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Actigraphy Sleep Patterns: Relationships To Body Mass Index And Physical Activity In Minority Children

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ACTIGRAPHY SLEEP PATTERNS:

RELATIONSHIPS TO BODY MASS INDEX AND PHYSICAL ACTIVITY IN MINORITY CHILDREN

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ACTIGRAPHY SLEEP PATTERNS: RELATIONSHIPS TO BODY MASS INDEX AND PHYSICAL ACTIVITY IN MINORITY CHILDREN. Kathryn E. Wynne, Karen B. Dorsey. Department of Pediatrics, Yale School of Medicine, New Haven, CT.

Previous research suggests that short sleep duration and later bedtimes are associated with higher body mass index (BMI) and lower physical activity levels in children. We sought to determine if objective measurements of sleep duration and timing were correlated with BMI percentile and/or physical activity measures in low-income, minority children. Data were collected from 104 8- to 10-year-old children (58% female; 88.5% Latino or African-American; 51.9% overweight or obese) who were instructed to wear an accelerometer at their waists for 1 week. Data were manually analyzed to determine average sleep-onset (i.e. bedtime), sleep-offset (i.e. wake time), sleep duration, and minutes of wake after sleep-onset (WASO) for each subject. Physical activity measures, calculated from previous analyses of these data, included minutes of moderate and vigorous physical activity (MVPA), sustained bouts of MVPA, minutes of vigorous physical activity (VPA), and bouts of VPA. We used Student t-tests and linear regressions to analyze relationships between BMI percentile, sleep variables, and physical activity variables. Only 7% of children averaged the recommended 10-11 hours of nightly sleep. We found a weak but statistically significant association between shorter sleep duration and increased BMI percentile. None of the other sleep variables, or any physical activity measure, was associated with increased BMI percentile. We conclude that minority children living in low-income communities, particularly those with a higher BMI, get less than the recommended nightly sleep. Future research needs to focus on causes and consequences of these sleep patterns to determine if increased sleep recommendations should be a routine part of obesity prevention and treatment strategies.

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1. Introduction

1.1 Prevalence of Childhood Obesity

Childhood obesity is a challenging and escalating concern that disproportionately affects minority and lower socioeconomic populations(1-3). The rates of obesity within the United States have reached alarming proportions. In 2007-2008, 9.5% of infants and toddlers were at or above the 95th percentile of weight-for-length charts, 31.7% of children and adolescents (aged 2- to 19-years) were at or above the 85th percentile of the BMI-for-age charts, qualifying as "overweight," and 16.9% were at or above the 95th percentile, qualifying as "obese"(4). Data demonstrate both ethnic and economic disparities. The prevalence of obesity among 2- to 5-year-old Latino children is 14.2% compared to 11.4% of African-American and 9.1% of non-Latino White children(4), and the prevalence of being either overweight or obese for children aged 10- to 17-years in families living below the poverty line is 44.8% compared with 22.2% of children of the same age range living above 400% of the poverty line(3). These statistics overlap in that Latino and African-American children are over-represented among the poor: 26.6% of Latino children live in poverty, compared to 27.4% of African-American and 9.9% of non-Latino Whites(5). Thus, Latino and African-American children are at a particularly high risk of being overweight or obese, a condition that often persists into adulthood and brings with it numerous complications.

1.2 Consequences of Childhood Obesity

Children who are either overweight or obese more often than not will continue to be overweight or obese as adults. This trend is even more pronounced in minority

populations. One study found that 3-year-old children with BMI values at the 95th percentile have a 58-71% likelihood of being overweight and a 15-24% likelihood of being obese at 35 years of age. This risk increases as children age, such that 17-year-olds who have a BMI at the 95th percentile have a 98-99% likelihood for being obese at 35 years of age(6). An epidemiologic literature review published in 1993 found that 26 to 41% of obese preschool-aged children and 42 to 63% of obese school-aged children became obese adults(7). Studies among minority children have found an even stronger association between childhood and adult obesity. Those who have the highest BMI as children have the highest risk becoming obese adults, and African-American children, particularly girls, have the highest risk for retaining obese status as they age regardless of the severity of obesity(8). A 17-year long study tracking 2,392 children (36% African-American) found that, among obese children aged 5- to 14-years, 65% of White girls, 84% of African-American girls, 71% of White boys, and 82% of African-American boys became obese adults(9). These study estimates vary depending on methodology and specific populations examined, but nevertheless demonstrate a consistent risk for future obesity that is increased for older children who are obese and for minority children.

Another important concern regarding childhood obesity is the morbidity and mortality associated with the disease, ranging from medical to psychological, and how these complications translate into adulthood. With obesity comes hypertension, hyperlipidemia, impaired glucose tolerance, type II diabetes, liver disease such as hepatic steatosis, orthopedic concerns such as Blount's disease, and pulmonary disease such as obstructive sleep apnea(10-12). In addition, obese children have significant psychological and social risks: they more often report a lower quality of life, negative

self-perceptions, and low self-worth, and have increased reports of behavior problems(10, 11, 13). As expected, a recent prospective study noted that overweight or obese children who remained obese as adults had increased risks of type II diabetes, hypertension, dyslipidemia, and carotid-artery atherosclerosis. Surprisingly, they also noted that the risks of these outcomes among overweight or obese children who became non-obese as adults were similar to those among persons who were never obese as children. While the population evaluated was predominantly White, a small percentage of African-American children were included in these results(14). Although this report emphasizes the benefit of improving weight status regardless of age, there are likely more factors to consider for minority populations. Several reports have found that while the higher prevalence of diabetes among minorities is in large part due to the higher rates of obesity, it cannot be accounted for by obesity alone. This suggests a multifactorial predisposition to this disease regardless of weight status, which likely involves both genetic and environmental factors(15).

Lastly, economic burden both on an individual level as well as societal level are of concern though not yet well described. The costs related to adult obesity have been well explored, but the costs related to pediatric obesity remain ambiguous. Some studies have found that obese children have higher medical bills with higher numbers of office visits when compared to non-overweight children, while other studies have found no difference between the two populations. Some studies suggest that higher costs do not become significant until children reach adolescence, when the cost of psychological treatment begins to play a larger role. Others have found higher costs for female adolescents but not for male(16). In consideration of childhood obesity trends, medical

and psychological complications, and economic worries, we must evaluate why this obesity epidemic is taking place as well as what are potential strategies for treatment and prevention, paying close attention to the communities most at risk for persistent obesity and its complications.

1.3 Causes and Correlations

The causes of this childhood obesity epidemic are believed to be numerous and complex, and are likely genetic as well as environmental. The rare case has a direct biological cause, e.g. hypothyroidism, leptin deficiency, while the vast majority result from a complex relationship between genetic proclivity and an environment that promotes an imbalance between calories consumed and calories spent. While genetics are believed to play a role, a simple genetic mutation cannot account for the recent dramatic increases in prevalence seen in industrialized countries around the globe(12). Rather, it is believed that there is a polygenetic disposition to obesity when the host is exposed to environmental conditions that promote excessive caloric intake and inadequate calorie expenditure. Examples of specific environmental conditions include the availability of infant formula feeding as opposed to breast-feeding, shorter and fewer school gym classes, and increased media screen time (e.g. television and video games) throughout childhood. On this list, shortened sleep duration and irregular sleep patterns may play significant but, as of yet, incompletely defined roles(12, 17). Considering this complex interaction among multiple risk factors, it is challenging for physicians to develop a single coherent strategy for the prevention of obesity or the treatment of overweight and obese patients.

1.4 Medical Diagnosis and Counseling has been Insufficient

The first step to approaching the obesity epidemic is to make a diagnosis, yet even at this first step doctors are struggling due to the lack of time for counseling and discussion and the belief that there are not adequate treatment options for patients(18, 19). Visits to a primary care provider for routine health maintenance provide an excellent opportunity for preventive efforts, evaluation, diagnosis and initiation of treatment plans(20). A national survey study in 2010 found that, when asked by self-report, 99% of pediatricians reported height and weight at each well visit, while only 52% assessed BMI percentile. Additionally, 23% of pediatricians felt that there are not any good treatment strategies for obesity, 67% reported they did not have time to counsel their patients regarding obesity, and, regardless of their opinions, 69% reported that dietician services and weight management programs are not covered by insurance(19). A separate study sought to determine the rates of diagnosis and treatment as well as types of treatment used among pediatricians in New Haven, Connecticut, by reviewing patient charts as opposed to physician self-report. Of six hundred randomly selected charts reviewed, most of which were of minority children, only 0.5% of charts reported the patient's BMI. Among the children at risk for being overweight or who already were overweight (totaling 39.8% of the patient population), 20.5% had a diagnosis included in the chart, and 16.9% had documented treatment. Most commonly, a regimen of diet (74%) and exercise (49%) was documented(21). In these instances, it appears that a disclosure of diagnosis occurred between physician and parent. A recent study asked parents of children who were at or above the 85th BMI percentile if their doctor had told them that their child was overweight. Only 22.4% of parents replied "yes." Factors associated

with positive recall were minority status, low-income, public health insurance, more frequent doctor visits, older children, and increasing obesity severity(22). While diagnosis and disclosure have clearly lagged, evidence suggests that this could change if physicians were not only aware of but also felt confident in the efficacy of available intervention strategies(19, 20).

