SLEEP DURATION, SLEEPINESS, OBESITY, AND RISK FOR HYPERTENSION IN YOUNG ADOLESCENTS: A MEDIATION MODEL

by

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ABSTRACT

HANNAH PEACH. Sleep duration, sleepiness, obesity, and risk for hypertension in young adolescents: A mediation model. (Under the direction of DR. JANE F. GAULTNEY)

Inadequate sleep has been identified as a risk factor for a variety of health consequences. Short sleep durations and daytime sleepiness, an indicator of insufficient sleep and/or poor sleep quality, have been identified as risk factors for hypertension in the adult population, but less evidence demonstrates such relations within child and early adolescent samples. Furthermore, mechanisms by which sleep influences blood pressure (BP) during youth are unclear. Using data from the Study of Early Child Care and Youth Development, the present study examined school-night sleep duration, weekend night sleep duration, and daytime sleepiness as predictors of hypertension in a sample of sixth graders (*N*=485) and tested body mass index (BMI) as a possible mediator. Findings demonstrated differential gender patterns in which daytime sleepiness predicted risk for hypertension among girls, while only school-night and weekend night sleep duration were significant predictors among boys. Additionally, sleep variables indirectly predicted both systolic and diastolic BP via BMI only among the total sample and boys. Findings provide clarification for the influence of sleep on BP during early adolescence and suggest the need for gender-specific designs in future research and application endeavors.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

BP	blood pressure
BMI	body mass index
CI	confidence interval
CSHQ	Children's Sleep Habits Questionnaire
DBP	diastolic blood pressure
NICHD	National Institute of Child Health and Human Development
OR	odds ratio
SBP	systolic blood pressure
SECCYD	Study of Early Child Care and Youth Development

CHAPTER 1: INTRODUCTION

In adults, inadequate sleep, which is often defined as sleep durations shorter than the recommended 7+ hours per night or poor sleep quality with or without the presence of a sleep disorder (Knutson, 2012), has been identified as a risk factor for a variety of health consequences, including hypertension (Croix & Feig, 2006; Wang, Xi, Liu, Zhang, & Fu, 2012). Although a substantial body of research has demonstrated a link between sleep disordered breathing and cardiovascular abnormalities in children and adolescents (Horne et al., 2011; Ka Li, Ng, & Yiu Fai, 2003; Li, Au, Ho, Fok, & Wing, 2009; Ng, Chan, Chow, Chow, & Kwok, 2005), very little research has been dedicated to the investigation of *characteristics of inadequate sleep* and elevated blood pressure (BP) within non-clinical samples of youth. While diagnoses of sleep disordered breathing comprise approximately 2% of children (American Academy of Sleep Medicine, 2008), a larger portion of youth suffer from more general sleep problems that can predict similar health consequences as disordered sleep. Considering adolescence marks a time period of changing sleep patterns and insufficient sleep due to biological, environmental, behavioral, and social influences (National Sleep Foundation, 2006; O'Brien & Mindell, 2005), with prevalence rates of adolescent sleep problems estimated at 25-40% (Mindell & Meltzer, 2008), examining the association of sleep problems with BP during late childhood/early adolescence would prove relevant.

Therefore, the goal of the present study was to expand current literature by examining sleep duration and daytime sleepiness as predictors of BP within a non-clinical sample of adolescents. As suggested by previous literature, quantity of sleep is a characteristic commonly researched as an indicator of sufficient restoration, and amounts of sleep required for optimal physiological function at all age groups have been well-established. Within the present study, "quantity" of sleep will be investigated via measures of both school-night and weekend sleep duration. Additionally, reported daytime sleepiness has been recognized as important in health research (Parker, Kutner, Bliwise, Bailey, & Rye, 2003). Daytime sleepiness serves as a behavioral manifestation of insufficient sleep and/or poor sleep quality and has been identified as an independent risk factor for hypertension (Goldstein, Ancoli-Israel, & Shapiro, 2004) and cardiovascular mortality (Empana et al., 2009) among older adults, although such research has not yet been expanded to younger populations. Considering such, reports of daytime sleepiness were examined as a predictor of BP within the present study.

Sleep duration and daytime sleepiness represent independent factors. Although both sleep quality and quantity are associated with daytime sleepiness, associations between sleep quality and daytime sleepiness are stronger than that of sleep duration and daytime sleepiness (Pilcher, Ginter, & Sadowsky, 1997). Furthermore, correlations between sleep quality and quantity are low in children and adolescents (Dewald, Meijer, Oort, Kerkhof, & Bögels, 2010); therefore daytime sleepiness reported in the absence of apparent sleep restriction indicates disordered/poor sleep quality distinct from quantity of sleep. Research has shown that daytime sleepiness and sleep duration are differentially affected by other factors (Hawkley, Preacher, & Cacioppo, 2010) and are independent

predictors of health and performance outcomes (Dewald et al., 2010; Empana et al., 2009; Goldstein et al., 2004).

