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Summertime Sleep and BMI in Urban Minority Girls: Relations to Physical Activity and Executive Functions

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SUMMERTIME SLEEP AND BMI IN URBAN MINORITY GIRLS: RELATIONS TO PHYSICAL ACTIVITY AND EXECUTIVE FUNCTIONS

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ABSTRACT

Urban minority youth, particularly females, are at high risk for increased weight gain during the summertime months, and may also experience insufficient sleep at this time. Few studies have objectively measured summertime sleep in this population or related sleep to weight gain during this season. The current study draws on a sample of 66 urban minority girls aged 10-to-14 who participated in a community-based summer day camp program promoting physical activity (PA). The study objectively characterizes sleep in this sample, both in unstructured and structured contexts. Additionally, the study examines potential pathways underlying summertime relations between sleep and weight, including PA and executive functions (EFs). Data were collected at a community-based summer day camp program at two time points: prior to beginning programming (T1; unstructured context) and during the final week of programming (T2; structured context). At both time points, participants experienced shorter nighttime sleep than is recommended for their age, and African American girls recorded significantly less sleep than Latina girls only when not engaged in programming. Furthermore, findings suggest that wake times may play a particularly influential role in youths' abilities to obtain adequate sleep. Mediation models were not significant, however, research with a larger sample is needed to adequately address mechanisms underlying relations between sleep and BMI. Overall, summertime sleep is an understudied health behavior that may be important to consider among minority youth.

CHAPTER ONE

INTRODUCTION

In the past several decades, obesity rates have reached epidemic proportions with approximately 17% of U.S. youth obese and even higher rates among minority females (Ogden, Carroll, Kit, & Flegal, 2014). This is concerning given that child obesity is associated with long-term health consequences, including many health conditions that were once thought only applicable to adults (e.g., high blood pressure, type 2 diabetes; Daniels, 2006). Moreover, adolescents who are obese tend to carry their obesity into adulthood (Ogden, Flegal, Carroll, & Johnson, 2002; Serdula et al., 1993), resulting in an increased burden of cardiovascular diseases, diabetes, and cancers. Current trends in the growth of child obesity rates project 65 million more obese adults in the United States by the year 2030, consequently amassing an additional $48-66 billion/year increase in medical costs for the treatment of preventable obesity-related diseases (Wang, McPherson, Marsh, Gortmaker, & Brown, 2011).

Although numerous contributing factors to child obesity have been identified, accumulating evidence suggests that poor sleep may contribute to excessive weight gain in youth. Researchers from the National Sleep Foundation recently established an updated recommendation of 9-11 hours of sleep per night for school-age children (Hirshkowitz et al., 2015). Short sleep durations (i.e., less than nine hours per night for school-age children), later sleep onset times (i.e., first minute of sleep at night), and

earlier wake times are associated with increased risk for obesity in children and adolescents. Recent meta-analyses have revealed that short sleep durations specifically are strongly associated with both concurrent and future obesity risk among children (Cappuccio, Taggart, Kandala, & Currie, 2008; Chen, Beydoun, & Wang, 2008; Fatima, Doi, & Mamun, 2015; Patel & Hu, 2008), with effects lasting into adolescence (Magee, Caputi, & Iverson, 2013). In a longitudinal study of 2,281 children ages 3 to 12 years old, children who were reported by their parents to experience shorter durations of sleep, later sleep onset times, and earlier wake-times at baseline were more likely to be overweight five years later (Snell, Adam, & Duncan, 2007).

Several large-scale studies have provided evidence of dose-response relations between sleep and weight status, suggesting that shorter sleep may incrementally increase the likelihood of obesity. Gupta, Mueller, Chan, & Meininger (2002) examined a sample of 383 racially diverse adolescents ages 11-16, using actigrapy to measure sleep throughout the school year. Results suggested that for every hour of lost sleep at night, the odds of obesity increased by 80 percent. Similarly, two large cohort studies from Japan and Portugal collected parent-reports of child sleep during the school year (children ages 6-7 and 7-8, respectively) and found that participants' likelihood of obesity increased significantly for each hour of sleep lost on average per night (Padez, Mourao, Moreira, & Rosado, 2005; Sekine, Yamagami, & Handa, 2002).

At the same time, research reveals that obese youth are at increased risk for short sleep, later sleep onset times, and earlier wake times by nature of their weight status. Spruyt, Molfese, & Gozal (2011) used polysomnography (i.e., lab-based

neurophysiological sleep testing) to examine sleep in a community sample of 351 Caucasian children ages 6-10 years old and found a 1.5 to 2-fold increase in the likelihood of short sleep durations when a child was obese. Additionally, overweight children were more likely to go to bed later and wake up earlier during the school week, and were less likely to experience catch up sleep on the weekends. Beebe and colleagues (2007) also found differences in sleep onset time per weight status in a sample of 60 African American children ages 10-16 recruited from a weight management clinic. Using a multi-method strategy of polysomnography, actigraphy, and self-reported sleep measures, results indicated that overweight children on average fell asleep later and had more disrupted sleep than healthy-weight, age-matched controls, resulting in shorter sleep durations. Differences in sleep based on weight status may in part be due to problems such as sleep disordered breathing (i.e., obstructive sleep apnea), which is strongly associated with obesity (Marcus et al., 2012).

Although the association between insufficient sleep and obesity in youth has been well explored over the past decade, relations may be particularly important to consider among low-income, urban minority girls who experience higher rates of obesity. Specifically, 24% of 6-11 year old African American children and 26% of Hispanic children are obese, as compared to 13% of non-Hispanic white children and 9% of non-Hispanic Asian children (Ogden et al., 2014). Additionally, while most associations between sleep and obesity are fairly consistent, adolescent girls have at times failed to evidence significant relations between sleep and BMI in comparison to their male or

younger female counterparts, and therefore remain a group of particular interest (Thind et al., 2014).

In addition to being at elevated risk for obesity, minority youth also report later sleep onset times and shorter sleep durations than non-minority youth (Crosby, LeBourgeois, & Harsh, 2005; Spilsbury et al., 2004). Urban minority youth may experience higher prevalence of sleep problems (e.g., bedtime resistance, sleep anxiety, etc.) contributing to shorter nighttime sleep durations when compared to non-minority counterparts (Sheares et al., 2013). While studies have begun to elucidate the difference in sleep patterns between minority and non-minority youth, measurement in these studies often relies on child or parent-reported measures of sleep, which may result in over-estimates of sleep when compared to objective measures (Dayyat, Spruyt, Molfese, & Gozal, 2011). One recent study examined minority youth sleep using objective measures and found that minority adolescent girls slept 0.3 hours less per night on average than their male and younger female counterparts (Wong et al., 2013). Thus, while the aforementioned studies suggest shorter periods of sleep in minority youth, particularly girls, additional research using objective measures is needed to confirm findings and accurately characterize both sleep and relations between sleep and BMI in this population.

Summertime as a Unique Developmental Context

The summertime months are a unique context for youth. While summertime only constitutes about 25% of the year, youths' waking hours during the summer rival the number of hours spent in school throughout the entire academic year (Mahoney, 2011).

The summertime period is characterized by a lack of traditional school attendance and seasonal differences in weather and activity. The time off of school during the summer vacation period often means that youth's daily experiences differ substantially than when school is in session. Differences may be most pronounced in low-income, urban youth, who may lack access to structured summertime programming due to cost or availability. When youth are not enrolled in programming and spend the majority of their time in unstructured contexts during the summer, they may engage in more obesity-related health behaviors such as increased sedentary time (e.g., television viewing; Larson & Kleiber, 1993; Gershenson, 2013), decreased physical activity (McCue et al., 2013), and decreased sleep (Nixon et al., 2008). Summer care arrangements, in turn, have been shown to affect weight gain, such that children who spend their summer under parental care or care by other adults in an unstructured settings are at increased risk for obesity the following year (Mahoney, 2011).

Sleep and BMI relations may be particularly important to examine during summertime, when youth experience marked decreases in sleep (Nixon et al., 2008) and increased risk for obesity (Baranowski et al., 2014). During the summer months when youth are not in school, youth report later sleep onset times and significant decreases in sleep duration (Nixon et al., 2008), perhaps related to increased hours of daylight (Benefice, Garnier, & Ndiaye, 2004; Carson & Spence, 2010). Furthermore, youth gain weight at a faster rate during the summer compared to the school year, and this weight gain is particularly pronounced among urban minority girls (von Hippel, Powell, Downey, & Rowland, 2007). Indeed, summertime weight gain may be a significant

barrier to obesity prevention among high-risk youth. A recent five-year longitudinal study found that although overweight youth tended to decrease in BMI during the school year, these decreases were negated by subsequent summertime weight gain (Moreno, Johnston, & Woehler, 2013). Taken together, relations between sleep and BMI are critical to understand among this high-risk group (i.e., urban minority girls) during this high-risk season (i.e., summer) using objective assessments of sleep.

An important component of understanding sleep and BMI relations among urban minority girls during summertime is identifying pathways of influence. In a recent meta-analysis of summer changes in child weight gain, Baranowski and colleagues (2014) noted that studies have observed seasonal changes in both sleep and physical activity (PA) and called for more research linking sleep, PA, and BMI patterns in children across the school year and summertime. Little is known about the mechanisms through which sleep influences BMI, though several possible pathways have been proposed in the literature (Cappuccio et al., 2008; Sivak, 2006; Patel & Hu, 2008). Of many potential pathways (e.g., hormonal regulation, opportunity to eat, and thermoregulation), this study will explore the possibility of PA (Figure 1) and executive functions (EFs; Figure 3) as key mechanisms in the relation between sleep and BMI.

