

Autonomic Dysfunction as a Potential Pathogenic Mechanism
Underlying the Association between Sleep and Childhood Obesity

Denise Christina Jarrin

A Thesis
in
The Department
of
Psychology

Presented in Partial Fulfillment of the Requirements
for the Degree of Master of Arts (Psychology) at
Concordia University
Montreal, Quebec, Canada

September 2008

© Denise Christina Jarrin, 2008



Library and
Archives Canada

Published Heritage
Branch

395 Wellington Street
Ottawa ON K1A 0N4
Canada

Bibliothèque et
Archives Canada

Direction du
Patrimoine de l'édition

395, rue Wellington
Ottawa ON K1A 0N4
Canada

Your file Votre référence
ISBN: 978-0-494-45487-9
Our file Notre référence
ISBN: 978-0-494-45487-9

NOTICE:

The author has granted a non-exclusive license allowing Library and Archives Canada to reproduce, publish, archive, preserve, conserve, communicate to the public by telecommunication or on the Internet, loan, distribute and sell theses worldwide, for commercial or non-commercial purposes, in microform, paper, electronic and/or any other formats.

The author retains copyright ownership and moral rights in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

AVIS:

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque et Archives Canada de reproduire, publier, archiver, sauvegarder, conserver, transmettre au public par télécommunication ou par l'Internet, prêter, distribuer et vendre des thèses partout dans le monde, à des fins commerciales ou autres, sur support microforme, papier, électronique et/ou autres formats.

L'auteur conserve la propriété du droit d'auteur et des droits moraux qui protègent cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

In compliance with the Canadian Privacy Act some supporting forms may have been removed from this thesis.

Conformément à la loi canadienne sur la protection de la vie privée, quelques formulaires secondaires ont été enlevés de cette thèse.

While these forms may be included in the document page count, their removal does not represent any loss of content from the thesis.

Bien que ces formulaires aient inclus dans la pagination, il n'y aura aucun contenu manquant.


Canada

ABSTRACT

Investigation of Autonomic Dysfunction as a Potential Pathogenic Mechanism Underlying the Association between Sleep and Childhood Obesity

Denise Christina Jarrin

The prevalence of childhood obesity has increased almost threefold over the past two decades. Shorter sleep duration has been linked to greater body mass index and obesity, both cross-sectionally and in prospective longitudinal studies. Sleep loss has been found to be related to autonomic dysfunction. As well, obese youth exhibit markers of autonomic dysfunction characterized by reduced heart-rate variability (HRV). The aim of the present study was to investigate whether autonomic dysfunction is a pathogenic mechanism that mediates the association between shorter sleep duration and childhood obesity.

Participants included 122 youth (50.8% females) aged 8-17 years ($M=12.79$, $SD=2.19$). Youth wore an ambulatory monitor for 24-hours to assess HRV derived from continuous ECG recordings. They also completed an in-home polysomnograph sleep study ($n=57$) to assess objective sleep measures (e.g., fragmented sleep, sleep efficiency, and time spent in REM sleep). Subjective sleep measures (e.g., sleep duration, daytime sleepiness, and sleep quality) were obtained by child- and parent-report on validated questionnaires.

Shorter sleep duration and poorer sleep quality were found to be associated with greater adiposity. Autonomic dysfunction, as characterized by reduced HRV and parasympathetic activity also was found to be associated with greater adiposity. However, autonomic dysfunction was not found to mediate the association between sleep and obesity. Future research should aim to better understand the role of autonomic

dysfunction and its other indices (e.g., blood pressure), as well as hormonal and endocrine function across the course of sleep, as potential pathogenic mechanisms underlying the association between sleep and obesity in youth.

ACKNOWLEDGEMENTS

I would like to thank Dr. Jennifer McGrath for her guidance and support, as well as the members of the Pediatric Public Health and Psychology Laboratory for their enthusiasm and continued help with the Healthy Heart Project.

DEDICATION

This thesis is dedicated to my loving and supporting family. I love you.

TABLE OF CONTENTS

List of Figures	xi
List of Tables	xii
List of Abbreviations	xiv
INTRODUCTION.....	1
The Childhood Obesity Epidemic.....	1
Childhood Obesity and its Health Complications.....	2
Measuring Obesity	3
Defining Obesity in Youth.....	4
Association between Childhood Obesity and Sleep: Cross-Sectional Studies	5
Association between Childhood Obesity and Sleep: Prospective Studies.....	8
Potential Underlying Mechanisms	11
Autonomic Functioning	14
Autonomic Dysfunction and HRV.....	16
Measuring HRV	17
Time Domain Analysis	18
Frequency Domain Analysis.....	19
Absolute and Normalized HRV Values.....	20
Cardiovascular Autonomic Dysfunction and HRV	21
Sleep.....	24
Sleep Structure	24
Measuring Sleep.....	26
Sleep and its Correlates.....	28

Sleep in Youth.....	29
Sleep Problems in Youth	30
Sleep Loss and Sleep Structure.....	31
Adequate “Normal” Sleep and Autonomic Dysfunction.....	31
Inadequate Sleep and Autonomic Dysfunction.....	32
Autonomic Dysfunction and Obesity.....	34
Parasympathetic and Sympathetic Activity and Obesity	35
Animal Models.....	36
Human Studies.....	37
Autonomic Dysfunction and Childhood Obesity.....	38
Rationale for Present Study	41
The Present Study	42
Primary Hypotheses	43
METHOD	44
Participants.....	44
Measures	44
Anthropometric Data	44
Pubertal Stage	45
Ambulatory Physiological Recordings	46
Heart Rate Variability (HRV).....	46
Time Domain Analysis	47
Frequency Domain Analysis.....	47
Data Reduction.....	47

Subjective Sleep Measures	48
Perceived Sleep Quality	48
Sleep Duration	48
Children’s Sleep Habits Questionnaire (CSHQ).....	49
Pediatric Daytime Sleepness Scale (PDSS).....	49
Objective Sleep Measures.....	50
In-Home Sleep Study	50
Sleep Stage Criteria & Detention.....	50
Stage Wake	50
Stage 1.....	51
Stage 2.....	51
Stage 3.....	51
Stage 4.....	51
REM.....	52
Procedure	52
Data Preparation.....	54
Data Cleaning.....	55
Statistical Analyses	55
RESULTS	57
Sample Characteristics.....	57
Pubertal Status	57
Subjective Sleep Measures	57
Objective Sleep Measures.....	59

HRV Measures	59
Obesity Constructs	59
Subjective Sleep Constructs.....	60
Objective Sleep Constructs	60
Autonomic Dysfunction Constructs.....	60
Age Effects.....	60
Hypothesized Model Testing	62
Base Model	62
Model 1	63
Model 2	63
Model 3	64
Post-Hoc Exploratory Analyses.....	64
DISCUSSION	67
Strengths	72
Limitations and Recommendations.....	73
Future Directions	76
Conclusions.....	77
REFERENCES.....	78

LIST OF FIGURES

Figure 1: Structural equation base model: Influence of objective sleep measures on obesity	138
Figure 2: Structural equation model: Influence of objective sleep measures <i>and</i> autonomic dysfunction, made up of SDNN, on obesity	139
Figure 3: Structural equation model: Influence of objective sleep measures <i>and</i> autonomic dysfunction, made up of rMSSD, on obesity	140
Figure 4: Structural equation model: Influence of objective sleep measures <i>and</i> autonomic dysfunction, made up of pNN50, on obesity	141

LIST OF TABLES

Table 1: Participant Characteristics	116
Table 2: Means and Standard Deviations for Obesity Measures in Youth.....	117
Table 3: Self-Report Pubertal Status – Girls.....	118
Table 4: Self-Report Pubertal Status – Boys	119
Table 5: Means and Standard Deviations for Subjective Sleep Measures.....	120
Table 6: Means and Standard Deviations for Objective Sleep Study Measures.....	121
Table 7: Means and Standard Deviations for HRV Measures during Light NREM, Slow Wave Sleep, and REM Sleep	122
Table 8: HRV Measures in Healthy-Weight, Overweight, and Obese Youth during Sleep.....	123
Table 9: Inter-correlations among Obesity Measures.....	124
Table 10: Inter-correlations among Child- and Parent-Report Subjective Sleep Measures	125
Table 11: Inter-correlations among Objective Sleep Measures.....	126
Table 12: Partial Correlations between Objective Sleep Measures and Child-Report Subjective Sleep Measures	127
Table 13: Partial Correlations between Objective Sleep Measures and Parent-Report Subjective Sleep Measures	128
Table 14: Inter-correlations among HRV Measures.....	129
Table 15: Partial Correlations between Subjective Sleep Measures and Obesity Measures	130

Table 16: Partial Correlations between Objective Sleep Measures and Obesity Measures	131
Table 17: Partial Correlations between Objective Sleep Measures and HRV during Light NREM Sleep	132
Table 18: Partial Correlations between Objective Sleep Measures and HRV during Slow Wave Sleep	133
Table 19: Partial Correlations between Objective Sleep Measures and HRV during REM Sleep.....	134
Table 20: Partial Correlations between Obesity Measures and HRV Measures during Light NREM Sleep	135
Table 21: Partial Correlations between Obesity Measures and HRV Measures during Slow Wave Sleep	136
Table 22: Partial Correlations between Obesity Measures and HRV Measures during REM Sleep	137

LIST OF ABBREVIATIONS

ANS.....	Autonomic Nervous System
BMI.....	Body Mass Index
CDC	Centers for Disease Control
CFI	Comparative Fit Index
CSHQ.....	Children’s Sleep Habits Questionnaire
DEXA	Dual Energy X-ray Absorptiometry
ECG.....	Electrocardiogram
EEG.....	Electroencephalogram
EMG.....	Electromyogram
EOG	Electroculogram
GFI	Goodness of Fit Index
HF	High Frequency
HF _{nu}	High Frequency in Normalized Units
HRV	Heart-Rate Variability
IOTF.....	International Obesity Task Force
ITA.....	Intermittent Arousal Index
LF	Low Frequency
LF/HF Ratio	Low Frequency/High Frequency Ratio
LF _{nu}	Low Frequency in Normalized Units
MIA.....	Micro-Arousal Index
NNFI	Non-Normed Fit Index
NREM.....	Non-Rapid-Eye Movement

PDSS	Pediatric Daytime Sleepiness Scale
pNN50.....	Percent difference in successive Beat-to-Beat intervals greater than 50 ms
PNS	Parasympathetic Nervous System
PSG	Polysomnograph
REM.....	Rapid Eye Movement
RMSEA.....	Root Mean Square Error of Approximation
rMSSD	Square Root Mean Difference of Successive Beat-to-Beat intervals
R-R.....	Beat-to-Beat
RSA.....	Respiratory Sinus Arrhythmia
SA	Sinoartial
SDANN.....	Standard Deviation of the Average R-R Intervals for each 5 min Segment
SDNN.....	Standard Deviation of all Beat-to-Beat Intervals
SDNNI	Average Standard Deviation of all R-R Intervals for each 5 min Segment
SEM	Structural Equation Modeling
SNS.....	Sympathetic Nervous System
SPT.....	Sleep Period Time
SWS	Slow Wave Sleep
TP.....	Total Power
TST	Total Sleep Time
VLF.....	Very Low Frequency

INTRODUCTION

The Childhood Obesity Epidemic

Childhood overweight and obesity has been identified as an international epidemic (Ebbeling, Pawlak, & Ludwig, 2002; Hossain, Kavar, & El Nahas, 2007). The proportion of overweight and obese youth has been rising in both developed and developing countries at an astonishing rate (Wang & Lobstein, 2006). In the United States, one in every four youth aged 6-19 years old is overweight and 11% are obese (Nicklas, Baranowski, Cullen, & Berenson, 2001).

In Canada, using data from two nationally representative surveys, the prevalence of overweight and obesity increased substantially from 1981 to 1996 from 11-33% and 2-10%, respectively (Tremblay, Katzmarzyk, & Willms, 2002). Even more alarming, youth are becoming overweight and obese at a faster rate than adults (Flegal & Troiano, 2000; Tremblay et al., 2002; Willms, Tremblay, & Katzmarzyk, 2003), and as a result, adult-obesity related diseases are now being diagnosed in youth. Further, there is a tendency for obese children to remain obese adults (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997) with evidence supporting the idea of childhood obesity *tracking* (persistence of relative rankings) into adulthood. As example, childhood body mass index at ages 2-17 was associated with adult BMI 17.5 years later (Freedman, et al., 2005). These findings suggest that heavy children remain heavy as adults compared to thinner children.

Because obesity and its co-morbidities during childhood track into adulthood, there is an unprecedented impending threat of the economic burden on national health care costs. In the United States, ~300,000 deaths per year and over 30% of pediatric hospitalizations have been attributed to obesity and obesity-related co-morbidities

(Allison, Fontaine, Manson, Stevens, & Vanltallie, 1999; Wang & Dietz, 2002). Health costs associated with obesity have tripled from 1971-81 to 1997-99, representing more than \$127 million annually in the United States (Wang & Dietz, 2002).

Childhood Obesity and its Health Complications

Overweight and obesity confer risk to many health conditions such as chronic diseases, disability, and even premature death (Must & Anderson, 2000). Overweight and obese youth already show precursors for cardiovascular diseases, hypertension, increased plasma insulin concentrations, insulin resistance, impaired glucose tolerance (Freedman, Dietz, Srinivasan, & Berenson, 1999), and chronic inflammation (Ferguson, Gutin, Owens, Litaker, Tracy, & Allison, 1998; Ford, Galuska, Gillespie, Will, Giles, & Dietz, 2001). Excess weight has also been associated with Parkinson's disease, polycystic ovary disorder, gallbladder disease, asthma, sleep apnea, arthritis (knee and ankle) and orthopedic problems (Goulding, Jones, Taylor, Williams, & Manning, 2001; Must & Strauss, 1999; Wang & Dietz, 2002).

Although obesity is a risk factor associated with many health problems, risk depends on the location of excess adipose tissue. Central adiposity, an excess of adipose tissue mainly around the abdomen, and visceral obesity, an excess of adipose tissue inside the space within the intestines, stomach, and liver, are both associated with increased risk of impaired glucose tolerance (Blaha, Gebretsadik, Shintani, & Elasy, 2008), insulin resistance, type 2 diabetes (Caballero, 2003), atherogenic lipoprotein profiles, and cardiovascular diseases in youth (Flodmark, Sveger, & Nilsson-Ehle, 1994).

Interestingly, fat distributed in the lower portion of the body (i.e., hips, thighs, and buttocks) has lower health risks than central adiposity (Hartz et al, 1990). In addition to

physical health risks, psychological and mental disorders such as depression, low self-esteem, and social stigmatization are present in overweight and obese youth (Dietz, 1998; French, Story, & Perry, 1995; Must & Strauss, 1999). Thus, the dramatic rise in the prevalence of obesity and its related co-morbidities represents a severe threat to public health.

Measuring Obesity

Multiple techniques exist to accurately assess body composition and fat distribution. Considered the gold standard, dual energy X-ray absorptiometry (DEXA) scans estimate the distribution and the total amount of lean and fat mass as well as percent body fat (Mazess, Barden, Bisek, & Hanson, 1990). Underwater weighing measures body fat as the calculated difference between in- and out- of water measurements. While both of these techniques are reliable and valid, they are expensive procedures requiring specialized equipment, qualified personnel, and cumbersome procedures (Mazess et al., 1990). Bioelectrical impedance analysis applies a low electric current that flows through conductive (e.g., muscle, organs, body water) and non-conductive body tissue (e.g., fat) to assess the volume of lean and fat mass. It is a safe, noninvasive, and a validated method frequently used to assess percent body fat (Mazess et al., 1990).

Skinfold thickness and waist circumference are anthropometric measurements used to estimate body fat distribution. Skinfold thickness involves more invasive procedures that measure the amount of adipose tissue under the skin using standardized calipers by trained personnel. Waist circumference is less invasive and is considered a reliable indicator of central adiposity in youth (Blaha et al., 2008; de Ridder et al., 1992;

Fox, Peters, Armstrong, Sharpe, & Bell, 1993; Goran, Gower, Treuth, & Nagy, 1998; Lee, Song, & Sung, 2008; Taylor, Jones, Williams, & Goulding, 2000; Wells & Fewtrell, 2006). Waist circumference has high sensitivity (89%; correctly identifies youth with central obesity) and specificity (94%; correctly identifies youth without central obesity), compared to DEXA scans (Taylor et al., 2000).

Body mass index (BMI) is frequently used in epidemiological studies because it is an easy, noninvasive, and reliable indicator of overweight and obesity in youth (Barlow & Dietz, 1998; Kopelman, 2000). BMI is correlated with direct measures of fatness and it is validated against DEXA scans, underwater weighing (Freedman et al., 2005; Mei et al., 2002; Pietrobelli et al., 1998), waist circumference (Blaha et al., 2008; Savva et al., 2000), and skinfold thickness (Morrison, Barton, Biro, Daniels, & Sprecher, 1999; Morrison, Sprecher, Barton, Waclawiw, & Daniels, 1999). However, the usefulness of BMI values are limited as they do not indicate distribution of body fat or differentiate between fat and fat-free mass (Wells & Fewtrell, 2006).

Defining Obesity in Youth

Defining obesity in youth is challenging because body fatness depends on several factors, including sex, racial/ethnic backgrounds, age, and growth patterns (Rosner, Prineas, Loggie, & Daniels, 1998). In general, boys tend to show greater overweight and obesity prevalence rates than girls (Tremblay et al., 2002). Native American, Black, and Latin children are heavier and have greater abdominal and trunk adiposity than Caucasian children (Afghani & Goran, 2006). During childhood, growth patterns are similar between boys and girls; however, during adolescence, girls tend to gain weight composed mainly of fat, whereas boys gain fat-free mass (Maynard et al., 2001).

To account for the timing of growth patterns for boys and girls, the Centers for Disease Control and Prevention (CDC) developed cutoff values for age- and sex-specific BMI distributions (2000). Based on national surveys of American children, the CDC BMI growth charts reflect body composition and include percentile distributions for the weight status of children aged 2-18 years. The most recent growth charts (CDC, 2000) are considered the best reference on the established criteria for tracking growth patterns in youth (Kuczmarski et al., 2000), even though the curves are in part based on data from 1963-1995. BMI categories for youth include underweight (<5th percentile), healthy weight (5th to 85th percentile), overweight (>85th to ≤95th percentile), and obese (>95th percentile; CDC, 2000).

The International Obesity Task Force (IOTF) definitions are based on other BMI cutoff points developed as a screening tool to detect childhood obesity. They are based on the World Health Organization BMI categories used to identify overweight (BMI >24.9–29.9) and obese (BMI >30) adults (Kuczmarski, Carroll, Flegal, & Troiano, 1997). These adult cutoff points are linked with age- and sex- specific BMI cutoff points specific to childhood overweight and obesity (Cole, Bellizzi, Flegal, & Dietz, 2000). Both the CDC and IOTF criteria are the most commonly used BMI cutoff points used to assess body fatness in youth.

Association between Childhood Obesity and Sleep: Cross-Sectional Studies

Intriguingly, mirroring the increased trends of overweight and obesity, total sleep duration and sleep quality have decreased among children and adolescents across three decades (Iglowstein, Jenni, Molinari, & Largo, 2003). Recently, several cross-sectional

studies have found significant associations between decreased sleep duration and excessive weight status among youth.

von Kries and colleagues (2002) found a dose-dependent relation between increased adiposity (measured by BMI with height and weight and percent body fat with bioelectrical analyses) and reduced sleep duration in more than 6800 children aged 5-6 years. Children sleeping ≥ 11.5 hrs had the lowest risk of having excessive body fat and of being overweight/obese compared to children sleeping ≤ 10 hrs, even after adjusting for several confounders including parental obesity, socioeconomic status, birth weight, weight gain, time spent watching television, and snacking. Also, percent body fat in children sleeping ≤ 10 hrs per night doubled from 7-15.6%, compared to children sleeping ≥ 11.5 hrs each night.

Similarly, in the *Québec en Forme* Project (2006), Chaput and colleagues found that in 422 children aged 5-10 years, decreased sleep duration was significantly associated with greater body weight, waist circumference, and BMI in boys. Among girls, similar associations were observed, although the correlations did not reach significance. Even after controlling for potential risk factors associated with obesity (e.g., age, sex, parental obesity, parental education, family income, single parenthood, physical activity, breastfeeding time, and time spent watching television) results yielded that children sleeping only 8-10 hrs had significantly greater odds of being overweight/obese, compared to children sleeping >12 hrs each night. These findings have been replicated in multiple studies in toddlers and in children (cf. Gibson, Lambert, & Neate, 2004; Hui, Nelson, Yu, & Fok, 2003; Kagamimori et al., 1999; Padez, Mourao, Moreria, & Rosado, 2005; Sekine et al., 2002).

This relationship has also been reported among adolescents. Recently, Seicean and colleagues (2007) asked 529 adolescents aged 14-18 years to self report the number of hours they typically sleep and to estimate their height and weight to calculate BMI. A dose-response relationship between being overweight/obese and sleep duration was found. Even after adjusting for sex, age, irregular eating, health status, and caffeine intake, youth who reported sleeping <5 average had greater odds of being overweight, than youth who reported sleeping >8 hours per night. Even in cross-sectional studies using objective measures of sleep duration (versus self-report), the link between reduced sleep duration and obesity has been observed.

In the Heartfelt Study, Gupta and colleagues (2002) had 383 adolescents aged 11-16 years wear wrist actigraphs for 24-hours to continuously measure total sleep duration. Youth who slept fewer hours were heavier (greater BMI and percent body fat) than those who slept longer hours, even after adjusting for sex, age, race, and pubertal status. In fact, for every additional hour of sleep lost, the likelihood of being obese increased by 80%. In another study, Beebe and colleagues (2007) compared sleep of overweight and healthy youth between the ages of 10-17 years using multiple indices of sleep. Based on wrist actigraphy, overweight youth slept 38 minutes less, had poorer sleep quality and less continuous, more fragmented sleep compared to healthy controls. No significant differences between groups were observed with overnight polysomnograph (PSG). Overweight youth complained more about daytime sleepiness and reported sleeping ~30 minutes less on school nights and their parents perceived them as displaying more sleep problems (e.g., bedtime resistance, daytime sleepiness) compared to healthy controls.

Thus, heavier youth obtained less sleep, of poorer quality, and exhibited and complained of more sleep-related problems compared to their lean counterparts.

Collectively, these cross-sectional studies provided robust evidence suggesting that heavier youth sleep fewer hours than lean youth. Using multiple measures of adiposity (e.g., BMI, waist circumference, percent body fat) and sleep (e.g., actigraphy, PSG; Beebe et al., 2007; Gupta et al., 2002), a dose-response relation between short sleep duration and increased weight status was consistently observed in both children and adolescents. Additionally, although sex differences were reported in some studies (Eisenmann, Ekkekakis, & Holmes, 2006; Knutson, 2005), these results were inconsistent and pubertal status was not objectively measured, thus confounding the results.

In summary, multiple cross-sectional studies present strong evidence that corroborates the association between short sleep duration and childhood overweight/obesity. Unfortunately, it is impossible to identify a causal relationship and determine whether short sleep duration precedes the development of obesity or vice versa from cross-sectional studies. However, results from prospective studies provide evidence for a possible causal mechanism and suggest that short sleep duration predicts future development of overweight/obesity. Prospective studies examining the association between sleep duration and obesity are presented in the next section.

Association between Childhood Obesity and Sleep: Prospective Studies

Several studies have demonstrated a longitudinal relationship between the amount of sleep obtained and future development of obesity in youth (cf. Agras et al., 2004; Lumeng et al., 2007; Seicean, 2007). Reilly and colleagues (2005) followed 8234 children from birth to 7 years, and although sleep duration was not the main variable

being investigated, a dose-response relation between reduced sleep duration at age 30 months and obesity at age 7 years was found. Compared to toddlers sleeping ≥ 12.5 hrs per day, toddlers sleeping < 10.5 hrs had the highest odds ratio of becoming obese compared to toddlers sleeping 10.5-10.9 hrs or 11-11.9 hrs, even after controlling for numerous covariates (i.e., sex, socioeconomic status, birth weight, physical activity, and energy intake at 36 months).

Lumeng and colleagues (2007) examined sleep duration assessed by parental report when their child was 9 years and again at age 12 years. After controlling for sex, race, maternal education, change in sleep duration, and baseline BMI, reduced sleep duration at age 9 predicted greater BMI and overweight status at age 12. In fact 9-year olds were approximately 40% less likely to be overweight three years later based on every additional one hour of sleep they obtained. More importantly, even after controlling for changes in sleeping patterns during the transition from childhood to adolescence (Carskadon, Acebo, & Jenni, 2004), reduced sleep duration was independently linked to increased risk of future overweight status three years later.

Snell and colleagues (2007) found children who had short sleep duration, later bedtimes, and/or earlier wake times at age 3 had higher BMI values and greater risk of being overweight five years later, even after controlling for baseline BMI and overweight status. In fact, sleep duration at baseline accounted for 23.8% of the variance in BMI and 12.8% of the variance in overweight at follow-up.

Similar results have been reported for infant's sleep duration and future weight gain. Shorter sleep duration at 6 and 24 months predicted greater skinfold thickness and overweight status in children at 36 months (Taveras et al., 2008), even after taking into

account several covariates. In addition, frequent sleep problems such as irregular sleeping habits, trouble initiating sleep, and disruptive sleep during the first few years of life were associated with an increased risk of being overweight and almost a two-fold increase of being obese at age 21 (Al Mamun et al., 2007). Thus, these longitudinal results support the causal nature of the relationship between reduced sleep duration preceding the development of obesity, even after controlling for important covariates.

In a recent meta-analysis, Chen and colleagues (2008) examined the association between sleep duration and childhood obesity from twelve cross-sectional, three prospective, and two case-control studies. Across all studies, short sleep duration was associated with an increased risk for overweight/obesity for the entire general pediatric sample. For every hour of sleep obtained the probability of increased weight status decreased by roughly 9%. Sex differences were also examined; boys with short sleep duration had a greater risk of being overweight/obese than girls. However, no significant sex differences were found based on age and the studies did not consistently assess or control for pubertal status. Overall, this meta-analysis found a robust link between short sleep duration and increased weight status in youth.

In summary, these prospective studies had strong methodological designs, which included multiple time assessments, the use of validated sleep questionnaires, and large sample sizes. Therefore, the notion that short sleep duration precedes weight gain is supported by both cross-sectional and prospective studies. This relation does not appear short-lived, but rather encompasses significant predictive power for future risk of obesity. Evidence suggests that short sleep duration during childhood, especially during preschool years, may be a critical risk factor with long-term consequences for the development of

obesity several years later. Unfortunately, the mechanisms underlying the association between sleep duration and obesity have not been fully elucidated. Because sleep is highly integrated with several networks in the body it has been suggested that there exist several potential mediators underlying the link between sleep and obesity.

Potential Underlying Mechanisms

Some researchers have postulated that reduced sleep duration may be considered a stressor that leads to adverse modifications in several critical systems in the body. The *allostatic load* theory refers to the wear and tear on the body and has been used to explain the pathophysiological mechanisms underlying the relation between short sleep duration and weight gain (McEwen, 2006). During normal sleep and wake cycles, the neuroendocrine, immune, and autonomic nervous system adapt and change to maintain homeostasis or *allostasis* (McEwen, 2002). These systems reflect a highly interconnected network and even small changes in production (e.g., hormones) can cause damage to the entire network. Therefore, when sleep is reduced, changes within the network occur and an allostatic overload develops (McEwen, 2006). If this allostatic overload is prolonged (e.g., chronic sleep loss), it can have harmful behavioural, neurobiologic, and physiologic consequences (McEwen, 2006).

Sleep is recognized as an important regulator of hypothalamic-pituitary processes that affect food consumption, energy expenditure, and metabolism; all of which are associated with circadian rhythms (Laposky, Bass, Kohsaka, & Turek, 2008). Following acute and chronic sleep restriction, animals show increased basal corticosterone and adrenocorticotrophic hormone concentrations (Meerlo et al., 2002) and adult humans show increased basal adrenocorticotropin and cortisol concentrations (Steiger, 2002). The

regulation and variation of cortisol concentrations are altered, with significantly greater cortisol levels in the morning (Capaldi et al., 2005), late afternoon, and early evening having been reported following acute and chronic sleep loss (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005). In addition, increased basal cortisol secretion is associated with longer sleep latency, poor perceived sleep quality, more fragmented sleep, and poor sleep patterns in adults (Vgontzas et al., 2003) and children (Hatzinger et al., 2007).

Glucose metabolism is also found to be influenced by sleep loss, with short sleepers (≤ 5 hrs) having an increased risk of diabetes up to 22 years later, compared to long sleepers (7-8 hrs; Ayas et al., 2003; Nilsson et al., 2004). Experimental studies show that sleep restriction leads to significantly elevated glucose concentrations and reduced insulin sensitivity, even after controlling for food intake (Spiegel et al., 2005). In fact, the values of glucose concentrations obtained during sleep restriction were similar to those observed in individuals with impaired glucose tolerance and patients with type 2 diabetes. Further, loss of slow wave sleep is associated with reduced insulin sensitivity, reduced compensatory insulin secretion, and an overall poor glucose homeostasis in healthy young men (Tasali et al., 2008).