CHAPTER 2: LITERATURE REVIEW

2.1 Sleep Characteristics and BP

The few studies that have examined adolescent BP and sleep characteristics beyond sleep disorder diagnoses have yielded mixed results. While two studies suggested a link from shorter sleep (Mezick, Hall, & Matthews, 2012) and poor sleep quality (Javaheri, Storfer-Isser, Rosen, & Redline, 2008) to elevated BP after controlling for the influence of associated covariates, other research has found no such association (Bayer, Neuhauser, & von Kries, 2009). Other studies have failed to demonstrate a link between BP and other characteristics of sleep problems, such as snoring (Kaditis et al., 2005). Data from the 2003-2006 National Health and Nutrition Examination Survey (Ostchega et al., 2009) suggested that approximately 13% of American youth ages 8-17 years old are classified as at risk for hypertension, and prevalence rates may be even higher at the present time. Considering that hypertension is a leading cause of death in the United States (Roger et al., 2011) and BP levels in early life can track into adulthood and predict hypertension and metabolic syndrome (Martikainen et al., 2011; Mezick et al., 2012; Sun et al., 2007), associations between poor sleep and elevated BP during youth must be confirmed and theoretical frameworks outlining possible underlying mechanisms of the relationship proposed.

Though the precise physiological mechanism by which sleep relates to BP has not yet been established, several pathways have been suggested within the literature,

including a reduction in nocturnal blood pressure dipping (Sayk et al., 2010). Other studies have suggested that shorter sleep leads to longer exposures to elevated sympathetic nervous system arousal (Huang et al., 2012; Sapolsky, 2004) and resulting waking physical and psychosocial stressors that generate increased salt retention (Gangwisch et al., 2006). Furthermore, inadequate sleep can lead to disrupted circadian rhythmicity and autonomic balance (Gangwisch et al., 2006), as well as diminished melatonin production (Zisapel, 2007), all of which affect BP. Most research has been conducted in adult samples; therefore it is unclear if these etiologies are also predictive of child and adolescent cases of elevated BP. Hypertension in older populations typically stems from stiffening of the arteries and increased peripheral resistance, which corresponds with aging. However, hypertension in adolescents is driven primarily by increased sympathetic activity and cardiac output (Foëx, Phil, & Sear, 2004); therefore mechanisms by which sleep contributes to elevated sympathetic activity and/or overworked cardiac output are plausible explanations for the sleep-BP link in early life. One such mechanism that has yet to be proposed is the indirect pathway through obesity, defined within children as a body mass index (BMI) at or above the 95th percentile for children of the same age and sex (Barlow, 2007).

2.2 Mediation Model and BMI

A substantial body of research has linked a variety of sleep disorders and characteristics of inadequate sleep to obesity in adults (Panossian & Veasey, 2012; Patel & Hu, 2008; Zimberg et al., 2012). While fewer studies have been conducted in children and adolescents, some research does suggest a significant association between sleep duration and obesity (Börnhorst et al., 2012; Garaulet et al., 2011; Gupta, Mueller, Chan,

& Meininger, 2002; Lytle, Pasch, & Farbakhsh, 2011; Nielsen, Danielsen, & Sørensen, 2011). Additionally, it has been proposed that the association between inadequate sleep and BMI is stronger during late childhood/early adolescence, and therefore children and adolescents may in fact be particularly vulnerable to the consequences of inadequate sleep (Danielsen, Pallesen, Stormark, Nordhus, & Bjorvatn, 2010; Knutson, 2012). Aside from the influence of fatigue on reduced physical activity, studies have shown that inadequate sleep can contribute to the development of obesity by affecting levels of hormones leptin and ghrelin, which are involved in appetite regulation, by reducing energy expenditure, and by increasing subjective appetite (Knutson, 2012).

However, some research has yielded null findings (Calamaro et al., 2010; Hassan, Davis, & Chervin, 2011), and other studies have demonstrated contrasting gender differences in which sleep duration is associated with obesity only among female adolescents (Lowry et al., 2012) or demonstrated stronger patterns among males (Knutson, 2005; Storfer-Isser, Patel, Babineau, & Redline, 2012). Such differential patterns among boys and girls, possibly stemming from hormonal differences, may serve as a viable explanation for null findings in samples that did not conduct separate analyses for both genders.

Identifying contributors to the development of childhood/adolescent obesity is essential, for obesity serves as a significant and widely recognized risk factor for concurrent elevated BP (Babinska, Kovacs, Janko, Dallos, & Feber, 2012; Ma et al., 2012; Oduwole, Ladapo, Fajolu, Ekure, & Adeniyi, 2012; Zhang, Shi, Huang, Feng, & Ma, 2012), as well as adult hypertension (Sabo, Lu, Daniels, & Sun, 2010). The excessive weight that characterizes obesity places strain on cardiac output, which in turn

drives elevations in BP. Obesity can also lead to metabolic abnormalities and chronic kidney diseases, both of which contribute to the development of hypertension (Narkiewicz, 2006). Considering the increasing prevalence of insufficient sleep among youth (National Sleep Foundation, 2006; O'Brien & Mindell, 2005), as well as obesity rates estimated at 17% (12.5 million) of U.S. children and adolescents aged 2-19 years (Ogden & Carroll, 2010) this sleep-obesity relationship serves as a plausible indirect pathway to elevated BP.