Sleep, Physical Activity, and BMI

While physical activity (PA) has been highlighted as a salient factor in the childhood obesity epidemic (Ekelund, 2013; Epstein & Goldfield, 1999; Kahn et al., 2002; Kelley, Kelley, & Pate, 2014), fewer studies have examined sleep and PA in relation to child BMI. Global public health authorities agree that children ages 5-18

years should engage in at least 60 minutes of moderate-to-vigorous physical activity (MVPA) on at least 5 days per week (World Health Organization, 2010; U.S. Department of Human Health and Services). Children who meet both PA and sleep recommendations are less likely to be obese than children who meet only one or neither of these recommendations (Laurson, Lee, Gentile, Walsh, & Eisenmann, 2014), suggesting that both sleep and PA have significant influence on obesity. There are inverse associations between both sleep duration and obesity (Chen et al., 2008) and PA and obesity in children (Chaput et al., 2014; Ness et al., 2007); however, pathways of influence between sleep, PA, and BMI are unclear.

Although some studies suggest that sleep may influence levels of PA (Baranowski et al., 2014; Patel & Hu, 2008), other literature suggests that PA may influence sleep (Delisle, Werch, Wong, Bian, & Weiler, 2010). Short sleep durations are associated with feelings of fatigue and sleepiness (Dinges, Pack, & Williams, 1997), and researchers hypothesize that experiencing fatigue and sleepiness may lead to reduced levels of PA (Patel & Hu, 2008). One study demonstrated that elevated levels of disturbed sleep were related to lower PA levels in adolescents (Gupta et al., 2002), while another study showed that children who slept more were less sedentary (Gomes et al., 2014). On the other hand, youth engaging in more vigorous PA have been shown to report longer sleep durations (Delisle et al., 2010), suggesting that links between sleep and PA may be reciprocal. Although research has begun to confirm hypothesized associations between sleep and PA, some recent studies have failed to find relations between objective measures of sleep and PA in children (Soric et al., 2014). When relations have been

found and explored, few studies have considered the ways in which these relations may help explain links between sleep, PA, and BMI.

Current literature evidences mixed findings regarding the effect of summertime on youth's levels of PA (Carson & Spence, 2010). Some research suggests that youth engage in significantly less MVPA over the summer when compared to the school year (Baranowski, Thompson, DuRant, Baranowski, & Puhl, 1993; McCue, Marlatt, Sirard, & Dengel, 2013), but these studies are limited due to small samples taken from contexts with extreme climates, environments, and differing structures of the school year. As such, findings require further investigation among different regions and samples. Low-income urban minority youth may be at particularly high risk for summertime declines in PA as they may lack access to safe play environments when not in school (Ergler, Kearns, & Witten, 2013). Additionally, as youth mature in adolescence, minority girls show substantial decreases in PA and increases in sedentary time in comparison to males and non-minority females (Belcher et al., 2010; Kimm et al., 2002; Nelson, Neumark-Stzainer, Hannan, Sirary, & Story, 2006). Studies have illustrated high prevalence rates of obesity, insufficient sleep, and reduced PA levels during adolescence in minority girls (Kimm et al., 2002; Wong et al., 2013; Treuth et al., 2009), and these factors may be related. However, the role of PA as a mediator between summertime sleep and BMI (Figure 1) has yet to be empirically tested, particularly among minority girls.

Although some literature has compared school-year levels of sleep, BMI, and PA to summertime levels of these variables (Baranowski et al., 1993; von Hippel et al., 2007; Nixon et al., 2008), little-to-no research has examined sleep, PA, and BMI throughout the

summer. Within the summertime context, research shows that summer care arrangements can have a strong impact on health outcomes among youth (Mahoney, 2011; Parente, Sheppard, & Mahoney, 2012). Whereas adolescents not involved in summer programming experience decreases in PA and increases in BMI (McCue et al., 2013), youth who participate in structured summer activities record higher levels of PA than their peers in unstructured contexts (Tovar et al., 2010). Organized activity involvement across the entire summertime season has been shown to prevent widely-observed weight gain during summer (Parente et al., 2012). Unfortunately, low-income, minority youth often do not have access to affordable structured programming when they are not in school, due to a variety of financial and logistic barriers (Harvard Family Research Project, 2004; Goerge & Chaskin, 2004; Sanderson & Richards, 2010). As such, few studies have been able to examine the impact of structured summertime programming on minority youth.

Furtheremore, whereas some studies have captured the ability of organized activities to change health behaviors such as PA (Bohnert, Ward, Burdette, Silton, & Dugas, 2014; Tovar et al., 2010), few-to-no studies have examined how program-related changes in health behaviors may impact sleep. PA may influence sleep by more readily bringing about feelings of fatigue and sleepiness at night (Patel & Hu, 2008). Indeed, research demonstrates that more vigorous PA is associated with longer sleep durations in adolescents (Delisle, Wech, Wong, Bain, & Weiler, 2010). In the context of summer program-related changes in PA, it is possible that participants will experience earlier sleep onset times and longer sleep durations at night, which may contribute to BMI at this

time. This study will take an additional step toward understanding summertime changes in child BMI by examining changes in sleep and PA during the summer in the context of a structured summer camp program promoting PA.

Sleep, Executive Functions, and BMI

Another pathway through which sleep may influence obesity is through executive functions (EFs). Broadly defined, EFs are cognitive processes that underlie goal-directed behavior (Best & Miller, 2010). EFs rely on prefrontal and parietal regions of the brain to facilitate planning and organization, as well as problem solving skills, and include domains of shifting and inhibition (Miyake et al. 2000; Wong, Mayberry, Bishop, Maley, & Hallmayer, 2006). Shifting relates to the ability to adapt behavior in response to situation or context, while inhibition refers to the ability to control an automatic response or impulse (Davidson, Amso, Cruess, & Diamond, 2006).

To date, the most convincing research linking sleep and obesity evidences strong links between short sleep and increased caloric intake (Beebe et al., 2013; Garaulet et al., 2011; Nishiura, Noguchi, & Hashimoto, 2010). However, studies acknowledge that dietary patterns can only partially explain relations between sleep and obesity (Nishiura et al., 2010). Increased dietary intake after short sleep is often linked to food-related disinhibition (Burt, Dube, Thibault, & Gruber, 2014), which may reflect underlying deficits in EF after poor sleep.

EF impairments, particularly difficulties with inhibitory control and shifting cognitive sets, may serve as underlying mechanisms that help to explain associations between obesity and sleep. Poor sleep quality may be associated with EF deficits in the

form of lower inhibition, slower processing speed, and more cognitive errors overall (Esposito et al., 2013; Goel, Rao, Durmer, & Dinges, 2009). Experimental studies support that short sleep is associated with poorer EFs including inhibition and shifting (Durmer & Dinges, 2005; Sadeh, Gruber, & Raviv, 2003), and inhibition and shifting deficits have been consistently associated with obesity in children, adolescents, and adults (Smith, Hay, Campbell, & Trollor, 2011). Specifically, lack of sleep may lead to an inability to regulate reward and punishment, particularly related to food choices and an inability to shift or inhibit behavior in response to a cue (i.e., feeling full) (Beebe et al., 2007; Durmer & Dinges, 2005; Fagundo et al., 2012). A recent study revealed that short sleep duration and later sleep onset time were associated with greater eating in response to external cues for food, emotional eating, and binge eating, respectively, which may be manifestations of EF deficits (Burt et al., 2014). Research exploring relations between EFs and obesity has similarly found that poor shifting and inhibition skills are often related to unhealthy eating behaviors (Fagundo et al., 2012; Maayan, Hoogendoorn, Sweat, & Convit, 2011). Finally, while poor sleep is often linked to lower cognitive functioning (Durmer, & Dinges, 2005), recent studies have shown that this relation is particularly strong in obese children (Tan, Healey, Schaughency, Dawes, & Galland, 2014). Few studies, however, have explicitly examined EFs as a mechanism underlying associations between sleep and BMI. Though one study found that sleep extension improves EF capabilities in obese adults (Lucassen et al., 2014), this has been henceforth unexplored in children, and these findings have not been experimentally tied back to weight status.

Although studies evidence links between sleep, EFs, and weight status, relations have yet to be explored within a summertime context. This study will take an important step by testing a cohesive model of summertime relations between sleep, EFs, and BMI. Additionally, the study will explore change in EFs throughout the summer in the context of a camp program promoting PA, and examine whether change in EFs are associated with changes in sleep and BMI at this time in sample of urban minority girls.

Specific Aims and Hypotheses

The current study draws on a sample of 10-to-14 year old low-income urban minority girls participating in a community-based summer day camp program promoting physical activity (PA). This multi-method study seeks to gain a better understanding of relations between general levels of summertime sleep, PA, executive functions (EFs), and BMI, as well as relations between these variables over time in the context of program-related changes in PA. Specifically, aims of this study include:

1. *Describe summertime sleep (i.e., duration, sleep onset time, and wake time) among minority girls using objective measures.* It is hypothesized that girls in our sample will obtain shorter sleep duration than is recommended for their age (i.e., less than nine hours per night).
2. *To examine relations between sleep and zBMI.* It is hypothesized that shorter sleep durations and later sleep onset times will be associated with higher levels of zBMI. Associations will be tested separately for each sleep measure (i.e., duration, sleep onset time, and wake time).

3. *To examine relations between changes in sleep, BMI, MVPA, and EFs over the summer in the context of a summer camp promoting PA.* See Table 1 for hypothesized relations between change variables. It is hypothesized that there will be negative associations between changes in sleep and changes in BMI, such that decreases in sleep over the summer will be associated with greater increases in BMI, and increases in sleep over the summer will be associated with decreases (or less of an increase) in BMI. Additionally, it is hypothesized that there will be positive associations between changes in sleep and changes in MVPA, such that increases in MVPA will be associated with increases in sleep. It is hypothesized that there will be negative associations between change in MVPA and change in BMI, such that increases in MVPA will be associated with decreases (or less of an increase) in BMI. It is hypothesized that there will be positive associations between changes in sleep and changes in EF, such that increases in sleep will be associated with increases in EF, and decreases in sleep will be associated with decreases in EF over the summer. Finally, it is hypothesized that there will be negative associations between changes in EF and changes in BMI, such that increases in EF will be associated with decreases (or less of an increase) in BMI, and decreases in EF will be associated with greater increases in BMI. Associations will be tested separately for all measures of sleep (i.e., duration, sleep onset time, and wake time), MVPA, and EF (i.e., D-KEFS inhibition time, D-KEFS shifting time, D-

KEFS total errors in inhibition, D-KEFS total errors in shifting, BRIEF-SR inhibition, and BRIEF-SR shifting).