1.5 Treatment and Prevention Studies

For the patients with obesity who are recognized and diagnosed, treatment has been very difficult with only modest improvements in weight. In addition, these studies frequently fail to include large numbers of minority children who are most in need of attention. Treatment and prevention often blend together as there are 2 major types of prevention: primary prevention of initial overweight/obese status, versus secondary prevention of weight regains following a loss, or simply avoidance of additional weight gain in already overweight/obese individuals(23). Thus, secondary prevention can also be considered treatment.

Treatment interventions within healthcare settings have focused on discussions of weight status, diet, and exercise, as well as referrals to intensive treatment programs. Despite seeming ineffective at producing change, a simple discussion with a parent that defines and clarifies his or her child's weight status is an important first step that can influence his or her actions. For example, one study found that the parents most likely to already be making healthy lifestyle changes were those who perceived that their child's weight was in fact a real health problem(24). While encouraging, this kind of discussion has not been shown to result in weight loss. Numerous studies examining more complex

treatment intervention have been performed. A Cochrane review of treatment options found 64 randomized controlled trials: 12 focused on physical activity, 6 focused on diet, 36 focused on behaviorally oriented treatment programs, and 10 focused on pharmacological interventions. Despite a wide variety of study designs, quality, and outcome measures, authors report that a family-based combined lifestyle interventions approach (including diet, activity, and behavior counseling) when compared to standard care or self-help, does produce clinically small but significant changes in weight and health status of children and adolescents. However, the majority of studies examined have a sampling bias, targeting efforts at middle-class White families who are able and willing to attend multiple counseling sessions over several weeks, and so conclusions regarding minority children living in low-income communities cannot be made(25).

Given the difficulty in treating obesity even when complex interventions are administered, preventive efforts are critical. Despite agreement on this important concept, prevention has been difficult to enact. Interventions have included school-based, community-based, and family-based programs, and most often focus on diet, physical activity, social support, or a combination of these. Based on meta-analyses performed, there have been some significant improvements in dietary and physical activity behaviors resulting from school-based(26, 27), and family based(27, 28) interventions. As might be expected, studies spanning such large cohorts are not purely "primary prevention" in design given the inclusion of overweight and obese individuals, but nonetheless these results are relevant. One review sought to evaluate studies examining minority populations (both non-overweight and overweight/obese children were included) and found 7 such studies. Conclusions from these studies point to that fact that interventions

can be beneficial to a small extent, but are especially so when objectives are specific, when interventions extend across school environments as well as into the general community, and when they are culturally relevant. However, improvements are often not reflected in maintaining healthy BMI measurements for non-overweight children nor in reducing BMI for already overweight/obese children during the study timeline(29). A Cochrane review focusing entirely on primary prevention found 22 studies to analyze, of which 10 studies lasted at least 12 months in duration, while 12 lasted between 12 weeks and 12 months. Results are similar to the studies mentioned above: when considered as a whole, several studies do see results in terms of improved diet and exercise patterns, but these benefits often do not translate into preventing non-overweight individuals from becoming overweight or obese over the course of the study. Additionally, given the heterogeneity of study design, combining findings statistically is not possible(30). Overall, these moderate successes have been unable to keep up with increased prevalence, and thus are unlikely to work effectively by themselves.

As both primary prevention and treatment studies have proven only slightly effective, researchers and clinicians are beginning to reexamine strategies based on both their timing and content. Given that 25% of preschool-aged children are already overweight, it appears that a crucial opportunity for prevention has already passed. By age 5 years, children have consumed thousands of meals and snacks, thus establishing personal preferences, and have become accustomed to the food culture of their homes and families. However, a relatively small number of prevention studies have been performed in preschool-aged children and even fewer prior to this age. Results thus far have not brought about specific suggestions for practice, but continue to investigate parenting

recommendations and methods to encourage children to naturally prefer healthier choices(31). A systematic review of 10 mixed primary prevention and treatment studies, namely nutrition and activity counseling, aimed at children less than 2 years of age found that no intervention improved weight status, and the quality of studies ranged from fair to poor(32). From this review as well as previously discussed evidence, it is clear that focusing on nutrition and physical activity alone is not enough. Instead, a larger variety of interventions must be combined and can include newer passive and active interventions. Policymakers and advocates, for example, are trying to initiate banning cartoon characters from food items such as cereal boxes as well as enacting soda taxations as passive interventions(33, 34). Researchers and physicians are also beginning to consider active interventions that are targeted to populations most at risk and that expand upon the traditional focus on dietary and sedentary behaviors. An example of this is by investigating the relationship between other lifestyle factors such as sleep and obesity.

1.6 Sleep and Obesity

Sleep duration and timing are modifiable risk factors that are being investigated given initial evidence supporting their relationship to obesity. It is important to investigate this link given observed trends: as the prevalence of obesity has increased over time, the duration of children's sleep has significantly decreased. Over the past 100 years, these decreases have occurred across all age groups and for both genders with a median estimate at 0.75 minutes lost nightly per year, indicating a change of over an hour lost per night over this time period. Decreases in sleep duration are most pronounced

among older children, particularly boys, and on school nights(35). In addition to these observed trends, sleep is an important factor to investigate because evidence suggests that compliance with sleep recommendations is easier to achieve than recommendations regarding diet and physical activity(36-38). As supporting evidence continues to be gathered, counseling about healthy sleep habits may become an important component of both preventing and treating childhood obesity. At this point in time, there is insufficient evidence to support the idea that increased sleep duration in children will help alleviate the obesity epidemic, and thus the American Academy of Pediatrics does not include a full night's sleep in their prevention of obesity policy statement(11, 39). They do, however, make reference to a possible link between short sleep and obesity in their policy statement titled "Children, Adolescents, Obesity and the Media," encouraging further research into relationships and potential mechanisms(40). Undoubtedly, obtaining further information is critical prior to making general recommendations. In order to begin investigating sleep in relation to obesity, we must first understand what normal sleep should look like.

1.7 Normative Sleep and Current Recommendations

Sleep is important for many reasons, and what constitutes a healthy duration and timing of sleep based on a child's age can be estimated based on normative data and other sleep studies. Aside from concerns regarding metabolic balance and obesity, inadequate sleep during childhood has been associated with a wide array of physical and psychosocial deficits, including impaired concentration and learning, impaired academic performance, attention deficit hyperactivity disorder, mood disorders such as anxiety and

depression, impaired motor skills, increased alcohol and drug use, increased risk of accidents and injuries, and impaired overall health and immune function(38). Sleep is regulated by two internal processes: the circadian rhythm that directs internal sleep-wake cycles, and the homeostatic process, which regulates sleep timing, length and depth based upon the timing, duration, and quality of the individual's previous sleep history. In early infancy, sleep is most strongly influenced by hunger and satiety, but light-wake cycles take control by 2-3 months of age(41). A longitudinal study of Swiss children from 1974 through 1993 observed an average 24-hour summed sleep duration of 14.2 hours (standard deviation 1.9 hours) at 6 months of age decrease to an average of 8.1 hours (standard deviation 0.8 hours) at 16 years of age. During this period, daytime napping dramatically decreased, from 100% of newborns napping, to 50.4% at age 3 years, down to 0.9% at age 7 years. Other estimates of napping among 7-year-old White children in the United States have ranged up to 13%, possibly reflecting population differences versus different patterns of nighttime and daytime sleep in that U.S. children may sleep less during the night. Trends from cohorts of children recruited in 1974, 1979, and 1986 reveal a decrease in mean sleep duration over time that results from later bedtimes with unchanged wake times(42). However, during the middle childhood period, decreased sleep duration begins to be associated with reported daytime sleepiness, suggesting that normative measures may not reflect physiological demand. Thus, more appropriate recommendations of needed sleep must be estimated from this data. Laboratory-based investigations in fact do indicate that total sleep needs do not change from later childhood into adolescence, yet adolescents get significantly less sleep than younger school-aged

children(41). In the United States, survey-based estimates range from 45% to 98% of adolescents getting less than 8 hours of sleep(43, 44).

Recently, emphasis has been placed on studying normative sleep data in ethnically diverse groups of children, which, thus far, have revealed some consistent differences. The Tuscon Children's Assessment of Sleep Apnea study has provided a comparison of 6- to 11-year-old children of White and Latino backgrounds based on one night of polysomnography. Small but significant differences were observed, including lower mean total sleep time for Latino children (averaging 12.7 fewer minutes per day) and a higher percentage of younger Latino children taking daytime naps (23% versus 13% at age 7 years)(45). Another study comparing White and African-American children aged 2- to 8-years based on parental survey found that African-American children also follow a more gradual decline in napping frequency and duration. Among 8-year-olds, 39.1% of black children still napped, compared to only 4.9% of white children. While napping occurred more often for black children, their average length of nocturnal sleep was lesser, thus average total sleep time did not significantly differ between the two groups. This trend of equal total weekly sleep duration among racial/ethnic groups persisted for each year of age studied(46).