2.3 Present Study

While other studies have identified associations between sleep, BMI, and BP, a mediation model is not yet well examined within the literature. For example, a comprehensive literature review (Knutson, 2012) addressed the effect of sleep on obesity, diabetes, cardiovascular disease, and hypertension, and although the link between obesity and hypertension is discussed, it is never proposed that obesity may serve as an additional indirect pathway from sleep to BP. Specifically in adolescents, only two studies have examined the effect of sleep on both BMI and BP. Narang et al. (2012) found sleep as a risk factor for cardiovascular health, including elevated BP and obesity, and Wells et al. (2008) demonstrated a link from sleep duration to both BP and BMI. Although research strongly acknowledges that all three constructs are interrelated and findings have *suggested* obesity as a plausible pathway between sleep and BP, to the best of our knowledge no studies have empirically tested the theory that sleep is related to BP partially through the pathway of obesity within early adolescence. Instead, obesity is treated as a confounding variable in studies of adolescent sleep and BP (Li, Au, Ho, Fok,

& Wing, 2009), and researchers simply adjust for BMI during analyses rather than addressing a possible mechanistic role within the relationship.

CHAPTER 3: STUDY GOALS

Considering the biological mechanisms that connect inadequate sleep to obesity, and the repeatedly demonstrated link between obesity and risk for hypertension, the goal of the present study was to clarify the three-way relationship by first examining associations and odds ratios within an early adolescent sample, then testing mediation models (see Figure 1) in which the relationship between inadequate sleep and elevated BP in adolescence is partially mediated by BMI. Considering the differing hormonal changes that occur for boys and girls during the pubertal period that marks adolescence, as well as the varying patterns of the sleep-BP relationship demonstrated within the literature, the present study tested the proposed model within a total sample of sixth graders and separately by gender.

CHAPTER 4: STUDY METHODS

4.1 Participants

The present study analyzed secondary data from the Study of Early Child Care and Youth Development (SECCYD). This longitudinal study was initiated by the National Institute of Child Health and Human Development (NICHD) and conducted at 10 different U.S. research sites. The study was designed to examine the relationship between child care experiences, child development, and well-being. Initial data collection began in 1991, where families were recruited to participate in the study during hospital visits to mothers shortly after the birth of a child. A conditionally random sample of 3,015 families was chosen. Within the initial sample, 1,364 eligible families were interviewed when the child was one month old, and these children were followed until three years of age (1991-1994). Phase II (1995-1999) yielded data for 1,226 children from 54 months through first grade; Phase III (2000-2004) yielded data for 1,061 children from second grade through sixth grade; and Phase IV (2005-2007) yielded data for 1,009 children from seventh grade through ninth grade. More extensive information regarding the recruitment, selection process, and eligibility requirements is available from previous publications (see http://www.nichd.nih.gov/research/supported/seccyd/bibliocfm; http://secc.rti.org).

The present study included data from Phase III, specifically when the children were in the sixth grade (n = 485). In the current study, only participants providing full

data at this time frame usable for statistical analysis were included (see Table 1 for descriptive data).

4.2 Control Measures

Demographics. Gender and race/ethnicity were reported during the first phase of data collection. Family income was measured as parental report of the total pre-tax family income at grade 6 (2002-2003).

Physical Activity. The present study included a measure of the average minutes per day each study child spent in moderate activity all day. Physical Activity Monitoring was used to measure activity over a period of 7 days during a typical school week. Each study child wore a single channel accelerometer that collected movement data by recording multiple accelerations, or changes in the child's total body movement over a defined period of time, that indicated level of physical activity. Monitors were not worn at night while sleeping or during water related activities. A full day of activity monitor data were defined as the time frame beginning with the first nonzero accelerometer count after 5 a.m. and ending with whichever of the following criteria came first: 60 consecutive minutes of zero counts after 9 p.m.; 30 consecutive minutes of zero counts after 10 p.m.; or the last nonzero count prior to midnight. Accelerometer counts were computed as the total number of minutes calculated for each day, and invalid days were coded and removed. The number of minutes spent per day in moderate activity (3 – 5.9 units of metabolic equivalent of task) were divided by the number of minutes spent wearing the monitor; this provided a measure of the percentage of time engaged in moderate activity while the monitor was worn, as well as a means of comparing across the varying lengths of time the monitor was worn by different children and by the same

child on various days. Data were averaged across all valid days for which the study child had data, yielding a measure of average minutes spent per day in moderate activity, which was used as a measurement of exercise in the present study. Intracorrelations and 95% confidence intervals were used to analyze data from a sample of 30 children who wore the monitors for 12 hours per day for 6 days, demonstrating adequate stability (r= 0.81-0.84) when 6 days of data were used and acceptable correlations (r = 0.75-0.78, CI = 0.60-0.88) when at least 4 days of data were used.

Pubertal Development. The Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988) measured stages of pubertal development, with separate forms used for boys and girls. Mothers responded to a series of 5 questions evaluating the degree to which a specific physical change (i.e. pimply skin, growth spurt, breast development, menstrual cycle, deepening of voice, and body/facial hair) had occurred in their child. Response scales ranged from 1 (specified development has not started) to 4 (development seems complete), and also provided a "don't know" response, which was recoded as missing data. The item regarding female participants' first menstrual period was recoded as 4=Yes and 1=No. Mean scores were computed for complete data, with higher scores indicating more advanced stages of pubertal development.