4. *To examine the fit of a mediation model in which MVPA mediates relations between sleep and zBMI.* It is hypothesized that PA will mediate relations between sleep and zBMI (Figure 1). Three versions of the model will be tested account for all possible relations between data at T1 and T2 (see Table 2). The first version of the model will test T1 MVPA as a mediator of relations between T1 sleep and T1 zBMI. The second version of the model will test T1 MVPA as a mediator of relations between T1 sleep and T2 zBMI. The third version of the model will test T2 MVPA as a mediator of relations between T1 sleep and T2 zBMI. All models will be tested separately for each sleep measure (i.e., duration, sleep onset time, and wake time).

Figure 1. Proposed Model of Physical Activity as a Mediator between Sleep and Body Mass Index Using Summertime Measures

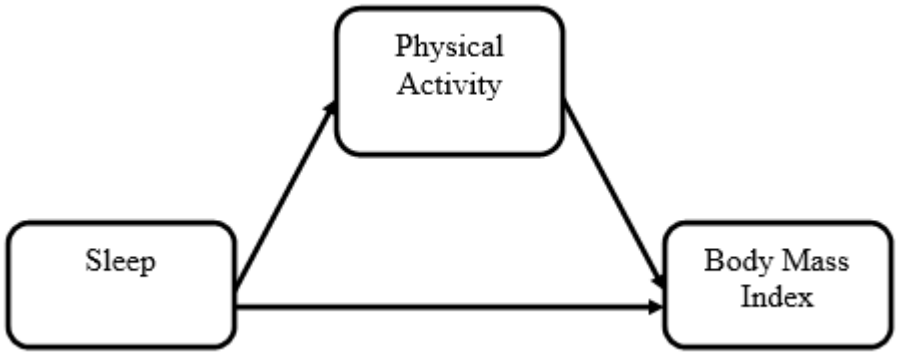


Table 1. Hypothesized Relations between Change Variables

	Δ Sleep	Δ BMI	Δ PA	Δ EFs
Δ Sleep	X	-	+	+
Δ BMI	-	X	-	-
Δ PA	+	-	X	X
Δ EFs	+	-	X	X

Notes. The symbol (+) represents a hypothesized positive correlation, whereas the symbol (-) represents a hypothesized negative correlation. An (X) denotes that the correlation will not be examined in the current study

5. *To examine the fit of a competing mediation model in which sleep mediates relations between MVPA and zBMI at T1*. The competing hypothesis is that sleep will mediate relations between MVPA and zBMI (Figure 2). This model will be tested in Form 3 (see Table 2), examining T2 sleep as a mediator of relations between T1 MVPA and T2 zBMI. The model will be tested separately for each sleep measure (i.e., duration, sleep onset time, and wake time).

Figure 2. Proposed Competing Model of Sleep as a Mediator between Physical Activity and Body Mass Index Using Summertime Measures

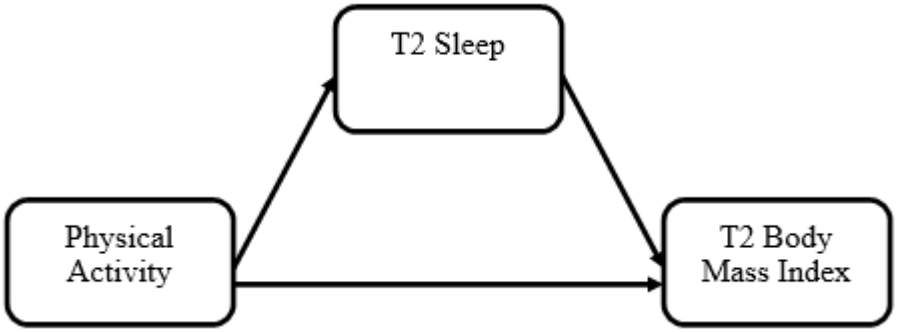
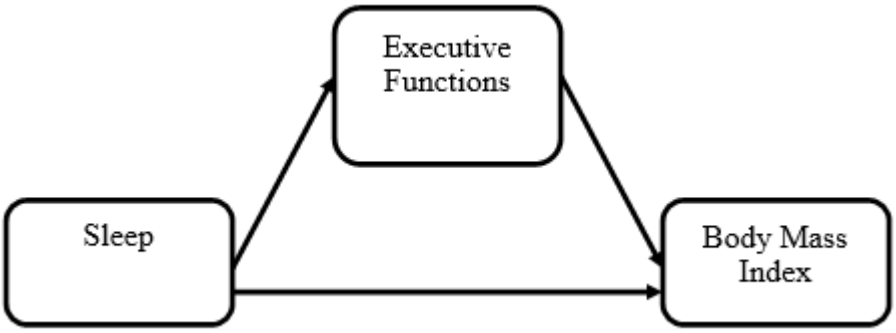


Table 2. Pattern of Variables Tested in Mediation Models to Account for All Possible Relations between Data at T1 and T2

	Independent Variable	Mediator	Dependent Variable
Form 1	T1	T1	T1
Form 2	T1	T1	T2
Form 3	T1	T2	T2

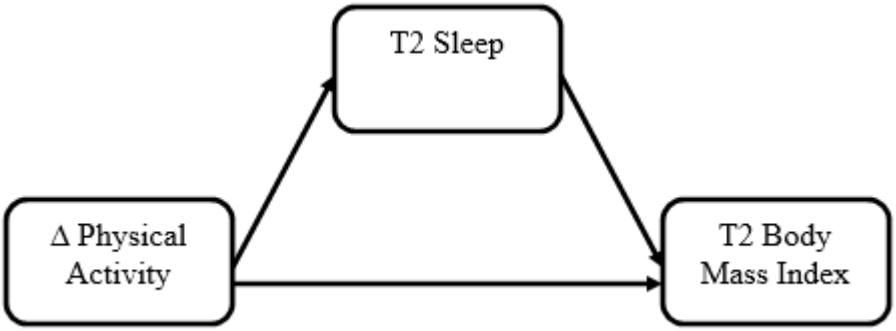
6. *To examine the fit of a mediation model in which EFs mediate relations between sleep and zBMI.* It is hypothesized that EFs will mediate relations between sleep and zBMI (Figure 3). Three versions of the model will be tested to account for all possible relations between data at T1 and T2 (see Table 2). The first version of the model will test T1 EFs as a mediator of relations between T1 sleep and T1 zBMI. The second version of the model will test T1 EFs as a mediator of relations between T1 sleep and T2 zBMI. The third version of the model model will test T2 EFs as a mediator of relations between T1 sleep and T2 BMI. Each version of the model will be run separately for each sleep measure (i.e., duration, sleep onset time, and wake time) and EF measure (i.e., D-KEFS inhibition time, D-KEFS shifting time, D-KEFS total errors in inhibition, D-KEFS total errors in shifting, BRIEF-SR inhibition, and BRIEF-SR shifting).

Figure 3. Proposed Model of Executive Functions as a Mediator between Sleep and Body Mass Index Using Summertime Measures



7. *To examine the fit of a mediation model in which sleep at T2 mediates relations between change in MVPA and zBMI at T2.* It is hypothesized that sleep at T2 will mediate relations between changes in MVPA and T2 zBMI in the context of a summer camp program promoting physical activity physical activity (Figure 4). The model will be run separately for each measure of sleep (i.e., duration, sleep onset time, and wake time).

Figure 4. Proposed Model of Sleep at Time 2 as a Mediator between Program-Related Change in Physical Activity (T2-T1) and Body Mass Index at Time 2



CHAPTER TWO

METHOD

Participants

Participants (*n*=66) in this study were 10-14 year old urban girls (*M* = 11.80 years, *SD* = 1.02) enrolled in a summer day-camp program. Participants' parents identified them as African American (n = 38, 58%), Latina (n = 22, 33%), Caucasian (n = 1, 2%), Asian-American (n = 1, 2%), and Other/Missing (n = 4, 5%), and 100% were female. Analyses using race/ethnicity compared only Latina and African American participants, due to small samples of other races.

Participants included in this study are a subset of a larger study that was designed to evaluate the effectiveness of the 4-week community-based summer program. In order to be included in the current study, participants had to attend an initial orientation session where anthropometrics were measured, individually administered EF measures were completed, and accelerometers were given with instructions. Girls must have then returned the accelerometer with at least four days of valid PA data and three nights of valid sleep data. Although 97 unique participants enrolled in the study across all three waves, only 66 met the inclusion criteria of complete BMI, PA, EF, and sleep data for analyses at T1. A further subset of participants (*n* = 44) met inclusion for complete data at T1 and T2, and these participants were used for analyses involving both time points.

Subsets of participants (n = 66 and n = 44) did not differ significantly from each other or from the full sample (N = 97) in terms of basic demographic characteristics including age, race/ethnicity, or outcome variables including T1 sleep, and both time points of PA, EF, and BMI variables.

Intervention

The community-based summer day camp program targeted girls who reside in urban Chicago neighborhoods with few resources and high numbers of ethnic and low-income youth. The program lasted four weeks and included six hours of activities each day, from 9 AM to 3 PM. The daily schedule included three 50-minute morning sessions (i.e., two sports-based, PA lessons and one health/leadership activity), a 40-minute lunch break, 60 minutes of pool time, 45 minutes of team physical activity, as well as an additional 10-minute snack break. Each session provided instruction and PA through a variety of traditional and non-traditional sports and fitness activities. Participants were provided transportation to and from the camp program each day.

Procedure

Data was first collected during the summer of 2012, with a second wave collected during the summer of 2013, and a final wave collected during the summer of 2014. In all three years, pre-programming data (Time 1; T1) was collected several weeks prior to the start of the camp program at an orientation open house, and follow-up data (Time 2; T2) was collected during the final week of the day camp program. Prior to all waves of data collection, participants who were already enrolled in the summer program were sent an informational packet with consent forms in either English or Spanish and a cover letter

explaining the study. The cover letter invited participants and their families to attend a program orientation day prior to the start of the summer camp program, where program participants will come to learn more about the community organization and their programs, as well as complete T1 measures if they chose to participate. Participants had the option of returning their consent forms by mail with their program registration form to the community organization, or could bring it directly to the open house orientation day. All program participants were called and invited to the open house orientation day as well.