With these data in mind, the National Sleep Foundation offers sleep duration recommendations with the understanding that individuals have varying needs. It is important to recognize that these recommendations are based on limited data and while widely cited are not uniformly agreed upon(47). Generally, they currently recommend 12-18 hours of sleep for newborns (0-2 months), 14-15 hours for infants (3-11 months), 12-14 hours for toddlers (1-3 years), 11-13 hours for preschoolers (3-5 years), 10-11

hours for school aged children (5-11 years), 8.5-9.25 hours for teens (11-17 years), and 7-9 hours for adults(48).

1.8 Theoretical Pathways linking Sleep and Obesity

With this understanding of normal sleep, research has begun to investigate the biochemical pathways that may relate decreased sleep and obesity, many of which have been supported by adult sleep-restriction studies. First, sleep loss results in increased sympathetic activity as well as increased evening cortisol and daytime growth hormone levels, which increases glucose resistance and decreases glucose tolerance(49-51). Decreased sleep has also been associated with dysregulation of hormonal appetite control(52). With sleep loss, leptin, a satiety factor, is decreased, and ghrelin, a hunger-promoting hormone, is increased. As these two hormones become misbalanced, the body's ability to relay information about caloric needs is compromised. One theory regarding the cause of this misbalance is that there may be increased activity of neurons producing the excitatory peptides orexins, which normally promote waking and feeding(38, 49). Beyond this appetite dysregulation, sleep loss allows for more hours awake that can be spent taking in calories as well as a sense of tiredness that may be related to decreased energy expenditure during the day(37, 38, 49-53). One study of adults in 2008 found that decreased sleep can lead to increased snack intake as opposed to meals with the composition of the snacks favoring higher carbohydrate content(54), while a separate study with adolescents in 2010 similarly noted that short sleep was significantly associated with a 2.1-fold increased odds of consuming 475 kcal more calories per day almost entirely from snacks as well as increased calories from fats(55).

Considering these variable pathways, researchers have used different methods with which to gather sleep information to see if these theories might expand to larger, varying populations of individuals.

1.9 Methods of Gathering Sleep Data

In order to most accurately analyze the potential link between short sleep duration and obesity, it is important to gather quality data via subjective and, more importantly, objective methods. Given restrictions in study design, no study has been able to find a causal link between short sleep duration and obesity. Studies have demonstrated that subjective reports, both self and parental, tend to be inaccurate(56-59). Nonetheless, these studies have the advantage of being applied across very large populations and so must be considered. Thus far, few studies have looked at the duration of sleep over the course of multiple days, and few measure sleep patterns objectively(60). Given the lack of objective evidence, new studies are moving in this direction via two basic options: polysomnography and accelerometry. The gold standard of objective sleep assessment is polysomnography, which measures electroencephalography, electro-oculography, electromyography, and electrocardiography patterns simultaneously over a period of time to provide detailed information regarding sleep-wake states and the five sleep stages. Polysomnography is expensive, laboratory-based, and difficult to implement over the course of several nights. In addition, for children in particular, polysomnography can be uncomfortable and stress-inducing so that results may not reflect what sleep is like in their normal home environment. Given these disadvantages, researchers are more frequently using accelerometers. Actigraphs are small watch-like devices worn most

commonly at the wrist or waist that collect data gathered by an internal accelerometer. The accelerometer counts the number of accelerations greater than 10% of the force of gravity (0.1 g) in preset time intervals (e.g. 1 minute epochs, 30-second epochs). Data is collected continuously over an extended period of time and can subsequently be uploaded to a computer for analysis. The advantages of actigraphy are that it provides a cost-effective method for studying sleep within the natural environment over the course of multiple days and nights, and can be administered easily across a much larger population. As compared to the gold standard of polysomnography, actigraphy provides a reliable and valid alternative for detecting sleep in healthy adults and children(61-63).

For physical activity assessment, the actigraph is worn at the waist, while for sleep it has been classically worn on the non-dominant wrist(64-66). However, research has proven that waist placement in children does not significantly affect assessment of sleep patterns(67, 68). Algorithms or manual counting of activity measures around sleep time can be applied to the collected data in order to determine sleep/wake timing and nighttime awakenings(62, 64, 67, 69, 70). The number of nights needed for analysis based on practice parameter guidelines is three(62), although other studies in children have suggested that five may be needed for better accuracy of results(71).

There are important disadvantages in using actigraphy for assessing sleep in children. Compared to polysomnography, actigraphy has a low sensitivity for detecting wakefulness. An alert child lying still in bed may be categorized as asleep based on motion counts, but if brain activity were to be measured, the child would be recognized as awake. In addition, movements during sleep, either real or artifact (for example, co-sleeping with an individual that moves or placing a wrist actigraph on the chest or

stomach where it will move with breathing motions), can be misinterpreted as being awake, and thus sleep quality can be underestimated. For children with sleep disorders, these opportunities for improper estimation can be an important drawback, but in healthy children this does not affect results(66, 72, 73). Third, some studies have shown that a small number of children consistently have differences in polysomnography and actigraphy results(67). To prevent misinterpretation of results, it is thus important to have a large subject population. Finally, despite the fact that actigraphy has been used to study sleep patterns for over 25 years, there is still considerable variability in how data is collected and processed. There are multiple actigraphic devices available along with varied algorithms and methods for interpreting data. In this setting, results among studies sometimes cannot be compared, limiting the usefulness of data. Despite these disadvantages, actigraphy has been accepted as a viable and useful alternative to polysomnography with numerous advantages as described (61, 63, 64, 74).

1.10 Results of Subjective Sleep Research

While studies within adult populations have failed to find a convincing correlation between short sleep and obesity, subjective studies with children often, but not always, report a positive cross-sectional association(17, 38, 60, 75-78). A meta-analysis by Cappuccio *et al* compiled 12 subjective studies and found that among 30,002 children aged 2- to 20-years world-wide, the odds ratio for short sleep duration and obesity was 1.89(79). A systematic review and meta-analysis by Chen *et al* compared 11 subjective studies from around the world, some of which Cappuccio had also analyzed, and concluded three main points. First, when compared to children with longer sleep

duration, children with shorter sleep duration had a 58% higher risk for being overweight or obese, and children with the shortest sleep duration had an even higher risk at 92%. This risk was reduced by 9% for each additional hour of sleep. Second, they noted a linear dose–response relationship between sleep and obesity risk in children under the age of 10 years. Third, results suggested boys had a stronger inverse association than girls (OR = 2.50 vs. 1.24)(80). Some longitudinal survey-based studies have analyzed future obesity risk by analyzing sleep duration at multiple ages. One study found that short sleep duration in predominantly White 6th graders was an independent risk factor for being overweight. They also noted that short sleep duration for these same children in 3rd grade, regardless of their weight status at that time, was an independent risk factor for being overweight in 6th grade(81).

Nevertheless, while the majority of subjective studies do support a correlation between short sleep duration and childhood obesity, there exist some that do not. A recent survey of 13,568 teens aged 12- to 18-years found that short sleep duration was not linked to obesity, although greater than 2 hours of television time per day and depression were linked(82). A cross sectional study in Ohio sought to determine if obesity severity was associated with short sleep in a population of 133 already obese adolescents. They collected sleep data by self-report as well as objectively via actigraphy and found no association between short sleep duration and BMI(83). In addition, Horne has argued that the link likely does exist, but that it is weak and takes years of short sleep to cause any change in BMI(84).

1.11 Results of Objective Sleep Research

Though limited in number, studies that have begun to look at short sleep duration and its relationship to obesity objectively suggest that a link does in fact exist. A study by Spruyt *et al* examines sleep patterns of 308 4- to 10-year-old children with wrist actigraphs over the course of one week. Researchers found that all children averaged 8 hours of sleep per night, but obese children were less likely to experience "catch- up" sleep on weekends. They also examined metabolic blood tests for a subset of participants and found that the combination of shorter sleep with variable sleep patterns was associated with adverse metabolic patterns developing even in non-obese participants, offering a potential mechanism of association(85). A prospective, longitudinal study in New Zealand looked at actigraphic data over five days and found that each additional hour of sleep at ages 3- to 5-years was associated with a reduction in BMI of 0.48 as well as a reduced risk of being overweight (0.39) at age 7 years(78). This study corroborates survey-based studies with similar predictive findings. A separate longitudinal study in New Zealand found that among 519 7-year-old children who wore a waist-bound actigraph for only one night, having fewer than 9 hours of sleep was an independent risk factor for being overweight/obese, unrelated to physical activity and levels of television time(86). This correlation has been shown true in minority adolescents as well: in a study of 471 adolescents (42.7% minority, predominantly African-American), shorter sleep duration measured via at least 3 nights of actigraphy was associated with decreased insulin sensitivity, minority race and increased BMI(87). In a cross sectional tri-ethnic sample of 383 adolescents aged 11- to 16-years (33.7% Non-Latino White, 36% African-American, 30.1% Latino), obese adolescents had significantly less sleep than their non-

obese peers, with the odds increasing by 80% for each hour of sleep lost. However, objective data was collected via actigraphy for only one night, thus while suggestive, the data are of limited value(88). No similar studies using objective measures have been done in younger minority children.

