AN OVERVIEW OF SLEEP AND OBESITY
IN CHILDREN AND ADOLESCENTS

By

JULIA MELLO DAVIS

A Thesis Submitted to The Honors College
In Partial Fulfillment of the Bachelors Degree
With Honors in

Nutritional Sciences

THE UNIVERSITY OF ARIZONA

MAY 2013

Approved by:

Dr. Jennifer A.Teske
Department of Nutritional Sciences
The University of Arizona Electronic Theses and Dissertations
Reproduction and Distribution Rights Form

The UA Campus Repository supports the dissemination and preservation of scholarship produced by University of Arizona faculty, researchers, and students. The University Library, in collaboration with the Honors College, has established a collection in the UA Campus Repository to share, archive, and preserve undergraduate Honors theses.

Theses that are submitted to the UA Campus Repository are available for public view. Submission of your thesis to the Repository provides an opportunity for you to showcase your work to graduate schools and future employers. It also allows for your work to be accessed by others in your discipline, enabling you to contribute to the knowledge base in your field. Your signature on this consent form will determine whether your thesis is included in the repository.

<table>
<thead>
<tr>
<th>Name (Last, First, Middle)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Davis, Julia Mello</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Degree title (eg BA, BS, BSE, BSB, BFA):</th>
</tr>
</thead>
<tbody>
<tr>
<td>BS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Honors area (eg Molecular and Cellular Biology, English, Studio Art):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutritional Sciences</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date thesis submitted to Honors College:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Title of Honors thesis:</th>
</tr>
</thead>
<tbody>
<tr>
<td>An overview of Sleep and Obesity in Children and Adolescents</td>
</tr>
</tbody>
</table>

The University of Arizona Library Release Agreement

I hereby grant to the University of Arizona Library the nonexclusive worldwide right to reproduce and distribute my dissertation or thesis and abstract (herein, the "licensed materials"), in whole or in part, in any and all media of distribution and in any format in existence now or developed in the future. I represent and warrant to the University of Arizona that the licensed materials are my original work, that I am the sole owner of all rights in and to the licensed materials, and that none of the licensed materials infringe or violate the rights of others. I further represent that I have obtained all necessary rights to permit the University of Arizona Library to reproduce and distribute any nonpublic third party software necessary to access, display, run or print my dissertation or thesis. I acknowledge that University of Arizona Library may elect not to distribute my dissertation or thesis in digital format if, in its reasonable judgment, it believes all such rights have not been secured.

[ ] Yes, make my thesis available in the UA Campus Repository!

<table>
<thead>
<tr>
<th>Student signature:</th>
<th>Date: 4/30/13</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Thesis advisor signature:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date: 4-30-2013</th>
</tr>
</thead>
</table>

[ ] No, do not release my thesis to the UA Campus Repository.

<table>
<thead>
<tr>
<th>Student signature:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Last updated: 04/01/13
ABSTRACT

Children across the world are more obese than ever before in history.¹ Children are beginning to manifest weight-associated diseases previously only seen in adulthood.² With obese children likely remaining so into adulthood, the implications of childhood obesity are significant and long lasting.¹ This underscores the importance of understanding its development so that it may be reversed. One aspect that has paralleled the rise in obesity is a decline in sleep duration.³ Epidemiologic studies suggest short sleep might play a role in weight gain and the subsequent development of obesity among children and adolescents. Sleep deprivation is associated with hormonal alterations that favor appetite stimulation and dysregulation of glucose metabolism. Additionally, decreased physical activity is associated with chronic short sleepers. Through these mechanisms, chronic short sleep may be one aspect of the obesity epidemic that can be targeted. This paper will address the body of evidence regarding the relationship between short sleep and obesity in children and adolescents.
INTRODUCTION

Obesity has become an epidemic in the United States and across the world. According to a report by the CDC’s National Center for Health Statistics, 17% of children aged 2-19 are obese representing a three-fold increase in the last thirty years. There are major medical and psychosocial implications of childhood obesity. Medically, obesity is a risk factor for the development of numerous conditions including Type II diabetes, metabolic syndrome, sleep apnea, fatty liver disease, and polycystic ovary syndrome. Additionally, low self-esteem and negative body image may result in depression and decreased quality of life in obese individuals.

Obesity is a multifactorial disease with influences from genetics, diet, and lifestyle. The recent rise in obesity has correlated with an increasing trend toward sleep curtailment. Particularly across childhood and adolescence, there has been a significant shift toward later weekday bedtimes resulting in total nightly sleep declines. These changes in sleep patterns may be one causative factor of this epidemic. Previous literature reviews published from 2008 to 2012 reported significant associations between sleep duration and obesity through childhood and adolescence. In a 2008 meta-analysis encompassing data on 30,002 participants aged 2-20 years, a consistent increased risk of obesity was shown among children sleeping less than 10 hours per night. This was supported by a second meta-analysis of children and adolescents in which short sleepers were 58% more likely than long sleepers to be overweight. According to Patel and Hu, “both cross-sectional and cohort studies of children suggest short sleep duration is strongly and consistently associated with concurrent and future obesity.” While the cross-sectional studies have reliably shown an inverse relationship between sleep duration and greater BMI in children, some of the longitudinal studies have had mixed results where some found an association between short sleep and future weight gain while others did not. These differences will be explored in this review.

Younger people may be more sensitive to the physiological and behavioral mechanisms of weight gain associated with shortened sleep. This would account for the inconsistent results of studies conducted in adolescent and adult populations. It may also be that weight gain occurs at or near the onset of shortened sleep and
subsequently plateaus after a threshold weight is reached.\textsuperscript{10} This may contribute to a lack of association in longitudinal studies of older populations. If weight gain does plateau, BMI and adiposity measures at a follow-up period may not correlate to short sleep duration earlier in life since the critical period of weight gain following sleep curtailment might not be considered in the timeframe.\textsuperscript{10} Since the role of sleep seems more critical at younger ages, this paper will focus on studies involving children and adolescents.

