

SLEEP, OBESITY, AND THE ADOLESCENT POPULATION;
A LITERATURE REVIEW

by
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Abstract

This literature review highlights the current state of adolescent obesity and lack of adequate sleep. Moreover, it focuses on the relationship between the two epidemics. The results indicate that the probability of sleep deprivation inversely affecting body mass index is quite high, and is most likely dose dependent. Because current evidence in the adolescent population is limited, recommendations include further research that is age and gender specific. Health care practitioners must be involved in the implementation and evaluation of the data on hand, and act in a fashion that empowers the adolescent to create positive changes in their life.

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Dedication

To my husband, Mahmud, for his day to day love and support. To my children, Jamal, Aysha, Malik, and Zakiyah, for their infinite smiles and hugs. To my parents, Connie and Carl Anderson, for their endless encouragement. To my friend, Marilee Walker, for our early morning runs and lots of laughter. Thank you.

Introduction

Childhood obesity is a pressing concern within the United States. Approximately nine million children over six years old are considered obese (Institute of Medicine of the National Academies [IOM], 2005). According to Healthy people 2010, the proportion of overweight children, aged six to 19 years, has risen from 10 to 15% between the years 1994 to 2000 (United States Department of Health, 2000). The dramatic increase is alarming to health practitioners worldwide, considering obesity is a strong predictor of cardiovascular disease (Lauderdale & Knutson, 2006). Childhood obesity is associated with several unhealthy conditions, including high blood pressure, abnormal glucose and cholesterol levels, depression, social discrimination, and behavioral problems (IOM, 2005). Along with the medical risks associated with obesity, is the overwhelming cost to our health care system as a result of the chronic disease process it creates. National health care expenditures linked to obesity-related diseases range from 98 to 124 billion dollars yearly (IOM, 2005). Nationwide attention has called for immediate interventions to slow the pandemic. Children are especially important to consider since obesity tracks into adulthood, having the potential to profoundly affect the number of health problems and amount of health care expenditure.

Concurrently, as the obesity trend has overwhelmed the nation, there has been a documented decline in the number of hours of sleep children receive nightly. In 1910, the average adolescent slept 9.1 hours each night, and it can be assumed that this number approximates the natural biological sleep need of the adolescent (Knutson, 2005). However, as early as 1994, it was noted that adolescents' sleep duration had decreased to 7.4 hours average per night (Knutson). This was recently confirmed in the 2006 Sleep in America Poll by the National Sleep foundation [NSF] (2006). Many factors such as television, computers, video

games, school, cellular phones, and the pace of contemporary life have influenced this trend. The consequences of lack of sleep include excessive daytime sleepiness, and interference with an adolescent's safety and health. Researchers have found that decreased sleep duration is more common in children who are overweight.

Problem

In recent years, as the trend in adolescent obesity has magnified, many studies have focused on isolating the contributory, modifiable risk factors. Some studies have identified an association between decreased sleep duration in adolescents and an increase in body mass index (BMI) (Al Mamun et al., 2007; Biggs & Dollman, 2007; Eisenmann, Ekkekakis, & Holmes, 2006; Gupta, Mueller, Chan, & Meininger, 2002; Hasler et al., 2004). Laboratory studies in adults have indicated a possible biological mechanism for this association, as well as support for the hypothesis that sleep restriction is related to obesity in adults (Taheri, Lin, Austin, & Mignot, 2004; Chaput, Despres, Bouchard, & Tremblay, 2007). However, to date, the findings of the studies that have examined this correlation in children have been inconsistent; most notably, they have assessed children of varying age ranges, used multiple different sleep scales and ratings, and erratically defined obesity (Lauderdale et al., 2006).

Purpose

The purpose of this paper is to provide a comprehensive literature review on the association between decreased sleep duration and the increasing obesity epidemic in the adolescent population.

Conceptual Framework

This paper utilizes the theory of thermogenesis as the foundation for the conceptual framework. Thermogenesis is the process of heat production. It is the means by which the body burns fat. Total energy expenditure can be broken down into three categories: 1) obligatory energy expenditure needed to perform the basic cellular and organ functions; 2) adaptive thermogenesis induced by diet or cold exposure; and 3) physical activity (Lowelle, 2004).

According to the theory of thermogenesis and weight control, weight control is the balance between caloric intake and energy expenditure (Maville & Huerta, 2001). Hence, obesity results from overeating, inactivity, or a combination of both. In light of decreased sleep duration, less sleep time offers more time and opportunity to eat, thus contributing to an increase in weight. Also, decreased sleep leads to daytime fatigue, which leads to decreased activity, which leads to weight gain (Chaput, Lord et al., 2007). A more recent consideration in the theory of thermogenesis is that decreased sleep time creates alterations in hormonal levels and activities that may directly affect hunger and appetite, thus affecting the energy balance of the body (Taheri, 2006; Chaput, Lord et al., 2007).

The scientific study of sleep began in 1953, at the University of Chicago, with the discovery of REM sleep (University of Chicago Medical Center, 2004). Since that time, further projects on sleep focused on its effects on the brain. Over the last ten years, researchers have begun to look at the link between the lack of sleep and hormonal and metabolic functions. Sleep exerts a profound effect on hormones and metabolism. Prolactin (PRL) and growth hormone (GH) levels strikingly increase during sleep, while cortisol and thyroid stimulating hormone levels decrease during periods of sleep (Van Cauter, Holmback, & Knutson, 2007). When sleep is interrupted, or of poor quality, the hormones are conversely affected and a documented

decrease in PRL and GH can be seen, as can an increase in cortisol and TSH levels (Van Cauter et al., 2007).

Growth hormone secretion is dependent on sleep duration and quality, with its levels markedly decreased during times of sleep restriction. As it is normally a critical element to nocturnal lipolysis, the decrease is a possible contributing factor to the development of obesity (Singh, Drake, Roehrs, Hudgel, & Roth, 2005). Whereas cortisol's lipogenic property may contribute to weight gain when the cortisol levels increase during sleep restriction (Singh et al.). In addition, chronic sleep loss can impair glucose tolerance, decrease insulin sensitivity and increase sympathetic tone (Spiegel, 1999). These are "well recognized risk factors for insulin resistance, obesity, and hypertension" (Singh et al., 2005, p. 361).

An association between sleep debt and altered leptin and ghrelin secretion has also been reported (Spiegel, 1999). Leptin and ghrelin are known as the "hunger hormones" (Magee, 2005). Leptin, made by adipocytes, decreases your appetite. Ghrelin, released in the stomach, signals hunger and increases appetite. Leptin has been implicated as a primary hormone in the process of thermogenesis, and its circulating levels have a distinct circadian rhythm. It has been shown that sleep deprivation suppresses leptin levels, altering a person's appetite (Singh et al., 2005). Simultaneously, as leptin levels decrease with sleep deprivation, levels of the counter-hormone, ghrelin, increase (Spiegel, 1999; Spiegel et al., 2004; Van Cauter et al., 2007). "The changes in leptin and ghrelin with sleep restriction could, therefore, provide a powerful dual stimulus to food intake that may culminate in obesity" (Taheri et al., 2004, p. 215).

In studies documenting the relationship between total sleep time and leptin/ghrelin levels, sleep time has varied from short duration deprivation found in the acute setting (Spiegel et al., 2004, 2007), to chronic sleep restriction found in the general population (Chaput, Dupres et al.,

2007; Taheri et al., 2004). Taheri and colleagues (2004) found that people who routinely slept five hours or less had a ghrelin level of 15% more and a leptin level 15% lower than those with seven hours sleep. These numbers were consistent with the laboratory findings in the studies by Spiegel and colleagues (2004; 2007), and again reinforced in the cross sectional study completed by Chaput, Dupres et al. (2007).