1.12 Considering the Timing of Sleep

A relatively new idea is that, in addition to short sleep duration, sleep timing, and specifically being late to bed, may be positively associated with increased weight and obesity. An Australian survey study compared 2,200 children aged 9- to 16-years in 4 groups based upon self-report: early to bed/early to rise, early to bed/late to rise, late to bed/early to rise, and late to bed/late to rise. They found that children who went to sleep late and woke up late were 1.5 times more likely to be obese than those who went to bed early and rose early. Of children with later bedtimes, 29% were overweight or obese while only 21% of children with earlier bedtimes were overweight or obese. They also noted that the late/late group watched about 48 minutes more of television and had 30 minutes fewer of vigorous physical activity per day as compared to the early/early children(89). "Chronotype" studies, which examine sleep timing preferences, have noted similarly that "morning-type" children have lower BMIs than "evening-type" children(90) and that "morning-type" children eat fewer calorically dense foods and caffeinated beverages(91). As these studies have been based on subjective evidence, conclusions cannot yet be drawn. In addition, these studies have focused on non-minority populations and so results cannot be generalized to a wider population.

1.13 Study Purpose

The overall purpose of this study is to determine if objective measurements of sleep duration and/or sleep timing are correlated with BMI percentiles and/or measures of physical activity in a group of 8- to 10-year-old Latino and African-American children living in a poor, urban community. We have chosen this population given that low-income minority children have the highest risks of obesity and its complications, yet objective data regarding their sleep is significantly lacking. In addition, very few studies have objectively examined sleep timing in relationship to obesity in children and very few studies have objectively examined sleep variables as related to physical activity in children.

2. Hypothesis

Shorter sleep duration and later bedtimes are significantly associated with higher BMI percentiles and lower levels of physical activity in children.

3. Specific Aims

Aim 1: To determine the relationship between sleep duration and BMI percentile.

Aim 2: To determine the relationship between sleep timing and BMI percentile.

Aim 3: To determine the relationship between sleep duration and daytime physical activity levels.

Aim 4: To determine the relationship between sleep timing and physical activity levels.

4. Methods

4.1 Participants

This study is a secondary analysis of data collected between September 2004 and June 2005 from 113 children between the ages of 8 and 10 years of age. Approval for this study was obtained from the Human Investigation Committee at the Yale School of Medicine. The present study did not gather any new data, but instead re-examined this previously collected data in regards to the children's sleep patterns as well as their physical activity. Subjects were enrolled to participate in a study assessing physical activity from a single community health center in New Haven, Connecticut. The parents of a total of 222 children were approached by their clinicians and 175 agreed to discuss the study with an on-site researcher. Participants were excluded if they had neurological or orthopedic impairments, an acute illness that might limit physical activity, attention deficit hyperactivity disorder or other conduct disorder (n=35). Twenty-seven declined

participation. Written informed consent from parents and verbal assent from children were obtained prior to enrollment.

4.2 Demographics and Anthropometrics

Sex, age, race, insurance coverage, and ethnicity were obtained per parent report. Weight was measured to the nearest 0.1 kg using a Seca digital scale (Hamburg, Germany). Height was measured to the nearest 0.1 cm using a stadiometer (Hamburg, Germany). Height and weight were measured twice and the values were averaged. BMI (kg/m^2) was calculated from the average height and weight values. BMI percentile was determined using the Centers for Disease Control and Prevention (CDC) growth charts. Children were categorized as non-overweight (NOW, $< 85^{th}$ BMI percentile for age and sex, overweight (OW, $\geq 85^{th}$ and $< 95^{th}$ BMI percentile for age and sex), and obese (OB $\geq 95^{th}$ BMI percentile for age and sex).

4.3 Actigraphy

Each participant was fitted with an Actical accelerometer (Minimitter, Bend, OR). The device was affixed to an elastic belt and worn at the waist. Participants were instructed to wear the belt and attached device all day and all night for 7 days, removing it only for periods of swimming or bathing (actual average length of proper wear = 8.26 nights, range of 5 to 14 nights). Participants were excluded from analysis if fewer than 5 days of complete daytime data (defined as at least 12 hours of motion data between 7:00 AM and 11:00 PM on weekdays and between 6:00 AM and 11:00 PM on weekend days) and/or fewer than 5 nights of complete nighttime data (defined as having clear evening

and morning transitions of sleep and wake as well as overnight activity to verify its status of being worn as opposed to removed during sleep)(n=9).

4.4 Actigraphic Sleep Assessment

A total of 104 participants provided at least 5 days and nights that met criteria for inclusion in analysis. All data were automatically converted to counts per minute, and subsequently manually analyzed by one rater. Sleep logs were not obtained during the collection of study data given its initial purpose for daytime physical activity assessment, thus sleep analysis was performed by visually evaluating graphic representation of motion in conjunction with examining activity counts per minute (Figure 1). The following measures of sleep were assessed: sleep-onset (defined below), sleep-offset (defined below), sleep time (time between sleep-onset and sleep-offset), wake after sleep-onset (WASO, number of minutes scored as wake after sleep-onset and before sleep-offset), and sleep duration (sleep time minus minutes of WASO). Sleep-onset was defined as the first minute of the first 5 consecutive minutes of zero counts that remained sustained, as suggested in previous studies(69, 70). Activity counts during sleep time were evaluated at 3 different thresholds: ≥20, ≥40, and ≥80 activity counts per minute. These values represent standard thresholds commonly used in actigraphic sleep software and represent low, medium, and high sensitivities for detecting sleep(69, 70, 73). Minutes during which activity counts exceeded each of the 3 thresholds for at least 2 consecutive minutes were summed and subtracted from each night's total sleep time (and thus qualified as WASO). Any activity lasting less than 2 minutes was presumed motion during continued sleep. Sleep-offset was assessed by determining definite awake period,

defined as at least 5 consecutive minutes of activity greater than 80 activity counts per minute, and working backwards to find the last 5 consecutive minutes of sleep. The last minute of that period was defined as sleep-offset(69, 70). Weekend data was obtained from Friday and Saturday nights. Every child had at least 1 weekend night of data included for analysis (7 had 1 night; average of 2 nights, range of 1-3 nights).

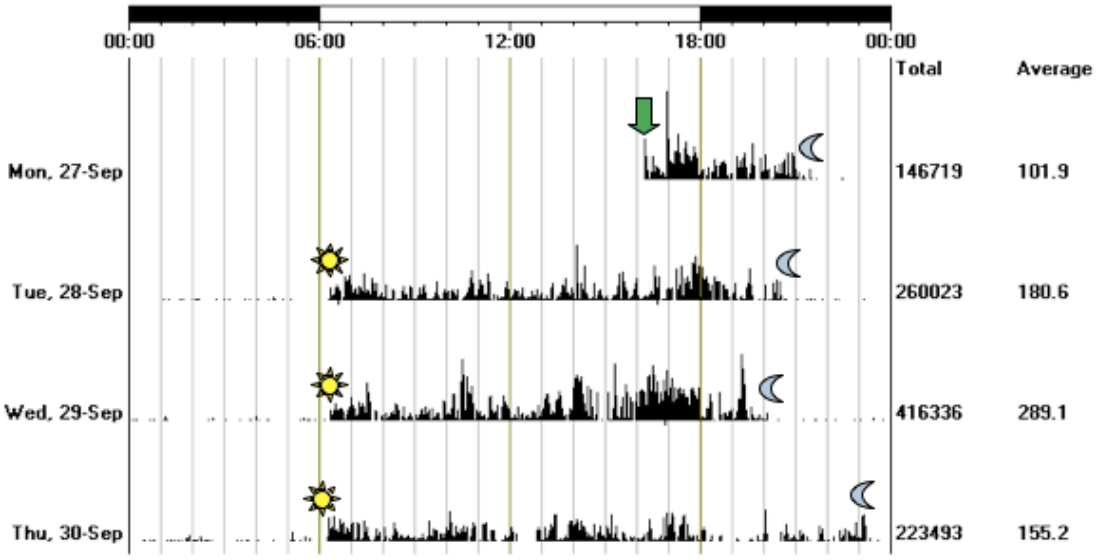


Figure 1. Sample Actigraphy*

*This figure represents the first 4 days of graphic activity for one participant. The date is in the left hand column, time of day is represented horizontally and is labeled at the top, and activity counts per epoch (set at 1 minute) are represented vertically as black bars, with increasing height of bars reflecting higher activity counts per epoch. The right hand column displays total activity counts for the 24 hour period and the average activity count per epoch.

From this graph, we can determine that the participant was fitted with an actigraph soon after 16:00 on 9/27 (Marked ⬇). Sleep-onset is marked ☾ and sleep-offset is marked ☼ Sleep-onset the first night is estimated around 21:45 and is determined to the minute by looking at exact activity counts in an Excel file of this data. Sleep-offset on the morning of 9/28 is estimated around 6:15, and is again determined by looking at exact activity counts. Very small activity counts during sleep represent movement and confirm that the participant is wearing the belt overnight. On 9/28, sleep-onset is estimated around 20:45, and sleep-offset around 6:15 the following morning. On 9/29, sleep-onset is estimated around 20:10 with sleep-offset the following morning around 6:15. On 9/30, sleep-onset occurs later, estimated around 23:15. The remainder of the file is not shown.