The biological need for sleep during childhood changes during growth and development.\textsuperscript{16} According to the National Sleep Foundation, sleep need decreases as age increases in the following trends: infants from 3-11 months old require 14-15 hours; toddlers from 1-3 years old require 12-14 hours; preschoolers from 3-5 years old require 11-13 hours; school-age children from 5-10 years old require 10-11 hours; and adolescents from 10-17 years old require 8.5-9.25 hours.\textsuperscript{17} These ranges represent the average amount of sleep required for most children that age; however, there is some variation within these ranges as some children need more sleep while others need less.\textsuperscript{17} At younger ages, naps are required to achieve the greater amount of recommended sleep. Nighttime sleep consolidates during the first year of life, and a significant decline in napping occurs between 1.5 years and 4 years of age.\textsuperscript{16} Thus, it is appropriate to use total sleep time (TST) in a 24-hour period when reporting sleep for younger populations from infancy to preschool age. By school age, children’s sleep has consolidated so that it is achieved solely at night.\textsuperscript{16} From this point on, total nightly sleep duration (SD) is a more appropriate measure since “napping does not appear to be a substitute for nighttime sleep in terms of obesity prevention.”\textsuperscript{19} Despite the National Sleep Foundation’s guidelines, there is no clear consensus for the length of short sleep used in the studies for the various age groups.

Sleep was determined both subjectively and objectively by the studies included in this review. In the subjective measurements, sleep data was self-reported or reported by the parents of the children through various means.\textsuperscript{4, 19-78} Questionnaires ranged from a single question, such as “How many hours of sleep do you usually get a night”\textsuperscript{22} to standardized questionnaires with multiple questions such as the validated School Sleep Habits Survey and the Pittsburg Sleep Quality Index.\textsuperscript{60, 79} Questionnaires were
conducted in person, over the phone, or though written forms. Sleep diaries were used in several studies in which the subjects recorded bedtimes and wakeup times, from which SD was calculated. Objective sleep measurements were obtained through two methods: actigraphy and polysomnography. Actigraphy measures gross motor activity in which periods of continuous inactivity are equated with sleep. In this method, wrist accelerometers were used to measure sleep in the study participants for 1-7 days. Polysomnography (PSG), by far the most accurate and expensive method of sleep determination, was used to define sleep architecture in addition to SD. This method can be used to measure sleep efficiency and rapid eye movement (REM) density.

Several anthropometric measurements were reported in the studies. Body mass index z-scores (BMI) are calculated from height and weight then adjusted for sex and age on growth charts. BMI was considered in all of the studies. The heights and weights were taken by trained professionals or self-reported by the study participants or their parents. The CDC’s Expert committee defines childhood overweight as a BMI between the 85th and 95th percentiles on growth charts. Children with a BMI greater than the 95th percentile are considered obese. These parameters for overweight and obesity were implemented in 2007 to replace the definitions provided by the 1998 expert committee which classified the 85th-95th percentiles as “risk of overweight” and greater than the 95th percentile as overweight. Consequently, older studies use the term “overweight” where more recent studies use “obesity.” In the longitudinal studies, changes in body weight or BMI were also measured. Additional anthropometric measurements often considered include circumference at the hip, waist (WC), and mid-arm and skinfold thickness at various landmarks (e.g. triceps and subscapular). Body fat composition was determined through dual-energy X-ray absorptiometry (DEXA) and bioelectrical impedance.

CROSS-SECTIONAL STUDIES

Observational studies have been conducted to look at the general association between SD and body weight in various childhood populations around the world. The general consensus of these cross-sectional studies is a significant negative association
between SD and body weight. Table 1 summarizes the findings of several cross-sectional studies focused on in this review.

In all but eleven of the studies, an inverse relationship between SD or TST was observed. In the Toyama Study conducted on 3 year old children, a dose-response relationship was found where children sleeping less than 9 hours were 57% more likely to be obese, 9-10 hours were 34% more likely to be obese, and 10-11 hours were 20% more likely to be obese when compared to children sleeping 11 hours or more a night. This relationship was seen in older populations as well, but at shorter sleep durations. In study of 14-18 year old high school students, the age and gender adjusted odds ratio of overweight was 8.53 for sleeping less than 5 hours, 2.79 for 5-6 hours, 2.81 for 6-7 hours, and 1.29 for 7-8 hours when compared to students sleeping more than 8 hours per night. In another study of 16-19 year olds, adolescents sleeping less than 7 hours per night were more likely to have a higher BMI, percent body fat, total body fat mass, and fat mass index as assessed using bioelectrical impedance. This study chose a single value to define short sleep. This value varied from study to study. For example, short sleep was defined as sleeping less than 8 hours in a study of 12-17 year olds. Despite the more inclusive definition, it was still found that short SD was associated with increased odds of being overweight or obese.

In the Quebec Adiposity and Lifestyle Investigation in Youth study of children aged 8-10, U-shaped relationships between SD and BMI, WC, and percent body fat were found after adjusting for age, sex, pubertal status, parental education, family income, and parental BMI. In this study, sleep was measured with actigraphy over the course of 7 days, and sleep was defined as short when less than 10 hours and long when 12 or more hours. Diet and physical activity levels were also considered. While this is one of the few studies to find a U-shaped relationship, the fact that many studies analyzed two groups (short sleepers and adequate sleepers) means that U-shaped relationships could be present but not detected.

Two studies used PSG for determining sleep characteristics. In one of the studies including 335 participants aged 7-17 years, PSG was measured for three consecutive nights, and it was found that shorter TST, reduced REM sleep, and
reduced sleep efficiency were all associated with being overweight after adjusting for demographics, pubertal status, and psychiatric diagnosis. It was suggested by the authors that the reduction in REM sleep, more so than TST, might result in endocrine alterations that lead to the development of obesity. In a case-control PSG study, SD was not significantly different between obese and healthy weight participants when measured with one night of PSG. This study also utilized actigraphy on weeknights and weekends and found that the obese group slept significantly less on the weekends. Since only one night of PSG was recorded and PSG requires a non-normative sleep environment by nature, the lack of association between SD measured by PSG and obesity in this study should not be given much weight.