A multi-method assessment strategy using questionnaires, anthropometric measures, individually administered neuropsychological tests, and accelerometers was used at both T1 and T2. Participants completed questionnaires in small groups with trained research assistants, completed neuropsychological tests individually with a trained research assistant, and had anthropometric measurements (i.e., height and weight) taken in a semi-private location. In addition, participants wore accelerometers (ActiGraph GT3X accelerometers; Pensacola, FL) attached at the waist for one week at T1 and one week at T2.

Measures

Anthropometrics. Weight of the participant was measured without shoes on and dressed in light clothing to the nearest 0.1 kg using a digital scale. Height was measured using a SECA stadiometer without shoes and head held in the Frankfort plane to the nearest 0.1 cm. This data was used to calculate BMI according to the following formula: BMI = kg/m^2. BMI *z*-scores (zBMI) were calculated based on age and gender-specific

CDC growth charts (Kuczmarski et al., 2002). zBMI was used for all analyses observing single time point data in order to best compare BMIs of girls across the study's age range. For analyses using data at multiple time points (i.e., analyses utilizing change scores) BMI was used in order to obtain the most sensitive measure of change over time (Himes, 2009).

Accelerometry. Participants were given an accelerometer (Actigraph GT3X; Pensacola, FL), worn at the waist, positioned just behind the right hip. The Actigraph GT3X has the capability to measure both daytime activity levels and nighttime sleep-wake patterns with no need to move or reattach the device (Kinder et al., 2012); this is advantageous for collecting both PA and sleep data from the community-based youth sample. Although accelerometry worn at the waist may be less sensitive than measures at the wrist (Hjorth et al., 2012), sleep measurement research supports the use of waist-worn sleep measures in children (Barreira et al., 2015; Kinder et al., 2012; Tudor-Locke, Barreira, Schuna, Mire, & Katzmarzyk, 2013). Participants wore accelerometers both during programming and outside of programming hours for a total of one week. Participants were instructed to only remove accelerometers when bathing and during the 60 minutes of pool time per day during programming. Research assistants were onsite during programming to ensure that accelerometers were correctly positioned on the right hip after pool time.

Sleep. The waist-worn accelerometer was used to record and estimate sleep duration between sleep onset time and morning wake time. For sleep analyses, 60-second epochs of motion were used. Sleep variables were derived using Tudor-Locke,

Barreira, and colleagues' recently developed algorithms, which are based on the standard Sadeh algorithm (Sadeh, Sharkey, & Carskadon, 1994) and were developed to better differentiate periods of sleep from periods of sedentary and non-wear time (Barreira et al., 2015; Tudor-Locke et al., 2013). A validation study demonstrated that the algorithms produced more specific estimates of sleep than sleep logs alone, but estimates were not significantly different from methodologies utilizing sleep logs in conjunction with accelerometers (Barreira et al., 2015). For the current study, participants were required to have at least three nights of captured sleep to be included in analyses. Using data from all available nights, three sleep variables were derived: (1) sleep onset time, or the first minute of sleep at night; (2) wake time, or the first minute of 10 consecutive minutes scored as non-sleep in the morning, and (3) nightly sleep duration, or the number of minutes scored as sleep between sleep onset time and wake time.

Physical activity. Accelerometer data were downloaded and passed through a customized Visual Basic EXCEL macro (Trost et al., 2013) designed to infer non-wear time and to determine the amount of time spent in sedentary, light, moderate and vigorous PA. A valid day of PA monitoring was defined as having nine or more hours of wear time. Moderate and vigorous activity levels were defined using published cut-points for children and adolescents (Rice & Trost, 2013). Data are reported as minutes of moderate-to-vigorous physical activity (MVPA).

Executive functions. Executive function measures of inhibition and shifting were collected from individually-administered neuropsychological tests and self-report questionnaires.

Delis-Kaplan Executive Function System (D-KEFS). Neuropsychological tests included the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) Color-Word Interference Test. The Color-Word inference test provides objective measures of inhibition and shifting by measuring an automatic response (word reading) in favor of a novel response (color naming), which was first developed by Stroop (1935). Additionally, the test includes an interference/switching condition which assesses cognitive flexibility (i.e., shifting) by asking participants to switch back and forth between reading words and naming dissonant ink colors. Raw scores are provided for both completion time and errors in these tests, with higher scores denoting worse performance. The D-KEFS has strong psychometric properties with internal consistency coefficients ranging from 0.62 to 0.77 for children 10-to-14 years of age, and test-retest reliability from 0.77 to 0.90 across the four conditions included in the test (Delis et al., 2001). From this test, four variables were obtained: total time on the inhibition task, total time on the shifting task, total errors in inhibition, and total errors in shifting.

Behavior Rating Inventory of Executive Function, Self-Report (BRIEF). Self-reported problems with EFs were assessed through the Behavior Rating Inventory of Executive Function, Self-Report (BRIEF-SR; Guy, Isquith, & Gioia, 2004). Participants completed the inhibit and shift items of the BRIEF-SR (i.e., “I have trouble getting used to new situations” and “I interrupt others”), with responses choices ranging from never, sometimes, or often. The BRIEF-SR is well validated and subscales have demonstrated a good internal consistency for all domains ($\alpha = .96$) and for the scales ($\alpha = .72$ - $.96$) (Guy,

Isquith, & Gioia, 2004). From these surveys, two variables were obtained: BRIEF-SR inhibition and BRIEF-SR shifting.

Change scores. For all analyses investigating change over time, change scores were created by subtracting variables at T1 from T2. Change scores were calculated based on individual participant means for all sleep, PA, and EF variables (i.e., sleep duration, sleep onset time, MVPA, D-KEFS inhibition time, D-KEFS shifting time, D-KEFS total errors in inhibition, D-KEFS total errors in shifting, BRIEF-SR inhibition, BRIEF-SR shifting), and for individual participant BMI.

Residuals. For all mediation analyses examining T2 variables, standardized residuals were created to quantify the difference in the T2 variable from what would be expected given the value of the variable at T1. The residual was created by performing a regression analysis in which the T1 level of the variable was used as the predictor of the T2 level of the variable, and each participant was given an individual residual score identifying deviation at T2 from the expected change. Standardized residuals were used in all mediation analyses utilizing T2 data in order to account for T1 levels of the variable.

Analytic Plan

Descriptive analyses, including means and standard deviations, were run for all study variables, and correlations were utilized to examine relations at T1, T2, and for change variables (i.e., T2-T1). Analyses of variance tests (ANOVAs) and t-tests were run to determine significant differences across time points, as well as differences in sleep variables based on weight status and ethnicity (i.e., African American and Latina),

respectively. To test the fit of hypothesized meditational models of PA (Figures 1-4), bootstrapping methodology was used (Hayes, 2009; Shrout & Bolger, 2002). Each of the hypothesized mediational models were tested in three versions to account for all possible relations between data at T1 and T2 (see Table 2).

CHAPTER THREE

RESULTS

Data Preparation

Data was first examined for the influence of outliers and skewness (Tabachnick & Fiddel, 1996). For D-KEFS variables, outliers were removed when inhibition or inhibition/switching times were greater than three standard deviations from the mean, including participants who timed out (i.e., obtained 180 seconds). All EF variables that had a skewness statistic greater than 1.2 were transformed using a square root transformation. The D-KEFS total errors in inhibition and D-KEFS total errors in switching were transformed as a result of these preliminary analyses.

Participants were required to have at least three nights of valid sleep data and one day of valid PA data (i.e., at least nine hours of wear time). Failure to meet wear time criteria for sleep or PA measures resulted in exclusion of the participant from data analyses. From the full sample of 97 participants, 44 participants had full accelerometer data and were included in all analyses, 22 participants had complete T1 data and incomplete T2 data, and were therefore only included in T1 analyses, and 31 participants had incomplete accelerometer data at both T1 and T2 and were excluded from all analyses. For missing data on the BRIEF-SR, if a participant responded to at least 80% of items on the scale, a mean imputation procedure was used to fill in the missing item(s) based on the participant's response to other items on the scale. For D-KEFS measures,

one participant was missing an inhibition/switch score at T1 only and therefore was not included in analyses involving inhibition/switching time at T1.

Descriptive Statistics

Descriptive statistics, including means, standard deviations, and correlations for study variables are listed in Tables 3 and 4 for T1 and T2, respectively. At T1, participants recorded a mean zBMI of 1.01, placing the sample in the 75th percentile as a whole, indicating normal weight status. In terms of individual participants' weight status at T1, 3% of participants were underweight (*n*=2), 41% were healthy weight (*n*=27), 24% were overweight (*n*=16), and 32% were obese (*n*=21).

Describing Summertime Sleep

At T1 (i.e., prior to the start of programming), participants went to bed at 12:15 AM (*SD* = 1:24), awoke at 9:06 AM (*SD* = 1:23), and slept for approximately 8 hours and 49 minutes per night (*SD* = 64 minutes). At T2 (i.e., during the final week of programming), participants went to bed at 11:11 PM (*SD* = 0:52) and got up at 7:42 AM on average (*SD* = 0:54), obtaining 8 hours and 29 minutes of sleep per night (*SD* = 60 minutes). Correlation analyses revealed that sleep onset time was negatively associated with sleep duration at both T1 and T2, such that participants who went to bed later obtained shorter sleep. Wake time was positively associated with sleep duration at both T1 and T2, such that participants who woke up later tended to obtain longer sleep.