Lack of sleep may upset the neuroendocrine balance of leptin and ghrelin. These changes have been shown to cause feelings of hunger, leading to increased eating. As hypothesized, an inverse relationship between decreased sleep duration and body mass index (BMI) is noted. Simply put, if a person does not get an adequate amount of sleep, they are too tired to exercise, they have too much free time to eat, and their hormonal imbalance promotes consumption of high calorie foods. The diagram in Appendix A depicts the conceptual framework of thermogenesis and weight control in relation to inadequate sleep patterns.

Definition of Terms

The literature review was conducted using a CINAHL, PUB-MED, and GOOGLE search of the words: Sleep; Obesity; Overweight; Adolescent; Body Mass Index (BMI); Leptin; and Ghrelin for articles written in English between the years of 1998 and 2008.

Adolescent: Culturally, the ages of adolescence vary. In the United States, adolescence is generally considered to begin around the age of 12 years and end around 19 years.

Body Mass Index (BMI): is a number calculated from a person's height and weight. It is a reliable indicator of body fat in people. It does not measure the body fat directly, but is seen as an alternative for direct measures (such as underwater weighing and dual energy x-ray

absorptiometry) of body fat. It is calculated as weight in kilograms divided by height in meters squared: $\text{weight (kg)} / [\text{height (m)}]^2$.

Ghrelin: Ghrelin is a neuropeptide produced by the endocrine cells, found in the stomach (Liddle, 2007). It stimulates the secretion of growth hormone and appetite, and generates a positive energy balance that can lead to weight gain (Liddle, 2007).

Leptin: Leptin is a member of the cytokine family. It is produced primarily in fat cells, and reduces food intake in a variety of ways (Bray, 2007).

Overweight: ‘Overweight’ refers to an excess of body weight.

Obesity: ‘Obesity’ refers to an excess of fat (Klish, 2007).

Sleep: the body’s rest cycle. It is a natural suspension of voluntary activity for any purpose.

Literature Review

Adult Studies

Between the years of 2004 to 2007, ten independent research studies have been reported within industrialized countries, analyzing the relationship between the increasing obesity trend in adults and chronic sleep deprivation (Bjorvatn et al., 2007; Buscemi, 2005; Chaput, Lord, et al. 2007; Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005; Hasler et al., 2004; Kohatsu et al., 2006; Patel, Malhotra, White, Gottlieb & Hu, 2006; Singh et al. 2005; Taheri et al., 2004; Verona et al., 2005). An eleventh study, reported in 2000 is included, as it provided the seminal data to which the later studies were compared (Vioque, Torres & Quiles 2000). Among the projects, three were longitudinal in nature (Hasler et al., 2004; Patel et al., 2006; Taheri et al., 2004). The remaining eight were cross-sectional in design. Specific populations included adult

women, rural adults, urban adults, obese participants, and internal medicine patients. The ages of the groups varied greatly – from 15 years and up (Vioque et al., 2000), to participants 50 years and older only (Chaput, Lord et al., 2007). Limitations among these studies included the issues of non-representative samples, lack of routine adjustment for co-morbid factors, the reporting of sleep time, and the definition of sleep times.

Obesity was consistently defined as a body mass index (BMI) ≥ 30 in all of the eleven adult studies examined for this review. The definition of sleep time or sleep duration varied greatly among the studies, although all relied heavily on self report as the indicator of total sleep time. Bjorvatn et al. (2007) specified “sleep duration” as “time in bed... minus self-reported sleep latency”, and likewise analyzed sleep duration during the workweek separately from sleep duration during free time (p. 67). Taheri and colleagues (2004) combined self-reported sleep measures in the forms of mailed questionnaires and six-day sleep diaries, along with a one-night polysomnography test to evaluate the degree of average nightly sleep duration in a longitudinal design.

Three studies categorized sleep duration into hourly blocks: <5 hours, 6 - 6.9 hours, 7 – 7.9 hours, 8 – 8.9 hours, and 9> hours (Bjorvatn et al., 2007; Patel et al., 2006; Singh et al., 2005). Sleep duration categories were similarly divided by Kohatsu et al. (2006), but with the lower end as less than six hours duration. The average sleep duration of the adult population was discovered to be seven to eight hours by multiple projects (Kohatsu et al., 2006; Patel et al., 2006; Singh et al., 2005; Taheri et al., 2004; Verona et al., 2005). In three cross sectional and one longitudinal study the mean BMI of the participants ranged from 27.2 – 30 kg/m² (Kohatsu et al., 2006; Lauderdale et al., 2006; Singh et al., 2005; Verona et al., 2005;). The lowest BMI

corresponded to sleep duration of seven to eight hours (Bjorvatn et al., 2007; Patel et al., 2006; Singh et al., 2005; Taheri et al., 2004).

Kohatsu and others (2006) revealed a negative correlation between sleep duration and BMI after adjusting for multiple covariates, including sex, age, and sleep-related breathing disorders. In fact, they discovered an increased BMI unit of 0.42 for every one hour decrease in sleep duration. [A 0.42 BMI unit for a person 70 inches tall is equal to 2.29 pounds (Kohatsu et al., 2006)]. Verona et al. (2005) adjusted for participants with diabetes mellitus, hypertension, arthritis, and gastroesophageal reflux disease after noting that participants with those disorders “had a significantly greater BMI than other participants ($P < .5$)” (p. 25). Final observations from this study concluded that obese participants ($BMI > 30$), slept less than those with a $BMI < 25$. Again, there was a dose dependent association. A mean BMI increase of 5.4 corresponded with a decrease of one hour sleep per week. A significant dose dependent increase in BMI associated with sleep loss from the baseline of 7.7 hours was also observed by Taheri et al. (2004). “An increase in BMI from 31.3 to 32.4 (+3.6%) corresponded approximately to an average nightly sleep duration decrease from 8 h to 5 h” (Taheri et al., p. 212). The study conducted in Spain in 2000 revealed an associated 24% decrease in obesity for every additional hour of sleep time over six hours (Vioque et al., 2000). The NHANES I analysis also showed a negative association with a change in BMI for each additional hour of sleep, however they report the association as “small and statistically insignificant” (Gangwisch et al., 2005)

The Hordaland Health Study, while adjusting for covariates such as gender, smoking and BMI, concluded similarly to the other cross sectional studies noting a “clear association between short sleep duration and obesity” (Bjorvatn et al., 2007, p.1). Hasler and colleagues (2004) revealed significant inverse relationship between sleep duration and BMI in all cross

sectional analyses of their study with the exception of the participants at age 40. Interestingly, the longitudinal trend, when comparing the association between short sleep duration and later obesity, as well as the association between short sleep duration and previous obesity, was significant ($P < .05$).