Sleep also influences metabolic hormones associated with appetite. Two of many appetite-regulating hormones include the appetite-stimulating ghrelin and appetite-inhibiting leptin. Ghrelin is a hormone that is mainly synthesized in the stomach. It stimulates caloric intake, down regulates energy expenditure, and conserves fat in the body (Horvath, Diano, Sotonyo, Heiman, & Tschop, 2001). In humans, ghrelin levels increase before food intake (Wren et al., 2001) decrease after meals, and increase again approximately 1.5-2 hours after eating, in parallel with feelings of hunger (Shinya et al.,

2005). Leptin is a hormone that is synthesized in adipose tissue. It is released into the blood and regulates food intake and energy metabolism (Sunter, O'Shea, & Bloom, 1997). Fasting and energy restriction decrease leptin levels and increase sensations of hunger (Keim, Stern, & Havel, 1998; Tritos & Mantzoros, 1997). Eating results in elevated levels of leptin (Tritos & Mantzoros, 1997), inducing satiety, decreasing appetite, and increasing energy expenditure (Campfield, Smith, Guisez, Devos, & Burn, 1995; Pelleymounter et al., 1995).

Circulating leptin and ghrelin levels show opposing patterns with leptin secretion increasing approximately 50% during the night compared to the day (Sinha, Sturis et al., 1996; Tritos & Mantzoros, 1997) and ghrelin levels decreasing during the second half of the sleep period (Dzaja et al., 2004; Sinha, Ohannesian, et al., 1996). However, short sleepers (5 hrs) showed 18% lower leptin levels and 15% higher ghrelin levels compared to long sleepers (8 hrs) the following morning, even after controlling for BMI (Taheri, Lin, Austin, Young, & Mignot, 2004). Following acute and chronic sleep deprivation, Spiegel and colleagues (2004) found young healthy men showed an earlier acrophase of leptin (by 2 hrs), a 26% reduction in the nocturnal acrophase of leptin, and an overall 19% decrease in mean leptin concentrations, irrespective of caloric intake, physical activity, and BMI. Further, hunger increased by 24% and appetite for high caloric-dense foods such as sweets, salty snacks, and starchy foods increased by 23%. Short sleep duration is associated with unhealthy eating behaviours (e.g., reduced intake of vegetables, irregular eating habits, snacking between meals, and increased food seasoning; Imaki, Hatanaka, Ogawa, Yoshida, & Tanada, 2002; Ohida, Kamal, Uchiyama, et al., 2001). This suggests that short sleep duration adversely affects the

appetite-regulating hormones which are interestingly also associated with changes in perceived hunger and appetite.

Numerous studies have provided evidence demonstrating that sleep loss adversely influences a variety of physiological processes important in metabolism and hunger regulation (Spiegel, Leproult, & Van Cauter, 1999). Therefore, sleep loss, through multiple pathophysiological alterations, offers a plausible biological link as an indirect, yet vital role in the development and maintenance of obesity. However, given that autonomic dysfunction is related with adverse physiological correlates which in turn confer risk to obesity, autonomic dysfunction has been proposed as *one* potential mediator elucidating the association between sleep and the etiology of obesity.

Autonomic Functioning

The autonomic nervous system (ANS) is the neural part of the nervous system which is responsible for homeostasis in the body, vital organs (e.g., lungs, kidneys), endocrine and exocrine glands, as well as the cardiovascular system (Furness, 2006). The ANS is comprised of two branches: the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). The PNS and SNS work in balance on almost every organ of the body with differing yet, complimentary effects.

The PNS is derived from the cranial nerves as well as in the spinal cord and is associated with the conservation of energy, allowing the body to “rest and digest” (Snitker, Macdonald, Ravussin, & Astrup, 2000). Activation of the PNS involves a reduction in heart rate, cardiac output, and blood pressure, facilitates digestion, and restores energy (Snitker et al., 2000). In the PNS, acetylcholine is the neurotransmitter

responsible for many of these effects on the body; especially slowing of the heart rate (Berntson et al., 1997).

The SNS also derives from the spinal cord and prepares the body for the “fight or flight” response (Snitker et al., 2000). This involves vasoconstriction (reducing blood flow), increasing metabolic changes of fat and glucose, and higher blood pressure, cardiac output, and heart rate (Snitker et al., 2000). In the SNS, sympathetic neurotransmitters, such as the catecholamines epinephrine and norepinephrine, increase cardiovascular functioning when stimulated (i.e., heart rate; Berntson et al., 1997). The SNS affects adipose tissue, thermoregulation, and resting metabolic states (Tentolouris, et al., 2003).

The ANS is the regulator of organ function throughout the body, including temperature, respiration, digestion, blood pressure, and heart rate (Berntson et al., 1997). The sinoatrial (SA) node, referred to as the heart’s pacemaker, generates electrical signals throughout the heart allowing it to contract and pump blood (Spiers, Silke, McDermott, Shanks, & Harron, 1993). Specifically, the ANS controls the frequency at which the SA node triggers the initiation of each heart beat. Each time the SA node fires, a tall spike, called an R-wave, is detected by an electrocardiogram (ECG). An ECG is an electrical signal waveform used to discriminate and quantify normal and abnormal beat-to-beat changes in the heart between the two branches of the ANS (Berntson et al., 1997). The number of R-waves per minute provides the average heart rate for that minute (Berntson et al., 1997).

There are five points (P, Q, R, S, and T) on the ECG waveform. These points follow a specific order and correspond to specific events occurring in the heart. The P-

point represents the contraction of the heart's atria pumping the blood into the heart's ventricles. The time it takes for the heart's ventricles to fill up with blood is represented by the P-Q interval. Then, the electrical signal divides into the right and left branches on the heart's septum. This is depicted by the Q-point. The electrical signal leaves the right and left bundle branches via the Purkinje fibers and spreads rapidly across the ventricles causing them to contract. The left ventricle contracts an instant before the right ventricle and is represented by the R-wave. The S-point represents the contraction of the heart's right ventricle. Finally, the relaxation of the heart's ventricles is denoted by the T-point and then the cycle continues.

Autonomic Dysfunction and HRV

In healthy individuals, the beat-to-beat (R-R) intervals, (i.e., one R-wave to the next R-wave) are not evenly spaced (Berntson et al., 1997). These beat-to-beat fluctuations in heart rate are measured by heart rate variability (HRV). HRV is influenced by several factors, including age (Shannon, Carley, & Benson, 1987), gender, ethnicity (Sloan et al., 2008), circadian rhythms (Malpas & Purdie, 1990), exercise (Berntson et al., 1997), and postural changes (Pomeranz et al., 1985). HRV generally decreases between the ages of 5 and 10 years and continues to decrease with age (Finley, Nugent, & Hellenbrand, 1987).

Because the cardiovascular system is modulated by the ANS, HRV is regarded as a useful, reliable, and noninvasive parameter to assess the sympathetic and parasympathetic functions of the ANS in newborns, children, adolescents, and adults (Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology [TFESCNASPE], 1996). Fast-variations (high HRV) reflect

parasympathetic (vagal) modulation and slower variations (low HRV) reflect a sympathetic dominance over the parasympathetic tone (Berntson et al., 1997). The overall balance between the sympathetic tone and the parasympathetic tone of the autonomic state is referred to as *sympathovagal balance* and can also be assessed with HRV (Goldberger, 1999).

Measuring HRV

Currently there are several methods and techniques used to assess the ANS. The gold standard is selective pharmacological blockade of either the SNS or the PNS (Berntson et al., 1997). Typically, researchers use beta-blockers to block the SNS and atropine to block the PNS. For example, blocking the parasympathetic branch will alter the heart's response or functional state and thus, provide information on basal contribution of that branch to the autonomic control of the heart (Berntson et al., 1997). Similarly, pharmacological blockade can also provide an estimated index of the opposite branch's contribution (i.e., sympathetic activity) based on the remaining cardiac control after the blockade of the initial branch (i.e., parasympathetic activity; Berntson et al., 1997). Although pharmacological blockades are expensive and have some limitations, such as incomplete blockades these direct measures, after cautious selection of drugs or dosages, provide valid and accurate assessments of cardiac autonomic function (Berntson et al., 1997). Based on this method, frequency components of HRV have been established (Akselrod, et al., 1985; Grossman & Kollai, 1993).

Other accurate, yet more invasive techniques have been developed to assess ANS. During stressful states, the SNS is activated, and large quantities of epinephrine and norepinephrine are released into the body, causing an increase in blood pressure and

cardiovascular activity. Thus, based on plasma, turnover, and urinary excretion of catecholamine concentrations (Liatis, Tentolouris, & Katsilambros, 2004) as well as cardiac norepinephrine spillover, autonomic function can be assessed (Ewing, Martyn, Young, & Clarke, 1985). Microneurography is another approach which directly measures muscle sympathetic nerve activity (Eckberg & Sleight, 1992) and is associated with cardiac norepinephrine spillover (Wallin et al., 1992).

Less invasive methods (e.g., orthostatic tests, Valsalva maneuver) also can be used to assess heart rate and blood pressure responses (Berntson et al., 1997). Although there are numerous specialized techniques, the two most commonly and widely used approaches to quantify HRV and autonomic function are *time* and *frequency* (power spectral) *domain analyses* (Berntson et al., 1997). Because accurate readings of the R-waves are required for time and frequency domain analyses, continuous ECG recordings are used to calculate HRV (Berntson et al., 1997).

Time Domain Analysis. HRV time domain methods are based on statistical calculations. There are two types of variables: indices derived from the direct measurement of R-R intervals and indices derived from the differences between successive R-R intervals (Spiers, et al., 1993). Time domain methods derived from direct measurement include the standard deviation of all the R-R intervals (SDNN), the standard deviation of the average R-R intervals for each 5 minute segment within a specified time period (SDANN), and the average standard deviation of all R-R intervals for each 5 minute segment within the ECG recording time (SDNNI; Kleiger, Stein, Bosner, & Rottman, 1992).

Time domain methods derived from the differences between R-R intervals include the percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart (pNN50) and the square root mean difference (in ms) of successive R-R intervals (rMSSD; Kleiger et al., 1992). Although all time domain indices provide information on the variability of the heart rate fluctuations, they are not identical; each index provides unique information. For example, the SDNN provides information on the total variance (power); and, the rMSSD and pNN50 reflect high frequency fluctuations (parasympathetic tone) and are inversely correlated with sympathovagal balance (Kleiger et al., 1992).

Frequency Domain Analysis. Frequency domain analysis provides precise information on the number of heart rate fluctuations per second (Lahiri, Prince, Kannankeril, & Goldberger, 2008). The Fast Fourier Transform algorithm is used to calculate the maximum variability in the heart's rhythm based on a range of frequency-specific oscillations of the R-R intervals (Lahiri et al., 2008; Spiers et al., 1993). These frequencies, when categorized into power bands, reflect different branches of the cardiac system. Total power (TP) covers all the frequencies and is used as an index of all of the variance over the recording period (Malliani, Pagani, & Lombardi, 1994). Very low frequency (VLF) ranges from 0.0033–0.04 Hz and has been reported to be influenced by thermoregulatory processes (Kitney, 1980). Low frequency (LF) ranges from 0.04–0.15 Hz, and although many researchers have proposed that the LF reflects solely sympathetic activity, other studies indicate that LF reflects the aggregate influences of *both* sympathetic and parasympathetic branches of the ANS (Akselrod et al., 1981; Berntson et al., 1997). High frequency (HF) power ranges from 0.15–0.40 Hz and represents the

parasympathetic activity of the ANS affecting the SA node (Berntson et al., 1997; Pomeranz et al., 1985).

Another frequency component is respiratory sinus arrhythmia (RSA), which ranges from 0.15–0.40 Hz (Berntson et al., 1997). Because the rate of breathing influences the frequency at which the SA node fires, and thus influences the heart rate, slow (e.g., exhalation) or fast (e.g., inspiration) breathing will produce differences in HRV (Berntson et al., 1997). For example, during inspiration heart rate increases, while during expiration, heart rate decreases (Berntson et al., 1997). RSA is mediated by several parasympathetic parameters, such as cardiac vagal tone (the differences in mean HRV between a resting baseline and a complete vagal blockade), baroflex activity, and respiratory function, and thus, is generally used as an indicator of the parasympathetic branch of the ANS (Berntson et al., 1997).

Although HRV can be assessed by various techniques, time- and frequency-domain analyses demonstrate significant associations between both types of analyses. Carrasco and colleagues (2001) found HF power was correlated with SDNN, pNN50, and rMSSD. LF power, reflecting both sympathetic and parasympathetic tone, correlated with SDNN, pNN50, and rMSSD. LF/HF ratio, indicative of sympathovagal balance, showed an inverse association with SDNN, pNN50, and rMSSD. Thus, either time or frequency HRV quantification methods can reliably assess autonomic influences on the cardiovascular system.

Absolute and Normalized HRV Values. The LF and HF variables quantified by frequency domain analyses can be assessed in *absolute values* (milliseconds squared) or *normalized units* (fractional or percentage). Absolute values are the raw values generated

by the Fast Fourier Transform algorithm and reflect the modulation of either parasympathetic (HF) or both sympathetic and parasympathetic modulation (LF) of the ANS. However, because absolute values tend to show a long-tailed right skewed distribution and are more difficult to interpret, normalized values are preferred (Burr, 2007).

Normalized units are not direct calculations of raw data, but rather are secondary analyses computed only after LF and HF bands are statistically calculated (Burr, 2007). Normalized values, denoted with the suffix “*nu*” (e.g., *LFnu* and *HFnu*), provide relatively easy interpretations of the proportion of each frequency bands, while controlling for total power within the power spectrum (Lahiri et al., 2008; Rabbia et al., 2003). Thus, given the appeal and ease of normalized values, many researchers report HRV measures in normalized units. Normalized units do not provide unique information about the modulation of either sympathetic or parasympathetic tones, but instead provide information on the *overall* modulation of both branches of the ANS (Burr, 2007). Consequently, problems in data interpretation have been noted (Busek et al., 2005; Robert et al., 2006; Sforza et al., 2007). Thus, it is highly recommended that researchers report both absolute and normalized values of the frequency power bands in order to describe completely the distribution of power in spectral components (TFESCNASPE, 1996).

Cardiovascular Autonomic Dysfunction and HRV

When assessing the cardiovascular system, an equal balance between the sympathetic and parasympathetic branches of the ANS represents a healthy autonomic function in the heart. However, when there is a dysregulation between the two branches

of the ANS, *cardiovascular autonomic dysfunction* develops (Messerli, Nunez, Ventura, & Snyder, 1987). Cardiovascular autonomic dysfunction, a marker of negative cardiovascular risk (e.g., stroke, coronary heart disease, myocardial infarction, angina, etc.), is characterized by a substantial decrease in HRV, denoted by reduced parasympathetic input (indicative of an inactive or damaged PNS) and an elevated sympathetic tone (Berntson et al., 1997).

The decreased vagal tone and increased sympathetic tone confer risk for ventricular fibrillation, a medical condition which causes disruptions in the contractions of the ventricles of the heart, preventing blood circulation and even causing death (Billman & Hoskins, 1988; Hull et al., 1990; Molgaard, Sorensen, & Bjerregaard, 1991). Conversely, an increased vagal tone is considered a protective factor because as it increases, the electrical signal that stimulates coordinated contractions in the ventricles of the heart allows blood to pump into the body (Berntson et al., 1997; Stein & Kleiger, 1999).

Autonomic dysfunction among adult patients is associated with chronic renal failure (Agarwal, Anand, Sakhuja, & Chugh, 1991), essential hypertension (Radaelli et al., 1994), diabetes (Stein & Kleiger, 1999), coronary artery disease (Dekker et al., 2000), left ventricular dysfunction (Nolan et al., 1992) congestive heart failure (Casolo, et al., 1989; Mortara et al., 1994), and obesity (Aronne, Mackintosh, Rosenbaum, Leibel, & Hirsch, 1997). Decreased parasympathetic tone is a significant risk factor for sudden cardiac death in post myocardial infarction patients (Hartikainen, Fyhrquist, Tahvanainen, Lansimies, & Pyorala, 1995), and increased sympathetic activity has been

linked to a five-fold increased likelihood of mortality in post-myocardial infarction patients up to 2½ years later (Bigger et al., 1992; Wolf, et al., 1978).

Even among healthy adult populations, prospective studies show that dampened HRV is a significant predictor of the development of hypertension (Liao et al., 1996), new cardiac events, as well as morbidity, and mortality, even after controlling for other clinical co-morbidities (Dekker et al., 1997; Tsuji et al., 1996). In youth, autonomic dysfunction is associated with diabetes (Akinci, Celiker, Baykal, & Tezic, 1993), atrial septal defect (Finley, Nugent, Hellenbrand, Craig, & Gillis, 1989), and obesity (cf. Kaufman et al., 2007; Rabbia et al., 2003). Interestingly, autonomic dysfunction is also linked with sleep disorders in adults and in children, including chronic fatigue syndrome (Stewart, Weldon, Arlievsky, Li, & Munoz, 1998), sleep-disordered breathing (O'Brien & Gozal, 2005), obstructive sleep apnea, and insomnia (Bonnf & Arand, 1998).

Indeed, based on this relation, recent data suggests that sleep loss (i.e., reduced sleep duration), through the dysregulation of ANS, may be a risk factor involved in the development of eventual health problems (Meerlo, Sgoifo, & Suchecki, 2008). Given that sleep is considered a time for the heart to rest and reduce its workload, sleep loss can actually increase the heart's workload, especially during specific sleep stages when the vagal tone relaxes the heart and cardiac output (Wolk, Gami, Garcia-Touchard, & Somers, 2005). In fact, sleep loss is associated with several adverse health conditions, including psychiatric disorders (Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997), heart disease (Ayas et al., 2003), and obesity (Patel, Malhotra, White, Gottlieb, & Hu, 2006). Thus, it is postulated that autonomic dysfunction is one pathogenic mechanism underlying the relation between sleep and obesity.

Sleep

One-third of human life is spent sleeping; and therefore, it is no surprise that sleep has a profound impact on fundamental homeostatic mechanisms in the body (Thompson, Ackermann, & Horner, 2001). Sleep is an intricately organized process regulated by the *two-process model* which involves the interaction between homeostatic and circadian processes (Achermann & Borbely, 2003; Daan, Beersman, & Borbely, 1984). The homeostatic process attempts to keep the amount of prior wakefulness and sleep balanced, so that during wakefulness sleep propensity (sleepiness) increases and will compensate for sleep loss (Achermann & Borbely, 2003).

The other component of sleep involves the circadian timing system, which regulates behavioural and physiological processes (Rosenwasser & Turek, 2005). It is regulated by the suprachiasmatic nucleus (Weaver, 1998) and follows a rhythmic pattern over 24 hours (Laposky et al., 2008). Circadian markers (e.g., melatonin and core body temperature; Cajochen & Dijk, 2003) and clock genes have been associated with “early birds” (morning preference) or “night owls” (evening preference; Archer et al., 2003; Duffy, Dijk, Hall, & Czeisler, 1999). Circadian rhythms affect the timing and structure of sleep (Dijk & Czeisler, 1995).

Sleep Structure

Sleep is composed of two major states: rapid eye movement (REM) and non-rapid eye movement (NREM). REM is further divided into tonic and phasic states. Tonic sleep occurs throughout REM sleep and includes muscle atonia and increased brain activity (Goldsmith, Casola, & Varenbut, 2006). Conversely, phasic states appear intermittently during REM sleep and are characterized by bursts of rapid eye movements,

muscle twitches, and vasoconstriction (Goldsmith et al., 2006). REM is characterized by the greatest frequency and intensity of dreaming and is associated with the consolidation of memory and learning (Maquet, 2001). Generally, during REM sleep there is an increase in heart rate, blood pressure, and respiration compared to NREM sleep (Goldsmith et al., 2006; van Eekelen, Varkevisser, & Kerkhof, 2003).

NREM sleep is subdivided into four stages: 1, 2, 3 and 4. Stage 1 reflects the transition from wake state to sleep. Stages 1 and 2 are referred to as light NREM sleep because the arousal threshold is lower. Conversely, stages 3 and 4 are referred to as deep or slow-wave-sleep (SWS), as these stages are characterized by an increase in slow wave activity or delta waves and the arousal threshold is higher (Gais, Plihal, Wagner, & Born, 2000). In particular, SWS is recognized as providing the most quality and restorative sleep and is involved in memory transfer and consolidation from daytime activity (Gais et al., 2000; Stickgold, 2005; Stickgold, Hobson, Fosse, & Fosse, 2001). During NREM sleep, brain activity, heart rate, cardiac output, breathing, and blood pressure decrease and are stable compared to wakefulness and REM sleep (Goldsmith et al., 2006; van Eekelen et al., 2003).

Throughout the night, sleep is divided into a 90-110 minute cycle of REM and NREM sleep that is repeated 4-6 times during the night. During the earlier portion of sleep, NREM sleep is predominant compared to REM sleep, with approximately 2-5% in Stage 1, 45-50% in Stage 2 (characterized by sleep spindles and K complexes), 5-8 % in Stages 3 and, 10-15% in Stage 4 (Rechtschaffen & Kales, 1968). As the night progresses, REM sleep becomes gradually longer and is predominant compared to

NREM sleep, occupying 20-25% of sleep (Rechtschaffen & Kales, 1968; Thompson et al., 2001).

Measuring Sleep

There are several ways to assess sleep duration and sleep quality. Most are subjective methods with questions asking participants to estimate their bed- and wake-times, the number of hours of sleep they obtain each night, and to rate their sleep quality on a Likert scale. Validated questionnaires are also used to determine sleep problems related to falling asleep, snoring, night terrors, parasomnias, and daytime sleepiness. Other methods include sleep diaries, which ask the participant to record the activities they are doing throughout the entire day, including what time they started and ended the activity (e.g., sleeping, etc.). Sleep diaries are usually filled out for 4-7 days to determine typical sleep/wake patterns in participants. Sleep diaries are a reliable method for estimating the amount of time spent on an activity and have been cross-validated with objective measures of sleep duration (Gaina, Sekine, Chen, Hamanishi, & Kagamimori, 2004; Robinson, 1985).

Wrist actigraph is an objectively measured method based on a non-invasive small device worn like a watch on the non-dominant arm for 5-7 days (Lockley, Skene, & Arendt, 1999). It monitors and calculates the participant's overall motor activity during the day and night, and calculates "resting" and active periods. Sleep duration and sleep quality based on disturbed or active movements during the night can be assessed and are considered reliable measures of sleep (Lockley et al., 1999).

Because there are distinct physiologic processes that occur during sleep, it is deemed most accurate to capture and categorize distinct sleep stages with

polysomnograph (PSG), the gold standard of measuring sleep. PSG continuously records physiological parameters, including brain wave activity and eye activity, that define wakefulness and sleep and is used for diagnosing a wide range of sleep disorders (e.g., obstructive sleep apnea), measuring sleep duration, sleep efficiency, and sleep latency (e.g., sleepiness), as well as quantifying sleep architecture (i.e., percent of time spent in each sleep stage) in adults and children (Acebo et al., 1996).

PSG is usually conducted during a single night in a laboratory or hospital setting by a trained technician to ensure the quality of the study. The minimal requirements for a sleep study typically include at least one channel for electroencephalogram (EEG) to detect cortical brain activity, two electroculograms (EOG) to detect right and left eye movements, and one electromyogram (EMG) to detect submental muscle and postural movements (Rechtschaffen & Kales, 1968). In addition, other measures can be used including a nasal cannula to measure airflow, pulse oximeter to measure blood oxygen levels, and an ECG monitor to measure heart rate. Trained sleep technicians manually review the raw data on consecutive epochs lasting 30 seconds and score sleep stages based on the standard rules of Rechtschaffen and Kales (1968). PSG is also used to measure daytime sleepiness, referred to as a state with an increased risk of falling asleep (Fallone, Owens, & Deane, 2002). Sleep latency is the time (in min) it takes to fall asleep; the shorter the latency, the greater sleepiness (Carskadon et al., 1986).

Although PSG in the laboratory is the gold standard of measuring sleep, it is expensive, invasive, and may affect the quality of sleep because it is conducted in an unfamiliar setting. Recently due to technological advancements in developing ambulatory equipment, there has been an increase in the use of unattended home PSG

recordings; that is, no certified technician attaching the electrodes, but simply teaching the participants how to attach the electrodes. Evidence suggests that home PSG sleep studies, with proper signal detection and low impedance, can be used to obtain accurate physiological data, in spite of age, sex, and BMI of participants (Iber et al., 2004; Kapur et al., 2000).

Sleep and its Correlates

Quality uninterrupted sleep is associated with physical growth and health, disease resistance, injury prevention, and emotional stability (Carskadon, 1999; Kelman, 1999). Conversely, lack of quality sleep adversely impacts daily living, reduces quality of life, and is associated with poor health status (Leger, Scheuermaier, Philip, Paillard, & Guilleminault, 2001). Disturbed sleep and longer sleep latency predict an increased all-cause mortality, even after adjusting for age, gender, medical comorbidities, and psychological disorders (Dew et al., 2003).

Unfortunately, due to the changes of modern day life, the importance of sleep has been less emphasized. Over the past 40 years sleep duration has decreased by 1-2 hours (Kripke, Simons, Garfinkel, & Hammond, 1979; National Sleep Foundation [NSF] Survey, 2000; 2001). The number of young adults sleeping less than 7 hours has increased more than two-fold from 15.6% in 1960 to 37.1% in 2001 (Kripke et al., 1979; NSF Survey, 2000; 2001). Currently, based on recommended sleep duration for youth (NSF, 2006), 15 million youth do not obtain the necessary amount of sleep (Smaldone, Honig, & Byrne, 2007).

Adequate sleep requirements are age-dependent, with younger children needing more sleep and older youth needing less sleep (Heussler, 2005). According to the

National Sleep Foundation (NSF, 2006) children aged 5-12 years should be getting 9-11 hours each night and adolescents should be getting approximately 8.5-9.5 hours each night (Carskadon, 1999). Given that sleep is a critical time in which important physiological processes occur, it is no surprise that sleep problems contribute a large portion to the economic burden of direct (e.g., managing sleep disorders), indirect (e.g., work-related injuries, production loss), and non-financial health costs (e.g., premature death, diminished quality of life); and thus, sleep is especially critical for the healthy development of children (Hillman, Murphy, Antic, & Pezzullo, 2006).

Sleep in Youth

Processes related to maturation and environment influence and change sleep structure and sleep duration from childhood to adolescence. In general, total sleep duration decreases from ~15 hours at 6 months to 8 hours at 16 years (Iglowstein et al., 2003). During the first year, daytime sleeping gradually decreases and night time sleeping gradually increases, incorporating 84% of total sleep duration; thereafter a pronounced reduction in night time sleep is observed (Iglowstein et al., 2003). Five to 6 year-olds sleep ~11 hours (Iglowstein et al., 2003; Wolfson, 1996), 7-9 year-olds sleep ~10.5 hours, and 10-12 year-olds sleep 9.3 hours each night (Howard & Wong, 2001; Iglowstein et al., 2003; Stein et al., 2001). Additionally, across development, children show decreased REM sleep and increased SWS (Carskadon & Dement, 1987; Heussler, 2005).

During adolescence, hormonal, developmental, and social changes accompany marked changes in sleep patterns (Anders, Carskadon, Dement, 1980). Adolescents' have later bed times (11 p.m. or later) but unchanged wake times, have longer sleep

latencies, and experience more awakenings and fragmented sleep compared to children (Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998; Gau & Soong, 2003; Iglowstein et al., 2003). In addition, sleep duration reduces from 9 to 8.1 hours each night, although their sleep need does not decrease (Carskadon, 1999).

Sleep Problems in Youth

Recent studies have found that almost 50% of adolescents report sleeping 1-2 hours less per night than the recommended 9 hours per night and ~60% report feeling too tired or sleepy during the day (NSF Survey, 2006). Daytime sleepiness is associated with adversely impacting youth's daily activities, mood, driving, and academic performance (Fallone et al., 2002). About 20% of students report falling asleep while doing their homework, 25% fall asleep during school, and 14% are late or miss school at least once a week due to oversleeping (NSF Survey, 2006).

Furthermore, worldwide studies have estimated that between 20-41% of children (Archbold, Pituch, Panjabi, & Chervin, 2002; Sadeh et al., 2000) and 6-37% of adolescents (Liu, Uchiyama, Okawa, & Kurita, 2000) report sleep problems related to initiating and maintaining sleep, daytime sleepiness, and poor sleep quality. Additionally, poor sleep quality is associated with disturbances in behaviour and mood in children (Lavignen et al., 1992; Sadeh, Gruber, & Raviv, 2003). Other problems include parasomnias (e.g., nightmares, night terrors, sleep walking) and bedtime resistance (e.g., stalling to go to bed or frequent times getting out of bed before sleeping) (Owens, Spirito, & McGuinn, 2000; Sadeh et al., 2000; Stein et al., 2001).