Some studies only found significant relationships in certain age sub-groups of their study population. In a study by Shi with children aged 5-15, an inverse association was found in 5-13 year olds while no association was found in the 13-15 year old group. In this study, short SD was defined as less than 9 hours across both groups. Consequently, the fact that no association was observed among 13-15 year olds could be explained by the study’s definition of short sleep, since 8.5-9.25 hours of sleep is the recommendation by the National Sleep Foundation for this age group. Therefore, 13-15 year olds that slept 8.5-9 hours would get adequate sleep but be included with the short sleepers. Age differences were also reported in a study of German children in which the participants were separated into a 3-10 year old group and a 10-17 year old group. Sleep was negatively associated with obesity in the younger group while no association was seen in the older group. One possible explanation for this variation is that sleep was reported using different methods. In this study, the younger age group reported sleep as the average TST per day; whereas, the older age group reported SD from the previous night. As a result, directly comparing the sleep outcomes may be inappropriate.

Significant gender differences were reported in seven studies to the extent that associations were observed in one sex but not the other. Several studies found significant associations among females but not males. In two adolescent studies, U-shaped associations were found only in females. In a study of 10-20 year old adolescent twins, sleeping less than 8 hours and more than 9 hours was
found to be associated with greater adiposity and higher BMI in females. Total adipose, as well as central adiposity, was more strongly associated with SD than the association of BMI with total sleep. In another study, average school night SD less than or equal to 4 hours and greater than 9 hours was associated with a higher likelihood of obesity among 13-18 year old female students. Inverse relationships were found in a few studies among girls only. In a study of students aged 12-13 years, sleep shorter than 7 hours was found to be significantly associated with overweight in females. In another study with 6-20 year olds stratified by age, SD was inversely associated with BMI in both genders, but when using the study’s defined short sleep (<10 hours for ages 6-10 and <9 hours ages 10-20), the association between short sleep and a higher risk of overweight/obesity only held statistical significance in girls. A similar outcome was seen in a study with 3 year olds in which girls sleeping less than 9 hours were more likely to be obese. These outcomes may be influenced by the definition of short sleep used. It has been theorized that boys are more susceptible to weight gain due to sleep deprivation through mechanisms that have not yet been determined. From an evolutionary standpoint, “girls may be biologically more resilient to environmental stressors and may require greater sleep deprivation to be affected negatively in comparison to boys.” If this is the case, shortened sleep may be affecting boys at a SD longer than the cut-off values used for short SD in these studies.

Three studies found inverse associations in males alone. In a study of adolescents in grades 7-12, each additional hour of sleep in males was associated with a 0.08 decrease in BMI in a linear regression, and logistic regression reported that each additional hour of sleep was associated with a 10% reduced risk of being overweight. No significant association was found in females using either form of regression. In another study, overweight, WC, and SD were studied a sample of 7-15 year olds. In boys, it was found that SD had a inverse graded relationship with WC, and sleeping less than 8 hours had 3.1 times greater odds of overweight than boys sleeping 10 or more hours. In girls, a post hoc analysis showed a significant association between a SD of 8-9 hours and mean BMI and WC when compared to girls sleeping 9 or more hours, but the overall pattern was not as clear as what was seen in boys. In a third study, a clear inverse relationship was found in boys but not girls. If the authors tested for a U-
shaped relationship in girls as opposed to an inverse relationship as was found in boys, statistical significance might have been reached. This is supported by the study’s findings that girls sleeping less than 9 hours and more than 10 hours were more likely to be overweight. 40

In the adolescent studies reporting variations between genders, the onset of puberty may play an important role in the differences.23, 31, 47, 48, 63 Knutson suggested that the gender differences might stem from physiological changes during puberty.48 According to Sun and colleagues, the changes in body composition during puberty, in which males experience increases in muscle mass and decreases in fat mass while females experience an increases in fat mass, may explain why sleep duration impacts weight gain in males and females differently.47 Interestingly, studies that found associations only in boys or only in girls both cite changes stemming from puberty as a possible cause of the difference. This highlights the lack of comprehensive understanding of the biological mechanisms linking puberty, sleep, and body weight.

Several studies have looked into the differences between SD during the school week and over the weekend in school-age children. School start times necessitate earlier wake-up times on school days resulting in a shorter SD if bedtimes remain the same. Studies have shown that normal weight children are more likely to experience catch-up sleep on weekends and holidays than overweight and obese children.57, 79, 90, 91 These studies concluded that compensatory weekend sleep is independently associated with a decreased risk of overweight and obesity.57, 60, 90, 91 Furthermore, in a study including 4-10 year olds, obese children were shown to have greater night-to-night sleep variances when measured using a week of actigraphy.90 There are also gender differences in the sleep/wake cycles on weekdays and weekends. Boys have been shown to wake up earlier and sleep less in general when given the opportunity for compensatory sleep.79

LONGITUDINAL STUDIES

The role of short sleep on weight changes later in life has been explored in longitudinal studies. Table 2 summarizes eleven of these studies in which SD or TST at a baseline age and BMI after a follow-up period were looked at for associations. A wide
variety of factors were also considered at both ends of the analysis to help determine causality.

