despite these differences, NES patients and controls consumed similar volumes of the liquid meal (979 ± 417 g vs. 859 ± 459 g, respectively). Furthermore, post-meal hunger and fullness ratings were not different between the two groups. Taken together, the change in hunger for a given volume of food was less in the NES patients compared to the controls during a daytime meal. This suggests that NES patients may have weaker daytime satiety signals.

In a second study, investigators used Ecological Momentary Assessment to assess hunger states in NES patients (Boseck, Engel et al. 2007). NES patients reported higher hunger sensations at night compared to controls; however, they also reported higher morning hunger levels that contrasts the morning anorexia commonly reported by NES patients. In a final study, prior to bariatric surgery, daytime hunger was lower in NES versus controls. Post-surgery, hunger decreased for all patients but it decreased less in NES patients (Morrow, Gluck et al. 2008).

Conclusions regarding the eating patterns and behaviours of NES

Taken together, NES is characterized by a circadian delay in energy intake that manifests as evening hyperphagia and/or nocturnal ingestions. Moreover, the eating patterns associated with NES can be described as “evening grazers”, with less discrete meals and a considerable amount of energy consumed over the evening. NES is sometimes accompanied by greater total energy intake but total daily differences may not exist because of the reduced morning energy intake by some individuals with the disorder or the relationship may be moderated by body weight. Eating behaviour traits (restraint, disinhibition and hunger) may not be a characteristic of NES. This could be that the TFEQ does not capture the temporal nature of the eating pathology in NES patients. Appetite states may be more informative and studies demonstrated an altered appetite response to a meal and differences in daytime and evening appetite states in NES and controls.

1.5: The course of NES

It is important to understand the course of a disorder in order to determine the health impact of the associated symptoms. As mentioned, there are very few studies that have examined NES longitudinally and none, to our knowledge, that have done so prospectively. NES seems to be a chronic disorder and most patients report that their symptoms persist > 5 years (de Zwaan, Roerig et al. 2006). The mean reported duration of NES symptomatology is approximately 10 years (Spaggiari, Granella et al. 1994; Marshall, Allison et al. 2004; Cleator, Judd et al. 2013), but it may be longer in treatment-seeking obese patients (mean 17.4 yrs) (Marshall, Allison et al. 2004).

It seems that the onset of NES symptomatology occurs during adulthood. In two studies, the mean onset of NES symptomatology was reported to occur during mid-adulthood (33 .7yrs, range 18 – 52 yrs (Spaggiari, Granella et al. 1994) and 33.2 yrs (de Zwaan, Roerig et al. 2006)). However, it is not uncommon for the symptoms to onset during childhood. In 30 severely obese NES patients, 6 reported their symptoms onset during childhood and 6 reported they onset during adolescence (Cleator, Judd et al. 2013). Likewise, 20% of another sample (*n* = 106) reported their symptoms onset before 19 years of age (de Zwaan, Roerig et al. 2006).

Only two studies have examined night eating behaviours in a child population. In a large sample (*n* = 1979), Lamerz et al., (Lamerz, Kuepper-Nybelen et al. 2005a) reported that 1.1% of parents reported that their child ate at night. Furthermore, our group has shown that no children (8 – 10 yrs) met all NES screening criteria (Lundgren, Drapeau et al. 2012).

These studies indicate that certain night eating behaviours can onset during childhood but that for the majority of the sufferers, the syndrome likely onsets much later. These data are sparse and are mostly based on retrospective reports. Thus, more prospective studies are needed to quantitatively assessment of the reproducibility, i.e., the stability, of NES symptoms are needed to fully understand the course of the disorder.

1.6: Potential health consequences of NES

As depicted in sections 1.2 and 1.3, NES is more prevalent in populations with increased body weight or in populations with other psychopathologies. However, this by no means suggests NES leads to these health problems and in truth, the potential health consequences of NES are currently uncertain. As described in section 1.2, uncoupled feeding and sleeping rhythms may negatively impact health, e.g., digestion, metabolic and cardiac problems which could theoretically occur in NES. However, the identification of NES-associated health consequences necessitates longitudinal studies that are currently lacking in the NES literature. For now, the health "consequences" are predominantly adverse health associations that based on cross-sectional studies or clinical case studies. These associations indicate that NES is associated with aspects of ill health, but to date, there are no longitudinal epidemiological data of NES.

Chronodisruption

Food and nutrients are non-photic zeitgebers, which means that food can synchronize the circadian system. Indeed, feeding-induced changes in metabolic parameters can alter the rhythm of clock gene expression in the peripheral tissues (kidneys, lungs, adipose tissue etc.) which occurs without impacting the SCN, the central pacemaker in the brain (Mendoza 2007; Vollmers, Gill et al. 2009). As an extreme example, if food were only available at night, the peripheral clocks would independently regulate certain bodily rhythms and, under specific

circumstances, it would be possible that the peripheral clocks could override the SCN and become responsible for the 'daily' rhythm.

It is possible that eating at night may lead to chronodisruption in the NES patient, which in turn could further impair the health of the individual. For example, if food is eaten at an atypical time, this could lead to internal circadian desynchrony between the central and peripheral clocks (O'Reardon, Stunkard et al. 2004; Goel, Stunkard et al. 2009). Chronodisruption is hypothesized as an etiological factor of obesity and metabolic disease (Froy 2010; Garaulet, Ordovas et al. 2010), depression (Srinivasan, Pandi-Perumal et al. 2009), insomnia (Srinivasan, Pandi-Perumal et al. 2009), and cancer (Reiter, Tan et al. 2007). On the contrary, it is also possible chronodisruption is the cause rather than the consequence of night eating but that the symptom (night eating) reinforces the chronodisruption.

Indeed, there is behavioural and biological evidence of chronodisruption in NES patients. Behaviourally, the feeding rhythm in NES patients is delayed 1.5 hours (Goel, Stunkard et al. 2009) and they experience insomnias and have elevated depressive symptoms, both of which are common side effects of circadian rhythm disorders and associated with circadian problems (Daimon, Yamada et al. 1992; Lam 2008). More specifically, diurnal mood variations, such as the evening-worsening mood in NES, are indicative of circadian rhythm problems (Boivin, Czeisler et al. 1997). Biologically, NES patients had lower values of nocturnal melatonin and leptin levels and higher nocturnal cortisol compared to controls (Birketvedt, Florholmen et al. 1999a). In a different study, NES patients had phase-delayed rhythms of melatonin (-1.1h), cortisol (-0.7h, ns), insulin (-2.8hrs), leptin (-1h) and glucose (-12.4hrs) had a phased-advanced rhythm of ghrelin (5hrs), compared to controls (Goel, Stunkard et al. 2009). NES patients also had differences in rhythm amplitudes, i.e., rhythm robustness, such as reduced amplitudes in the rhythms of cortisol (-25.7%), ghrelin (-49.6%), and insulin (-57.7%). Furthermore, as mentioned above, rhythms that are typically in synch were uncoupled in the NES group, i.e., glucose and insulin and ghrelin and leptin.

Obesity

The relationship between NES and obesity is elaborated in the manuscript presented in chapter 3 (Gallant, Lundgren et al. 2012b). Therefore, this section will report on the general

conclusions of the association between NES and obesity and will discuss more recent perspectives on this issue.

Although studies demonstrate that NES patients do not necessarily consume excess energy (section 1.2), these studies are few and there is potential for a great deal of error in energy reporting. It would be expected that NES patients consume more not only because they eat during the night but also because they report obesity-related eating patterns (emotional and stress-related eating, irregular eating, grazing etc.) and because they report disrupted sleep. The latter could contribute to excess energy intake through altered appetite control, independent of nocturnal ingestions (Spiegel, Tasali et al. 2004; Taheri, Lin et al. 2004; Chaput, Despres et al. 2007).

The phenomenon that the timing of food intake can influence body weight is discussed in several recent reviews (Allison, Goel et al. 2014; Garaulet and Gomez-Abellan 2014). Epidemiological studies have demonstrated evening eating as a risk factor for obesity (Baron, Reid et al. 2011; Wang, Patterson et al. 2013). Moreover, while on a weight loss program, participants who consumed more of their daily food intake earlier in the day lost more weight than those who consumed more of their intake later in the day (Garaulet, Gomez-Abellan et al. 2013; Jakubowicz, Barnea et al. 2013). Under this hypothesis, excessive evening and night eating *per se* could lead to unfavorable changes in energy balance and obesity.

Despite a mainly consistent relationship between NES and obesity, only two studies examined the longitudinal association between night eating behaviours and weight gain and both studies were conducted in already obese populations. It is imperative to further investigate this issue, particularly in different populations, if night eating behaviours are to be considered a risk factor for obesity.

Metabolic consequences

This issue was briefly discussed in section 1.2 and will be discussed in more detail in Chapter 5. Concisely, eating during the night may lead to unfavourable metabolic changes. Indeed, in both NES patients and in an experimental study, night eating and eating late at night, respectively, lead to alterations in glucose and insulin (Qin, Li et al. 2003; Allison, Ahima et al. 2005). However, whether these metabolic changes lead to metabolic health problems over the long-term is currently unknown and despite the close connection between the circadian system

and metabolic homeostasis (Bass and Takahashi 2010; Marcheva, Ramsey et al. 2013), few researchers have investigated the relationship between NES and metabolic diseases.

In one study, metabolic syndrome criteria were not different between obese patients with and without NES (Calugi, Dalle Grave et al. 2009a). Similarly, baseline metabolic features were not different between NES and non-NES diabetic patients participating in a lifestyle intervention study (Allison, Crow et al. 2007). In contrast with these studies, Morse et al., (Morse, Ciechanowski et al. 2006) observed that the physical health was worse in of diabetics with evening hyperphagia compared to diabetics without this eating behaviour. Indeed, sleep disturbances (52% vs. 30%) and diabetic neuropathy (46% vs. 33%) were more prevalent in those with evening hyperphagia and they had greater odds of being obese (OR (CI) = 2.6 (1.5 – 4.5)), of reporting more diabetic-related complications (OR (CI) = 2.6 (1.5 – 4.5)) and of having an A1C > 7% (OR (CI) = 2.2 (1.1 – 4.1)). To date, our knowledge of the relationship between NES and metabolic disease is limited to a few cross-sectional analyses.

Depression

NES patients generally report more depression symptoms than controls, regardless of BMI or metabolic status (Figure 1.9). NES patients have a pooled average depression score of 16.6, which is considered “moderately depressed” (Beck, Steer et al. 1988). Moreover, the prevalence of a lifetime diagnosis of depression was high in NES (56%) (de Zwaan, Roerig et al. 2006) and higher in young adults with NES compared to those without (25.5% va 10%) (Runfola, Allison et al. 2014).

The association between NES and depressive symptoms may be biased because major depressive disorder (MDD) is usually an exclusion criterion in NES studies. Without this exclusion criterion, a large percent of NES patients also met criteria for MDD (18.8% to 52.6%) (de Zwaan, Roerig et al. 2006; Lundgren, Allison et al. 2008a). Furthermore, 21.3% of MDD patients also met NES criteria (Kucukgoncu, Tek et al. 2014b) and individuals with MDD had significantly greater odds of meeting NES criteria compared to non-depressed controls (35.2% vs. 19.2%, OR = 2.64, 1.5 – 4.6)(Orhan, Ozer et al. 2011).

Like the other NES health associations, it is uncertain if NES leads to depression or vice versa. Theoretically, NES could physiologically impact depressive symptoms directly through circadian dysregulation. Chronodisruption is believed to lead to depression (Srinivasan, Pandi-

Perumal et al. 2009). It could also lead to depression indirectly through the impact that night eating has on sleep knowing that insomnia is a diagnostic criterion for MDD and sleep troubles usually precedes depression (Lam 2008). The relationship could also be a cycle between evening hyperphagia and an evening-worsening mood, similar to the self-perpetuating relationship between negative affect and binge eating.

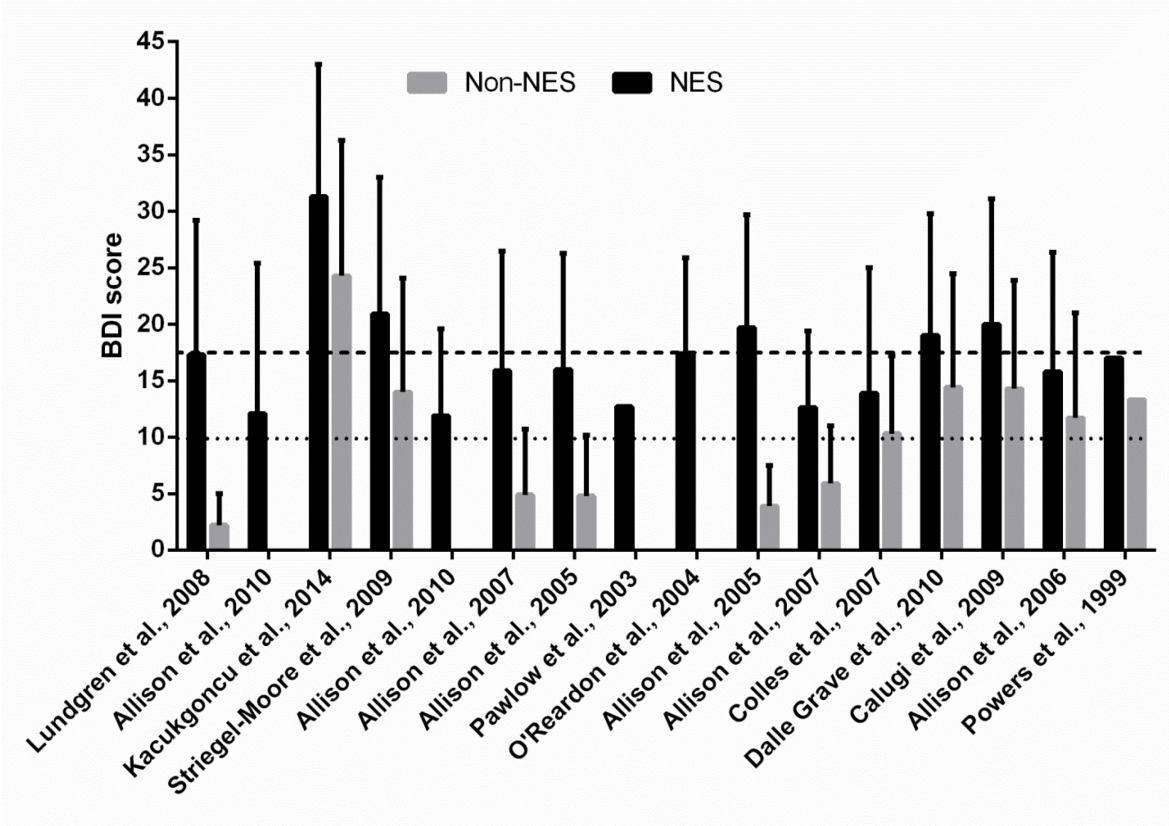

Figure 1.9. Beck Depression Inventory (BDI) scores (Beck, Steer et al. 1988) between NES and controls. Studies are ordered from smallest to greatest sample mean BMI (left to right). Thick dotted line is the overall mean for the NES samples; the thin dotted line is the overall mean for the control samples. In most studies, NES scores were significantly higher than from controls (Powers, Perez et al. 1999; Pawlow, O'Neil et al. 2003; O'Reardon, Ringel et al. 2004; Allison, Ahima et al. 2005; Allison, Grilo et al. 2005; Allison, Wadden et al. 2006; Allison, Crow et al. 2007; Allison, Grilo et al. 2007; Colles, Dixon et al. 2007; Lundgren, Allison et al. 2008a; Calugi, Dalle Grave et al. 2009b; Striegel-Moore, Franko et al. 2009; Allison, Lundgren et al. 2010b; Dalle Grave, Calugi et al. 2010a; Kucukgoncu, Tek et al. 2014a).

Regardless of the direction of the relationship, it seems that the negative health impacts of depression and NES may be additive. In MDD patients, those with NES had higher depression scores, greater BMI, reported more sleep disturbances, and reduced sleep quality (Kucukgoncu, Tek et al. 2014b). Likewise, depressed evening (>50% after 7PM) and late-night eaters (>11pm) had reported sleep troubles (onset insomnia, being tired) and had more of a loss of appetite than non-depressed late-night eaters (Striegel-Moore, Franko et al. 2008). Thus, NES could theoretically lead to depression or the two disorders in concert may further impair the physical and psychological health of the individual.

Perceived stress and anxiety

NES is consistently associated with stress and was originally describes as "a special diurnal response to stress characteristic of some obese persons" (Stunkard, Grace et al. 1955b). In this study, 90% of the night eating cases occurred during times of life stress. Other reports also document increased stress in NES. NES patients reported higher scores of perceived stress compared to controls and to US norms (Pawlow, O'Neil et al. 2003; Lundgren, Allison et al. 2008b), 61% of NES patients reported psychological distress compared to 15% of controls (Napolitano, Head et al. 2001) and night eating symptoms were positively related with perceived stress scores in university students (Wichianson, Bughi et al. 2009). Moreover, NES patients also met criteria for anxiety disorders: 10.4 - 50% panic disorder, 17.5 - 20% general anxiety disorder (Spaggiari, Granella et al. 1994; de Zwaan, Roerig et al. 2006).

NES does co-occur with perceived stress and stress-related psychopathologies but the relationship between NES and stress is complex. It is possible that the relationship is cyclic because, on the one hand, stress is a potential underlying mechanism of NES while on the other, does NES causes distress in the individual. This is confirmed by the presence of distress as diagnostic criterion for the disorder, which is confirmed in testimonies of individuals with NES. The following is a excerpt taken from an online NES self-help forum:

> "I am a 48 y old woman, I have been night eating for 30 yrs, since age 18. It has ruined my life. I am at the end of my rope.... It seems like ANOTHER PERSON gets up and starts shoving food into my mouth at 12am, 1a, 2a...I wake up bloated & DEAD...I have lost jobs, relationships, etc. over the yrs. Now I am just held up in my house, afraid to even fall asleep at night. I used to be productive & pleasant. Now I am a total wreck." - Anonymous

Psychological stress and the impact that biological stress has on cognition (heightened emotional function), appetite control and reward behaviour can lead to habitual excess energy intake and obesity (Dallman 2010). In rats, repeated exposure to chronic stress lead to hyperphagia, weight gain and visceral adiposity (Tamashiro, Sakai et al. 2011). Furthermore, chronic stress can directly impact the circadian system, which could potentially exacerbate night eating symptoms.

Sleep problems

As reported in section 1.2, NES patients frequently report sleep troubles (Napolitano, Head et al. 2001; Morse, Ciechanowski et al. 2006; Rogers, Dinges et al. 2006; Lundgren, Allison et al. 2008a; Cleator, Abbott et al. 2013).and were shown to have reduced sleep efficiency (Rogers, Dinges et al. 2006)yet with similar sleep onset and offset times (O'Reardon, Ringel et al. 2004; Rogers, Dinges et al. 2006).

Despite greater measured and perceived sleep troubles, it is not certain if NES patients experience greater daytime fatigue. In two studies, daytime fatigue was not greater in NES patients compared to controls (Rogers, Dinges et al. 2006; Lundgren, Allison et al. 2008a) yet in others, NEQ scores were positively correlated with scores of daytime sleepiness (Olbrich, Muhlhans et al. 2009; Cleator, Abbott et al. 2013); however, this relationship was mediated by sleep quality in one of the two studies (Cleator, Abbott et al. 2013).

The insomnias associated with NES may impair the health of the individual. Reduced sleep duration has been associated with obesity (Cappuccio, Taggart et al. 2008; Patel 2009) and weight gain (Patel and Hu 2008; Spaeth, Dinges et al. 2013), depression (Srinivasan, Pandi-Perumal et al. 2009), and cardiovascular disease (Cappuccio, Cooper et al. 2011). Sleep loss also results in increased appetite (Spiegel, Tasali et al. 2004) and altered appetite hormone control (Spiegel, Tasali et al. 2004; Taheri, Lin et al. 2004; Chaput, Despres et al. 2007) which may perpetuate the nocturnal eating cycle.

Conclusion of the health consequences of NES

There are several possible avenues through which NES could negatively impact health (Figure 1.10). NES may directly impact metabolic health if night eating behaviours lead to excess energy intake. NES may also directly impact metabolic health through the metabolic

consequences associated with night eating *per se*, i.e., eating during a time reserved for sleep and fasting.

NES may indirectly influence metabolic and psychological health through the impact of night eating on circadian regulation and sleep. Indeed, NES, by its definition, does cause distress and sleep troubles and is associated with depression and stress but the direction of these relationships is uncertain and may even be cyclic. Moreover, because of the interrelationships between temporal eating patterns, chronodisruption and psychological and metabolic problems, it is difficult to tease out the causes and consequences of night eating behaviours.

Despite some evidence that suggests night eating behaviours may lead to weight gain, the study populations were already very obese. Moreover, the metabolic health associations and consequences of NES are currently unknown. There is an urgent need for more longitudinal studies on NES and the associated symptoms to determine whether these eating behaviours are indeed a risk factor for obesity and metabolic disease. Furthermore, these aspects need to be investigated in populations with varying body weights and ages to help isolate potential mediating factors.

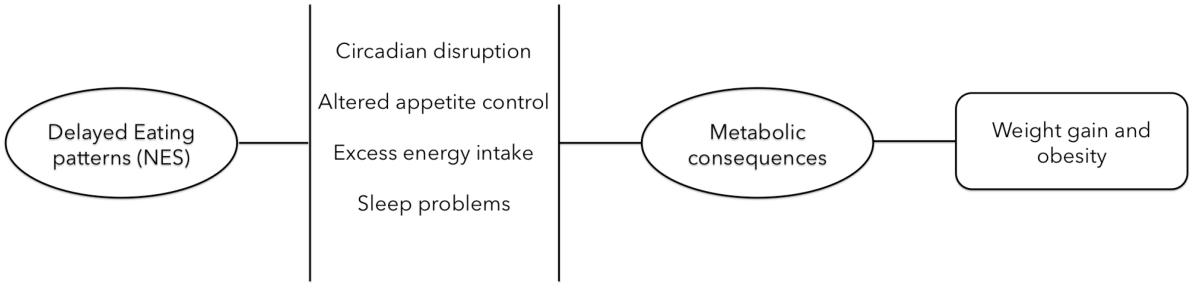

Figure 1.10. Schematic diagram of the theoretical pathways through which a delayed eating pattern could impact metabolic and psychological health.

1.7: Clinical utility of NES

NES often co-occurs with other psychiatric disorders. Indeed, the prevalence of NES (12.3%) and an night eating behaviours (40%), i.e. > 25% and/or > 3 nocturnal ingestions per week, was higher in a psychiatric populations compared to the general population (Lundgren, Allison et al. 2006a; Lundgren, Rempfer et al. 2010). From a clinical perspective, it is important to distinguish NES from other psychopathologies, such as Major Depressive Disorder, anxiety disorders and other eating disorders, such as Binge Eating Disorder, Bulimia Nervosa, and Anorexia Nervosa.

Binge Eating Disorder (BED)

There is overlap between NES and BED, particularly in obese samples (Figure 1.11). Night eating behaviours are prevalent in BED and night eaters report binges more frequently than non-night eaters (Lundgren, McCune et al. 2011; Grilo, Milsom et al. 2012). Despite the overall, it is generally accepted that NES and BED are distinct disorders that affect similar populations (Stunkard and Allison 2003). Indeed, BED and NES are both related to the serotonergic system and have HPA-axis abnormalities that reveal fundamental similarities (Kaye 2008; Stunkard, Allison et al. 2009b); however, a heritability study of revealed that, although NES and BED are related, particularly in women, they remain independent from each other (Root, Thornton et al. 2010). Moreover, although NES and BED severity are correlated (Harb, Levandovski et al. 2012; Dalle Grave, Calugi et al. 2013), binge eating severity was not a distinguishing factor of NES (Calugi, Dalle Grave et al. 2009a; Dalle Grave, Calugi et al. 2010b). Similarities and differences are summarized in Figure 1.12.

The most notable distinguishing factors between NES and BED are related to eating patterns and behaviours. Firstly, NES but not BED is consistently associated with a delayed pattern of energy intake (evening hyperphagia, nocturnal eating, breakfast skipping) (Stunkard and Allison 2003; Allison, Grilo et al. 2005). In contrast, Individuals with BED report more objective binge episodes than individuals NES (Allison, Grilo et al. 2005; Runfola, Allison et al. 2014). Compared to NES, BED patients are more consistently associated with disinhibited eating (Napolitano, Head et al. 2001; Allison, Grilo et al. 2005; Colles, Dixon et al. 2007) and susceptibility to hunger (Allison, Grilo et al. 2005; Colles, Dixon et al. 2007).

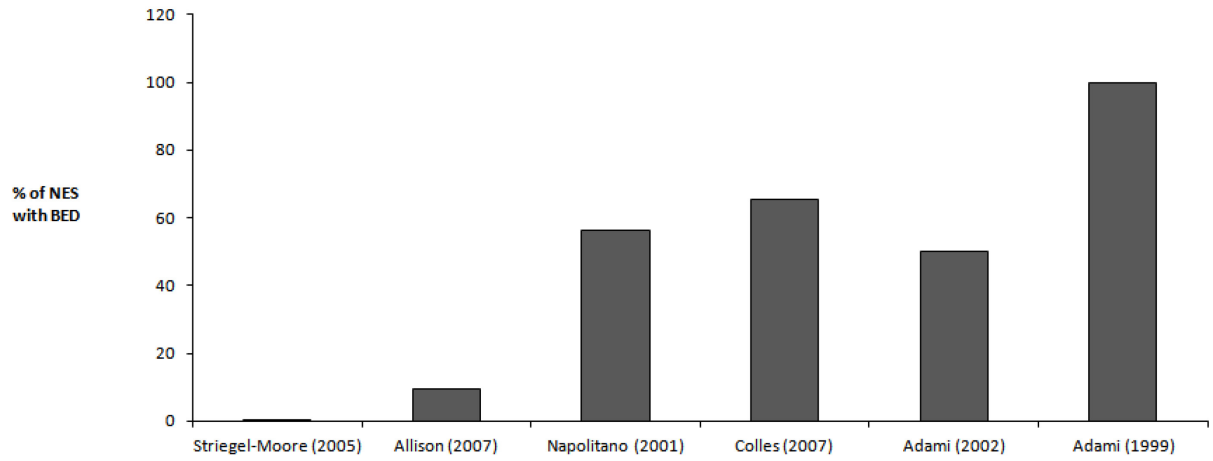

Figure 1.11. Percent of NES patients that also meet criteria for BED, according to sample mean BMI (NES and BED diagnosed by interview) (Adami, Meneghelli et al. 1999; Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Striegel-Moore, Dohm et al. 2005; Allison, Crow et al. 2007; Colles, Dixon et al. 2007).