Evidence suggests a possible gender specific association between sleep duration and obesity. Kohatsu et al. (2006) noted no significant interaction between sleep duration and sex, nor sleep duration and age. Neither did the results of Chaput, Lord et al. (2007) reveal a correlation between sleep duration and BMI in older women. Buscemi (2005) noted an increased likelihood of obesity with decreased sleep duration (<7hrs), (OR = 2.93) in the overall population, however included a U-shaped curve association in women as compared to men. The NHANES I analysis revealed a slight gender difference in that women with a sleep duration less than seven hours were “progressively more likely to be obese as their sleep durations decreased. Men who slept six or fewer hours per night were more likely to be obese than those who slept seven hours per night” (Gangwisch et al., 2005, p. 1292). The research continued to note that gender differences continued when sleep durations of eight to nine hours were compared with subjects who slept seven hours, including a U shaped curve for women (Gangwisch et al.). Data gathered by Taheri et al. (2004) also revealed a U-shaped curve association between sleep duration and BMI. The Hordaland Health Study revealed similar findings “where BMI increases in subjects with sleep duration below 6 hours and above 9 hours” (Bjorvatn et al., 2007, p. 73). Longitudinal findings by Patel et al. (2006) noted that while adjusting for age and BMI, an average weight difference of 1.14kg in women sleeping less than six hours was revealed at baseline. “Not only was a difference in baseline weight apparent but also weight increased more rapidly in those sleeping the least” (Patel et al., p. 950). This established a relative risk of a 15 kg

weight gain over 16 years to be 1.32 (95% CI = 1.19, 1.47) for women sleeping five hours nightly.

The general conclusions to the afore-mentioned eleven studies overwhelmingly support the epidemiological hypothesis that decreased sleep duration in the adult population is related to increased obesity. While the studies demonstrated inconsistent control of confounding variables, variation in sample age groups, and definitions of sleep time, significant correlations between sleep duration and obesity were discovered. The groundwork created by these studies has provoked further investigations as to the relation between sleep duration, age, and obesity. If, as was noted by the longitudinal study by Hasler et al. (2007), lack of sleep in the younger aged population leads significantly to obesity at that age and later in life, then it is pertinent to study this hypothesis in the adolescent population. Research into the neurohormonal and metabolic functions related to sleep deprivation in the adolescent population may reveal a functional and chronic debilitation that is unable to correct itself as a person ages. The initial focus is to verify that there is a similar relationship between sleep debt and obesity in the adolescent population.

Childhood Studies

Thirteen studies were conducted during the years 1999 to 2008 investigating the relationship of sleep duration and body mass index in children (Agras, Hammer, McNicolas & Kraemer, 2005; Al Mamun et al., 2007; Biggs & Dollman, 2007; Chaput, Brunet, & Tremblay, 2006; Eisenmann et al., 2006; Gupta et al., 2002; Kagamimori, Yamagami, Sokejima, Numata, & Handa, 1999; Knutson, 2005; Landis & Parker, 2007; Lumeng, et al., 2007; Reilly et al., 2005; Sekine et al., 2002; von Kries, Toschke, Wurmser, Sauerwald & Koletzko, 2002;). A meta-analysis involving many of these studies was recently published (Chen, Beydoun, and Wang; 2008). Of the 14 projects, six studies specifically focused on the adolescent population. Goals of

these studies ranged from 1.) identifying risk factors for childhood obesity, 2.) assessing the relationship between sleep and obesity, and 3.) addressing the hypothesis that obesity is inversely related to sleep deprivation.

Although all thirteen studies defined overweight and obesity in terms of body mass index, there remained an inconsistent pattern of classification. No uniform, world-wide definition of obesity in children has been agreed upon. Trends are difficult to compare without a common standard. The international survey conducted by Cole, Bellizzi, Flegal & Dietz (2000) *Establishing a standard definition for child overweight and obesity world wide*, served as one obesity definition. As noted by the authors, as a child ages, their body mass index varies widely. In order to compare populations, a “cutoff point related to age is needed to define childhood obesity, based on the same principle at different ages” (p. 1240). In the United States, the 95th percentile of the body mass index is often used as the cutoff point for childhood obesity (CDC, 2007). In contrast, Cole and others proposed a definition from “pooled international data for body mass index and linked to the widely used adult obesity cut off point of 30kg/m². The definition is less arbitrary and more international than others, and should encourage direct comparison of trends in childhood obesity worldwide” (p. 1242). Four of the eleven studies used the international definition of overweight and obesity (Chaput, Brunet et al., 2006; Sekine et al., 2002; Eisenmann et al., 2006; Al Mamun et al., 2007). The 2000 CDC growth charts, in which, overweight is defined as BMI \geq 95th percentile for age and gender, was utilized in four of the studies (Knutson, 2005; Landis & Parker, 2007; Lumeng, et al., 2007; Reilly et al., 2005). The German study designated “overweight” as a BMI \geq 90th percentile for age and gender and “obese” as a BMI \geq 97th percentile, and included measurements of body fat in the definition (von Kries et al., 2002). Gupta et al. (2002) also combined body mass index and body fat to finalize

their definition of “obese”: a BMI \geq 85th% with a body fat of \geq 25%(boys) or \geq 30%(girls). The longitudinal study completed by Agravas et al. (2004) only supplied a definition of “overweight”, consistent with a BMI \geq 85th% for age and gender. Australian researchers included waist circumference in their definition of obesity, with values based on percentile ranges (Eisenmann et al., 2006; Biggs and Dollman, 2007).

There was significant variation in the definition of sleep duration and the methods used to obtain the data. A wide discrepancy of subject age in the thirteen studies was noted, and as is understood, sleep needs of children vary by age. One cross sectional study focused on toddlers (Kagamimori et al., 1999). Three cross sectional studies focused on the school aged child, from five to ten years old (Chaput, Brunet et al., 2006; Sekine et al., 2002; von Kries et al., 2002), while two cross sectional studies combined these age parameters (Agravas et al., 2005; Reilly et al., 2005). Pre-adolescents were the focus of one study (Lumeng et al., 2007). Three studies used teenaged participants (Gupta et al., 2002; Knutson, 2005; Landis & Parker, 2007), with two studies merging the school age and adolescent groups, including children aged seven to 15 years old (Biggs & Dollman, 2007; Eisenmann et al., 2006). The longitudinal study by Al Mamun et al. (2007) spanned the ages from six months to 21 years.

Average sleep duration in 24 hours during infancy (six months of age) is 14.2 hours (Iglowstein et al., 2002). The total sleep time decreases to 8.1 hours by age 16 (Iglowstein et al.). In most studies the total sleep duration time is obtained by parent report; although with older children it is often by self report. As reported by Iglowstein et al. (2002), parent report of total sleep time, although somewhat overestimated in time duration, has demonstrated reliability with actigraph recordings.

Most people believe that adolescents need less sleep than younger children. In fact, according to Kelman (1999) and Dahl and Lewin (2002), adolescents need more sleep. This need arises from the intense physical, hormonal, intellectual and social changes they are experiencing. In addition, the basic sleep-wake pattern is altered (Kelman; Dahl & Lewin). Adolescents spend less time in the deep sleep stage, and more time in the light sleep stage. There is also a decrease in the amount of REM sleep as compared to young children, which is the stage that restores brain energy. The circadian rhythm of the adolescent changes also. Whereas the rhythm cycles in approximately 24 hours from age six months to 14 years, it slows in mid-adolescence to a 26 to 30 hour “day”. Hence, some teens may go to bed much later, and have difficulty waking early in the morning for school and social responsibilities. The circadian rhythm returns to the 24 hour cycle in a person’s late 20s to early 30s (Kelman, 1999; Dahl & Lewin, 2002). Given this information, recommendations by the National Sleep Foundation are for at least 9.25 hours of sleep nightly for adolescents aged 14 years and up (NSF, 2006). These recommendations are echoed by Chen, Bouyden, and Wang (2008).