These sleep problems may confer risk for sleep debt/deprivation, daytime sleepiness, prolonged sleep delay onset, and insomnia in both children and adolescents

(Hoban & Chervin, 2001), all which have been linked to emotional and behavioural problems as well as biological and physiological processes.

Sleep Loss and Sleep Structure

Following sleep loss, only up to 85% of sleep can be recovered, suggesting that short sleepers may carry a sleep debt (Klerman & Dijk, 2005). In a clinical study, healthy adults whose sleep was restricted to 4, 6, or 8 hours over 2 weeks showed no signs of compensation in either sleep duration or time spent in SWS during sleep opportunities (Van Dongen, Maislin, Mullington, & Dinges, 2003). During sleep debt conditions, a significant decrease in the time spent in light NREM (Stages 1 and 2) and REM sleep occurs and this is compensated for with an increase time spent in SWS (Stages 3 and 4). Conversely, during sleep recovery, an increase time spent in light NREM and REM sleep occurs (Spiegel et al., 1999; 2004). These changes reflect alterations in sleep structure, with the suppression of REM sleep and preservation of SWS during sleep restriction, and an increase rebound of REM sleep during sleep recovery.

Adequate “Normal” Sleep and Autonomic Dysfunction

The ANS regulating cardiovascular function demonstrates distinct changes as a result of sympathetic and parasympathetic influences throughout the sleep cycle. PSG and ECG monitoring are used to identify rapid fluctuations of cardiac activity during sleep stages. During NREM sleep, the parasympathetic system exerts its greatest influence on the cardiac system with higher HF and lower LF components compared to REM (Ako et al., 2003; Bonnet & Arand, 1997; Burgess, Trinder, & Kim, 1996; Elsenbruch et al., 1999; Trinder et al., 2001). From Stages 1-4 there is a linear increase

in parasympathetic activity (van Eekelen et al., 2003) and a progressive and significant reduction in LF/HF ratio (Toscani et al., 1996). In particular, during Stage 2 there is a significant increase in sympathovagal balance (e.g., greater sympathetic activity and reduced parasympathetic activity) compared to SWS (Toscani et al., 1996).

Conversely, during the transition to REM sleep, a decrease in parasympathetic contribution and an increase in sympathetic dominance on the cardiac autonomic balance is observed (Berlad, Shlitner, Ben-Haim, & Lavie, 1993; Bonnet & Arand, 1997; Trinder et al., 2001; van Eekelen et al., 2003; Vanoli et al., 1995). Compared to SWS, REM is characterized by high HRV (Morgan et al., 1996), high LF_{nu} and LF/HF ratio (Ako et al., 2003), as well as high SDNN values (Brandenberger, Buchheit, Ehrhart, Simon, & Piquard, 2005).

In general, both parasympathetic and sympathetic activity contribute to cardiac modulation during normal sleep. However, there is sleep-stage dependence; vagal tone increases during NREM sleep, especially during SWS, and sympathovagal balance, that is sympathetic activation, increases during REM sleep.

Inadequate Sleep and Autonomic Dysfunction

Evidence in animals and humans has demonstrated a profound relation between sleep loss and autonomic dysfunction. Recently, Sgoifo and colleagues (2006) deprived rats of sleep for 48 hours by placing them on a slowly rotating wheel. Compared to controls, sleep deprived rats showed significantly reduced HRV, SDNN, and rMSSD values, reflective of parasympathetic withdrawal, during dark phases and especially during light phases. In addition, sleep deprived rats showed elevated heart rate for the following two days during sleep recovery. This experimental data demonstrates that

sleep loss alters cardiac autonomic function, inducing vagal withdrawal and sympathetic activation, and has lasting effects, particularly during resting conditions.

Similar results have been found in humans, with sleep loss leading to autonomic dysfunction. Numerous studies have demonstrated that total and partial sleep deprivation leads to significant increases in heart rate, blood pressure (Lusardi et al., 1996; Ogawa et al., 2003; Tochikubo et al., 1996), sympathovagal balance (Tochikubo et al., 1996), norepinephrine and epinephrine levels (Irwin et al., 1999; Tochikubo et al., 1996), and a significant decrease in HRV upon awakening compared to baseline (Ewing, Neilson, Shapiro, Stewart, & Reid, 1991).

In another experimental study, (Spiegel et al., 2004) 11 healthy men underwent 6 nights of 4 hours of sleep (sleep debt) and 6 nights of 12 hours of sleep (sleep recovery). Dampened 24-hour HRV was observed during sleep debt and sympathovagal balance significantly increased during sleep recovery. This sympathetic activation was especially seen in the morning (9 a.m.-1 p.m.) and afternoon (1-5 p.m.), suggesting that reduced sleep negatively influences cardiac autonomic function, especially in the early part of the day following sleep loss. Similar results were seen among healthy men who slept 4 hours for 5 consecutive nights (American Academy of Sleep Medicine, 2007).

Franco and colleagues (2003) compared HRV measures in 12 infants in either a sleep deprived (for 2 hours before napping) or control condition. Sleep deprived infants had significantly reduced HRV during REM sleep and significantly elevated LF power and LF/HF ratio during NREM sleep compared to the control condition ($p < .01$). Evidence suggests that even losing 2 hours of sleep has the potential to change cardiac

autonomic function. However, due to the limited sample size (low power), no statistical differences in the HF power were observed.

Brief arousals during sleep are also associated with an increase in heart rate and blood pressure (Ekstedt et al., 2004; Sforza et al., 2004) and both heart rate and blood pressure can remain elevated for up to 40 sec after the arousal (Blasi et al., 2003; Sforza et al., 2006). Compared to normal sleepers, individuals with sleep apnea or insomnia have an elevated heart rate and a progressive increase in sympathetic activity during sleep and wakefulness, (Narkiewicz & Somers, 2003) as well as a decrease in parasympathetic tone (Bonnet & Arand, 1997). Tasali and colleagues (2008) examined HRV in 9 healthy adults after three consecutive nights of SWS suppression, where acoustic stimuli of different frequencies and intensities were administered during SWS. LF_{nu} and LF/HF ratio were significantly elevated and HF_{nu} was significantly reduced after SWS suppression compared to baseline, even after controlling for breathing frequency and total sleep and wake time.

Indeed, data demonstrates that reduced, disturbed, and fragmented sleep are associated with autonomic dysfunction. In fact, being in a state of sleep debt alters the normal modulations of the ANS, and even losing 2 hours of sleep requires greater sympathetic activation to awaken and maintain wakefulness for the rest of the day.

Autonomic Dysfunction and Obesity

The imbalance between the parasympathetic and sympathetic systems of the ANS has been proposed to play a role in the onset of obesity in animal models and in humans. However, there are inconsistent findings in terms of where (i.e., PNS/SNS) the changes on the ANS occur and how they contribute to the etiology of obesity. Some studies

report changes in both PNS and SNS (Aronne et al., 1997; Emdin et al., 2000; Karason, Molgaard, Wikstrand, & Sjostrom, 1999); others report changes only in the PNS or only in the SNS (Richter, Geiss, Aleksic, & Schwandt, 1996; Rossi et al., 1989; Valensi et al., 1998); while others report *no* changes in either branch (Petretta et al., 1995; Piccirillo et al., 1996; Piha, Ronnema, & Koskenvuo, 1994).

Parasympathetic and Sympathetic Activity and Obesity. Regardless of methodology or sample size, reduced parasympathetic activity has been consistently found to be associated with obesity (Laederach-Hofman, Mussgay, & Ruddel, 2000; Richter et al., 1996; Rossi et al., 1989; Valensi et al., 1998). Parasympathetic underactivity has been found to be inversely associated with an increased percent body fat (Peterson, Rothschild, Weinberg, Fell, McLeish, & Pfeifer, 1988), BMI (Freeman, Weiss, Roberts, Zbikowski, & Sparrow, 1995), triglyceride concentrations (Sajadieh, Nielson, Rasmussen, Hein, & Hansen, 2005), and body weight (Hirsch, Leibel, Mackintosh, & Aguirre, 1991). In fact, a decline in HF power is associated with a 10% increase in body weight (Hirsch et al., 1991).

There is evidence suggesting an association between sympathetic activity and obesity, however, the mechanism by which sympathetic activation promotes obesity is unclear. However, because SNS influences the body through direct (e.g., β -receptors) and indirect effects (e.g., cardiovascular system), data suggests that sympathetic activation may contribute to the development of obesity. Findings are inconsistent as some research suggests low sympathetic activation leads to obesity and other research suggests that high sympathetic activation leads to obesity (Ravussin & Tataranni, 1996).

Animal Models. Numerous studies have investigated the link between autonomic dysfunction and obesity in animal models. One hypothesis is the *MONA LISA Hypothesis*, an acronym for “Most Obesities kNown Are Low In Sympathetic Activity” (Bray, 1991). The MONA LISA Hypothesis is based on animal studies which indicate that reduced SNS activity is associated with and is a causal risk factor for obesity (Bray, 1991; Bray, York, & Fisler, 1989). In genetically obese (e.g., *ob/ob* mouse and the *fa/fa* rat) and *ventromedial* hypothalamus-lesioned rodents (i.e., hypothalamic obesity) brown adipose tissue showed significantly decreased sympathetic and increased parasympathetic activity. Rodents also exhibited decreased energy expenditure and increased food intake and weight gain (Knehans & Romsos, 1983; Levin et al., 1983; Niiijima et al., 1984). Lesions to the *lateral* hypothalamus showed elevated sympathetic activity and reduced food intake and body weight (Bray, 1991). While, lesions to the *paraventricular* nucleus showed no significant changes in either autonomic function or food intake (Fukushima, Tokunaga, Lupien, Kemnitz, & Bray, 1987). These findings suggest that functions of the ANS are uniquely altered in both genetic and hypothalamic forms of obesity in rodents, yet depend on the location where damage to the hypothalamus is found.

In contrast, Landsberg (1986) suggests that sympathetic activation may contribute to the development of obesity. Experimental animal studies demonstrate that fasting suppresses sympathetic activity (Young & Landsberg, 1977), while increased food intake stimulates sympathetic activity (Young, Saville, Rothwell, Stock, & Landsberg, 1982). Recently, the sympathetic neural transmitter neuropeptide Y, an important orexigenic peptide involved in the production of white adipose tissue (Kalra & Kalra, 2004), has provided new evidence in elucidating the role of autonomic dysfunction in the

development of obesity. Neuropeptide Y is associated with increased food intake, especially of carbohydrate-rich foods, and has been found in the brain in many forms of experimental obesity (Kalra & Kalra, 2004). Kuo and colleagues (2007) demonstrated that in mice, under conditions of stress (i.e., cold and/or aggression) and a high-fat diet, the release of neuropeptide Y lead to the development of abdominal obesity within two weeks compared to controls. These findings support the notion that SNS plays an important role in the development of obesity and suggest that stress, (and hence activation of the SNS and withdrawal of the parasympathetic activity) may be a potential catalyst.

Human Studies. Several studies suggest that autonomic dysfunction is related to obesity in humans. Similar to most animal models, some research suggests that reduced sympathetic activity precedes obesity. In a prospective study of Pima Indians, decreased baseline measures of SNS activity (i.e., low urine epinephrine and norepinephrine concentrations) were associated with weight gain and the development of central obesity 3 years later (Spraul et al., 1993; Tataranni, Young, Bogardus, & Ravussin, 1997). Given that Pima Indians are genetically susceptible to obesity and diabetes and have low rates of hypertension, these results cannot be generalized to other populations (Tataranni et al., 1997). Recently, Flaa and colleagues (2008) found reduced concentrations of epinephrine to stress tasks at baseline predicted increased changes in BMI and waist circumference 18 years later in Caucasians. This may suggest that low SNS activity is involved in the etiology of obesity in animal models and in humans.

Conversely, other research suggests that sympathetic activation leads to obesity since sympathetic activation is associated with increasing body weight (Masuo, Mikami,

Ogihara, & Tuck, 2001; Vaz, Jennings, Turner, Cox, Lambert, & Esler, 1997), blood pressure, heart rate, and cardiac output (Reaven, Lithell, & Landsberg, 1996). Masuo and colleagues (2003) demonstrated that increased blood pressure and resting plasma norepinephrine at baseline predicted more than a 10% increase in BMI and subsequent weight gain over a 5-year period. In fact, greater baseline norepinephrine concentrations predicted individuals who were resistant to losing weight and predicted individuals who experienced rebound weight gain, even after successfully losing body weight over 24 months (Masuo et al., 2005). Branth and colleagues (2007) also demonstrated that sympathetic activation, triggered by stress, predicted the development of abdominal obesity, increased body fat and blood pressure in sailors 5 months later, despite regular exercise and a controlled diet.

These conflicting results may be attributable to methodology. The studies used different indices of sympathetic activation (i.e., norepinephrine concentrations, blood pressure, heart rate, etc.) and although all reflect sympathetic activation, differences were observed depending on the physiological outcome variable of interest (i.e., cardiac system, renal system, and adipose tissue; Davy & Orr, 2008).

Autonomic Dysfunction and Childhood Obesity

Nagai and colleagues (2003; 2004) had 42 obese and healthy youth wear an ECG monitor for 4.5-5 minutes. Based on absolute HRV values, heavier children showed reduced TP, LF ($p < 0.05$), and HF ($p < 0.01$) compared to controls. Consistent with the adult literature, obese children showed reduced parasympathetic activity. The authors claimed that based on the LF band, only sympathetic activity was significantly reduced among obese children. However, because the LF encompasses both sympathetic and

parasympathetic activity, these conclusions should be interpreted with caution. Unfortunately, neither time-domain analyses nor the LF/HF ratio were included in these studies, which could have provided additional information on the modulation and sympathovagal balance of the ANS in children.

Recently, Kaufman and colleagues (2007) investigated cardiac functioning of overweight/obese and healthy youth aged 10-13 years old. Based on 15 minutes of HRV data, overweight/obese youth showed signs of autonomic dysfunction with a sympathetically driven cardiac system (LF/HF ratio and $LFnu$) and a hypoactive parasympathetic tone ($HFnu$, SDNN, and rMSSD values) compared to healthy controls. Similar findings were found by Riva, *et al* (2001) when HRV was analyzed for 24 hours in non-diabetic obese ($n=23$) and in healthy adolescents ($n=14$). Overall, obese youth had lower SDNN, pNN50, and rMSSD values, reflective of reduced parasympathetic activity, compared to lean controls. Sympathetic activity showed an inverse pattern, with obese youth showing greater $LFnu$ and LF/HF ratio values, especially during the night compared to healthy youth.

Martini and colleagues (2001) had 32 non-diabetic obese youth and 13 healthy sex-matched controls wear an ECG Holter monitor for 24 hours. Obese youth had significantly lower pNN50, rMSSD values, and $HFnu$ values, and greater sympathovagal balance (LF/HF ratio) during the night compared to healthy controls. Also, BMI was inversely correlated with night time $HFnu$ values. Thus, obese youth showed significant parasympathetic under-activity and sympathetic over-activity, especially during nocturnal hours compared to their lean counterparts.

Rabbia and colleagues (2003) compared cardiac autonomic function in 50 non-diabetic obese youth and 12 healthy controls. Unlike previous studies, youth were categorized into recently, intermediate, and long-term obese groups; youth who snored or had sleep apnea were excluded from the study. Based on 24 hour HRV recording, obese youth from all duration categories had lower SDNN, pNN50, rMSSD, and TP values compared to healthy controls. Particularly, during the night period, rMSSD and HF ν values were significantly lower as well, LF ν and LF/HF ratio values were significantly elevated among obese youth compared to healthy controls. Consistent with previous findings, Rabbia and colleagues (2003) found obese youth show autonomic dysfunction. Interestingly, even after excluding youth with snoring and sleep apnea, this aberrant pattern was seen especially during nocturnal hours.

These cross-sectional studies provide support for the association between reduced HRV, characterized by diminished parasympathetic function, and a predominance of sympathetic function particularly during “sleep” periods in youths with excess adipose tissue. Increased adiposity may prevent the heart’s ability to rest and reduce its workload during sleep, thus, predisposing obese youth to cardiovascular risk.

Clearly, there is strong evidence supporting a link between autonomic dysfunction and obesity in adults and children. Some studies suggest that low sympathetic activation is a risk factor for future weight gain and others suggest that the increase in SNS activity may contribute to obesity. These inconsistent findings may be attributed to differences in methodology. However, since findings consistently suggest that parasympathetic activity is reduced and because autonomic function reflects the constant balance between both parasympathetic and sympathetic activity, it is difficult to assume that both branches are

inactive. Rather, it makes logical sense to have one branch under-active (parasympathetic) and the other over-active (sympathetic). The proposed mechanism underlying autonomic dysfunction (reduced parasympathetic activity and elevated sympathetic activity) is likely the increased secretion of catecholamines, which in turn increases food intake, weight gain, and a reduction in energy expenditure. Collectively, these studies have provided evidence suggesting a potential pathway, whereby autonomic dysfunction contributes to the pathogenesis of obesity.

Rationale for Present Study

Based on the studies presented, the potential pathogenic mechanism underlying the association between and obesity is autonomic dysfunction. First, there is strong evidence linking short sleep duration and obesity. Research findings have demonstrated that reduced sleep duration predicts future weight gain and obesity in youth. Second, based on experimental studies, sleep loss has been proposed as a causal risk factor in the development of autonomic dysfunction, such that reduced sleep alters autonomic functioning for up to 2 days after sleep debt. Third, because autonomic dysfunction is associated with obesity; increased sympathetic activation and its correlates have been implicated as the underlying link between sleep and obesity. By combining experimental and epidemiological data, it seems conceivable that autonomic dysfunction may promote increased food intake by stimulating the SNS, which in turn leads to increased fat deposition and the onset and maintenance of obesity. Considering that reduced sleep is a hallmark of modern society, it is no surprise that obesity prevalence rates have increased so dramatically.

Although researchers have begun to explore this adverse trajectory of losing sleep, exhibiting a decrease in HRV, eating, and gaining weight in adults, the association between these factors remains to be examined in children and adolescents. To date, there are no studies which have measured cardiac functioning while experimentally manipulating youth's sleep duration (e.g., depriving and extending sleep). The majority of studies in youth have been cross-sectional and prospective, and while these studies have provided convincing findings on the link between short sleep duration and weight gain, many were not purposely designed to evaluate sleep (i.e., sleep was not the primary variable of interest). Furthermore, most of these studies obtained sleep information about duration and quality via questionnaires, or sleep diaries from child- and parent-reports instead of using objective measures (e.g., wrist actigraph, PSG).

Interestingly, while researchers have started to explore individual factors such as sleep-related behaviours and beliefs and knowledge about sleep hygiene, all which may play important roles in linking sleep with the likelihood of becoming obese, there is still a paucity of research aimed at elucidating the pathophysiological mechanisms underlying the link between sleep and obesity.

The Present Study

The present study is designed to extend the understanding of the underlying mechanisms of autonomic function during different sleep stages in children and adolescents of varying weight status. Specifically, the goal of the study is to evaluate whether autonomic dysfunction is one of the pathophysiological mechanisms underlying the association between sleep and obesity in children and adolescents. The results of this study will provide new knowledge about how autonomic dysfunction may mediate the

association between short sleep duration and obesity in boys and girls. There are primary and secondary hypotheses associated with the objective of the present study.

Primary Hypotheses

First, it is hypothesized that there will be a negative linear relation between sleep duration and obesity indices, such that youth with shorter sleep duration will have greater obesity values, measured by anthropometric measures (e.g., BMI, BMI percentile, waist circumference, percent body fat).

Second, it is hypothesized that short sleep duration will predict autonomic dysfunction, as measured by HRV in both time and frequency analyses. That is, SDNN, rMSSD, pNN50, and HF power values, reflective of parasympathetic under-activity, will be lower; and, LF power and LF/HF ratio, reflective of sympathetic activation, will be greater among short sleepers compared to long sleepers.

Third, it is hypothesized that greater indices of autonomic dysfunction will predict obesity in youth, such that autonomic dysfunction (e.g., reduced HRV and parasympathetic activity and increased sympathetic activation) will be observed among heavier youth compared to lean youth.

Finally, it is hypothesized that autonomic dysfunction will partially mediate the association between short sleep duration and obesity, even after controlling for potential covariates, such as age, sex, and pubertal status.

METHOD

Participants

Healthy children and their parents were recruited using flyers posted in the community and distributed to elementary and high schools affiliated with the English Montréal School Board. Letters were sent to principals who agreed to have materials distributed by classroom teachers. Exclusionary criteria included reported use of medications with cardiovascular effects, a history of cardiovascular complications, serious medical conditions, or psychopathology. A total of 122 children (50.8% girls) aged 8 to 17 years old ($M = 12.81$, $SD = 2.19$) participated in the study. There were 43 of families that participated and a total of 31 siblings. Parents were aged 30 to 65 years ($M = 45.44$, $SD = 7.3$) and were predominately mothers (78.9%). The institutional review board of Concordia University approved the study procedure (UH2005-077-2).

Measures

As part of the larger Healthy Heart Project, children and their parents completed measures assessing smoking, sedentary behavior, physical activity, culture, stressful life events, and diet. Parents also completed a general health and cardiovascular health history form for themselves and for their child(ren). Only the measures and procedures relevant to my thesis are described below.

Anthropometric Data. Trained research assistants measured children's waist and hip circumference, height, and weight. All anthropometric measures were taken while the children were dressed in light clothing. While the children were standing, waist circumference was measured at the narrowest part of the body, midway between the lowest rib cage and the iliac crest, with a standard measuring tape. Hip circumference

was measured at the widest part of the body, over the buttocks with a standard measuring tape. Height was measured using a standard stadiometer, at maximal breath with shoes off. Waist and hip circumferences and height were measured in duplicate, to the nearest 0.1 cm by two trained research assistants; the average of the two measurements was used.

Weight and percent body fat were measured with a bioelectrical impedance scale (Tanita Body Composition Analyzer BF-350). BMI was calculated based on the formula weight (m)/height squared (m²). BMI percentiles were determined using the growth charts published by the U.S. Centers for Disease Control and Prevention (2000) which plot BMI with respect to age and sex (Ogden et al., 2002).

Pubertal Stage. Pubertal status was determined based on the validated parent- and self-report Growing and Changing Questionnaire (“Avon Longitudinal Study of Parents and Children” [ALSPAC], 2006) adapted from the Tanner questionnaire (Marshall & Tanner, 1969). There are five Tanner stages from prepubertal (P1) to complete sexual maturity (P5). The intermediate stages (P2, P3, P4) reflect progressive changes of external genitalia and pubic hair. Girls rated their pubertal stage based on a series of five standard illustrations with descriptive text on breast size and shape as well as pubic hair distribution. In addition, for girls, the age and date of their first period and the length of menstruation cycle were assessed.

Boys rated their pubertal stage based on a series of five standard illustrations with descriptive text on the size and shape of the scrotum, testes, and penis, as well as on pubic hair distribution. For boys, changes in voice and hair growth in armpits were also assessed. A sixth response option was provided for participants who did not know.

Consistent with ethical guidelines, participants were given the option of refusing to answer any questions they did not want to.

Compared to the gold standard of determining pubertal status through invasive assessments by pediatricians, this method was deemed most suitable and practical. The Tanner questionnaire has demonstrated moderate kappa coefficient agreement and thus the Growing and Changing questionnaire (“ALSPAC”, 2006) was used. The reliability for breast or penis Tanner staging was 0.48 (95% CI: 0.31 - 0.64) between self-report Tanner ratings and clinical pediatric physician ratings (Taylor, Whincup, Hindmarsh, Lampe, Odoki, & Cook, 2001). A kappa coefficient agreement of 0.67 (95% CI: 0.49 - 0.87) was found for pubic hair development in boys and girls using the Tanner questionnaire and physician ratings (Taylor et al., 2001).

Ambulatory Physiological Recordings. The LifeShirt (Vivometrics®) is an ambulatory monitor worn by children for 24-hours. The LifeShirt has received approval from Health Canada to measure continuous electrocardiogram (ECG), respiratory patterns (pulse oximetry), posture, and electroencephalogram (EEG). The LifeShirt is a noninvasive shirt worn underneath the children’s clothes in their natural environment at home and school, thereby permitting ecologically valid measures of physiological parameters with minimal inconvenience. The LifeShirt system is made from a comfortable CoolMax garment embedded with two calibrated respiratory inductive plethysmography bands that measured changes in lung volume.

Heart rate variability (HRV). HRV was derived from the ambulatory ECG LifeShirt recorder that the children wore while sleeping in their own bed at home. The ambulatory monitor is a 3-lead, single channel ECG used to measure heart rate from

which HRV can be derived. HRV indices were derived in two ways: by time domain analysis and by frequency (power spectral) domain analysis sampled at 200 Hz. Time- and frequency-domain analyses were calculated from continuous ECG recordings during the children's sleep period.

Time Domain Analysis. HRV time domain analysis quantified the successive normal R-R intervals recorded over the children's sleep period. Time domain variables include standard deviation of normal R-R intervals (SDNN) and the square root mean difference of successive R-R intervals (rMSSD). rMSSD is a measure of parasympathetic nervous system activity and is inversely correlated with the sympathovagal LF/HF ratio (Berntson et al., 1997). The pNN50 is the average number of times per hour in which the difference in successive R-R intervals are greater than 50 ms. All time domain indices were calculated in ms, excluding the pNN50 which was calculated as a percentage (Berntson et al., 1997).

Frequency Domain Analysis. Given that HRV is composed of various frequencies, Fast Fourier transformation of R-R interval oscillations isolated two major frequency components: low frequency (LF, 0.04-0.15 Hz) and high frequency (HF, >0.15 Hz) (Malliani, Pagani, Lombardi, & Cerutti, 1991). LF/HF ratio was also derived as an indicator of sympathovagal balance.

Data Reduction

Artifacts are waves, or groups of waves, which are not due to electrical activity but are produced as a result of technical problems, participant movement, or environmental disturbances. Even ECG artifacts as short as 2 minutes in duration have been shown to increase error in measuring HRV (Berntson & Stowell, 1998; Xia et al.,

1993). Artifacts were identified and removed in the ECG waveform on the R-Waves and R-R Interval periods using the VivoLogic Software (VivoMetrics ®) and verified by visual inspection from a qualified trained professional. Based on VivoLogic Software, linear interpolation algorithms assigned cutoff values to successive R-R intervals so that any R-R values that fell above or below the cutoff values were removed.

Premature contractions and irregular heart beats were also identified and removed from the data based on VivoLogic Software algorithms and verified by visual inspection from a qualified trained professional. The VivoLogic algorithm was based on three rules; first the order of intervals should be normal. Second, the difference between the large interval and the smaller interval must exceed 100 ms. Third, the sum of the large interval and the smaller interval should be within 10% of the sum of the previous and following normal intervals. Finally, the value of the previous normal interval should be within 20% of the value of the following normal interval.

Subjective Sleep Measures

Perceived Sleep Quality. Children rated their sleep quality on a scale of 1 to 10 (1=very bad to 10=very good) twice; once for their typical night's sleep and again for the night they completed the sleep study.

Sleep Duration. Children reported their usual wake and sleep times for school nights (Sunday through Thursday) and weekend nights (Friday and Saturday). Parents also reported their child's usual wake and sleep times on school and weekend nights. Sleep duration for school and weekend were calculated as the difference between parent- and self-report sleep and wake times. Hence, four independent sleep duration measures were derived (school and weekends based on parent and self-reports).

Children's Sleep Habits Questionnaire (CSHQ). Parents completed the CSHQ (Owens et al., 2000). This questionnaire is used to evaluate children's sleep patterns over a one week interval and includes 43 questions to assess sleep problems such as difficulty in initiating and maintaining sleep, napping, and daytime sleepiness. Each question was rated on a 3-point scale that described the frequency (rarely, sometimes, and usually) of the sleep problems. For example, "in the last week, my child is ready to go to bed at bedtime". Scores for each item were summed to produce a total score that ranged from 0 to 135. A score of 41 or above is considered to be indicative of more sleep-related problems, poor sleep quality, and more daytime sleepiness. The CSHQ has demonstrated test-retest reliability, validity, and internal consistency (Cronbach's alpha community sample = 0.68, clinical sample = 0.78; Owens et al., 2000).

Pediatric Daytime Sleepiness Scale (PDSS). The PDSS (Drake et al., 2003) is a scale for pediatric populations to assess daytime sleepiness. Although the PDSS scale has not been validated using objective measures, it demonstrates acceptable reported levels of internal consistency (Cronbach's alpha =.81) and split-half reliability (Cronbach's alpha =.80) among middle-school aged children. Eight self-rated questions assessed daytime sleepiness and alertness in children based on the frequency (never, seldom, sometimes, frequently, always) of reporting, for example, "how often do you get sleepy or drowsy while doing your homework?". Each question was answered on a 0 to 4 scale, and summed to calculate a global daytime sleepiness score. Total scores ranged from 0 to 32. A total score of 20 or more reflected a very "sleepy" child. The PDSS score was used as a subjective indicator of daytime sleepiness in children.