Psychological factors also distinguish the two disorders. BED is associated moreso with weight-related psychological factors, such as shape/weight dissatisfaction (Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Allison, Grilo et al. 2005; Colles, Dixon et al. 2007; Fischer, Meyer et al. 2012; Runfola, Allison et al. 2014), preoccupation and guilt (Jarosz, Dobal et al. 2007). Those with BED had higher daily anxiety while those with NES had higher nocturnal anxiety (Sassaroli, Ruggiero et al. 2009) and depression scores were higher in binge eaters compared to NES patients (Colles, Dixon et al. 2007) but not when compared to individuals with BED (Allison, Grilo et al. 2005).

Interestingly, emotional eating may one factor that connects the two disorders. One study demonstrated an interaction effect between NES and emotional eating on binge eating frequency; NES severity was related to binge eating frequency only in emotional eaters (Meule, Allison et al. 2014). In line with this, some studies have found that individuals with NES and BED present more psychopathology than individuals with NES alone (Napolitano, Head et al. 2001; Colles, Dixon et al. 2007).

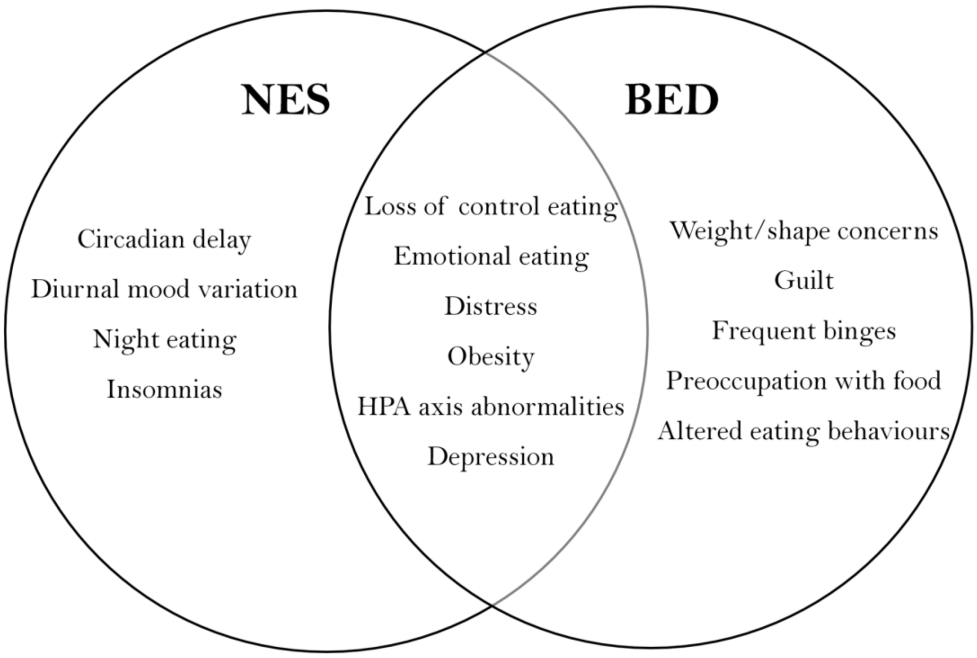

Figure 1.12. Similarities and differences in clinical characteristics between binge eating disorder (BED) and night eating syndrome (NES) (Adami, Meneghelli et al. 1999; Napolitano, Head et al. 2001; Adami, Campostano et al. 2002; Striegel-Moore, Dohm et al. 2005; Allison, Crow et al. 2007; Colles, Dixon et al. 2007).

Bulimia nervosa and Anorexia nervosa

The overlap between NES and other eating disorders has been less studied but the limited results shows some overlap between NES and other eating disorders. The prevalence of NES was 40.6% among those with BN and 9.4% among those with AN (Lundgren, McCune et al. 2011) and night eating behaviours were prevalent in BN patients (38.7% evening hyperphagia, 12.9% nocturnal ingestions) (Lundgren, Shapiro et al. 2008). Moreover, 11% of young adults who met NES criteria reported a lifetime diagnosis of AN (Runfola, Allison et al. 2014). Thus, the significant overall between NES and BN, and the fact that night eating behaviours are common in individuals with eating disorders raises some possible problems concerning the clinical utility of NES. As stated by Lundgren et al. “Perhaps when night eating occurs in the context of refusal to maintain body weight or compensatory behaviours, night eating behaviour is best conceptualized as part of the eating pathology associated with AN and BN. In this case, diagnoses of AN or BN with night eating features would be more appropriate than a co-morbid NES diagnosis.” (Lundgren, McCune et al. 2011). Night eating behaviours in

the absence of other eating behaviours would, however, merit a diagnosis of NES (Striegel-Moore, Franko et al. 2009).

Overlap with other Psychiatric disorders

Psychopathology is common in NES samples and overall with other disorders is common. Forty percent of psychiatric patients, e.g., Schizophrenia, bipolar disorder, MDD, reported either evening hyperphagia (>25%) or nocturnal ingestions greater than 3 times per week (Lundgren, Rempfer et al. 2010). In young adults with a research diagnosis of NES, 16.4% reported being diagnosed with an anxiety disorder compared to 7% of the controls (Runfola, Allison et al. 2014). Non-obese night eaters were more likely to meet thresholds for Axis I disorders (unipolar mood disorders and anxiety disorders) (Lundgren, Allison et al. 2008a) and psychological distress was also more prevalent among those with NES compared to controls (Napolitano, Head et al. 2001). Higher prevalence of lifetime ADHD was reported in the NES group compared to controls (20% vs. 4.7%) (Runfola, Allison et al. 2014). Furthermore, in a descriptive study of NES patients, the highest lifetime prevalence of DSM-IV disorders were major depression (55.6%), PTSD (18.1%), current dysthymia (11.3%), BED (14.2%) and panic disorder (10.4%) (de Zwaan, Roerig et al. 2006).

1.8: Mechanistic underpinnings of NES

Heritability and possible genetic underpinnings of NES

As discussed in greater detail in section 1.2, genetics could be involved in the relationship between NES and health. Several studies have demonstrated night eating behaviours to have a potential genetic underpinning. First-degree relatives of NES patients were almost five times more likely to themselves have NES (Lundgren, Allison et al. 2006b). Likewise, we found that NEQ scores were low to moderately heritable in parents and offspring and that correlations in NEQ scores were stronger between the mother and the offspring compared to the father and the offspring (Lundgren, Drapeau et al. 2012). Additionally, in twins correlations were moderate for night eating symptoms and coefficients were stronger in monozygotic compared to dizygotic twin pairs, which indicates additive genetic effects in the propagation of these behaviours (Root, Thornton et al. 2010). Moreover, additive genetic effects potentially explained 35% of the variance in night eating behaviours in women and 44% in men (Root, Thornton et al. 2010). These studies suggest genetics could be involved in night eating

behaviours but to date, there are no genes associated with NES in humans. Indeed, candidate gene studies are needed to advance this area.

Chronic stress and the HPA axis

NES patients commonly report their symptoms onset coincides with a traumatic life event or during a period of high life stress (Stunkard, Grace et al. 1955c; Spaggiari, Granella et al. 1994; de Zwaan, Roerig et al. 2006). Often, NES patients are also chronically stressed and demonstrate elevated cortisol levels or altered cortisol rhythms compared to controls (Table 1.5). Thus, the stress-response system is believed to be involved in the onset of night eating.

The hypothalamic-pituitary-adrenal axis (HPA axis) of the stress response system is an important player in chronic biological stress. During the stress response, a stressor (perceived or physiological) leads to a cascade of direct neurohumeroal stimulation terminating with the release of the glucocorticoid cortisol from the adrenal cortex which, among other functions, increases arousal and metabolism and suppresses sleep and digestion (Chrousos 2009). Glucocorticoids, in turn, terminate the stress response by affecting the secretion of CRH and ACTH (Charmandari, Tsigos et al. 2005). If the negative feedback loop fails to terminate the response, if the stress system is repeatedly activated, or if the system responds excessively, the tissues may be exposed to increased or prolonged these neurohumoral factors and leads to chronic biological stress (Charmandari, Tsigos et al. 2005; Chrousos 2009). An overactive HPA-axis and chronic stress can lead to increased food intake, insomnia, depression, anxiety and to obesity (Chrousos 2009).

Interesting to NES, there is much crosstalk between the circadian and stress response systems and chronic stress may interfere with these connections. The secretion of glucorcorticoids from the adrenal cortex is tightly controlled by the SCN (dependent and independent of the HPA axis) and, consequently, they follow a robust circadian rhythm that follows the activity phase (in humans, high morning, low nighttime levels) (Dickmeis 2009; Nader, Chrousos et al. 2010). In this way, the SCN is believed to use glucocorticoids to communicate with the periphery both directly and indirectly via their ability to entrain peripheral clocks in the liver, kidney and heart (Balsalobre, Brown et al. 2000; Mendoza 2007; Cermakian and Boivin 2009; Girotti, Weinberg et al. 2009; Huang, Ramsey et al. 2011; Prasai, Pernicova et al. 2011) through glucocorticoid receptor signalling (Mavroudis, Scheff et al. 2012). Thus,

glucocorticoids keep the genetic “clockwork” of the SCN and periphery in sync – they are one example of humeral signals that convey time information to peripheral clocks (Pezuk, Mohawk et al. 2012).

Table 1.5. Cortisol in NES and controls

Authors	Study Design	Results
Birketvedt et al., (1999a)	24-h neuroendocrine study; no night eating permitted	Higher nocturnal cortisol among NES
Birketvedt et al., (2002)	CRH-induced cortisol and ACTH responses	NES had lower ACTH (47%) and cortisol responses (71%)
Allison et al., (2005)	24-h neuroendocrine study; night eating permitted	No difference in nocturnal cortisol or prolactin
Morrow et al., (2008)	Fasting cortisol pre- and post-bariatric surgery	Higher fasting afternoon cortisol levels 5 months post-surgery in NES patients
Geliebter et al., (2012)	Stress and hunger response to a cold pressor test (CPT)	Higher baseline and CPT response in night eaters; no differences in response after controlling for baseline
Geliebter et al., (2002)	Cortisol response to a CPT and dexamethasone administration	NES patients had higher baseline cortisol, higher cortisol response to CPT (mediated by depression) and reduced cortisol suppression (controlling for depression)
Allison et al., (2013)	Salivary cortisol measures in the morning, before lunch, before supper and before bedtime	Women with NES had higher cortisol levels, particularly before lunch and supper; evening cortisol levels were related to night eating symptoms

Glucocorticoids can impact the periphery independent of the central clock because they are also released in response to stressors. Moreover, in chronic stress glucocorticoids are repeatedly or excessively secreted in response to a stressor which may attenuate the glucocorticoid rhythm and increase its number of daily pulses (Dickmeis 2009), a phenomenon seen in chronically stressed individuals (Nader, Chrousos et al. 2010). The loss of this usually

robust glucocorticoid signal could lead to the momentary uncoupling or chronic disorganization of the central and peripheral clocks. This is because glucocorticoids can entrain peripheral clocks but they cannot directly entrain clocks in the CNS because the SCN does not have glucocorticoid receptors (Prasai, Pernicova et al. 2011). Indeed, a recent study has hypothesized and demonstrated mathematically that changes in the cortisol rhythm (amplitude, frequency) may lead cells to become desynchronized, “uncoupling them from the systemic circadian network” (Mavroudis, Scheff et al. 2012). Uncoupled central and peripheral clocks is a hypothesized mechanism of NES (O'Reardon, Ringel et al. 2004; Goel, Stunkard et al. 2009).

The importance of intact peripheral clocks in feeding was demonstrated in flies. Xu and colleagues reported that peripheral clocks in the digestive and metabolic tissues regulated the feeding rhythms in *Drosophila* (Xu, Zheng et al. 2008). Specifically, they observed that flies that lacked clocks in these tissues increased feeding at night. Thus, as the authors state, clocks in these tissues would normally regulate the timing of feeding and inhibit nighttime eating (Xu, Zheng et al. 2008). Moreover, when feeding is uncoupled from sleep (restricted feeding paradigm in rodents), the peripheral clocks become entrained to the meal (and not light/dark) and lead to food-anticipatory activity (FAA), i.e., behaviours that indicate the animal is ready to eat (Mistlberger 2011). Interestingly, individuals who consumed nocturnal ingestions had greater 24-h spontaneous physical activity (SPA) measures, even during the night, which the authors concluded was representative of altered circadian rhythms or disrupted sleep (Gluck, Venti et al. 2011). It is possible that the SPA could also be a form of food-anticipatory behaviour, but studies have yet to investigate FAA in humans. Because of the connection between feeding and activity rhythms, studying the physical activity patterns in NES may shed light on this disorder. Along these lines, exercise may indeed be able to synchronize certain peripheral clocks (Atkinson, Edwards et al. 2007; Edwards, Reilly et al. 2009).

More simplistic explanations could involve the direct impact that stress has on appetite and sleep hormones and behaviours. Higher evening glucocorticoids, observed in NES (Allison, Lundgren et al. 2013) and chronically stressed individuals (Nader, Chrousos et al. 2010), may inhibit melatonin (possibly directly at the pineal gland) and leptin secretion (possibly at the adipose tissue), which in turn, leads to disturbed sleep and increased hunger (Birketvedt, Florholmen et al. 1999a). Indeed, cortisol has been shown to suppress melatonin, particularly at night (Monteleone, Fuschino et al. 1992). Healthy young men were stressed by exercising

during the late evening (22h40 – 23h). The exercise led to an increase in cortisol, which was followed by an attenuated nocturnal melatonin surge.

Another behavioural model could be that evening and nighttime stress or negative affect may lead to an evening-specific habit of coping with stress through eating rather than a cognitive approach (Dallman 2010). Indeed, it has been suggested that individuals may engage in night eating behaviours as a maladaptive coping strategy for perceived stress, strategies that involve an emotional focus (Wichianson, Bughi et al. 2009). Interestingly, psychological stress in rats elicited a greater stress response in the early rest phase ("evening") and a physical stress elicited a greater response in the early activity phase ("morning") (Dickmeis 2009).

Diet and endocrine/metabolic disease

An unhealthy diet that precedes obesity may also be a factor in the etiology of NES. High-fat diets in mice led to changes in the rhythms of peripheral and central clock gene expression and changes in behavioural patterns, such as increases in locomotor activity and more frequent feeding behaviour during the "night" in addition to attenuated locomotor and feeding rhythms. Interestingly, these changes preceded the onset of obesity (Kohsaka, Laposky et al. 2007). Additionally, mice fed a high-fat diet to induce weight gain were less able to readjust after simulated jet-lag, concluding altered circadian synchronization to light (Mendoza, Pévet et al. 2008). Reciprocally, mice with genetically-induced obesity, such as in strains of mice with "type 2 diabetes" and mice with leptin deficiencies, demonstrated altered circadian rhythms of clock gene expression in the nucleus of the solitary track (Kaneko, Yamada et al. 2009) and in the adipose tissue and liver (Ando, Yanagihara et al. 2005).

Interestingly, although to date there are insufficient data to support these hypotheses, an underlying metabolic state may exacerbate night eating symptoms. In line with this, Van Somener et al., stated a hypothesis that "with an irregularly timed zeitgeber a robust rhythm can only be accomplished by a strong endogenous oscillator… or with a weak endogenous oscillator a robust rhythm can only be accomplished by a strong and regularly timed zeitgeber." In this context, this would mean that individuals with attenuated rhythms to start (metabolic disease, for example) might not be able to counteract the impact of peripheral entrainment to nocturnal meals. However, for now, these are all quite unsupported claims.

To date, the cause of NES is unknown. Nonetheless, there are several interesting hypotheses that merit exploration. However, the complex and cyclic relationships between circadian regulation, stress, metabolism and behaviour will make it difficult to tease out the exact cause of NES. More longitudinal studies are needed and factors that precede the onset of NES need to be identified, suggesting that studies in younger populations need to be initiated.

1.9: Treatment of NES

Although not numerous, the published NES treatment modalities are diverse. They range from pharmacological treatments to more holistic approaches. The treatments aim to change behaviour by acting on brain chemistry but also through behavioural psychotherapy and education. NES symptoms have also improved second to weight loss surgery.

Behaviour modification and psychotherapy

Cognitive behavioural therapy has been used to treat NES. Twelve weeks of individual psychotherapy sessions together with a weight loss/maintenance diet significantly reduced nocturnal ingestions (from 8.7 to 2.6/ week), evening hyperphagia (from 35% to 24.9%), NES and improved depressive symptoms and quality of life (Allison, Lundgren et al. 2010a). Also, the patients lost an average of 3.1 kg of body weight.

In an intense, in-patient behavioural weight loss trial, 35 NES patients and controls were put on a low calorie diet, exercised and participated in psyho-education group sessions for 21 days (Dalle Grave, Calugi et al. 2011). At 6 months follow-up, only 22.8% of the NES patients reported evening hyperphagia (>25%) and/or nocturnal ingestions of food > 3 times/week. Moreover, 51.4% of the NES patients reported no nocturnal ingestions during the past 3 months. Weight loss (BMI reduction) was similar between NES and controls (post inpatient: -1.9 vs. -1.5, 6 months: -2.7 vs. -3.3 kg/m^2). These improvements in NES symptomatology were not related to body weight per se because changes in BMI were not different between those who retained an NES diagnosis at follow-up compared to those who no longer met NES criteria. However, a main limitation of this study is that the authors could not determine whether it was the exercise, the psyhoeducation or the diet that resulted in these improvements.

Pharmaceutical treatment

Anticonvulsants

In a case-study report, two patients with NES were treated with topiramate, an antiseizure/anticonvulsant drug (Winkelman 2003). One of the NES patients reported reduced nocturnal ingestions frequency (1/night to 1/week) and lost 33 lbs whereas for the other patient, nocturnal ingestions completely remitted from 1/night, with a 15 lbs weight loss. For this patient, when the dose was lowered due to eye twitching, nocturnal ingestions recommenced at a frequency of 2/week.

In another case study, an obese woman was treated with topiramate for night eating in addition to sleep walking and post-traumatic stress. The treatment stopped the night eating, sleep walking and post-traumatic stress symptoms and she lost 70 lbs. (Tucker, Masters et al. 2004)

Serontonin-related drugs

Sertraline, a selective serotonin reuptake inhibitor (SSRI) that creates serotonin homeostasis has been studied in NES treatment. In a randomized, placebo-controlled trial of Sertraline in 34 NES patients, NES symptoms improved more for the Sertraline group compared to the placebo group; after 8 weeks, nocturnal ingestions dropped from 8.3/wk to 1.6/wk in the Sertraline group whereas they went from 6.4 to 5.5/wk in the placebo group. Likewise, the extent of evening hyperphagia went from 47.3% to 14.8% in the treated group compared to 44.7 to 31.6% in the placebo group (O'Reardon, Allison et al. 2006).

In open-label trials, 12-week administration of Sertraline resulted in reduced nocturnal ingestions (1.5 to 0.5 per night), reduced extent of evening hyperphagia (51.6% to 25.7%). In addition, 5 of the 17 patients no longer met NES criteria after the treatment and these individuals experienced significant weight loss (4.8kg) (O'Reardon, Stunkard et al. 2004). Similar results were observed in NES patients treated with Sertraline and by which their NES symptoms were assessed via questionnaires and telephone interviews. NEQ scores were reduced, the frequency of evening hyperphagia, nocturnal awakenings and nocturnal ingestions of food were also reduced. Their mood also improved. This also resulted in a 3kg body weight loss (Stunkard, Allison et al. 2006).

In an earlier case study, 7 patients were treated for a mean of 11 months by fenfluramine, an anorectic drug that promotes serotonin secretion (Spaggiari, Granella et al. 1994). Of the 7 patients, 1 recovered completely Symptoms improved for 5 patients (50% reduction in frequency of nocturnal ingestions, and a >70% decrease in extent of evening hyperphagia). However, this drug is no longer on the market due to secondary cardiovascular effects.

Other anti-depressants

Agomelatine is a selective agonist of type I and type II melatonin receptors and an antagonist of serotonin 5-HT (2C) receptors, has recently been used in patients with MDD, SAD and bipolar disorder with success by improving sleep and depressive symptoms (Dolder, Nelson et al. 2008). It has created phase advances in core body temperature in humans (Krauchi, Cajochen et al. 1997; Schulz and Steimer 2009). In 5 case-reports, 3 month administration of Agomelatine resulted in reduced symptom severity (NEQ scores decreased by 10 units), improved mood, fewer weekly nocturnal ingestions of food (from 12 to 6.4/wk), and an mean 3.6kg weight loss (Milano, De Rosa et al. 2013).

Other treatments

Herbal medications

Herbal medications, specifically melatonin or tryptophan, have been tried as well, with minimal success (O'Reardon, Peshek et al. 2005).

Light therapy

One patient with depression and who also met NES criteria was treated with light therapy (10, 000 lux white light for 30 minutes for 14 consecutive days). The treatment resulted in improvements in depressive symptoms (no longer met DSM-VI criteria for MDD) and the patient no longer experienced nightly nocturnal ingestions, morning anorexia and evening hyperphagia of > 50% NES (Friedman, Even et al. 2004). This protocol also improved the mood and reduced the night eating symptoms in an obese woman (Friedman, Even et al. 2002). However, after the light therapy was stopped, night eating symptoms returned although the depressive symptoms did not. Light treatment again stopped her night eating symptoms.

Relaxation therapy

Abbreviated Progressive Muscle Relaxation Therapy (APRT) was also used in the treatment of NES with the aim of reducing stress and anxiety (Pawlow, O'Neil et al. 2003). One week of daily APRT exercises had lowered stress, anxiety and depression in NES patients and their hunger ratings had shifted to an earlier circadian rhythm – higher morning hunger and lower late evening hunger.

Bariatric surgery

Several studies examined NES and night eating behaviours before and after bariatric surgery. Generally, the prevalence of NES decreased post-surgery, except for one study (Adami, Meneghelli et al. 1999)(Table 1.6). One study reported that patients reported less frequent and calorific nocturnal ingestions post-surgery than pre-surgery (Hsu, Betancourt et al. 1996). In the study by Latner and colleagues, nocturnal ingestions went from 2.84/week before gastric bypass to 0.35/week after and the prevalence of nocturnal ingestions of 3/week for 3 months decreased from 55% to 2% post-surgery. Thus, the limited data suggest that bariatric surgery may reduce night eating symptoms but the mechanisms that lead to these improvements have yet to be studied.

Table 1.6. NES prevalence before and after weight loss surgery

Author/year	**Pre-surgery NES n(%)**	**Post-surgery NES n(%)**	**Follow-up duration (yrs)**	**New NES cases n (% post-NES)**
Adami et al. (1999)	5 (7.9)	4 (6.3)	3	0
Colles et al. (2008)†	22 (17.1)	10 (7.8)	1	6 (60)
Hsu et al. (1996)	10 (42)	2 (8.3)	1 – 1.5	0
Hsu et al. (1997)	9 (33)	3 (11.1)	3.5	n/a
Latner et al. (2004)	36 (55)	1 (2)*	1 – 1.5	n/a

† postoperative reductions in nocturnal ingestions reported by 4 patients

Treatment conclusions

Night eating symptomatology can be improved through pharmacological and intensive behavioural treatment. To date, cognitive behaviour therapy and pharmacological studies seem to improve NES symptomatology to a similar degree.

1.10: Conclusions and Research problem

The literature on the Night Eating Syndrome is evolving and our understanding of this syndrome is increasing. Diagnostic criteria have been standardized, some health correlates have been established (i.e. obesity) and mechanistic pathways have been hypothesized. Despite these advances, important areas of research have not been investigated. The metabolic characterisation of the disorder is largely unexplored and the health consequences of night eating symptoms are currently unknown in members of the general population. Prospective studies are needed to assess the potential health impact of NES, particularly in mixed populations where variability in symptoms and disease are present. Moreover, night eating symptoms have been understudied in childhood populations and the health behaviour correlates of these symptoms are also unknown.

Thus, the present study aimed to investigate the behavioural and metabolic characterization of night eating symptoms in a target sample of the general population that includes a child and their biological parents. Moreover, certain methodological aspects need to be considered to accomplish these objectives. The Night Eating Questionnaire (NEQ) has yet to be used in a child population and its validity in this population is required. Moreover, if the NEQ will be used prospectively, the long-term reproducibility of the NEQ must be assessed.

The specific research objectives and hypotheses were:

1. To review the literature and synthesize the relationship between NES and obesity and to make a connection between NES, obesity and chronodisruption

2. To measure the agreement between the child's and parents' reports of the child's night eating symptoms and to measure the agreement between the child and parent reports and a 3, 24-hour dietary recall completed by the child

Hypothesis the parent and child NEQ would be related but the parent report of the child's night eating symptoms would be more valid than the child report

3. To investigate the relationship between night eating symptoms and physical activity profiles in children

   Hypothesis: children who reported a delayed pattern of energy intake (as per the NEQ) would also show a delayed pattern of physical activity

4. To synthesize the literature on the diet quality of late eaters and its impact on health, to describe the diet quality of children with night eating symptoms in QUALITY and to report on common foods consumed during nocturnal ingestions among QUALITY night-eaters (adults and children)

   Hypothesis: Nocturnal ingestions would be comprised of high carbohydrate foods, less healthy foods

5. To investigate the association between metabolic disease and markers of metabolic health and night eating symptoms in adults

   Hypothesis: night eating symptoms would be associated with obesity, metabolic syndrome and type II diabetes in men and women

6. To investigate the reproducibility, that is stability, of night eating symptoms and to examine differences according to baseline body weight status

   Hypothesis: Night eating symptoms would be reproducible across time but less reproducible in obese individuals compared to healthy weight individuals

7. To investigate the association between baseline night eating symptoms and weight change in adults

   Hypothesis: Night eating symptoms would be associated with weight gain

Chapter 2: Methods

2.1: The cohort

The QUALITY study (**QU**ébec **A**dipose and **L**ifestyle **I**nves**T**igation in **Y**outh) is a prospective study of the genetic, biological, environmental and psycho-social determinants and cardiometabolic consequences of paediatric obesity in Canadian youth (Lambert, Van Hulst et al. 2011). Families were recruited through primary schools in the Quebec City and Montreal areas. Close to 600 families with one child aged 8-10 years and one parent being overweight or obese (based on BMI and/or waist circumference) have participated in both phase I (634 families, 48% of eligible families) and Phase II (564 families; 89% retention rate). Phase I baseline data were collected between September 2005 and December 2008 when the children were 8 – 10 years. Phase II data were collected between September 2008 and March 2011 (children aged 10 – 12 years). Data for Phase III are currently being collected. Data collection centers were the Laval Hospital in Québec City and the St-Justine Research Centre in Montreal.

2.2: Measured data

Data was collected during one clinical testing day. A full list of the measured variables can be found in Table 2.1. Anthropometric data were measured for parents, and children. Body composition was measured using Dual Energy X-ray absorptiometry for the children only.