Table 3. Descriptives and Correlations between Main Study Variables at T1

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	_												
2. T1 zBMI	-.16	_											
3. T1 BMI percentile	-.06	.90**	_										
4. T1 Sleep Duration	.09	-.09	-.03	_									
5. T1 Sleep Onset Time	.11	-.01	-.03	-.40*	_								
6. T1 Wake Time	.19	-.05	-.04	.39**	.70**	_							
7. T1 MVPA	.03	-.08	-.03	.24	-.27*	-.26*	_						
8. T1 BRIEF-SR Inhibition	-.09	.01	.04	-.14	.29*	.19	.01	_					
9. T1 BRIEF-SR Shift	-.28*	-.01	-.01	-.10	.02	-.05	-.04	.52**	_				
10. T1 D-KEFS Inhibition Time	-.44**	.35**	.30*	-.15	.15	-.04	-.10	-.02	.14	_			
11. T1 D-KEFS Inhibition/Shift Time	-.34**	.30*	.32*	-.01	-.16	-.16	.07	-.09	.01	.66**	_		
12. T1 D-KEFS Inhibition Total Errors	-.01	-.08	-.06	-.07	-.01	-.05	-.04	-.18	-.04	.30*	.32**	_	
13. T1 D-KEFS Inhibition/Shift Total Errors	.13	.09	.08	-.17	.08	-.05	.02	-.02	.16	.28*	.47**	.29*	_
M	11.80	1.01	75.46	529.15	0:15	9:06	13.62	6.78	7.04	70.96	75.86	1.81	2.14
SD	1.02	.99	26.90	64.29	1:24	1:23	18.23	4.66	3.49	19.99	20.57	.80	.89

* $p < 0.05$ level; ** $p < 0.01$ level.

Table 4. Descriptives and Correlations between Main Study Variables at T2

	1	2	3	4	5	6	7	8	9	10	11	12
1. T2 zBMI	_											
2. T2 BMI percentile	.96*	_										
3. T2 Sleep Duration	-.20	-.22	_									
4. T2 Sleep onset time	.07	.06	-.52**	_								
5. T2 Wake Time	-.10	-.15	.56**	.39**	_							
6. T2 MVPA	-.18	-.15	-.06	-.17	-.18	_						
7. T2 BRIEF-SR Inhibition	.08	.05	-.06	-.19	-.17	-.01	_					
8. T2 BRIEF-SR Shift	.03	.01	.10	-.05	.08	.05	.68**	_				
9. T2 D-KEFS Inhibition Time	.21	.13	.19	-.18	.06	.29	.13	.08	_			
10. T2 D-KEFS Inhibition/Shift Time	.39*	.38*	.07	-.23	-.08	.27	.31*	.14	.76**	_		
11. T2 D-KEFS Inhibition Total Errors	.19	.11	.19	-.05	.24	-.27	.13	.01	.27	.18	_	
12. T2 D-KEFS Inhibition/Shift Total Errors	.18	.16	.00	-.14	-.10	.03	.20	-.02	.29	.41**	.44*	_
M	.97	75.69	509.51	11:11 PM	7:42 AM	43.32	7.35	7.51	65.06	70.38	1.47	1.38
SD	1.03	26.58	60.45	0:52	0:54	34.38	5.56	3.93	19.26	24.51	.86	.90

* $p < 0.05$ level; ** $p < 0.01$ level.

ANOVAs and t-tests were utilized to examine differences in sleep based on weight status and race/ethnicity (i.e., African American and Latina). There were no significant differences in sleep variables based on weight status at either time point. However, there were significant differences based on ethnicity (Table 5). Latina participants recorded significantly longer sleep durations ($t(58)=-2.84$, $p = .006$) and earlier sleep onset times ($t(58) = 2.48$, $p = .016$) compared to African American girls at T1. Specifically, Latina girls experienced 45 additional minutes of sleep and went to bed 51 minutes earlier than African American participants on average. At T2, Latina girls no longer recorded significantly longer sleep durations, earlier sleep onset times, or later wake times than African American participants.

Table 5. Sleep Variables Broken Down by Participant Ethnicity (African American and Latina Participants Only)

	African American	Latina
T1 *n*	38	22
T1 Sleep Duration*	518.14 (57.78)	563.93 (63.95)
T1 Sleep Onset Time*	12:29 AM (1:29)	11:38 PM (1:29)
T1 Wake Time	9:08 AM (1:21)	9:02 AM (1:24)
T2 *n*	26	14
T2 Sleep Duration	503.50 (65.55)	534.96 (42.08)
T2 Sleep Onset Time	11:14 PM (1:00)	10:59 PM (0:39)
T2 Wake Time	7:38 AM (0:57)	7:54 AM (0:55)

* denotes significant a difference between races

Relations between Sleep and BMI, PA, and EFs

Correlational analyses examined relations between sleep and BMI variables, MVPA, and EF variables at T1 and T2 (Tables 3 and 4). There were no significant relations between sleep and BMI variables at T1 or T2. Sleep variables were associated with MVPA at T1, but not T2. Specifically, sleep onset time was negatively associated

with MVPA at T1, indicating that participants recording earlier sleep onset times also recorded more MVPA. Wake time was also negatively associated with MVPA at T1, indicating that participants recording earlier wake times also recorded more MVPA. Interestingly, sleep duration was not associated with MVPA at T1. Relations between sleep variables and MVPA were not significant at T2. There was also a significant association between sleep variables and EF variables at T1, but not T2. Specifically sleep onset time was positively associated with BRIEF-SR inhibition score at T1, indicating that later sleep onset times were associated with more problems with inhibition. Relations between sleep variables and EFs were not significant at T2.

Though there were no significant relations between BMI variables and MVPA, significant associations emerged between BMI and EF variables at both T1 and T2. Specifically, zBMI and BMI percentile were positively associated with time taken to complete the D-KEFS inhibition task and D-KEFS switching tasks at both T1 and T2. These associations indicate that participants with higher zBMIs had more limited inhibition and shifting abilities than participants with lower zBMIs both before programming and during programming.

Relations between Changes in Sleep, BMI, PA, and EFs

Change scores were calculated for sleep and BMI variables, MVPA, and EF variables from T1 to T2. Figures 5-15 in Appendix A depict the distribution of change across participants. T-tests were run to analyze the significance of change from T1 to T2. Means, standard deviations, t-tests, and correlations between change variables are listed in Table 6. Participants recorded 24 fewer minutes of sleep on average at T2 when

compared to T1. Participants also went to bed 58 minutes earlier on average at T2 than T1. Changes in sleep onset time were negatively correlated with changes in sleep duration, such that participants who experienced greater decreases in sleep onset time (i.e., going to bed earlier at T2), experienced greater increases (or less of a decrease) in sleep duration between time points. Participants woke up 1 hour and 22 minutes earlier on average at T2. Changes in wake time were positively correlated with changes sleep duration, such that participants who experienced increases (or less of a decrease) in wake time experienced greater increases (or less of a decrease) in sleep duration. Changes in sleep variables were not significantly correlated with changes in BMI or changes in MVPA. Similarly, changes in BMI were not significantly correlated with changes in MVPA.

Changes in sleep duration were significantly correlated with changes in EFs, specifically the time taken to complete the D-KEFS inhibition task. Participants were 4.6 seconds faster on average on the D-KEFS inhibition task at T2 than T1. Changes in sleep duration were positively associated with changes in the amount of time taken to complete the D-KEFS inhibition task, indicating that participants experiencing greater increases (or less of a decrease) in sleep duration recorded greater decreases (i.e., quicker times) on the D-KEFS inhibition task at T2, representing better inhibitory skills.

Table 6. Descriptives and Correlations between Change Variables (T2-T1)

	1	2	3	4	5	6	7	8	9	10	11
1. ΔBMI	_										
2. ΔSleep Duration	-.08	_									
3. ΔSleep Onset Time	.23	-.52**	_								
4. ΔWake Time	.13	.53**	.45**	_							
5. ΔMVPA	-.25	.10	.09	.21	_						
6. ΔBRIEF-SR Inhibition	-.18	.09	.05	.15	.03	_					
7. ΔBRIEF-SR Shifting	-.13	.01	-.11	-.09	.10	.48**	_				
8. ΔD-KEFS Inhibition Time	-.18	.33*	-.33*	.01	.02	.04	.08	_			
9. ΔD-KEFS Inhibition/Shift Time	-.11	.24	-.16	.11	.13	-.09	-.10	.35*	_		
10. ΔD-KEFS Inhibition Total Errors	.22	.21	-.23	-.02	-.30*	.00	-.13	.10	.00	_	
11. ΔD-KEFS Inhibition/Shift Total Errors	-.23	-.08	.13	.05	.08	.25	-.03	.03	.05	.14	
M	.24	-24.23†	-0:58†	-1:22†	31.68†	.58	.28	-4.64†	-5.91†	-.41†	-.83†
SD	.41	78.66	1:16	1:17	37.56	3.84	2.98	10.75	18.75	1.02	.92

* $p < 0.05$ level; ** $p < 0.01$ level.

†denotes significant change from T1 to T2 ($p < 0.05$)