The Toyama Birth Cohort Study, initiated in the 1990s, served as a basis for two of the foundational studies completed. The purpose of this cohort project was to study the lifestyles and health of children of Toyama, Japan (Kagamimori et al., 1999). Kagamimori and colleagues explored the relationship between childhood obesity and environmental factors, including total sleep duration. This study included 9668 children, aged three years, who were born in 1998, as subjects. The environmental factors observed included meal intake, snack intake, concerns of nutrition value, physical activity and outdoor play, bed/wake time and total sleep duration, and family dynamics. Of the lifestyle and social characteristics, irregular snack intake, physical inactivity and reduced sleeping hours were statistically significant indicators.

Sleep data were obtained via parental report. Total sleep duration was calculated by subtracting “going to bed time” and “wake up” time; less than 10 hours was then designated as “reduced sleep hours” for this cohort of three year old children. Obesity was defined as a Kaup index, “alternatively called the body mass index” > 18 . This is comparable to the CDC guidelines of a BMI $\geq 95^{\text{th}}$ % for girls or boys aged 36 months (Centers for Disease Control and Prevention, 2007). A multivariate logistic-related lifestyle analysis was used. The characteristics were compared between obese and nonobese children, with results showing that reduced sleeping hours were “significantly higher [for obese children] compared to nonobese children” ($P < .001$) (Kagamimori et al., 1999; p. 238).

The second analysis of the Toyama Birth Cohort Study included 8274 children (4194 males, 4080 females), aged six to seven (Sekine et al., 2002). The goal was to examine the relationship between childhood obesity and parental and lifestyle factors. Again, obesity was defined based on body mass index; this time, however, on percentiles designated by Cole et al. (2000). Parental obesity was one of the comparison factors, and it was defined as a BMI ≥ 25 kg/m². Sleep duration was obtained via parental report in questionnaire form, with attention given to wake up time, bedtime, and sleeping hours, although the prior two were not entered into the model for analysis. Per authors’ report the questionnaire was “considered to have moderate to high reproducibility” (Sekine, 2002, p. 165). Multivariate logistic analysis was conducted, adjusting for age, sex and parental obesity. A “significant dose-relationship between late bedtime or short sleeping hours and obesity [was noted] whereas wakeup time was not significantly related to obesity” (Sekine, 2002, p. 167). The adjusted odds ratio, in comparison to ≥ 10 hours sleep, were as follows:

- 9-10 hours sleep: OR = 1.49 95% CI = 1.08 – 2.14
- 8-9 hours sleep: OR = 1.89 95% CI = 1.34 – 2.73
- < 8 hours sleep: OR = 2.87 95% CI = 1.61 – 5.05

German researchers, von Kries, Toschke, Wurmser, Sauerwald and Koletzko (2002), conducted a cross sectional study to assess the relationship between total sleep duration and obesity in five to six year old children from southern Germany. In total, 6862 children participated in the project. Of those, 2109 were five-year-olds, the rest were six years of age. The measurement outcomes of the study included “overweight”, defined as a body mass index > 90th percentile adjusted for age and gender; and “obesity”, defined as > 97th percentile BMI adjusted for age and gender; and excessive body fat, defined as fat mass > 90th percentile for children aged 5-6 years. Questionnaires regarding sleep habits were completed by the parents, with total sleep time calculated by a difference between the reported awake time from the reported to bed time. Sleep hours were categorized out as < 10 hours, 10.5 – 11 hours, > 11.5 hours. Multiple cofounders, including parental obesity, single parenthood, maternal smoking, snacks, and watching tv, were adjusted for in the odds ratio distribution.

The average duration of sleep for the Bavarian children was between 10.5 – 11 hours nightly. The authors concluded a “dose dependent decrease in the proportion of overweight and obese children” (von Kries, et al., 2002, p. 712) with increased hours of sleep. The proportion of overweight decreased from 14.5% in children sleeping for less than 10 hours, to 7.4% in children sleeping 11.5 hours or longer. The proportion of obesity decreased from 5.4% to 2.1%, respectively. The risk of obesity was reduced by more than half in children sleeping more than 11.5 hours in comparison to those sleeping less than ten hours (von Kries et al).

A cross sectional study, completed in the Canada with children involved in the ‘Quebec en Forme’ (QEF) Project was published by Chaput, Brunet, and Tremblay in 2006. The QEF Project is a government and private partnership promoting a healthy lifestyle, with a mission to “improve health and global autonomy of children and family” (Chaput, Brunet et al., 2006, p. 1081). The sample population included 422 children (211 boys; 211 girls), in first, second and fourth grade during the year 2003. The mean age of the children was 6.6 years for boys and 6.5 years for girls. A questionnaire survey was administered by telephone to the parents of the participants. Two of two of the questions on sleep were: “When does your child usually go to bed during the week?” and “When does your child usually get up in the morning during the week?”. Distinct time options were given for each question. Total sleep time was calculated by the difference between bedtime and time for getting up. Obesity was defined according to the Cole definition with BMI as the index. This study also included anthropometric measurements of the sample population, including waist circumference. The prevalence of overweight in the sample population was 20% in boys and 24% in girls.

To evaluate the strength of the relationship between potential risk factors and childhood obesity, logistic regression analysis was performed. Next, multivariate logistic regression analysis was performed by gender, and the odds ratios were adjusted for age and parental obesity. While using the 12-13 hour sleep duration as reference, adjusted odds ratio for those with 8 to 10 hours of sleep was 3.45 (95% CI: 2.61, 4.67). These authors observed a “significant negative association” between sleep duration and body weight ($P < .01$), BMI ($P < .01$), and waist circumference ($P < .01$). The relationship was more significant in males than females, even though a general trend was appreciated (P -values did not exceed .10) (Chaput, Brunet et al., 2006, p. 1083).

Three cross sectional studies concluded that there was an inverse relationship between sleep duration and BMI in school aged to adolescent aged boys (Biggs and Dollman, 2007; Eisenmann et al., 2006; Knutson, 2005). The relationship for girls in the three studies was not significant. The average age of the child studied in Knutson's project was 16.6 years old. A total of 4486 students (2199 males, 2287 females) were included in the analysis. Surveys administered at home were part of a greater longitudinal study, "The National Longitudinal Study of Adolescent Health". Both parents and adolescents participated in completing the survey during the summer of 1996. Habitual sleep duration was assumed by asking the survey question, "How many hours of sleep do you usually get?" (Knutson, p. 831). The average reported total sleep time was 7.7 hours nightly for boys (7.6 hours for girls). Obesity was defined based on the CDC definition. Within the total participant population, 13% of the males and 10% of the females met the definition of "overweight". Variables including age, race, physical activity and inactivity, and parental education level, were adjusted for when completing the analysis. A linear regression model was used to predict BMI, and a logistic regression model was used to predict obesity, and in both, sleep duration was a significant predictor in males only. "According to the logistic regression, every hour increase in sleep duration is associated with a 10% reduction in risk of being overweight in males" (Knutson, p. 832).

Eisenmann et al. (2006) examined the relationship of both body mass index and waist circumference to sleep duration in Australian children aged seven to 15 years old. The authors chose to include waist circumference as a second index to BMI, as one that "is an independent risk factor for several chronic disease states" (Eisenmann et al., p. 957).