Unhealthy Eating Habits. The Child's Eating Habits and Body Self Image questionnaire, which included items slightly modified from the Youth Risk Behavior Survey (Brener et al., 2002) generated an index of unhealthy eating habits as reported by the study child. Four items pertaining to the consumption of unhealthy meals, snacks, and drinks were summed, with scores ranging from 0-12. Higher scores indicated more

unhealthy eating habits. The raw items used to create the index score had modest internal reliability (Cronbach's alpha = .59).

Depressive symptoms. Depression was measured via the Children's Depression Inventory Short Form (Kovacs, 1992), a widely used 10-item questionnaire with a response scale ranging from 0-2. Items 3, 4, 5, 6, and 10 are reverse coded and responses are summed, yielding a total score ranging from 0-20 with higher scorings indicating more depressive symptoms. Scores above 8 for girls and above 10 for boys are considered "well above average." The instrument yielded adequate internal reliability within this sample (Cronbach's alpha = .73).

4.3 Outcome Measures

Blood Pressure. Continuous measures of systolic BP (SBP) and diastolic BP (DBP), as well as dichotomized hypertension classifications, were included in the present study. BP was measured by a nurse practitioner during the annual Health and Physical Development Assessment of the study. To preserve uniformity in data collection across sites, BP was taken with the child seated, using the non-dominant arm, and measured via a blood pressure cuff and stethoscope. Readings were taken a second time if the child appeared anxious or if the initial readings were high. When two readings were reported, the value of the analysis variable was set to the value of the second reading; this yielded raw measures of both SBP and DBP. Using BP standards for children and adolescents based on sex, age, and height, the present study computed BP percentiles from growth charts published by the Centers for Disease and Prevention (CDC; 2000). Using a formula provided by the Fourth Report of the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents (National High Blood Pressure

Education, 2004), separate SBP and DBP percentiles were calculated as a function of age and sex. In children, prehypertension is defined as an average SBP or DBP level that is greater than or equal to the 90th percentile for sex, age, and height, while hypertension is defined as greater or equal to the appropriate 95th percentile (National High Blood Pressure Education, 2004). Less than 3% of the sample was classified as hypertension, therefore BP measurements were dichotomized as normotensive (SBP and DBP < 90th percentile) or at risk for hypertension (SDP or DBP ≥ 90th percentile).

Body Mass Index. Continuous BMI measures, as well as a dichotomized classification of obesity were included in the present study. BMI was computed from height and weight measurements recording during laboratory visits. Height was given in inches and centimeters; weight was given in pounds and kilograms. BMI was calculated by original researchers and included computed gender and age adjusted percentiles for BMI. Based on national recommendations (Barlow, 2007), the present study used BMI percentiles measurements to create a dichotomous variable to classify participants as obese (BMI percentile ≥95) or non-obese (<95%).

4.4 Sleep Measures

Daytime Sleepiness. Daytime sleepiness was measured via a subscale of the My Child's Sleep Habits scale, which is a set of questions from the Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000). Measures were reported by each study child's mother. Four items referring to daytime sleepiness (i.e. "My child seems tired during the day") with response scales ranging from 1 (usually) to 3 (rarely) were reverse scored and averaged, such that higher scores indicated more daytime

sleepiness problems. The raw items used to create the problem score had modest internal reliability (Cronbach's alpha = 0.65).

Sleep Duration. Sleep duration was reported by the study child using a scale developed by the original study researchers that included items adapted from the CSHQ. Questions were administered to study children in an interview format and included items regarding children's bedtimes, amount of sleep, and difficulties going to sleep. The present study included measures of (1) sleep duration on school nights, as well as (2) sleep duration on weekend nights.

4.5 Data Analysis Plan

To determine the impact of school-night sleep duration, weekend sleep duration, and daytime sleepiness on the odds of being obese, logistic regressions were performed with dichotomized obesity classification as the outcome. To determine the unique impact of school-night sleep duration, weekend sleep duration, and daytime sleepiness on the odds of being prehypertensive, three logistic regressions were performed with dichotomized hypertension classification as the outcome. To account for related constructs, sleep variables (school-night sleep duration, weekend night sleep duration, and daytime sleepiness) were regressed onto gender, income, race/ethnicity, unhealthy eating habits, daily physical activity, depressive symptoms, and pubertal status. The residualized primary variables were then entered into the logistic regressions. Analyses were conducted within the full sample, as well as separately within only boys and only girls.

To test the proposed mediation model that suggests sleep has an indirect effect on BP via associations with obesity, path analysis was conducted within the full sample, and

bootstrapping determined confidence intervals (CIs) for indirect paths. CIs excluding zero were interpreted as statistically significant. Correlations of school-night sleep duration and weekend sleep duration with daytime sleepiness were low, which corresponds with prior research suggesting low or modest associations of sleep duration with sleep quality and daytime sleepiness (Dewald et al., 2010). Furthermore, school-night and sleep duration yielded correlations of varying strengths with BMI and BP, therefore we examined the unique effects of each sleep component; path analyses were run separately such that only one sleep variable at a time served as the exogenous variable. These yielded three models that examined the effect of school-night sleep duration on BMI and BP, weekend night sleep duration on BMI and BP, and daytime sleepiness on BMI and BP. To identify possible differential patterns between genders, separate path analyses were also conducted within gender subgroups. Original BMI scores were analyzed as the mediator, and raw systolic and diastolic BP were analyzed as outcome variables. Again, analyses controlled for gender, income, race/ethnicity, unhealthy eating habits, daily physical activity, depressive symptoms, and pubertal status by regressing BMI and the sleep variables onto the aforementioned variables, then entering the residualized primary variables into the path analysis.