Figure 5. Distribution of Change in BMI

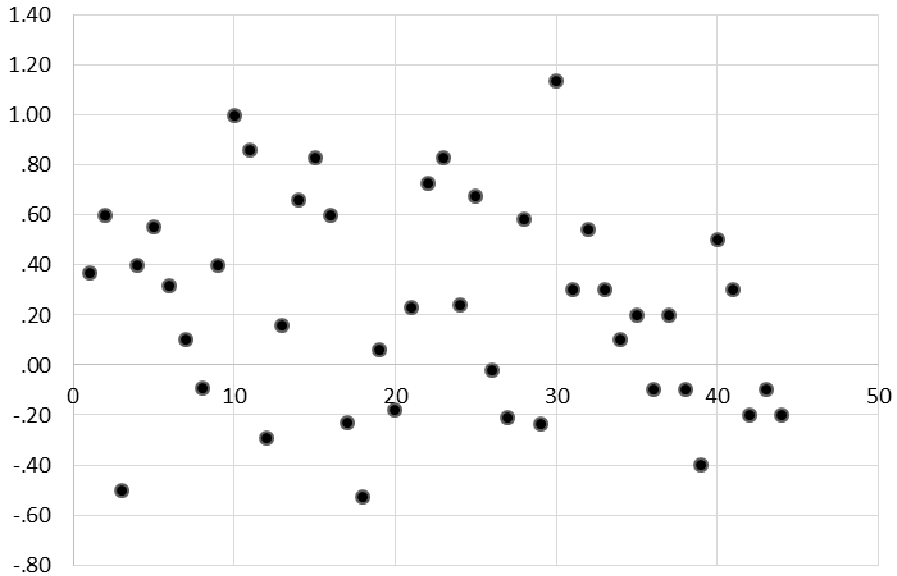


Figure 6. Distribution of Change in Sleep Duration

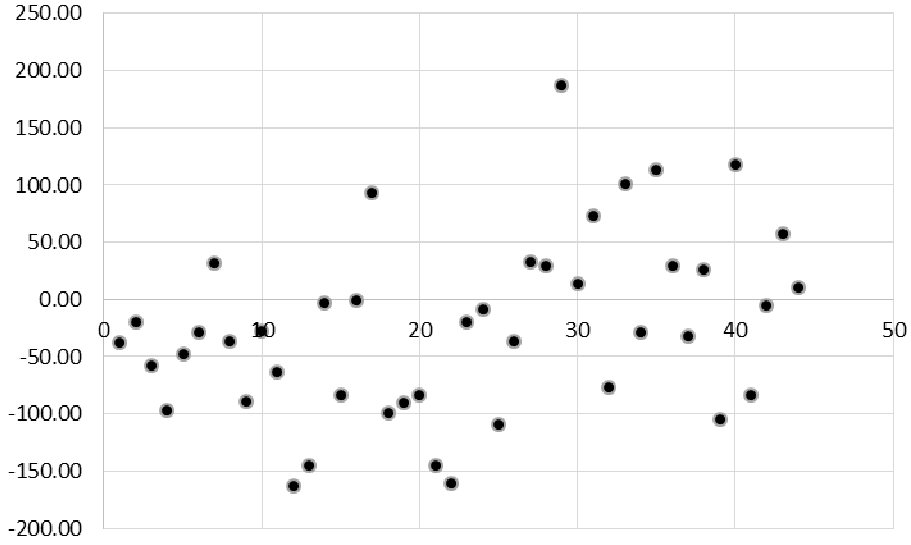


Figure 7. Distribution of Change in Sleep Onset Time

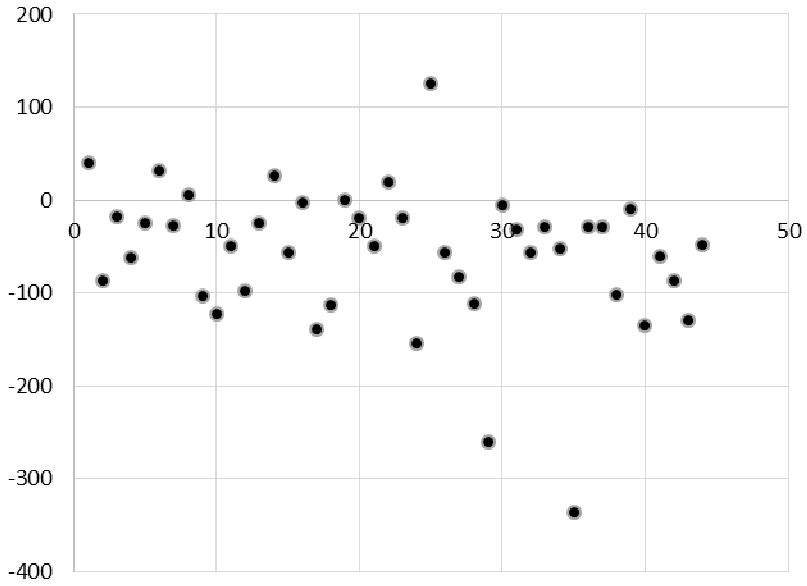


Figure 8. Distribution of Change in Wake Time

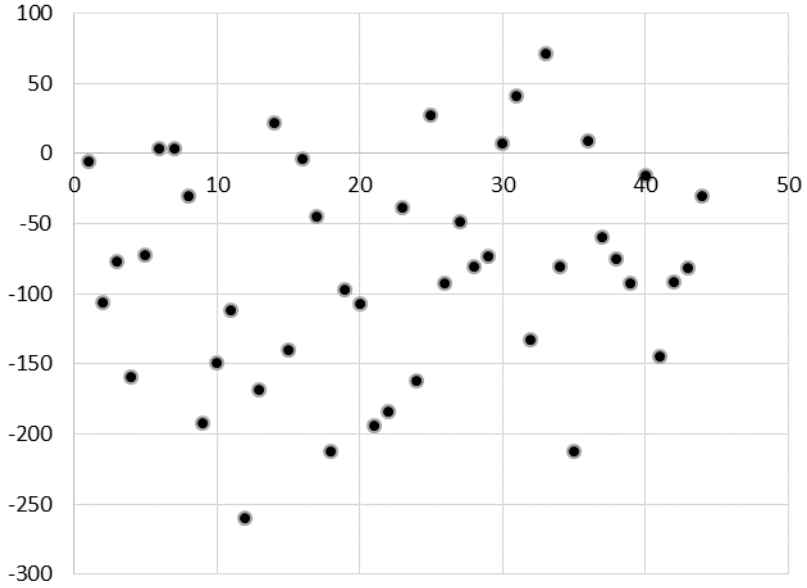


Figure 9. Distribution of Change in MVPA

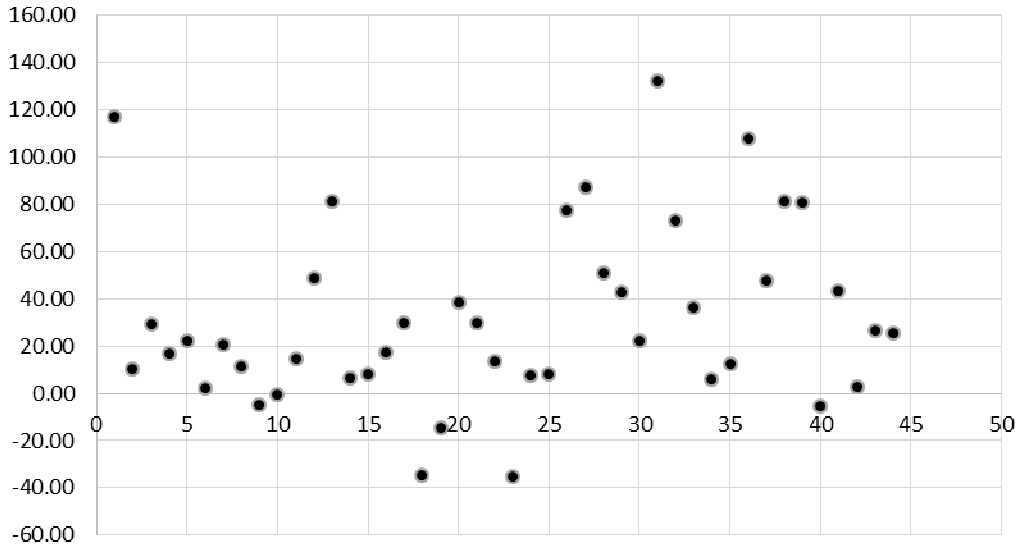


Figure 10. Distribution of Change in BRIEF-SR Inhibition

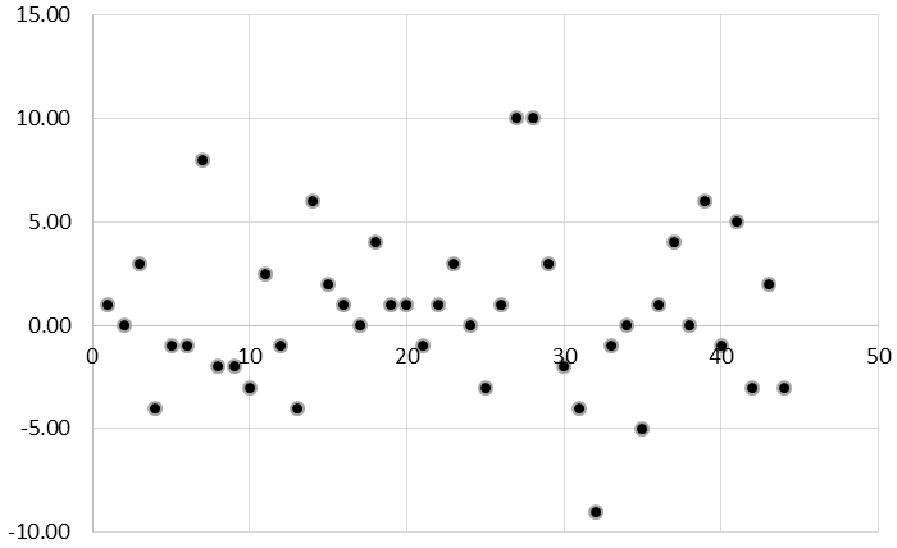


Figure 11. Distribution of Change in BRIEF-SR Shift

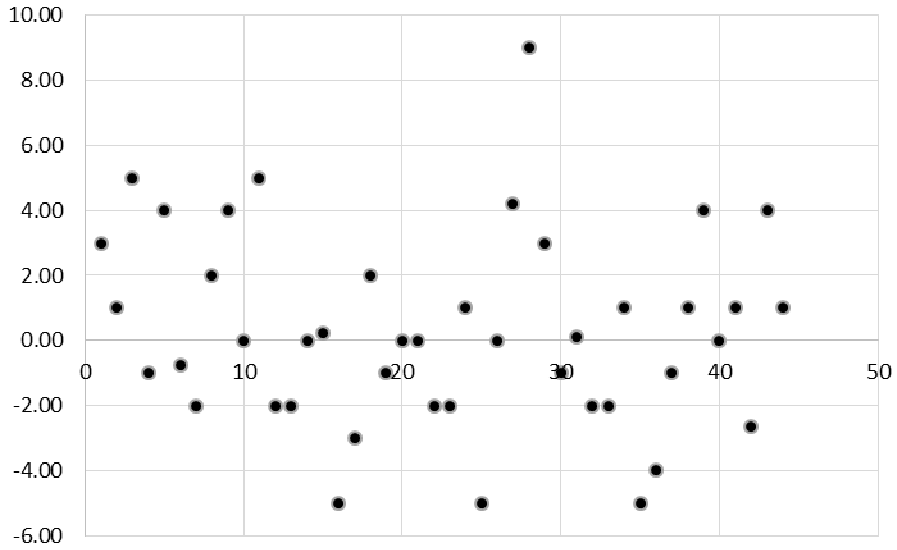


Figure 12. Distribution of Change in D-KEFS Inhibition

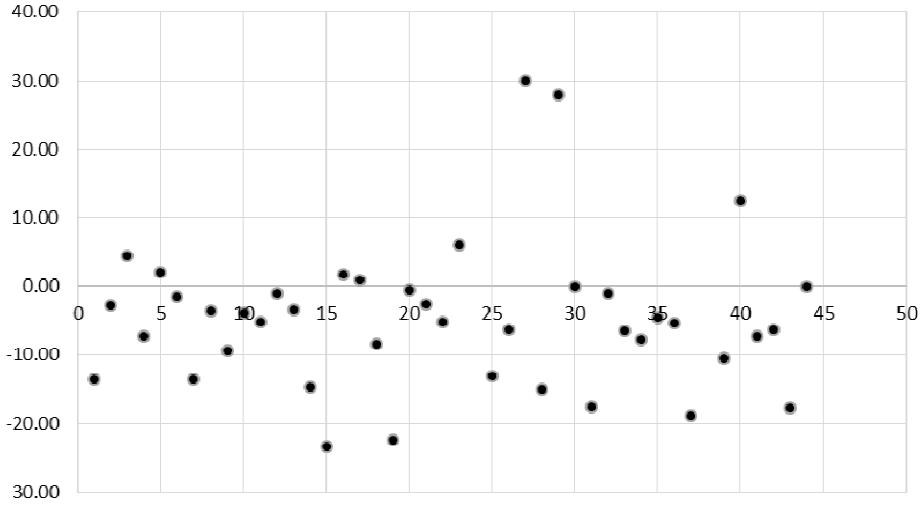


Figure 13. Distribution of Change in D-KEFS Inhibition/Switching

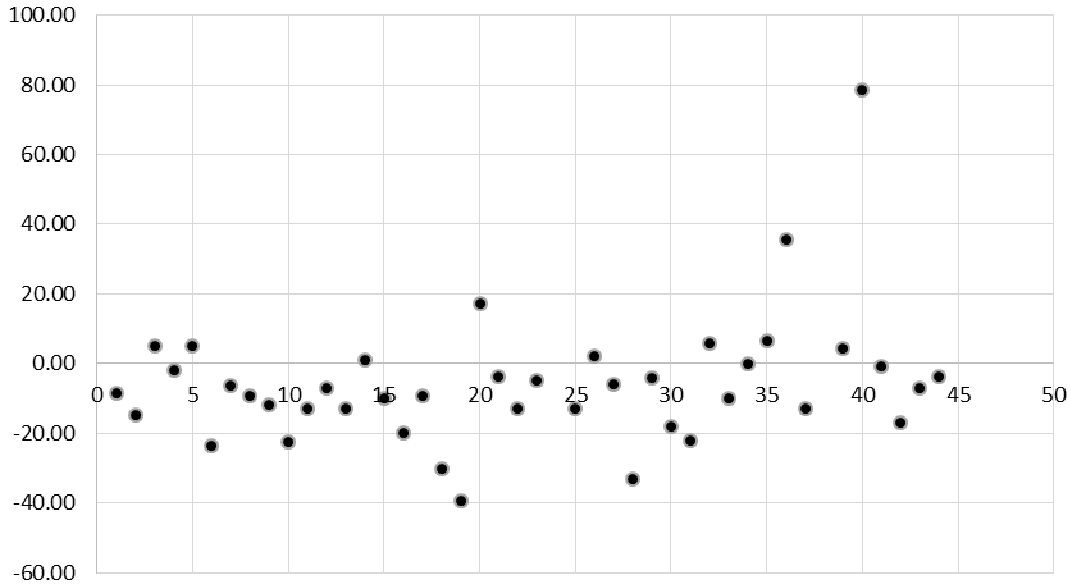


Figure 14. Distribution of Change in D-KEFS Inhibition Total Errors

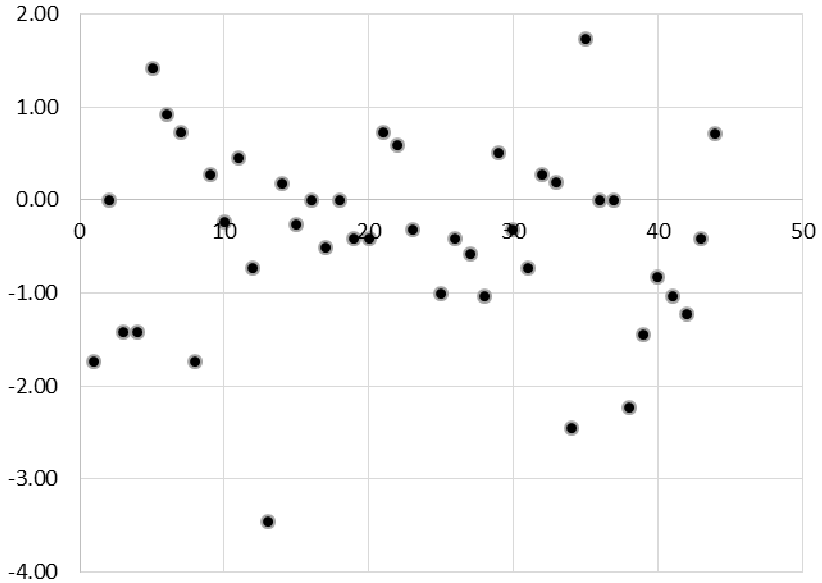
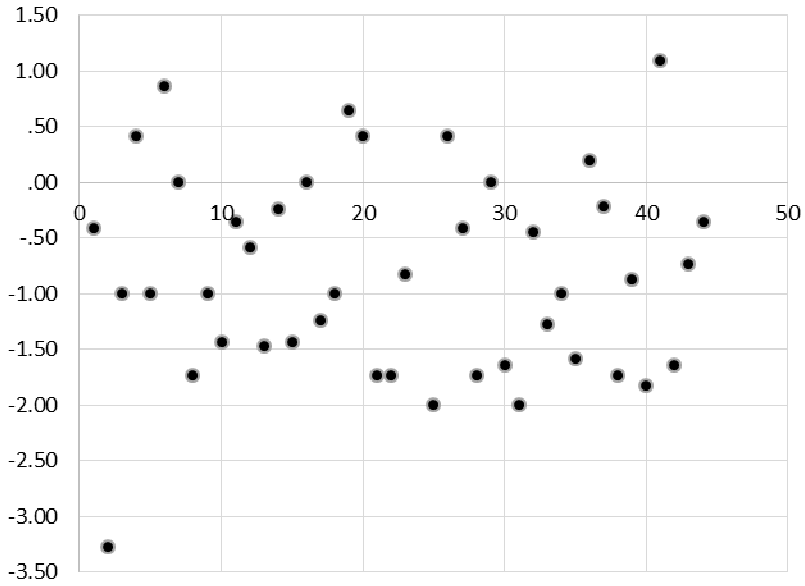


Figure 15. Distribution of Change in D-KEFS Inhibition/Switching Total Errors



Mediation Models

Bootstrapping analyses, including bias corrected (BC) confidence intervals (CI's, 95%), were used to test all proposed mediation models (see Preacher & Hayes, 2008; Preacher, Rucker, & Hayes, 2007) using a bootstrapped sample of n = 5,000. In the bootstrapping approach, the original study sample *n* is used as a population reservoir from which numerous pseudo (bootstrap) samples of *N* participants are randomly sampled with replacement from the original *n*. Next, for each bootstrap sample, *a* (the effect of the independent variable on the mediator) and *b* (the effect of the mediator on the dependent variable) are estimated from the samples, and the product of the path coefficients are recorded. These first two steps are then repeated a total of *k* times (Hayes, 2009, recommends *k*=5,000). Upon completion, this procedure results in *k* estimates of the indirect effect. The overall distribution of the indirect effect functions as an approximation of the sampling distribution of the indirect effect. The *k* estimates are