4.5 Actigraphic Physical Activity Assessment

All daytime motion data were collected, calculated, and analyzed as previously described(92, 93). Details on how these variables were defined can be found in the original publication(94). For the purpose of this study, we re-examined 4 measures of physical activity: daily minutes of moderate and vigorous physical activity (MVPA), daily number of bouts of MVPA (defined as MVPA sustained for > 1 minute), daily minutes of vigorous physical activity (VPA), and daily number of bouts of vigorous physical activity (defined as VPA sustained for > 1 minute)(94).

4.6 Statistics

Descriptive analysis of participant characteristics was performed using proportions for categorical variables (gender, race/ethnicity, insurance) and means and standard deviations for continuous variables (age, BMI, BMI percentile). We looked for potential differences in subject characteristics among weight groups by using chi-square for categorical variables and Student t-tests for continuous variables. We also calculated means and standard deviations for sleep variables including sleep-onset, sleep-offset, WASO, and sleep duration. There were reported separately for the whole sample, for weekdays compared to weekend days, for females compared to males, and for NOW compared to OW/OB participants. Next, we performed Student t-tests to determine whether each of the sleep variables (mean sleep durations calculated by subtracting minutes of WASO based on each of the 3 different activity count thresholds [20, 40, 80], sleep-onset, sleep-offset, and WASO at each of the 3 activity thresholds) differed according to sex or weight groups (NOW compared with OW/OB). To determine

whether or not there were differences in categories of average sleep duration (<9 hours, 9-9.49 hours, and >=9.5 hours) based on weight group, we used a 3-category chi-square test. We also performed simple linear regressions to determine the association between these same sleep variables and BMI percentiles in the whole sample. We performed these analyses to determine whether there were differences in mean sleep duration, sleep-onset, sleep-offset, and/or WASO for different sexes or weight groups or an association between any of these variables and BMI percentile in the sample.

For physical activity assessments, we used descriptive statistics to determine the mean daily MVPA, mean daily VPA, and mean daily number of bouts of MVPA and VPA. Finally, we examined the associations between these 4 physical activity variables and 3 sleep variables (mean sleep duration, sleep-onset, and sleep-offset) using simple linear regression. All analyses were performed using Microsoft Excel 2007.

When we examined average sleep duration based on subtracting minutes of WASO derived from the three activity thresholds (20, 40, and 80 activity counts per minute), our results did not differ aside from absolute values as shown, and thus we based our discussed sleep durations on a conservative 80 counts per minute threshold.

4.7 Explanation of the Student's Involvement

All participants were enrolled, anthropometric data was collected, and actigraphs were fitted and collected prior to the student's involvement. The student developed the hypothesis and aims of the study, re-examined all raw actigraphic data, and manually evaluated all sleep parameters to create new sleep variables. The student performed

statistical analyses of descriptive data, Student t-tests, chi-square calculations, and linear regressions and prepared this original manuscript under the guidance of the thesis advisor.

5. Results

5.1 Participants

Our sample of participants was made up predominantly of Latino and African-American children receiving state or Medicaid insurance. Slightly more than half were female, and about half were overweight or obese. Overall, there were no significant differences in demographic characteristics when comparing NOW versus OW/OB participants (Table 1).

Table 1. Participant Characteristics*

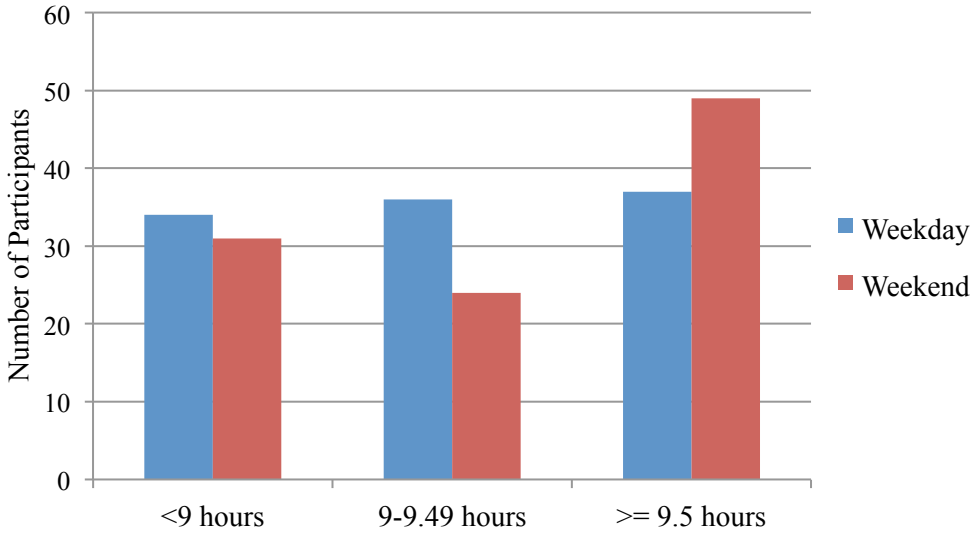
	NOW	OW/OB	Total
N (%)	50 (48.1)	54 (51.9)	104
Age, Mean in years (SD)	9.3 (0.9)	9.5 (0.9)	9.4 (0.9)
Female, N (%)	32 (64)	26 (48.1)	58 (55.8)
BMI, kg/m^2, Mean (SD)**	16.7 (1.4)	23.5 (3.4)	20.3 (4.3)
BMI Percentile, Mean (SD)**	50.6 (21.2)	94.7 (3.7)	73.5 (26.7)
Race/Ethnicity, N (%)			
Black	5 (10)	8 (14.8)	13 (12.5)
Latino	38 (76)	41 (75.9)	79 (76)
White	1 (2)	4 (7.4)	5 (4.8)
Multi-ethnic	5 (10)	1 (1.9)	6 (5.7)
Other	1 (2)	0 (0)	1 (1)
Insurance, N (%)			
Medicaid	38 (76)	45 (83.3)	83 (79.8)
Uninsured	6 (12)	3 (5.6)	9 (8.7)
Private	6 (12)	6 (11.1)	12 (11.5)

*This table compares participant characteristics among non-overweight (NOW) and overweight (OW)/obese (OB) children. There were no statistically significant differences between weight groups with respect to age, gender, race/ethnicity, or insurance type. Note: N = number. SD = Standard Deviation.
**Differences in body mass index (BMI) and BMI percentile were significant ($P < 0.0001$). There were 20 overweight (7 male and 13 female) and 34 obese (21 male and 13 female) participants.

5.2 General Patterns of Sleep

Participants averaged 9.3 hours (9 hours: 18 minutes) of sleep across all nights, with no significant differences between sexes. Across all nights, 6.7% of children obtained the recommended 10-11 hours of sleep (28.6% female; 57% NOW). On weekdays alone, 7.7% of children obtained the recommended sleep (25% female; 62.5% NOW). On weekends alone, 14.4% of children obtained the recommended sleep (64% female; 48% NOW) and 3.8% obtained more than recommended (75% female; 50% NOW). Sleep duration was shorter on weekdays as compared to weekends (Graph 1). Average sleep-onset was 22:10 (or 10:10 PM). On weekends, all participants went to sleep later, averaging sleep-onset at 22:51 (10:51 PM). On weekdays females went to sleep significantly later than males at a difference of 25 minutes (P=0.04). Sleep-offset averaged 7:36 AM for all participants across all nights, though on both weekdays and weekends, females slept in longer than males, averaging 23 and 24 minutes later, respectively (P=0.02). On weekends, all participants slept in with an average sleep-offset at 8:27 AM, with no significant difference between sexes. As expected, WASO decreased with the increased activity threshold, with females having small but significantly more WASO on all nights using 40 count threshold (12 versus 10 minutes) and on weekends using the 80 count threshold (10 versus 7 minutes)(Table 2).

Graph 1. Categories of Sleep Duration for All Participants on Weekdays versus Weekends*



*This graph demonstrates categories of average sleep duration for all participants on weekdays versus weekends. Sleep duration is determined based on 80 Count Threshold.