Inverse associations were seen in several of the longitudinal studies.\textsuperscript{4, 20, 33, 64-68, 70, 72-74, 85, 86, 92, 93} Many of these studies were prospective cohorts in which the children were followed from birth for as few as 6 months to as many as 32 years.\textsuperscript{19-21, 64, 65, 68, 71, 73, 86, 92, 93} In a New Zealand birth cohort of 1,037 participants, shorter sleep times at ages 5, 7, 9, and 11 years old, which were averaged to create a composite mean SD during childhood, were associated with higher BMI at age 32.\textsuperscript{65} This study, with the longest follow-up duration to date, adjusted for adult sleep time and several confounding factors including early childhood BMI, sex, socioeconomic status, parental BMI, television viewing, smoking, parental control, and adult physical activity.\textsuperscript{65}

In a study of children aged 3-12 at baseline, children who slept 10-11 hours of time diary sleep had a lower BMI after a five-year follow-up.\textsuperscript{4} When the children were stratified into age subgroups, the authors concluded that “sleep matters more for younger children's BMI and overweight status than older children's BMI and overweight status” independent of gender.\textsuperscript{94} While this study still found an association in the older age subgroup, three other studies found no association when following adolescent populations.\textsuperscript{39, 41, 76} In a study of adolescents from aged 10-16 at baseline and followed for two years, there was no longitudinal association between sleep and changes in BMI or percent body fat.\textsuperscript{76} In the National Longitudinal Study of Adolescent Health, a survey of 13,568 adolescents aged 12-18 at baseline, there was no association between getting less than 6 hours of sleep and obesity after a 1-2 year follow-up.\textsuperscript{41} In the follow-up survey of this study, SD was not even associated with increased BMI cross-sectionally.\textsuperscript{41} The authors speculated that onset of puberty may play a role in these results.\textsuperscript{41} This study also used a single, self-reported question to determine SD, which makes the results less conclusive.

Significant age differences were seen in two of the studies that looked at children and adolescents.\textsuperscript{19, 39} In one study, children 0-13 years old at baseline were divided into two groups: 0-4 years old and 5-13 years old.\textsuperscript{19} An inverse association between SD and BMI was shown after a five-year follow-up in the younger group; whereas, there was no association between SD and BMI among 5-13 years olds over the same five-year
follow-up period. In another study, 474 first graders (7.3 years old) and 1,030 fourth graders (10 years old) in the Obesity and Metabolic Disorders Cohort in Childhood registry were surveyed after a two-year period. Shorter sleep among the first graders was a predictor of high BMI after two years, while there was no association between SD in 4th graders and their future BMI. Short sleep was, however, associated with higher BMI in a cross-sectional analysis of the older population. These findings support the supposition that sleep duration is more important at younger ages.

Only two studies found no longitudinal association in populations 11 years old or younger. In 3,844 infants who were followed every two years for a total of six years, sleep duration earlier in life was not associated with BMI at 6-7 years. In fact, the sleep durations remained similar across BMI categories cross-sectionally until 6-7 years old, when obese children were found to sleep 30 minutes less than their counterparts. This study took multiple measurements of sleep using parental diaries and adjusted for baseline weight when sleep was measured. The authors concluded that there might be an unaccounted for factor such as sleep apnea that influences weight at older ages which in turn decreases sleep duration. In the Early Childhood Longitudinal Study, approximately 11,400 children were followed from kindergarten to fifth grade. While it was found that each additional hour of sleep was associated with a 0.06 decrease in initial BMI, sleep was not associated with changes in BMI over time.

Gender was a significant modifying factor in only one of the longitudinal studies. In this birth cohort of 1,015 children, the average SD from ages 2.5-6 years old was surveyed annually. Among boys only, sleeping less than 10 hours per night earlier in life was associated with a two fold increase in the odds of being obese at 6 years old when compared to boys sleeping more than 11 hours per night. This association was found after controlling for a broad range of factors including the child’s characteristics, parental characteristics, family demographics, and socioeconomic characteristics. The authors suggested that the gender differences might stem from poorer overall sleep quality in boys as well as other unknown behavioral and biological mechanisms.
POTENTIAL MECHANISMS

There have been several mechanisms proposed to explain the relationship between shortened sleep and obesity. Three pathways have been posited to explain how inadequate sleep amount may lead to increased body weight: increases in appetite, alterations in glucose metabolism, and decreases in energy expenditure. Three pathways have been posited to explain how inadequate sleep amount may lead to increased body weight: increases in appetite, alterations in glucose metabolism, and decreases in energy expenditure.94

Shortened sleep is often accompanied by changes in appetite regulating hormones. Sleep deprivation results in decreased leptin levels and elevated ghrelin levels.95 Leptin is an appetite-reducing hormone secreted by white adipose tissue postprandially and at night.94 Decreases in leptin result from increased sympathetic nervous activity accompanying sleep curtailment.96 Ghrelin is an appetite stimulating hormone produced in the stomach that is present in high levels during fasting states and when sleeping.94 Interestingly, this hormone is generally higher in shorter sleepers even though it is produced during sleep.94 Ghrelin prolongs postprandial glucose responses and stimulates the release of growth hormone.94 The consequence of these combined hormonal alterations is an increase in appetite resulting in increased caloric intake.

Orexins are neuropeptides that also increase with decreased sleep and serve to up-regulate appetite.94 The orexin system is stimulated by ghrelin and inhibited by leptin.94 Consequently, increases in ghrelin and decreases in leptin, occurrences associated with shortened sleep, trigger the orexin system, further intensifying appetite.94