Objective Sleep Measures

In-Home Sleep Study. Each child underwent a full-night sleep study in their own home with the LifeShirt BioSomnia Electroencephalogram (EEG) Device (VivoMetrics®) in combination with a horizontal electrooculogram (EOG). The BioSomnia EEG device measured changes in the duration and amplitude of electrical activity in the brain to identify distinct brainwave frequencies and was used to determine sleep stages within NREM and REM sleep. The EOG records electrical activity of eye movements and was used to determine slow and rapid eye movements. The sleep study included a single channel EEG (FPz, A1, ground), as well as one EOG electrode on the right temple, with a sampling frequency of 200 Hz per channel.

EEG has specific frequency bands including alpha (8-12 Hz), theta (3.5 to 7.5Hz), and delta (0.1-3 Hz) associated with specific sleep stages. Digital sleep data were stored for later analysis. Sleep staging and arousals were scored in 30-second segments (epochs) based on Rechtschaffen and Kales criteria (1968; adapted by VivoLogic software) as well as with visual detection by a qualified trained sleep technician. Sleep stages included wake, NREM (stage 1, stage 2, stage 3, and stage 4) and REM for each epoch.

Sleep Stage Criteria & Detection

Stage Wake. Wake stage is the wakefulness state. It is defined by alpha activity and or low voltage and mixed frequency EEG. Alpha is associated with being alert (while eyes are closed) and with relaxation (deep breathing). Alpha distribution is typically localized and is mainly detected during the wake stage. The transition from wakefulness to Stage 1 is defined by a reduction in the amount, amplitude, and frequency

of alpha activity. Theta is slow brainwave activity and represents the transition between wakefulness and sleep states. Its distribution can generally be either diffused or localized to one side of the brain (Rechtschaffen & Kales, 1968).

Stage 1. Stage 1 lasts from 1 to 7 min and occurs during the earlier portion of the stage. Stage 1 is characterized by the presence of slow eye movement, a moderately low voltage, mixed frequency EEG (alpha) activity ranging from 2 to 7 cycles per second (cps), and vertex sharp waves (Rechtschaffen & Kales, 1968).

Stage 2. Stage 2 is defined by the presence of sleep spindles that must be present for at least half a second. In addition, the presence of K complexes (EEG waveforms with a negative sharp wave followed by a positive wave) must be present for over half a second (Rechtschaffen & Kales, 1968).

Stage 3. This stage is defined by delta waves of 2cps or slower which are present for at least 20% but not more than 50% of the epoch (Rechtschaffen & Kales, 1968). Delta, the highest amplitude and the lowest brainwave frequency, is associated with diminished arousal levels and relaxed states (Rechtschaffen & Kales, 1968). Delta can be detected during NREM sleep stages 3 and 4 (deep or slow wave sleep). Its distribution is typically widespread and dispersed. Sleep spindles may or may not be present in this stage.

Stage 4. This stage is defined by delta waves of 2cps or slower which are present for more than 50% of the epoch. Stage 4 is predominately composed of delta (slow wave) activity high in amplitude (Rechtschaffen & Kales, 1968). Sleep spindles may or may not be present in Stage 4. In general, during NREM sleep HRV is elevated and or is stable compared to wakefulness and REM sleep (Goldsmith et al., 2006).

REM. This stage is defined by the simultaneous presence of moderately low voltage, mixed EEG activity (“saw-tooth” waves), and episodic rapid eye movements. REM is similar to Wake (but the frequency is approximately 1-2 cps slower) and to Stage 1 (but no vertex sharp waves are present). No sleep spindles or K complexes are present during REM (Johnson, Lubin, Naitoh, Nute, & Austin, 1969).

From these recordings, standard sleep measures included: 1) sleep period time, which includes only the time spent in light NREM, SWS, and in REM, 2) total sleep time, which includes all sleep stages and arousals, 3) sleep efficiency, the percentage of time in bed spent asleep, 4) sleep latency (the amount of time it takes to fall asleep, in minutes), 5) fragmented sleep (the number of awakenings lasting at least 15 seconds), 6) the intermittent arousal index (sum of duration of all awakenings between sleep onset and sleep offset) and the 7) micro-arousal index (the average number of arousals per hour of sleep). In addition, the percent of time spent in light NREM, SWS, and REM sleep were derived. The LifeShirt has a sensitivity of 0.83 and specificity of 0.87 with traditional polysomnograph (Orr & Goodrich, 2003).

Procedure

A telephone screening interview was conducted to ensure children met inclusionary criteria (e.g., no serious psychopathology) and two sessions were scheduled at the laboratory, three days apart. During the first session, the study protocol was described and explained to parents and their children. If the parents and children agreed to participate, parental informed consent and child assent were obtained. Next, parents and children were separated into different rooms and each completed a set of questionnaires; this prevented parents and children from obtaining answers from each

other on their respective questionnaires. After the children completed their questionnaires, they were brought into the medical room where two trained research assistants completed a finger prick blood test (i.e., cholesterol levels) and collected anthropometric measurements (i.e., waist and hip circumference and height and weight).

Next, children were fitted with the ambulatory monitor (Vivometrics® LifeShirt) by the research assistants. Three ECG electrodes were placed in a Lead II configuration: two electrodes (active electrodes) were placed one-inch under each of the shoulders and one electrode was placed on the left side of the child's abdomen (ground electrode). Instructions for operating the LifeShirt were explained and troubleshooting tips were provided to both the parent and child. Also at this time, the research assistants provided instructions to parents and children on the sleep study. In addition, instruction sheets and a contact phone number were provided in the event that the parent and or child had questions during the 24 hour monitoring session.

The sleep study took place at the child's home. Before the child went to sleep, the parent or child attached an auxiliary EEG device (LifeShirt Oxford BioSomnia) to the LifeShirt. The parent or child then placed three EEG electrodes directly onto the skin of the child's forehead (FPz), left mastoid (A1 referent), and nape of the neck (ground electrode), as well as one EOG electrode on the child's right temple. This 4-lead configuration permitted minimally invasive polysomnograph (PSG) data collection of brain wave activity and eye movement to be conducted while the child slept in his or her own bed. In addition, a finger clip (pulse oximeter) was placed on the child's index finger to measure blood oxygen saturation levels throughout the night.

When the child awoke, the sleep study was considered complete. Upon awakening from the sleep study, the parent or child removed the EEG and EOG electrodes and the child continued wearing the LifeShirt for the remainder of the 24 hours during a normal school day. Upon completing the 24 hours, the LifeShirt was removed by the parent or child. During the second session, the parent and child returned the LifeShirt equipment and completed a feedback form to report their experience with the LifeShirt and the sleep study.

Lastly, as compensation for their time, participants received \$60, a feedback sheet about their own heart health, and an educational healthy lifestyle packet.

Data Preparation

ECG data captured during the sleep study were analyzed with VivoLogic software and by visual inspection. VivoLogic was used to calculate frequency- (LF, HF, LF/HF ratio, LF_{nu} , and HF_{nu}) and time- (SDNN, rMSSD, and pNN50) domain indices for every 30-second epoch during sleep. Thirty-second epochs were chosen to permit matching HRV data with the appropriate sleep stage (e.g., Stages 1-4) scored using the Rechtschaffen and Kales (1968) criteria. For each individual participant, VivoLogic produced a “sleep stage” excel file output organized by 30-second intervals and their respective sleep stages scored from Stages 1-4 and REM. Next, for each participant, VivoLogic produced a “HRV” excel file output, again organized by 30-second epochs that coincided with the sleep output. Frequency domain variables were analyzed with Fast Fourier Transform at 128 Hz with a resample rate of 10. The HRV output was merged with the sleep output to create one master file for each participant. The number of 30-second intervals in the output ranged from 679-1605 across participants.

Data Cleaning

HRV data identified as artifacts by VivoLogic algorithms were immediately removed from the master file for each participant. From the 57 participant files, 43 (75%) did not have any artifacts identified by VivoLogic software, thus no intervals were removed; 9 files (16%) had $\leq 1\%$ of the data identified as artifacts (e.g., from 1253 to 1250 intervals); 2 files (4%) had 2% of data identified as artifacts (e.g., from 1159 to 1147 intervals); 2 files (4%) had $\sim 8\%$ of data identified as artifact (e.g., from 1090 to 1018 intervals); and, one file had 15% of data identified as artifact (e.g., from 992 to 839).

The mean HRV values for each frequency- and time-domain variables were calculated. HRV values that were above or below four standard deviations from the mean were visually inspected in the raw data screen, cleaned, and replaced with the average HRV value for the respective sleep stage for each individual. Following the removal of artifacts and outliers, HRV variables in Stages 1 and 2 were aggregated to represent light NREM sleep, while Stages 3 and 4 were aggregated to represent SWS. REM sleep had its own HRV values.

Statistical Analyses

Assumptions of multivariate normality were met, such that all univariate distributions were normal and all bivariate scatterplots were linear and homoscedastic. Outliers and missing data were replaced with the mean for that particular variable. All analyses were cross-sectional and performed using SPSS 15.0. A p -value of <0.05 was considered significant for all analyses.

Structural equation modeling (SEM), using Lisrel Version 8.8 (Joreskog & Sorbom, 2001), was used to test the mediation models. SEM is a complete approach to modeling multiple interrelated observations simultaneously (Kline, 1998). In the present study, the maximum likelihood estimation method of SEM was used and the listwise deletion method was used to deal with missing and or incomplete data. Manifest (observed) variables and latent (unobserved) variables were included into the models and tested to see if the models explained the data (goodness of fit). The following indices were used to assess the goodness of fit of the models: 1) Comparative Fit Index (CFI $\geq .90$), 2) Non-Normed Fit Index (NNFI; $\geq .90$), 3) Root Mean Square Error of Approximation (RMSEA $\geq .05$ to $.08$), and 4) Goodness of Fit Index (GFI $\geq .90$; Bentler, 1990). Chi-square statistic, degrees of freedom, and R^2 were used to determine mediation in the models.

RESULTS

Sample Characteristics. The characteristics of the parents and children who participated in the study are outlined in Table 1. The majority of the parents were mothers (78.9%) averaging 45.4 years ($SD = 7.3$), were married (68.5%), obtained an undergraduate degree (37.8%), and reported a household income of \$60,000 to \$69,000 (14.6%). There were 122 children (50.8% female), aged 8 to 17 years ($M = 12.81$, $SD = 2.19$) who participated. On average, youth's waist circumference was 73.14 cm ($SD = 9.52$), hip circumference was 91.34 cm ($SD = 10.68$), weight was 118.66 lbs ($SD = 30.53$), BMI was 21 kg/m^2 ($SD = 3.97$), percent body fat was 22% ($SD = 9.62$), and BMI percentile was 63.91 ($SD = 25.07$; see Table 2). Consistent with other North American population studies, our sample consisted of 8% obese children and 18% overweight children based on CDC BMI growth charts (CDC, 2000).

Pubertal Status. Based on child-report, pubertal status for girls' breast and hair development was Tanner stage 3.25 ($SD = 1.46$) and 3.27 ($SD = 1.72$), respectively. On average, 80.3% endorsed having hair in armpits and 66.1% have begun their menstrual periods (see Table 3). Pubertal status for boys' penis and hair development was Tanner stage 2.83 ($SD = 1.62$) and 3.02 ($SD = 1.52$), respectively. On average, 62.3% endorsed having hair in armpits and 29.5% report changes in their voice (see Table 4).

Subjective Sleep Measures. Children reported sleeping an average of 8 h 56 min ($SD = 59 \text{ min}$) on school nights and 9 h 57 min ($SD = 1 \text{ h } 17 \text{ min}$) on weekend nights. Based on child-report, the mean Pediatric Daytime Sleepiness Score was 13.96 ($SD = 5.5$; see Table 5). Parents reported their children sleep an average of 9 h 07 min ($SD = 56 \text{ min}$) on school nights and 10 h 09 min ($SD = 1 \text{ h } 09 \text{ min}$) on weekend nights. The

intraclass correlations (ICC) between child- and parent-report of sleep duration on school nights was 0.70 (CI 95% 0.59 - 0.78, $p < .001$); on weekend nights was 0.42 (CI 95% 0.26 - 0.56, $p < .001$). Based on parent-report, the mean score on the CSHQ total sleep disturbance score was 40.51 ($SD = 5.9$); the remaining subscales are shown in Table 5.

From the original 122 children recruited for this study, 82 (66.7%) completed the in-home sleep study; the remaining 33.3% did not participate in the in-home sleep study either because the sleep recorder was not yet acquired so they had no opportunity to complete it ($n = 35$) or because they refused to complete the sleep study ($n = 5$). One-way ANOVA statistics showed no significant differences between youth who did and did not complete the sleep study based on age ($F_{(1, 121)} = 0.57, p = .49$), waist circumference ($F_{(1, 121)} = 0.09, p = .77$), or child-report of sleep duration on school nights ($F_{(1, 121)} = 0.04, p = .85$). These groups also did not differ on pubertal status [Girls: breast development ($F_{(1, 60)} = 0.11, p = .74$); hair growth ($F_{(1, 59)} = 0.11, p = .74$); the start of menstruation ($F_{(1, 61)} = 0.01, p = .93$); Boys: pubertal penis development ($F_{(1, 59)} = 0.35, p = .56$); hair growth ($F_{(1, 59)} = 0.01, p = .92$)]. Only one female participant reported menstruating on the night of her sleep study.

Of the 82 children who completed the sleep study, 24 (30%) experienced problems with the sleep recorder (e.g., sleep recorder did not work, batteries died, sensor fell off and lost EEG and/or ECG signal for more than 60 consecutive minutes during sleep) leaving 57 (70%) participants with complete and sufficient EEG and ECG data, defined as more than 5 hours of sleep with clean EEG and ECG signals. No differences were found in perceived sleep quality for typical nights ($M = 6.75, SD = 2$) compared to

the in-home sleep study ($M = 6.66$, $SD = 2.32$; $F_{(1, 120)} = 0.86$, $p = .36$) for those who did and did not complete the sleep study.

Objective Sleep Measures. Table 6 summarizes the data for all in-home sleep study parameters. On average, the sleep period time was 517.03 min ($SD = 77.75$) and the total sleep time was 467.28 min ($SD = 100.90$). The average number of awakenings lasting at least 15 seconds was 50.91 ($SD = 30.22$). Children spent 23.40% ($SD = 8.17$) in light NREM, 20.19% ($SD = 8.57$) in SWS, and 45.07% ($SD = 17.69$) in REM sleep. The mean sleep efficiency was 89.37% ($SD = 11.39$) and sleep latency was 1.97 min ($SD = 2.83$). No sex differences were found for any of these objective sleep parameters (e.g., sleep period time, total sleep time, etc.).

HRV Measures. The mean values for HRV measures during light NREM, SWS, and REM sleep are summarized in Table 7. In general, frequency- (LF, HF) and time-domain (SDNN, rMSSD, pNN50) HRV indices were highest during REM sleep and lowest during SWS. Table 8 shows differences in HRV variables in normal-weight, overweight, and obese children during light NREM, SWS, and REM sleep. In general, normal-weight youth had the highest indices of parasympathetic activity compared to overweight and obese youth.

Obesity Constructs. Inter-correlations among obesity variables indicated that waist circumference ($r_{\text{avg}} = 0.80$, $p < .001$), hip circumference ($r_{\text{avg}} = 0.80$, $p < .001$), and BMI ($r_{\text{avg}} = 0.81$, $p < .001$) were the strongest indices associated with all of the obesity variables. Percent body fat ($r_{\text{avg}} = 0.64$, $p < .001$) corresponded the least with the remaining obesity variables (see Table 9).

Subjective Sleep Constructs. Inter-correlations among subjective sleep variables indicated that child-report perceived sleep quality ($r_{\text{avg}} = 0.25, p < .05$), child-report sleep duration ($r_{\text{avg}} = 0.19, p < .05$), and child-report daytime sleepiness ($r_{\text{avg}} = 0.26, p < .05$) were highly inter-correlated with other subjective sleep measures.

Objective Sleep Constructs. Inter-correlations among objective sleep variables indicated that fragmented sleep ($r_{\text{avg}} = 0.36, p < .05$), time spent in REM ($r_{\text{avg}} = 0.33, p < .05$) and sleep efficiency ($r_{\text{avg}} = 0.36, p < .01$) were found to be highly related with other objective sleep variables (see Table 11). Some subjective sleep measures were also found to correlate with objective sleep measures. Child-report sleep duration on school nights only was negatively correlated with sleep efficiency ($r = -0.31, p < .05$) and time spent in REM sleep ($r = -0.28, p < .05$; Table 12). Parent-report sleep duration on school nights was correlated with time spent in light NREM ($r = 0.30, p < .05$); as well, fragmented sleep was positively associated with night awakenings ($r = 0.27, p < .05$; Table 13).

Autonomic Dysfunction Constructs. Inter-correlations among the frequency-domain HRV variables indicated that HF ($r_{\text{avg}} = 0.69, p < .01$) and the time-domain HRV variables SDNN ($r_{\text{avg}} = 0.53, p < .01$), rMSSD ($r_{\text{avg}} = 0.72, p < .01$), and pNN50 ($r_{\text{avg}} = 0.67, p < .01$) were the HRV variables best representing parasympathetic activity and together were negatively correlated with the sympathovagal balance LF/HF ratio index ($r_{\text{avg}} = -0.54, p < .01$; Table 14).

Age Effects. Partial correlations were computed to assess the relation between obesity measures and sleep measures while controlling for age. On average, a negative linear relation was found between obesity measures (waist and hip circumference,

weight, BMI, percent body fat, BMI percentile) with child-report sleep quality ($r_{\text{avg}} = -0.33, p < .05$) and parent-report on CSHQ sleep disturbance ($r_{\text{avg}} = -0.38, p < .05$). And inversely, a positive association between obesity measures and child-report daytime sleepiness ($r_{\text{avg}} = 0.19, p = \text{n.s.}$) and parent-report on CSHQ daytime sleepiness ($r_{\text{avg}} = 0.33, p < .05$) was found (see Table 15). Obesity measures were also significantly correlated with objective sleep measures. As shown in Table 16, the majority of the objective sleep measures were inversely correlated with obesity measures. These results support the hypothesis that sleep (shorter sleep duration and poorer sleep quality) is negatively associated with obesity.

While controlling for age, partial correlations showed that the time spent in SWS was positively correlated with LF/HF ratio ($r = 0.35, p < .05$) and LFnu ($r = 0.37, p < .05$), and was negatively correlated with HFnu ($r = -0.30, p < .05$; see Table 17). Sleep efficiency was negatively associated with LF ($r = -0.29, p < .05$), HF ($r = -0.28, p < .05$), and SDNN ($r = -0.26, p < .05$) during SWS. Fragmented sleep was negatively correlated with LF/HF ratio ($r = -0.28, p < .05$) and LFnu ($r = -0.25, p < .05$), and positively correlated with HFnu ($r = 0.27, p < .05$). These correlations during SWS are presented in Table 18; during REM are in Table 19. Although there are significant relations among sleep and HRV measures, these results were not in the hypothesized direction.

Partial correlations, while controlling for age, showed that HRV variables: LF, SDNN, rMSSD, and pNN50 in light NREM, SWS, and REM sleep were significantly negatively correlated with obesity measures (see Tables 20-22). No significant correlations were found for LFnu, HFnu, or LF/HF ratio with the obesity measures.

These results partially support the hypothesis that autonomic dysfunction is associated with obesity.

Hypothesized Model Testing

Three latent variables were created: obesity, autonomic dysfunction, and sleep. Although partial correlations showed strong associations between frequency-domain HRV variables and the sleep and obesity measures, only SDNN, rMSSD, and pNN50 variables were included as manifest variables. Because SEM is extremely sensitive to sample size and multicollinearity among items, our small sample size precluded analyses with the frequency-domain variables. Thus, three models were tested for partial mediation while controlling for age. The latent variables sleep and obesity made up the base model.

Base Model. The latent variable obesity was made up of three manifest variables that were found to best represent the construct: waist circumference, hip circumference, and BMI. In the measurement model, waist circumference had a loading of 0.90, hip circumference had a loading of 0.97, and BMI had a loading of 0.88. Goodness of fit indices suggest the measurement model was a good fit with the data (CFI = 0.99, NNFI = 0.99, RMSEA = 0.00, GFI = 0.99). The latent variable sleep was made up of three manifest variables that were found to best capture the construct: sleep efficiency, fragmented sleep, and time spent in REM. In the measurement model, sleep efficiency had a loading of 0.70, fragmented sleep had a loading of 0.55, and time spent in REM had a loading of 0.97. The measurement model showed a good fit (CFI = 0.88, NNFI = 0.65, RMSEA = 0.21, GFI = 0.94). Although time spent in SWS and REM were also

found to be highly related with other indices, due to multicollinearity, these manifest variables could not be included in the SEM models.

The structural equation revealed a moderate association between sleep and obesity with an $R^2 = 0.14$ ($\chi^2 = 15.75$, $df = 10$). This path model was a good fit with the data (CFI = 0.94, NNFI = 0.92, RMSEA = 0.10, GFI = 0.93; see Figure 1).

Model 1. In the first model, the latent variable autonomic dysfunction was composed of manifest variables: SDNN values during light NREM, SWS, and REM sleep. SDNN during light NREM had a factor loading of 0.93, during SWS had a loading of 0.95, and during REM sleep had a loading of 0.99. The measurement model showed strong goodness of fit indices (CFI = 1.00, NNFI = 1.02, RMSEA = 0.00, GFI = 0.99). When a path from sleep to autonomic dysfunction was added into the model, results showed 3% of the variance in autonomic dysfunction was explained by sleep ($\chi^2 = 19.56$, $df = 13$) and goodness of fit indices were high (CFI = 0.94, NNFI = 0.91, RMSEA = 0.09, GFI = 0.91). When a path from autonomic dysfunction to obesity was added, an $R^2 = 0.15$ ($\chi^2 = 10.34$, $df = 10$) was found. Goodness of fit indices were high (CFI = 0.99, NNFI = 0.99, RMSEA = 0.02, GFI = 0.95). When all three paths between latent variables were included, 25% of the variance in obesity was explained by both sleep and autonomic dysfunction ($\chi^2 = 37.70$, $df = 28$) with an acceptable fit with the data (CFI = 0.96, NNFI = 0.94, RMSEA = 0.07, GFI = 0.88; see Figure 2).

Model 2. A second model was run with autonomic dysfunction based on the rMSSD manifest variables for light NREM (factor loading of 0.99), SWS (factor loading of 0.96), and REM sleep (factor loading of 0.96). The measurement model had a good fit (CFI = 0.98, NNFI = 1.00, RMSEA = 0.00, GFI = 0.98). The path from sleep to

autonomic dysfunction did not produce a significant association ($R^2 = 0.03$, $\chi^2 = 14.65$, $df = 13$). The path from autonomic dysfunction to obesity produced an $R^2 = 0.12$ ($\chi^2 = 4.82$, $df = 10$; CFI = 0.99, NNFI = 1.03, RMSEA = 0.00, GFI = 0.98). When all paths were added, 24% ($\chi^2 = 27.00$, $df = 28$) of the variance in obesity was accounted for (CFI = 0.99, NNFI = 0.98, RMSEA = 0.00, GFI = 0.91; see Figure 3).

Model 3. A third model was run with autonomic dysfunction based on the pNN50 manifest variables from light NREM (factor loading of 0.96), SWS (factor loading of 0.99), and REM sleep (factor loading of 0.95). The measurement model had a good fit (CFI = 1.00, NNFI = 1.00, RMSEA = 0.00, GFI = 0.99). The path from sleep to autonomic dysfunction did not reveal a significant association ($R^2 = 0.00$, $\chi^2 = 21.01$, $df = 13$) even though data showed modest fit with the model (CFI = 0.95, NNFI = 0.92, RMSEA = 0.10, GFI = 0.90). The path from autonomic dysfunction to obesity produced a significant association and good fit ($R^2 = 0.11$, $\chi^2 = 13.92$, $df = 10$; CFI = 0.98, NNFI = 0.96, RMSEA = 0.08, GFI = 0.93). Lastly, when all paths were added, sleep and autonomic dysfunction explained 25% of the variance ($R^2 = 0.25$, $\chi^2 = 38.85$, $df = 28$; CFI = 0.96, NNFI = 0.94, RMSEA = 0.08, GFI = 0.88; see Figure 4).

Post-Hoc Exploratory Analyses. Due to issues with multicollinearity, sample size, and power, post-hoc exploratory analyses were conducted with multiple linear regression to evaluate the frequency-domain HRV variables while controlling for age. In parallel with the SEM analyses, a base model consisting of time spent in REM and BMI was first tested. Time spent in REM significantly predicted BMI ($F_{(1, 54)} = 4.91$, $p = .03$) and explained 8% of its variance. When LF/HF ratio during REM was added to the model, time spent in REM did not significantly predict LF/HF ratio ($F_{(1, 54)} = 0.30$, $p =$

.58; $R^2 = 0.01$); and LF/HF ratio did not significantly predict BMI ($F_{(1, 54)} = 0.01, p = .91$; $R^2 = 0.00$). These multiple regression exploratory models were repeated for LF/HF ratio during SWS and light NREM (data not shown) and similar results were found; these results do not support the mediation hypothesis.

These multiple regression exploratory models were repeated for LF and HF during light NREM, SWS, and REM. Although LF during light NREM ($F_{(1, 54)} = 6.95, p = .01$; $R^2 = 0.11$), SWS ($F_{(1, 54)} = 5.03, p = .02$; $R^2 = 0.09$), and REM ($F_{(1, 54)} = 7.21, p = .01$; $R^2 = 0.11$) predicted BMI, time spent in REM did not significantly predict LF in light NREM ($F_{(1, 54)} = 1.69, p = .19$; $R^2 = 0.03$), SWS ($F_{(1, 54)} = 1.84, p = .18$; $R^2 = 0.03$), or in REM ($F_{(1, 54)} = 0.64, p = .43$; $R^2 = 0.01$). When sleep and LF during light NREM were added into the model, again both contributed to BMI ($F_{(1, 53)} = 4.69, p = .006$; $R^2 = 0.17$), but each of their variance decreased by 2% (LF: $R^2 = 0.09, p = .02$; REM: $R^2 = 0.06, p = .06$). Similar results were observed for LF during SWS (LF: $R^2 = 0.07, p = .06$; REM: $R^2 = 0.06, p = .06$). Finally when time spent in REM and LF in REM were simultaneously added to the model they individually contributed to predicting BMI (LF: $R^2 = 0.11, p = .02$; REM: $R^2 = 0.07, p = .04$) and suggested partial mediation as the beta coefficient for time spent in REM decreased by 1%. These results suggest only some support for partial mediation.

When HF variables were included in the models, HF in light NREM ($F_{(1, 54)} = 6.86, p = .01$; $R^2 = 0.11$), SWS ($F_{(1, 54)} = 7.38, p = .001$; $R^2 = 0.12$), or in REM ($F_{(1, 54)} = 5.70, p = .02$; $R^2 = 0.10$) significantly predicted BMI; however, time spent in REM did not significantly predict HF in light NREM ($F_{(1, 54)} = 0.81, p = .37$; $R^2 = 0.02$), SWS ($F_{(1, 54)} = 0.92, p = .34$; $R^2 = 0.02$), or REM sleep ($F_{(1, 54)} = 1.19, p = .28$; $R^2 = 0.02$). When

sleep and HF during light NREM were added into the model, again both contributed to BMI, but each of their variance decreased by 1% (HF: $R^2 = 0.10$, $p=.01$; REM: $R^2 = 0.07$, $p=.05$). Similar results were observed for HF during SWS (HF: $R^2 = 0.08$, $p=.03$; REM: $R^2 = 0.07$, $p=.05$). Finally, when time spent in REM and LF in REM were simultaneously added to the model they individually contributed to predicting BMI (HF: $R^2 = 0.08$, $p=.03$; REM: $R^2 = 0.07$, $p=.05$) and the variance attributed to time spent in REM decreased by 1% and the variance attributed to HF decreased by 3%. The results suggest only some support for partial mediation. However, the results are to be interpreted with caution due to possible suppression effects (see Discussion).

DISCUSSION

The aim of the present study was to examine whether autonomic dysfunction, measured by HRV, mediated the relation between sleep and childhood obesity. Consistent with previous cross-sectional literature (cf. Beebe et al., 2007; Gupta et al., 2002; Sekine et al., 2002; von Kries et al., 2002), our results demonstrate a clear association between short sleep duration, poor sleep quality, and more sleep complaints among youth with greater adiposity indices. Heavier youth reported overall poorer sleep quality, reduced sleep duration, and complained more of daytime sleepiness than their lean counterparts, even after controlling for age. Based on parent-report, heavier youth had shorter sleep durations and exhibited more sleep problems, including bedtime resistance, sleep disturbance, and daytime sleepiness compared to lean youth.

Parent-reported sleep problems, such as sleep-onset delay, sleep anxiousness, and sleep-disordered breathing were less consistently associated with obesity. These differences may be attributable to the variation of observable symptoms; for example, most children in our sample reported that they slept alone in their own bedroom, which would make parent report on sleep-onset delay difficult; whereas, daytime sleepiness is a more “noticeable” sleep problem (Carskadon et al., 1999).