Table 2. General Sleep Characteristics for All Participants*				
	All	Females	Males	P-value**
Sleep Duration in Hours, Mean (SD)				
All Nights, 20	9.13 (0.51)	9.15 (0.48)	9.1 (0.56)	0.65
Weekdays, 20	9.08 (0.62)	9.07 (0.56)	9.1 (0.69)	0.83
Weekends, 20	9.25 (0.92)	9.35 (0.91)	9.13 (0.92)	0.23
All Nights, 40	9.22 (0.51)	9.25 (0.47)	9.2 (0.56)	0.61
Weekdays, 40	9.17 (0.63)	9.16 (0.56)	9.18 (0.71)	0.86
Weekends, 40	9.36 (0.92)	9.46 (0.92)	9.24 (0.92)	0.22
All Nights, 80	9.3 (0.51)	9.33 (0.47)	9.26 (0.57)	0.51
Weekdays, 80	9.24 (0.63)	9.24 (0.55)	9.24 (0.72)	0.98
Weekends, 80	9.45 (0.93)	9.56 (0.93)	9.32 (0.92)	0.20
Sleep-Onset in Military Time (SD in Hours:Min)				
All Nights	22:10 (0:57)	22:19 (0:57)	21:59 (0:56)	0.08
Weekdays	21:55 (1:00)	22:06 (0:57)	21:41 (1:01)	0.04‡
Weekends	22:51 (1:13)	22:54 (1:14)	22:49 (1:13)	0.73
Sleep-Offset in Military Time (SD in Hours:Min)				
All Nights	7:36 (0:54)	7:46 (0:56)	7:23 (0:50)	0.03‡
Weekdays	7:15 (0:55)	7:26 (0:57)	7:02 (0:50)	0.02‡
Weekends	8:27 (1:13)	8:37 (1:13)	8:15 (1:11)	0.13
WASO in Minutes, Mean (SD)				
All Nights, 20	17 (6.44)	18 (5.99)	16 (6.77)	0.05
Weekdays, 20	16 (7.11)	17 (6.56)	14 (7.6)	0.1
Weekends, 20	21 (11.18)	22 (11.7)	18 (10.23)	0.07
All Nights, 40	11 (4.91)	12 (4.65)	10 (5)	0.02‡
Weekdays, 40	10 (5.34)	11 (5.17)	9 (5.4)	0.06
Weekends, 40	14 (8.87)	15 (9.47)	12 (7.77)	0.05
All Nights, 80	7 (3.56)	7 (3.45)	6 (3.6)	0.05
Weekdays, 80	6 (3.67)	7 (3.65)	6 (3.67)	0.19
Weekends, 80	8 (7.04)	10 (7.37)	7 (6.31)	0.03‡

*This table describes mean baseline sleep characteristics (sleep duration at 3 activity count thresholds: 20, 40, 80; sleep-onset; sleep-offset; and wake after sleep onset (WAS0) for the same 3 activity count thresholds) in total as well as divided into weekdays and weekends for all individuals as well as split into sexes. Note: SD = Standard Deviation. Min = Minutes.

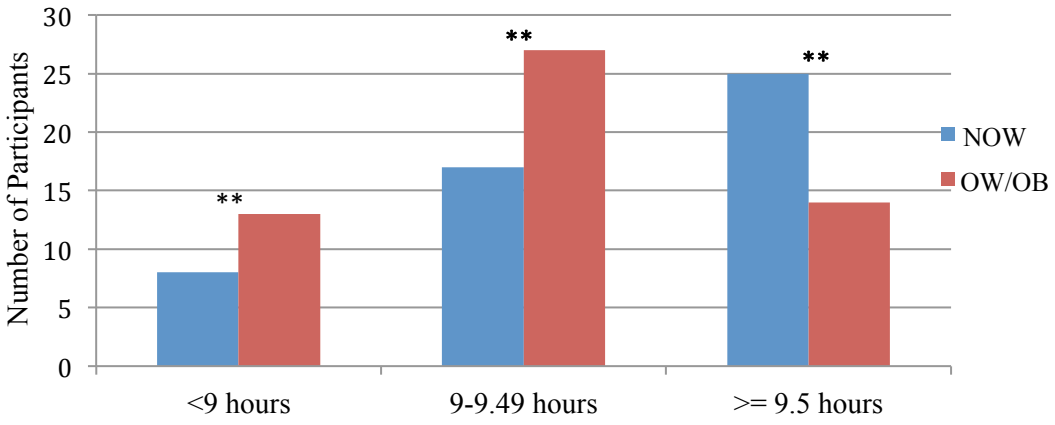
**P-values are calculated as female/male comparisons across rows via student t-test.

‡A P-value < 0.05 is considered significant.

5.3 Sleep and Weight Status

In comparing the 2 weight groups (NOW and OW/OB) across 3 categories of sleep duration (<9, 9-9.49, and >=9.5 hours), a greater proportion of NOW participants slept significantly longer than their OW/OB peers across all nights (P=0.04)(Graph 2). In addition, total sleep duration was significantly associated with BMI percentile via linear regression (Coefficient=-0.005, SE=0.002, P=0.015). On weekdays, NOW again had significantly more sleep than their OW/OB peers via regression calculation (12.6 minute difference; Coefficient=-0.005, SE=0.002, P=0.019). On weekends, sleep durations were not significantly different between weight groups (Coefficient=-0.003, SE=0.003, P=0.335). Both weight groups had similar WASO counts (Table 3). Although there were trends for NOW participants to have earlier sleep-onset across all nights, later sleep-offset on weekdays, and earlier sleep-offset on weekends when compared to OW/OB participants, differences were not statistically significant. These results remained non-significant in linear regression models (Table 4).

Graph 2. Categories of Mean Sleep Duration in NOW versus OW/OB Participants*



*This graph demonstrates categories of mean sleep duration in non-overweight (NOW) versus overweight (OW)/obese (OB) participants across all nights. Sleep duration is determined based on an 80 Count Threshold. No child had an average duration >11 hours.

**Differences in NOW versus OW/OB groups are statistically significant via 3 category chi-square test (P=0.04). A $P < 0.05$ is considered significant.

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Table 3. Sleep Duration and WASO in NOW versus OW/OB Participants*			
	NOW	OW/OB	P-value**
Sleep Duration in Hours, Mean (SD)			
All Nights	9.39 (0.48)	9.22 (0.54)	0.09‡
Weekdays	9.35 (0.62)	9.14 (0.63)	0.08‡
Weekends	9.49 (0.88)	9.41 (0.97)	0.66
WASO in Minutes, Mean (SD)			
All Nights, 20	18 (6)	17 (6.7)	1
Weekdays, 20	16 (7.14)	15 (7.07)	0.3
Weekends, 20	20 (10.65)	21 (11.76)	0.88
All Nights, 40	11 (4.74)	11 (5.11)	0.86
Weekdays, 40	11 (5.34)	10 (5.36)	0.75
Weekends, 40	13 (8.77)	14 (9.02)	0.6
All Nights, 80	7 (3.55)	7 (3.57)	0.3
Weekdays, 80	6 (3.65)	6 (3.71)	0.61
Weekends, 80	8 (6.98)	9 (7.11)	0.42

*This table demonstrates mean sleep duration and mean wake after sleep onset (WASO) minutes at 3 activity count thresholds across all nights, weekdays only, and weekends only in non-overweight (NOW) versus overweight (OW)/obese (OB) participants. All sleep duration data is based on 80 Count Threshold. Note: SD = Standard Deviation.

**P-values are calculated comparing NOW and OW/OB across rows via Student t-test.

‡ These P-values are non-significant via Student t-test, but are significant when comparing sleep duration to Body Mass Index percentile via regression: All nights: (Coefficient=-0.005, SE=0.002, P=0.015), Weekdays: (Coefficient=-0.005, SE=0.002, P=0.019). A P-value < 0.05 is considered significant.

Table 4. Sleep Timing in NOW versus OW/OB Participants*				
	NOW	OW/OB	P-value**	Regression‡
Sleep-Onset				
All Nights	22:08 (1:04)	22:12 (0:50)	0.74	CE=0.0003 SE=0.0006 P=0.61
Weekdays	21:53 (1:05)	21:56 (0:55)	0.82	CE=0.0003 S=0.0006 P=0.65
Weekends	22:42 (1:22)	23:00 (1:03)	0.21	CE=-0.002 SE=0.001 P=0.09
Sleep-Offset				
All Nights	7:38 (1:04)	7:34 (0:45)	0.74	CE<-0.001 SE<0.001 P=0.72
Weekdays	7:20 (1:06)	7:10 (0:43)	0.36	CE=-0.0001 SE=0.0001 P=0.29
Weekends	8:19 (1:20)	8:34 (1:06)	0.32	CE=0.0002 SE=0.0002 P=0.26

*This table compares mean sleep-onset and sleep-offset timing in non-overweight (NOW) versus overweight (OW)/obese (OB) participants. All times are in Military time (SD in Hours:Min). Note: SD = Standard Deviation. Min = minutes. CE = Coefficient. SE = Standard Error. P = P value.
**P-values compare sleep times for NOW versus OW/OB participants across rows via Student t-test.
‡Regression calculations compare sleep timing and Body Mass Index Percentile.

5.4 Weight Status, Sleep and Physical Activity

During the day, females and males spent comparable amounts of time in each of the 4 measures of physical activity: daily minutes of MVPA, bouts of MVPA, daily minutes of VPA, and bouts of VPA (Table 5). As previously described (92, 93), NOW participants had higher levels of each of the 4 physical activity measures. These results were not significant when compared across the 2 weight groups via Student t-test, but significant differences were seen when comparing daily minutes of VPA, bouts of MVPA, and bouts of VPA to BMI percentile via regression (Table 6). However, sleep duration was not significantly associated with any of the 4 physical activity measures. Similarly, sleep timing was not significantly associated with any of the physical activity measures (Table 7).