CHAPTER 5: RESULTS

5.1 Preliminary Analyses

Among the total sample with complete data, descriptive information and correlations among primary variables (see Table 2) demonstrated that all variables yielded reasonable means and variability, and overall primary variables correlated with one another within the expected direction. Average sleep durations exceeded the nationally recommended 8.5-9.25 hours per night for this age group (National Sleep Foundation, 2009). Of the total sample, 13.8% were classified as at risk for hypertension; 14.1% of boys and 13.6% of girls were classified as at risk for hypertension. In the total sample, 18.1% were classified as obese, with 19.8% of boys and 16.7% of girls classified as obese. BMI among girls (20.79) and boys (20.67), was not significantly different, $t(483) = -.29$, $p = .77$. Additionally, measures of SBP (girls: 104, boys: 103.99), $t(483) = -.02$, $p = .99$, and DBP (girls: 62.02, boys: 61.68), $t(483) = -.40$, $p = .69$, were not significantly different between boys and girls. Only weekend sleep duration was slightly higher among boys, $t(483) = -2.48$, $p = .01$, while school-night sleep duration, $t(483) = -.98$, $p = .33$, and daytime sleepiness did not differ, $t(483) = -.58$, $p = .56$.

To ensure participants with missing data did not differ from those included in the present study, descriptive information and correlations of the primary variables were compared between participants with complete data and participants excluded from present analyses. Although excluded participants yielded higher average family income

($91,681.37) and slightly higher unhealthy eating habits (3.43), all other means and standard deviations were similar to those of the present study. Overall, correlations were also within similar strength and direction as the present study, although correlations of weekend sleep duration with BMI and SBP no longer reached statistical significance.

5.2 Planned Analyses

Among the total sample, logistic regressions (see Table 3) revealed that school-night sleep duration and daytime sleepiness significantly predicted risk for obesity and both independently accounted for 3% of the total variance. Every one hour decrease in school-night sleep increased the odds of being obese by 38%, while every one unit increase in reported daytime sleepiness increased the odds by 294%. Differential patterns emerged when analyzing logistic regressions among gender subgroups. For girls, none of the sleep variables significantly predicted risk for obesity. For boys, both school-night and weekend sleep durations significantly predicted risk for obesity, with school-night sleep duration uniquely accounting for 2% of the total variance and weekend sleep duration uniquely accounting for 7% of the total variance. Every hour decrease in school-night sleep duration increased the odds of being obese by 36%, and every hour decrease in weekend sleep duration increased odds by 27%.

When regressing risk for hypertension onto sleep variables, logistic regressions among the total sample revealed only daytime sleepiness as a significant predictor, accounting for 3% of the total variance. For every one unit increase in daytime sleepiness, the risk for hypertension increased approximately three-fold (O.R. = 3.99). Again, differential patterns emerged among gender subgroups. For girls, only daytime sleepiness was a significant predictor and accounted for 3% of the total variance in risk

for hypertension. For every one unit increase in daytime sleepiness, risk for hypertension increased by 287%. Oppositely, school-night and weekend sleep duration were significant predictors of risk for hypertension among boys, with school-night sleep duration uniquely accounting for 4% of the total variance and weekend sleep duration uniquely accounted for 12% of the total variance. For every hour decrease in school-night sleep duration, the risk for hypertension increased by 43%. For every hour decrease on weekend sleep duration the risk for hypertension increased by 35%.

Eighteen separate path models were conducted (see Table 4); separate path models were analyzed for each sleep variable predicting SBP and DBP, and these analyses were examined within the total sample and the gender subgroups. Within the total sample, all path models accounted for 17-18% of the total variance in SBP and 9-10% of the variance in DBP. School-night sleep duration, weekend sleep duration, and daytime sleepiness all independently predicted BMI, and in turn BMI significantly predicted both SBP and DBP in all path models. While none of the sleep variables yielded significant direct effects on SBP or DBP, school-night sleep duration yielded a significant indirect effect on SBP (CI [-1.87, -.42]) and DBP (CI [-1.02, -.25]) via BMI. Weekend sleep duration also yielded a significant indirect effect on SBP (CI [-1.01, -.29]) and DBP (CI [-1.59, -.17]) via BMI. Lastly, daytime sleepiness yielded a significant indirect effect on SBP (CI [.20, 5.47)] and DBP (CI [.05, 3.06]) via BMI.

Differing patterns emerged between the gender subgroups (see Table 4). In girls, all path models accounted for 15-16% of the total variance in SBP and 8% of the total variance in DBP. In boys, all path models accounted for 20% of the total variance in SBP and 11% of the total variance in DBP. In girls, only daytime sleepiness had a significant

direct effect on BMI, while in boys only school-night and weekend night sleep duration significantly predicted BMI. BMI significantly predicted SBP and DBP for boys and girls in all path models. In girls, only school-night sleep duration had a significant direct effect on SBP, and the path weight was not in the expected direction (i.e., longer sleep duration was associated with higher SBP). Among girls, there were no significant indirect effect of school-night sleep duration (CI [-1.35, .35]), weekend sleep duration (CI [-.70, .12]) or daytime sleepiness (CI [-.05, 5.67]) on SBP. Additionally, there were no significant direct effects or indirect effects of school-night sleep duration (CI [-.87, .12]), weekend sleep duration (CI [-.49, .03]), or daytime sleepiness (CI [.06, 4.49]) on DBP.