Several studies have observed dietary intake in relation to SD. Landis and colleagues demonstrated that food cravings were more prevalent in those with less nocturnal sleep,97 while Tatone-Tokuda and colleagues found that children with short sleeping patterns from 2.5-6 years were less likely to have healthy dietary intakes at 6 years old.71 Short sleeping children were also less likely to eat fruits and vegetables, boys were more likely to eat meats, and girls were more likely to drink soda than their longer-sleeping counterparts.71 In an adolescent study using 8 hours of average weekday sleep as a critical value, short sleepers consumed more calories from fat and less calories from carbohydrates after adjusting for confounding factors.82 These small changes in feeding behavior may cumulatively contribute to abnormal weight gain and consequent increases in BMI and adiposity.
Glucose metabolism may also be affected by short sleep. Glucose intolerance as a result of insulin insensitivity is negatively associated with SD.\textsuperscript{74} Cortisol and growth hormone levels, which are influenced by sleep, are insulin counter-regulatory hormones that modulate the “circadian rhythms of glucose metabolism.”\textsuperscript{94} Dysregulation of glucose metabolism may result in weight gain as a result of insulin resistance, elevated GH, and elevated cortisol. The studies measuring hormonal changes as a byproduct of sleep deprivation have almost exclusively been conducted in adults.\textsuperscript{94} The two studies in children and adolescents have found less clear associations.\textsuperscript{43,98} The study conducted by Hitze and colleagues in children and adolescents did not find an association between short sleep and glucose metabolism other than suggesting that insulin resistance resulted from overweight status.\textsuperscript{43} These findings were supported another study in 6-12 year olds; however, it was found that shorter SD may be associated with early metabolic abnormalities via increases in fasting C-peptide.\textsuperscript{98}

Decreased energy expenditure might result from inadequate sleep. Shortened sleep results in tiredness and fatigue, which in turn decreases daytime physical activity and exercise choices.\textsuperscript{13} In addition to reduced physical activity, non-exercise activity thermogenesis may be negatively impacted by reduced sleep.\textsuperscript{99} This would increase the likelihood of positive energy balances resulting in weight gain.

**LIMITATIONS IN SLEEP DETERMINATION**

Most studies determined sleep in children or adolescents with parental questionnaires. While this method may be the easiest and most cost effective, accuracy may be hindered by poor recall and likely overestimates sleep duration. When parents report their child’s sleep, bedtime may be used in the place of actual sleep time. The same might be true of the time that the child gets out of bed in relation to the actual wake-up time. If parents were to report sleep in this manner, a systematic overestimation of sleep duration would be expected to skew the results.

In adolescent populations, SD was frequently self-reported. Participants reporting SD tend to underestimate their actual sleep possibly due to social pressures to “do more and sleep less.”\textsuperscript{4} Self-reported sleep may also reflect quality of sleep, psychosocial factors, perceived sleep, and societal influences on appropriate sleep.
lengths as opposed to actual sleep duration. In 2007, Knutson compared sleep from time diaries to self-reported sleep in 10-17 year old adolescents and found a weak correlation between the two. Diary sleep was more than an hour longer than self-reported sleep; however, only two random days were chosen for the diary in this study, and non-compliance becomes an issue when used for longer durations. Also, in a time diary, wakefulness during the night and length of time falling asleep may not be accounted for. A longer study would better elucidate the accuracy of sleep diaries and the correlation between average sleep and diary sleep. Nevertheless, this study draws into question the validity of using subjective measures of sleep determination in the studies.

The number and type of questions asked in questionnaires might also influence the sleep durations obtained. Questions such as “how many hours a night do you usually sleep a night” or “how many hours of sleep did you get last night” may be less accurate than obtaining reported bedtimes and wakeup times. Recall biases play a more significant role when only a single question is asked.

Many of the studies used the previous night of sleep to represent the person’s normal sleeping patterns. By only using one day of data, results may not be indicative of normal sleeping durations. As mentioned earlier, school-age individuals sleep exhibit different sleep patterns during the week as opposed to the weekend. Consequently, the day of the week and the time of the year that the study was conducted may play a significant role in the results.

While objective measures of sleep such as actigraphy and PSG are considered to be the “gold standard,” both methods have limitations. In actigraphy, there may be a non-compliance bias, in which obese or unhealthy individuals are less likely to comply. This would be easier to address than the problems associated with subjective measures. PSG is the only method for determining type of sleep, but it is expensive and not conducive to long-term measurements. Due to the intrusive nature of the PSG, sleep measurements may not be representative of normal sleep patterns. Despite these limitations, objective sleep measures are still more thorough, and actigraphy should be considered when accurate sleep durations are required.
ADDITIONAL LIMITATIONS

A majority of the studies relied on parental or self-reported measures of height and weight in order to calculate BMI. Without validated measurements, these values may not provide a realistic picture of actual BMI, as parents may estimate heights and weights or use previously measured values instead of taking new measurements. Even if new measurements are taken, the quality of scale and tape measure used was well as the precision taken when measuring height may result in inaccurate values.

In many of the studies, BMI was the only anthropometric measurement used. BMI provides part of the picture of obesity, but not all weight gain should be treated equally. While it is convenient to use BMI as a surrogate of body fatness, it is limited in that BMI does not contain a direct measure of adiposity. This is particularly important, as it is the accumulation of fat mass, particularly in the abdominal region, in obesity that is associated with many of its comorbidities. In children, the relationship between BMI, percent fat composition, and percent fat-free body composition are complicated by differences in growth rates and maturity level, as BMI is adjusted for chronological age instead of biological age. The limitations of BMI are particularly apparent during puberty as “BMI may not be an equally valid marker of adiposity for both sexes, and this may play a role in the observed sex difference in the association between sleep and BMI.” Increases in BMI throughout childhood are generally driven by increases in fat free mass in both genders. This occurs up until adolescence, after which higher BMI in girls is a result of increases in total body fat.

The use of chronological age as opposed to biological age is another limitation in many of the studies. By not considering Tanner Stage of development, many of the studies do not take into account that the onset of puberty occurs about two years earlier in girls than in boys. There is also a high degree of variation in the age of onset within each gender. By not considering the body compositional changes associated with puberty, a potential lurking variable is introduced into the results.