Table 5. General Daily Physical Activity Characteristics for All Participants*

	All	Females	Males	P-Value**
MVPA in Min, Mean (SD)	120.31 (50.4)	122.72 (48.4)	117.27 (53.2)	0.59
Bouts of MVPA, Mean (SD)	99.8 (57.95)	101.23 (57.8)	97.98 (58.72)	0.78
VPA in Min, Mean (SD)	24.28 (20)	25.18 (21.29)	23.14 (18.41)	0.61
Bouts of VPA, Mean (SD)	14.23 (16.81)	15.4 (18.83)	12.76 (13.65)	0.43

*This table demonstrates the mean daily minutes spent in moderate and vigorous physical activity (MVPA) and vigorous physical activity (VPA) as well as mean daily number of bouts of MVPA and VPA for all participants and based on sex. Note: Min = minutes. SD = Standard Deviation.
**P-values compare female/male sex across rows via Student t-test.

Table 6. Daily Physical Activity Characteristics in NOW versus OW/OB Participants*				
	NOW	OW/OB	P-value**	Regression‡
MVPA in Min, Mean (SD)	130 (51.8)	111.32 (47.49)	0.06	CE=-0.36 SE=0.18 P=0.05
Bouts of MVPA, Mean (SD)	110.74 (60.62)	89.65 (53.95)	0.06	CE=-0.43 SE=0.21 P=0.04†
VPA in Min, Mean (SD)	27.47 (22.51)	21.32 (17.04)	0.12	CE=-0.15 SE=0.07 P=0.04†
Bouts of VPA, Mean (SD)	17.59 (20.04)	11.12 (12.55)	0.05	CE=-0.16 SE=0.06 P=0.01†

*This table demonstrates mean daily minutes spent in moderate and vigorous physical activity (MVPA) and vigorous physical activity (VPA) as well as mean daily number of bouts of MVPA and VPA for now-overweight (NOW) versus overweight (OW)/obese (OB) participants. Note: Min = Minutes. SD = Standard Deviation. CE = Coefficient. SE = Standard Error. P = P value.

**P-values compare NOW versus OW/OB participants across rows via Student t-test.

‡Regression calculations compare physical activity measures to Body Mass Index percentile.

† A P value < 0.05 is considered significant.

Table 7. Sleep Duration and Timing Compared to Physical Activity Measures*				
	MVPA	Bouts of MVPA	VPA	Bouts of VPA
Sleep Duration				
All Nights	CE=-0.0006 SE=0.001 P=0.548	CE=1E-5 SE=0.0009 P=0.991	CE=0.002 SE=0.003 P=0.489	CE=0.003 SE=0.003 P=0.261
Weekdays	CE=8.21E-5 SE=0.001 P=0.948	CE=0.0006 SE=0.001 P=0.582	CE=0.003 SE=0.003 P=0.336	CE=0.005 SE=0.004 P=0.207
Weekends	CE=-0.002 SE=0.002 P=0.258	CE=-0.001 SE=0.002 P=0.477	CE=0.0006 SE=0.005 P=0.897	CE=0.0005 SE=0.005 P=0.92
Sleep-Onset				
All Nights	CE=3.48E-5 SE=7.86E-5 P=0.659	CE=5.85E-5 SE=6.82E-5 P=0.393	CE=0.0002 SE=0.0002 P=0.227	CE=0.0003 SE=0.0002 P=0.248
Weekdays	CE=3.11E-5 SE=8.2E-5 P=0.705	CE=5.18E-5 SE=7.12E-5 P=0.469	CE=0.0002 SE=0.0002 P=0.283	CE=0.0002 SE=0.0002 P=0.313
Weekends	CE=-5.5E-5 SE=0.0001 P=0.584	CE=-8.1E-6 SE=8.72E-5 P=0.926	CE=5.6E-5 SE=0.0002 P=0.825	CE=8.89E-5 SE=0.0003 P=0.768
Sleep-Offset				
All Nights	CE=-1.4E-5 SE=7.49E-5 P=0.856	CE=4.08E-5 SE=6.5E-5 P=0.532	CE=0.0003 SE=0.0002 P=0.151	CE=0.0004 SE=0.0002 P=0.097
Weekdays	CE=3.87E-5 SE=7.59E-5 P=0.612	CE=7.88E-5 SE=6.57E-5 P=0.233	CE=0.0003 SE=0.0002 P=0.067	CE=0.0004 SE=0.0002 P=0.051
Weekends	CE=-0.0001 SE=9.87E-5 P=0.147	CE=-6E-5 SE=8.66E-5 P=0.492	CE=2.85E-5 SE=0.0003 P=0.91	CE=0.0001 SE=0.0003 P=0.73

*This table demonstrates regression analyses comparing sleep variables listed in the left-hand column (Sleep Duration, Sleep-onset, and Sleep-offset across all nights, weekdays, and weekends) with physical activity measures listed across the top row (moderate and vigorous physical activity (MVPA), bouts of MVPA, vigorous physical activity (VPA), and bouts of VPA). All calculations are not statistically significant. All sleep durations are based on an 80 Count Threshold. Note: CE = Coefficient. SE = Standard Error. P = P-value.

6. Discussion

6.1 Main Findings

Our study is one of only a few to look objectively at the relationship between sleep and BMI in minority children living in low-income communities. In addition, it is one of even fewer to compare these factors to objective measures of physical activity. We found that few children obtained recommended amounts of sleep in this population regardless of their weight status. However, overweight and obese children had slightly shorter sleep compared with non-overweight peers. Despite this pattern of shorter sleep and higher BMI, sleep-onset, sleep-offset, and WASO did not significantly differ between weight groups. In addition, none of the measures of physical activity were linked to our sleep variables.

Our finding that short sleep duration was associated with higher BMI, more so on weekdays than weekends, was statistically significant but weak as evidenced by a small regression coefficient. Other studies have found stronger associations between short sleep and BMI(79, 80), which may be explained by a couple of factors. There may be fewer low-income minority children who obtain longer sleep durations. In our study, the average sleep duration was about an hour shorter than what is recommended with very few children obtaining more than 10 hours of sleep. Thus, there was little variation in sleep duration, making identifying causes of differences difficult in our small sample. Other studies have shown shorter sleep duration among minority children compared with White peers. For example, one study that compared White and Latino children found that, on average, Latino children slept about 12.7 minutes fewer each night regardless of weight status(45). It is also important to note that in our study, OW/OB children had

significantly less sleep on weekdays but on weekends all children had comparable longer sleep durations. This finding is in direct contrast to one study that noted all children, who wore actigraphs for 1 week, had similar sleep durations during the week but that obese children had less "catch up" sleep on weekends(73). Sleep timing was not discussed by the authors, so comparing sleep patterns with our study is not possible. In addition, the population evaluated in that study lived in Kentucky and had only 20% African-American children, and 10% "Other." Thus, it is possible that sampling and environmental differences between the 2 groups explain this variation in "catch up" sleep on weekends based on BMI.

We also noted that the majority of children in our study did not obtain the recommended 10 to 11 hours of nightly sleep, even during longer weekend sleep. This finding is concerning not only because of how it may influence weight status, but also because sleep loss in childhood has been linked to many adverse consequences. This list includes but is not limited to: impaired concentration and learning, impaired academic performance, attention deficit hyperactivity disorder, mood disorders such as anxiety and depression, impaired motor skills, increased alcohol and drug use, increased risk of accidents and injuries, and impaired overall health and immune function(38). Our estimates of sleep duration (which range from 9.13 to 9.3 hours depending on which threshold of activity one uses) are similar to most estimates of sleep duration for children in this age group of varying ethnicities, though slightly higher than other objective estimates. A sampling of subjective studies have the following estimates: 9.8 hours for 8-year-old mostly White children(46); 9.55 hours in a national sample of 3rd graders and 8.97 for these same children in 6th grade(81); and 9.4, 9.2 and 9.1 hours in a national

survey of children aged 8, 9, and 10 years respectively(95). A sampling of objective studies have lower estimates of average sleep duration: 8.5 hours for 8-year-old Finnish children(96), 8 hours for a sample of 50% White, 50% Latino children aged 6- to 11-years(56); 9.1 hours and 8.2 hours for 8- to 9- and 10- to 11-year-old White children, respectively(97); and 8 hours in a sample of White and African-American 4- to 10-year-old children(85). These averages are affected by many factors, some within and some beyond control of the studies obtaining them, from method of sleep measurement, to combining weekday and weekend estimates, to demographic and socioeconomic factors. Understanding how these factors fit together makes it difficult to understand how to interpret absolute estimates of sleep duration. Nevertheless, one major theme does emerge: the majority of school-aged children in the U.S. do not reach the 10-11 hour sleep recommendation.