Data for the analysis was taken from a cross sectional study conducted in 1985, The Australian Health and Fitness Survey. Data from 3203 males and 3121 females, aged 7 to 16

years were included in this study. The subjects were stratified into three age groups, 7 to 10.9 years old, 11 to 13.9 years old, and 14 to 16.5 years old (Eisenmann et al., 2006). Sleep duration data were obtained by questionnaire. Each child was asked “ What time did you go to bed last night and what time did you wake up this morning?” (Eisenmann et al., p. 957). Four categories of sleep duration were designated by calculating the difference between bed time and awake time (< 8 hours; 8-9 hours; 9-10 hours; > 10 hours). Obesity was based on body mass index percentiles as defined by the Cole et al. study. Waist circumference was measured to the nearest 0.1 cm.

Analysis was conducted both within the three age groups and in the total sample population. The prevalence of overweight for the sample population was 9.8%, and that of obesity 1.6%. The average total sleep time was 9.5 hours. The highest risk for obesity occurred in the older population; with little significant trends noted for the female gender. After linear and logistic regression analysis, “the results indicate significant associations between sleep duration and mean BMI and WC in both sexes; however, a stronger association and an inverse graded response between sleep duration and mean BMI and WC and risk for overweight was found in males but not females” (Eisenmann et al., p. 960). The findings of this study were similar to the dose-dependent results of the Sekine et al., (2002) and von Kries et al. (2002) projects on children, and the analysis of the NHANES I by Gangwisch et al. (2005).

Eisenmann and colleagues (2006) acknowledged limitations to their conclusion due to lack of physical activity or dietary data in the analysis. A retrospective analysis, conducted by Biggs and Dollman (2007), aimed to re-examine Eisenmann’s findings and include physical activity and diet as covariates, while delineating sleep times across the age groups. Data were obtained from 1307 children aged 9 – 11.9 years old, and 2361 children aged 13-16 years old,

who participated in the 1985 Australian Schools Health and Fitness Survey. “After controlling for diet and physical activity, results from this study concur with Eisenmann and colleagues” (Biggs and Dollman, 2007, p. 1839). A shortened sleep duration continued to remain a significant predictor of obesity, measured by BMI and waist circumference in boys, but not in girls. The concluding results demonstrated that future research must consider gender when analyzing the connection between sleep duration and obesity.

Pre-adolescent students were studied by Lumeng et al. (2007). The main goal of this study was to determine whether short sleep duration is associated with obesity in pre-adolescent students, independent of race and social economic status. A longitudinal relationship was evaluated between children during their 3rd grade and 6th grade years (ages 9 to 12 years old). The sample included 785 children, of which 18% were overweight in the 6th grade. Overweight was defined as a BMI \geq 95th percentile for age and gender per CDC guidelines. Sleep duration data were obtained using the Children’s Sleep Habits Questionnaire (CSHQ), completed by the mothers. On average, the children slept nine hours per night. Covariates included gender, race, and social economic status, quality of home environment, parental discipline strategies, and child behavior problems. Again, analysis revealed a dose dependent relationship. “For every additional 1 hour of sleep in 6th grade, the child was [about] 20% less likely (95%CI: 2%-35%) to be overweight in 6th grade. For every additional 1 hour of sleep in 3rd grade, the child was [about] 40% less likely (95%CI: 1%-64%) to be overweight in 6th grade” (Lumeng, p.1025).

An early study by Gupta, Mueller, Chan, and Meininger (2002) considered the relationship between sleep quality and obesity in the adolescent population. A cross sectional design sampled 383 children aged 11 to 16 years old. Sleep quality was assessed as the total sleep time and sleep disturbance time, both measured by wrist actigraphy. Obesity was defined

with a combination of percent body fat and body mass index. A logistic regression analysis of obesity and sleep quality revealed a dose-dependent relationship, in which “the odds ratio associated with sleep time was 0.2, implying that for every hour of increased sleep time the odds of obesity decreases by 80%” (Gupta et al., 2002, p. 767). Confounding variables, such as age, gender, behavioral activities (physical activity, alcohol use, drug use, birthcontrol use, pregnancy) were adjusted for, with final results revealing a slight gender based difference. “Obesity occurs more frequently at younger ages in boys” ($P < .01$). Of important note in this study, subsequent logistic regression analysis supported that obesity itself was not related to sleep disturbances, however daily physical activity was directly related (Gupta et al.).

Landis and Parker (2006) conducted a retrospective chart review to specifically focus on the relationship between total sleep time and obesity in the adolescent population. The chart review was of 870 adolescents seen in a sleep clinic for sleep complaints. Complaints consisted of snoring, insomnia, sleep apnea, restless leg syndrome, and excessive daytime sleepiness. Sleep time was documented by polysomnography testing in the sleep lab, with set bedtime and awake time hours. Overweight and obese definitions were based on the CDC recommendations, 85 – 94th percentile for age/gender; $\geq 95^{\text{th}}$ percentile respectively. Actual participant height and weight were taken by parent report.

Over fifty percent of the sample population was overweight; the mean age for the group was 14.3 +/- 1.8 years. The authors did note that the overweight participants were significantly older than the mean age. While controlling for age, correlational analysis looked at the relationship between BMI and sleep stages, revealing that BMI was associated with sleep disturbances rather than total sleep time (Landis & Parker, 2007).

The three longitudinal studies on sleep debt and childhood obesity cover the wide range of childhood ages. Reilly et al. (2005) gauged school aged obesity at age seven years by data collected when the children were three years old. Agras et al. (2005) studied children from birth to 9.5 years of age. Al Manum et al. (2007) began data collection with 6 month old infants and followed them through their 21st birthday. All three studies revealed a prevalence for obesity in later years in relation to poor sleep quality in younger years, even after variables were adjusted for.

The Avon Longitudinal Study of parents and children in the United Kingdom, provided the basis for the prospective cohort study conducted by Reilly et al. (2005). The original cohort was formed in 1992, in which data was collected from questionnaires and medical records. Data was collected from 7758 children. The outcome measure of the study was obesity at age seven years, defined by a body mass index $\geq 95^{\text{th}}$ percentile for gender and age (Reilly et al., 2005, p. 1).

Multivariate analysis was conducted in three phases using multivariable binary logistic regression models. Final results were adjusted for gender, maternal education, and estimated energy intake. Twenty five potential risk factors were suggested in young (< 3 years of age) children, and eight were associated with obesity in the final models. Short sleep duration (< 10 hours per night) at age 3 years old was one of the identified risk factors, with an odds ratio of 1.45 (95% CI 1.10, 1.89). Other early life risk factors that lead to obesity by grade school years included parental obesity, television time, and weight gain in the first year. The prevalence for obesity did not differ significantly between the genders. "Sleep duration in children aged 30 months was independently associated with prevalence of obesity at age seven. Children with the lowest quarters of sleep duration (<10.5 hours and 10.5 – 10.9 hours) were more likely to be

obese at the age of seven than children in the highest quarter (>12 hours)” (Reilly et al., 2005, p. 3).

In a prospective study of 150 children, Agras, Hammer, McNicholas and Kraemer (2004) looked to establish risk factors in the development of obesity in school-aged children. The authors chose to look at many established and hypothesized risk factors for the development of childhood overweight, including parental obesity, feeding behaviors, early weight gain, maternal return to work, 24 hour calorie intake, activity, temperament, sleep time, maternal weight gain, parental concerns about child’s weight, and parenting behaviors. Of this list of potential factors, five resulted as independent factors for childhood overweight: parental obesity, parental concerns about weight, child temperament, child tantrums over food, and decreased childhood sleep time.