For children, dietary data were assessed by three 24-hour dietary recalls. A registered dietician administered these to the child over the telephone. The recall covered two week days and one weekend day and used the *Multiple Pass Approach Method* (Moss 2001) which has been validated in children (Johnson, Driscoll et al. 1996). Several quality checks were also performed during the following weeks by telephone (for more details see reference 6). The nutritionist was blind to the NEQ responses of the child. The dietary data were compiled using the CANDAT software (Godin London On. 2007) and converted to nutrients using the 2007b Canadian Nutrient File (Health Canada, Canadian Nutrient File 2007).

For children, physical activity data was collected objectively via 7 consecutive days of accelerometry (Actigraph LS 7164 activity monitor, Actigraph LLC, Penacola, FL, USA). Also, self-report physical activity data were collected for the 7 days during which the accelerometer was worn in addition to general physical activity participation.

Table 2.1. Measured variables in the QUALITY cohort.

	Phase I		Phase II	
	Child	Parent	Child	Parent
Resting heart rate (HR)	X	X	X	X
Resting blood pressure (BP)	X	X	X	X
Antrhopometrics	X	X	X	X
Weight	X	X	X	X
Height	X	X	X	X
Waist circumference	X	X	X	X
Hip circumference	X		X	
Triceps and subscapular skinfold thickness	X	X	X	X
Tanner Stage	X		X	
Accelerometry	X		X	
Dietary intake (3 X 24-h recalls)	X			
Fasting blood chemistry	X	X	X	X
Glucose	X	X	X	X
Insulin	X	X	X	X
Lipid profile	X	X	X	X
C-reactive protein	X		X	
Leptin and adiponectin	X		X	
Stored fasting plasma (future analyses)	X	X	X	X
Stored DNA	X	X		
Stored urine sample (future analyses)	X		X	
Oral glucose tolerance test	X		X	
Salivary cortisol	X		X	
HR variability (3-h Holter monitoring)	X		X	
Body composition (fat mass, fat free mass, % body fat), fat distribution, and bone mineral density using DEXA	X		X	
Carotid intima-media thickness (ultrasound)	X		X	
Cardiac ultrasound (ventricular mass)				
Aerobic fitness (incremental exercise test)	X		X	
Dental and oral examination	X		X	
Direct observations of built environment (school/residential)	X			

(Lambert, Van Hulst et al. 2011)

2.3: Questionnaires

Parents were self-administered several questionnaires pertaining to socio-demographic information, their health and the health of their child, and their lifestyle behaviours (physical activity and diet habits). The children were interviewer-administered questionnaires pertaining to health (depression, stress) and lifestyle (including physical activity participation).

The Night Eating Questionnaire (NEQ)

The NEQ (Allison, Lundgren et al. 2008b) was introduced mid-way during phase I data collection. For this study, the NEQ was translated from English to French and then back translated to ensure translation accuracy. The NEQ was self-administered to parents and administered to the children over the telephone a mean 3 months after the clinical testing day. We felt that if the interviwerer administered the NEQ to the children, this would ensure the child would be able to fully comprehend the questions and it would also alleviate some of the burden placed on the child during the testing day. We aimed to call each child within three weeks of the testing day, but that was not always possible due to time delays in between the testing day and when we received the file, unable to contact the family etc. Also, we changed research assistants during the summer of 2008 in which created a setback in calling lag times.

The NEQ is a research tool used to identify NES symptoms. It is comprised of 14 close-ended items assessed with 5-point likert scales. Items pertain to the proposed research diagnostic criteria of NES, e.g., evening hyperphagia (caloric consumption after the evening meal), nocturnal ingestion, morning anorexia, mood, insomnia. The NEQ is written at a 5.9 grade level and if required, the interviewer clarified the questions for the children to increase understanding. If the items are summed (excluding item 13), the total score, the NEQ score, represents a continuous measure of symptom severity. The NEQ score can range from 0 to 52.

The NEQ has been validated in adults and a screening cut-off value of 25 has been suggested for adults, but the positive predictive value increases considerably with a cut-off of 30 (40.7% vs. 72.7%) (Allison, Lundgren et al. 2008a). Moreover, the presence and absence of NES diagnostic criteria can be identified to estimate the prevalence of NES and its criteria. More recently, a study assessed the validity of the NEQ and other screening questions in correctly identifying individuals with the full diagnostic criteria of NES based on the NESHI (Allison, Lundgren et al. 2014). The sensitivity estimates of two screening questions (“How often

do you get up at night to eat" and "What proportion of your daily food intake takes place after the evening meal?") were 0.62 and 0.63 and the specificity estimates were 0.56 and 0.50 for men and women, respectively.

Parent NEQ report

Because the NEQ has never been used in a child population, an abbreviated version of the questionnaire was developed by this research team to be completed by parents to report the night eating symptoms of their child. This brief questionnaire comprises eight close-ended questions with a maximum score of 24 (five-point Likert scales where only 6 questions make up the final score). Five of the eight questions included on the parent NEQ report are identical to the child NEQ. The parent NEQ report was self-administered to the parents on the clinical testing day. When possible, we suggested it be completed by both parents or by the mother. We chose the mother because we felt the mother is generally more familiar with the child's behaviour at home.

Chapter 3: Validity of the Night Eating Questionnaire in Children

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Abstract

Objective: To measure the construct validity of the Night Eating Questionnaire (NEQ) in children against a parent report of child night eating syndrome (NES) symptoms (NEQ report) and a 3-day dietary recall. Methods: NEQ of 304 children from the QUALITY (QUébec Adipose and Lifestyle InvesTigation in Youth) cohort were compared to a parent report and 3-day dietary recall. Results: Child NEQ scores were related to the parent NEQ report ($\rho = 0.30$ $p < 0.0001$) yet there were inconsistencies between responses concerning sleep troubles. Total child NEQ scores, but not parent NEQ report scores, were associated with dietary manifestations of NES such as increased evening ($\rho = 0.20$, $p < 0.001$) and reduced morning intake ($\rho = -0.12$, $p < 0.05$). Discussion: The NEQ score is related to eating patterns of NES in children and is more informative than a parent report, specifically when used in conjunction with dietary recall.

Résumé

L'objectif de cette étude était d'évaluer la validité de certains aspects du Night Eating Questionnaire (NEQ) chez les enfants. Méthodes: Les symptômes du syndrome de fringale nocturne (SFN) des enfants ont été mesurés à l'aide du NEQ auprès de 304 enfants de la cohorte QUALITY (Québec Adipose and Lifestyle Intervention in Youth). Les réponses ont été comparées avec les symptômes (des enfants) rapportés par le parent (parent NEQ report) et 3 rappels alimentaires de 24 heures complétés par l'enfant. Résultats: Les scores du NEQ des enfants étaient liés aux scores NEQ rapporté par le parent (parent NEQ report; $\rho = 0{,}30$ $p < 0{,}0001$). Toutefois, différentes réponses ont été notées entre les enfants et les parents concernant les questions reliées aux troubles du sommeil. Le score du NEQ de l'enfant, mais n'ont pas le score rapporté par le parent (parent NEQ report), a été associé à une plus grande consommation d'aliments en soirée ($\rho = 0{,}20$, $p < 0{,}001$) et une plus faible consommation le matin ($\rho = -0{,}12$, $p < 0{,}05$). Discussion: Le score du NEQ est associé à des habitudes alimentaires caractéristiques du SFN chez les enfants et est plus informatif que le score rapporté par le parent, en particulier lorsqu'il est utilisé en conjonction avec un rappel alimentaire.

Introduction

The Night Eating Syndrome (NES) (Stunkard, Grace et al. 1955a) is a circadian delay in food intake. The diagnostic criteria for NES have recently been standardized (Allison, Lundgren et al. 2010b) and include evening hyperphagia (EH) (>25% total daily calories after the evening meal) and/or nocturnal ingestions (NI) (> 3 per week) with additional clinical features such as insomnia, morning anorexia (MA), increased evening cravings and depressed mood. NES diagnostic criteria can be measured using the Night Eating Questionnaire (NEQ). This research tool measures the severity of NES symptomatology and has been validated in adults (Allison, Lundgren et al. 2008b).

To date, few studies have investigated NES in children. All extant studies focus on excessive evening intake or NI and have not measured the full NES diagnostic criteria (Striegel-Moore, Thompson et al. 2004b; Lamerz, Kuepper-Nybelen et al. 2005b). Using the NEQ in children may better identify NES symptoms, as this instrument measures the syndrome in its entirety. To our knowledge, no study has reported data using the NEQ in children. Studying NES in children is important, as the age of onset of the syndrome and its development over time are currently unexplored areas. Additionally, as the NEQ is used in clinical contexts for treating obesity and other eating disorders, it is important to assess its utility in a child population with the growing concern of child obesity.

Although the prevalence of circadian delayed eating behaviours may be very low in young children, the accuracy of the NEQ can be tested and responses can be compared to child NES symptoms described by the parent. The purpose of this study, therefore, is to measure the validity of the NEQ in children and to identify the most appropriate tool to measure NES symptoms in children.

Methods

Three hundred and four children participated in this study. Participants were a sub-sample of cycle 1 of the QUALITY (Québec Adipose and Lifestyle InvesTigation in Youth) study - a longitudinal investigation of children at risk for obesity. This cohort has been described elsewhere (Lambert, Van Hulst et al. 2011). All parents provided signed informed consent. The ethics committees of Laval University and Centre Hospitalier Universitaire Sainte-Justine approved this study.

Child night eating symptoms were measured by an interviewer-administered NEQ (Allison, Lundgren et al. 2008a) to the child by telephone (*Child NEQ*) and by a parent report of child NES symptoms (*parent NEQ report*, Appendix A). Dietary data were measured by three 24-hour dietary recalls administered to the child using the *Multiple Pass Method* (Moss 2001). Data were compiled and transformed using the CANDAT software (Godin London On. 2007) and the 2007b Canadian Nutrient File (Canadian Nutrient File 2007).

Statistical analyses were performed using JMP 7.0 statistical software (SAS, Cary, NC). Internal consistency of both child and parent questionnaires were measured with standardized Cronbach's alpha. The convergent validity of the NEQ was evaluated by comparing the NEQ total scores and item responses with EH and MA as per the dietary recall. EH and MA were identified by the relative energy intakes of the evening snack and of the breakfast, respectively.

For agreement analyses, the energy content of the evening snack was transformed into percentage of total caloric intake: 0%, 1-25%, >26%. Breakfast consumption on all 3 dietary recalls was coded as having consumed breakfast as was consuming breakfast before 09h00 on the child NEQ and parent NEQ report. Spearman ρ correlation coefficients were used to examine all continuous relationships and were adjusted for age, BMI and sex of the child. Percent agreements with kappa coefficients were used to quantify the concordance between the child NEQ and parent NEQ report as well as between the questionnaires and the dietary recall.

Results

Three hundred and four families completed the NEQ (child NEQ and parent report) (child mean age 9.5 ± 1.0, BMI 19.1± 3.9; %F = 46). The median child NEQ score was 8 (IQR 4) with a range of 0 – 21. No child met the full diagnostic criteria for NES (Allison, Lundgren et al. 2010b). The internal consistencies of the child NEQ and parent NEQ report were 0.54 and 0.35, respectively. The prevalence of NES defined EH (> 25% total intake) was 0.3% (n = 1) as per the Child NEQ, 5.6% (n = 17) as per the dietary recall and 1.3% (n = 4) as per the parent NEQ report. The prevalence of NI was 5.1% (n = 9) as per the Child NEQ and 2.1% (n = 3) as per the parents.

The convergent validity of the child NEQ and the parent NEQ report are found in 3.1. There was a positive association between the child NEQ total score and the mean proportion of calories eaten after the evening meal. Questions pertaining to evening cravings, EH, and

evening-worsening mood were most responsible for this relationship (ρ = 0.11 – 0.18, *p* = 0.07 - 0.002). Together, these questions represented 29% of the variance in evening intake. There was a negative association between the relative breakfast intake and the child NEQ scores. The question pertaining to an evening-worsening mood was most responsible for this relationship (ρ = -0.13, *p* = 0.03). No significant relationships were observed for the parent NEQ report.

Table 3.1. Spearman ρ correlation coefficients for relationship between child NEQ and parent NEQ report and NES dietary manifestations in children

Dietary Recall (*n* = 304)	Child NEQ				Parent NEQ report			
	Total Score		Question response[1]		Total Score		Question response[1]	
	ρ	*p*	ρ	*p*	ρ	*p*	ρ	*p*
Intake after evening meal, % mean total daily kcal	0.20	0.0004	0.10	0.08	0.09	0.14	0.09	0.14
Morning consumption, % mean total daily kcal	-0.12	0.04	-0.13	0.02	0.02	0.72	n/a	n/a

Adjusted for sex, age, BMI.

[1]see question 2 Appendix A for evening intake and methods for morning consumption

n/a not measured in parent NEQ report

Table 3.2 shows the percent agreement between the child NEQ and parent NEQ report as well as between the questionnaires and the dietary recall. Overall, percent agreement between the questionnaires and the dietary recall were low. Percent agreement for EH between the two NEQ questionnaires was good with a "fair" kappa coefficient. There was high agreement for NI frequency and breakfast timing but response variability was low. Agreement was lowest concerning sleep onset and maintenance insomnia. Child NEQ total scores were significantly correlated with total scores from the parent NEQ report (ρ = 0.32, *p* < 0.0001).

Table 3.2. Concordance (percent agreement) between NEQ questionnaires (child and parent report) and between the NEQ questionnaires and the dietary recall (n = 304)

	Agreement (%)		Kappa coefficient	
	Child NEQ[3]	Parent NEQ report[3]	Child NEQ	Parent NEQ report
Evening intake[1]				
0%	21	21.8		
1-25%	38.2	43.3	0.18	0.29
>25% (EH)	0	0		
Breakfast consumption[2]				
Yes	90.1	91.4	0.02	0.003
No	1.0	0.3		
Parent NEQ report				
Breakfast timing	94.9	-	-0.02	-
Evening hyperphagia	70.0	-	0.37	-
Onset insomnia	47.0	-	0.16	-
Maintenance insomnia	41.7	-	0.18	-
Nocturnal ingestions	97.2	-	-0.01	-

[1]Percentage of mean total caloric intake of the evening snack as per the dietary recall and NEQ

2As per dietary recall, *yes* indicates reported breakfast on all three recalls and breakfast before 09h00 on NEQ

[3]Corresponding answers on the NEQ questionnaires

Discussion

No studies have reported using the NEQ in a young population. The purpose of this study was to compare the NEQ in children to dietary recall and a parent report of child NES symptoms. The child NEQ total score is related to greater evening and reduced morning intake, albeit no children expressed full NES criteria. The child NEQ scores reflect the scores of the parent NEQ report with the greatest disagreement pertaining to sleep troubles. The total score of the child NEQ is informative when measuring night eating symptoms in children and should be used together with dietary data to confirm EH, as suggested for adults by Allison et al. (2008b).

Internal consistencies of both the child NEQ and the parent NEQ report were weak. This was expected as both questionnaires were designed to identify symptoms of NES, not to evaluate higher order constructs. Likely reasons for the low internal consistency are the reduced response variability for certain questions and that no children expressed full NES criteria.

The total child NEQ score, and not that of the parent NEQ report, was associated with a delayed pattern of dietary intake, even in the absence of children reporting full NES criteria. The EH item, however, had low agreement with the dietary recall revealing the concept of "25% of total intake" is difficult for children to understand. Importantly, more items than just the EH item were related to delayed eating. Nonetheless, the dietary recall may be more accurate in describing EH as results obtained from this method are similar to those found in another study. In the present study, 5.6% of the children had reported consuming >25% total energy intake on one occasion whereas 8.9% of 11 year-old girls had reported this eating behaviour on one day (3-day dietary records) (Striegel-Moore, Thompson et al. 2004b). The child NEQ total score was also related to a decreased relative breakfast intake. Only one item however, evening-worsening mood, was responsible for the relationship. Accordingly, in adults MA was a low discriminator of NES when analyzed by item response theory analysis (Allison, Engel et al. 2008). The agreement for breakfast consumption between the questionnaires and the dietary recall were moderate to high. The utility of this measure is questionable in children, as parents often require their child to eat breakfast regardless of their level of morning appetite. Regarding NI, the present findings are similar to a study where 1.1% of parents reported their 6-year old child ate at night (Lamerz, Kuepper-Nybelen et al. 2005b). Unfortunately, this item could not be compared to dietary recall because the nutritionists did not prompt for nocturnal ingestions. Taken together, the additional items in the child NEQ, compared to the parent NEQ report, are

pertinent in identifying a delayed eating pattern and justify the use of the total NEQ score in combination with dietary data to identify this pattern of intake.

The total scores from the child NEQ and parent report were related but the strength of the relationship was not as strong as expected and percent agreement was low regarding insomnia. Agreement tended to be related to response variability in that questions with low response variability had high agreement whereas responses with high variability had lower agreement. It is therefore possible that the children are more aware of their sleeping problems than are their parents. This is consistent with results from studies where eating behaviour and binge eating symptoms were not congruent between parent and child reports (Steinberg, Tanofsky-Kraff et al. 2004), specifically when problems actually existed (Johnson, Grieve et al. 1999). The subjective nature of this question and the disparity between the parent and child report indicate the child NEQ may be more informative for this item.

The main limitation of this study is not being able to validate the NEQ in children in full as the prevalence of delayed eating behaviour is very low. An additional limitation is the inability to assess the validity of NI in this population, as this was not prompted in the dietary recall. Moreover, dietary reporting in children is also not without error yet the multiple pass method has been shown to be the most accurate method of dietary assessment in children (Burrows, Martin et al. 2010).

Conclusion

For the first time, the Night Eating Questionnaire has been used in children. It has demonstrated a range of scores, although less variability than that found in adults. The total score is related to clinical features of NES such as increased proportional evening intake and lower proportional morning consumption. A parent report of the child's night eating behaviour was comparable to that indicated by the children themselves yet dietary recall may best illustrate the evening hyperphagia criterion. Responses related to sleep troubles were in most disagreement between the child NEQ and parent report. Taken together, this study emphasizes the need to use the child NEQ total score with dietary data to measure night eating symptoms in children.

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Chapter 4: Daily physical activity profiles of children with delayed eating behaviours

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Abstract

The night eating syndrome (NES) is a delayed pattern of energy intake. It is unknown if symptoms associated with this syndrome are accompanied by a delayed pattern of physical activity. This study examines the relationship between physical activity patterns and delayed eating behaviours in children. Children from the QUALITY cohort (*n* = 269, 45% female, age: 8 – 11yrs) completed the Night Eating Questionnaire (NEQ), which measures NES symptoms on a continuous scale and identifies single NES symptoms. Daily accelerometer data were transformed into mean counts/wear-time minute for each hour of the day. Children with high NEQ scores had higher levels of daily ($p < 0.001$) and evening physical activity ($p = 0.05$) and reached 75% of their total daily physical activity 20 minutes later than children with low NEQ scores ($p < 0.05$). Excessive evening snacking and a strong urge to eat in the evening/at night were the symptoms most related to these physical activity patterns. Children with delayed eating behaviours had higher levels of physical activity in the late morning and evening and a delayed physical activity pattern compared to children with no/fewer symptoms. Future research is needed to determine if physical activity plays a role in the onset or maintenance night eating.

Résumé

Le syndrome de fringale nocturne (SFN) est caractérisé par un délai du cycle alimentaire. Il n'est pas connu si les symptômes associés à ce syndrome sont aussi accompagnés par un délai du cycle d'activités physiques. Cette étude examine la relation entre le profil d'activités physiques et les symptômes du SFN chez les enfants. Des enfants de la cohorte QUALITY (n = 269, 45% de filles, 8 à 11ans) ont complété le Night Eating Questionnaire (NEQ), qui mesure ce syndrome sur une échelle continue (score global) en plus d'identifier les critères diagnostiques. Les données d'accélérométrie (7 jours) ont été transformées en moyennes de comptes par minute/temps pour chaque heure de la journée. Les enfants avec un score élevé de NEQ avaient un niveau d'activités physiques quotidien plus élevé ($p < 0,001$) de même qu'un niveau d'activités physiques plus élevé en soirée ($p = 0,05$). De plus, ces enfants ont atteint 75% de leur pratique d'activités physiques quotidienne totale environ 20 minutes plus tard que les enfants présentant un score de NEQ plus faible ($p < 0,05$). L'hyperphagie en soirée et une forte envie de manger le soir et la nuit étaient les deux critères les plus associés à ces habitudes d'activités physiques. Les enfants présentant des symptômes du SFN avaient un niveau d'activité physique plus élevé en soirée et un profil d'activités physiques retardé par rapport aux enfants qui avaient moins de symptômes. Des recherches futures sont nécessaires pour déterminer si l'activité physique joue un rôle dans l'apparition ou l'entretien de ce syndrome.

Introduction

Childhood obesity is a major health concern and there is a current need to recognize novel factors implicated in weight gain. The circadian aspect of behaviour is emerging as a potential obesity risk factor (Garaulet, Ordovas et al. 2010). Accordingly, the night eating syndrome (NES) (Stunkard, Grace et al. 1955c) – a syndrome of misaligned eating and sleeping patterns – is associated with obesity (Gallant, Lundgren et al. 2012b).

Compared to controls, individuals with NES report a delayed eating pattern of approximately 1.5 hrs (Goel, Stunkard et al. 2009), such that they start eating later in the day, overeat in the evening (> 25% of total energy intake) and/or wake up at night to eat. Aside from the core symptoms of excessive evening snacking and night eating, other important clinical symptoms of NES were recently specified: morning anorexia, onset and maintenance insomnias, a depressed mood, a strong urge to eat in the evening and into the night and the belief that one must eat in order to fall asleep or resume sleep (Allison, Lundgren et al. 2010b). Recently and for the first time, night eating was examined in a population-based cohort of children (aged 8-10 yrs) (Lundgren, Drapeau et al. 2012) using the Night Eating Questionnaire (NEQ) (Allison, Lundgren et al. 2008a). Although no children in this cohort reported the presence of all NES symptoms (Lundgren, Drapeau et al. 2012), NEQ total scores, a measure of symptom severity, were variable and were associated with a relatively delayed pattern of energy intake, i.e., relatively less energy intake was consumed for breakfast and more after the evening meal (Gallant, Lundgren et al. 2012b).

The causes of late night eating in NES are currently unknown, although biological and behavioural models have been hypothesized (Stunkard, Allison et al. 2009b) including involvement of the circadian system (O'Reardon, Ringel et al. 2004; Goel, Stunkard et al. 2009). Physical activity could be implicated as exercise can entrain the circadian rhythm by producing either a phase advance or a phase delay depending on timing (Mistlberger and Skene 2005; Atkinson, Edwards et al. 2007). Only one study has reported the daily physical activity profile associated with NES, and this was in adults in a 24-hour in-patient study with findings of greater spontaneous physical activity in night eaters (Gluck, Venti et al. 2011). It remains to be tested if physical activity patterns are different in individuals with a delayed pattern of energy intake or with specific delayed eating behaviours. The aim of this study was to investigate the relationship between circadian physical activity patterns and delayed eating behaviours in children at risk of obesity.

Methods

Quality Study

Data were drawn from cycle 1 of the QUALITY study (Québec Adipose and Lifestyle InvesTigation in Youth) – a prospective cohort of 630 families that investigates obesity risk factors in children (Lambert, Van Hulst et al. 2011). Families were recruited through schools and eligibility symptoms consisted of a Caucasian child aged 8-10 years with at least one obese biological parent based on BMI (≥ 30 kg/m^2) or waist circumference (>102 cm men, >88cm women). Body weight and height were measured during a clinical testing day. Socioeconomic status was determined by reported family income. All parents signed informed consent for their child and the ethics committees of Laval University and the Centre Hospitalier Universitaire de Sainte-Justine approved this study.

Physical Activity

Children's physical activity was measured using 7-day accelerometry (Actigraph LS 7164 activity monitor, Actigraph LLC, Pensacola, FL US) during the week following the baseline clinical testing day. The Actigraph is a waist-mounted, uniaxial accelerometer that detects vertical accelerations (magnitude and frequency) and was set at 1-minute movement epochs for the current study. A valid day was defined as ≥ 10 hours of recorded activity time per day (Troiano, Berrigan et al. 2008). Children were excluded from the analysis if they had < 4 valid days (Colley, Garriguet et al. 2011). Non-wear time was identified by ≥ 60 minutes of consecutive zeros with 1 or 2 consecutive interruptions (1 - 2 minutes) with activity counts > 0 but ≤ 100 counts per minute (cpm) (Troiano, Berrigan et al. 2008). Non-wear time was subtracted from wear time. Hourly counts (i.e., 12:00 – 12:59) were divided by hourly wear time (after non-wear time was removed) to give hourly counts/min for each hour of the day, for all 7 days. Mean counts per minute for each hour were calculated from available valid days (hour "09:00" represents activity from 09:00 to 09:59). Total daily physical activity levels were estimated with the equations developed by Evenson and colleagues (Evenson, Catellier et al. 2008), as recommended for the Actigraph (Trost, Loprinzi et al. 2011), where sedentary activity < 100cpm, light activity >100 but < 2296 cpm, moderate activity ≥ 2296 but > 4012 cpm and vigorous activity ≥ 4012 cpm. The Actigraph has been validated in children with a sensitivity of 77and 68% for moderate and vigorous physical activity, respectively (Evenson, Catellier et al. 2008). Nocturnal rest phase, the time in hours at night when the accelerometer was inactive, was estimated by subtracting accelerometer start time (the time of the first activity counts (> 50

cpm) from the previous night's accelerometer stop time (time of the last daily activity counts (< 50 cpm) for each day (except for the first day). Data on physical activity patterns were presented in absolute (count/min) and relative terms (% physical activity counts per wear time/ 4-hour period). For each day, the child self-reported their participation in swimming, cycling or diving, as these activities would not be captured by the Actigraph accelerometer. In addition, the child reported the number of organized sports teams of which he or she was a member.

Delayed eating behaviours

Delayed eating behaviours were assessed with the Night Eating Questionnaire (NEQ). This questionnaire, which has been validated in children (Gallant, Lundgren et al. 2012a), was administered, in 2008-9, to children over the telephone by trained interviewers. The NEQ measures night eating symptoms through 14 close-ended questions, 5-point Likert scales, with 2 items related to morning appetite, 2 items related to depressive symptoms, 2 items related to sleep troubles, 3 items related to excessive evening eating,4 items related to night eating behaviour. The NEQ score is the sum of these 14 items (maximum 52). As suggested in adults (Allison, Lundgren et al. 2008a), the question pertaining to excessive evening snacking (i.e., consuming ≥ 25% calories after the evening meal) was adjusted by using data from multiple-pass dietary recall (described elsewhere (Gallant, Lundgren et al. 2012a)). As previously reported, no children in this cohort met the criteria for NES (Lundgren, Drapeau et al. 2012), but the NEQ total score ranged from 0 – 21 (maximum possible score 52). For analysis, children were categorized by a median split of the NEQ score (high NEQ: 8 – 21, low NEQ: 0 – 8, mutually exclusive groups).