Since obesity is a multifactorial disease, it is important to adjust for confounding factors when trying to determine an association between sleep and obesity. Each of the studies had varying inclusion and exclusion parameters and adjusted for a wide variety of factors in various combinations. Due to the lack of uniformity across the studies, the
results are not entirely comparable. Essential confounding factors appear to be age, gender, and pubertal status. While feeding behavior and physical activity are two of the proposed mechanisms by which sleep may impact weight, independently adjusting for these factors allows for a clearer picture of the sleep-obesity relationship. Additionally, when choosing study participants or correcting for factors in large studies, race, socioeconomic status, health status, sleep disordered breathing, medication use, parental education, parental BMI, electronic usage, and sedentary behaviors are important to take into account. With longitudinal studies it is also important to consider initial BMI or other adiposity measurements at baseline.

Very large study populations are most able to accommodate all of the factors that have to be taken into account while still achieving statistically significant results. If a large study is not financially feasible, a study design with a very selective population that inherently adjusts for said factors is the next best choice. While more global conclusions cannot be made with these studies, the results within the population can be more conclusive regarding the role of sleep in the development of obesity.

**CONCLUSION**

The mounting body of evidence supports an association between shortened sleep and higher body weight in children. Despite the variability in defining short sleep and methods of measuring it, almost all of the cross-sectional studies have found a negative correlation while using a wide variety of covariates. Unfortunately, the direction of causality cannot be determined because the extent of causation, common responses, and confounding factors cannot be determined through cross-sectional studies alone. When considering the confounding factors between sleep and obesity, they can be explanatory, mechanistic, or lurking variables. It is likely that the relationship between sleep duration and obesity is bimodal, with short sleep leading to obesity through various mechanisms and obesity leading to shortened sleep though obstructive sleep apnea and other medical and somatic issues. By using prospective randomized controlled studies, causality can be addressed most effectively so that the mediating and moderating factors can be identified and explored. Future prospective studies
should be conducted using the most objective sleep measures such as actigraphy and direct measurement of adiposity.

Emphasis should be placed on understanding the mechanisms by which sleep impacts obesity particularly in regard the role of gender and puberty. The exact role of gender should be explored further with a focus on determining the mechanisms by which differences occur. The variable onset of puberty in the study populations may also play a significant role. Future studies should consider the differences between weekend and weekday sleep when studying these populations. If these studies continue to show that inadequate sleep leads to weight gain, sleep can be considered a modifiable risk factor for obesity. If shortened sleep is not just associated with but causative of higher body weight in children, sleep interventions focusing on obtaining adequate sleep early in life can be implemented to help reverse the childhood obesity epidemic and its lifelong implications.
<table>
<thead>
<tr>
<th>First author</th>
<th>Year</th>
<th>Study Population</th>
<th>Age Range</th>
<th>Methods to Measure Sleep</th>
<th>Short Sleep Definition</th>
<th>Main Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beebe, D.</td>
<td>2007</td>
<td>82 US adolescents; 60 from a weight management clinic (67% females) and 22 healthy controls (64% females)</td>
<td>10-16.9 years</td>
<td>PSG, actigraphy, and questionnaire</td>
<td>Not defined</td>
<td>The overweight participants and the healthy controls had no differences in SD relationship via PSG or questionnaire. Overweight adolescents slept significantly less (p=.001) on non-school nights in the multivariate actigraphy findings.</td>
</tr>
<tr>
<td>Chaput, J</td>
<td>2011</td>
<td>550 children in the Quebec Adiposity and Lifestyle Investigation in Youth (QUALITY) study (299 males and 251 females)</td>
<td>8-10 years</td>
<td>Actigraphy</td>
<td>&lt;10</td>
<td>Short SD is associated with overweight and obesity with an OR of 2.05 when adjusted for caloric intake, physical activity, age, sex, Tanner stage, parental education, family income, and parental BMI.</td>
</tr>
<tr>
<td>Eisenmann, J</td>
<td>2006</td>
<td>6,324 adolescents from the Australian Health and Fitness Survey (3,203 males and 3,121 females)</td>
<td>11-16.5 years</td>
<td>Questionnaire</td>
<td>&lt;8</td>
<td>In boys, SD had an inverse graded relationship with WC, and a SD less than 8 hours had an OR of 3.1 for overweight compared to boys sleeping 10 or more hours. No clear pattern was seen in girls.</td>
</tr>
<tr>
<td>Kleiser, C</td>
<td>2009</td>
<td>10,021 participants in KiGGS survey</td>
<td>3-17 years</td>
<td>Questionnaire</td>
<td>Not defined</td>
<td>Sleep was negatively associated with obesity among 3-10 year olds. In the same multivariate analysis, no association was found in 10-17 year olds.</td>
</tr>
<tr>
<td>Knutson, K</td>
<td>2005</td>
<td>4,486 adolescents in the National Longitudinal Study of Adolescent Health (2,199 males and 2,289 females)</td>
<td>16.7 years</td>
<td>Questionnaire</td>
<td>Not defined</td>
<td>SD was negatively associated with overweight in a linear and logistic regression in males. No significant association was found in females using either form of regression.</td>
</tr>
<tr>
<td>Knutson, K</td>
<td>2007</td>
<td>1,546 adolescents from the Child Development Supplement of the Panel of Income Dynamics (767 males and 779 females)</td>
<td>10-19 years</td>
<td>Questionnaire and sleep diary (2 days)</td>
<td>Not defined</td>
<td>SD from the sleep diary was not significantly associated with overweight. Self-reported questionnaire SD was associated with overweight, but the association was not linear. Sleep diary values were &lt;1 longer than questionnaire sleep.</td>
</tr>
<tr>
<td>Liu, X</td>
<td>2008</td>
<td>335 children and adolescents (55.2% male)</td>
<td>7-17 years</td>
<td>PSG</td>
<td>Not defined</td>
<td>Shorter TST, reduced REM sleep, and reduced sleep efficiency were all associated with overweight. One hour of less TST had an adjusted OR of 1.85, and one hour less of REM sleep had an OR of 2.91. Reduced REM sleep may play a more important role than shorter TST.</td>
</tr>
<tr>
<td>Lowry, R</td>
<td>2012</td>
<td>30,451 adolescents in the Youth Risk Behavior Survey (51.4% male)</td>
<td>13-18 years</td>
<td>Questionnaire</td>
<td>≤8</td>
<td>SD ≤4 and &lt;9 was associated with increased likelihood of obesity in females. There was no significant association between obesity and SD in males.</td>
</tr>
<tr>
<td>Morley, B</td>
<td>2012</td>
<td>12,188 adolescents from the National Secondary Students' Diet and Activity survey (53% male)</td>
<td>12-17 years</td>
<td>Questionnaire</td>
<td>≤8</td>
<td>Short SD on a school night was associated with increased odds of 1.22 of being overweight or obese.</td>
</tr>
</tbody>
</table>