Sleep-timing patterns and WASO observed in our study were not significantly associated with BMI. In this setting, small changes in more than one of these sleep variables compounded to create a significant difference in sleep duration when compared to BMI. Non-significant trends from our data suggest that the longer sleep obtained by NOW children resulted from both slightly earlier bedtimes and slightly later wake times during the week. This result differs somewhat from chronotype studies, which have generally found that early-to-bed/early-to rise children are the slimmest(89, 90). In a setting where all children do not obtain enough sleep, it is possible that these chronotype patterns are not able to fully emerge. It is also possible that these differences are reflective of the populations sampled since most studies have a predominance of White children from more affluent families. However, the sleep-timing differences between

weight groups found in our study were statistically non-significant, so further information would be needed prior to drawing any conclusions. Weekday sleep timing was slightly but significantly different between sexes, however, with females both going to bed and waking up slightly later, leading resultant sleep durations to be similar. Some studies have noted that the association between short sleep and increased BMI in children is stronger in males, but this finding has been inconsistent, and differences in sleep timing do not seem to be noted, but are also not often reported(80). Gender differences aside, the sleep-onset and sleep-offset times themselves in our sample are similar to other estimates of sleep timing for this age group. The 2004 Sleep in America Poll, a parent-report based survey, estimated weekday bedtimes at 9:03 PM, 9:21 PM, and 9:26 PM for 8-, 9-, and 10-year-olds, respectively(95). These early times likely reflect some degree of inaccurate estimation by parents. Objective studies, on the other hand, have found estimates close to our own, ranging from 10:06 PM for 8-year-olds to 10:42 PM for 6th graders(41). Weekday sleep-offset estimates from the parent-based survey were more similar to our own findings as well as those of others: the poll noted 7:06 AM, 7:06 AM, and 6:59 AM wake times for the same ages, while objective studies have noted estimates of 7:26 AM for 8-year-olds and 6:54 AM for 6th graders. It is not surprising that sleep-offset times are more similar given that children have similar timing for the start of the school day. Differences in bedtime, however, may reflect different personal patterns as well as home and family environments.

Our study is one of only a few to compare objective sleep variables to objective daytime physical activity in children, and, similar to most, we found no association between any of the sleep variables (sleep duration, sleep-onset, and sleep-offset) with any

of the physical activity measures (average daily amount of MVPA, VPA, bouts of MVPA, or bouts of VPA). Since separate studies have shown that both short sleep and physical activity are associated with BMI, our result appears to delineate the two as independent risk factors for obesity(11, 79, 80, 93). In contrast, studies that assess physical activity subjectively do tend to find a link between shorter sleep and physical inactivity. For example, a survey of 68,288 U.S. children aged 6- to 17-years found that children experiencing inadequate sleep had a 55% higher odds of physical inactivity(98). Another survey-based study of 7- to 9-year-old Portuguese children also found a link between short sleep and less daytime physical activity(99). A study of Australian adolescents generated more complex results: they found that early-risers spent more minutes in MVPA than late-risers, as assessed via an activity recall software (89). They also found that when comparing late-to-bed/late-to-rise adolescents (who had significantly higher BMIs) to their early-to-bed/early-to-rise peers, the late/late group had significantly fewer minutes spent in MVPA, despite having similar sleep durations. In our smaller sample, sleep times were less variable, making such group comparisons not possible. Sleep timing in this population of adolescents also varied considerably from ours, suggesting that the age difference may make comparison of results more complicated depending on parental control. However, the few objective studies have been less clear, and more often reflect results similar to our own. One study prospectively followed 150 children from birth to 9.5 years in order to identify risk factors for becoming OW/OB. Shorter sleep at ages 3 through 5 years, almost all of which resulted from less daytime napping, predicted future weight gain, and short sleep at these ages was also associated with concurrent decreased physical activity(100). This

study found no association between physical activity and weight status, possibly because they did not separate types of physical activity (i.e. moderate versus vigorous). Nevertheless, the link that they noted between shorter daytime sleep and less overall physical activity may reflect this younger age group when compared to our own. A more recent study in Finland that specifically focused on sleep and physical activity found, contrary to their hypothesis, that children who slept less had higher levels of physical activity. Additionally, they noted a higher level of physical activity during the day was significantly associated with poorer sleep that night (defined by more nighttime awakenings), and that poorer sleep during the night was significantly associated with higher levels of activity the following day(96). The authors questioned whether innate levels of physical activity are reflected not only in increased daytime activity but also in increased nighttime activity. Our study is not able to comment on this hypothesis because there was little variability in sleep quality between participants as evidenced by similar WASO minutes. The two other objective studies found results similar to our own: one assessed White children in Quebec with an average age of 9.5 years(101) and the other assessed 7-year-olds in New Zealand(86). Both found that a 7-day actigraphy trial failed to find any association between short sleep and physical activity, although they did find an association between shorter sleep and increased BMI. These varying results may reflect population differences, but objective studies generally agree that the link between physical activity and sleep is not direct.

6.2 Strengths and Weaknesses

Our study has some important strengths as well as limitations. Our study analyzes an important clinical question within an often-missed subpopulation of children who are at the highest risk for developing obesity and its complications(4, 15). Given the complexity of treating obesity, it is critical that we understand the differences that may exist within variable patient populations in order to provide targeted and effective care. Additionally, we collected objective sleep data over the course of multiple days, with both weekend and weekday comparisons for each child. We have included sleep-timing variables, which are often not reported but in fact are vital in understanding the characteristics of short sleep patterns and what recommendations would be most efficient at addressing the problem. Furthermore, comparing objective sleep data with objectively measured physical activity data is an angle of research that has yet to be fully explored in children. The distinction between whether or not short sleep and decreased physical activity are associated is important because if they are independent risk factors, they need to be addressed separately via targeted strategies.

Despite these strengths, there are weaknesses to consider. We did not ask our study participants to maintain a sleep/wake log to correlate data from actigraphy due to the initial purpose of this data to assess the relationship between physical activity and obesity. Thus, our assessment of sleep intervals could not be verified using parent-report of sleep patterns, potentially resulting in inaccurate sleep-onset or sleep-offset times. This weakness would result in our nighttime sleep duration estimations to be potentially longer or shorter than they actually were. However, there is no reason to suspect bias in assessment of sleep timing based on BMI percentile. Any error in assessment would be

distributed randomly across weight groups and therefore likely not affect our results. While such error is not ideal, studies suggest that the assessment of sleep duration with and without sleep logs may introduce small differences that may be statistically but not clinically significant(70). Without logs, we also were unable to measure daytime sleep. This weakness is actually not unique to our study and in fact studies in this age group rarely measure daytime sleep. However, given that minority children have been shown to continue napping at older ages(45, 46, 102), this weakness may be more pertinent to our study. Without other comparison studies, we cannot determine to what degree this would have affected our results. Third, actigraphs in general have their own disadvantages as discussed earlier(61, 64), and in the present study one rater analyzed actigraphy data manually. This method was beneficial in order to eliminate rater variability, but it means that our methods may have introduced human error. Nonetheless, there exist several studies that analyze manual sleep assessment with actigraphs, and thus its use is clearly a viable option(69, 70). In addition, when sleep intervals were assessed the rater was blind to the weight group of the subject. Thus, any error introduced would likely be evenly distributed among weight groups and not introduce bias into our analysis. Fourth, the population of children in this study is representative of some communities including our own, but is not representative of the U.S. as a whole. This limits the ability to generalize our results. Fifth, our sample size of 104 children limits us in detecting small differences, such as a potential relationship between sleep-onset or sleep-offset and BMI percentile, as well as investigating significant but weak findings. Lastly, studying sleep patterns in overweight and obese children may be complicated by comorbidities of sleep disordered breathing. The prevalence of these diseases during this period of

childhood is very low, even within studied Latino populations(103), and the fact that our WASO estimates remained low suggest that this possibility was not likely to play a large role in our results.

6.3 Implications

Our results highlight the importance of advocating for longer sleep for all children, particularly by encouraging earlier bedtimes. This is especially important for overweight or obese children, or those at risk for becoming so, and minority children given heightened risk profiles. Even for non-overweight children, decreased sleep can lead to concurrent hormonal imbalances that can lead to future weight problems and resultant complications(38). How to help parents attain this goal for children requires multiple strategies, which include establishing sleep routines and minimizing distractions such as televisions in the bedroom(104). Whether or not counseling about healthy sleep should be a mainstay of prevention and treatment strategies for obesity remains unclear. While many advocate that it should be(37), others feel we should not incorporate this aspect of counseling until studies are more definitive(84). The best way to settle this decision would be to conduct a randomized, longitudinal obesity prevention study in a large group of varied ethnicities and races starting in infancy, of which only one arm of the study would include sleep counseling. For quality assurance, the children would need to have their sleep patterns periodically assessed objectively in order to verify that sleep counseling actually resulted in prolonged sleep and to what degree. Thus far, longitudinal observational studies examining sleep and obesity have provided promising

results in that short sleep is associated with future obesity(76, 78, 81, 100), and so a longitudinal interventional study is a natural next step.

Given slight variations in our results when compared to the literature, our study highlights two other important needs: targeting studies toward low-income minority populations and reporting actigraphy results thoroughly so that studies can be adequately compared. First, low-income minority children are an extremely important demographic to consider in the obesity epidemic, and existing evidence, including that which is presented here, show differences in sleep patterns that warrant further investigation. Second, in order to make the most of already collected research data, findings need to be presented in a manner that allows for future comparisons and meta-analyses. This includes thorough reporting of methodology, particularly regarding participants and actigraphy details such as algorithms and thresholds, as well as thorough reporting of results, including actual values of sleep durations and timings in addition to statistical analyses. Inclusion of these details will allow for researchers to assess how short sleep develops and whether the link between short sleep and obesity results from similar patterns within varying ages and populations of children. With this subsequent research, we can move toward optimizing both prevention and treatment strategies in this epidemic of obesity that will undoubtedly affect our society for generations to come.

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