The presence or absence of each NES symptom was identified by item frequency/severity responses on the NEQ as follows: excessive evening snacking ≥ 25% total energy intake after the evening meal, night eating ≥ sometimes waking to eat in the middle of the night, morning anorexia = no morning appetite at all or eat for the first time after noon, a strong urge to eat in the evening or during the night ≥ often/very much, the belief that one must eat in order to sleep ≥ somewhat, sleep troubles ≥ usually have trouble getting to sleep and/or wake up at night more than 1/week, depressed mood ≥ feeling somewhat blue and/or mood worsens in the evening or at night.

Statistical analysis

Repeated measures MANOVA controlling for nocturnal rest phase was used to test if physical activity patterns differed between high NEQ children and low NEQ children and between children with or without each NES symptoms. Post-hoc Student's t-tests were used to determine the hours that physical activity counts were statistically different between groups. The relative (%) distribution of physical activity between groups was assessed by grouping physical activity counts into 4-hour blocks covering relevant daily periods (morning, afternoon, early and late evening). ANCOVA was used to test for significant differences between the proportions of total physical activity that was undertaken during each of these periods while controlling for nocturnal rest phase (hrs).

Results

Forty-six children were removed because they did not have four valid days of accelerometer time (≥10 hours), leaving 269 children for analysis. There were no statistically significant differences in mean age, mean BMI, sex distribution or NEQ scores between children with and without four valid days (data not shown). There were no differences in the reported occurrence of swimming, diving or cycling between low and high NEQ score groups.

Children with high NEQ scores had statistically higher counts per minute compared to children with low NEQ scores. Consequently, children with high NEQ scores had higher daily amounts of vigorous and moderate, and to a lesser extent, light physical activity (Table 4.1). This difference remained statistically significant after controlling for nocturnal rest phase (hrs).

Repeated measures analyses revealed different patterns of daily physical activity between children with high and low NEQ scores; children with high NEQ score had higher cpm during the afternoon and evening (F (18, 248) = 1.64, p = 0.05, Figure 4.1). Controlling for BMI, socioeconomic status, mean activity morning start time and total activity duration time did not affect the results (data not shown).

Table 4.1. Participant characteristics.

Characteristic	Low NEQ (0 – 8) *n* = 133	High NEQ (8 – 21) *n* = 136
Age, yrs	9.57 (0.97)	9.48 (0.98)
BMI, kg/m^2	19.4 (3.9)	19.0 (4.1)
Female, %	47%	46%
Obese, %	21%	17%
NEQ score	6 (3)	10 (3)
Activity start time	07:40 (0.65)	07:56 (0.78)*
Activity stop time	21:00 (0.82)	21:00 (0.83)
Nocturnal rest phase†, hrs	10.4 (0.77)	10.5 (0.76)
Counts per wear-time, counts/min	554.90 (152.9)	629.18 (209.7)**
Daily activity duration, hrs	13.6 (0.78)	13.5 (0.76)
Sedentary time, min	356.45 (81.6)	342.93 (84.9)
Light activity time, min	376.55 (67.4)	398.42 (60.9)**
Moderate activity time, min	34.09 (15.8)	38.29 (18.1)*
Vigorous activity time, min	11.60 (8.85)	15.67 (14.0)**

All physical activity measures are 7-day means (SD); NEQ score median (IQR)

†Nocturnal rest phase: duration (hrs) of accelerometer inactivity during the night

Low and High NEQ groups are significantly, as noted, * $p < 0.05$ ** $p < 0.01$

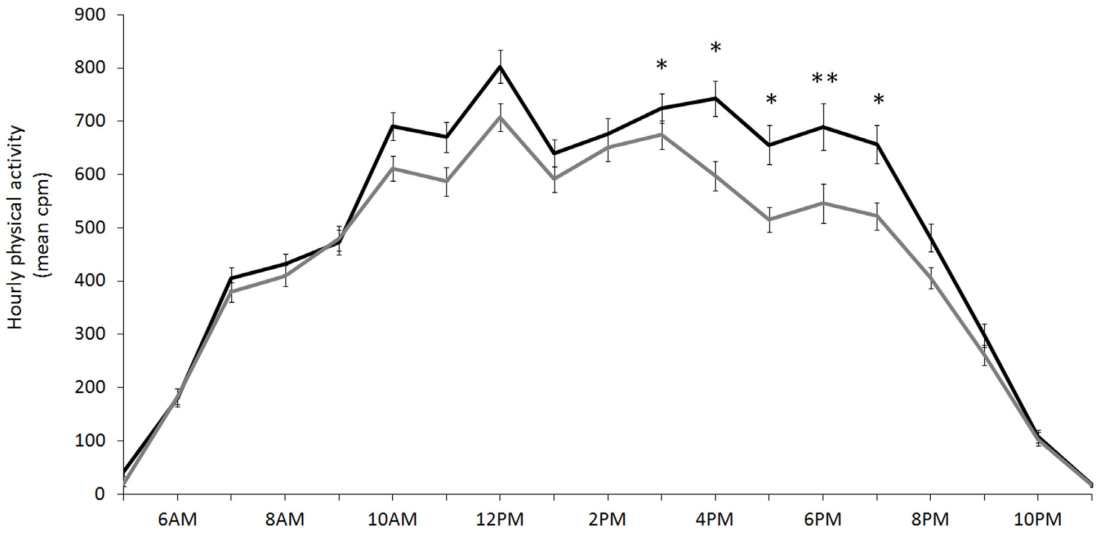

Figure 4.1. Mean (7-d) counts-per-minute (cpm) in children with high (black, n = 136) and low (grey, n = 133) NEQ scores (median split). Repeated measures MANOVA significant group by time effect, F (18, 248) = 1.64, p = 0.05. Posteriori analyses: *p < 0.05, **p < 0.01.

The proportion of total physical activity that occurred in the morning was higher in children with low NEQ scores compared to children with high NEQ scores (17.8 *vs*. 16.2%, p = 0.01). Conversely, the proportion of total physical activity in the evening was higher in children with high NEQ scores compared to children with low NEQ scores (17.0 *vs.* 15.6%, p = 0.04). Results remained statistically significant after controlling for nocturnal rest phase (hrs). Children with a high NEQ score attained 75% of their total physical activity 20 minutes later than children with a low NEQ score (5:33PM vs. 5:52PM, Figure 4.2).

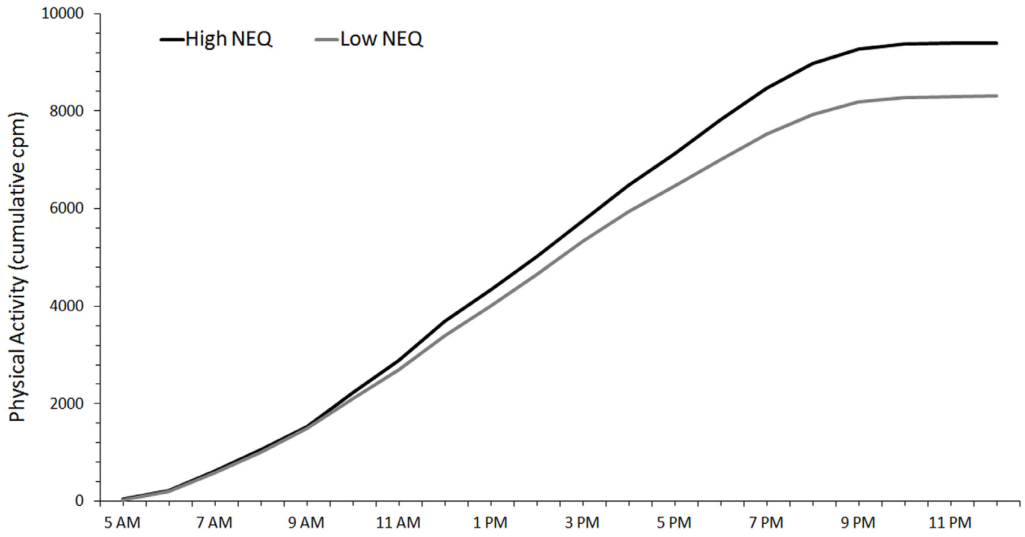

Figure 4.2. Cumulative mean physical activity counts per minute (cpm) for children with a high (n = 136, grey line) and low (n = 133, grey line) NEQ scores. Children with a high NEQ score reached 75% of their total daily physical activity approximately 20 minutes later than children with a high NEQ score (5:52 PM vs. 5:33 PM).

Different physical activity patterns were observed among children reporting core NES symptoms including excessive evening snacking ($F(18, 248) = 1.72$, $p = 0.04$) and night eating ($F(18, 248) = 2.22$, $p = 0.004$), compared to children not reporting these symptoms (Figure 4.3). One child reported a need to eat in order to fall asleep and this child's physical activity profile was also statistically different from the other children who did not report this symptom (F (18, 248) = 4.80, $p < 0.0001$). Children who reported a strong urge to eat in the evening or at night had higher physical activity counts throughout the day compared to children who did not report this symptom (group effect: F (1, 265) = 4.33, $p = 0.04$), but the patterns were not significantly different (group by time effect: F (18, 248) = 1.15, $p = 0.30$). Repeating analyses without this child did not affect the other results. No other NES symptoms were related to physical activity patterns.

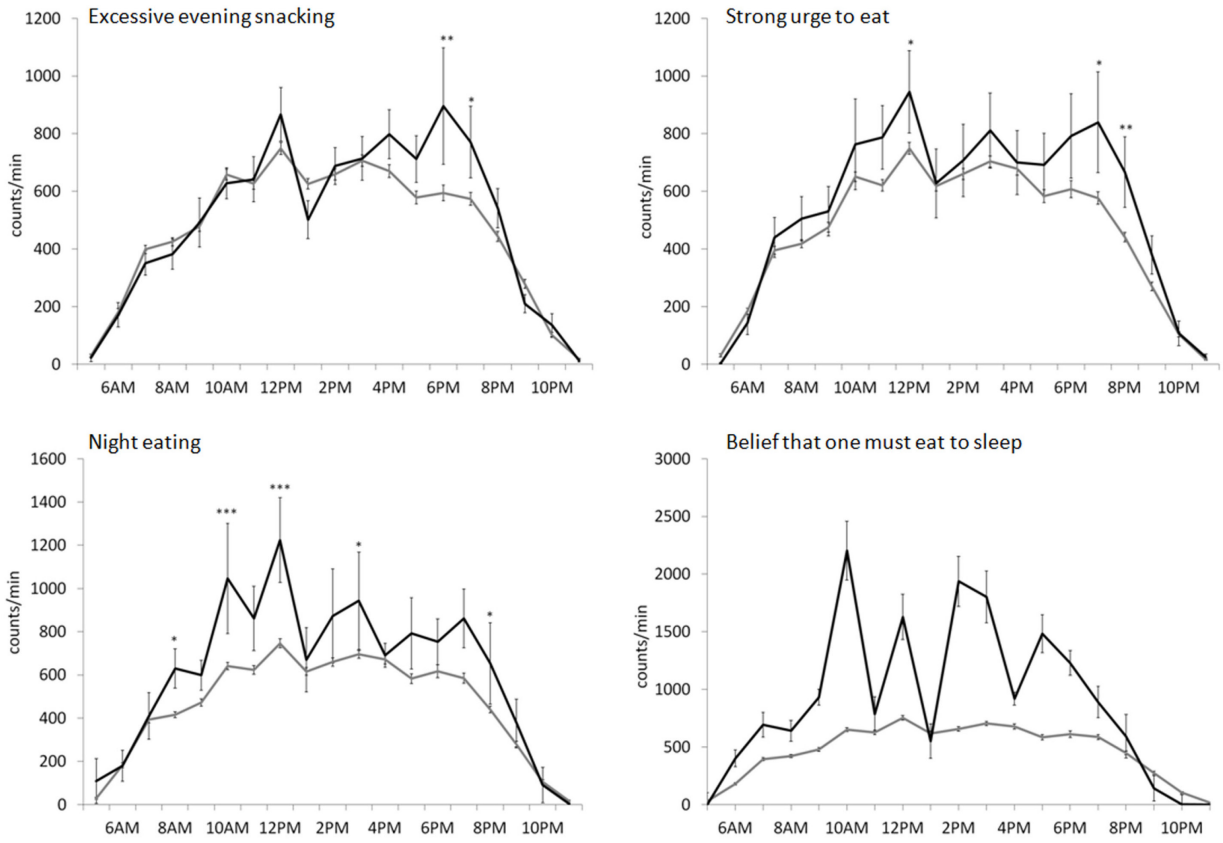

Figure 4.3. Average physical activity counts per minute (cpm) for each hour for children with and without delayed eating behaviours, clockwise from top-left: 1. excessive evening snacking (black line ≥ 25% total caloric intake after the evening meal (*n* = 18), gray line < 25% (*n* = 251)), group by time effect: $F(18, 248) = 1.72$, $p = 0.04$; 2. a strong urge to eat in the evening or at night (black line ≥ often/somewhat strong cravings/urges to eat after supper or at night (*n* = 12), grey line = not at all/ a little (*n* = 257)), group effect: $F(1, 265) = 4.33$, $p = 0.04$, group by time effect, NS; 3. night eating (black line ≥ sometimes waking at night to eat (*n* = 6), grey line = no night eating (*n* = 263)), group by time effect: $F(18, 248) = 2.22$, $p = 0.004$; belief that one must eat in order to fall asleep (black line ≥ somewhat a belief one must eat in order to fall back asleep (*n* = 1), grey line = no such belief (*n* = 268)), group by time effect: $F(18, 248) = 4.80$, $p < 0.0001$.

Discussion

This study aimed to investigate the relationship between daily physical activity patterns and delayed eating behaviours in children at risk of obesity. Results show that children with delayed eating behaviours, measured by the NEQ, have higher levels of total physical activity that is accentuated during the early evening compared to children with fewer symptoms. Moreover, children with high NEQ scores present a delayed pattern of physical activity compared to children with low NEQ scores. Core delayed eating behaviours (i.e., the presence of excessive evening snacking or night eating) were the NES symptoms most associated with accentuated physical activity patterns in the evening.

Our results are consistent with those of Gluck and colleagues (Gluck, Venti et al. 2011) who reported higher 24-h spontaneous physical activity in individuals who ate at night. The authors hypothesized that the higher nocturnal activity in night eaters may indicate restless sleep and increased arousals, and thus a potential circadian problem. Similarly, results from this study indicate a relative delay in physical activity that is associated with delayed eating behaviours; children with a high NEQ score attained 75% of their total physical activity 20 minutes later than children with a low NEQ score (5:33PM vs. 5:52PM).

Whether this delay has clinical significance is uncertain; unlike in a previous study (Ogbagaber, Albert et al. 2012), BMI was not implicated in the delay in physical activity observed in this study (results persisted after controlling for BMI and there were not significance BMI differences between NEQ groups). The present results in combination with the previous report that children with high NEQ scores show a delayed pattern of energy intake (Gallant, Lundgren et al. 2012a), demonstrate a relative delayed behaviour profile in children with delayed eating behaviours. None of these children had NES and the delays are presented herein are small. The implication of physical activity in night eating merits further examination, particularly among individuals diagnosed with NES since the impairment in the circadian system may increase the risk of obesity (Garaulet, Ordovas et al. 2010).

Although the delay in physical activity is short (~ 20 min), it could theoretically be involved in delayed eating behaviours. Exercise is a zeitgeber, i.e., it can synchronize the circadian rhythm and can create a circadian phase shift (Buxton, Lee et al. 2003; Mistlberger and Skene 2005; Atkinson, Edwards et al. 2007). The intensity, duration and timing of physical activity necessary to generate a circadian phase shift are not known. However, both long, low-

intense activity and short, high-intense activity can have similar effects on phase shifts (Atkinson, Edwards et al. 2007). In fact, cycling at 70% VO_2max for 30 min creates a phase shift of 1-h (Edwards, Waterhouse et al. 2002). In the present study, it is uncertain if the amount of evening activity observed in children with delayed eating behaviours is enough to produce a zeitgeber effect since the evening physical activity was light (data not shown) (Evenson, Catellier et al. 2008). There are no data on the circadian effect of evening exercise (Atkinson, Edwards et al. 2007) and no studies examined the impact of habitual daily physical activity patterns on circadian rhythm. The latter is important if effect cumulates over time such that habitual patterns over days (or longer) may have a larger effect on phase-shifting than the effects of short-term interventions (Baehr, Eastman et al. 2003).

Another hypothesis is that higher evening physical activity in children with delayed eating behaviours may increase hunger sensations during this time and thus promote energy intake in the evening. That NES symptoms related to eating are associated with this activity pattern, and not the symptoms related to sleep or mood, also support this. However, it is important to note that there is also large variability in dietary compensatory responses to physical activity (King, Horner et al. 2012) and a recent review concluded that, although there is some connection between energy intake and expenditure in children, the former is not dependant on the latter (Thivel, Aucouturier et al. 2012). Nonetheless, if increased evening activity leads to increased hunger at night, this pattern could lead to more severe delayed eating behaviours.

This study has several limitations. The data are cross sectional limiting causal inference. Moreover, no children met all research diagnostic criteria and thus are not representative of individuals who report the full syndrome; however, as in other eating disorders such as binge eating disorder, sub-threshold symptoms are a risk factor for developing the full syndrome later in life (Stice, Marti et al. 2012). Prospective studies will allow us to understand this relationship better, but for now, there is value in examining the association between the presence of delayed eating behaviours and physical activity patterns. Moreover, data regarding the time of the evening meal was not available and it is not known if children who reported excessive evening snacking ate their last meal at an earlier time than children who did not report this behaviour. Data regarding sleep duration was also not available and had this variable had to be estimated from the accelerometer start and stop times (nocturnal rest phase). Finally, additional measures of the timing of circadian rhythms, such as melatonin levels, would support these associations and would better quantify circadian phase differences.

In conclusion, children with delayed eating behaviours, in particular excessive evening snacking and night eating, have higher overall physical activity that is accentuated in the late morning and evening, which translates into delayed physical activity patterns compared to children without symptoms.

Competing interests

'The author(s) declare that they have no competing interests'

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Chapter 5: Nutritional aspects of late and night eating

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Abstract

The timing of food intake has been investigated as a novel factor in the etiology, maintenance and treatment of obesity. Indeed, consuming a large proportion of food later in the day and into the night has been associated with higher body weight and may even impair weight loss. The diet quality of late-eaters may be a factor involved in these relationships. Moreover, the nutritional characteristics of the foods consumed during the night may negatively affect metabolic and circadian rhythms that are required for optimal health. This review will first examine the diet quality of late-eaters and describe common foods consumed as nocturnal snacks. Second, this review will briefly acknowledge the potential adverse metabolic and circadian effects of consuming certain foods very late in the evening or during the night.

Résumé

Le moment de la prise alimentaire semble un nouvel élément dans l'étiologie, la maintenance et le traitement de l'obésité. En effet, la consommation d'une grande proportion de nourriture plus tard dans la journée et dans la nuit a été associée à un poids corporel plus élevé et pourrait même nuire à la perte de poids. La qualité du régime des individus qui mangent en soirée et durant la nuit peut être un facteur impliqué dans ces relations. En outre, les caractéristiques nutritionnelles des aliments consommés au cours de la nuit peuvent influencer négativement les rythmes métaboliques et circadiens, des processus qui ont un rôle important à jouer dans le maintien d'un bon état de santé. Le but de cet article sera d'abord d'examiner la qualité de la diète des individus qui consomment une grande proportion de leur apport calorique totale en soirée et pendant la nuit (late-eaters) et de décrire le type d'aliments consommés pendant les fringales nocturnes. Par la suite, les effets métaboliques et circadiens potentiellement négatifs associés à la consommation de certains aliments en soirée ou pendant la nuit seront discutés brièvement.

Introduction and context

The temporal distribution of daily food intake tends to be culturally determined; however, epidemiological reports demonstrate similarities in the distribution of food intake within similar cultures. For example, Canadians consume 18% of total energy intake (TEI) for breakfast, 24% for lunch, 36% for dinner and 23% outside discrete meals (Garriguet 2007) whereas Americans reported 24%, 37% and 39% for each discrete meal, respectively (Wang, Patterson et al. 2013). Nonetheless, there is variability in the distribution of food intake across daily meals between individuals and studies have begun to investigate the relationship between the former and body weight.

Observational and experimental reports suggest that the temporal distribution of food intake may be a factor implicated in obesity. Briefly a greater proportion of food intake in the evening was associated with greater BMI, an increased risk of overweight/obesity and reduced weight loss, even after controlling for important covariates such as physical activity, TEI and sleep duration (Baron, Reid et al. 2011; Garaulet, Gomez-Abellan et al. 2013; Wang, Patterson et al. 2013). Furthermore, experimental manipulation of the temporal distribution of food intake during a weight loss intervention demonstrated greater weight loss success when a greater proportion of food was consumed earlier in the day compared to later in the day (Jakubowicz, Barnea et al. 2013). These data are supported by the association between obesity and extreme delayed eating patterns, such as is observed in night eating syndrome (NES) (Gallant, Lundgren et al. 2012b). Thus, emerging evidence has associated a delayed temporal distribution of food intake with measures of body weight and body weight control.

The potential mediators of the relationship between a delayed temporal distribution of food intake and obesity are still uncertain. Different nutritional aspects of the diet could be involved; however, diet quality, independently of TEI, has not often been considered. The aim of this review was to examine the diet quality of late-eaters and the foods consumed as nocturnal snacks and to briefly acknowledge the potential adverse metabolic and circadian effects of consuming these foods very late in the evening or during the night.

Diet quality of late- and night-eaters

Observational studies

Reports indicate that there is a general rhythm of macronutrient intake throughout the day. Indeed, morning meals tend to be relatively high in carbohydrate, midday meals tend to be relatively high in protein and evening meals tend to be relatively high in fat (de Castro 1987; Westerterp-Plantenga, MJ et al. 1996). Moreover, energy density seems to be lowest in the morning and highest in evening (de Castro 2004) and energy density predicts TEI (de Castro 2007). With this information, it could be expected that individuals who eat later during the day, may consume foods higher in dietary fat and energy density. Studies have examined the nutritional qualities of the diet in individuals who report a delayed temporal distribution of food intake, i.e., late- or night-eaters.

In one observational study, adult late-sleepers, who were also late-eaters (35% of TEI after 20h00), consumed fewer daily servings of fruit and vegetables (1.9 vs. 3.4) and consumed greater weekly servings of sugar soda (4.5 vs. 1.3), fast food meals (5.2 vs. 3.0) and caffeinated beverages (13.0 vs. 7.3), compared to early-eaters (20% of TEI after 20h00) (Baron, Reid et al. 2011). Despite these differences, TEI and the relative macronutrient contribution to the diets were similar in early- and late -eaters (Baron, Reid et al. 2011). Similarly, relative macronutrient intakes were not different between early- eaters (<33% of TEI between 17h00 – 0h00) and late-eaters (≥33% than <33% of TEI between 17h00 – 00h00) but in contrast with the former study, there were no differences in daily servings of fruit or vegetable across groups (Wang, Patterson et al. 2013). However, eating ≥33% after 17h00 may be a typical eating pattern in this population (US) and not indicative of late-eaters considering a mean of 39% of TEI was consumed for dinner as observed in the US population (Wang, Patterson et al. 2013). This population-based research is too limited to make any firm conclusions regarding the diet quality associated with a delayed temporal distribution of food intake. Thus, examining the diet quality in known late- and night-eaters, such as shiftworkers, evening chronotypes or individual with nigh eating syndrome, may shed more light on this issue.

The case of shiftworkers

Shiftworkers, although not necessarily habitual late-eaters, are a good group to investigate diet quality of late- or night-eaters. Accordingly, individuals working at night are frequently required to consume food at an atypical time, i.e., late in the evening and or during

the night. It is well documented that shiftworkers are at greater risk for health problems, namely metabolic and cardiovascular complications and cancer (Wang, Armstrong et al. 2011). Because of these health implications, the diets of shiftworkers have been extensively investigated. Most reviews have concluded that although there are obvious differences in eating patterns between shiftworkers and regular workers e.g., meal size, meal frequency, meal timing, nevertheless the diet quality in general is not different (Antunes, Levandovski et al. 2010; Lowden, Moreno et al. 2010; Canuto, Garcez et al. 2013).

The case of evening chronotypes

In contrast to shiftworkers, other individuals may be more biologically driven to eat later in the day. This includes individuals characterized as an evening chronotype, an "evening-type". Evening chronotypes are individuals with an innate evening preference and who perform optimally in the evening. This chronotype is identified by validated self-report questionnaires and/or behavioural or physiological rhythms (Horne and Ostberg 1976; Roenneberg, Kuehnle et al. 2007). Several studies have reported associations between an evening preference and late-eating (Garaulet, Gomez-Abellan et al. 2013; Lucassen, Zhao et al. 2013; Reutrakul, Hood et al. 2013). Late-eaters were more likely to be evening chronotypes (Garaulet, Gomez-Abellan et al. 2013) and evening chronotypes consumed 50% more energy after 20h00 than morning-types (Lucassen, Zhao et al. 2013). Moreover, evening chronotypes consumed a greater percent of their TEI for dinner (43% vs. 32%), which was also 2-hours later than morning-types (Reutrakul, Hood et al. 2013). Thus, evening chronotypes represent an interesting phenotype to investigate diet quality of late-eaters.

Several studies have explicitly investigated the diet quality of the evening chronotype (Fleig and Randler 2009; Sato-Mito, Sasaki et al. 2011; Sato-Mito, Shibata et al. 2011; Kanerva, Kronholm et al. 2012). These studies reported less healthy diets among these individuals. Specifically, adolescent evening chronotypes reported greater consumption of fast foods and caffeinated drinks and lower consumption of dairy products compared to morning chronotypes (Fleig and Randler 2009). Also, adult evening chronotypes consumed less fish and fruit and consumed more chocolate and soft-drinks than their morning chronotype peers (Kanerva, Kronholm et al. 2012). Moreover, in this latter study and others, a low score on the circadian preference scale (low score is indicative of an evening chronotype) was associated with a greater relative fat intake and less relative protein and carbohydrate intakes, fewer servings of dairy products and less relative intakes of many vitamins and minerals, e.g. calcium, potassium,

vitamin A, vitamin D (Sato-Mito, Sasaki et al. 2011; Sato-Mito, Shibata et al. 2011; Kanerva, Kronholm et al. 2012). In contrast with these studies, there were no differences in the macronutrient content of the diets between evening and morning chronotypes in two studies (Garaulet, Gomez-Abellan et al. 2013; Lucassen, Zhao et al. 2013); however, diet quality was not the primary outcome of these studies and additional nutritional aspects of the diet were not assessed. Taken together, there is evidence showing that evening chronotypes types may have less healthful diets compared to morning chronotypes.