(Continued on next page)
<table>
<thead>
<tr>
<th>1st author</th>
<th>Year</th>
<th>Study Population</th>
<th>Age Range</th>
<th>Methods to Measure Sleep</th>
<th>Short Sleep Definition</th>
<th>Main Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ochiai, H.</td>
<td>2012</td>
<td>3,433 children from the Saitama Prefecture, Japan (1,781 males and 1,652 females)</td>
<td>9-10 years</td>
<td>Questionnaire</td>
<td>&lt;10</td>
<td>There was an inverse relationship between SD and overweight in boys. There was no significant inverse association in girls, but girls sleeping &lt;9 hours or ≥10 were more likely to be overweight.</td>
</tr>
<tr>
<td>Seicean, A.</td>
<td>2007</td>
<td>529 adolescents from Bay High School, Bay Village, OH, USA</td>
<td>14-18 years</td>
<td>Questionnaire</td>
<td>≤8</td>
<td>There was an inverse (dose-response) relationship between SD and overweight. The age and gender adjusted OR of overweight was 8.53 for sleeping &lt;5 hours, 2.79 for 5-6 hours, 2.81 for 6-7 hours, and 1.29 for 7-8 hours when compared to students sleeping more than 8 hours per night.</td>
</tr>
<tr>
<td>Sekine, M.</td>
<td>2002</td>
<td>8,941 children in the Toyama Birth Cohort study (4,590 males and 4,351 females)</td>
<td>3 years</td>
<td>Questionnaire</td>
<td>≤11</td>
<td>There was a dose-response relationship between short sleeping hours and obesity was found. The adjusted OR for obesity was 1.57 for sleeping &lt;9 hours, 1.34 for 9-10 hours, 1.20 for 10-11 hours, and when compared to children sleeping 11 or more hours per night.</td>
</tr>
<tr>
<td>Sekine, M.</td>
<td>2002</td>
<td>8,274 children Toyama Birth Cohort study (4,194 males and 4,080 females)</td>
<td>6-7 years</td>
<td>Questionnaire</td>
<td>≤10</td>
<td>There was a dose-response relationship between short sleeping hours and obesity. The adjusted OR of obesity was 2.87 for sleeping &lt;8 hours, 1.89 for 8-9 hours, and 1.49 for 9-10 hours when compared to those sleeping 10 or more hours per night.</td>
</tr>
<tr>
<td>Shaikh, W.</td>
<td>2009</td>
<td>489 Gujarati Indian adolescents (297 males and 192 females)</td>
<td>16-19 years</td>
<td>Questionnaire</td>
<td>&lt;7</td>
<td>BMI, percent body fat, fat mass, and fat mass index were significantly lower in adolescents getting an average of ≥7 hours of sleep a night for the previous year.</td>
</tr>
<tr>
<td>Shi, Z.</td>
<td>2010</td>
<td>3,495 children and adolescents from the South Australian Monitoring and Surveillance System (50.3% boys)</td>
<td>5-15 years</td>
<td>Questionnaire</td>
<td>&lt;9</td>
<td>Short SD is associated with increased obesity in children 5-13. No significant association between short SD and obesity was found in children aged 13-15.</td>
</tr>
<tr>
<td>Sugimori, H.</td>
<td>1997</td>
<td>351 children in the Toyama Study; 117 obese children (66.4% males) and 234 controls (50.4% males)</td>
<td>3 years</td>
<td>Questionnaire</td>
<td>≤9</td>
<td>In a matched pair analysis, short SD in girls correlated to an increased risk of obesity (OR=2.24). No association was found in boys.</td>
</tr>
<tr>
<td>Sun, Y.</td>
<td>2009</td>
<td>5,753 students from the Toyama prefecture (2,842 male and 2,911 female)</td>
<td>12-13 years</td>
<td>Questionnaire</td>
<td>&lt;7</td>
<td>Short SD in girls correlated to an increased risk of overweight (OR= 1.81). No association was found in boys.</td>
</tr>
<tr>
<td>Yu, Y.</td>
<td>2007</td>
<td>500 Chinese twins (273 males and 227 females)</td>
<td>10-20 years</td>
<td>Questionnaire and sleep diary (7 days)</td>
<td>&lt;8</td>
<td>SD of &lt;8 and ≥9 was associated with higher adiposity measures in females after adjusting for covariates. The association was stronger for truncal fat and waist circumference than for BMI. No consistent association was found in males. The questionnaire and diary reported the same SD, statistically.</td>
</tr>
<tr>
<td>1st author</td>
<td>Year</td>
<td>Study Participants</td>
<td>Follow-up</td>
<td>Main Conclusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>------</td>
<td>---------------------</td>
<td>-----------</td>
<td>----------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Agras, W.64</td>
<td>2004</td>
<td>150 children from a San Francisco Bay Area Birth cohort (74 males and 76 females)</td>
<td>0 years</td>
<td>Less TST at 3-4 years old was a risk factor for overweight at 9.5 years in a significant negative relationship. Children who become overweight at 9.5 years slept 30 minutes less.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bell, J.19</td>
<td>2010</td>
<td>1,930 children from Panel Survey of Income Dynamics Child Development Supplements; 822 0-4 years (48% female) and 1,108 5-13 years (50% female)</td>
<td>0-13 years</td>
<td>Shorter SD at baseline was strongly associated with increased risk of subsequent overweight or obesity (OR=1.80). No association was found at 5-13 years at baseline in the follow-up.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calamaro, C.41</td>
<td>2010</td>
<td>13,568 adolescents from the National Longitudinal Study of Adolescent Health survey (6,612 males and 6,956 females)</td>
<td>12-18 years</td>
<td>No statistically significant longitudinal relationship was found between short SD at baseline and risk of obesity after adjusting for BMI at baseline, age, gender, race, and parental income.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hiscock, H.21</td>
<td>2011</td>
<td>7,201 infants and children; 3,357 infants (51% male) and 3,844 children (52% male) in the Longitudinal Study of Australian Children</td>
<td>0-1 year and 4-5 years</td>
<td>No statistically significant longitudinal relationship was found between TST at baseline and risk of obesity. The only cross-sectional association was in 6-7 year olds who slept ~30 min less than their normal weight counterparts.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Landhuis, C.65</td>
<td>2008</td>
<td>1,037 children in the Dunedin Multidisciplinary Health and Development Study (535 males and 502 females)</td>
<td>5 years</td>
<td>Shorter average SD (using SD from 5, 7, 9, and 11 years) was associated with higher adult BMI values in a sex-adjusted linear regression controlling for early BMI, childhood SES, parental BMI, television viewing, physical activity, and moderate BMI in 1st graders in the two year follow-up.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lee, H.39</td>
<td>2012</td>
<td>1,504 children in the Obesity and Metabolic Disorders Cohort in Childhood registry; 474 1st graders (48% male) and 1,030 4th graders (49% male)</td>
<td>7.3 years and 10 years</td>
<td>Short SD predicted increased BMI in 1st graders in the two year follow-up. No longitudinal association was found in 4th graders.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lytle, L.76</td>
<td>2012</td>
<td>648 adolescents from the IDEA study and the ECHO study (320 males and 328 females)</td>
<td>10-16 years</td>
<td>No association was found between changes in SD at baseline and changes in BMI and percent body fat in the follow-up. Greater hours of sleep by children was significantly associated with lower BMIs in 1st graders.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miller, D.69</td>
<td>2011</td>
<td>11,400 children in the Early Childhood Longitudinal Study (49% male) and the ECHO study (48% male) and 1,105 children aged 3-4 years</td>
<td>4-6 y</td>
<td>No association was found between changes in SD at baseline and changes in BMI in the follow-up. Greater hours of sleep by children was significantly associated with lower BMIs in 1st graders.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snell, E.4</td>
<td>2007</td>
<td>1,414 children from the IDEA study</td>
<td>3-12 years</td>
<td>No association was found between changes in SD at baseline and changes in BMI in the follow-up. Greater hours of sleep by children was significantly associated with lower BMIs in 1st graders.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tatone-Tokuda, F.71</td>
<td>2008</td>
<td>1,015 children in the Quebec Longitudinal Study of Child Development (47% male)</td>
<td>0 years</td>
<td>No association was found between changes in SD at baseline and changes in BMI in the follow-up. Greater hours of sleep by children was significantly associated with lower BMIs in 1st graders.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Touchette, E.68</td>
<td>2008</td>
<td>1,138 children in the Quebec Longitudinal Study of Child Development (47% male)</td>
<td>0 years</td>
<td>No association was found between changes in SD at baseline and changes in BMI in the follow-up. Greater hours of sleep by children was significantly associated with lower BMIs in 1st graders.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
REFERENCES