then used to generate a percentile-based bootstrap confidence interval (typically 95%), for which the cut points exclude (□/2) x 100% (typically 0.05) of the values from each tail of the distribution of the indirect effect. If zero is not included between the lower and upper bound of the confidence interval, then it is acceptable to claim that the indirect effect is not zero (Hayes, 2009; Shrout & Bolger, 2002). The bootstrapping technique has notable strengths in that inferences can be made based on estimates of the actual indirect effects themselves, it makes no assumptions about the shape of the sampling distribution or the indirect effects, and no estimates of standard error are needed. Thus, bootstrapping is considered to be the most valid and powerful method for examining indirect effects in mediation (Hayes, 2009).

Sleep, PA, and BMI. Models of mediation were tested to determine whether relations between sleep variables and zBMI were mediated by MVPA (see Figure 1). Three versions of the model were tested to account for all possible relations between data at T1 and T2 (see Table 2). Each version of the model was tested with all sleep variables, yielding 9 unique tests of mediation. None of these models with MVPA as a mediator yielded significant results.

As shown in Figure 2, a competing mediation model was also tested (e.g., sleep mediates relations between MVPA and zBMI). The model was tested with all sleep variables, yielding 3 unique tests of mediation. When testing T2 sleep onset as a mediator of relations between T1 MVPA and T2 zBMI, there was a significant direct effect of the mediator (T2 sleep onset) on the dependent variable (T2 zBMI). However, there were no significant total or direct effects of the independent variable (T1 MVPA)

on the dependent variable (T2 zBMI) or the independent variable (T1 MVPA) and the mediator (T2 sleep onset), so this is not considered significant mediation. None of the other tested models with sleep as a mediator yielded significant results.

Sleep, EFs, and BMI. Models of mediation were also tested to determine whether relations between sleep and zBMI were mediated by EFs (see Figure 3). Three versions of the model were tested to account for all possible relations between data at T1 and T2 (see Table 2). Each version of the model was tested with all possible combinations of EF and sleep variables, yielding 54 unique tests of mediation. None of the tested meditational models yielded significant results suggestive of mediation.