The case of night eating syndrome (NES)

NES is a pathological example of chronic late- or night-eating. In individuals with NES, late-eating manifests as a delayed circadian distribution of food intake (≥ 25% of TEI after the evening meal) and/or nocturnal ingestions of food (Allison, Lundgren et al. 2010b). Indeed, the circadian rhythm of individuals with NES is delayed by approximately 1.5 hr (Goel, Stunkard et al. 2009) and is accompanied often by disturbed and inefficient sleep, albeit a typical sleep onset and offset (O'Reardon, Ringel et al. 2004; Rogers, Dinges et al. 2006). Moreover, these individuals often report reduced morning appetite (Stunkard, Grace et al. 1955c) and a specific diurnal variation in mood whereby their mood worsens as the day progresses (Birketvedt, Florholmen et al. 1999a). NES is also associated with obesity (Gallant, Lundgren et al. 2012b) and poor diabetic management (Morse, Ciechanowski et al. 2006). NES is uncommon and is only estimated to affect 0.5% to 5.7% of the general population (Rand and Kuldau 1986; Rand, Macgregor et al. 1997; Striegel-Moore, Dohm et al. 2005; Colles, Dixon et al. 2007; Lundgren, Drapeau et al. 2012). Because of this, the disorder is understudied and very few have examined the nutritional quality of the diet or of the foods consumed at night in these chronic late-eaters.

Indeed, data are very limited regarding the diet quality associated with NES or night eating behaviours e.g., nocturnal ingestions, evening hyperphagia. Individuals with evening hyperphagia and/or who eat late at night (anything past 11PM) reported a lower relative daily intake of protein (-0.5%) and a greater daily intake of sodium (+350mg) than individuals without these eating behaviours (Striegel-Moore, Franko et al. 2008). These differences were more pronounced when restricted to very late night eaters whereby these individuals consumed 500mg more of sodium, 0.9% less protein and 33.8mg more of cholesterol than those who did not report this pattern of eating. In contrast, there were no differences in daytime macronutrient intakes between individuals with and without NES (Allison, Ahima et al. 2005) or between individuals with and without nocturnal ingestion (Gluck, Venti et al. 2008a).

Late- and night-eaters: results from Quality cohort

Research from our group has observed that children with night eating symptoms consumed a less healthy diet than children without these symptoms (Table 5.1). We measured night eating symptoms with the Night Eating Questionnaire (NEQ) in children (aged 8 – 10yr) participating in a prospective cohort investigating obesity risk factors in youth (the QUALITY cohort (Lambert, Van Hulst et al. 2012)). The NEQ measures night eating symptom severity on a continuous scale and can identify specific night eating behaviours (Allison, Lundgren et al. 2008a). In addition to the NEQ, three 24-hour diet recalls were administered by telephone and 317 children had both NEQ and dietary data. Important to the context of this review, the NEQ score in children has previously been associated with a delayed meal-related distribution of energy intake (Gallant, Lundgren et al. 2012a). After excluding 143 under-reporters using methods described by Huang et al., (Huang, Roberts et al. 2005) and Noel et al., (Noel, Mattocks et al. 2010), children with higher NEQ scores had a greater percent of fat intake, greater sodium intake, fewer servings of fruits and vegetables (Table 5.1) and a higher occurrence of unhealthy snacks (high sodium, high saturated fat, low fruit and vegetable, etc.) compared to children with lower NEQ scores (Figure 5.1). These results suggest that children with night-eating symptoms, and a delayed meal-related distribution of energy intake, report less healthy eating habits.

Night eating behaviour, such as nocturnal ingestion of food, may also be a behaviour associated with particular unhealthy eating habits. Also from the QUALITY cohort, children who reported nocturnal ingestions of food (n = 9) reported higher total relative intakes of fat (37% vs. 32%, p = 0.009) compared to children without this behaviour. The greater fat intake tended to come from daytime and evening snacks rather than meals.

In contrast to habitual intake, the foods consumed during nocturnal ingestion tend to be consistently rich in carbohydrates, such as breads and cereal product and sweets. In the second wave of testing for QUALITY (2008 – 2010, 953 adults (53% female) and 516 pre-adolescents (10 – 12 years old, 45% female), 88 adults (9%) and 26 children (5%) reported nocturnal ingestion of food. Accordingly, the most frequently reported foods consumed during nightly awakenings were sugary foods and sweets, breads and cereal products and dairy products (Table 5.2). Children also reported a high frequency of fruit consumption that was not observed among adults. There were no differences in food choices between men and women,

except for a tendency for fewer women to report consuming sugary foods and sweets upon awakening (43% vs. 27%).

Table 5.1. Nutritional data regression models (standard least squares) with the dependent variable NEQ score

Dietary characteristics	NEQ score 1st tertile	NEQ score 2nd tertile	NEQ score 3rd tertile	*p*-value for linear trend
NEQ score (range 0 – 21)	4.5 ± 1.5	8.0 ± 0.9	12.3 ± 2.2	-
Age, yrs	9.6 ± 1.0	9.5 ± 1.0	9.6 ± 1.0	0.86
BMI *z* – score	0.6 ± 1.0	0.4 ± 1.0	0.6 ± 1.0	0.31
Energy intake, kcal	1774 ± 348	1861 ± 412	1873 ± 350	0.27
Fat, %	31 ± 4	33 ± 4	35 ± 5	**0.005**
Carbohydrate, %	53 ± 6	53 ± 5	50 ± 7	0.11
Saturated Fat, %	35 ± 4	36 ± 5	37 ± 5	0.78
Protein, %	17 ± 4	16 ± 2	16 ± 3	0.21
Sodium, mg	2468 ± 792	2616 ± 814	2785 ± 639	**0.07**
Calcium, mg	870 ± 225	923 ± 294	932 ± 311	0.79
Fruit and vegetable, servings/day	5.3 ± 2.1	4.8 ± 2.1	4.6 ± 1.7	**0.03**
Dairy products, servings/day	1.8 ± 0.8	2.1 ± 0.9	2.1 ± 1.0	0.43
Grain products, servings/day	4.5 ± 1.5	5.1 ± 1.8	4.7 ± 1.4	0.91
Meat and alternatives, servings/day	2.1 ± 0.8	2.1 ± 0.9	2.3 ± 1.1	0.29
High-sugar beverages, ml/day	108 ± 105	105 ± 139	123 ± 139	0.17
Snacks, no./day	4.6 ± 1.6	4.9 ± 2.3	5.1 ± 2.1	0.14

Data are means ± SD

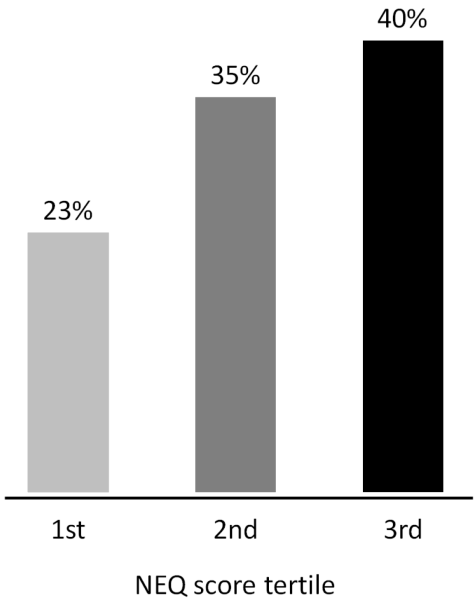

Figure 5.1. Percent of children who reported eating snacks (n = 142) with at least one unhealthy snack, separated by NEQ score tertiles (χ^2 = 3.6, *p* = 0.17). An unhealthy snack was based on the saturated fat and sodium content of the snack and could not be a fruit or vegetable or a dairy product.

Reports from other studies are in agreement with our observations that foods consumed during the night tend to be rich in carbohydrates. This phenomenon is observed in individuals with NES where the carbohydrate content of the nocturnal meal was reported to be 70.3% in one study (Birketvedt, Florholmen et al. 1999a) and 61.5% in another (Gluck, Venti et al. 2008a), values much higher than the carbohydrate component of daytime meals (46.6%). Nocturnal ingestions in sleeping-related eating disorder were also reported to be rich in carbohydrates but were also sometimes foods that were both high-carbohydrate and high-fat (breads, pies, sweets etc.) (Winkelman 1998; Vetrugno, Manconi et al. 2006; Howell, Schenck et al. 2009). Likewise, shiftworkers tended to eat more carbohydrates during the night shift than when they were working the day shift (100g more, 51 vs. 46% of total energy intake) (de Assis, Kupek et al. 2003). A final example is that pre- adolescent girls chose high-carbohydrate foods (53.8 – 68.8% of energy from carbohydrates) when the foods were consumed after 11PM (Striegel-Moore, Thompson et al. 2004a). Taken together, there seems to be a consistent trend of choosing foods rich in carbohydrate, when foods are eaten late in the evening or during the night.

Table 5.2. Percent of adults and children who reported consuming these foods during nocturnal ingestions children (10 – 12 yrs)

	Individuals who reported consuming these foods during nocturnal ingestions, *n* (%[†])	
	Adults (*n* = 88)	Children (*n* = 26)
Sugars and sweet foods	**31 (35)**	**8 (31)**
Fats and fatty foods	6 (7)	1 (4)
Salt and salty foods	6 (7)	1 (4)
Dairy products	**45 (51)**	**12 (46)**
Fruits	13 (15)	**11 (42)**
Vegetables	1 (1)	0 (0)
Meats	2 (2)	0 (0)
Breads and cereal products	**28 (32)**	**13 (50)**
Soft drinks	7 (8)	0 (0)
Chocolate	8 (9)	0 (0)

50% female in adults, no sex differences in food choices

[†]Percent of individuals who reported nocturnal ingestions

Diet quality of late and night eating: metabolic and circadian effects beyond energy value

There may be metabolic consequences of eating certain foods late at night, beyond those related to TEI *per se*. There is evidence of exaggerated glucose and insulin responses to foods eaten during the late evening or night, likely attributable to relative insulin resistance during this time (Hampton, Morgan et al. 1996; Knutsson, Karlsson et al. 2002). More specifically, high carbohydrate foods with a high glycemic index (GI) may further exaggerate the glucose response to the late-evening meal (Morgan, Shi et al. 2012). Indeed, postprandial glucose area under the curve (AUC) was highest after a high GI evening meal compared to a low GI evening meal (Morgan, Shi et al. 2012). Moreover, the relative insulin resistance at night (Van Cauter, Shapiro et al. 1992) may also lead to increased postprandial plasma triglyceride (TAG) concentration by the reduced activation of lipoprotein lipase, a TAG-clearing enzyme. This has been shown in several studies that examined the postprandial response to foods consumed during either a simulated (Hampton, Morgan et al. 1996; Ribeiro, Hampton et al.

1998) or real-life shift-work context (Lund, Arendt et al. 2001). These authors concluded that high-fat meals should also be avoided during the night, at least in the unadapted shift-worker.

Although this suggests eating a certain type of food in the evening or night could compromise metabolic health, other studies have shown different results. The macronutrient composition of a nocturnal meal had no impact on postprandial metabolism in a cross-over study where men were fed a high carbohydrate (65% carbohydrate, 20% fat) and a high fat (40% carbohydrate, 45% fat) nocturnal meal for seven days. Indeed, despite several altered postprandial hormone responses after the nocturnal meals (pancreatic peptide, thyroid-stimulating hormone, cortisol and leptin), no differences were attributed to the macronutrient composition of the diet (Holmbäck, Forslund et al. 2003). Therefore, it seems that eating during the night may produce metabolic consequences that may be worsened by intake of unhealthy, palatable foods (high sugar, high fat).

In addition to the metabolic challenge of a nocturnal meal, certain nutrients may feedback more strongly to the circadian system and entrain (synchronize or desynchronize) the peripheral circadian system, particularly when food is consumed during an atypical circadian phase (Mazzoccoli, Pazienza et al. 2012). Briefly, this atypical feeding pattern may influence rhythms of gene expression in peripheral tissue (and their downstream metabolic and humeral targets) and uncouple these rhythms from the central circadian pacemaker in the brain, the suprachiasmatic nucleus, thus creating internal desynchronization and leading to metabolic disease (Hastings, Reddy et al. 2003). Reviews have elaborated on this topic (Froy 2007; 2010; Garaulet and Madrid 2010; Shibata, Tahara et al. 2010; Cagampang and Bruce 2012; Stenvers, Jonkers et al. 2012; Tahara and Shibata 2013) and the term “chrono-nutrition” has been used to describe the relationships between feeding, nutrition and the circadian system (Tahara and Shibata 2013).

Most evidence on these relationships stems from animal studies and indicates that molecules related to the regulation of glucose metabolism seem to be involved in this phenomenon. Indeed, the insulin signal seems to be an important player in the entrainment of the expression rhythms of clock genes in the peripheral tissues (Tahara and Shibata 2013). However, insulin may not be an *essential* player as high blood glucose levels led to a phase advance in the expression of genes that regulate tissue-specific circadian rhythms (clock genes) in insulin deficient rats and mice (Young, Wilson et al. 2002; Oishi, Kasamatsu et al. 2004).

Accordingly, adenosine monophosphate-activated protein kinase, an enzyme which maintains energy balance, is another potential mediator between the nutrient signals and circadian clocks (Lamia, Sachdeva et al. 2009). Moreover, dietary salt may indirectly influence the circadian entrainment through its effect on glucose absorption (Oike, Nagai et al. 2010; Tahara and Shibata 2013). Chemicals involved in glucose metabolism seem to be important players in the entrainment of peripheral circadian clocks; however, there is still much work needed in this area, particularly among humans.

Nutrients influencing glucose metabolism are predicted to have a strong effect on metabolic and humeral rhythms in humans. In one study, a high carbohydrate meal (75% CHO, 1600 kcal) was served either for breakfast (08:30) or for dinner (21h30). The morning meal impacted the central pacemaker because it phased-advanced the core body temperature (CBT) by 1 hour. In contrast, the evening meal had no effect on the CBT but it either shortened or attenuated the melatonin rhythm (Wirz-Justice, Krauchi et al. 1998; Krauchi, Cajochen et al. 2002). However, it cannot be determined whether this effect was a due to the carbohydrate content of the meal or due to the energy content of the meal *per se.* For example, it is not known if a similar effect would be observed if the meal was high in fat or was balanced in macronutrient content. In a further study, diurnal rhythms of appetite hormones were altered after restricting carbohydrates to the evening while controlling energy intake throughout the rest of the day (Sofer, Eliraz et al. 2013). In accordance with this, altered rhythms of appetite hormones have been observed among individuals with NES. Individuals with NES had attenuated rhythms of cortisol, ghrelin and insulin, phase-delayed rhythms of leptin, insulin and cortisol and phase-advanced rhythms of ghrelin (Goel, Stunkard et al. 2009). Importantly, the leptin-ghrelin rhythms and the glucose-insulin rhythms were uncoupled, which indicates internal physiological desynchronization (Goel, Stunkard et al. 2009). Future studies in humans are needed to determine if specific nutrients can create clinically significant changes in circadian regulation in humans and, if so, the extent of their impact on metabolic health and energy balance. Moreover, it is important to test if nutrients or nutritional strategies can be used to minimize or reverse the effect of circadian desynchronization, a problem thought to occur in individuals who eat at night.

Conclusions and future directions

The literature regarding the temporal distribution of food intake and body weight control is beginning to emerge. However, there are too few studies to make any firm conclusions regarding the diet quality of late- or night-eaters. Most studies have focused on the diet quality of evening chronotypes and results seem to be consistent, i.e., evening chronotypes eat less healthy diets. In contrast, almost no studies have examined the diet quality of individuals with NES, a more severe disorder of late-eating and, consequently, conclusions cannot be made. Nonetheless, individuals who consume food very late in the evening and into the night tend to select high-carbohydrate foods during these eating occasions. Palatable foods, such as foods with a high glycemic index and/or high fat foods, that are eaten during the night may negatively influence postprandial metabolism and may have a negative impact on the peripheral circadian system, creating internal desynchronization. Future research needs to focus on aspects of chrono-nutrition in humans, including the impact of late-eating or night-eating on health, body weight control and the circadian system. This information could help to develop novel nutritional interventions to treat NES or at least minimize the negative metabolic impact of meals eaten late in the evening.

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Chapter 6: Night eating behaviour and metabolic health 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.

Résumé

Contexte: La désynchronisation entre l'alimentation et les habitudes de sommeil et un sommeil de piètre qualité a été associée à l'obésité et des anomalies métaboliques. Cette étude a examiné les associations entre les symptômes du syndrome de fringale nocturne (SFN) et les paramètres métaboliques chez des adultes qui ont participé à l'étude de cohorte QUALITY. Méthodes: Les symptômes du SFN ont été évalués chez 310 femmes (âge = 40,3 ± 5,1 ans, IMC = 28,8 ± 6,2 kg / m^2) et 305 hommes (âge = 42,5 ± 5,9 années, l'IMC = 30,3 ± 5,0 kg / m^2) à l'aide du night eating questionnaire (NEQ). Des mesures anthropométriques, le profil lipidique et la pression artérielle ont été utilisés pour identifier la présence ou non du syndrome métabolique (MetS), tandis que la présence ou non du diabète de type 2 (DT2) a été auto-rapportée. Des corrélations et des comparaisons de cas (SFN/contrôle) ont été utilisées pour évaluer la prévalence des symptômes du SFN chez les individus présentant ou non le MetS ou le DT2. Résultats: Les scores du NEQ étaient positivement corrélés à l'IMC. Les scores NEQ étaient négativement corrélés avec la pression artérielle chez les femmes et positivement corrélés avec le tour de taille et les niveaux de triglycérides sanguins chez les hommes, et ce,

même après ajustement pour l'IMC. Le MetS était associé à l'anorexie matinale chez les hommes et chez les femmes de même qu'à une forte envie de manger seulement chez les femmes. Le DT2 a été associé à une humeur dépressive chez les femmes et à de l'insomnie chez les hommes. Conclusion: Les symptômes du SFN sont associés à un IMC plus élevé et un profil métabolique détérioré. Les recherches futures sont nécessaires pour déterminer si le SFN peut être une cause reliée au développement de l'obésité et des maladies métaboliques.

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 et al. 1955c). 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, Lundgren et al. 2010b). 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, Lundgren et al. 2010b).

Night eating behavior has been associated with weight gain over time (Andersen, Stunkard et al. 2004a), diabetic complications (Morse, Ciechanowski et al. 2006), and individuals seeking weight loss have a higher prevalence of night eating behavior compared to general population samples (Gallant, Lundgren et al. 2012b). 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 hour phase delay), ghrelin (5.2 hour phase advance), glucose (11.6 hour phase advance/ 12.4 hour phase delay), and insulin (2.8 hour delay) (Goel, Stunkard 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.

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 and colleagues

(Turek, Joshu 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 and colleagues (Salgado-Delgado, Angeles-Castellanos et al. 2010) demonstrated that, compared to control rats, rats required to be active for eight hours 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 and colleagues (Wang, Armstrong et al. 2011) reported that rotating and night shift work are associated with an increase in metabolic syndrome risk (odds ratios ranged from 1.46 - 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, Li 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 and colleagues (Scheer, Hilton et al. 2009) forced a 12-hour circadian misalignment in 10 adults. Compared to the baseline phase, the misalignment resulted in an increased mean arterial pressure, an increased post-prandial 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, Zalcman et al. 2007; Knutson, Spiegel et al. 2007; Spiegel, Tasali et al. 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 and Tremblay 2012). This literature is particularly relevant to NES, because although sleep timing is not circadian advanced or delayed (Rogers, Dinges et al. 2006), people with NES often have sleep onset

and/or sleep maintenance insomnia (Allison, Lundgren et al. 2010b) and reduced sleep efficiency (Rogers, Dinges 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, Lundgren et al. 2012b) and persons taking antipsychotic medications (Lundgren, Allison et al. 2006a). Notably, despite the provisional status of NES in the psychiatric nomenclature as an Other Specified Feeding or Eating Disorder (American Psychiatric Association 2013b) 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, Lundgren et al. 2010b) would be associated with obesity, metabolic syndrome and type 2 diabetes in men and women.

Materials and methods

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, Van Hulst 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 > 88cm for women, or 102cm 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 ethics review boards of Laval University and CHU Ste. Justine approved the study.

Assessment

Night Eating Behavior. The Night Eating Questionnaire (NEQ; (Allison, Lundgren et al. 2008a)) 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 that 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 0-52 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 the testing day.

The NEQ can be used as a continuous measure, describing the severity of the NES symptoms (Allison, Lundgren et al. 2008a), or it can be used to assess symptoms of NES based on research diagnostic criteria (Allison, Lundgren et al. 2010b; Lundgren, Drapeau 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, Drapeau 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 feeling blue, the mood is lower in the early evening or late evening/nighttime.

Anthropometric measurements.

Height was measured to the nearest 0.1cm with a stadiometer. Weight was measured to the nearest 0.1kg with an electronic scale and body mass index (BMI) was calculated (kg/m^2). Waist circumference was measured at the middle point between the iliac crest and the lowest rib with participants standing straight up and wearing a hospital gown.

Metabolic Syndrome and Type 2 Diabetes Components.

The metabolic syndrome was classified according to ATP III criteria (Grundy, Cleeman et al. 2005). Type 2 Diabetes was diagnosed with a self-report questionnaire. Blood was obtained by venipuncture after an overnight fast. Glucose, HDL cholesterol, and triglycerides were measured using a Synchron LX®20 chemistry analyzer (Beckman Coulter Brea, CA, USA). Blood pressure was assessed with participants in the sitting position using an oscillometric instrument (Dinamap XL, model CR9340, Critikon Company, Tampa, FL, USA). The average of the last three of five measures (1-min interval) was used in the analyses. Medication use was self-reported and if an individual was medicated for a particular metabolic syndrome component, they were considered positive for that component. Medication use was not considered in the diagnosis of diabetes (self-report of medical diagnosis).

Statistical Analyses

T-tests were used to compare men and women on continuous measures e.g., metabolic parameters, anthropometric values. All analyses were segregated by sex because of significant sex differences in these variables and sex differences in the metabolic syndrome diagnostic criteria (2002) (Table 6.1). Partial correlations, controlling for BMI, were used to examine the statistical relationship between NEQ scores and components of the metabolic syndrome and type 2 diabetes. Logistic regression analyses were used to predict metabolic syndrome and diabetes diagnoses from NEQ scores when controlling for BMI. Because of the low base-rate of night eating syndrome symptoms in this population, Fischer's exact test examined cross-tabulations between metabolic syndrome and diabetes diagnoses and specific NES symptoms. All statistical analyses were performed with using SPSS software (SPSS, Inc., Chicago IL) and statistical significance was set to a $p < 0.05$.

Results

Participant demographic characteristics by sex are presented in Table 6.1. There was significant overlap between diagnoses for diabetes and the metabolic syndrome: 13% of women

with the metabolic syndrome also had diabetes and 77% with diabetes also had the metabolic syndrome. For men, 11% of those with the metabolic syndrome had diabetes and 88% with diabetes also had the metabolic syndrome.

Table 6.1. Participant demographic characteristics by sex.

Variable	Women n = 310	Men n = 305	*p* value
Age (years), mean ± SD	40.3 ± 5.1	42.5 ± 5.9	< 0.001
BMI (kg/m^2), mean ± SD	28.8 ± 6.2	30.3 ± 5.0	< 0.01
Obese, %	39%	51%	0.004
Median household revenue, $/year	$45000	$45000	0.96
Physically active[†],%	72%	73%	0.88
Daily smoker, %	8%	11%	0.15
Daily alcohol consumption, %	2%	6%	0.02
Evening or night shift worker, %	1.3%	1.6%	0.49
NEQ total score, mean ± SD	10.7 ± 4.6	10.9 ± 4.9	0.69
Range (0-52 points)	1-35	3-26	
Type 2 Diabetes, %	4.2%	5.6%	0.45
Metabolic Syndrome, %	24.9%	44.9%	< 0.001
Metabolic Syndrome and Type 2 Diabetes Components, mean ± SD	3.2%	5.0%	0.31
Waist circumference (cm)	92.42 ± 13.97	104.87 ± 13.1	< 0.001
Triglycerides (mmol/L)	1.25 ± 0.71	1.76 ± 1.16	< 0.001
HDL Cholesterol (mmol/L)	1.34 ± 0.32	1.11 ± 0.24	< 0.001
Systolic blood pressure (mm Hg)	105.68 ± 11.30	116.99 ± 12.03	< 0.001
Diastolic blood pressure (mm Hg)	61.99 ± 7.62	69.32 ± 8.68	< 0.001
Fasting glucose (mmol/L)	5.35 ± 0.81	5.74 ± 1.17	< 0.001

[†]Based on self-report and defined as ≥60 minutes of moderate (>3.0 METs) and/or vigorous (>6.0 METs) physical activity per week during the past 3 months and/or a physically strenuous occupation.

Relationship between Night Eating and Metabolic Health in Women

NEQ scores and BMI were significantly correlated (r = 0.29, p <0.001). Significant negative partial correlations, controlling for BMI, were found between the NEQ score and systolic and diastolic blood pressures (Table 6.2). As a follow up, we compared the NEQ scores of women with and without anti-hypertensive medication prescriptions; there were no significant differences between groups (p = 0.63).

Table 6.2. Correlations between NEQ score and metabolic health components when controlling for BMI (kg/m^2).

	Partial correlation with NEQ score r, (p)	
Variable	Women (n = 310)	Men (n = 305)
Waist Circumference	-0.02, (0.63)	0.19, (<0.01)
Blood Pressure		
Systolic	-0.14, (<0.05)	0.02,(0.75)
Diastolic	-0.12, (<0.05)	0.05, (0.40)
Triglycerides	0.00, (0.96)	0.15, (<0.01)
HDL Cholesterol	-0.04, (0.49)	-0.01, (0.91)
Fasting Glucose	0.00, (0.88)	0.0, (0.63)

Logistic regression analyses controlling for BMI revealed that the NEQ score did not significantly predict diagnoses for metabolic syndrome (OR (95% CI) = 0.99 (0.93 – 1.1) or type 2 diabetes (OR (95% CI) = 1.0 (0.94 – 1.2). A significantly higher proportion of women with metabolic syndrome, compared to those without the diagnosis, reported morning anorexia and urges to eat after dinner or in the middle of the night (Table 6.3). Diabetic women were significantly more likely to report depressed mood than non-diabetic women. None of the other NES symptoms differed between groups.

Five women reported night/evening work and an additional 12 women reported rotating shift work. There were no statistically significant differences in the frequency of metabolic syndrome or type 2 diabetes by work category. Similarly there were no statistically significant differences for the mean BMI or NEQ scores by work category, and none of the rotating or evening/night workers reported evening hyperphagia or nocturnal ingestions of food.

Furthermore, when excluding all rotating/evening/night shift workers from the correlation, logistic regression, and cross-tab analyses all significant findings persisted.

Table 6.3. Prevalence of NES Symptoms in Women With and Without Metabolic Syndrome and Type 2 Diabetes.