In boys, none of the sleep variables had a significant direct effect on SBP or DBP, and daytime sleepiness yielded no significant indirect effects on SBP (CI [-1.67, 7.99]) or DBP (CI [-.92, 4.16]). However, school-night sleep duration yielded significant indirect effects on both SBP (CI [-3.42, -.74]) and DBP (CI [-1.82, -.34]) via BMI. Similarly, weekend sleep duration demonstrated significant indirect effects on SBP (CI [-.1.57, -.48]) and DBP (CI [-.88, -.24]) via BMI.

CHAPTER 6: DISCUSSION

5.1 Summary of Main Findings

Findings from the present study demonstrate that both sleep quantity and daytime sleepiness, an indicator of insufficient/poor quality sleep, serve as risk factors for obesity and high blood pressure in a sample of sixth graders, although the relative contribution of each sleep predictor varied by gender. To our knowledge, this is the first study to examine a mediation model of sleep, BMI, and risk for hypertension in young adolescents and to examine such relations separately between boys and girls. Despite several limitations of the present study, findings yield empirical and application implications, as well as directions for future research.

When examining sleep characteristics as predictors of dichotomized risk for obesity and hypertension within the total sample, results suggest both (school-night) sleep duration and daytime sleepiness increase the odds of being classified as obese, and also increase the odds of being classified as prehypertensive/hypertensive. Additionally, all three sleep variables yield significant indirect effects of DBP and SBP through associations with BMI. However, further analyses among the gender subgroups suggest varying patterns are emerging, therefore conclusions regarding a sleep-BMI-BP relationship without considering gender differences may be misleading.

Differential gender patterns suggest the risk of developing hypertension among girls may be more sensitive to insufficient/poor quality sleep indicated by daytime

sleepiness, while shortened sleep durations may be more important for weight and BP management among boys. School-night and weekend sleep duration served as significant risk factors of obesity classification and raw BMI measurements among boys. Among girls, however, no sleep variables served as significant risk factors for dichotomous classifications of obesity; only daytime sleepiness significantly predicted raw BMI scores. Such findings are in line with prior studies that have shown stronger effects of sleep duration on BMI in males (Knutson, 2005; Storfer-Isser et al., 2012) and suggest that future research must consider additional characteristics of sleep, beyond sleep quantity, as contributors to obesity. Additionally, research must explore identification of the underlying mechanisms by which various sleep characteristics could have unique and differing effects on boys versus girls.

Furthermore, sleep characteristics as predictors of raw BP measures and hypertension classification yielded contrasting patterns between boys and girls. For boys, both school-night and weekend sleep duration increased the odds of being classified as obese and also yielded significant indirect effects on both SBP and DBP. While daytime sleepiness yielded no effects on dichotomous or raw measurements of BP among boys, daytime sleepiness increased the odds of hypertension classification by nearly three-fold among girls. Contrary to expectations, school-night sleep duration had a significant positive effect on SBP among girls, which the present study was unable to explain. However, findings indicating differential gender patterns do not emerge from mere differences in means among these primary variables; although some studies suggest BMI or BP measurements are higher among one gender (Juhász et al., 2010), our results are suggesting differing underlying mechanisms by which sleep influences BMI, which in

turn affects BP measurements among boys and girls. Future studies should replicate and expand upon the present findings to identify how and why sleep quantity differentially influences BMI among boys and indicators of insufficient/poor quality sleep yield physiological effects among girls.

6.2 Strengths and Limitations

Within the present study, a rich data set allowed for the control of important contributing factors to both BMI and BP, including demographic variables such as race/ethnicity and family income, as well as behavioral and physiological influences, including physical activity, unhealthy eating habits, pubertal development, and depression. By controlling for these variables, analyses yielded the unique and independent effects of sleep on obesity and risk for hypertension above and beyond the influence of these related factors. Effect sizes can therefore be interpreted as exclusively due to the influence of sleep. Although research suggests the influences of non-disordered sleep in younger populations are modest in size, weekend sleep quantity among boys accounted for 7% and 12% of the variance in obesity and hypertension classification, respectively. Such effect sizes reflect relatively strong influences, particularly considering that vital contributors such as diet and exercise were held constant. Additionally, considering mixed findings within the adolescent health literature, the present analyses provide support for prior research demonstrating a link between sleep and obesity (Börnhorst et al., 2012; Garaulet et al., 2011; Gupta et al., 2002; Lytle et al., 2011; Nielsen et al., 2011), as well as sleep and high BP (Javaheri, et al., 2008; Mezick et al., 2012), in younger adolescents.

While the present findings yield additional clarification for the influences of sleep on BMI and BP among young adolescents, several limitations of the present study are evident and provide direction for future research. Firstly, while BP and BMI measurements met the "gold-standard" of objective calculation, all sleep measures within the data set were self or parental-report. School-night and weekend sleep may be less reliable as a "typical" measure of sleep if the study child's parents are divorced and home environments differ from week to week. Additionally, daytime sleepiness served only as a behavioral indicator of insufficient sleep and/or poor sleep quality, and while daytime sleepiness serves as a predictor of a variety of health outcomes (e.g., Parker et al., 2003), additional research should include more narrowed measures of such constructs. Future studies can replicate the present analyses using objective measures of sleep, such as actigraphy or polysomnography.