10. Magee, C. A.; Caputi, P.; Iverson, D. C., Short sleep mediates the association between long work hours and increased body mass index. J Behav Med 2011, 34, 83-91.


53. Taylor, A. W.; Winefield, H.; Kettler, L.; Roberts, R.; Gill, T. K., A population study of 5 to 15 year olds: full time maternal employment not associated with high BMI. The importance of


Sherriff, A.; Avon Longitudinal Study of, P.; Children Study, T., Early life risk factors for obesity

67. Rutters, F.; Gerver, W. J.; Nieuwenhuizen, A. G.; Verhoef, S. P.; Westerterp-Plantenga,
M. S., Sleep duration and body-weight development during puberty in a Dutch children cohort.
Int J Obes (Lond) 2010, 34, 1508-14.

68. Touchette, E.; Petit, D.; Tremblay, R. E.; Boivin, M.; Falissard, B.; Genolini, C.;
Montplaisir, J. Y., Associations between sleep duration patterns and overweight/obesity at age 6.

69. Miller, D. P., Associations between the home and school environments and child body

Kagamimori, S., Analysis of factors that influence body mass index from ages 3 to 6 years: A

Montplaisir, J. Y., Sex differences in the association between sleep duration, diet and body

Touchette, E., Short sleep duration and body mass index: a prospective longitudinal study in
preadolescence. In Am J Epidemiol, The Author 2011. Published by Oxford University Press on
behalf of the Johns Hopkins Bloomberg School of Public Health: United States, 2011; Vol. 173,
pp 621-9.

In Obesity (Silver Spring), United States, 2008; Vol. 16, pp 1651-6.

H., Shorter sleep duration is associated with increased risk for being overweight at ages 9 to 12

M.; Mantzoros, C. S., Association of maternal short sleep duration with adiposity and
cardiometabolic status at 3 years postpartum. Obesity (Silver Spring) 2011, 19, 171-8.

76. Lytle, L. A.; Murray, D. M.; Laska, M. N.; Pasch, K. E.; Anderson, S. E.; Farbakhsh, K.,
Examining the Longitudinal Relationship Between Change in Sleep and Obesity Risk in

77. Lytle, L. A.; Pasch, K. E.; Farbakhsh, K., The relationship between sleep and weight in a