	Metabolic syndrome			Type 2 Diabetes		
	Yes n (%)	No n (%)	p	Yes n (%)	No n (%)	p
Evening Hyperphagia	0 (0.0)	3 (1.3)	NS	0 (0.0)	3 (1.0)	NS
Nocturnal Ingestions of Food	1 (1.3)	1 (0.4)	NS	1 (7.7)	1 (0.3)	NS
Morning Anorexia	9 (11.7)	5 (2.2)	0.002	1 (7.7)	13 (4.4)	NS
Urge to Eat at Night	21 (27.3)	34 (14.7)	0.01	4 (30.8)	51 (17.2)	NS
Initial or Sleep Maintenance Insomnia	38 (49.4)	108 (46.6)	NS	6 (46.2)	141 (47.5)	NS
Need to Eat to Return to Sleep at Night	1 (1.3)	3 (1.3)	NS	1 (7.7)	3 (1.0)	NS
Depressed Mood	36 (46.8)	91 (39.2)	NS	10 (76.9)	118 (39.7)	0.01

NS = Not statistically significant; NES, night eating syndrome. 13% of women with the metabolic syndrome also had diabetes; 77% with diabetes also had the metabolic syndrome.

Relationship between Night Eating and Metabolic Health in Men

As in women, BMI and NEQ scores were significantly correlated (r = 0.12, p = 0.04). Significant positive partial correlations controlling for BMI were observed between NEQ scores and both waist circumference and triglycerides (Table 6.2). Post-hoc multivariate regression analysis with both measures indicated that waist circumference (F (1,1): 4.0, $p < 0.05$) and blood triglycerides (F (1,1): 7.1, $p < 0.01$) were independently related to NEQ scores.

Logistic regression analyses controlling for BMI revealed that the NEQ score did not significantly predict diagnoses for metabolic syndrome (OR (95% CI) = 1.0 (0.96 – 1.1) or type 2 diabetes (OR (95% CI) = 1.0 (0.91 – 1.1). A significantly higher proportion of men with metabolic syndrome, compared to those without the diagnosis, reported morning anorexia (Table 6.4). Diabetic men, compared to those without the diagnosis, were significantly more likely to report

difficulty initiating or maintaining sleep. None of the other NES symptoms differed between groups.

Table 6.4. Prevalence of NES Symptoms in Men With and Without Metabolic Syndrome and Type 2 Diabetes.

	Metabolic Syndrome			Type 2 Diabetes		
	Yes n (%)	No n (%)	p or NS	Yes n (%)	No n (%)	p or NS
Evening Hyperphagia	3 (2.2)	4 (2.4)	NS	0 (0.0)	7 (2.4)	NS
Nocturnal Ingestions of Food	1 (0.7)	2 (1.2)	NS	1 (5.9)	2 (0.7)	NS.
Morning Anorexia	17 (12.4)	10 (6.0)	0.04	1 (5.9)	26 (9.0)	NS
Urge to Eat at Night	33 (24.1)	31 (18.5)	NS	6 (35.3)	58 (20.1)	NS
Initial or Sleep Maintenance Insomnia	63 (46.0)	62 (36.9)	NS	11 (64.7)	114 (39.6)	0.04
Need to Eat to Return to Sleep at Night	2 (1.5)	3 (1.8)	NS	1 (5.9)	4 (1.4)	NS
Depressed Mood	43 (31.4)	48 (28.6)	NS	5 (29.4)	86 (29.9)	NS

NS = Not statistically significant; NES, night eating syndrome; 11% of men with the metabolic syndrome had diabetes; 88% of men with diabetes also had the metabolic syndrome.

Seven men reported night/evening work and an additional 25 men reported rotating shift work. There were no statistically significant differences in the frequency of metabolic syndrome or type 2 diabetes by work category, and there were no statistically significant differences in the mean BMI or NEQ scores by work category. None of the rotating or evening/night workers reported nocturnal ingestions of food; two rotating shift workers reported evening hyperphagia. When excluding all rotating/evening/night shift workers from the correlation, logistic regression, and cross-tab analyses all significant findings persisted, except for the correlation between NEQ scores and BMI ($r = .11$; $p = 0.11$).

Discussion

This study demonstrated a relationship between components of night eating syndrome and metabolic health. Consistent with previous research, NEQ scores, a measure of symptom

severity, were significantly positively correlated with BMI (Lundgren, Smith et al. 2010). Although NEQ scores did not predict the presence of metabolic syndrome or type 2 diabetes, participants with these metabolic conditions experienced proportionately more symptoms of NES compared to their non-affected peers.

The NEQ score was also associated with markers of metabolic health in the current study, although the relationships differed by sex. When controlling for BMI, higher NEQ scores were associated with lower blood pressure in women and a larger waist circumference and higher triglycerides in men. The negative relationship between the NEQ score and blood pressure (controlling for BMI) among women was unexpected and cannot be explained by the use of anti-hypertension medication. For men, waist circumference and blood triglycerides were related to NEQ score and persisted after controlling for BMI. Because of the small effect sizes, these correlations may not be clinically meaningful but taken together the pattern suggests that circadian misaligned eating and sleeping may contribute to aspects of altered metabolic health in men, although cause cannot be tested in this cross-sectional study.

The patterns of association between NES symptoms and metabolic syndrome and between NES symptoms and type 2 diabetes differed. Although few participants met criteria for evening hyperphagia or nocturnal ingestions of food, the closely related symptoms of morning anorexia (males and females) and the urge to eat at night (females only) were reported more frequently among participants with metabolic syndrome than those without it. The association between morning anorexia and metabolic syndrome strengthened after the removal of individuals with diabetes, likely because the latter group may eat more regularly in the morning as prescribed for management of their diabetes.

The only symptoms of NES that occurred more frequently among participants with vs. without type 2 diabetes were insomnia (males only) and depression (females only). Because insomnia and depressed mood are not unique to NES, these findings do not necessarily suggest that NES per se is detrimental to health. The insomnia findings in particular, however, provide additional evidence that poor quality sleep is associated with impaired glucose regulation (Speigel et al., 2009). Additionally, if the diabetic participants were more regimented in their eating patterns in order to regulate their glucose, it could explain why there was no association between eating patterns and diabetes, but there was such an association found when comparing those with vs. without MetS (i.e., morning anorexia).

The design of the current study does not allow us to explore the mechanisms by which NES symptoms might lead to the development of metabolic syndrome or type 2 diabetes, or vice versa. Fortunately, because of the longitudinal nature of the study we will be able to assess whether individuals with metabolic syndrome and diabetes develop night eating symptoms and/or if those with night eating symptoms at baseline develop metabolic abnormalities or an increase in their severity over time. The current literature on this topic is sparse but indicates that the prevalence of NES is not elevated among diabetics (3.8%) (Allison, Crow et al. 2007) and may (Morse, Ciechanowski et al. 2006) or may not (Allison, Crow et al. 2007) be associated with diabetic complications.

Although this study has strengths, including objectively measured metabolic variables, large sample size, and comprehensive night eating assessment using a validated measure, there are limitations that should be noted. First, although the NEQ has been validated in several samples, it is a self-report symptom measure and some degree of participant under and over reporting of symptoms is expected. Diabetes diagnosis was also self-report but research has shown high agreement between self-report and medical records for diabetes (Okura, Urban et al. 2004). We have no data, however, to suggest that persons with and without metabolic syndrome or type 2 diabetes would be any more or less likely to under/over report on the NEQ or vice versa. Moreover, the overall pattern of results, in addition to those reported by Morse et al., (Morse, Ciechanowski et al. 2006) support that the relationships are accurate and not a reflection of classification methodology. Second, few males and females in this sample reported full threshold NES or the core criteria of evening hyperphagia or nocturnal ingestions of food, and the mean NEQ scores were low (mean = 10 points out of 52 for males and females). Although there was significant variability in NEQ scores in men (range 3-26 points) and women (range 1-35 points), these findings are not necessarily generalizable to those with more severe NES symptoms. Similarly, the average body mass index of participants in this sample was overweight (women = 28 kg/m^2) and minimally obese (men = 30 kg/m^2). More obese samples might show a different pattern of association between night eating symptoms and metabolic health, even when controlling for BMI.

Finally, because the relationship between NES and other sleep, mood, and eating disorders is not fully understood, it is possible that the NES symptoms that are most strongly associated with obesity and poor metabolic health are attributable to other conditions. For example, in the current sample, a greater proportion of males with type 2 diabetes reported

insomnia than males without type 2 diabetes. Similarly, a higher proportion of females with type 2 diabetes reported depressed mood than those without type 2 diabetes. Arguably, however, both insomnia and depressed mood often co-occur or are secondary to other conditions such as life stress. Future studies are necessary to replicate these findings in people who meet full threshold criteria for NES and to compare the health profiles of individuals with NES to individuals who report sleep/mood problems without evening/night eating behavior.

In summary, this is the first study to report the association between night eating syndrome symptoms and objectively measured components of metabolic health in a sample of the general population. Significant associations were found in both men and women between NEQ total score and BMI. Both men and women with metabolic syndrome were more likely than those without metabolic syndrome to report morning anorexia, and for females there was an additional association between food cravings during the evening/night and metabolic syndrome. The pattern of association was different for type 2 diabetes, with insomnia (males) and depressed mood (females) being the only symptoms of NES to co-occur with a diabetes diagnosis. Of note, with the exception of morning anorexia, the pattern of association between night eating symptoms and metabolic health was quite different between males and females. These findings contribute to a growing literature on the relationship among circadian misalignment, poor sleep, and metabolic health. Future research using both human and animal models is necessary to determine the physiological mechanisms by which NES symptoms and metabolic health are associated.

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Chapter 7: Long-term reproducibility of the Night Eating Questionnaire in adults

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Abstract

Aims: To assess the long-term reproducibility of the Night Eating Questionnaire (NEQ) in adults and to describe differences attributed to body weight status. Methods: The NEQ assesses night eating symptom severity and estimates the prevalence of night eating syndrome (NES) diagnostic criteria. The NEQ was administered twice to 517 adults (53% female, 45% obese) participating in the QUALITY study: at baseline (2005 – 2008) and two years later. Intra-class correlation coefficients and kappa coefficients were used to quantify the agreement for the NEQ total score, the individual NEQ items and the NES criteria for the total sample and separated by baseline weight status (BMI < or ≥ 30 kg/m^2). Results: The NEQ score was reproducible for the sample whereby 69% of the variability between assessments was attributed to inter-individual differences. Most NEQ items were also reproducible. Agreement for the NES criteria ranged from fair to moderate and was limited by their low prevalence. The items and criterion related to mood were not reproducible. Generally, reproducibility was stronger in the non-obese group compared to the obese group. Conclusion: The NEQ score and individual items were reproducible in an adult sample without NES. The reproducibilities of the NES criteria were moderate to fair but were limited by the non-clinical population.

Résumé

L'objectif de cette étude était d'évaluer la reproductibilité à long terme du questionnaire sur le syndrome d'hyperphagie nocturne (NEQ) chez les adultes et évaluer l'impact du statut pondéral sur cette reproductibilité. Méthodes: Le night eating questionnaire (NEQ) qui évalue les symptômes du syndrome de fringale nocturne (SFN) a été utilisé pour évaluer la prévalence du SFN et de ses symptômes. Le NEQ a été administré à deux reprise soit à la phase 1 (2005 - 2008) et à la phase 2 (2008-2010) auprès de 517 adultes (53% de femmes, 45% d'obèses) participant à la cohorte QUALITY. Les coefficients de corrélation intra-classe (CCI), la pondération et les coefficients de kappa normalisés ont été utilisés pour quantifier l'adéquation des réponses entre ces deux temps pour le score total du NEQ, les questions individuelles du NEQ et les critères cliniques du SFN. Les analyses ont été effectuées sur l'échantillon total, puis en fonction du statut pondéral (indice de masse corporelle, IMC < ou ≥ 30 kg/m^2). Résultats: Le score de NEQ était reproductible pour l'ensemble de l'échantillon et 69% de la variabilité entre les évaluations a été attribuée à des différences inter-individuelles. La plupart des questions individuelles du NEQ étaient également reproductibles. L'adéquation des réponses entre les deux temps pour les critères cliniques du NES variait de passable à modérée et la reproductibilité a été limitée par la faible prévalence de nombreux critères

cliniques du SFN. Les questions reliées à l'humeur ont démontré la plus faible reproductibilité. En général, la reproductibilité a été plus forte dans le groupe non-obèse, comparativement au groupe obèse. Conclusion: Le score de NEQ et la plupart des questions individuelles sont reproductibles dans un échantillon d'adultes sans SFN. La reproductibilité plus faible des critères cliniques du SFN peut être expliquée par la population étudiée.

Introduction

Night eating syndrome (NES) is a disorder characterized by a circadian delayed pattern of energy intake (evening hyperphagia and nocturnal awakenings with ingestions of food), strong cravings for food in the evening and at night, morning anorexia, insomnia and a depressed mood or evening-worsening mood (Stunkard, Grace et al. 1955c). NES is associated with comorbid psychopathology (Lundgren, Allison et al. 2008a; Vinai, Allison et al. 2008b; Vander Wal 2012) and the delayed eating behaviours consequent to this disorder may be an obstacle for body weight control (Gluck, Geliebter et al. 2001; Andersen, Stunkard et al. 2004a; Gluck, Venti et al. 2008a), diabetic management (Morse, Ciechanowski et al. 2006) and are associated with components of the metabolic syndrome (Gallant, Drapeau et al. 2014).

Estimates of the prevalence of NES in samples of the general population range from 0.5 to 5%, but are largely dependent on the diagnostic criteria or the operational definition, which have not always been uniform (Rand, Macgregor et al. 1997; Andersen, Stunkard et al. 2004a; Striegel-Moore, Franko et al. 2006b; Colles, Dixon et al. 2007; Striegel-Moore, Franko et al. 2008; Lundgren, Drapeau et al. 2010). To advance the research on NES, standardized research diagnostic criteria have been proposed (Allison, Lundgren et al. 2010b). As a result of these efforts, NES is included in the 5th version of the Diagnostic and statistical manual of mental disorders as an other specified feeding or eating disorder (American Psychiatric Association 2013a).

The night eating questionnaire (NEQ) is a research tool that was developed to aid in the diagnosis of NES and to assess NES symptom severity (Allison, Lundgren et al. 2008a). NES symptom severity is assessed by a global NEQ score which represents a higher-order factor of NES symptomatology (Allison, Lundgren et al. 2008a). In a validation study, the total NEQ score was related to percent of daily calories consumed after supper, frequency of nocturnal ingestions, and scales of depression, stress and sleep troubles (Allison, Lundgren et al. 2008a).

The internal consistency of the NEQ score was 0.70 and an NEQ > 25 had a high negative predictive value (95.2%). Furthermore, individual NES criteria can be derived from the NEQ; the latter have been used in large cohorts to estimate the prevalence of NES (Lundgren, Drapeau et al. 2012; Runfola, Allison et al. 2014).

Clinicians who work with individuals who suffer from NES have recognized that symptoms remit and relapse over time but that the disorder is chronic. It is common for patients to report symptoms to persist beyond 5 years (de Zwaan, Roerig et al. 2006) and the mean duration of symptoms has been reported to range from 7.4 to 10.4 years (Spaggiari, Granella et al. 1994; Marshall, Allison et al. 2004; Cleator, Judd et al. 2013).

Despite the chronicity of night eating symptoms, to our knowledge no studies have examined the long-term reproducibility, i.e., stability, of the NEQ or the derived NES criteria. Knowing the degree of reproducibility of the NEQ, defined as the intra-individual variability in self-reported NES symptoms under changing conditions (Bartlett and Frost 2008), could have both methodological and clinical benefits. First, this information will help quantify the stability of night eating symptoms. Second, it will provide information regarding the items of the NEQ that measure emotional and appetite states versus more stable traits and last, it will provide information regarding the ability of this tool to be used to track reported symptoms in both experimental and clinical contexts.

Furthermore, in view of the consistent relationships between night eating symptoms and obesity (Colles and Dixon 2012; Gallant, Lundgren et al. 2012b), it is uncertain if the reproducibility of this questionnaire differs among populations of different body weights. This could be specifically important in the context of tracking self-reported symptoms within the context of a weight-related intervention in order to evaluate the efficacy of a weight loss intervention.

The objective of this study was to assess the two-year reproducibility in NEQ items and score and the derived NES criteria in a sample of adults and to describe differences attributed to baseline weight status.

Methods

Participants

Participants included adults participating in the QUALITY (Québec Adipose and Lifestyle InvesTigation in Youth) study, which is an ongoing prospective cohort study investigating the natural course and risk factors for obesity in children (Lambert, Van Hulst et al. 2011). For the QUALITY cohort, parents and their 8 – 10 year old child were recruited through schools. Eligibility criteria included being Caucasian with at least one obese biological parent based on BMI (≥ 30 kg/m^2) or waist circumference (>102 cm men, >88cm women). Families were required to participate clinical testing days at baseline (2005 – 2008) and two years later at follow-up (2008 – 2011). Participant characteristics were recorded and anthropometric measurements (body weight, height) were made according to standardized procedures. Obesity was defined as a BMI ≥ 30 kg/m^2. Only the parents of this cohort were included in the present study. This multi-centre project was approved by the ethics committees of Laval Hospital (Quebec) and the Centre Hospitalier Universitaire de Sainte-Justine (Montréal). All parents provided informed consent.

Night Eating Symptoms

Adults were auto-administered the Night Eating Questionnaire (NEQ) (Allison, Lundgren et al. 2008a) twice (baseline and two-year follow-up). The NEQ contains 14 items that are assessed on 5-point frequency or severity ordinal likert scale. Thirteen of the items can be summed to give a total score (NEQ score) that is a continuous measure of symptom severity (maximum possible score of 52). One item (item 13) distinguishes nocturnal ingestions related to night eating syndrome from those of sleep-related eating disorders, and this item is not included in the total score. NES research diagnostic criteria were also derived from the NEQ (derived NES criteria). NEQ items were grouped according to specific symptoms and dichotomized according to pre-determined cut-offs to indicate the presence or absence the NES criteria (Table 7.1) (Lundgren, Drapeau et al. 2012; Gallant, Drapeau et al. 2014).

Statistical analysis

T-tests were used to examine differences in baseline characteristics between weight groups. The intra-individual discrepancy and variability in NEQ scores between time points was assessed with paired t-tests, a Bland-Altman plot of the mean versus the difference in scores (Bland and Altman 1986) and Bartlett's test to test for homogeneity of variance across weight

groups. Intra-individual agreement in NEQ scores was quantified by intra-class correlation coefficients (ICC) (Shrout and Fleiss 1979).

Agreement of the NEQ items were quantified with weighted kappa coefficients. Weighted kappas were used to take into account the ordinal scales of the NEQ items (Spitzer, Cohen et al. 1967; Cohen 1968). Quadratic weights were assigned to disagreements that were farther apart, such that going from “never” to “always” would have an exponentially greater weight than going from “never” to “sometimes”.

Normal (unweighted) kappa coefficients assessed agreement for the presence/absence of the NES criteria. The prevalence of certain criteria were low in this cohort which automatically lowers the kappa coefficient and may not represent the actual agreement (Viera and Garrett 2005). For this reason, maximum kappa (κ_{max}) coefficients were also reported to aid with interpretation (Dunn 1989; Sim and Wright 2005). The κ_{max} is the maximum kappa possible for the reported prevalence, i.e., holding the marginal totals of the cross-tabulation constant. Interpretations for the normal kappa coefficients were as follows: ≤ 0 = poor, 0.01 – 0.20 = slight, 0.21 – 0.40 = fair, 0.41 – 0.60 = moderate, 0.61 – 0.80 = substantial and ≥ 0.81 = almost perfect agreement (Landis and Koch 1977). The systematic divergence in NES criteria was assessed by McNemar tests or Wilcoxin signed rank tests for small cell numbers (<5).

Table 7.1. Items severity and frequency cut-offs from the Night Eating Questionnaire used to measure night eating syndrome criteria

Night eating symptoms	Night Eating Questionnaire items	Coded as present
Core symptoms		
Evening hyperphagia	*How much of your daily food intake do you consume after supper?*	≥ 26 – 50%
Nocturnal ingestions (with awareness)	*When you get up in the middle of the night, how often do you snack?*	≥ Half the time
	AND	
	Other than to use the bathroom, how often do you wake up at least once in the middle of the night?	≥ More than once a week
Clinical features		
Morning anorexia	*How hungry are you usually in the morning?*	= Not at all
	AND/OR	
	When do you usually eat for the first time?	≥ 12:01PM
Evening /nighttime food cravings	*Do you have cravings or urges to eat snacks after supper, but before bedtime?*	≥ Very much so
	AND/OR	
	Do you have cravings or urges to eat snacks when you wake up at night?	≥ Very much so
Belief that one must eat to return to sleep†	*Do you need to eat in order to get back to sleep when you awake at night?*	≥ Somewhat
Insomnia	*How often do you have trouble getting to sleep?*	≥ Usually
	AND/OR	
	Other than only to use the bathroom, how often do you get up at least once in the middle of the night?	≥ More than once a week
Depressed mood	*Are you currently feeling blue or down in the dumps?*	≥ Somewhat
	AND/OR	
	When you are feeling blue is your mood lower in the...	Early or late evening/nighttime

Results

Participant characteristics

The sample included 517 adults (53% female). Mean age and BMI at time 1 were 41.6 ± 5.5 yrs and 29.5 ± 5.6 kg/m^2, respectively. Forty-five percent of the sample was obese at time 1. Mean age was not different between the non-obese and obese groups (p = 0.48). The mean BMI of the non-obese group was 25.6 kg/m^2, which was significantly smaller than the mean BMI of the obese group (34.2 kg/m^2, p < 0.0001). Body weight and height was missing for one individual.

The reproducibility of the NEQ score is reported in Table 7.2. Overall, NEQ scores differed by ± 7 units in 95% of the sample (range -13 to 15) and there was no systematic increase or decrease in NEQ scores between the two assessments (p = 0.24, Table 7.2). The Bland-Altman plot demonstrated that there was greater intra-individual variability in scores in individuals who had a greater mean score (Figure 7.1). The ICC for the NEQ score was moderate and intra-individuals variability explained 31% of the total variance in NEQ scores between assessments (Table 7.2).

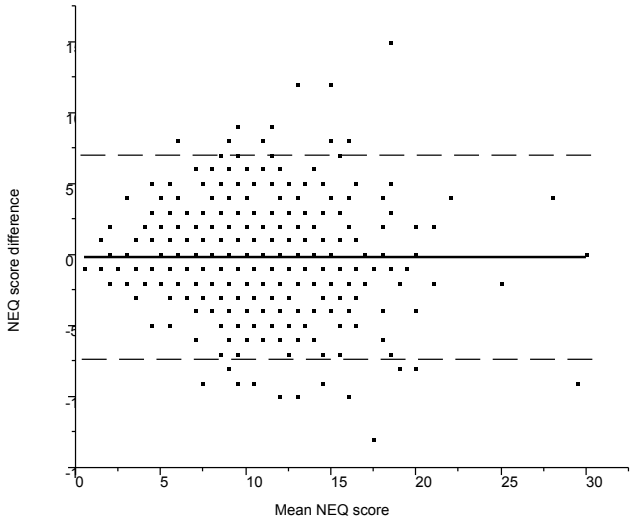

Figure 7.1. The Bland-Altman plot of the intra-individual mean Night Eating Questionnaire (NEQ) score for time 1 (2005 – 2008) and time 2 (2008 – 2011) versus the intra-individual difference between the two scores. Solid line is the overall sample mean and the dashed lines are ± 2 SD associated with the overall mean.

When examined by weight group, NEQ scores were higher in the obese group compared to the health/overweight group at time 1 (t (513) = 5.0, $p < 0.0001$) and time 2 (t (513) = 2.7, p= 0.008). The NEQ score systematically decreased between time 1 and time 2 in the obese group only ($p < 0.006$, Table 7.2). Accordingly, the ICC was strong for the non-obese group and moderate the obese group (Table 7.2) and the intra-individual variability explained 29% and 34% of the total variance in NEQ scores between assessments, respectively. Moreover, the variance of the difference was greater in the obese group compared to the non-obese group (p = 0.005); scores differed by ± 6.4 units in 95% of the non-obese group compared to ± 7.6 units in 95% of the obese group. However, in both weight groups the Bland-Altman plot demonstrated patterns of greater intra-individual variability in individuals who had a greater mean scores (figures not shown).

Table 7.2. Discrepancy, variance and intra-class correlation coefficients (ICC) for Night Eating Questionnaire scores (NEQ) between time 1 (2005 – 2008) and time 2 (2008 – 2010).

	Total sample (n = 517)	Non-obese (n = 283)	Obese (n = 233)
NEQ score, mean ± sd			
Time 1	10.8 ± 4.7	9.9 ± 4.3	11.9 ± 4.8
Time 2	10.6 ± 4.4	10.1 ± 4.2	11.2 ± 4.6†
Mean difference	-0.2	0.25	-0.69
Variance of the difference	12.5	10.2	14.6*
ICC (1,1)	0.69	0.71	0.66

†Significantly lower than time 1, p = 0.006

*Unequal variance compared to BMI < 30, using Bartlett's test for homogeneity at p = 0.005

Reproducibility of NEQ items

Because weighted kappa coefficients are mathematically identical to ICC, they can be interpreted as a correlation. For almost half the items (items 2, 3, 8, 9, 10, 12), at least 60% of the variability in item responses across assessments was attributed to inter-individual variability (Table 7.3). In contrast, items 6 and 7 were the least reproducible and thus 74% of the variability

attributed to intra-individual variability. The only item to systematically diverge across assessments was item 2 where, despite a high reproducibility, the mean score was lower on the second assessment (p = 0.04), 23 individuals reported a later breakfast timing on the second assessment whereas 43 reported an earlier breakfast timing on the second assessment.

Item reproducibility was generally stronger for the non-obese group compared to the obese group (Table 7.3). The non-obese group had stronger agreement for evening cravings for food (item 3), initial insomnia (item 8) and the level of consciousness during nocturnal ingestions (item 13). The items relating to mood had slight agreement for all participants, regardless of weight category.

Reproducibility of NES criteria

Overall, reproducibility ranged from fair to moderate. The kappa for evening hyperphagia was 0.40 (Table 7.4). The low agreement was also corroborated by a systematic divergence in the presence of this behaviour; more individuals reported this behaviour at time 2 compared to time 1 (p = 0.03). However, the low maximum kappa for evening hyperphagia reflected the low prevalence of this behaviour and demonstrated the upper limit of intra-individual agreement that was possible in this sample (normal kappa was 61% of maximum kappa).

Reproducibility for nocturnal ingestions of food was stronger at 0.50. Unlike for evening hyperphagia, the presence of this behaviour did not systematically diverge across assessments but consistent with evening hyperphagia, the maximum kappa was low at 0.75, indicating the true agreement may be greater.

The most reproducible clinical criteria were insomnia and a belief that one must eat in order to sleep (moderate agreement for both). The low maximum kappa for a belief that one must eat to sleep reflects the low prevalence of this criterion (1%). The presence of clinical criteria did not systematically diverge across assessments.