Based on the in-home sleep PSG study, heavier youth had shorter sleep duration and sleep latency, they spent less time in light NREM and SWS, and they spent more time in REM sleep, than their lean counterparts. Previous studies suggest that the amount of delta waves and the time spent in SWS are heritable and stable individual traits (Franken & Tafti, 2003; Linkowski, 1999; Tan, Campbell, & Feinberg, 2001). Additionally, in mice, mutations in the circadian *Clock* gene lead to obesity (Turek et al.,

2005). It is plausible that a clock gene encoding for these sleep parameters may also be involved in the development of obesity and other related diseases such as diabetes and insulin resistance within particular populations.

Lean youth showed fewer, but longer micro-arousals compared to heavier youth who showed more, but shorter micro-arousals. Micro-arousals are more likely to occur during REM than in NREM sleep (Halasz, Terzano, Parrino, & Bodizs, 2004). Heavier youth spent a larger portion of sleep in REM which suggests that the sleep they did obtain was non-restorative (Halasz et al., 2004). In addition, micro-arousals are associated with daytime sleepiness (Sforza, Chapotot, Pigeau, Naitoh, & Buguet, 2004) which explains why heavier youth and their parents reported more daytime sleepiness symptoms.

Similar to past research, an association between objective sleep measures and autonomic dysfunction was found. As expected, as the number of awakenings increased and as sleep efficiency decreased, LF (absolute, but not normalized) values increased indicative of greater arousals and fragmented sleep (Halasz et al., 2004). Unlike previous findings, greater time spent in SWS was associated with $LFnu$ and LF/HF ratio, as well as reduced $HFnu$. Unexpectedly, we found that greater HF (absolute), a marker of parasympathetic activity, was also associated with markers of poor sleep quality (time spent in SWS), fragmented sleep, and greater sympathovagal balance during sleep. These findings were not consistent across all HRV indices, as no relation was observed between time-domain variables and objective sleep measures. These are counter-intuitive and may be attributable to other factors, such as the measurement technique or design (described below in limitations).

Using subjective sleep measures, an association between sleep and autonomic dysfunction was also observed. Poorer perceived sleep quality was associated with reduced rMSSD, pNN50, and HF (absolute and normalized) values as well as with greater LF/HF ratio and $LFnu$, suggesting that youth with reduced parasympathetic activity and increased sympathetic activity perceive their sleep as poor. Daytime sleepiness complaints were associated with reduced pNN50 values; sleep disturbance reported by parents was also related with reduced SDNN and HF (absolute) values during sleep. These results suggest that less restorative sleep is associated with reduced parasympathetic activity and autonomic dysfunction.

Sloan and colleagues (2005) have demonstrated that psychosocial stressors (i.e., daily hassles, lower socioeconomic status, chronic stress, etc.) can dampen cardiac parasympathetic activity and increase risk of adverse health complications. Similarly, Hall and colleagues (2004) found that a speech stress task is related to significantly reduced parasympathetic activity and more fragmented sleep in stressed individuals compared to controls. Thus, given that daily stressors can impact both autonomic function and sleep, subjective sleep measures may provide a better indicator of overall sleep compared to one night with the sleep study. Although the in-home sleep provided detailed information on objective sleep measures that could not be assessed with questionnaires, these results highlight the importance of obtaining information from multiple sources.

Similar to previous findings, youth with greater adiposity evidenced autonomic dysfunction. Throughout sleep, significantly dampened HRV was observed among heavier youth, with lower SDNN, rMSSD, and pNN50 values. Based on the frequency-

domain variables (LF and HF), parasympathetic activity was also significantly reduced throughout sleep. These results are similar to other studies that computed HRV variables in absolute values (vs. normalized units; Nagai et al., 2003; 2004).

Compared to studies using normalized HRV values (Kaufman et al., 2007; Rabbia et al., 2003), *HFnu* results were consistent, with lower values among heavier youth. However, *LFnu* values were also lower among heavier youth; in fact, healthy-weight youth had greater values. Given that there is evidence that the LF band of HRV is not exclusively associated with sympathetic activity, but also with parasympathetic activity (Berntson et al., 1997), these results suggest that youth with excess adiposity show reduced parasympathetic activity. There was, however, a non-significant trend indicating an elevated LF/HF ratio among heavier youth during sleep, which may suggest sympathetic activation.

Rabbia and colleagues (2003) found that duration of obesity was related to autonomic dysfunction. Specifically, youth who were obese for a longer period showed a reduction in *LFnu* and LF/HF ratio, meaning an under-active sympathetic activation; whereas, recently obese youth (shorter duration) showed greater *LFnu* and LF/HF ratio values, indicating an over-active sympathetic drive and an under-active parasympathetic tone. In the present study, we did not control for duration of obesity and it is likely that our obese sample may have been obese for some time, which may partly explain our results.

Inconsistent with our hypothesis, the results of this study do not fully support the mediation model that autonomic dysfunction is a potential pathophysiological mechanism underlying the association between objective sleep measures and obesity indices.

Objective sleep measures, such as fragmented sleep, sleep efficiency, and time spent in REM were directly related to obesity measures (BMI, waist and hip circumference). However, the relation between objective sleep measures and both time- and frequency-domain HRV measures were not significant. It is plausible that the duration of sleep problems may affect autonomic dysfunction, such that the longer sleep problems are experienced, the more autonomic dysfunction occurs. Therefore, because objective measures are based on a single night of recording they did not tap into the frequency and severity of potential sleep problems. Nevertheless, while both autonomic dysfunction and sleep correlated with obesity, direct mediation was not supported.

The results must be interpreted with caution as there is some evidence of statistical suppression. Unlike the mediation model which predicted that the association between sleep and childhood obesity would decrease after autonomic dysfunction was controlled for (i.e., added in the model), the results of the SEM models were opposite this, such that the addition of autonomic dysfunction increased the predictive validity (variance) in obesity attributed to sleep. Because sleep and HRV are two highly interdependent variables, meaning that certain sleep stages naturally exhibit certain preponderance of the ANS parasympathetic or sympathetic activity than in other stages (i.e., REM sleep shows more sympathetic activation and less parasympathetic activation vs. NREM sleep), examining HRV in the context of time in REM inherently masks its effects. In other words, because youth with greater adiposity were observed to spend more time in REM sleep, this inherently means that they also have greater sympathetic activation and less parasympathetic activity than those spending more time in SWS.

These findings provide only some support the role of autonomic dysfunction in the association between sleep and obesity.

Strengths

There were several methodological strengths in the present study. First, our study included a sample that was representative of the general population, with prevalence rates of healthy-weight, overweight, and obese youth similar to population-based studies (Tremblay et al., 2002). Unlike previous studies that include samples of 10-13 years, the age range of our participants consisted of a wide age range (from 8-17 years).

Second, unlike previous cross-sectional studies, we included multiple indicators of obesity, including body fatness (BMI), fat distribution (waist and hip circumference), and percent body fat, providing us with a more comprehensive measure of obesity. This study also included a comprehensive assessment of sleep with several sleep measures assessed subjectively from multiple informants (e.g., child and parent) and objectively with an in-home sleep study. This multi-model approach likely provides more valid and reliable measures of obesity and sleep.

Third, this was the first study to have children and adolescents complete an ambulatory in-home sleep study while simultaneously measuring continuous ECG and EEG. The LifeShirt system, in conjunction with the BioSomnia sleep recorder, are advanced technological equipment that permit the collection of continuous physiological data with minimal intrusion. Distinct from formal sleep studies which are conducted in different settings (i.e., hospital or laboratory), our procedure and method allowed children and adolescents to complete a comprehensive sleep study in their natural environment. Studies have cross-validated in-home sleep studies against laboratory PSG (Ancoli-Isreal,

Kripke, Mason, & Messin, 1981; Redline, Tosteson, Boucher, & Millman, 1991) providing support that in-home sleep studies are ecologically valid.

Fourth, due to the technology used, continuous HRV data was extrapolated and examined within various sleep stages. These stage-specific analyses provided important information and insight about autonomic function during sleep in youth.

Lastly, unlike ANOVA and regression analyses, SEM analyses (conducted whenever possible) allowed for the simultaneous examination of latent variables made of several manifest variables. The use of this statistical method provided a means of maximizing the multi-modal data for sleep, obesity, and autonomic function obtained from our sample. Despite these methodological strengths, there were limitations which may have affected our results.

Limitations and Recommendations

One limitation of the present study was the way the ECG data was cleaned and prepared. HRV measures were aggregated to synchronize with specific sleep stages and thus coded into 30-second epochs. Typically, HRV measures are analyzed based on at least 5 minute recording periods (Berntson et al., 1997). Using shorter intervals to capture autonomic dysfunction masked subtleties in HRV and likely reduced our power to detect a relation between objective sleep measures and autonomic dysfunction. It would be optimal to have used software which analyzed HRV data at larger intervals to include greater power frequency bands within each sleep stage; unfortunately, this software does not yet exist.

Second, because the early portion of sleep is mainly made up of NREM sleep, which is driven by the parasympathetic branch, and conversely, the later portion of sleep

is composed of REM sleep, which is dominated by the sympathetic activity, aggregating sleep stages together may have skewed or biased the results by assuming that sleep stages are homogenous. As example, it may not have been representative to aggregate REM from early sleep with REM from later sleep because although time spent in REM was controlled for, time effects were not. Clearly, given that both sleep (Laposky et al., 2008) and HRV (Massin, Maeyns, Withofs, Ravet, & Gerard, 2000) are influenced by circadian rhythms, this may be a potential drawback and should be considered in future studies. More complex statistical analyses should be applied to include time effects/order to control for this redundant variance in each unique variable.

Third, in the present study we analyzed HRV during sleep; most studies deprive or restrict sleep in their participants for several days and investigate autonomic dysfunction the following days (cf. Spiegel et al., 1999; 2004; 2005). Compared to controls, narcoleptic patients exhibit similar autonomic function during sleep, however there are significant differences in autonomic function during wakefulness (before sleep) between the groups (Ferini-Strambi et al., 1997), suggesting that autonomic function impairment may be influenced by both sleep and wake cycles. It is therefore recommended that both sleep and wake cycles be investigated; however, this would be difficult as it becomes more invasive and intrusive for participants (Katz et al., 2002; Scholle et al., 2003).

The fourth weakness in this study was that healthy children underwent the in-home sleep study for *one* night under normal sleeping conditions. The in-home sleep study was performed only once and results may have been influenced by the *first night effect*. The first night effect has been observed in children and adolescents, which results

in reduced sleep duration, sleep quality, and sleep continuity (Emslie et al., 2001). Future studies should consider an adaptation night to permit acclimatization to the recording equipment during sleep.

Fifth, through several experimental studies, partial and chronic sleep deprivation have been shown to alter the concentration levels of several hormone levels, including cortisol, ghrelin, leptin, and insulin in adults (Spiegel et al., 1999; 2004). In particular, because leptin levels are associated with the activation of the SNS, inducing an increase in heart rate, blood pressure (Shek, Brands, & Hall, 1998), and sympathetic outflow to kidneys and adipose tissue (Haynes, Sivitz, Morgan, Walsh, & Mark, 1997), leptin should be examined. Unfortunately, in the present study no hormones were measured. It is highly recommended that future studies investigate these physiological changes in hormone levels during sleep in youth.

The sixth limitation was the modest sample size, which due to power limitations precluded some of the statistical analyses. Because the BioSomnia sleep recorder is new technology, several participants experienced problems associated with correctly operating the equipment and we quickly learned gel-based electrodes were needed to adequately capture the EEG signals; these technical problems reduced our sample size by about 30%. The reduced sample size and inevitably loss of power may explain why frequency-domain HRV variables could not be used as manifest variables in the SEM model. However, the average sample size in standard PSG sleep studies with youth is about 60 (ranges from 24 to 87; e.g., Beebe et al., 2007; Kirov et al., 2004; O'Brien et al., 2004). In addition, because we obtained multiple measures within each participant, the reliability and stability of PSG derived variables increased.

Lastly, although we statistically controlled for the effects of age (sex and pubertal status did not differ), we did not control for other important covariates including food intake, physical activity, and duration of obesity.

Future Directions

To date, this was the first study that investigated the association between autonomic dysfunction, sleep, and obesity in children and adolescents using objectively assessed PSG data. Given that autonomic dysfunction, as measured by HRV, did not show evidence of partially mediating the association between sleep and obesity, it is important to consider how other indicators of autonomic dysfunction may play a role. For example, blood pressure typically drops by 10-20% during sleep and this drop is referred to as “dipping” (Pickering, 1990). Interestingly, individuals who do not show dipping (non-dippers) exhibit less time in SWS (Loredo, Nelesen, Ancoli-Israel, & Dimsdale, 2004) and are at greater risk for hypertension, left-ventricular hypertrophy, and mortality, independent of 24-hour blood pressure and obstructive sleep apnea (Ohkubo et al., 2002; Verdecchia et al., 1990). This suggests another potential autonomic dysfunction pathway that should be further investigated.

Although sleep is linked with multiple physiological changes, (i.e., metabolic, endocrine, and cerebral activity) and psychosocial factors (i.e., stress, socio-economic status), there is little information available as to the role between sleep and obesity, especially within a pediatric population. In future studies it is important to examine other metabolic and hormonal profiles (e.g., cortisol, ghrelin, leptin, insulin, etc.) across specific sleep stages (i.e., SWS and REM sleep). Much of this work has been conducted in lean healthy young men and results cannot be generalized to children and adolescents.

Investigating the metabolic and endocrine functioning across sleep stages in children may elucidate other physiologic responses that confer risk for the development and maintenance of obesity.

Conclusion

Given that obesity is a risk factor for multiple chronic diseases, disability, and premature death, better understanding of the pathogenesis of obesity is of great importance. While robust data support an association between sleep and obesity, the mechanisms underlying their relation are complex and unclear. The present study was aimed to develop new knowledge about autonomic dysfunction as one such possible mechanism.

In summary, short sleep duration, poor fragmented sleep quality, more time in REM sleep, and less time in SWS were associated with greater adiposity in children and adolescents. Similarly, autonomic dysfunction, characterized by reduced HRV and parasympathetic activity was associated with greater adiposity in youth. Autonomic dysfunction did not directly mediate the relation between sleep and obesity. Future research should further examine how autonomic functioning over the course of sleep in synchronization with hormonal and endocrine functioning, affect the relation between sleep and obesity.

REFERENCES

- Acebo, C., Millman, R. P., Rosenberg, C., Cavallo, A., & Carskadon, M. A. (1996). Sleep, breathing, and cephalometrics in older children and young adults. *Chest, 109*, 664-672.
- Achermann, P., & Borbely, A. A. (2003). Mathematical models of sleep regulation. *Frontiers in Bioscience 8*, 683-693.
- Afghani A., & Goran, M. I. (2006). Racial differences in the association of subcutaneous and visceral fat on bone mineral content in prepubertal children. *Calcified Tissue International, 79*, 383-388.
- Agarwal, A., Anand, I. S., Sakhuja, V., & Chugh, K. S. (1991). Effect of dialysis and renal transplantation on autonomic dysfunction in chronic renal failure. *Kidney International, 40*, 489-495.
- Agras, W. S., Hammer, L. D., McNicholas, F., & Kraemer, H. C. (2004). Risk factors for childhood overweight: A prospective study from birth to 9.5 years. *Journal of Pediatrics, 145*, 20-25.
- Akinci, A., Çeliker, A., Baykal, E., & Teziç, T. (1993). Heart rate variability in diabetic children: Sensitivity of the time- and frequency-domain methods. *Pediatric Cardiology, 14*, 140-146.
- Ako, M., Kawara, T., Uchida, S., Miyazaki, S., Nishihara, K., Mukai, J., et al. (2003). Correlation between electroencephalography and heart rate variability during sleep. *Psychiatry and Clinical Neurosciences, 7*, 59-65.

- Akselrod, S., Gordon, D., Madwed, J. B., Snidman, N. C., Shannon, D. C., & Cohen, R. J. (1985). Hemodynamic regulation: Investigation by spectral analysis. *American Journal of Physiology- Heart and Circulatory*, 249, H867-H875.
- Akselrod, S., Gordon, D., Ubel, F. A., Shannon, D. C., Berger, A. C., & Cohen, R. J. (1981). Power spectrum analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science*, 213, 220-222.
- Allison, D. B., Fontaine, K. R., Manson, J. E., Stevens, J., & Vanltallie, T. B. (1999). Annual deaths attributable to obesity in the United States. *The Journal of the American Medical Association*, 282, 1530-1538.
- Al Mamun, A., Lawlor, D. A., Cramb, S., O'Callaghan, M., Williams, G., & Najman, J. (2007). Do childhood sleeping problems predict obesity in young adulthood? Evidence from a prospective birth cohort study. *American Journal of Epidemiology*, 166, 1368-1373.
- American Thoracic Society: Standards and indications for cardiopulmonary sleep studies in children. (1996). *American Journal of Respiratory Critical Care Medicine*, 153, 866-878.
- Ancoli-Israel, S., Kripke, D. F., Mason, W., & Messin, S. (1981). Comparisons of home sleep recordings and polysomnograms in older adults with sleep disorders. *Sleep*, 4, 283-291.
- Anders, T. F., Carskadon, M. A., & Dement, W. C. (1980). Sleep and sleepiness in children and adolescents. *Pediatric Clinics of North America*, 27, 29-43.
- Archbold, K. H., Pituch, K. J., Panjabi, P., & Chervin, R. D. (2002). Symptoms of sleep

- disturbances among children at two general pediatric clinics. *Journal of Pediatrics*, 140, 97-102.
- Archer, S. N., Robilliard, D. L., Skene, D. J., Marcel, S., Williams, A., Arendt, J., et al. (2003). A length polymorphism in the circadian clock gene *Per3* is linked to delayed sleep phase syndrome and extreme diurnal preference. *Sleep*, 26, 413-415.
- Aronne, L. J., Mackintosh, R., Rosenbaum, M., Leibel, R. L., & Hirsch, J. (1997). Cardiac autonomic nervous system activity in obese and never-obese young men. *Obesity Research*, 5, 354-359.
- Ayas, N. T., White, D. P., Manson, J. E., Stampfer, M. J., Speizer, F. E., Malhotra, A., & Hu, F. B. (2003). A prospective study of sleep duration and coronary heart disease in women. *Archives of Internal Medicine*, 163, 205-209.
- Avon Longitudinal Study of Parents and Children (ALSPAC). (2006). *The Growing and Changing Questionnaire*. Retrieved September 2006, from <http://www.bristol.ac.uk/alspac/documents/ques-cb16a-mum-and-daughter-at-8.pdf>.
- Barlow, S. E., & Dietz, W. H. (1998). Obesity evaluation and treatment: Expert committee recommendations. *Pediatrics*, 102, e29.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Pathology*, 51, 1173-1182.
- Beebe, D. W., Lewin, D., Zeller, M., McCabe, M., MacLeod, K., Daniels, S. R., & Amin,

- R. (2007). Sleep in overweight adolescents: Shorter sleep, poorer sleep quality, sleepiness, and sleep-disordered breathing. *Journal of Pediatric Psychology, 32*, 69-79.
- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238-246.
- Berlad, I. I., Shlitner, A., Ben-Haim, S., & Lavie, P. (1993). Power spectrum analysis and heart rate variability in Stage 4 and REM sleep: Evidence for state-specific changes in autonomic dominance. *Journal of Sleep Research, 2*, 88-90.
- Berntson, G. G., Bigger, J. T., Eckberg, D. L., Grossman, P., Kaufman, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34*, 623-648.
- Berntson, G. G., & Stowell, J. R. (1998). ECG artifacts and heart period variability: Don't miss a beat! *Psychophysiology, 35*, 127-132.
- Bigger Jr. J. T., Fleiss, J. L., Steinman, R. C., Rolnitzky, L. M., Kleiger, R. E., & Rottman, J. N. (1992). Frequency domain measures of heart period variability and mortality after myocardial infarction. *Circulation, 85*, 164-171.
- Billman, G. E., & Hoskins, R. S. (1988). Cocaine-induced ventricular fibrillation: Protection afforded by the calcium antagonist verapamil. *The Federation of American Societies for Experimental Biology, 2*, 2990-2995.
- Blaaha, M. J., Gebretsadik, T., Shintani, A., & Elasy, T. A. (2008). Waist circumference, not the metabolic syndrome, predicts glucose deterioration in type 2 diabetes. *Obesity, 16*, 869-874.

- Blasi, A., Jo, J., Valladares, E., Morgan, B. J., Skatrud, J. B., & Khoo, M. C. K. (2003). Cardiovascular variability after arousal from sleep: Time-varying spectral analysis. *Journal of Applied Physiology*, *95*, 1394-1404.
- Brandenberger, G., Buchheit, M., Ehrhart, J., Simon, C., & Piquard, F. (2005). Is slow wave sleep an appropriate recording condition for heart rate variability analysis? *Autonomic Neuroscience: Basic and Clinical*, *121*, 81-86.
- Bray, G. A. (1991). Obesity, a disorder of nutrient partitioning: The Mona Lisa Hypothesis. *Journal of Nutrition*, *121*, 1146-1162.
- Bray, G. A., York, D. A., & Fisler, J. S. (1989). Experimental obesity: A homeostatic failure due to defective nutrient stimulation of the sympathetic nervous system. *Vitamins and Hormones*, *45*, 1-125.
- Bonnet, M. H., & Arand, D. L. (1997). Heart rate variability: Sleep stage, time of night, and arousal influences. *Electroencephalography and Clinical Neurophysiology*, *102*, 390-396.
- Bonnet, M. H., & Arand, D. L. (1998). Heart rate variability in insomniacs and matched normal sleepers. *Psychosomatic Medicine*, *60*, 610-615.
- Burgess, H. J., Trinder, J., & Kim, Y. (1996). Cardiac parasympathetic nervous system activity does not increase in anticipation of sleep. *Journal of Sleep Research*, *5*, 83-89.
- Burr, R. L. (2007). Interpretation of normalized spectral heart rate variability indices in sleep research: A critical review. *Sleep*, *30*, 913-919.
- Busek, P., Vankova, J., Opavsky, J., Salinger, J., & Nevsimalova, S. (2005). Spectral analysis of heart rate variability in sleep. *Physiological Research*, *54*, 369-376.

- Caballero, A. E. (2003). Endothelial dysfunction in obesity and insulin resistance: A road to diabetes and heart disease. *Obesity Research, 11*, 1278-1289.
- Cajochen, C., & Dijk, D. J. (2003). Electroencephalographic activity during wakefulness, rapid eye movement and non-rapid eye movement sleep in humans: Comparison of their circadian and homeostatic modulation. *Sleep and Biological Rhythms, 1*, 85-95.
- Campfield, L. A., Smith, F. J., Guisez, Y., Devos, R., & Burn, P. (1995). Recombinant mouse ob protein: Evidence for a peripheral signal linking adiposity and central neural networks. *Science, 269*, 546-549.
- Carskadon, M. A. (1999). When worlds collide: Adolescent need for sleep versus societal demands. *Phi Delta Kappan, 80*, 348-353.
- Carskadon, M. A., Acebo, C., & Jenni, O. G. (2004). Regulation of adolescent sleep: Implications for behavior. *Annals of the New York Academy of Sciences, 1021*, 276-291.
- Carskadon, M. A., & Dement, W. C. (1987). Daytime sleepiness: Quantification of a behavioral state. *Neuroscience and Biobehavioral Reviews, 11*, 307-317.
- Carskadon, M. A., Dement, W. C., Mitler, M. M., Roth, T., Westbrook, P. R., & Keenan, S. (1986). Guideline for the multiple sleep latency test (MSLT): A standard measure of sleepiness. *Sleep, 9*, 519-524.
- Carskadon, M. A., Wolfson, A. R., Acebo, C., Tzischinsky, O., & Seifer, R. (1998). Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. *Sleep, 21*, 871-881.

- Capaldi II, V. F., Handwerger, K., Richardson, E., & Stroud, R. L. (2005). Associations between sleep and cortisol responses to stress in children and adolescents: A pilot study. *Behavioral Sleep Medicine, 3*, 177-192.
- Casolo, G., Balli, E., Taddei, T., Amuhasi, J., & Gori, C. (1989). Decreased spontaneous heart rate variability in congestive heart failure. *The American Journal of Cardiology, 64*, 1162-1167.
- Chang, P. P., Ford, D. E., Mead, L. A., Cooper-Patrick, L. & Klag, M. J. (1997). Insomnia in young men and subsequent depression. *American Journal of Epidemiology, 146*, 105-114.
- Chaput, J-P., Brunet, M., & Tremblay, A. (2006). Relationship between short sleeping hours and childhood overweight/obesity: Results from the 'Québec en Forme' Project. *International Journal of Obesity, 30*, 1080-1085.
- Chen, X., Beydoun, M. A., & Wang, Y. (2008). Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. *Obesity, 16*, 265-274.
- Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. *British Medical Journal, 320*, 1-6.
- Daan, S., Beersman, D. G. M., & Borbely, A. A. (1984). Timing of human sleep: Recovery process gated by a circadian pacemaker. *American Journal of Physiology, 246*, 161-178.
- Davy, K. P., & Orr, J. S. (2008). Sympathetic nervous system behavior in human obesity. *Neuroscience and Biobehavioral Reviews*, doi:10.1016/j.neubiorev.2008.05.024.
- de Ridder, C. M., de Boer, R. W., Seidell, J. C., Nieuwenhoff, C. M., Jeneson, J. A.,

- Bakker, C. J., et al. (1992). Body fat distribution in pubertal girls quantified by magnetic resonance imaging. *International Journal of Obesity Related Metabolic Disorders*, 16, 443-449.
- Dekker, J. M., Crow, R. S., Folsom, A. R., Hannan, P. J., Liao, D., Swenne, C. A., et al. (2000). Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: The ARIC Study. *Circulation*, 102, 1239-1244.
- Dekker, J. M., Schouten, E. G., Klootwijk, P., Pool, J., Swenne, C. A., & Kromhout, D. (1997). Heart rate variability from short electrocardiographic recordings predicts mortality from all causes in middle-aged and elderly men. *American Journal of Epidemiology*, 145, 899-908.
- Dew, M. A., Hoch, C. C., Buysse, D. J., Monk, T. H., Begley, A. E., & Houck, P. R. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine*, 65, 63-73.
- Dietz, W. H. (1998). Health consequences of obesity in youth: Childhood predictors of adult disease. *Pediatrics*, 101, 518-525.
- Dijk, D. J., & Czeisler, C. A. (1995). Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *Journal of Neuroscience*, 15, 3526-3538.
- Drake, C., Nickel, C., Burduvali, E., Roth, T., Jefferson, C., & Badia, P. (2003). The Pediatric Daytime Sleepiness Scale (PDSS): Sleep habits and school outcomes in middle school children. *Sleep*, 26, 455-458.

- Duffy, J. F., Dijk, D. J., Hall, E. F., & Czeisler, C. A. (1999). Relationship of endogenous circadian melatonin and temperature rhythms to self-reported preference for morning or evening activity in young and older people. *Journal of Investigative Medicine*, 47, 141-150.
- Dzaja, A., Dalal, M. A., Himmerich, H., Uhr, M., Pollmacher, T., & Schuld, A. (2004). Sleep enhances nocturnal plasma ghrelin levels in healthy subjects. *American Journal of Physiology Endocrinology and Metabolism*, 286, E963–E967.
- Ebbeling, C., Pawlak, D., & Ludwig, D. (2002). Childhood obesity: Public-health crisis, common sense cure. *The Lancet*, 360, 473-482.
- Eckberg, D. L., & Sleight, P. (1992). *Human baroreflexes in health and disease*. Oxford, UK: Clarendon Press, 216-226.
- Eisenmann, J. C., Ekkekakis, P., & Holmes, M. (2006). Sleep duration and overweight among Australian children and adolescents. *Acta Paediatrica*, 95, 956-963.
- Ekstedt, M., Akerstedt, T., & Sodestrom, M. (2004). Microarousals, during sleep are associated with increased levels of lipids, cortisol and blood pressure. *Psychosomatic Medicine*, 66, 925-931.
- Elsenbruch, S., Harnish, M. J., & Orr, W. C. (1999). Heart rate variability during waking and sleep in healthy males and females. *Sleep*, 22, 1067-1071.
- Emslie, G. J., Armitage, R., Weinberg, W. A., Rush, J., Mayes, T. L., & Hoffmann, R. F. (2001). Sleep polysomnography as a predictor of recurrence in children and adolescents with major depressive disorder. *International Journal of Neuropsychopharmacology*, 4, 159-168.
- Ewing, D. J., Martyn, C. N., Young, R. J., & Clarke, B. F. (1985). The value of

cardiovascular autonomic function tests: A ten year experience in diabetes.

Diabetes Care, 8, 491-498.