Overweight for this study, was defined as BMI above the 85th percentile for sex at age 9.5 years. Sleep duration was assessed annually by parent report at ages two to five years, as part of the multiple questionnaires that accompanied this longitudinal study. Logistic regression was used in the first stage of analysis to examine the association between the proposed risk factors and BMI results. Statistically significant ($P < .05$) risk factors were pursued further. In this study, 25.3% of the participants were clinically overweight at the age of 9.5 years. Hours of sleep were negatively related with overweight ($P < .01$). A decrease of 30 minutes less than average was all that was needed to increase the proportion of obesity. This difference was due to daytime sleep loss, rather than a decreased duration of sleep at night (Agras, Hammer et al., 2005). The authors hypothesized that young children with low daytime sleep length have low activity levels, which affects weight by reduced calorie expenditure; the low activity level children may sleep less during the day because they are less tired.

The final longitudinal study used a population birth cohort of 2494 children born in the 1980s in Brisbane, Australia (Al Mamun et al., 2007). This study collected data regarding sleep habits at ages six months, two years, and four years, and examined the prospective relationship with obesity in those children at age 21 years. This study observed the potential effect sleep problems in youth had on young adult obesity. The mothers and their children were enrolled in the study during their prenatal care, and followed by maternal questionnaire forms at infants age of six months, five years, 14 years, and 21 years. In addition, the children completed detailed physical exams, cognitive and developmental assessments at ages 5, 14 and 21 years old. Health, welfare, and lifestyle questionnaires were completed by the subjects at ages 14 and 21 years of age.

Of the 2494 participants involved in this analysis, half were male. Overweight at age 21 years was defined as a body mass index 25.0-29.9, and obese as a BMI ≥ 30 ; defined according to the World Health Organization criteria. The definition of sleep problems was dependent on the age of the child. During the six-month follow up questionnaire, mothers were asked, "How often does your child have the following problems?" with "Sleeplessness" as one of the options. Response categories included: almost every day; a few times a week; a few times a month; rarely; never. At the five-year follow up, mothers were asked to think about their child's sleep patterns between the ages of two and four years of age. They were questioned, "... did s/he have irregular sleeping habits?" Response categories included: sometimes; rarely; never. Sleep duration was not measured for this study. Rather, it was extrapolated that the duration of a child's sleep was related to the quality or perceived problems of a child's sleep patterns as observed by their mother.

Potentially confounding factors were acknowledged and adjusted for, ranging from maternal and family characteristics to environmental factors. Dietary intake and physical activity were accounted for at age 14 years old. Multiple linear regression models determined the mean difference in BMI according to sleeping problems. Final analysis revealed no association between sleep disturbances at 6 months of age and BMI at 21 years of age. However, the “mean BMI at age 21 years increased with increasing frequency of experiencing sleeping problems at ages 2-4”, as did the prevalence for overweight and obesity (Al Mamun et al., 2007, p. 1370). The odds ratio for obesity in young adulthood after experiencing sleep difficulties in childhood was 1.90 (95% CI 1.24, 2.93). There was no gender disparity noted.

Limitations

Limitations between the reported studies are similar, and have been previously stated. By addressing some of the basic limitations, future studies conducted in similar fashions will be increasingly reliable. The majority of the studies are cross-sectional in design; this in itself is inherently restrictive. Such study results cannot establish short sleep time as a causative factor in obesity. As obesity appears to be causally associated with sleep apnea and other physiological disorders, “it is unclear from cross-sectional studies whether obesity results in reduced hours of sleep (reverse causality) or vice versa” (Al Mamun et al., 2007, p. 1370).

While reviewing the projects, it became overwhelmingly evident that there is no concrete definition for many of the themes that make up the collection of data. The definition of obesity varied from study to study. The definition of overweight is at times interchanged with obesity. Unlike the adult research projects, which consistently use the definition of body mass index to categorize under/normal/overweight and obesity, the childhood definitions varied from BMI based on United States percentiles for gender and age, to universal definition that included

international data and created percentile curves based on the adult indexes. Others included waist circumference and body fat percentages. It is difficult to adequately compare data between studies when the basic definition of their outcome measurement differs.

Along with the discrepancy in the definition of obesity, is the variations found in sleep definitions and data collection. Data regarding sleep time was obtained by various tools, including parental report, adolescent report, wrist actigraphy and polysomnography. Some data was based on time in bed, while others were based on “sleep quality”. Furthermore, as has been noted, children at different ages require different amounts of sleep time; to compare a seven year old who receives less than seven hours of sleep nightly to a child aged 13 years who receives that same amount of sleep is highly inaccurate. It remains unclear, however, whether the association is between obesity and duration of sleep, or with sleep disturbance of another type. As noted by Al Mamun et al. (2007), “if the key exposure of interest is duration of sleep, then futures studies should directly assess this factor, and methods should be developed for determining the best methods of accurately determining sleep duration” (p. 1372).

The studies included multiple factor analysis in order to depict variables that have the potential to influence energy balance in children. Often energy intake was not included in the variable analysis, which could confound the results. Psychiatric and somatic problems, including depression and obstructive sleep apnea were not included either; both are issues which become more prevalent as a child matures. They are also issues that have the potential to significantly affect a person’s body mass index.

Strengths

Strengths among the various studies include the similar results when compared among each other. Although the cross-sectional designs cannot establish causality, the likelihood of a

relationship between the two factors is significant. Repeated epidemiological investigations, with improved controls, and the convergence of evidence will “help to improve our understanding of the link between sleep and obesity” (Singh et al. 2005). The combination of multiple large population studies, in conjunction with laboratory studies, points toward a unique physiological interaction between sleep and metabolism.

Summary of Literature Review

The studies presented provide provocative insights into the association between sleep duration and obesity across the age spans. According to Taheri (2006) there is now sufficient evidence to support a negative effect of short sleep on energy balance, stating “good sleep could be part of the obesity prevention approach” (p.881). A meta-analysis involving many of the previous studies, reportedly showed a “clear association between sleep duration and the risk for overweight or obesity in children. The risk declined with more sleep” (Parsons, 2008). Final results of the meta-analysis revealed that for every additional hour of sleep a child obtained, the risk of being overweight declined by 9% (Chen, Bouyden, & Wang, 2008).

Several of the large population studies were able to identify a dose-response relationship between decreased sleep time and obesity (Vioque, 2000; Singh et al. 2005; Taheri et al., 2004; Gupta et al., 2002; Reilly et al., 2005; Knutson, 2005; Sekine et al., 2002; von Kries et al., 2002). Results from the studies conducted on preschool and school aged children were consistent with a linear dose-response relationship, in comparison to the findings in adolescent studies, which were much less consistent. In addition, the adolescent studies tended to manifest a gender difference, revealing a significant odds ratio in boys only (Biggs & Dollman, 2007; Eisenmann et al., 2006; Gupta et al. 2002; Knutson, 2005). Interestingly, the longitudinal childhood studies identified that short sleep duration during early childhood years predicted obesity in later years

(Agras et al.2005; Al Mamun et al., 2007; Reilly et al., 2005). Appendix B lists the key findings of the childhood and adolescent studies that reported associations between short sleep time and obesity.