Table 7.3. Weighted kappa coefficients (κ_w) for the agreement between baseline (2008 – 2010) and follow-up (2008 – 2010) Night Eating Questionnaire (NEQ) items in the total sample and by weight status

NEQ item	Total sample (n = 517)	Non-obese (n = 283)	Obese† (n = 233)
	κ_w	κ_w	κ_w
1 – morning appetite	0.59	0.60	0.58
2 – morning meal	0.65	0.66	0.63
3 – evening cravings	0.62	**0.66**	**0.55**
4 – control over eating	0.51	**0.54**	**0.43**
5 – evening hyperphagia	0.55	0.55	0.52
6 – current mood state	0.26	**0.19**	**0.34**
7 – evening worsening mood	0.26	**0.30**	**0.19**
8 – onset insomnia	0.60	**0.65**	**0.54**
9 – maintenance insomnia	0.66	0.68	0.63
10 – awakening hunger	0.61	0.61	0.62
11 – belief must eat to sleep	0.50	**0.54**	**0.44**
12 – night eating frequency	0.67	0.68	0.66
13 – level of consciousness	0.50	**0.58**	**0.41**
14 – control over night eating	0.47	0.47	0.45

Items in bold > 0.10 units difference between non-obese and obese

†Body weight and height missing for one individual

Reproducibility was stronger in the non-obese group for 4/7 NES criteria. Agreement for evening hyperphagia could not be compared across groups because no one in the non-obese group reported this behaviour at time 1. For two components of morning anorexia, skipping breakfast was more reproducible in the obese group (0.65, substantial) than the non-obese

group (0.39, fair). Both weight groups had slight agreement for the lack of morning appetite (non-obese = 0.27 vs. obese = 0.17). For the components of a depressed mood, a current depressed mood was not reproducible in either weight group (kappa of 0) and an evening-worsening mood was more reproducible in the non-obese group (0.30) than the obese group (0.20). There was no systematic divergence in the presence of most criteria in either weight group except for strong evening cravings for food which was less prevalent in the second assessment in the obese group only (31% versus 24%, $p = 0.03$). This could explain the weaker agreement for this criterion in this group.

Table 7.4. Normal kappa coefficients and the maximum kappa possible for the reported prevalence of the NES criteria

	Total Sample (n = 517)		By weight status			
			Non-obese (n = 283)		Obese† (n = 233)	
NES criteria	κ	κ_{max}	κ	κ_{max}	κ	κ_{max}
Evening hyperphagia	0.40	0.66	-	-	0.43	0.71
Nocturnal ingestions	0.50	0.75	0.50	0.50	0.50	1.0
Morning anorexia	0.41	0.98	0.24	0.89	0.54	0.94
Evening food cravings	0.49	0.95	0.51	0.88	0.44	0.83
Must eat to sleep	0.57	0.86	0.50	1.0	0.66	0.66
Insomnia	0.58	0.96	0.61	0.97	0.54	0.95
Depressed mood	0.27	0.99	0.31	0.93	0.22	0.95

κ, kappa coefficient; κ_{max}, maximum kappa possible for the prevalence of the criterion

†Body weight and height missing for one individual

Discussion

The objective of this study was to assess the two-year reproducibility of the Night Eating Questionnaire (NEQ score and items) and the derived NES criteria in a sample of adults and to describe differences attributed to baseline weight status. Overall, night eating symptom severity was reproducible and 70% of the variability in the NEQ score was attributable to inter-individual differences. Most of the NEQ items were also reproducible, particularly the items relating to the frequency of nocturnal ingestions and nocturnal awakenings and the timing of the morning meal. Most derived NES diagnostic criteria had moderate reproducibility. NEQ items or NES criteria related to mood was the least reproducible. Generally, the NEQ and the derived NES criteria were less reproducible for obese participants compared to non-obese participants, which could reflect greater symptom fluctuation across time in this weight group.

This study demonstrated that the NEQ score, a continuous measure of night eating symptom severity, was substantially reproducible over two years in a non-clinical adult sample. Indeed, the inter-individual variability in NEQ scores was greater than intra-individual variability. Thus, the degree of night eating symptom severity seems to be a stable characteristic of an individual. However, NEQ was less reproducible in more symptomatic individuals (as per the Bland-Atman plot). Knowing this, the mild night eating symptomatology observed in the present sample could partially explain the substantial reproducibility of the NEQ score. Stated differently, it could be the absence of night eating symptoms that is highly reproducible. Therefore, the reproducibility of NEQ scores greater > 34 needs to be assessed to complement the results presented herein.

Importantly, the mean NEQ score did not systematically diverge across assessments in the overall sample. This suggests that global night eating symptoms do not fluctuate greatly over two years in middle-aged adults or do not worsen with age. Additionally, this indicates that a first assessment of the NEQ does not systematically influence a second assessment two years (no lingering retest effects). Likewise, Moizé et al., reported that an two-week interval that separated NEQ assessments did not systematically influence NEQ scores in a Spanish version of the questionnaire (Moize, Gluck et al. 2012).

The majority of the individual NEQ items were reproducible across assessments, particularly those relating to frequency of nocturnal ingestions, nocturnal awakenings and the timing of the morning meal. Based on the use of quadratic weights, this indicates that it was

uncommon for an individual to diverge greatly in their responses on these items and suggests these symptoms are stable across time. Like with the NEQ score, the lack of severe and frequent symptoms in the current sample could impact the reproducibility of the items. However, despite the low prevalence of severe or frequent symptoms, i.e., 1 – 2 % reporting nocturnal ingestions, breakfast skipping, onset insomnia and a current depressed mood, we still observed variability in the reproducibility of these items. This suggests that although some symptoms are under-represented in this sample, some symptoms are more reproducible across time than others (night eating vs. a depressed mood for example).

The normal kappas used to measure agreement for the NES criteria can be interpreted as the consistency in the categorization of more general symptoms. Most criteria had at least moderate agreement across assessments, although the confidence limits were wide and the lower limit was below 0.40 which has been suggested as the clinical limit of acceptable agreement (Sim and Wright 2005). In effect, no criteria had substantial or excellent agreement. This could indicate that the cut-offs were not distinct enough, leaving room for divergences in symptom severity/frequency, particularly in individuals who reported moderate symptoms. However, the cut-offs were likely adequate since few individuals reported night eating behaviours, which is to be expected in a sample of the general community. The lack of substantial agreement could also be an indication that the prevalence was too low for certain NES criteria. Indeed, eating behaviours unique to NES, such as evening hyperphagia, nocturnal ingestions and a belief that one must eat in order to sleep, had moderate maximum kappa coefficients indicating that agreement would be stronger in a more symptomatic sample.

The reproducibility for evening hyperphagia was lower than for nocturnal ingestions. This could be because there was a systematic increase in evening hyperphagia in the second assessment. Participants have previously found it difficult to report the proportion of total daily energy eaten after the evening meal on the NEQ (Allison, Lundgren et al. 2008a). But because this was an observational study (no intervention) and because the time interval between the two tests was two years, this reduces the likelihood that the increase in evening hyperphagia was attributed to improved comprehension of this item. This divergence could indicate that delayed eating may become more prevalent as adults age (and who also become heavier), particularly in samples at risk of obesity such as in the current study. Indeed, delayed eating may be characteristic of some obese individuals (Allison, Goel et al. 2014). Thus, fluctuations in evening hyperphagia may be related to other factors whereas nocturnal ingestions may be a more stable

and reproducible characteristic of NES. The systematic divergence in self-reported evening hyperphagia should be considered when tracking night eating symptoms over time.

The least reproducible NEQ items or NES criteria were those related to mood. The low reproducibility indicates that these items and the derived criterion likely captured mood states as opposed to trait depression. This is logical for item 6 that directly questions the current mood of the individual, and this symptom was not reproducible when assessed as an item or a dichotomous symptom. In contrast, item 7 assesses habitual diurnal mood fluctuations and should not vary depending on the current mood of the individual. Then again, it is uncertain if diurnal mood variations are a stable characteristic of mood and thus reproducible. In a longitudinal study of 39 depressed patients, diurnal mood variations were irregular and individuals reported both morning- and evening-worsening mood variations (Gordijn, Beersma et al. 1994). In contrast with the current study, Moizé et al., found that the short-term reliability of these items were high ($r > 0.80$) in a non-NES sample (Moize, Gluck et al. 2012). Thus, the low reproducibility from this study could represent more long-term fluctuations in depressive symptoms. Because evening-worsening mood is characteristic of NES, the long and short-term reproducibility of this item could be better assessed in a NES sample.

Like with the depressed mood, no morning appetite, one of the two components of morning anorexia, had very low reproducibility. Thus, when dichotomized, morning appetite may actually reflect an appetite state that is not reproducible across time and that may be dependent on some other emotional (or environmental) factor, regardless of body weight.

When assessed by weight status, the NEQ was generally less reproducible for the obese group. Indeed, this group had greater intra-individual variability in NEQ scores and scores were systematically lower on the second assessment compared to the first. Moreover, the reproducibility was weaker for most NEQ items and NES criteria, particularly for those related to mood, sleep and evening food cravings, all symptoms that are associated with body weight. In accordance with this, Moizé et al., found that the NEQ items were reliable in an sample characterized with obesity (Moize, Gluck et al. 2012). Thus, it is possible that the lower reproducibility observed in the obese group in the current study are more attributable to symptom fluctuations as this group was also more symptomatic. Accordingly, the agreement was weaker in criteria that were more prevalent in this group (evening hyperphagia, strong urge to eat and insomnia).

One exception was the obese group had greater reproducibility in morning anorexia criterion that was attributable to the greater reliability of breakfast skipping in the obese group. Skipping breakfast is associated with obesity and this may be a more stable behaviour in this weight group (Ma, Bertone et al. 2003). The role of morning anorexia in NES has previously been discussed (Gluck, Geliebter et al. 2001; Allison, Engel et al. 2008). It may be best to assess this criterion with the timing of the morning meal as this seems to be a more reproducible behaviour, particularly in obese individuals who are more susceptible of the disorder.

Despite the importance of assessing questionnaire reproducibility in various different populations, the main limitation of this study is the lack of symptomatic individuals. In one way, this was overcome by assessing the reproducibility across different weight groups. Even though the obese group was more symptomatic than the non-obese group, this sample did not have the full disorder but only reported isolated symptoms. Thus, the results presented herein cannot be generalized to more symptomatic samples. However, they can complement other studies that wish to assess the NEQ reproducibility in NES patients. Furthermore, the two-year time interval between assessments can be both and strength and a limitation in the current study. On the one hand, it is clinically important to assess symptom reproducibility over a long period of time as this gives a quantified idea of the stability of these behaviours, while reducing the likelihood of retest effects. On the other hand, we cannot determine if the lack of reproducibility in some criteria and items was a result of actual changes in self-reported symptoms or if they were attributed to categorization methods to derive the NES criteria.

Conclusions

This study demonstrated that the NEQ score and the NEQ items are reproducible over two years in adults. The reproducibility of most of the derived NES criteria were also reasonable. The items and criteria relating to mood, in addition to morning appetite criterion, were the least stable and thus seem to measure mood and appetite states rather than more stable traits. Lastly, the NEQ was less reproducible in obese persons but whether this was attributed to their greater NES symptomatology or to a unique aspect of an obese state cannot be determined.

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Chapter 8: Night eating symptoms and two-year weight change in parents enrolled in the QUALITY cohort

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Abstract

Background/Objective: The timing of food intake may be implicated in weight gain. This study tested the hypothesis that night eating symptoms are associated with weight gain in adults. Subjects/Methods: Parents participating in QUALITY completed the Night Eating Questionnaire (NEQ) at baseline (2005 – 2008). Height and weight were measured at baseline and again two years later (2008 – 2010) for 388 parents (59% female, mean (sd) age: 41.8 ± 5.7, mean (sd) BMI: 29.6 ± 5.7). Linear regression models were used to test the associations between baseline night eating symptoms (NEQ scores, night eating behaviours) and percent change in each of BMI and waist circumference (WC). Results: A high NEQ score predicted a small increase in percent change in BMI in non-obese parents but a decrease among those who were severely obese. Nocturnal ingestions of food and morning anorexia predicted increases in percent change in both BMI and WC. Conclusion: Nocturnal ingestions of food may be a risk factor for weight gain in adults.

Résumé

Contexte / Objectif: Des données récentes suggèrent que le moment de la prise alimentaire pourrait être impliqué dans le gain de poids. Cette étude avait comme objectif d'évaluer l'impact des symptômes du syndrome de la fringale nocturne (SFN) sur la prise de poids chez les adultes. Sujets / Méthodes: Les parents participant à la cohorte QUALITY ont complété le night eating questionnaire (NEQ) à deux reprises, soit à la phase 1 (2005 - 2008) et à la phase 2 (2008 - 2010). La taille et le poids ont été mesurés et les données du NEQ étaient disponibles pour 388 des parents (59% de femmes, moyenne (SD) âge: 41,8 ± 5,7, moyenne (DS) IMC: 29,6 ± 5,7). Des modèles de régression linéaire ont été utilisés pour tester les associations entre les symptômes du SFN (scores du NEQ, critères cliniques du SFN) et le changement relatif de l'indice de masse corporelle (IMC) entre la phase 1 et 2. Résultats: Un score élevé au NEQ prédisait une légère augmentation relative de l'IMC chez les parents non obèses mais une diminution relative de l'IMC chez les parents avec obésité sévère. Par ailleurs, les ingestions nocturnes et l'anorexie matinale prédisaient une augmentation relative d'IMC et de circonférence de la taille. Conclusion: Les ingestions nocturnes peuvent être un facteur de risque de gain de poids chez les adultes.

Introduction

There is growing interest in the relationship between body weight and the timing of food intake (Allison, Goel et al. 2014; Garaulet and Gomez-Abellan 2014). Greater food intake in the evening has been associated with an increased risk of overweight and obesity (Wang, Patterson et al. 2013), a higher BMI (Baron, Reid et al. 2011), as well as less weight loss during a weight-loss interventions (Garaulet, Gomez-Abellan et al. 2013; Jakubowicz, Barnea et al. 2013).

Night eating syndrome (NES), a disorder characterized by a circadian delay in food intake, is associated with obesity (Colles and Dixon 2012; Gallant, Lundgren et al. 2012b). Individuals with NES generally have higher body weights (Aronoff, Geliebter et al. 2001; Colles, Dixon et al. 2007) and it may impede body weight control (Gluck, Geliebter et al. 2001; Andersen, Stunkard et al. 2004a; Gluck, Venti et al. 2008a). Furthermore, night-eaters gained more weight over 3 – 6 years than non-night-eaters (Andersen, Stunkard et al. 2004a; Gluck, Venti et al. 2008a) and individuals with NES lost less weight during a restrictive weight-loss intervention than individuals without NES (4.4 vs. 7.3 kg) (Gluck, Geliebter et al. 2001). In contrast, having NES did not impede weight loss or weight loss maintenance after an intensive inpatient behavioural weight-loss intervention (Dalle Grave, Calugi et al. 2011). Thus, the limited evidence available suggests that NES and night-eating behaviour may be implicated in body weight changes but additional studies are needed.

The aim of this study was to test the hypothesis that night eating symptom severity and NES diagnostic criteria are associated with two-year weight gain in parents.

Methods

Study population

Participants included parents participating in the QUALITY (Québec Adipose and Lifestyle InvesTigation in Youth) family study, an ongoing prospective cohort investigation of the natural course and risk factors for childhood obesity (Lambert, Van Hulst et al. 2011). Families (both biologic parents and one child) were recruited in elementary schools in Montreal and Quebec City, Canada. Eligibility criteria included being Caucasian, child aged 8-10 years, and at least one obese biological parent (BMI ≥ 30 kg/m^2 or waist circumference >102 cm in fathers, >88cm in mothers). Parents participated in two testing days approximately two years apart, at baseline in 2005 – 2008 and again at follow-up in 2008 – 2011. This project was approved by

the ethics committees of Laval Hospital (Quebec) and the Centre Hospitalier Universitaire de Sainte-Justine (Montréal).

Anthropometric measures

Body weight and height were measured according to standardized protocols (Lohman 1988) using a stadiometer for height and an electric scale for weight. Body mass index (BMI) was calculated as weight (kg/m^2). Waist circumference (WC) was measured at the mid-point between the iliac crest and the last rib. Non-obese was defined as a BMI < 30 kg/m^2, obesity as a BMI from 30 – 34.9 kg/m^2 and severe obesity as a BMI ≥ 35 kg/m^2. Abdominal obesity was defined by the American Heart Association/National Heart, Lung, and Blood Institute cut-offs for women (<88cm) and men (<102cm) (NIH 1998). Parents with missing anthropometric data at follow-up were excluded (*n* = 261).

Night eating symptoms

Parents completed the self-administered Night Eating Questionnaire (NEQ) (Allison, Lundgren et al. 2008a) on testing days. The NEQ was introduced mid-way through the baseline testing period, but to all parents at follow-up. This current study uses the subsample of parents with both baseline and follow-up NEQ data.

The NEQ is a 14-item scale that measures NES severity as a continuous score (range = 0 - 52) (Allison, Lundgren et al. 2008a). Item 13 (level of consciousness of nocturnal ingestions) is not included in the total score, but is used to distinguish nocturnal ingestions common to NES from nocturnal ingestions common to sleep-related eating disorders. Each item in the NEQ can be dichotomized according to pre-determined severity cut-offs and grouped according to specific night eating symptoms. The specific night eating symptoms are then coded as present or absent depending on the level of severity.

Evening hyperphagia was defined as > 25% of total energy intake after the evening meal. Nocturnal ingestions was defined as eating upon awakening at night at least sometimes with frequent (>3/week) nocturnal awakenings. A strong urge to eat in the evening or at night was defined as often or always feeling a strong urge to eat in the evening or a very strong urge to eat upon awakening at night.

The definitions of night eating symptoms, based on NEQ item cut-offs, are found in Table 8.1. Other studies have used similar definitions but the cut-off for nocturnal ingestions was less severe in the current study (Elsadek, Hamid et al. 2014; Gallant, Drapeau et al. 2014; Runfola, Allison et al. 2014). The NEQ has been validated in adults (Allison, Lundgren et al. 2008a).

Table 8.1. Definitions of night eating symptoms

Night Eating Symptom	**Definition base on the NEQ**
Evening Hyperphagia	> 25% total energy intake after the evening meal
Nocturnal ingestions	Sometimes eating upon awakening with frequent night awakenings (> 3/week)
Morning anorexia	No morning appetite at all and/or eating for the first time after 12:01 PM
Strong evening/nighttime food cravings	Strong food cravings and urges to eat after supper and/or when awaken from sleep
Belief that one must eat in order to sleep	A need to eat to fall back asleep
Sleep troubles	Frequent sleep onset or maintenance problems (usually, > 1/week)
Depressed or evening-worsening mood	Currently feeling blue and/or mood is lowest in the early or late evening

NEQ, Night Eating Questionnaire

Covariates

Demographic and lifestyle data were collected in self-administered questionnaires. Covariates included age, family income, smoking status, average weekly frequency of alcohol consumption during the last 12 months, shift work and voluntary weight change.

Statistical analysis

The outcomes of interest in this analysis included percent change in each of BMI and WC over two years. Linear mixed models were used to assess the association between the continuous NEQ score and each of percent change in BMI and percent change in WC. In all

models, a random "family" effect was used to correct for clustering within families. Values of the anthropometric measures at baseline were included in all models, first as an interaction term and thereafter as an independent variable. Group size permitting, significant results were additionally adjusted for covariates. Missing values for covariates were replaced with the mean value of that variable.

Results

Sample characteristics

The analytic sample included 388 parents (59% female) after 261 parents (38% female) were excluded because of missing anthropometric data at follow-up. BMI, WC and NEQ scores at baseline were not different between parents with and without anthropometric data at follow-up. Descriptive characteristics of the sample are presented in Table 8.2. The mean percent change in BMI over two years was 1.4 ± 6.3 (range: -31.1 to 36.8) and the mean percent change in WC over two years was 0.24 ± 6.1 (range: -22.7 to 36.4).

Night eating symptom severity (NEQ score)

We tested a NEQ score X baseline BMI interaction term, which indicated that the relationship between NEQ and percent change in BMI tended to be positive in parents with a BMI < 30, neutral in obese parents and negative in severely obese parents (β (SE) = -0.05 (0.01), $p < 0.0001$). The interaction term remained statistically significant after adjusting for all covariates ($p < 0.0001$). When stratified by body weight category, only the negative association in the severely obese group was significant ($n = 59$, β (SE) = -0.71 (0.23), $p = 0.04$). There NEQ X baseline WC interaction term did not predict percent change in WC over two years ($p = 0.14$). NEQ score did not predict percent change in WC after controlling for baseline WC ($p = 0.84$).

Specific night eating symptoms

We examined the association between each specific night eating symptom (i.e., nocturnal ingestions (n=21 parents reported this symptom), morning anorexia (n=21), sleep onset or maintenance problems (n=178), evening food cravings (n=78) and evening depressed mood n=127)) and percent change in BMI over two years adjusting for baseline BMI. Two of the five associations tested were statistically significant or close to statistical significance including: (i) the association between nocturnal ingestions and percent change in BMI ($p = 0.04$) and (ii) the association between morning anorexia and percent change in BMI ($p = 0.07$) (Table 8.3).

Table 8.2. Baseline characteristics of parents participating in QUALITY (n = 388)

	Parents (*n* = 388)
Age, yrs, mean ± sd	41.2 ± 5.7
Female, %	59
BMI, kg/m^2, mean ± sd	29.6 ± 5.7
Weight status, %	
Overweight	31
Obese	31
Waist circumference, cm, mean ± sd	98.3 ± 14.9
Abdominally obese[§], %	74
Current smoking status, %	
Non-smoker	87
Occasional smoker	5
Daily smoker	8
Alcohol consumption, ≥ 4 drinks/week %	14
Family income, $/year, mean sd	45 166
Shiftwork[†], %	9
NEQ score, mean ± sd	10.7 ± 4.5
NEQ score, range	1 – 26

female ≥ 88cm; male ≥ 102 cm (NIH 1998)

†17% of parents did not reply to this question

We also examined the association between each specific night eating symptom and percent change in WC over two years adjusting for baseline WC. Morning anorexia was the only symptom that associated with an increase in percent change in WC (β (SE) = 1.31 (0.66), *p*=

0.048, Table 8.3). Because of the small number of parents who reported nocturnal ingestions and morning anorexia, these models could not be adjusted for additional covariates (beyond baseline BMI).

Table 8.3. Adjusted beta coefficients for the relationship between the percent change in each of BMI and WC over two years and individual night eating symptoms among parents in the QUALITY cohort (n = 388)

Reported night eating symptoms	*n*†	Percent change in BMI		Percent change in WC	
		β (SE)	*p* - value	β (SE)	*p* –value
Nocturnal ingestions	21	1.44 (0.70)	**0.04**†	0.53 (0.68)	0.44
Morning anorexia	21	1.22 (0.68)	0.07	1.31 (0.66)	**0.048**‡
Sleep troubles	178	0.32 (0.31)	0.31	0.11 (0.31)	0.71
Strong evening food cravings	78	0.04 (0.40)	0.93	-0.25 (0.39)	0.52
Depressed mood	127	0.28 (0.32)	0.40	0.25 (0.32)	0.44

†Number of parents reporting this symptom

Mean percent changes in BMI and WC associated with night eating symptoms are illustrated in Figures 8.1 and 8.2. The percent change in BMI and WC associated with evening hyperphagia and a belief that one must eat in order to sleep could not be assessed because of the low prevalence of these symptoms in this sample (*n* = 6 and 1, respectively).

Interactions between night eating symptom severity and sleep problems

Individuals who reported frequent nocturnal awakenings with ingestions of food at least sometimes had a higher percent change in both anthropometric measures (BMI (3.8 ± 5.3%) and WC (0.94 ± 5.5%) than those who reported frequent nocturnal awakenings without ingestion of food (BMI: 1.2 ± 6.5%, WC: 0.10 ± 6.0%) and those without frequent nocturnal awakenings (BMI: 1.4 ± 6.2%, WC: 0.26 ± 6.3%). However, the findings did not attain statistical significance (BMI: *p* = 0.12 and WC: *p* = 0.49).

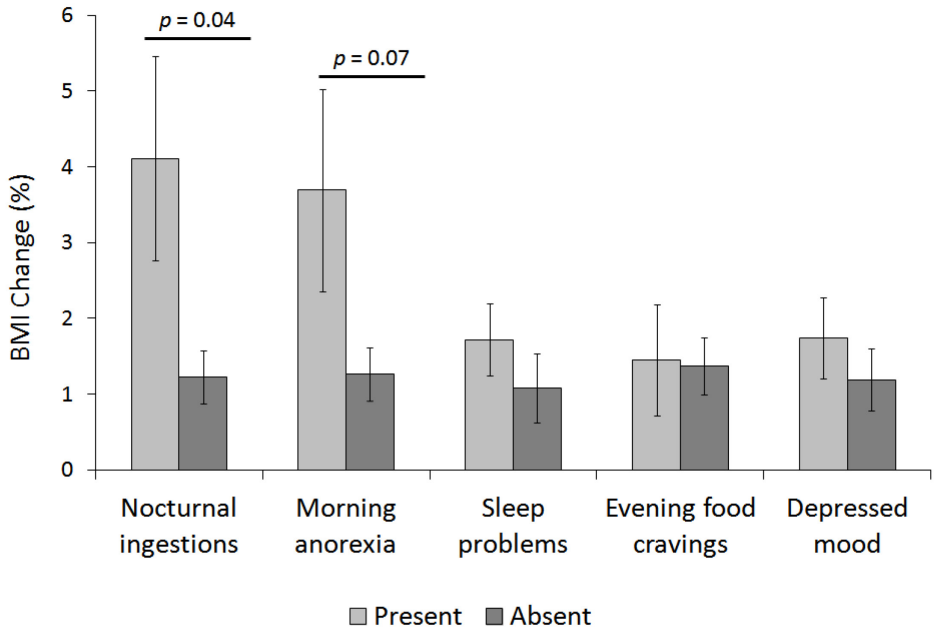

Figure 8.1. Percent change in BMI over two years according to night eating behaviours in parents (n = 388), adjusted for baseline BMI. Number of participants with symptoms: 21 parents reported nocturnal ingestions, 21 reported morning anorexia, 178 reported sleep onset or maintenance problems, 78 reported strong evening food cravings and 127 reported current or evening worsening depressed mood.

Discussion

The aim of this study was to examine the longitudinal association between night eating symptoms and body weight change over two years in parents participating in the QUALITY study. Baseline body weight moderated the association between night eating symptom severity and two-year weight change. Specifically among severely obese parents, a high NEQ score was associated with a lower percent change in BMI. However, a high NEQ scores was associated with a higher percent change in BMI in the non-obese (although the latter did not reach significance). In contrast, parents who reported nocturnal ingestions of food had a 3% greater increase in BMI than parents who did not report this behaviour. Furthermore, parents who reported morning anorexia had a 2.6% greater increase in waist circumference.