Secondly, the present study proposes a limited model; we are not addressing bidirectionality or disordered sleep within this study, therefore additional research is needed to clarify if BMI and high BP are in fact influencing sleep quality or quantity in some way. Furthermore, this research only demonstrates *patterns* of the influences of sleep within young adolescent boys and girls. We cannot conclude causality or identify underlying physiological mechanisms from the present study. However, the clarification of such patterns is necessary in order to devote further research to causal mechanisms, and the present study provides justification for future research to not only examine causal pathways from insufficient sleep to higher BMI and BP, but to propose how and why such pathways vary between boys and girls. For example, studies in adults suggest sex hormones may play a role in heart health (Coulter, 2011), and although this study

controlled for pubertal development, future research can examine hormones as a possible underlying mechanism by which the sleep-BMI-BP relationship differs between adolescent boys and girls.

6.3 Implications and Future Research

Findings from the present study yield both research and applicatory implications. The clarification of sleep characteristics as predictors of obesity and hypertension classification meaningfully contributes to a body of literature generating contrasting findings. Additionally, analyses supported the proposed mediation model in which sleep quantity and daytime sleepiness yield indirect effects on BP through associations with obesity. The proposed model provides information regarding a possible mechanism by which sleep may influence BP at an early age. Considering early adolescence provides a period of opportunity for the prevention of or early intervention for hypertension (Mezick et al., 2012), the clarification of the mechanisms underlying the sleep-BP link may inform the design of such prevention/intervention efforts.

Furthermore, the emerging differential patterns between boys and girls, while preliminary findings, may suggest that application efforts as well as future research should be gender-specific. Efforts to lengthen regular sleep duration among boys may be protective against increasing BMI and BP levels, while daytime sleepiness among girls may serve as an identifiable risk factor for such physiological consequences. Future research and application efforts within the adolescent field should consider sleep as a significant influence on health that may contribute to the prevention of obesity and hypertension development.

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Table 1: Demographic information

Variable	Total			Girls			Boys		
	M (*N*)	SD	Range	M (*N*)	SD	Range	M (*N*)	SD	Range
N	(485)			(258)			(227)		
Race									
White	(402)			(206)			(196)		
Black	(53)			(36)			(17)		
Asian	(6)			(4)			(2)		
Other	(24)			(12)			(12)		
Income	82422.68	67265.48	2500-5.5[5]	78798.45	65362.73	2500-5.5[5]	86541.85	69279.17	2500-5.5[5]
Physical Activity	81.80	27.62	4.75-220.71	76.30	25.89	4.75-182.14	88.06	28.24	29.25-220.71
Eating Habits	3.18	2.20	0-12	3.09	2.16	0-12	3.29	2.25	0-12
Pubertal Dev.	1.85	.66	1-3.80	2.19	.65	1-3.80	1.45	.38	1-3
Depression	1.43	2.27	0-19	1.51	2.35	0-14	1.35	2.19	0-19

Note. Values in parentheses indicate sample size. Pubertal Dev. = Pubertal Development.

Table 2: Descriptive statistics and correlations of blood pressure and predictors

	M	*SD*	Range	1	2	3	4	5	6
Total Sample									
1. BMI	20.73	4.83	11.60-39.26	(1.00)					
2. School-night Sleep	9.31	.76	7-12	-.17***	(1.00)				
3. Weekend Sleep	9.90	1.60	4-17.5	-.18***	.25***	(1.00)			
4. Daytime Sleepiness	1.12	.24	1-3	.12*	-.10*	.06	(1.00)		
5. Systolic Blood Pressure	104.49	12.42	11-138	.42***	-.04	-.10*	-.03	(1.00)	
6. Diastolic Blood Pressure	61.88	9.21	38-90	.31***	-.09*	-.06	.00	.43***	(1.00)
Girls									
1. BMI Percentile	20.79	4.64	13.96-37.37	(1.00)					
2. School-night Sleep	9.35	.80	7-12	-.12*	(1.00)				
3. Weekend Sleep	10.07	1.43	5-15	-.07	.29***	(1.00)			
4. Daytime Sleepiness	1.13	.25	1-3	.16*	-.13*	.04	(1.00)		
5. Systolic Blood Pressure	104.00	11.46	80-138	.39***	.06	-.13*	-.02	(1.00)	
6. Diastolic Blood Pressure	62.02	9.02	38-88	.29***	-.01	-.06	.04	.41***	(1.00)
Boys									
1. BMI Percentile	20.67	5.03	11.60-39.26	(1.00)					
2. School-night Sleep	9.28	.72	7.25-11	-.23***	(1.00)				
3. Weekend Sleep	9.71	1.76	4-17.5	-.27***	.21***	(1.00)			
4. Daytime Sleepiness	1.11	.22	1-2.25	.07	-.05	.08	(1.00)		
5. Systolic Blood Pressure	103.99	13.45	11-138	.45***	-.14*	-.07	-.04	(1.00)	
6. Diastolic Blood Pressure	61.68	9.44	40-90	.33***	-.19**	-.07	-.05	.45***	(1.00)

Note. $N = 485$. *indicates p<.05; **indicates p<.005; ***indicates p<.001.