Although the majority of the childhood studies included gender as a variable when analyzing their data and found no significant difference, there were five studies that revealed significant gender differences in the relationship between decreased sleep duration and obesity in the adolescent population (Biggs & Dollman, 2007; Chaput, Brunet, & Tremblay, 2006; Eisenmann et al., 2006; Gupta et al., 2002; Knutson, 2005). The relationship between sleep duration and obesity was more significantly marked in adolescent males than females. When compared with the adult studies, it is pertinent to note that adult men did not show an increased propensity to weight gain as sleep hours decreased when compared to women. Rather, three adult studies indicated a U-shaped curvilinear pattern in relation between sleep duration and BMI, where BMI increased in both male and female subjects with sleep duration below six hours and above nine hours (Singh et al., 2005; Taheri et al., 2004; Bjorvatn et al., 2007). In four other studies, there was a U-shaped curve for women only and monotonic trends in BMI in men (Gangwisch et al., 2005; Buscemi et al., 2005; Patel et al., 2006; Hasler et al. 2004). It is possible that more structured sleep durations should be advocated for, given the U-shaped curves. Also, there is potential evidence that women may need more gender specific guidelines to their sleep habits than young men, while young boys should be encouraged to simply lengthen their sleep hours.

Aside from gender, age has also been observed as a significant variable in the research. No consistent finding has been established, however. The longitudinal childhood studies revealed poor sleep quality or decreased sleep time in younger years, associated with obesity in

later years, again signifying that sleep habits need to be established early in life. A few adult longitudinal studies revealed a trend of decreased sleep in younger years and an increased BMI in subsequent years (Hasler et al., 2004; Gangwisch et al., 2005).

None of the child or adolescent studies included analysis of hormonal levels, such as leptin and ghrelin. As previously noted, studies have been conducted on adolescents and growth hormones, glucose levels, insulin levels, and cortisol levels. There is one known independent study on leptin levels in school aged children (Dencker et al., 2005). These studies have tended to replicate the adult counterparts, stating that hormones act consistently in the general populations, regardless of age. One must wonder, however, how the change in circadian rhythm during adolescence influences the levels of circulating hormones.

One of the crucial differences between the childhood and adolescent studies was the fashion in which sleep data were obtained. Studies conducted in younger children used parental report of sleep duration. Parental reporting of childhood sleep time is negligible in accuracy (Iglowstein et al., 2002; Knutson, 2007). It is doubtful that children are asleep the entire time they are in bed. Often “time in bed” is only roughly estimated, as an average for a child during the week. “This imprecision is likely to account for random misclassification and bias” (von Kries et al., 2002). In spite of this, their results are overall consistent, indicating that children whose parents report short sleep times were at a significant risk for obesity.

The findings are less consistent in the adolescent studies. Data gathered regarding sleep duration in adolescent studies was often by self report, although one study used wrist actigraphy (Gupta et al., 2002), and one study determined sleep duration by polysomnography (PSG) (Landis, 2007). Both of these methods (wrist actigraphy and PSG) have been shown to have significant validity and reliability in their use to document sleep duration; in fact, PSG has been

known as the ‘gold standard’ of sleep determination. Both wrist actigraphy and PSG, although with measured validity, are impractical for a large study, and are expensive tools.

According to Lauderdale et al. (2007), it is difficult to determine whether the inconsistent findings among the adolescent studies are due to the differences in sleep measurement tools or other factors, including study populations. In response to this dilemma, the authors attempted to measure reliability between two different measures of sleep duration, a 24 hour time diary and self reported usual sleep hours (Lauderdale et al.). For large, population based research studies, these methods are economically realistic. A weak correlation between the two methods was found. The time diary sleep was not associated with the subjects’ weight, whereas the self reported sleep duration was associated in an inverse U-curve pattern rather than a linear fashion. The inconsistency in this study reveals the need to establish a validated and reliable tool for measuring sleep duration; one that is feasible and economical for large scale studies.

It is paradoxical that a reduction in sleep, the most sedentary of all activities, would be associated with weight gain. It has been hypothesized that decreased sleep can lead to weight gain in three ways: 1) less sleep time means more awake time, which gives a child more time to eat; 2) less sleep time leads to less daytime energy, which is manifested in less physical activity, and 3) decreased sleep time has been associated with changes in the neurohormonal balances of a person (specifically increased ghrelin, decreased leptin levels) that stimulate hunger and appetite. The review of the literature has shown that there is plausible cause for the relationship between lack of sleep and increased obesity rates in the adolescent population.

Recommendations

Further research should consist of prospective studies that delineate specific age related sleep patterns and their relations to obesity. A universal definition of childhood obesity must be

agreed upon. With suggestion from the past longitudinal studies, it would be imperative that future consideration also discriminate between total sleep times between the genders. The intricate interactions between hormones, appetite, thermogenesis, sleep and weight gain should be targeted for future research. There are no current studies to replicate the findings of leptin and ghrelin changes in adolescents, as have been found in adults. Many of the studies have demonstrated a dose-response relationship between sleep debt and obesity, and have consequently also noted a significant relationship between increased sleep hours and decreased obesity. However, this has not been the goal of the studies. Further research should include this in their hypothesis. Addressing the hypothesis that adequate sleep time reduces a child's BMI is another study to consider. It is difficult to make generalizations from these studies to other age groups, however, having narrow age ranges, may be a considerable advantage by making the subjects more homogeneous and by addressing the physiologic changes that occur during their rapid growth period.

Nursing Considerations

To combat the obesity pandemic, nursing must recognize that weight management requires a holistic approach. With the increasing number of nurse practitioners caring for today's families, it is imperative that they have the tools and knowledge to combat leading health issues and promote beneficial lifestyle modifications. When assisting a patient to manage their weight, the practitioner needs to include a variety of factors that realistically can be modified. When assessing a patient's thermogenic balance, the energy expenditure and caloric intake can vary based on multiple aspects. It is imperative to include the assessment of a patient's weight, eating habits, exercise, and sleep patterns. By advocating for healthy lifestyles, with realistic modifiable risk reductions, patients can independently choose concrete solutions to their

problems. Sleep is an example of a modifiable item, amenable to teaching and increased knowledge by youth and their families.

It is also essential that the nurse realizes obesity is not simply an adult problem. Current research has given us a base for the overwhelming fact that our children are directly affected by and at risk for obesity. It is a chronic medical condition that has enormous implications on health, and can lead to many associated disease processes. When addressed early through holistic lifestyle modifications, there is great potential for preventing the obesity trajectory and improving population outcomes. Therefore, in the family practice setting, the nurse practitioner must advocate for a reasonable and prudent weight of all patients seen across the age continuum. This includes youth. By addressing the adolescent population, the nurse can more effectively intervene in the issue at hand by directly involving the young adult in their own health care. The current literature suggests that duration of sleep is important in the war on obesity. If this is established, it may call for the creation of educational materials emphasizing recommended amounts of sleep and how to achieve this.

Conclusion

The prevalence of adolescent obesity is increasing at an astonishing rate worldwide, and sleep has been identified as a possible contributory factor. The purpose of this review was to identify the current state of the evidence related to the association between decreased sleep duration and increased obesity in the adolescent population. Many studies among adults, children, and adolescents support the hypothesis that short sleep time is associated with weight gain. Evidence leans toward a dose dependent relationship and possible gender difference within the hypothesis. Increasing evidence in adults suggests that short sleep duration results in metabolic changes that may contribute to the development of obesity (Chaput, Lord, et al., 2007;

Spiegel, 2004; Taheri et al., 2004). Sleep duration appears to be important in the “regulation of body weight and metabolism by the modulation of key hormones such as leptin and ghrelin” (Taheri, 2006, p. 882). Addressing adequate sleep patterns in adolescents supports the evolving theory of thermogenic energy balance in weight control. With sleep duration as a potential modifiable risk factor, ensuring adequate sleep time in adolescence might have important clinical implications for the prevention and treatment of obesity.