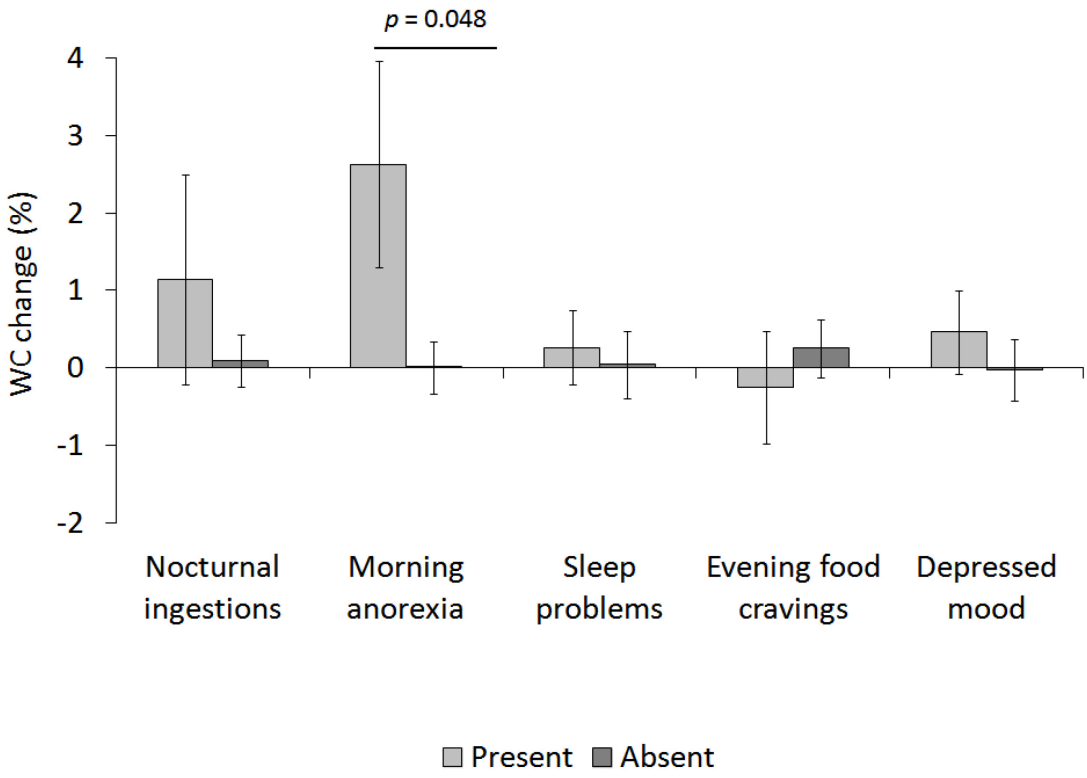

Figure 8.2. Percent change in waist circumference (WC) over two years according to specific night eating symptoms among parents (n = 388), adjusted for baseline WC. Number of participants with symptoms: 21 parents reported nocturnal ingestions, 21 reported morning anorexia, 178 reported sleep onset or maintenance problems, 78 reported strong evening food cravings and 127 reported current or evening worsening depressed mood.

In several cross-sectional analyses, night eating symptom severity, i.e., NEQ score, was positively associated with BMI (Lundgren, Smith et al. 2010; Harb, Levandovski et al. 2012; Moize, Gluck et al. 2012), including in a study from this cohort (Gallant, Drapeau et al. 2014). However, to our knowledge, this is the first study to investigate the association between change in BMI and night eating symptom severity (NEQ score). We did not find that more frequent or severe night eating symptoms, as indicated by a high NEQ score, were associated with an increase in BMI after two years in the full sample. However, we found that baseline BMI moderated the relationship between the NEQ score and percent change in BMI; a greater degree of night eating symptom severity predicted an increase in BMI in the non-obese parents whereas it predicted a decrease in the severely obese parents. Although it seems that night

eating symptom severity may lead to weight gain in non-obese parents, particularly in normal weight parents, the effect was small and was not significant when analysed separately. Thus, more studies are needed to confirm this finding.

Unexpectedly, the reverse was observed in severely obese parents; a high NEQ score predicted a decrease in BMI, independent of sleep problems. However, it is unlikely that more severe night eating symptomatology represented by a greater NEQ score, e.g., atypical eating patterns, poor appetite control, depressed mood, leads to weight loss. We postulated that individuals in this weight group, individuals who were more symptomatic and likely suffering from obesity-related co-morbidities were more likely to seek intensive weight loss treatment for their obesity over the course of the two years. This would explain the negative association between the NEQ score and percent change in BMI. However, we adjusted the results for voluntary weight loss and the results persisted. It is also possible that the night eating symptoms in this group attenuated over the two-years or that the individuals sought treatment especially for these symptoms that resulted in a decrease in BMI. The latter does not explain the very large changes in BMI observed in some individuals in this weight group (-32% change in BMI). Thus, further studies are needed to assess how night eating symptoms impact weight control in severely obese parents.

Despite a demonstrated link between NEQ scores and components of the metabolic syndrome in men (high fasting triglycerides, elevated waist circumference) (Gallant, Drapeau et al. 2014), the NEQ score did not predict a change in WC in the current study. It should be noted that the degree of night eating symptomatology was low in the current cohort (maximum NEQ score 26), which could have impacted the results. Thus, it is important to confirm these results in a more symptomatic sample.

Nocturnal ingestions of food was associated with an increase in BMI in parents. Indeed, parents who reported this behaviour had approximately a 3% greater increase in BMI after two years than parents who did not report this behaviour. This finding is in accordance with other studies that have reported greater weight gain in individuals who eat during the night (Andersen, Stunkard et al. 2004a; Gluck, Venti et al. 2008a). Gluck et al., reported that obese individuals who consumed nocturnal ingestions gained 4.5kg more over a mean of 3.5 years than those without this behaviour (Gluck, Venti et al. 2008a). Likewise, Andersen et al., reported that obese women who consumed nocturnal ingestions gained 4.3 kg more over 6 years (+4.6%

of baseline body weight) than obese women without this behaviour (Andersen, Stunkard et al. 2004a). The observed weight gain from the current study is comparable to these studies but slightly less (equivalent to approximately a +2.5% change in body weight). Because nocturnal ingestions of food was not associated with an increase in WC, this suggests that this type of eating may not favour the accumulation of abdominal adipose tissue.

Morning anorexia was related to percent change in WC, a marker of metabolic health. Along these lines, morning anorexia was more prevalent in parents with metabolic syndrome from this cohort (Gallant, Drapeau et al. 2014), which could possible explain this result. Other studies have found that breakfast skipping was associated with a worse metabolic profile in young adults, adolescents and children (Deshmukh-Taskar, Nicklas et al. 2010; Shafiee, Kelishadi et al. 2013). Moreover, in a randomized cross-over trial, skipping breakfast was associated with higher fasting and total cholesterol and impaired insulin sensitivity (Farshchi, Taylor et al. 2005). Thus, this suggests that morning anorexia (and skipping the morning meal) may be important in metabolic health, as it seems to favor abdominal adiposity. It should be noted that the weight change associated with nocturnal ingestions and morning anorexia seems to be independent from either symptom because only one parent reported both eating behaviours.

Nocturnal awakenings, i.e., sleep maintenance problems, are implicit in individuals who report nocturnal ingestions and could potentially explain the relationship between nocturnal ingestions and percent change in BMI. Indeed, some studies have demonstrated a relationship between sleep duration and quality and weight gain (Cappuccio, Taggart et al. 2008). However, in the current study, reported sleep onset and/or sleep maintenance problems were not associated with measures of weight change. This suggests that it is the nocturnal ingestions per se and not the nocturnal awakenings led to the increase in BMI. Despite this, when examined jointly, percent change in BMI in parents who reported nocturnal ingestions was not statistically different from the percent change in BMI in parents who reported nocturnal awakenings or controls, but the differences could become clinically meaningful over time.

The limitations of this study need to be considered. First, the symptoms identified herein are self-report and, for the most part, are isolated and thus the participants do not represent individuals with the full disorder (NES). It is possible that reporting several of the symptoms could have an additive effect on body weight. Moreover, the low frequency of certain night

eating symptoms needs to be considered and how this limits the results presented herein. Because of this, additional covariates could not be examined in models of nocturnal ingestions and morning anorexia. Nonetheless, these results contribute to a growing literature night eating as this study is a first step in understanding the prospective relationships between night eating symptoms and body weight gain in adults.

Conclusions

Night eating symptom severity does not consistently predict weight change over two years in healthy weight and obese adults from the general community and the association may depend on initial body weight. In contrast, nocturnal ingestions of food was associated with an increase in BMI and morning anorexia with an increase in WC. This study highlights the importance in considering night eating symptoms, particularly nocturnal ingestions of food, morning anorexia and sleep problems, in interventions aimed to prevent and treat obesity.

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Chapter 9: Conclusions

There is growing evidence that eating-related obesity risk factors are beyond energy content and diet quality. Researchers are beginning to investigate how and when individuals eat as possible contributors to weight gain. The Night Eating Syndrome (NES) is a disorder characterized by a circadian delay in food intake that manifest mainly as excessive evening intake, evening hyperphagia, and nocturnal ingestions of food. The disorder is rare in the general population (0.5 – 5%) but its prevalence increases in obese populations (9 – 19%). Because of the nature of the symptoms, e.g., eating during the biological rest phase, poor appetite control, disrupted sleep, this disorder could potentially lead to metabolic problems and weight gain. However, very few studies have examined the negative health consequences associated with NES.

As established in the literature review of this thesis, the metabolic characterization and long-term consequences associated with these symptoms are elements that are lacking in the NES literature. Specifically, these issues have not been investigated in non-treatment seeking individuals, such as in members of the general population. Treatment-seeking obese patients carry with them a variety of health symptoms and because of this, assessing night eating symptoms in the general population may shed light on the health consequences associated with sub-clinical symptoms.

In addition to the metabolic and weight-related aspects of NES, data regarding this disorder is lacking in children. Indeed, only one study (Lamerz, Kuepper-Nybelen et al. 2005a) has explored night eating symptoms in a child population and this study did not examine health correlates of these symptoms. Furthermore, the symptoms were parent-reported and not child-reported and the study limited the investigation to assessing only nocturnal ingestions of food. Because of this, assessment of the validity of the Night Eating Questionnaire (NEQ) in a child population is needed. Moreover, the behavioural characterization of these symptoms in a child population is necessary to better understand the etiology of the disorder.

Thus, the main objectives of this study were to investigate the behavioural and metabolic characterization of night eating symptoms children and their parents. Specific objectives included to review the literature on NES and obesity and late night eating, to validating the Night Eating Questionnaire (NEQ) in children, to examine the metabolic health of adults with night

eating symptoms and to examine the relationship between metabolic disease and night eating symptoms, to assess the long-term reproducibility of the night eating symptoms and to evaluate the impact of night eating symptoms on weight gain in adults.

Specifically, this study aimed to answer the following questions: Are night eating symptoms associated with metabolic and anthropometric variables in adults? Which is a better measure of night eating symptoms in children, a child- or parent-report? Is the NEQ associated with dietary data and other health behaviours in children? What do children and adults eat when they awaken from sleep? Are night eating symptoms related to weight gain in adults? Are night eating symptoms reproducible across time in adults?

Night eating symptoms in children: the NEQ and health behaviour correlates

The QUALITY cohort study was the first study to thoroughly investigate night eating symptoms in children with the Night Eating Questionnaire (NEQ). We found that, although no children met the screening criteria for the full disorder, there was variability in the global score of night eating symptom severity (NEQ score). Furthermore, the variability in this score was associated with a relative delayed pattern of energy intake; children with more frequent/severe night eating symptoms ate relatively more after supper and less in the morning. This demonstrated that the NEQ captured children who had a similar eating style as observed as those with NES, albeit to a much lesser degree. This result was also supported by the relative delay in physical activity that observed in children with night eating symptoms. Taken together, these studies revealed that global night eating symptom severity, the NEQ score, was associated with a relative delay in circadian behaviours, eating and physical activity.. But what is the clinical significance of the behavioural delay captured by the NEQ?

Although the behavioural delay associated with night eating symptoms was investigated in the context of validating the NEQ, this behavioural delay may also have a clinical significance. In line with this statement, emerging evidence in animals and human adults is linking a delayed pattern of energy intake with body weight, weight gain and energy metabolism (Allison, Goel et al. 2014; Garaulet and Gomez-Abellan 2014). Despite this and the observation that children with night eating symptoms showed signs of a less healthy diet, e.g., greater fat intake, less fruit and vegetable intake, and a tendency for less healthy snacks, these symptoms were not associated

with measures of body weight in cross-sectional analyses. This could be that the delay in food intake was only small, that the dietary differences did not translate into greater total energy intake or that the behavioural delay was also accompanied by greater daily minutes of light physical activity as observed in this study. Thus, for now, the behavioural delay captured by the NEQ, is not associated with negative weight-related health in children.

The behavioural delay captured by the NEQ is interesting from a methodological and conceptual standpoint. This suggests that the children who are eating relatively more in the evening (and less in the morning) are also participating in more physical activity in the evening. In these children, feeding and physical activity behaviours seem to be coupled, which is actually a feature of health, as depicted in humans (Scheer, Hilton et al. 2009) and in animal models (Salgado-Delgado, Angeles-Castellanos et al. 2010). Thus, the NEQ could have captured a certain evening "chronotype", i.e., the innate circadian preference of an individual's biological functioning, in healthy children which may not have negative health consequences at such a young age.

The studies presented herein were important as first investigations in night eating symptoms in children. However, because few children reported isolated night eating symptoms e.g., night eating behaviours, morning anorexia, further research is needed to accurately assess the health impact of these symptoms in a young population. The NEQ will also need to be validated against measures of sleep, against measures of mental health and, if possible, it will need to be validated in children with the full disorder.

Night eating symptoms in adults: metabolic health correlates, long-term reproducibility and impact on body weight gain

As elaborated in the review on obesity and NES (chapter 3) and in the review section of the nutritional aspects of late and night-eating (chapter 4), it is theorized that night eating symptoms, through several pathways, could lead to metabolic and weight-related health consequences in some individuals. Based on this information, we hypothesized that individuals with night eating symptoms would be more at risk for obesity, metabolic problems and be more likely to gain weight over time.

Night eating symptom severity in adults

In line with our hypotheses, we demonstrated that night eating symptom severity was associated with measures of metabolic health, both in cross-sectional and prospective analyses. However, the findings were less consistent than what was hypothesized and some sex differences were identified. As hypothesized, night eating symptoms were more frequent/more severe in adults with a greater BMI and the cross-sectional association between night eating symptom severity and metabolic health (adjusted for BMI), such as high blood triglycerides and elevated waist circumference, seemed to be specific to men.

Despite these cross-sectional associations between night eating syndrome severity and markers of metabolic health, night eating symptom severity did not predict two-year changes in body weight in the entire sample (in either men or women), which was contrary to our prospective hypotheses. However, there was an unexpected interaction effect with baseline body weight – it predicted a small weight gain in non-obese adults and a weight loss in severely obese adults. Interestingly, the NEQ total score was more reproducible across time in the non-obese adults, i.e., night eating symptoms were less stable in the obese group, which may explain this moderation effect.

Thus, adults with an elevated BMI, and men with elevated triglycerides and waist circumference independently from BMI, were characterized by more frequent and severe night eating symptom as indicated by a higher NEQ score. Furthermore, although the NEQ score does not seem to fluctuate greatly over two years, less so in the non-obese than the obese, these symptoms tended to predict weight gain only in the non-obese adults. Nonetheless, these results indicate that night eating symptom severity is more common in obese individuals and because of this may contribute to their body weight, however, perhaps only a small and indiscernible amount which is more noticeable in non-obese adults.

Night eating syndrome criteria

We hypothesized that isolated night eating symptoms, particularly nocturnal ingestions, evening hyperphagia, morning anorexia, a belief that one must eat in order to sleep, symptoms unique to NES, would be more prevalent in individuals with metabolic disease. Indeed, we found that the prevalence of morning anorexia, that is skipping breakfast and/or reporting no morning

appetite, was higher in those with metabolic syndrome than those without this disorder. Although not a core criterion of NES, morning anorexia suggests atypical circadian appetite control, and is often reported in individuals with the full disorder. Interestingly, one component of this symptom, breakfast skipping, was highly reproducible in obese adults of this cohort and thus it seems to be a fairly stable eating characteristic of obese individuals. In addition to these findings, morning anorexia was associated with an increase in waist circumference after two years but was mediated other lifestyle factors, namely alcohol consumption and smoking status. Thus, obese individuals and individuals with metabolic disease may be characterized by morning anorexia but the relationship between this behaviour and body weight control is complex and other factors need to be considered.

As we hypothesized in the chapter on late night eating, we theorized that night eating could be associated with metabolic problems and could predict weight gain. In contrast to our hypotheses, nocturnal ingestions was not more prevalent in individuals with metabolic syndrome or diabetes and this behaviour did not predict these diseases. Furthermore in longitudinal analyses, although nocturnal ingestions of food was associated with an increase in BMI over two years, the weight gain was not specific to abdominal adipose tissue. These cross-sectional and prospective results suggesting that metabolic health may not be impaired in night eaters beyond what is expected by general weight gain.

Although nocturnal ingestions of food predicted weight gain in adults, this behaviour was only moderately stable across time. This needs to be considered because if this behaviour was not *substantially* stable, could it have really be responsible for the associated weight gain? Because we used a low severity cut-off for this behaviour, i.e., “sometimes”, a non-consistent report across time (i.e., an unstable/irreproducible report), it would mean that the individual no longer reported nocturnal ingestions at all. It was, however, more reproducible across time than other symptoms. Because the nocturnal ingestions was related to weight gain after adjusting for multiple confounders, it is possible that the few individuals who reported nocturnal ingestions consistently across time were responsible for the greater weight gain observed among those that ate at night. However, this was not tested in any of the studies presented in this thesis. The lack of reproducibility in NES criteria could also be another reason why other night eating symptoms did not predict weight gain over time.

Strengths and Limitations

This is the first study to investigate night eating symptoms in a prospective cohort study. Furthermore, this is also the first study to investigate the full spectrum of the symptomatology in children. The strengths of this overarching study of night eating symptoms are therefore the large sample size and the family and longitudinal design aspects, in addition to the measured anthropometric, behavioural and metabolic variables.

The research presented herein needs to be considered in the context of the limitations. The main limitation of this study is the low frequency of certain night eating symptoms in the sample study. Because this cohort was characterized by obesity, many reported evening cravings for food, depressed mood and sleep problems. On the contrary, night eating behaviours were under-represented. It was believed that because this sample was considered an at-risk population based on parental obesity that night eating behaviours, i.e., nocturnal ingestions and evening hyperphagia, would be more common. However, this was not the case. The QUALITY cohort is a cohort that is comprised of well-educated and affluent community members, which may be played a role in the overall health of the population even though they were at risk. Furthermore, eating late is common in the Quebecois culture, which could have influenced the prevalence of evening hyperphagia. These two extremes, that is low and high prevalence of certain symptoms, likely impacted the variability and thus limits the true association between the symptom and weight gain and/or metabolic health. Indeed, in this thesis, night eating behaviours were examined mostly as isolated symptoms (or NES criteria) because few individuals reported several (>3) night eating symptoms. It is possible that when more than one symptom is present, the effect on metabolic health could be greater.

Along these lines, although cost-effective, the data is based on self-reported symptoms. Indeed, the NES criteria were derived from cut-offs that were not validated nor confirmed by a clinical interview. This aspect of the research design likely overestimated or underestimated the actual frequency of certain symptoms. However, unlike in other studies, evening hyperphagia was not overreported because very few individuals reported this behaviour. Furthermore, it has been suggested that the cut-offs used to identify NES criteria may be too narrow or specific (Runfola, Allison et al. 2014). This could be the case in a sample not at risk for obesity but we feel that, if anything, some cut-offs were not specific enough to identify individuals with severe sleep troubles, depressive symptoms or evening cravings.

Theoretical and research implications of this research

Although this thesis was exploratory in nature, some results presented herein may contribute to the underlying theory between feeding and activity rhythms in humans. Children are an interesting population to document activity rhythms because of their spontaneous behaviours and that they have fewer social constraints and responsibilities. As such, their activity rhythms could be seen as more natural than an activity rhythm of an adult. Indeed, the study on the physical activity profiles associated with night eating behaviours is one of the few studies in humans to examine this. These results support the connectedness of eating and physical activity rhythms and help lay the foundation for future studies which will examine the etiology of night eating.

Future studies

Although research on NES has been around since the mid 1950's, it is really only in its infancy. Studies on the etiology of this disorder are needed as are studies which will examine more long-term consequences of these symptoms (greater than two years). Indeed, this disorder can be an interesting model through which to explore the connection between obesity and circadian regulation.

In the short-term, the short-term reproducibility of the NEQ needs to be assessed in an English-speaking population and in more symptomatic individuals. The sleep and depressive components of the NEQ need to be validated in children and, if possible, the NEQ also needs to be validated in a young group of NES patients. Furthermore, the course of the disorder, i.e., the onset and symptom evolution, needs to be empirically quantified.

More long-term research should tease out the effects of chronic stress, circadian dysregulation and feeding behaviour in NES as well as in obesity. Indeed, there are gaps in our current understanding of these relationships, particularly when trying to translate animal research to humans. For example, can peripheral clocks, clocks that were disrupted through stress, lead to altered eating patterns per se? Or, are the nutrients from nocturnal ingestions, a meal used to cope with stress-induced insomnia, entraining the peripheral clocks which maintains the insomnia-night eating cycle?

Research related to obesity and the timing of food intake is also needed. Does grazing impact the circadian rhythm of feeding and appetite and is this eating style at the root of appetite-related problems and obesity? Are regular eaters less likely to become obese or to have altered eating patterns? Does changing the circadian timing of food intake impact body weight in a positive or negative way? For example, genetically obese mice with disrupted feeding patterns that were fed only at night (their biological day), showed weight- and metabolic-related health improvements (Salgado-Delgado, Angeles-Castellanos et al. 2010).

Along these lines, there are some interesting eating and activity-related treatment strategies for both NES and obesity that also merit investigation. Can temporally structured exercise improve night eating symptoms through its impact on the circadian system? Can very specific eating-related strategies reset the circadian clocks? For example, can fasting followed by temporally restricted feeding phase shift feeding rhythms and improve appetite control? Indeed, because of the rhythmic nature of our biological functions, including parameters of energy metabolism, modifying behaviour rhythms to improve their robustness could be a very interesting avenue through which to explore one aspect of the etiology of obesity and NES.

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Appendix A : Questionnaire Alimentaire (NES)

Consignes : Encerclez une seule réponse pour chaque question.

1. À quel point avez-vous généralement faim le matin?

0	**1**	**2**	**3**	**4**
Pas de tout	Très peu	Quelque peu	Modérément	Beaucoup

2. Quand mangez-vous habituellement pour la première fois de la journée?

0	**1**	**2**	**3**	**4**
Avant 9h00	9h01-12h00	12h01-15h00	15h01-18h00	Après 18h01

3. Est-ce que vous avez des envies soudaines de manger ou des rages d'aliments difficiles à contrôler après le souper mais avant d'aller vous coucher?

0	**1**	**2**	**3**	**4**
Pas de tout	Un peu	Quelquefois	Souvent	Très souvent

4. Quel contrôle avez-vous sur votre apport alimentaire entre le souper et le coucher?

0	**1**	**2**	**3**	**4**
Aucun	Un peu	Assez	Beaucoup	Énormément

5. Quel pourcentage de votre alimentation quotidienne (%) consommez-vous ***après*** le souper?

0	**1**	**2**	**3**	**4**
0% (aucun)	1-25% (jusqu'à un quart)	26-50% (environ la moitié)	51-75% (plus de la moitié)	76-100% (presque tout)

6. Vous sentez-vous présentement maussade et déprimé?

0	**1**	**2**	**3**	**4**
Pas de tout	Un peu	Assez	Beaucoup	Extrêmement

7. Lorsque vous vous sentez maussade et déprimé, le pire moment est :

0	**1**	**2**	**3**	**4**
Tôt le matin	En fin de matinée	Dans l'après midi	Début de soirée	Fin de soirée/la nuit

Cochez ici si votre humeur ne change pas au cours de la journée : ☐

8. Avez-vous de la difficulté à vous endormir?

0	1	2	3	4
Jaimais	Parfois	Souvent (environ 50% du temps)	Habituellement	Toujours

9. Combien de fois vous levez-vous au moins une fois durant la nuit?

0	1	2	3	4
Jaimais	Moins de 1fois/semaine	Environ 1 fois/semaine	Plus de 1 fois/semaine	Toutes les nuits

***********SI VOUS AVEZ RÉPONDU 0 À LA QUESTION 9, SVP ARRÊTEZ IÇI********

10. Lorsque vous vous levez la nuit, est-ce que vous avez envie de manger?

0	1	2	3	4
Pas de tout	Un peu	Assez	Beaucoup	Extrêmement

11. Lorsque vous vous levez la nuit, est-ce que vous avez besoin de manger pour vous rendormir?

0	1	2	3	4
Pas de tout	Un peu	Souvent	Très souvent	Toujours

12. Lorsque vous vous levez dans le milieu de la nuit, à quelle fréquence mangez vous?

0	1	2	3	4
Jaimais	Parfois	Souvent (environ 50% du temps)	Habituellement	Toujours

**********SI VOUS AVEZ RÉPONDU 0 À LA QUESTION 12, SVP ARRÊTER IÇI**********

13. Lorsque vous mangez dans le milieu de la nuit, à quel point êtes-vous conscient du fait que vous mangez?

0	1	2	3	4

Pas de tout	Un peu	Modérément	Beaucoup	Complètement

13.1 Si vous avez répondu 2, 3, ou 4, décrivez les aliments habituellement choisi :

- Sucre et aliments sucrés
- Gras et aliments gras
- Sel et aliments salés
- Lait et produits laitiers
- Fruits
- Légumes
- Viandes
- Pain et céréales
- Boisson gazeuse
- Chocolat
- Autres, svp préciser :

_____

14. Quel contrôle avez-vous sur votre apport alimentaire durant la nuit?

0	**1**	**2**	**3**	**4**
Pas de tout	**Un peu**	**Assez**	**Beaucoup**	**Complètement**

15. Depuis combien de temps avez-vous ce type de comportement alimentaire durant la nuit?

_____semaines _____mois _____années

16. À quel point le fait de mangé la nuit vous préoccupe-t-il?

0	**1**	**2**	**3**	**4**
Pas de tout	Un peu	Assez	Beaucoup	Extrêmement

17. À quel point le fiat de manger la nuit affecte-t-il votre vie?

0	**1**	**2**	**3**	**4**
Pas de tout	Un peu	Assez	Beaucoup	Extrêmement