Table 3: Logistic regressions of obesity and hypertension on sleep characteristics

	Total				Girls				Boys			
	b	S.E.	O.R.	R^2	*b*	S.E.	O.R.	R^2	*b*	S.E.	O.R.	R^2
Outcome of Obesity												
School-night Sleep	-.48**	.16	.62	.03	-.39	.22	.68	.02	-.44*	.20	.64	.02
Weekend Sleep	-.12	.08	.89	.01	-.08	.12	.93	.00	-.31**	.10	.73	.07
Daytime Sleepiness	1.37**	.45	3.94	.03	.56	.60	1.75	.01	.82	.66	2.27	.01
Outcome of Hypertension												
School-night Sleep	-.07	.18	.93	.00	.26	.23	1.30	.01	-.57*	.24	.57	.04
Weekend Sleep	-.07	.08	.93	.003	-.11	.13	.90	.01	-.44***	.11	.65	.12
Daytime Sleepiness	1.38**	.45	3.99	.03	1.35*	.60	3.87	.03	.89	.67	2.44	.01

Note. These analyses have been corrected for gender, race/ethnicity, income, physical activity, unhealthy eating habits, depression, and pubertal development. All predictors were entered into separate logistic regressions. *indicates p< .05, **indicates p<.01, ***indicates p<.001. *b* = unstandardized logistic regression coefficient; S.E. = Standard Error of *b*; O.R. = odds ratio. R^2 values are Nagelkerke pseudo-R^2 values.

Table 4: Direct, indirect, and total effects on blood pressure

	Total			Girls			Boys		
	BMI	SBP	DBP	BMI	SBP	DBP	BMI	SBP	DBP
School-night Sleep									
Total R^2		.18***	.10***		.16***	.08***		.20***	.11***
Direct	-.98***	.75	-.43	-.59	1.82*	.30	-1.52***	-.72	-1.48
Indirect	-	-1.11*	-.58*	-	-.59	-.35	-	-1.86*	-.91*
Total	-.98	-.36	-1.01	-.59	1.23	-.05	-1.52	-2.61	-2.39
BMI									
Direct	-	1.14***	.60***	-	1.01***	.59***	-	1.24***	.59***
Weekend Sleep									
Total R^2		.17***	.09***		.15***	.08***		.20***	.11***
Direct	-.56***	-.28	-.07	-.30	-.83	-.19	-.75***	.22	.05
Indirect	-	-.62*	-.34*	-	-.29	-.17	-	-.96*	-.49*
Total	-.56	-.90	-.41	-.30	-1.12	-.36	-.75	-.74	-.44
BMI									
Direct	-	1.10***	.61***	-	.95***	.58***	-	1.28***	.65***
Sleepiness									
Total R^2		.18***	.10***		.15***	.08***		.20***	.11***
Direct	2.10*	-3.86	-1.30	2.32*	-3.36	.00	1.80	-4.35	-3.03
Indirect	-	2.40*	1.30*	-	2.31	1.34	-	2.30	1.18
Total	2.10	-1.46	.00	2.32	-1.05	1.34	1.80	-2.05	-1.85
BMI									
Direct	-	1.14***	.62***	-	1.00***	.58***	-	1.28***	.66***

Note. These analyses have been corrected for gender, race/ethnicity, income, physical activity, unhealthy eating habits, depression, and pubertal development. Path weights derived from unstandardized regression coefficients. Sleepiness = daytime sleepiness. *indicates p< .05, **indicates p<.01, ***indicates p<.001. All significant indirect effects yielded confidence intervals that exclude zeros.

APPENDIX B: FIGURES

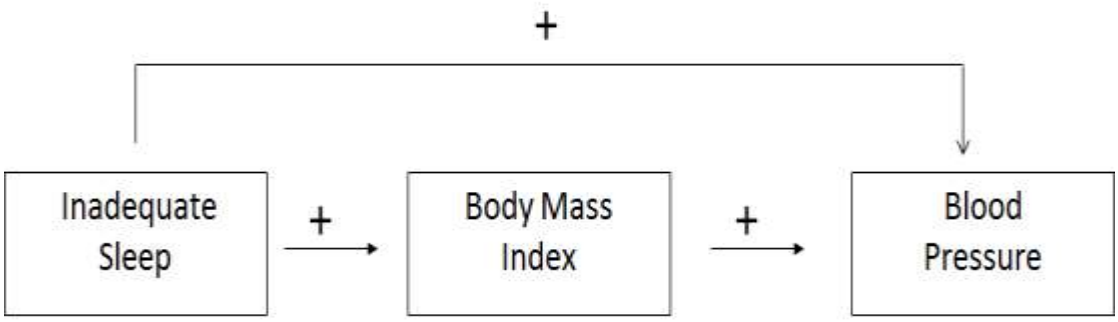

Figure 1. Mediation model in which inadequate sleep is partially mediated via associations with body mass index; the model does not account for possible bidirectionality. Analyses examined three separate models in which inadequate sleep was operationalized as school-night sleep duration, weekend sleep duration, and daytime sleepiness.