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Appendix A

Thermogenesis, Weight Control, and Sleep

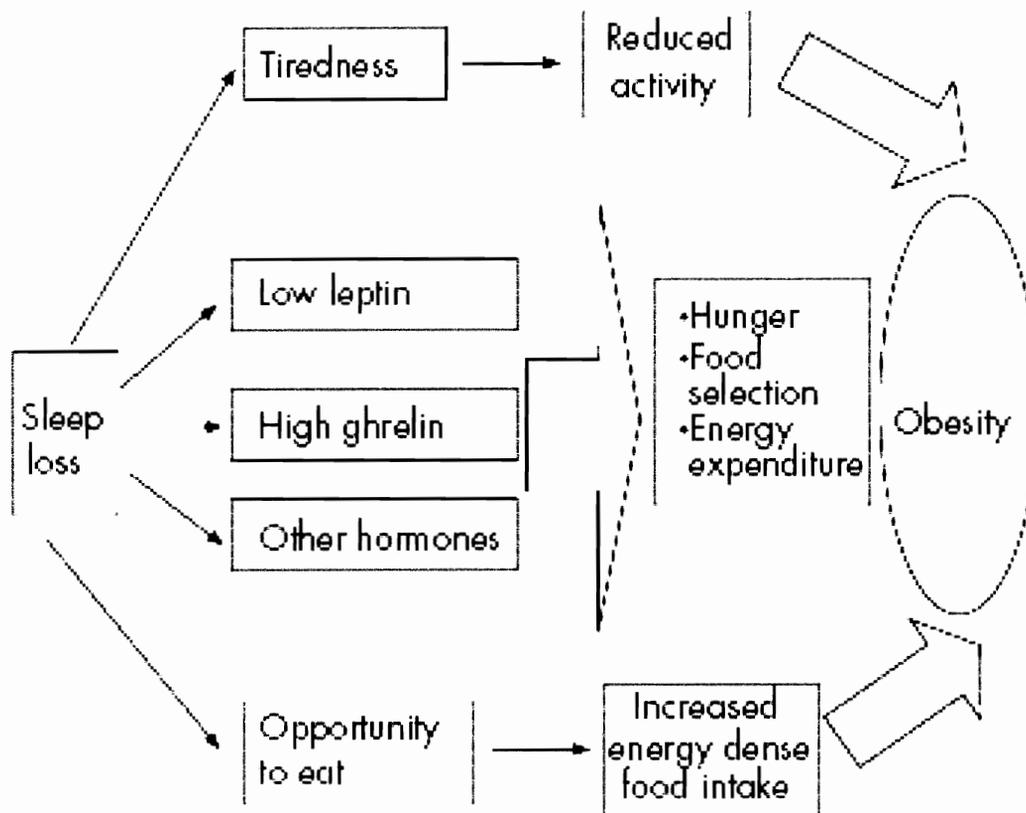


Figure 1 The potential mechanisms through which short sleep duration could result in obesity. Short sleep duration can affect both energy intake and energy expenditure. It results in tiredness that may hamper physical activity, and alters metabolic hormones to increase appetite and affect food selection. Additionally, extra time awake provides increased opportunity for food intake. Other potential mechanisms include effects of sleep on basal metabolic rate, thermic effect of food and non-exercise thermogenesis.

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APPENDIX B

SUMMARY OF MAJOR STUDIES REPORTING AN ASSOCIATION BETWEEN SHORT SLEEP DURATION AND OBESITY IN ADOLESCENTS

Authors	Study design	Subjects	Ave. Age	TST/BMI	Results
Knutson (2005)	Cross-sectional	4486	16	self report	dose dependent: every hour increase in sleep duration is associated with a 10% reduction in risk of being overweight; significant in males only OR 0.90 (CI 0.82 – 1.00)
Gupta et al. (2002)	Cross sectional	383	11-16	wrist actigraphy self report BMI+ BodyFat	26.6% obese; Ave TST = 7.68hrs statistically sign ($P < 0.000$) negative assoc of obesity and TST for every hour of increased sleep time the odds of obesity decreases by 80%”
Eisenmann et al. (2006)	Cross sectional	6324	7-15	self report CDC + WC	9.8% overweight/ 1.6% obese Average 9.5 hrs sleep inverse graded relationship between sleep duration and BMI and WC in boys; for boys, <8hrs sleep led to 3.1 times greater odds of obesity As compared to > 10hrs sleep
Landis & Parker (2006)	Retrospective	52	14.3	PSG CDC	53.9% obese/ Ave TST = 311 minutes BMI not associated with TST, but rather sleep disturbances
Biggs & Dollman (2007)	Retrospective	3669	9-11 13-18	self report BMI + WC	shortened sleep duration (<9hrs, age 9-11; <8hrs, age 13-18) significant predictor of obesity ($P < 0.007$) in males only
Al Manum et al. (2007)	Longitudinal	2494	6m, 2, 4 21 yrs	parental report @ age 6m, 2, 4 Cole	The OR for obesity at age 21 after experiencing sleep difficulties (age 2-4): 1.90 (95% CI 1.24, 2.93) – compared to no sleep problems
Lumeng et al. (2007)	Longitudinal	785	9-12 yrs	Children’s Sleep Habits Quest. CDC	18% overweight in 6 th grade; average 9 hrs sleep More likely to be male; Each additional hour of sleep, associated with 20% less likely to be obese in 6 th grade; in 3 rd grade = 40% less likely in 6 th grade

SUMMARY OF MAJOR STUDIES REPORTING AN ASSOCIATION BETWEEN SHORT SLEEP DURATION AND OBESITY IN CHILDREN

Authors	Study Design	Subjects	Age in years	TST/ BMI	Results
Kagamimori (1999)	Cross sectional	9668	3	parent report Kaup Index	frequency of < 10hrs sleep duration was greater in obese kids (29.3%) vs non-obese kids (13.7%) $P < 0.001$
Von Kries et al. (2002)	Cross-Sectional	6862	5-6	parent report BMI + %BodyFat	dose dependent decrease in prevalence for obesity: $\leq 10h$: 15%; ≥ 11.5 hr: 7% Sleeping four 11.5 hrs reduced risk of obesity to less than 1/2
Sakine et al. (2002)	Cross-sectional	8274	6-7	parent questionnaire/ pretested for validity Cole definition	dose response relationship compared to 10+hrs sleep < 8 hrs: OR= 2.87 (CI 1.61 – 5.05) 8-9hrs: OR= 1.89 (1.34 – 2.73) 9-10hrs OR= 1.49 (1.08 2.14) OR for boys > girls (both statistically sign.)
Agras et al. (2004)	Longitudinal	150	sleep at 3-5 weight @ 9.5	parent report CDC	overweight at 9.5 yrs inversely related to less daytime sleep at age 3-5 @ 9.5 yrs: 25.3% overweight/ 9% obese
Reilly et al. (2005)	Longitudinal	8234	Seep @ 3 weight @ 7	parent report	Short sleep duration (< 10 hours per night) at age 3 years old was one of the identified risk factors odds ration of 1.45 (95% CI 1.10, 1.89).
Chaput et al. (2006)	Cross sectional	422	5-10	parent questionnaire Cole	OR for 8-10hrs sleep 3.45 (CI 2.61-4.67) compared to 12- 13 hrs sleep Significant negative association in boys, not girls Overweight: Boys 20% Girls 24%