Ewing, D. J., Neilson, J. M., Shapiro, C. M., Stewart, J. A., & Reid, W. (1991). Twenty four hour heart rate variability: Effects of posture, sleep, and time of day in healthy controls and comparison with bedside tests of autonomic function in diabetic patients. *British Heart Journal*, 65, 239-244.

Fallone, G., Owens, J. A., & Deane, J. (2002). Sleepiness in children and adolescents: Clinical implications. *Sleep Medicine Reviews*, 6, 287-306.

Ferguson, M. A., Gutin, B., Owens, S., Litaker, M., Tracy, R. P., & Allison J. (1998). Fat distribution and hemostatic measures in obese children. *The American Journal of Clinical Nutrition*, 67, 1136-1140.

Ferini-Strambi, L., Spera, A., Oldani, A., Zucconi, M., Bianchi, A., Cerutti, S., & Smirne, S. (1997). Autonomic function in narcolepsy: Power spectrum analysis of heart rate variability. *Journal of Neurology*, 244, 252-255.

Finley, J. P., Nugent, S. T., & Hellenbrand, W. (1987). Heart-rate variability in children. Spectral analysis of developmental changes between 5 and 24 years. *Canadian Journal of Physiology and Pharmacology*, 65, 2048-2052.

Finley, J. P., Nugent, S. T., Hellenbrand, W., Craig, M., & Gillis, D. A. (1989). Sinus arrhythmia in children with atrial septal defect: An analysis of heart rate variability before and after surgical repair. *British Heart Journal*, 61, 280-284.

Flegal, K. M., & Troiano, R. P. (2000). Changes in the distribution of body mass index of adults and children in the U. S. population. *International Journal of Obesity*, 24, 807-818.

- Flodmark, C. E., Sveger, T., & Nilsson-Ehle, P. (1994). Waist measurement correlates to a potentially atherogenic lipoprotein profile in obese 12-14 year old children. *Acta Paediatrica*, 83, 941-945.
- Ford, E. S., Galuska, D. A., Gillespie, C., Will, J. C., Giles, W. H., & Dietz, W. H. (2001). C-reactive protein and body mass index in children: Findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *The Journal of Pediatrics*, 138, 486-492.
- Fox, K., Peters, D., Armstrong, N., Sharpe, P., & Bell, M. (1993). Abdominal fat deposition in 11-year-old children. *International Journal of Obesity Related Metabolic Disorders*, 17, 11-16.
- Franco, P., Seret, N., Van Hees, J. N., Lanquart, J-P., Groswasser, J., & Kahn, A. (2003). Cardiac changes during sleep in sleep-deprived infants. *Sleep*, 26, 845-848.
- French, S. A., Story, M., & Perry C., L. (1995). Self-esteem and obesity in children and adolescents: A literature review. *Obesity Research*, 3, 479-490.
- Freedman, D. S., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (1999). The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa Heart Study. *Pediatrics*, 103, 1175-1182.
- Freedman, D. S., Khan, L. K., Serdula, M. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2005). The relation of childhood BMI to adult adiposity: The Bogalusa Heart Study. *Pediatrics*, 115, 22-27.
- Freedman, D.S., Serdula, M.K., Srinivasan, S.R., & Berenson, G.S. (1999). Relation of

- circumferences and skinfold thickness to lipid and insulin concentrations in children and adolescents: The Bogalusa Heart Study. *American Journal of Clinical Nutrition*, 69, 308-317.
- Freedman, D. S., Wang, J., Maynard, L. M., Thornton, J. C., Mei, Z., Pierson Jr. R. N., et al. (2005). Relation of BMI to fat and fat-free mass among children and adolescents. *International Journal of Obesity* 29, 1–8.
- Freeman, R., Weiss, S. T., Roberts, M., Zbikowski, S. M., & Sparrow, D. (1995). The relationship between heart rate variability and measures of body habitus. *Clinical Autonomic Research*, 5, 261-266.
- Fukushima, M., Tokunaga, K., Lupien, J., Kemnitz, J. W., & Bray, G. A. (1987). Dynamic and static phases of obesity following lesions in PVN and VMH. *Regulatory, Integrative and Comparative Physiology*, 253, 523-529.
- Furness, J. B. (2006). The organisation of the autonomic nervous system: Peripheral connections. *Autonomic Neuroscience: Basic and Clinical*, 130, 1-5.
- Gais, S., Plihal, W., Wagner, U., & Born, J. (2000). Early sleep triggers memory for early visual discrimination skills. *Nature Neuroscience* 3, 1335-1339.
- Gaina, A., Sekine, M., Chen, X., Hamanishi, S., & Kagamimori, S. (2004). Sleep parameters recorded by actiwatch in elementary school children and junior high school adolescents: Schooldays vs weekends. *Sleep and Hypnosis*, 6, 66-77.
- Gau, S-F., & Soong W-T. (2003). The transition of sleep-wake patterns in early adolescence. *Sleep*, 26, 449-454.
- Gibson, S., Lambert, J., & Neate, D. (2004). Associations between weight status, physical

- activity, and consumption of biscuits, cakes and confectionary among young people in Britain. *Nutrition Bulletin*, 29, 301-309.
- Goldberger, J.J. (1999). Sympathovagal balance: How should we measure it? *American Journal of Physiology*, 4, H1273–H1280.
- Goldsmith, R. J., Casola, P., & Varenbut, M. (2006). Sleep complications in depression, anxiety, and psychotic disorders and their treatment. *Psychiatric Annals*, 36, 853-859.
- Goran, M. I., Gower, B. A., Treuth, M., & Nagy, T. R. (1998). Prediction of intraabdominal and subcutaneous abdominal adipose tissue in healthy prepubertal children. *International Journal of Obesity Related Metabolic Disorders*, 22, 549-558.
- Goulding, A., Jones, I. E., Taylor, R. W., Williams, S. M., & Manning, P. J. (2001). Bone mineral density and body composition in boys with distal forearm fractures: A dual-energy x-ray absorptiometry study. *The Journal of Pediatrics*, 139, 509-515.
- Grossman, P., & Kollai, M. (1993). Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: Within- and between-individual relations. *Psychophysiology*, 30, 486-495.
- Gupta, N. K., Mueller, W. H., Chan, W., & Meininger, J. C. (2002). Is obesity associated with poor sleep quality in adolescents? *American Journal of Human Biology*, 14, 762-768.
- Halász, P., Terzano, M., Parrino, L., & Bódizs, R. (2004). The nature of arousal in sleep. *Journal of Sleep Research*, 13, 1-23.
- Hall, M., Vasko, R., Buysse, D., Ombao, H., Chen, Q., Cashmere, J. D., et al. (2004).

- Acute stress affects heart rate variability during sleep. *Psychosomatic Medicine*, 66, 56-62.
- Hartikainen, J., Fyhrquist, F., Tahvanainen, K., Länsimies, E., & Pyörälä, K. (1995). Baroreflex sensitivity and neurohormonal activation in patients with acute myocardial infarction. *British Heart Journal*, 74, 21-26.
- Hartz, A., Grubb, B., Wild, R., Van Nort, J. J., Kuhn, E., Freedman, D., & Rimm, A. (1990). The association of waist hip ratio and angiographically determined coronary artery disease. *International Journal of Obesity*, 14, 657-665.
- Hatzinger, M., Brand, S., Perren, S., Stadelmann, S., von Wyl, A., von Klitzing, K., & Holsboer-Trachsler, E. (2007). Electroencephalographic sleep profiles and hypothalamic–pituitary–adrenocortical-activity in kindergarten children: Early indication of poor sleep quality associated with increased cortisol secretion. *Journal of Psychiatric Research*, 42, 532-543.
- Haynes, W. G., Sivitz, W. I., Morgan, D. A., Walsh, S. A., & Mark, A. L. (1997). Sympathetic and cardiorenal actions of leptin. *Hypertension*, 30, 619-623.
- Heussler, H. S. (2005). Common causes of sleep disruption and daytime sleepiness: Childhood sleep disorders II. *The Medical Journal of Australia*, 182, 484-489.
- Hillman, D. R., Murphy, A. S., Antic, R., & Pezzullo, L. (2006). The economic cost of sleep disorders. *Sleep*, 29, 299-305.
- Hirsch, J., Leibel, R. L., Mackintosh, R., & Aguirre, A. (1991). Heart rate variability as a measure of autonomic function during weight change in humans. *American Journal of Physiology*, 261, 1418-1423.

- Hoban, T. F., & Chervin, R. D. (2001). Assessment of sleepiness in children. *Seminars in Pediatric Neurology*, 8, 216-228.
- Horvath, T. L., Diano, S., Sotonyo, P., Heiman, M., & Tschop, M. (2001). Minireview: Ghrelin and the regulation of energy balance—A hypothalamic perspective. *Endocrinology*, 142, 4163-4169.
- Hossain, P., Kavar, B., & El Nahas, M. (2007). Obesity and diabetes in the developing world - A growing challenge. *The New England Journal of Medicine*, 356, 213-215.
- Howard, B. J., & Wong, J. (2001). Sleep Disorders. *Pediatrics in Review*, 22, 327-342.
- Hui, L. L., Nelson, E. A. S., Yu, L. M., & Fok, T. F. (2003). Risk factors for childhood overweight in 6- to 7-y-old Hong Kong children. *International Journal of Obesity*, 27, 1411-1418.
- Hull Jr., S. S., Evans, A. R., Vanoli, E., Adamson, P. B., Stramba-Badiale, M., Albert, D. E., et al. (1990). Heart rate variability before and after myocardial infarction in conscious dogs at high and low risk of sudden death. *Journal of the American College of Cardiology*, 16, 978-985.
- Iber, C., Redline, S., Kaplan Gilpin A. M., Quan, S. F., Zhang, L., Gottlieb, D. J., et al. (2004). Polysomnography performed in the unattended home versus the attended laboratory setting-Sleep Heart Health Study methodology. *Sleep*, 27, 536-540.
- Iglowstein, I., Jenni, O. G., Molinari, L., & Largo, R. H. (2003). Sleep duration from infancy to adolescence: Reference values and generational trends. *Pediatrics*, 111, 302-307.
- Imaki, M., Hatanaka, Y., Ogawa, Y., Yoshida, Y., & Tanada, S. (2002). An

- epidemiological study on relationship between the hours of sleep and life style factors in Japanese factory workers. *Journal of Physiological Anthropology and Applied Human Science*, 21, 115-120.
- Irwin, M., Thompson, J., Miller, C., Gillin, J. C., & Ziegler, M. (1999). Effects of sleep and sleep deprivation on catecholamine and interleukin-2 levels in humans: Clinical implications. *The Journal of Clinical Endocrinology & Metabolism*, 84, 1979-1985.
- Johnson, L., Lubin, A., Naitoh, P., Nute, C., & Austin, M. (1969). Spectral analysis of the EEG of dominant and non-dominant alpha subjects during waking and sleeping. *Electroencephalography Clinical Neurophysiology*, 26, 361-370.
- Joreskog, K. G., & Sorbom, D. (2001). *LISREL (Version 8.8). Structural equation modeling* [Computer Software]. Chicago: Scientific Software International.
- Kagamimori, S., Yamagami, T., Sokejima, S., Numata, N., Handa, K., Nanri, S., et al. (1999). The relationship between lifestyle, social characteristics and obesity in 3-year-old Japanese children. *Child: Care, Health & Development*, 25, 235-247.
- Kalra, S. P., & Kalra, P. S. (2004). NPY and cohorts in regulating appetite, obesity and metabolic syndrome: Beneficial effects of gene therapy. *Neuropeptides*, 38, 201-211.
- Kapur, V. K., Rapoport, D. M., Sanders, M. H., Enright, P., Hill, J., Iber, C., et al. (2000). Rates of sensor loss in unattended home polysomnography: The influence of age, gender, obesity, and sleep-disordered breathing. *Sleep*, 23, 682-688.

- Karason, K., Molgaard, H., Wikstrand, J., & Sjostrom, L. (1999). Heart rate variability in obesity and the effect of weight loss. *American Journal of Cardiology*, *83*, 1242-1247.
- Katz, E. S., Greene, M. G., Carson, K. A., Galster, P., Loughlin, G. M., Carroll, J., & Marcus, C. L. (2002). Night-to-night variability of polysomnography in children with suspected obstructive sleep apnea. *The Journal of Pediatrics*, *140*, 589-594.
- Kaufman, C. L., Kaiser, D. R., Kelly, A. S., Dengel, J. L., Steinberger, J., & Dengel, D. R. (2008). Diet revision in overweight children: Effect on autonomic and vascular function. *Clinical Autonomic Research*, *18*, 105-108.
- Kaufman, C. L., Kaiser, D. R., Steinberger, J., Kelly, A. S., & Dengel, D. R. (2007). Relationships of cardiac autonomic function with metabolic abnormalities in childhood obesity. *Obesity*, *15*, 1164-1171.
- Keim, N. L., Stern, J. S., & Havel, P. J. (1998). Relation between circulating leptin concentrations and appetite during a prolonged, moderate energy deficit in women. *The American Journal of Clinical Nutrition*, *68*, 794-801.
- Kelman, B. B. (1999). The sleep needs of adolescents. *Journal of School Nursing*, *15*, 14-19.
- Kitney, R. I. (1980). An analysis of the thermoregulatory influences on heart-rate variability. In R. I. Kitney & O. Rompelman (Eds.), *The Study of Heart-Rate Variability* (pp. 81-106). Oxford: Clarendon Press.
- Kirov, R., Kinkelbur, J., Heipke, S., Kostanecka-Endress, T., Westhoff, M., Cohrs, S., et al. (2004). Is there a specific polysomnographic sleep pattern in children with attention deficit/hyperactivity disorder? *Journal of Sleep Research*, *13*, 87-93.

- Kleiger, R. E., Stein, P. K., Bosner, M. S., & Rottman, J. N. (1992). Time domain measurements of heart rate variability. *Cardiology Clinics*, *10*, 487-498.
- Klerman, E. B., & Dijk, D. J. (2005). Interindividual variation in sleep duration and its association with sleep debt in young adults. *Sleep*, *28*, 1253-1259.
- Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York: Guilford Press.
- Knehans, A. W., & Romsos, D. R. (1983). Norepinephrine turnover in obese (ob/ob) mice: Effects of age, fasting, and acute cold. *American Journal of Physiology, Endocrinology and Metabolism*, *244*, 567-574.
- Knuston, K. L. (2005). Sex differences in the association between sleep and body mass index in adolescents. *Journal of Pediatrics*, *147*, 830-834.
- Kopelman, P. G. (2000). Obesity as a medical problem. *Nature*, *404*, 635-643.
- Kripke, D. F., Simons, R. N., Garfinkel, L., & Hammond, E. C. (1979). Short and long sleep and sleeping pills. Is increased mortality associated? *Archives of General Psychiatry*, *36*, 103-116.
- Kuczmarski, R. J., Carroll, M. D., Flegal, K. M., & Troiano, R. P. (1997). Varying body mass index cutoff points to describe overweight prevalence among U.S. adults: NHANES III (1988 to 1994). *Obesity Research*, *5*, 542-548.
- Kuczmarski, R. J., Ogden, C. L., Grummer-Strawn, L. M., Flegal, K. M., Guo, S. S., Wei, R., et al. (2000). CDC growth charts: United States. *Advanced Data*, *314*, 1-27.
- Kuo, L. E., Kitlinska, J. B., Tilan, J. U., Li, L., Baker, S. B., Johnson, M. D., et al. (2007). Neuropeptide Y acts directly in the periphery on fat tissue and mediates stress-induced obesity and metabolic syndrome. *Nature Medicine*, *13*, 803-811.

- Laederach-Hofman, K., Mussgay, M., & Ruddel, H. (2000). Autonomic cardiovascular regulation in obesity. *Journal of Endocrinology*, 164, 59-66.
- Lahiri, M. K., Prince, J., Kannankeril, J., & Goldberger, J. J. (2008). Assessment of autonomic function in cardiovascular disease. *Journal of the American College of Cardiology*, 51, 1725-1733.
- Landsberg, L. (1986). Diet, obesity and hypertension: An hypothesis involving insulin, the sympathetic nervous system, and adaptive thermogenesis. *QJM: An International Journal of Medicine*, 61, 1081-1090.
- Laposky, A. D., Bass, J., Kohsaka, A., & Turek, F. W. (2008). Sleep and circadian rhythms: Key components in the regulation of energy metabolism. *Federation of European Biochemical Societies Letters*, 582, 142-151.
- Lee, K., Song, Y-M., & Sung, J. (2008). Which obesity indicators are better predictors of metabolic risk?: Healthy twin study. *Obesity*, 16, 834-840.
- Léger, D., Scheuermaier, K., Philip, P., Paillard, M., & Guilleminault, C. (2001). Evaluation of quality of life in severe and mild insomniacs compared with good sleepers. *Psychosomatic Medicine*, 63, 49-55.
- Levin, B. E., Triscari, J., & Sullivan, A. C. (1983). Altered sympathetic activity during development of diet-induced obesity in rat. *American Journal of Physiology, Regulatory, Integrative and Comparative Physiology*, 244, 347-355.
- Liao, D., Cai, J., Barnes, R.W., Tyroler, H. A., Rautaharju, P., Holme, I., & Heiss, G. (1996). Association of cardiac autonomic function and the development of hypertension: the ARIC study. *American Journal of Hypertension*, 9, 1147-1156.
- Liatis, S., Tentolouris, N., & Katsilambros, N. (2004). Cardiac autonomic nervous

- system activity in obesity. *Pediatric Endocrinology Reviews*, 1, 476-483.
- Linkowski, P. (1999). EEG sleep patterns in twins. *Journal of Sleep Research*, 8, 11-13.
- Liu, X., Uchiyama, M., Okawa, M., & Kurita, H. (2000). Prevalence and correlates of self-reported sleep problems among Chinese adolescents. *Sleep*, 23, 27-34.
- Lockley, S. W., Skene, D. J., & Arendt, J. (1999). Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *Journal of Sleep Research*, 8, 175-183.
- Loredo, J. S., Nelesen, R., Ancoli-Israel, S., & Dimsdale, J. E. (2004). Sleep quality and blood pressure dipping in normal adults. *Sleep*, 27, 1097-1103.
- Lumeng, J. C., Somashekar, D., Appugliese, D., Kaciroti, N., Corwyn, R. F., & Bradley, R. H. (2007). Shorter sleep duration is associated with increased risk of being overweight at ages 9 to 12 years. *Pediatrics*, 120, 1020-1029.
- Lusardi, P., Mugellini, A., Preti, P., Zoppi, A., Derosa, G., & Fogari, R. (1996). Effects of a restricted sleep regimen on ambulatory blood pressure monitoring in normotensive subjects. *American Journal of Hypertension*, 9, 503-505.
- Malliani, A., Pagani, M., & Lombardi, F. (1994). Physiology and clinical implications of variability of cardiovascular parameters with focus on heart rate and blood pressure. *American Journal of Cardiology*, 73, 3C-9C.
- Malliani, A., Pagani, M., Lombardi, F. & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, 84, 1482-1492.
- Malpas, S. C., & Purdie, G. L. (1990). Circadian variation of heart rate variability. *Cardiovascular Research*, 24, 210-213.
- Mancia, G. (1993). Autonomic modulation of the cardiovascular system during

- sleep. *New England Journal of Medicine*, 328, 347–349.
- Marshall, W. A., & Tanner, J. M. (1969). Variations in the pattern of pubertal changes in girls. *Archives of Disease in Childhood*, 44, 291-303.
- Martini, G., Riva, P., Rabbia, F., Molini, V., Ferrero, G. B., Cerutti, F., Carra, R., & Veglio, F. (2001). Heart rate variability in childhood obesity. *Clinical Autonomic Research*, 11, 87-91.
- Massin, M. M., Maeyns, K., Withofs, N., Ravet, F., & Gerrad, P. (2000). Circadian rhythm of heart rate and heart rate variability. *Archives of Disease in Childhood*, 83, 179-182.
- Masuo, K., Katsuya, T., Fu, Y., Rakugi, H., Ogihara, T., & Tuck, M. L. (2005). Adrenergic receptor polymorphisms are related to the onset of weight gain and blood pressure elevation over 5 years. *Circulation*, 111, 3429-3434.
- Masuo, K., Kawaguchi, H., Mikami, H., Toshio Ogihara, T., & Tuck, M. L. (2003). Serum uric acid and plasma norepinephrine concentrations predict subsequent weight gain and blood pressure elevation. *Hypertension*, 42, 474-480.
- Masuo, K., Mikami, H., Ogihara, T., & Tuck, M. L. (2001). Weight reduction and pharmacologic treatment in obese hypertensives. *American Journal of Hypertension*, 14, 530-538.
- Maquet, P. (2001). The role of sleep in learning and memory. *Science*, 2, 1048-1052.
- Maynard, L. M., Wisemandle, W., Roche, A. F., Chumlea, W. C., Shumei S., Guo, S. S., & Siervogel, R. M. (2001). Childhood body composition in relation to body mass index. *Pediatrics*, 107, 344-350.
- Mazess, R. B., Barden, H., Bisek, J., & Hanson, J. (1990). Dual energy X-ray

- absorptiometry for total body and regional bone mineral and soft tissue composition. *American Journal of Clinical Nutrition*, 51, 1106-1112.
- McEwen, B. (2002). Sex, stress and the hippocampus: Allostasis, allostatic load and the aging process. *Neurobiology of Aging*, 23, 921-939.
- McEwen, B. (2006). Sleep deprivation as a neurobiologic and physiologic stressor: Allostasis and allostatic load. *Metabolism*, 55, S20-S23.
- Meerlo, P., Koehl, M., Van der Borght, K., & Turek, F. W. (2002). Sleep restriction alters the hypothalamic-pituitary-adrenal response to stress. *Sleep*, 14, 397-402.
- Meerlo, P., Sgoifo, A., & Suchecki, D. (2008). Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Medicine Reviews*, 12, 197-210.
- Mei, Z., Grummer-Strawn, L. M., Pietrobelli, A., Goulding, A., Goran, M. I., & Dietz, W. H. (2002). Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *American Journal of Clinical Nutrition*, 75, 978-985.
- Messerli, F. H., Nunez, B. D., Ventura, H. O., & Snyder, D. W. (1987). Overweight and sudden death. Increased ventricular ectopy in cardiopathy of obesity. *Archives of Internal Medicine*, 147, 1725-1728.
- Molgaard, H., Sorensen, K. E., & Bjerregaard, P. (1991). Circadian variation and influence of risk factors on heart rate variability in healthy subjects. *The American Journal of Cardiology*, 68, 777-784.

- Morgan, B. J., Crabtree, D. C., Puleo, D. S., Badr, M.S., Toiber, F., & Statrud, J. B. (1996). Neurocirculatory consequences of abrupt change in sleep state in humans. *Journal of Applied Physiology*, *80*, 1627-1636.
- Morrison, J. A., Barton, B. A., Biro, F. M., Daniels, S. R., & Sprecher, D. L. (1999). Overweight, fat patterning, and cardiovascular disease risk factors in black and white boys: The National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics*, *135*, 451-457.
- Morrison, J. A., Sprecher, D. L., Barton, B. A., Waclawiw, M. A., & Daniels, S. R. (1999). Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: The National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics*, *135*, 458-464.
- Mortara, A., La Rovere, M. T., Signorini, M. G., Pantaleo, P., Pinna, G., Martinelli, L., et al. (1994). Can power spectral analysis of heart rate variability identify a high risk subgroup of congestive heart failure patients with excessive sympathetic activation? A pilot study before and after heart transplantation. *British Heart Journal*, *71*, 422-430.
- Must, A., & Anderson, S. E. (2000). Establishing a standard definition for childhood overweight and obesity worldwide: International survey. *British Medical Journal*, *320*, 1240-1245.
- Must, A., & Strauss, R. S. (1999). Risks and consequences of childhood and adolescent obesity. *International Journal of Obesity Related to Metabolic Disorders*, *23*, S2-S11.

- Nagai, N., Matsumoto, T., Kita, H., & Moritani, T. (2003). Autonomic nervous system activity and the state and development of obesity in Japanese school children. *Obesity Research, 11*, 25-32.
- Nagai, N., & Moritani, T. (2004). Effect of physical activity on autonomic nervous system function in lean and obese children. *International Journal of Obesity, 28*, 27-33.
- Narkiewicz, K., & Somers, V. K. (2003). Sympathetic nerve activity in obstructive sleep apnoea. *Acta Physiologica Scandinavica, 177*, 385-390.
- National Sleep Foundation. 2000 “*Sleep in America*” Poll. Washington, DC: National Sleep Foundation; 2000.
- National Sleep Foundation. 2001 “*Sleep in America*” Poll. Washington, DC: National Sleep Foundation; 2001.
- National Sleep Foundation. 2006 “*Sleep in America*” Poll. Washington, DC: National Sleep Foundation; 2006.
- Nicklas, T. A., Baranowski, T., Cullen, K. W., & Berenson, G. (2001). Eating patterns, dietary quality and obesity. *Journal of the American College of Nutrition, 20*, 599-608.
- Nijjima, A., Rohner-Jeanrenaud, F., & Jeanrenaud, B. (1984). Role of ventromedial hypothalamus on sympathetic efferents of brown adipose tissue. *American Journal of Physiology, Regulatory, Integrative and Comparative Physiology, 247*, 650-654.

- Nilsson, P. M., Rööst, M., Engström, G., Hedblad, B., & Berglund, G. (2004). Incidence of diabetes in middle-aged men is related to sleep disturbances. *Diabetes Care, 27*, 2464-2469.
- Nolan, J., Flapan, A. D., Capewell, S., MacDonald, T. M., Neilson, J. M., & Ewing, D. J. (1992). Decreased cardiac parasympathetic activity in chronic heart failure and its relation to left ventricular function. *British Heart Journal, 67*, 482-485.
- O'Brien, L. M., & Gozal, D. (2005). Autonomic dysfunction in children with sleep-disordered breathing. *Sleep, 28*, 747-752.
- O'Brien, L. M., Mervis, C. B., Holbrook, C. R., Bruner, J. L., Klaus, C. J., Rutherford, J., et al. (2004). Neurobehavioral implications of habitual snoring in children. *Pediatrics, 114*, 44-49.
- Ogawa, Y., Kanbayashi, T., Saito, Y., Takahashi, Y., Kitajima, T., Takahashi, K., Hishikawa, Y., & Shimizu, T. (2003). Total sleep deprivation elevates blood pressure through arterial baroreflex resetting: A study with microneurographic technique. *Sleep, 26*, 986-989.
- Ogden, C. L., Kuczmarski, R. J., Flegal, K. M., Mei, Z., Guo, S., Wei, R., et al. (2002). Centers for Disease Control and Prevention 2000 Growth Charts for the United States: Improvements to the 1977 National Center for Health Statistics Version. *Pediatrics, 109*, 45-60.
- Ohida, T., Kamal, A. M. M., & Uchiyama, M., (2001). The influence of lifestyle and health status factors on sleep loss among the Japanese general population. *Sleep, 24*, 333-338.

- Ohkubo, T., Hozawa, A., Yamaguchi, J., Kikuya, M., Ohmori, K., Michimata, M. et al. (2002). Prognostic significance of the nocturnal decline in blood pressure in individual with and without high 24-h blood pressure: The Ohasama study. *Hypertension, 20*, 2183-2189.
- Orr, W. C., & Goodrich, S. (2003). An investigation of the accuracy of the Lifeshirt in comparison to standard Polysomnography. [Abstract]. *Associated Professional Sleep Societies Abstracts, 1020*.
- Owens, J. A., Spirito, A., & McGuinn, M. (2000). The children's sleep habits questionnaire (CSHQ): Psychometric properties of a survey instrument for school-aged children. *Sleep, 23*, 1043-1051.
- Padez, C., Mourao, I., Moreria, P., & Rosado, V. (2005). Prevalence and risk factors for overweight and obesity in Portuguese children. *Acta Paediatrica, 94*, 1550-1557.
- Patel, S. R., Malhotra, A., White, D. P., Gottlieb, D. J., & Hu, F. B. (2006). Association between reduced sleep and weight gain in women. *American Journal of Epidemiology, 164*, 947-954.
- Pelleymounter, M. A., Cullen, M. J., Baker, M. B., Hecht, R., Winters, D., Boone, T., et al. (1995). Effects of the obese gene product on body weight regulation in ob/ob mice. *Science, 26*, 540-543.
- Peterson, H. R., Rothschild, M., Weinberg, C.R., Fell, R. D., McLeisch, K. R., & Pfeifer, M. A. (1988). Body fat and the activity of the autonomic nervous system. *The New England Journal of Medicine, 318*, 1077-1083.
- Petretta, M., Bonaduce, D., De Filippo, E., Mureddu, G. F., Scalfi, L., Marciano, F., et al. (1995). Assessment of cardiac autonomic control by heart period variability in

- patients with early-onset familial obesity. *European Journal of Clinical Investigation*, 25, 826-832.
- Pickering, T. G. (1990). The clinical significance of diurnal blood pressure variations: Dippers and nondippers. *Circulation*, 81, 700-702.
- Pietrobelli, A., Faith, M. S., Allison, D. B., Gallagher, D., Chiumello, G., & Heymsfield, S. B. (1998). Body mass index as a measure of adiposity among children and adolescents: A validation study. *The Journal of Pediatrics*, 132, 204-210.
- Piha, S. J., Rönnemaa, T., & Koskenvuo, M. (1994). Autonomic nervous system function in identical twins discordant for obesity. *International Journal of Obesity*, 18, 547-550.
- Pomeranz, B., Macaulay, R. J., Caudill, M. A., Kutz, I., Adam, D., Gordon, D., et al. (1985). Assessment of autonomic function in humans by heart rate spectral analysis. *American Journal of Physiology- Heart and Circulatory Physiology*, 248, 151-153.
- Rabbia, F., Silke, B., Conterno, A., Grosso, T., DeVito B., Rabbone, I. et al. (2003). Assessment of cardiac autonomic modulation during adolescent obesity. *Obesity Research*, 11, 541-548.
- Radaelli, A., Bernardi, L., Valle, F., Leuzzi, S., Salvucci, F., Pedrotti, L. et al. (1994). Cardiovascular autonomic modulation in essential hypertension. Effect of tilting. *Hypertension*, 24, 556-563.
- Ravussin, E., & Tataranni, P. A. (1996). The role of altered sympathetic nervous system activity in the pathogenesis of obesity. *Proceedings of the Nutrition Society*, 55, 793-802.

- Reaven, G. M., Lithell, H., & Landsberg, L. (1996). Hypertension and associated metabolic abnormalities-the role of insulin resistance and the sympathoadrenal system. *The New England Journal of Medicine*, 334, 374-381.
- Rechtschaffen, A., & Kales, A. (1968). Proposed supplements and amendments to 'A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects', standard. *Psychiatry and Clinical Neurosciences*, 55, 305-310.
- Redline, S., Tosteson, T., Boucher, M. A., & Millman, R. P. (1991). Measurement of sleep-related breathing disturbances in epidemiologic studies. Assessment of the validity and reproducibility of portable monitoring device. *Chest*, 100, 1281-1286.
- Reilly, J. J., Armstrong, J., Dorosty, A. R., Emmett, P. M., Ness, A., Rogers, I., et al. (2005). Early life risk factors for obesity in childhood: Cohort study. *British Medical Journal*, 330, 1357-1364.
- Richter, W. O., Geiss, H. C., Aleksic, S., & Schwandt, P. (1996). Cardiac autonomic nerve function and insulin sensitivity in obese subjects. *International Journal of the Association for the Study of Obesity*, 20, 966-969.
- Robert J. J., Elsenbruch, S., & Orr, W. C. (2006). Sleep-related autonomic disturbances in symptom subgroups of women with irritable bowel syndrome. *Digestive Diseases and Sciences*, 51, 2121-2127.
- Robinson, J. P. (1985). *The validity and reliability of diaries versus alternative time use measures*. In F. T. Juster & F. P. Stafford (Eds.). *Time, goods, and well-being* (pp. 63-92). Ann Arbor, MI: Institute for Social Research.

- Rosenwasser, A. M., & Turek, F. W. (2005). Physiology of the mammalian circadian system. In M. H. Kryger, T. Roth, & W.C. Dement (Eds.), *Principles and Practices of Sleep Medicine* (pp. 351-362). Philadelphia, P.A: Elsevier.
- Rosner, B., Prineas, R., Loggie, J., & Daniels, S. R. (1998). Percentiles for body mass index in U. S. children 5-17 years of age. *Journal of Pediatrics*, *132*, 211-222.
- Rossi, M., Marti, G., Ricordi, L., Fornasari, G., Finardi, G., Fratino, P., et al. (1989). Cardiac autonomic dysfunction in obese subjects. *Clinical Science*, *76*, 567-572.
- Sadeh, A., Gruber, R., & Raviv, A. (2002). Sleep, neurobehavioral functioning and behavior problems in school-age children. *Child Development*, *73*, 405-417.
- Sadeh, A., Raviv, A., & Gruber, R. (2000). Sleep patterns and sleep disruptions in school-age children. *Developmental Psychology*, *36*, 291-301.
- Sajadieh, A., Nielson, O.W., Rasmussen, V., Hein, H. O., & Hansen, J. F. (2005). Prevalence and prognostic significance of daily life silent ischaemia in middle-aged and elderly subjects with no apparent heart disease. *European Heart Journal Advance Access*, *26*, 1402-1409.
- Savva, S. C., Tornaritis, M., Savva, M. E., Kourides, Y., Panagi, A., Silikiotou, N., et al. (2000). Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *International Journal of Obesity*, *24*, 1453-1458.
- Scholle, S., Scholle, H. C., Kemper, A., Glaser, S., Rieger, B., Kemper, G., & Zwacka, G. (2003). First night effect in children and adolescents undergoing polysomnography for sleep-disordered breathing. *Clinical Neurophysiology*, *114*, 2138-2145.

- Seicean, A., Redline, S., Seicean, S., Kirchner, H. L., Gao, Y., Sekine, M., et al. (2007). Association between short sleeping hours and overweight in adolescents: results from a US suburban high school survey. *Sleep Breath, 11*, 285-293.
- Sekine, M., Yamagami, T., Hamanshi, S., Handa, K., Saito, T., Nanri, S., et al. (2002). Parental obesity, lifestyle factors and obesity in preschool children: Results from the Toyama Birth Cohort Study. *Journal of Epidemiology, 12*, 33-39.
- Sekine, M., Yamagami, T., Handa, K., Saito, T., Nanri, S., Kawaminami, K., et al. (2002). A dose-response relationship between short sleeping hours and childhood obesity: Results from the Toyama Birth Cohort Study. *Child: Care, Health & Development, 28*, 163-170.
- Sforza, E., Pichot, V., Cervena, K., Barthelemy, J. C., & Roche, F. (2007). Cardiac variability and heart-rate increment as a marker of sleep fragmentation in patients with a sleep disorder: a preliminary study. *Sleep, 30*, 43-51.
- Sforza, E., Chapotot, F., Pigeau, R., Naitoh, P., & Buguet, A. (2004). Effects of sleep deprivation on spontaneous arousals in humans. *Sleep, 27*, 1068-1075.
- Sgoifo, A., Buwalda, B., Roos, M., Costoli, T., Merati, G., & Meerlo, P. (2006). Effects of sleep deprivation on cardiac autonomic and pituitary-adrenocortical stress reactivity in rats. *Psychoneuroendocrinology, 31*, 197-208.
- Shannon, D. C., Carley, D. W., & Benson, H. (1987). Aging of modulation of heart rate. *American Journal of Physiology. Heart and Circulatory Physiology, 253*, 874-877.
- Shek, E. W., Brands, M. W., & Hall, J. E. (1998). Chronic leptin infusion increases arterial pressure. *Hypertension, 31*, 409-414.

- Shinya, K., Toshihiro, N., Nobuatsu, N., Tetsuro, M., Muneki, T., Daisuke, Y., et al. (2005). Altered ghrelin and peptide YY responses to meals in bulimia nervosa. *Clinical Endocrinology*, *62*, 74–78.
- Sinha, M. K., Sturis, J., Ohannesian, J., Magosin, S., Stephens, T. W., Heiman, M. L., et al. (1996). Ultradian oscillations of leptin secretion in humans. *Biochemical and Biophysical Research Communications*, *8*, 228-733.
- Sloan, R. P., Huang, M-H., McCreath, H., Sidney, S., Liu, K., Williams, O. D., & Seeman, T. (2008). Cardiac autonomic control and the effects of age, race, and sex: The CARDIA study. *Autonomic Neuroscience*, *139*, 78-85.
- Sloan, R. P., Huang, M-H., Sidney, S., Liu, K., & Williams, O. D. (2005). Socioeconomic status and health: Is parasympathetic nervous system activity an intervening mechanism?. *International Journal of Epidemiology*, *34*, 309-315.
- Smaldone, A., Honig, J. C., & Byrne, M. W. (2007). Sleepless in America: Inadequate sleep and relationships to health and well-being of our nation's children. *Pediatrics*, *119*, S29-S37.
- Snell, E. K., Adam, E. K., & Duncan, G. J. (2007). Sleep and the body mass index and overweight status of children and adolescents. *Child Development*, *78*, 309-323.
- Snitker, S., Macdonald, I., Ravussin, E., & Astrup, A. (2000). The sympathetic nervous system and obesity: Role in aetiology and treatment. *Obesity Reviews*, *1*, 5-15.
- Spiegel, K., Knutson, K., Leproult, R., Tasali, E., & Van Cauter, E. (2005). Sleep loss: A novel risk factor for insulin resistance and Type 2 diabetes. *Journal of Applied Physiology*, *99*, 2008-2019.

- Spiegel, K., Leproult, R., L'Hermite-Baleriaux, M., Copinschi, G., Penev, P. D., & Van Cauter, E. (2004). Leptin levels are dependent on sleep duration: Relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. *The Journal of Clinical Endocrinology & Metabolism*, *89*, 5762-5771.
- Spiegel, K., Leproult, R., & Van Cauter, E. (1999). Impact of sleep debt on metabolic and endocrine function. *Lancet*, *354*, 1435-1439.
- Spiers, J. P., Silke, B., McDermott, U., Shanks, R. G., & Harron, W. G. (1993). Time and frequency domain assessment of heart rate variability: A theoretical and clinical appreciation. *Clinical Autonomic Research*, *3*, 145-158.
- Spraul, M., Ravussin, E., Fontvieille, A. M., Rising, R., Larson, D. E., & Anderson, E. A. (1993). Reduced sympathetic nervous activity. A potential mechanism predisposing to body weight gain. *Journal of Clinical Investigation*, *92*, 1730-1735.
- Steiger, A. (2002). Sleep and the hypothalamo-pituitary-adrenocortical system. *Sleep Medicine Reviews*, *6*, 125-138.
- Stein, P. K., & Kleiger, P. K. (1999). Insights from the study of heart rate variability. *Annual Review of Medicine*, *50*, 249-261.
- Stein, M. A., Mendelsohn, J., Obermeyer, W. H., Amromin, J., & Benca, R. (2001). Sleep and behavior problems in school-aged children. *Pediatrics*, *107*, e60.
- Stewart, J., Weldon, A., Arlievsky, N., Li, K., & Munoz, J. (1998). Neurally mediated hypotension and autonomic dysfunction measured by heart rate variability during head-up tilt testing in children with chronic fatigue syndrome. *Clinical Autonomic Research*, *8*, 221-230.

- Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature*, *437*, 1272- 1278.
- Stickgold, R., Hobson, J. A., Fosse, R., & Fosse, M. (2001). Sleep, learning, and dreams: Off-line memory reprocessing. *Science*, *294*, 1052-1057.
- Sunter, D., O'Shea, D., & Bloom, S.R. (1997). Leptin and recent advances in appetite regulation. *Tumour Marker Update*, *9*, 1-4.
- Tafti, M., & Franken, P. (2002). Genetic dissection of sleep. *Journal of Applied Physiology*, *92*, 1339-1347.
- Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *Plos Medicine*, *1*, 210-217.
- Tan, X., Campbell, I. G., Palagini, L., & Feinberg, I. (2000). High internight reliability of computer-measured NREM delta, sigma, and beta: Biological implications, *Biological Psychiatry*, *48*, 1010–1019.
- Tasali, E., Leproult, R., Ehrmann, D. A., & Van Cauter, E. (2008). Slow-wave sleep and the risk of type 2 diabetes in humans. *Proceedings of the National Academy of Sciences*, *105*, 1044-1049.
- Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology. (1996). Heart Rate Variability: Standards of Measurement, Physiological Interpretation, and Clinical Use. *Circulation*, *93*, 1043-1065.
- Tataranni, P. A., Young, B. A., Bogardus, C., & Ravussin, E. (1997). A low sympathoadrenal activity is associated with body weight gain and development of central obesity in Pima Indian men. *Obesity Research*, *5*, 341-347.
- Taveras, E. M., Rifas-Shiman, S. L., Oken, E., Gunderson, E. P., & Gillman, M. W.

- (2008). Short sleep duration in infancy and risk of childhood overweight. *Archives of Pediatrics and Adolescent Medicine*, 162, 305-311.
- Taylor, R. W., Jones, I. E., Williams, S. M., & Goulding, A. (2000). Evaluation of waist circumference, waist-to-hip ratio, and the conicity index as screening tools for high trunk fat mass, as measured by dual-energy X-ray absorptiometry, in children aged 3–19. *American Journal of Clinical Nutrition*, 72, 490-495.
- Taylor, S. J. C, Whincup, P. H., Hindmarsh, P. C., Lampe, F., Odoki, K., & Cook, D. G. (2001). Performance of a new pubertal self-assessment questionnaire: A preliminary study. *Paediatric and Perinatal Epidemiology*, 15, 88-94.
- Tentolouris, N., Tsigos, C., Perea, D., Koukou, E., Kyriaki, D., Kitsou, E., et al. (2003). Differential effects of high-fat and high-carbohydrate isoenergetic meals on cardiac autonomic nervous system activity in lean and obese women. *Metabolism*, 52, 1426-1432.
- Thompson, S. R., Ackermann, U., & Horner, R. L. (2001). Sleep as a teaching tool for integrating respiratory physiology and motor control. *Advances in Physiology Education*, 25, 29-44.
- Tochikubo, O., Ikeda, A., Miyajima, E., & Ishii, M. (1996). Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. *Hypertension*, 27, 1318-1324.
- Toscani, L., Gangemi, P. F., Parigi, A., Silipo, R., Raghianti, P., Sirabella, et al. (1996). Human heart rate variability and sleep stages. *The Italian Journal of Neurological Sciences*, 17, 437-439.
- Tremblay, M. S., Katzmarzyk, P. T., & Willms, J. D. (2002). Temporal trends in

- overweight and obesity in Canada, 1981-1996. *International Journal of Obesity*, 26, 538-543.
- Trinder, J., Kleiman, J., Carrington, M., Smith, S., Breen, S., Tan, N. et al. (2001). Autonomic activity during human sleep as a function of time and sleep stage. *Journal of Sleep Research*, 10, 253-264.
- Tritos, N. A., & Mantzoros, C. S. (1997). Leptin: Its role in obesity and beyond. *Diabetologia*, 40, 1371-1379.
- Tsuji, H., Larson, M. G., Venditti, F. J. Jr., Manders, E. S., Evans, J. C., Feldman, C. L., & Levy, D. (1996). Sleep and cardiovascular disease events. The Framingham Heart Study. *Circulation*, 94, 2850-2855.
- Turek, F. W., Joshu, C., Kohsaka, A., Lin, E., Ivanova, G., McDearmon, E., et al. (2005). Obesity and metabolic syndrome in circadian clock mutant mice. *Science*, 308, 1043-1045.
- van Eekelen A. P. J., Varkevisser M., & Kerkhof, G. A. (2003). Cardiac autonomic activity during human sleep: Analysis of sleep stages and sleep cycles. *Biological Rhythm Research*, 34, 493-502.
- van Dongen, H. P. A., Maislin, G., Mullington, J. M., & Dinges, D. F. (2003). The cumulative cost of additional wakefulness: Dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep*, 26, 117-126.
- Vanoli, E., Adamson, P. B., Ba-Lin, M. P. H., Pinna, G. D., Lazzara, R., & Orr, W. C. (1995). Heart rate variability during specific sleep stages a comparison of healthy subjects with patients after myocardial infarction. *Circulation*, 91, 1918-1922.

- Vaz, M., Jennings, G., Turner, A., Cox, H., Lambert, G., & Esler, M. (1997). Regional sympathetic nervous activity and oxygen consumption in obese normotensive human subjects. *Circulation*, *96*, 3423-3429.
- Verdecchia, P., Schillaci, G., Guerrieri, M., Gatteschi, C., Benemio, G., Boldrini, F., et al. (1990). Circadian blood pressure changes and left ventricular hypertrophy in essential hypertension. *Circulation*, *81*, 528-536.
- Vgontzas, A. N., Zoumakis, M., Bixler, E. O., Lin, H.-M., Prolo, P., Vela-Bueno, A., et al. (2003). Impaired nighttime sleep in healthy old versus young adults is associated with elevated plasma interleukin-6 and cortisol levels: Physiologic and therapeutic implications. *The Journal of Clinical Endocrinology & Metabolism*, *88*, 2087-2095.
- von Kries, R., Toschke, A. M., Wurmser, H., Sauerwald, T., & Koletzko, B. (2002). Reduced risk for overweight and obesity in 5- and 6-y-old children by duration of sleep- a cross-sectional study. *International Journal of Obesity*, *26*, 710-716.
- Wallin, B. G., Esler, M., Dorward, P., Eisenhofer, G., Ferrier, C., Westerman, R., & Jennings, G. (1992). Simultaneous measurements of cardiac noradrenaline spillover and sympathetic outflow to skeletal muscle in humans. *The Journal of Physiology*, *453*, 45-58.
- Wang, G., & Dietz, W. (2002). Economic burden of obesity in youths aged 6 to 17 years: 1979-1999. *Pediatrics*, *109*, 1-6.
- Wang, Y., & Lobstein, T. (2006). Worldwide trends in childhood overweight and obesity. *International Journal of Pediatric Obesity*, *1*, 11-25.
- Weaver, D. R. (1998). The suprachiasmatic nucleus: A 25-year retrospective. *Journal of*

- Biological Rhythms*, 13, 100-112.
- Wells, J. C. K., & Fewtrell, M. S. (2006). Measuring body composition. *Archives of Disease in Childhood*, 91, 612-617.
- Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997). Predicting obesity in young adulthood from childhood and parental obesity. *New England Journal of Medicine*, 337, 869-873.
- Willms, D. J., Tremblay, M. S., & Katzmarzyk, P. T. (2003). Geographic and demographic variation in the prevalence of overweight Canadian children. *Obesity Research*, 11, 668-673.
- Wolf, P. A., Dawber, T. R., Thomas, Jr. H. E., & Kannel, W. B. (1978). Epidemiologic assessment of chronic atrial fibrillation and risk of stroke. The Framingham Study. *Neurology*, 28, 973-977.
- Wolfson, A. (1996). Sleeping patterns of children and adolescents: Developmental trends, disruptions, and adaptations. *Child and Adolescent Psychiatric Clinics of North America*, 5, 549-568.
- Wolk, R., Gami, A., Garcia-Touchard, A., & Somers, V. (2005). Sleep and cardiovascular disease. *Current Problems in Cardiology*, 30, 625-662.
- Wren, A. M., Seal, L. J., Cohen, M. A., Brynes, A. E., Frost, G. S., Murphy, K. G., et al. (2001). Ghrelin enhances appetite and increases food intake in humans. *Journal of Clinical Endocrinology and Metabolism*, 86, 5992.
- Xia, R., Odemuyiwa, O., Gill, J., Malik, M., & Camm, A. J. (1993). Influence of recognition errors of computerised analysis of 24-hour electrocardiograms on the

measurement of spectral components of heart rate variability. *International Journal of Bio-medical Computing*, 32, 223-235.

Young, J. B., & Landsberg, L. (1977). Suppression of sympathetic nervous system during fasting. *Science*, 196, 1473-1475.

Young, J. B., Saville, M. E., Rothwell, N. J., Stock, M. J., & Landsberg, L. (1982). Effect of diet and cold exposure on norepinephrine turnover in brown adipose tissue of rat. *Journal of Clinical Investigation*, 69, 1061-1071.

Table 1.

Participant Characteristics (N=122)

	<i>N (Mean)</i>	<i>% (SD)</i>
Children		
Age (years)	(12.81)	(2.19)
Sex		
Female	62	50.8%
Male	60	49.2%
Completed Sleep Study with Sufficient Sleep Data		
Yes	57	47%
No	65	53%
Parents		
Age (years)	(45.44)	(7.31)
Sex		
Female	71	78.9%
Male	19	21.1%
Marital Status		
Single	11	12.4%
Married	61	68.5%
Seperated	2	2.2%
Divorced	12	13.5%
Widowed	3	3.4%

Table 2.

Means and Standard Deviations for Obesity Measures in Youth

	<u>Girls</u>		<u>Boys</u>		<u>Overall</u>	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
Waist (cm)	72.25	10.72	74.01	8.16	73.14	9.52
Hip (cm)	92.17	11.63	90.64	9.69	91.42	10.71
Weight (lbs)	115.82	31.50	121.83	29.66	118.77	30.63
BMI (kg/m ²)	21.33	4.66	20.66	3.13	20.99	3.98
Percent Body Fat (%)	26.72	8.51	17.15	8.31	21.97	9.66
BMI Percentile (%ile)	63.03	25.58	64.85	24.75	63.92	25.09

Table 3.

Self-Report Pubertal Status - Girls

	<i>N (Mean)</i>	<i>% (SD)</i>
Tanner Stage (Breast)	(3.25)	(1.46)
Stage 1	7	11.5%
Stage 2	3	4.9%
Stage 3	13	21.3%
Stage 4	24	39.3%
Stage 5	10	16.4%
Not sure	4	6.6%
Tanner Stage (Hair)	(3.27)	(1.72)
Stage 1	9	15%
Stage 2	4	6.7%
Stage 3	9	15%
Stage 4	13	21.7%
Stage 5	20	33.3%
Not sure	5	8.3%
Have you started to have hair growing in armpits?		
Yes	49	80.3%
No	12	19.7%
Missing	1	0.8%
Have you begun menstrual periods yet?		
Yes	41	66.1%
No	21	33.9%
Age of first menstrual period (years)	(11.99)	(1.03)

Note. $N=62$.

Table 4.

Self-Report Pubertal Status - Boys

	<i>N (Mean)</i>	<i>% (SD)</i>
Tanner Stage (Penis)	(2.83)	(1.62)
Stage 1	3	5%
Stage 2	14	23.3%
Stage 3	13	21.7%
Stage 4	10	16.7%
Stage 5	12	20%
Not sure	8	13.3%
Tanner Stage (Hair)	(3.02)	(1.52)
Stage 1	8	13.3%
Stage 2	9	15%
Stage 3	12	20%
Stage 4	16	26.7%
Stage 5	11	18.3%
Not sure	4	6.7%
Has your voice changed?		
No, it's the same	4	6.7%
Yes, occasionally	26	43.3%
Yes, it has now changed	18	30%
Not sure	12	20%
Have you started to have hair growing in armpits?		
Yes	38	63.3%
No	22	36.7%

Note. $N=60$.

Table 5.

Means and Standard Deviations for Subjective Sleep Measures (n=57)

	<i>Mean</i>	<i>SD</i>
Child-Report		
Overall Sleep Quality	6.56	2.01
Sleep Study Sleep Quality	6.66	2.32
School Sleep Duration	8 h 56 min	57 min
Weekend Sleep duration	10 h	1 h 13 min
Daytime Sleepiness	14.29	4.82
Parent-Report		
School Sleep Duration	9 h 07 min	56 min
Weekend Sleep duration	10 h 09 min	1 h 09 min
CSHQ Sleep Disturbance	41.19	5.23
CSHQ Bedtime Resistance	6.85	1.32
CSHQ Sleep Onset Delay	1.17	0.50
CSHQ Sleep Anxiousness	4.37	0.64
CSHQ Night Awakenings	3.23	0.56
CSHQ Parasomnias	7.46	0.82
CSHQ Daytime Sleepiness	12.77	3.31
CSHQ Sleep-disordered Breathing	3.17	0.46

Note. CSHQ = Children's Sleep Habits Questionnaire.

Table 6.

Means and Standard Deviations for Objective Sleep Study Measures (n=57)

	<i>Mean</i>	<i>SD</i>
Sleep Period Time (min)	517.03	77.75
Total Sleep Time (min)	467.28	100.91
Fragmented Sleep ^a	50.91	30.22
Light NREM Time (%) ^b	23.40	8.17
Slow Wave Sleep Time (%) ^c	20.19	8.57
REM Time (%)	45.07	17.69
Sleep Efficiency (%) ^d	89.37	11.39
Sleep Latency (min)	1.97	2.83
Intermittent Time Awake	44.87	34.16
Micro Arousal Index	9.09	6.28
Sleep Apnea Index	0.05	0.36

Note. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4.
^dTotal Sleep Time/Sleep Period Time.

Table 7.

Means and Standard Deviations for HRV Measures during Light NREM, Slow Wave Sleep, and REM Sleep (n = 57)

	<u>Light NREM</u>		<u>Slow Wave Sleep</u>		<u>REM</u>	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
LF	727.04	509.05	547.12	503.90	822.39	657.22
HF	2441.63	1871.19	2260.79	1878.48	2950.35	2540.29
LF/HF Ratio	0.37	0.16	0.28	0.13	0.37	0.17
LF <i>nu</i>	0.21	0.07	0.17	0.07	0.21	0.07
HF <i>nu</i>	0.68	0.08	0.73	0.08	0.68	0.08
SDNN	80.08	29.12	69.05	26.30	84.44	33.64
rMSSD	77.70	37.29	76.30	38.21	85.02	45.39
pNN50	0.39	0.22	0.42	0.24302	0.42	0.24

Note. LF = low frequency (ms^2); HF = high frequency (ms^2); LF*nu* = low frequency normalized unit; HF*nu* = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms^2); rMSSD = root mean square difference among successive R-R normal intervals (ms^2); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart.

Table 8.

HRV Measures in Healthy-Weight, Overweight, and Obese Youth during Sleep (n=57)

	Healthy-Weight (n=40)		Overweight (n=11)		Obese (n=5)	
	Mean	SD	Mean	SD	Mean	SD
BMI	19.42	2.14	23.86	1.69	28.86	6.49
Light NREM						
LF	811.58	538.07	450.19	304.55	620.94	517.14
HF	2853.12	2019.10	1337.40	965.99	1531.32	917.10
LF/HF Ratio	0.37	0.17	0.39	0.16	.038	0.11
LFnu	0.21	0.07	0.22	0.05	0.21	0.05
HFnu	0.68	0.09	0.68	0.07	0.67	0.07
SDNN	86.68	29.86	61.02	20.71	67.71	21.59
rMSSD	86.29	38.90	53.55	26.39	60.47	15.63
pNN50	0.43	0.22	0.24	0.20	0.32	0.09
Slow Wave Sleep						
LF	623.65	542.17	341.75	359.84	274.19	135.85
HF	2700.54	2021.15	1163.21	805.90	943.54	497.54
LF/HF Ratio	0.28	0.14	0.30	0.13	0.25	0.79
LFnu	0.17	0.07	0.18	0.06	0.15	0.03
HFnu	0.73	0.09	0.72	0.08	0.73	0.10
SDNN	75.87	26.12	50.72	18.88	49.63	10.71
rMSSD	85.29	39.65	52.66	25.93	51.23	11.72
pNN50	0.47	0.24	0.28	0.24	0.28	0.06
REM						
LF	940.72	710.77	479.01	404.47	567.44	376.18
HF	3437.33	2744.83	1803.23	1712.59	1530.70	1014.66
LF/HF Ratio	0.37	0.18	0.36	0.18	0.39	0.14
LFnu	0.21	0.07	0.20	0.06	0.22	0.45
HFnu	0.68	0.08	0.69	0.08	0.67	0.07
SDNN	92.27	34.14	64.39	27.49	64.31	18.84
rMSSD	94.46	47.44	62.41	36.74	58.69	19.10
pNN50	0.46	0.23	0.30	0.26	0.30	0.09

Table 9.

Inter-correlations among Obesity Measures

Subscales	1	2	3	4	5	6
Participants ($n=118$)						
1. Waist (cm)	-	.82**	.87**	.92**	.62**	.74**
2. Hip (cm)		-	.87**	.86**	.70**	.71**
3. Weight (lbs)			-	.87**	.52**	.74**
4. BMI (kg/m^2)				-	.76**	.81**
5. Percent Body Fat (%)					-	.64**
6. BMI Percentile (%ile)						-

Note. Partial correlations while controlling for age. ** $p < .001$ level (2-tailed).

Table 10.

Inter-correlations among Child- and Parent-Report Subjective Sleep Measures.

	1	2	3	4	5	6	7	8
Participants (<i>n</i> =57)								
1. Overall Sleep Quality ^a	-	.26*	.28*	.00	-.50**	.22	-.33*	-.30*
2. Sleep Study Sleep Quality ^a		-	-.18	.00	-.11	.10	.02	-.24*
3. School Sleep Duration ^a			-	-.08	.25	.35*	-.14	.01
4. Weekend Sleep Duration ^a				-	.05	.13	.32*	-.16
5. Daytime Sleepiness ^a					-	-.06	.31*	.33*
6. School Sleep Duration ^b						-	.23	-.23
7. Weekend Sleep Duration ^b							-	.19
8. CSHQ Sleep Disturbance ^b								-

Note. CSHQ = Children's Sleep Habits Questionnaire. ^aBased on child-report. ^bBased on parent-report.

Table 11.

Inter-correlations among Objective Sleep Measures

Subscales	1	2	3	4	5	6	7	8	9	10
	Participants (<i>n</i> =57)									
1. Sleep Period Time (min)	-	.74**	.19	.14	.22	-.11	-.07	-.07	-.09	-.08
2. Total Sleep Time (min)		-	-.07	-.14	.30*	.34*	.63**	-.07	-.28*	-.33*
3. Fragmented Sleep ^a			-	.61**	-.11	-.52**	-.46*	-.08	.85**	.68**
4. Light NREM Time (%) ^b				-	.21	-.80**	-.36**	-.08	.51**	.25
5. Slow Wave Time (%) ^c					-	-.39**	.28*	-.02	.24	.40**
6. REM Time (%)						-	.68**	.02	-.53**	-.25
7. Sleep Efficiency (%) ^d							-	.02	-.63**	-.51**
8. Sleep Latency (min)								-	.07	-.15
9. Intermittent Time Awake									-	.66**
10. Micro Arousal Index										-

Note. Partial correlations while controlling for age. . ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. **p*<.05 level (2-tailed). ** *p*<.01 level (2-tailed).

Table 12.

Partial Correlations between Objective Sleep Measures and Child-Report Subjective Sleep Measures

	Overall Sleep Quality	Sleep Study Sleep Quality	School Sleep Duration	Weekend Sleep Duration	Daytime Sleepiness
Participants ($n = 57$)					
Sleep Period Time (min)	.01	.11	-.02	.18	-.03
Total Sleep Time (min)	.06	.10	-.19	.15	-.21
Fragmented Sleep ^a	.03	-.06	.18	.14	.04
Light NREM (%) Time ^b	-.15	-.08	.25*	-.01	.23
Slow Wave Sleep (%) Time ^c	-.17	-.03	-.10	-.07	-.15
REM Sleep Time	.19	.10	-.28*	.04	-.20
Sleep Efficiency (%) ^d	.06	.03	-.31*	.09	-.24
Sleep Latency (min)	.13	-.29*	.07	.09	-.15
Intermittent Time Awake	-.01	-.15	.22	.16	.13
Micro Arousal Index	.04	-.06	.24	-.07	.04

Note. Partial correlations while controlling for age. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. * $p < .05$ level (2-tailed).

Table 13.

Partial Correlations between Objective Sleep Measures and Parent-Report Subjective Sleep Measures

	School Sleep Duration	Weekend Sleep Duration	CSHQ Sleep Disturbance	Bedtime Resistance	Sleep Onset Delay	Sleep Anxious	Night Awakening	Para- somnias	Sleep- disordered Breathing
Participants (<i>n</i> = 57)									
Sleep Period Time (min)	.12	.17	.01	.10	.30*	.00	-.04	-.10	.24
Total Sleep Time (min)	.04	.15	.07	.07	.20	-.03	.05	.03	.26*
Fragmented Sleep ^a	.21	.07	-.16	-.03	.12	-.12	.27*	-.21	-.10
Light NREM (%) Time ^b	.30*	.02	-.10	-.22	-.10	-.28*	-.12	-.03	-.14
Slow Wave Sleep (%) Time ^c	.08	.02	-.12	-.20	-.05	-.22	.02	.02	-.12
REM Sleep Time	-.20	-.07	.10	.19	.09	.22	.16	.13	.24
Sleep Efficiency (%) ^d	-.04	-.08	-.01	-.01	.01	-.01	.16	.19	.16
Sleep Latency (min)	.02	-.05	-.21	.08	-.05	-.02	-.04	.06	-.21
Intermittent Time Awake	.21	.16	-.11	.05	.11	-.02	-.14	-.19	-.17
Micro Arousal Index	.10	-.10	.10	.22	.17	-.01	-.21	-.16	.03

Note. Partial correlations while controlling for age. . CSHQ = Children's Sleep Habits Questionnaire. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. **p* < .05 level (2-tailed).

Table 14.

Inter-correlations among HRV Measures

	1	2	3	4	5	6	7	8
Participants ($n=57$)								
1. LF	-	.65**	.26*	.32*	-.23	.74**	.59**	.41**
2. HF		-	-.26*	-.17	.21	.92**	.93**	.78**
3. LF/HF Ratio			-	.97**	-.95**	-.30*	-.38**	-.53**
4. LF nu				-	.94**	-.22	-.28*	-.43**
5. HF nu					-	.26*	.29*	.43**
6. SDNN						-	.94**	.83*
7. rMSSD							-	.91**
8. pNN50								-

Note. LF = low frequency (ms^2); HF = high frequency (ms^2); LF nu = low frequency normalized unit; HF nu = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms^2); rMSSD = root mean square difference among successive R-R normal intervals (ms^2); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart.

* $p < .05$ level (2-tailed). ** $p < .001$ level (2-tailed).

Table 15.

Partial Correlations between Subjective Sleep Measures and Obesity Measures

	Waist (cm)	Hip (cm)	Weight (lbs)	BMI (kg/m ²)	Percent Body Fat (%)	BMI percentile (%ile)
Participants (<i>n</i> = 57)						
Child-Report						
Overall Sleep Quality	-.31*	-.33*	-.39*	-.37*	-.28*	-.27*
Sleep Study Sleep Quality	-.19	-.18	-.22	-.18	-.07	-.06
School Sleep Duration	-.05	-.19	-.10	-.20	-.18	-.09
Weekend Sleep Duration	-.02	.10	.08	.05	.20	-.08
Daytime Sleepiness	.15	.23	.16	.18	.24	.19
Parent-Report						
School Sleep Duration	-.18	-.18	-.17	-.22	-.05	-.08
Weekend Sleep Duration	.12	.12	.14	.20	.20	.04
CSHQ Sleep Disturbance	.36*	.42*	.36*	.42*	.34*	.43*
CSHQ Bedtime Resistance	.17	.20	.19	.23	.34*	.27*
CSHQ Sleep Onset Delay	.03	.04	-.03	.03	.23	.16
CSHQ Sleep Anxiousness	.04	.00	-.04	.09	.20	.29*
CSHQ Night Awakenings	.03	-.01	-.00	-.02	.02	-.01
CSHQ Parasomnias	.14	.19	.13	.21	.06	.20
CSHQ Daytime Sleepiness	.30*	.32*	.33*	.31*	.17	.37*
CSHQ Sleep-disordered Breathing	.13	.18	.15	.19	.25	.06

Note. Partial correlations while controlling for age. CSHQ = Children's Sleep Habits Questionnaire. **p* < .05 level (2-tailed).

Table 16.

Partial Correlations between Objective Sleep Measures and Obesity Measures

	Waist (cm)	Hip (cm)	Weight (lbs)	BMI (kg/m ²)	Percent Body Fat (%)	BMI percentile (%ile)
Participants (<i>n</i> = 57)						
Sleep Period Time	-.25	-.16	-.21	-.25	-.05	-.17
Total Sleep Time	-.15	-.02	-.07	-.14	.03	-.16
Fragmented Sleep ^a	-.29*	-.18	-.21	-.27*	-.08	-.20
Light NREM (%) Time ^b	-.22	-.21	-.16	-.19	-.16	-.13
Slow Wave Sleep (%) Time ^c	-.29*	-.27*	-.27*	-.26*	-.27*	-.24
REM Sleep Time	.29*	.32*	.29*	.29*	.27*	.19
Sleep Efficiency (%) ^d	.08	.14	.13	.11	.10	.02
Sleep Latency (min)	-.02	-.09	-.10	-.06	-.03	-.13
Intermittent Time Awake	-.16	-.10	-.14	-.14	.05	-.05
Micro Arousal Index	.07	.13	.08	.07	.16	.12

Note. Partial correlations while controlling for age. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. **p*<.05 level (2-tailed).

Table 17.

Partial Correlations between Objective Sleep Measures and HRV during Light NREM Sleep

	LF	HF	LF/HF Ratio	LFnu	HFnu	SDNN	rMSSD	pNN50
Participants (n = 57)								
Sleep Period Time (min)	.04	-.08	.04	-.00	.09	-.01	-.09	-.03
Total Sleep Time (min)	-.04	-.13	.12	.06	-.02	-.06	-.09	.03
Fragmented Sleep ^a	.27*	.15	-.13	-.11	.10	.23	.17	.13
Light NREM (%) Time ^b	.10	.09	-.00	.03	-.02	.13	.06	.00
Slow Wave Sleep (%) Time ^c	-.00	-.14	.35*	.37*	-.30*	-.10	-.16	-.12
REM Sleep Time	-.17	-.12	-.05	-.10	.05	-.13	-.06	.05
Sleep Efficiency (%) ^d	-.20	-.23	.18	.14	-.17	-.19	-.16	-.06
Sleep Latency (min)	-.05	.06	.09	.16	-.15	.02	.08	.13
Intermittent Time Awake	.15	.13	-.21	-.19	.20	.13	.12	.10
Micro Arousal Index	.17	.01	-.03	-.04	.01	.08	.01	-.05

Note. Partial correlations while controlling for age. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. LF = low frequency (ms²); HF = high frequency (ms²); LFnu = low frequency normalized unit; HFnu = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms²); rMSSD = root mean square difference among successive R-R normal intervals (ms²); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart. **p* < .05 level (2-tailed).

Table 18.

Partial Correlations between Objective Sleep Measures and HRV during Slow Wave Sleep

	LF	HF	LF/HF Ratio	LFnu	HFnu	SDNN	rMSSD	pNN50
Participants (<i>n</i> =57)								
Sleep Period Time (min)	-.08	-.09	-.09	-.11	.19	-.01	-.10	-.11
Total Sleep Time (min)	-.20	-.17	-.02	-.05	.08	-.10	-.13	-.06
Fragmented Sleep ^a	.04	.09	-.28*	-.25*	.27*	.15	.17	.13
Light NREM (%) Time ^b	.03	.08	-.00	.01	.00	.09	.09	-.10
Slow Wave Sleep (%) Time ^c	-.05	-.19	.24	.27	-.18	-.16	-.19	-.15
REM Sleep Time	-.18	-.13	-.04	-.08	.00	-.12	-.08	.01
Sleep Efficiency (%) ^d	-.29*	-.28*	.11	.09	-.13	-.26*	-.20	-.08
Sleep Latency (min)	-.09	.01	.01	.08	-.06	-.04	.03	.14
Intermittent Time Awake	-.00	.12	-.34*	-.32*	.33*	.14	.15	.14
Micro Arousal Index	.01	-.02	-.20	-.22	.22	.00	.01	-.02

Note. Partial correlations while controlling for age. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. LF = low frequency (ms²); HF = high frequency (ms²); LFnu = low frequency normalized unit; HFnu = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms²); rMSSD = root mean square difference among successive R-R normal intervals (ms²); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart. **p*<.05 level (2-tailed).

Table 19.

Partial Correlations between Objective Sleep Measures and HRV during REM Sleep

	LF	HF	LF/HF Ratio	LFnu	HFnu	SDNN	rMSSD	pNN50
Participants (<i>n</i> = 57)								
Sleep Period Time (min)	.10	-.03	.03	.08	-.02	-.01	-.04	-.08
Total Sleep Time (min)	.04	-.12	.11	.14	-.11	-.02	-.06	-.05
Fragmented Sleep ^a	.18	.20	-.15	-.14	.12	.16	.21	.10
Light NREM (%) Time ^b	.05	.17	.03	.03	-.03	.12	.12	-.01
Slow Wave Sleep (%) Time ^c	.05	-.17	.40*	.45*	-.41*	-.03	-.18	-.19
REM Sleep Time	-.10	-.14	-.07	-.10	.07	-.12	-.08	.01
Sleep Efficiency (%) ^d	-.10	-.23	.20	.19	-.20	-.12	-.16	-.11
Sleep Latency (min)	-.06	.03	.10	.10	-.10	.09	.06	.14
Intermittent Time Awake	.09	.18	-.20	-.20	.20	.10	.15	.08
Micro Arousal Index	.01	.01	-.13	-.13	.11	-.06	.00	-.05

Note. Partial correlations while controlling for age. ^aNumber of awakenings lasting at least 15 sec. ^bStages 1 and 2. ^cStages 3 and 4. ^dTotal Sleep Time/Sleep Period Time. LF = low frequency (ms²); HF = high frequency (ms²); LFnu = low frequency normalized unit; HFnu = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms²); rMSSD = root mean square difference among successive R-R normal intervals (ms²); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart. **p* < .05 level (2-tailed).

Table 20.

Partial Correlations between Obesity Measures and HRV Measures during Light NREM Sleep

	Waist (cm)	Hip (cm)	Weight (lbs)	BMI (kg/m ²)	Percent Body Fat (%)	BMI percentile (%ile)
Participants (<i>n</i> = 57)						
LF	-.32*	-.30*	-.28*	-.34*	-.32*	-.37*
HF	-.30*	-.33*	-.32*	-.34*	-.28*	-.32*
LF/HF Ratio	-.03	.04	.05	-.03	-.06	-.07
LF ν	-.02	-.02	.02	-.08	-.12	-.13
HF ν	-.02	-.04	-.08	.04	.09	.12
SDNN	-.33*	-.32*	-.28*	-.36*	-.33*	-.34*
rMSSD	-.33*	-.33*	-.30*	-.34*	-.29*	-.34*
pNN50	-.29*	-.27*	-.28*	-.26*	-.13*	-.25*

Note. Partial correlations while controlling for age. LF = low frequency (ms²); HF = high frequency (ms²); LF ν = low frequency normalized unit; HF ν = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms²); rMSSD = root mean square difference among successive R-R normal intervals (ms²); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart. **p* < .05 level (2-tailed).

Table 21.

Partial Correlations between Obesity Measures and HRV Measures during Slow Wave Sleep

	Waist (cm)	Hip (cm)	Weight (lbs)	BMI (kg/m ²)	Percent Body Fat (%)	BMI percentile (%ile)
Participants (<i>n</i> = 57)						
LF	-.21	-.27*	-.18	-.29*	-.41*	-.31*
HF	-.31*	-.34*	-.32*	-.35*	-.32*	-.31*
LF/HF Ratio	.06	.05	.12	-.03	-.18	-.11
LF ν	-.01	-.02	.05	-.09	-.23	-.18
HF ν	-.09	-.10	-.18	-.03	-.14	.14
SDNN	-.37*	-.39*	-.35*	-.40*	-.39*	-.36*
rMSSD	-.36*	-.35*	-.34*	-.36*	-.29*	-.34*
pNN50	-.32*	-.33*	-.32*	-.29*	-.19	-.26*

Note. Partial correlations while controlling for age. LF = low frequency (ms²); HF = high frequency (ms²); LF ν = low frequency normalized unit; HF ν = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms²); rMSSD = root mean square difference among successive R-R normal intervals (ms²); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart. **p* < .05 level (2-tailed).

Table 22.

Partial Correlations between Obesity Measures and HRV Measures during REM Sleep

	Waist (cm)	Hip (cm)	Weight (lbs)	BMI (kg/m ²)	Percent Body Fat (%)	BMI percentile (%ile)
Participants (<i>n</i> = 57)						
LF	-.31*	-.31*	-.28*	-.34*	-.34*	-.37*
HF	-.27*	-.30*	-.27*	-.31*	-.28*	-.28*
LF/HF Ratio	.04	.06	.08	-.01	-.06	-.14
LF nu	-.01	.01	.04	-.06	-.11	-.20
HF nu	-.01	-.05	-.07	.03	.07	.19
SDNN	-.34*	-.37*	-.31*	-.38*	-.37*	-.36*
rMSSD	-.31*	-.32*	-.28*	-.32*	-.28*	-.29*
pNN50	-.25*	-.31*	-.25*	-.25*	-.22	-.19

Note. Partial correlations while controlling for age. LF = low frequency (ms²); HF = high frequency (ms²); LF nu = low frequency normalized unit; HF nu = high frequency normalized unit; SDNN = standard deviation of normal to normal R-R intervals (ms²); rMSSD = root mean square difference among successive R-R normal intervals (ms²); pNN50 (%) = percentage of differences between successive R-R intervals that are greater than 50 milliseconds (ms) apart. **p* < .05 level (2-tailed).

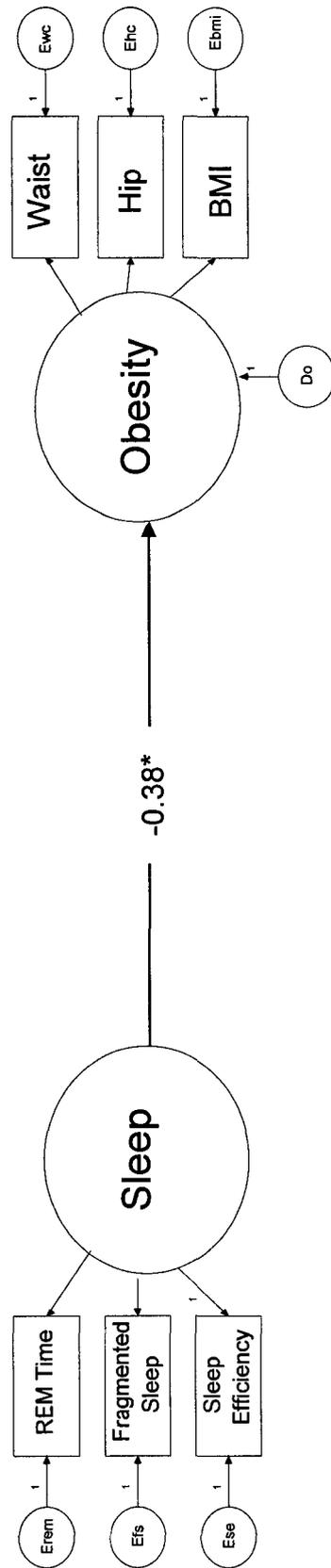


Figure 1. Structural equation base model: Influence of objective sleep measures on obesity. * $t > 1.96$ (2-tailed).

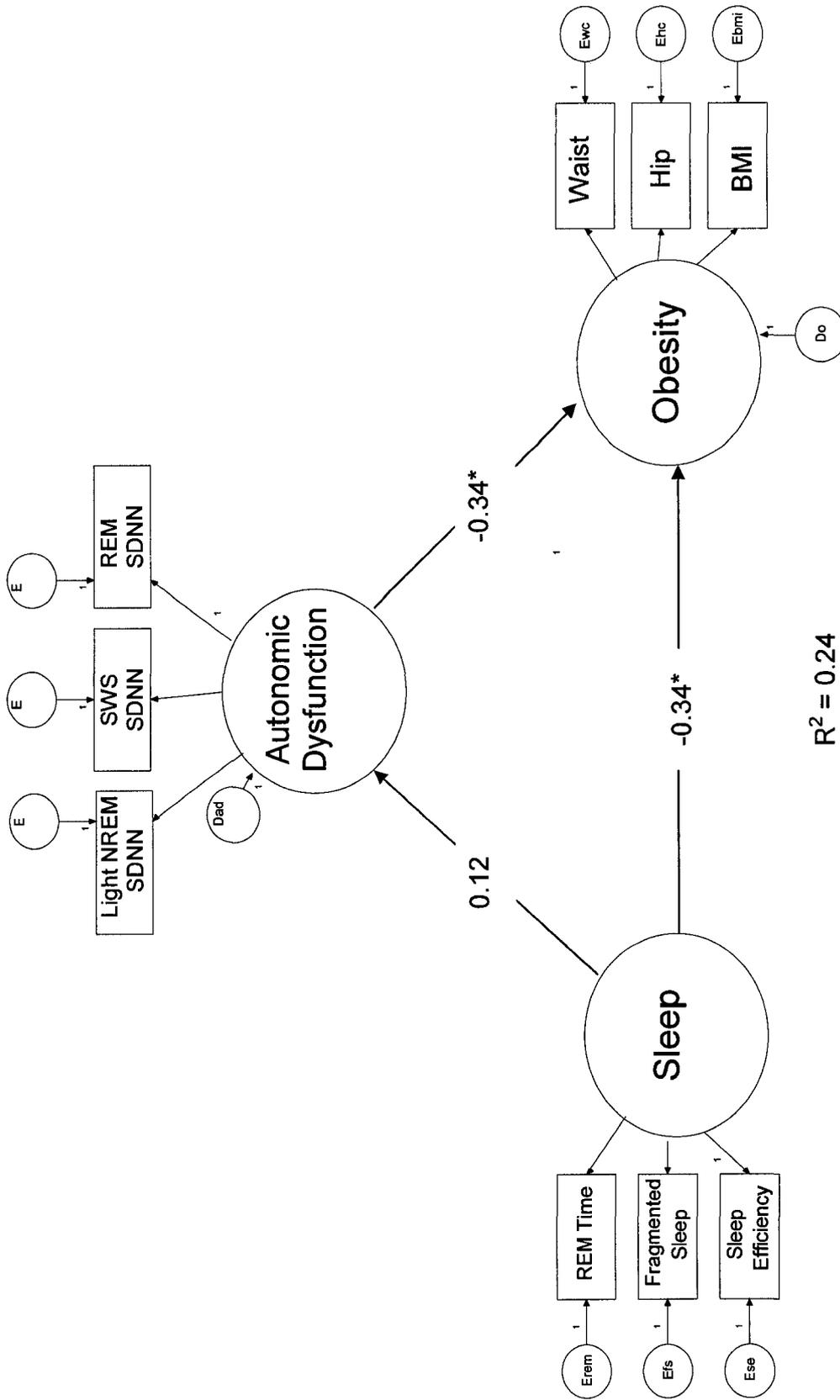


Figure 2. Structural equation model: Influence of objective sleep measures and autonomic dysfunction, made up of SDNN, on obesity. * $r > 1.96$ (2-tailed).

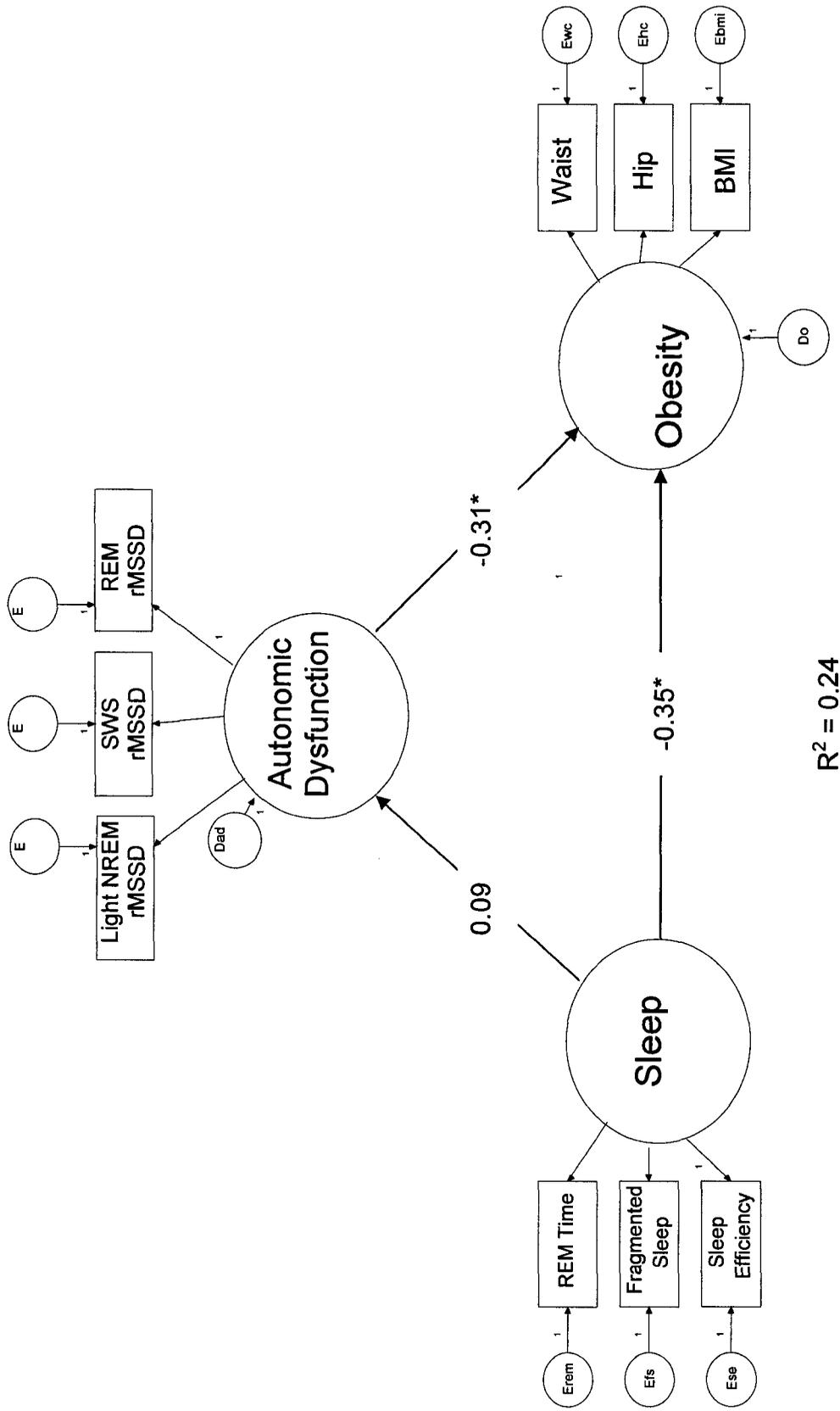


Figure 3. Structural equation model: Influence of objective sleep measures and autonomic dysfunction, made up of rMSSD, on obesity. * $P > 1.96$ (2-tailed).

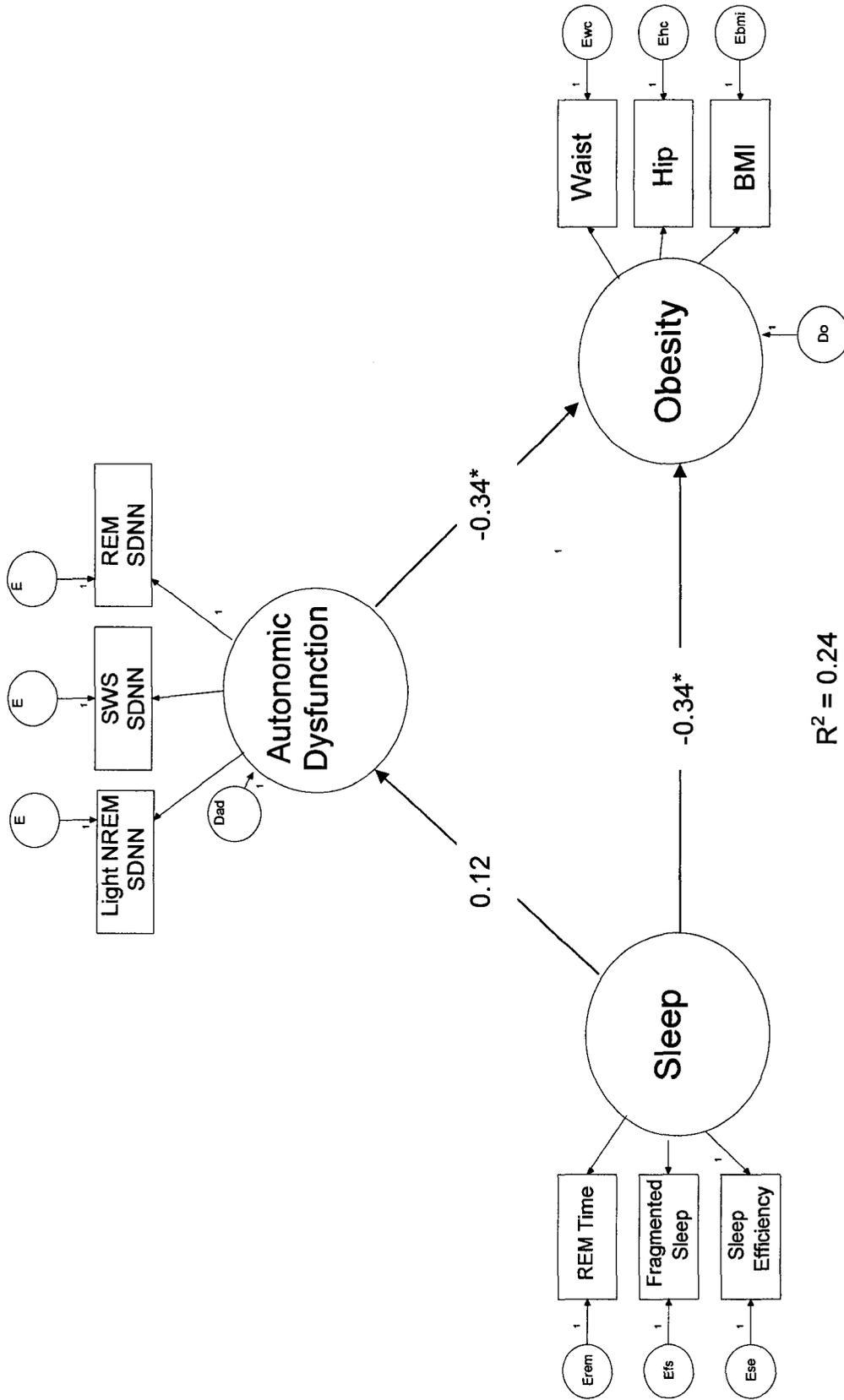


Figure 2. Structural equation model: Influence of objective sleep measures and autonomic dysfunction, made up of SDNN, on obesity. * $t > 1.96$ (2-tailed).