Change in PA, sleep, BMI. Models of mediation were used to test sleep variables at T2 as a mediator of relations between change in MVPA and T2 zBMI in the context of a summer camp program promoting physical activity physical activity (Figure 4). The model was run separately for each measure of sleep (i.e., duration, sleep onset time, and wake time) yielding 3 unique mediation models, however, none of the tested models yielded significant results suggestive of mediation.

CHAPTER FOUR

DISCUSSION

A primary aim of this study was to objectively characterize sleep patterns in a sample of urban minority girls during summertime, given the high risk for inadequate sleep and weight gain during the summer months among this demographic group. Although a handful of studies have suggested that youth experience shorter sleep durations during the summer months (Nixon et al., 2008, Bénéfice et al., 2004; Carson & Spence, 2010) and that minority youth sleep less than non-minority youth throughout the year (Crosby et al., 2005, Spilsbury et al., 2004; Wong et al., 2013), little is known about the relevance of summertime to sleep among urban minority girls. The National Sleep Foundation recommends 9-11 hours of sleep per night for school-age children (Hirshkowitz et al., 2015). Results from the current study utilizing objective measures suggest that low-income, urban, African American and Latina girls may sleep for a shorter period of time than is optimal for their age during the summertime months, as study participants averaged less than the recommended nine hours of sleep per night at both time points. These findings extend prior literature to provide more robust and objective evidence of short sleep durations among urban minority adolescents. Results also shed light on minority youth sleep during the summertime. Though researchers have hypothesized that minority youth may experience insufficient sleep during the summer months (Baranowski et al., 2014; Carson & Spence, 2010), this is the first study to

objectively examine that hypothesis. Prior research has demonstrated that adolescents who obtain even one hour less sleep per night than recommended experience up to an 80% increased likelihood of obesity (Gupta et al., 2002), putting much of the current sample at increased risk for obesity based on sleep patterns.

Although the design of the current study does not allow for examination of seasonal differences in sleep, the current sample reported shorter sleep durations than a comparable sample of urban minority girls during the school year (Spilsbury et al., 2004), suggesting the need for additional studies to examine ways in which summertime may be linked with decreases in sleep duration in this population. The current sample also experienced shorter sleep duration by 45 minutes than a study measuring summertime sleep durations in non-minority, non-urban youth using waist-worn actigraphy (Nixon et al., 2008), suggesting that urban minority youth may be particularly at risk for a lack of sleep during the summer months. Although the present study cannot determine whether differences are attributed to an urban environment, minority status, or seasonal variations, other research within urban samples has reported differences in sleep durations between minority and non-minority youth (Spilsbury et al., 2004).

Prior research has suggested that minority youth may sleep less due to more sleep problems, particularly bedtime resistance (Sheares et al., 2013). Indeed, the current study sample evidenced later sleep onset times by two hours than a large sample of non-minority, non-urban youth (Nixon et al., 2008). Furthermore, girls in the current study went to bed later on average than a sample of urban minority girls during the school year utilizing self-report measures (Spilsbury et al., 2004), highlighting the need for further

inquiry as to whether summertime may lead to even later sleep onset times among minority youth, especially girls. The disparity between sleep onset times and sleep durations in this sample and other comparable samples supports the hypotheses that summertime may be associated with late sleep onset times and short sleep for minority girls living in an urban environment.

Participants in this study recorded less sleep in the structured context (i.e., during summer programming), than in the unstructured context (i.e., before summer programming). Since sleep data in the unstructured context was collected during the initial stages of the summer vacation period, it is possible that participants who are normally short sleepers experienced a "sleep rebound," or an increase in sleep after a period of sleep restriction, during the first weeks of summer vacation (Binder, Hirokawa, & Windhorst, 2009). Sleep rebounds result in longer sleep than is typical for a short period of time, after which sleep returns to baseline levels.

Within the current sample, sleep differences were observed between race/ethnicity groups. Latina girls recorded earlier sleep onset times and more sleep than African American girls when not involved in structured programming. Researchers have suggested that the Latino "siesta culture" may buffer urban and sociocultural risk for poor sleep by promoting sleep during the day and thereby allowing for more sleep overall (Williams et al., 2015). While the current data measurement and analysis cannot determine whether Latina participants were sleeping more during the day (i.e., napping) than their African American counterparts, findings highlight the need for further investigation of sleep patterns among ethnic minority girls. Interestingly, according to the

"siesta" hypothesis, differences in sleep between Latinas and other race/ethnicities are most likely to emerge when the weather is hot and when youth are not in school (Williams et al., 2015), suggesting that Latino children may experience the most sleep as a result of "siestas" during unstructured time in the summer. Indeed, in our sample, Latina girls exhibited significant differences in sleep only in the unstructured context (i.e., when not in programming). On the other hand, the short sleep recorded by African American girls in this study is consistent with other studies in which African American youth reported shorter, more disrupted sleep than their other minority peers (Sheares et al., 2013; Williams et al., 2015). Researchers hypothesize that elements of the African American sociocultural context, such as lower socioeconomic status, more disadvantaged housing, and higher rates of comorbid health conditions, may serve as a particularly strong risk factors for poor sleep (Williams et al., 2015). However, studies have found that racial differences in sleep are minimized in urban environments, where sleep affecting factors such as noise, light, and crowding are often similar regardless of race (Gamaldo, McNeely, Shah, Evans, & Zonderman, 2015).

Although not a major aim of the study, several notable findings emerged regarding participant's sleep schedules during the summer. Youth in the current study shifted their sleep schedule considerably across time points, recording significantly earlier sleep onset times in the context of the summer camp program when compared to when not in programming. Although youth went to bed earlier during programming, they also woke up earlier, which may have been due in part to the program's scheduled start time of 9 AM each day. While both sleep onset time and wake time were substantially

earlier during programming, participants' wake times changed more than their sleep onset times, resulting in shorter sleep durations on average during programming. At both time points, sleep duration was more strongly associated with wake time than sleep onset time. While previous research has highlighted the importance of early sleep onset times for obesity prevention (Beebe et al., 2007; Snell et al., 2007), fewer studies have examined the relevance of wake times to obesity or obesogenic behaviors. However, a large body of research has examined the importance of school start times for adolescent wake time and total sleep duration (e.g., Kirby, Magi, & DiAnguilli, 2011; Owens, Belon, & Moss, 2010; Sadeh, Gruber, & Raviv, 2003; Wolfson, Spaulding, Dandrow, & Baroni, 2007). The current findings support research suggesting that wake times may play a particularly influential role in adolescents' abilities to obtain adequate sleep, and suggest that early start times for summertime programming may curtail participant sleep. Summer programs aimed at promoting adolescent health may benefit participants by adopting later start times to allow for later wake times and longer sleep durations. Additionally, programs may consider providing specific guidance to parents about the importance of implementing earlier bedtimes when programming is in session.

This study is the first to explore relations between sleep, PA, EFs, and zBMI during the summertime, both outside of structured programming and in the context of a structured summer day camp program. Results suggest that sleep schedules (rather than sleep duration) may also hold relevance for participants' activity level. When participants were not involved in structured programming, those who fell asleep earlier and woke up earlier were more physically active than those with sleep schedules shifted

later. In contrast, sleep duration was not associated with MVPA. Results suggest that sleep schedules, rather than sleep duration, may be related to PA during the summertime. Indeed, a recent review by Miller and colleagues (2015) highlighted shifted sleep schedules (i.e., later sleep onset times and later wake times) as an emerging risk factor for obesity beyond sleep duration, although more research is needed in children and adolescents. Shifted sleep schedules may put children and adolescents at risk for obesity through the impact of late sleep onset time and early wake times on health behaviors such as PA. Specifically, school-aged children who wake up later and go to bed later may spend a greater portion of their waking hours at night, and as a result may experience fewer opportunities to be physically active outdoors, especially in low-income urban neighborhoods where it may be unsafe to play outside after dark (Carver, Timperino, & Crawford, 2008). Conversely, it may be the case that earlier bedtimes and wake times for school-aged children during the summer are reflective of greater levels of family structure and routine (Anderson et al., 2015), which may positively impact on other health behaviors such as dietary intake and PA (Moreno et al, 2015).

In the context of structured programming, participants went to bed earlier, woke up earlier, and also obtained more MVPA. However, there were no significant correlations between these variables during programming, signifying that sleep onset time and wake time no longer related to levels of MVPA as they did in the unstructured context (i.e., before programming). While causation cannot be inferred with the current study design, it may be the case that the structured programming promoted activity in all girls, including those who were not as physically active in the unstructured context due to

late sleep onset times, late wake times, or overall fatigue. More research is needed to assess the relevance of sleep schedules, including sleep onset time, wake time, and sleep durations to physical activity, both in unstructured contexts and in the context of structured programming.

Contrary to hypotheses, sleep variables were not significantly related to zBMI before programming or during programming. Although a large body of literature supports relations between sleep and zBMI (Chen & Beydoun, 2008; Fatima et al., 2015; Patel & Hu, 2008), there are a few reasons why relations may not have been significant in the current sample. As previously stated, the sample is small, and therefore necessitated a large effect size to detect differences in sleep between participants of varying zBMIs. Furthermore, participants as a whole experienced less sleep than is recommended for their age at both time points, indicating that the entire sample is sleep-compromised. In the context of other powerful factors which may have impacted participant sleep, such as an urban environment and minority status, it is possible that individual levels of zBMI do not hold enough weight to emerge as a significantly associated with summertime sleep. Other studies have also failed to find relations between objectively measured sleep and BMI (Harrington, 2013).

Unexpectedly, zBMI was not associated with MVPA in this study. As relations between levels of activity and BMI are strongly supported in the literature (Jiménez-Pavón, Kelly, & Reilly, 2011), this finding may be the result of a small sample size. However, findings do correspond with previous literature demonstrating that early adolescent minority girls universally exhibit low levels of physical activity (Belcher et

al., 2010; Kimm et al., 2002; Nelson, Neumark-Stzainer, Hannan, Sirary, & Story, 2006). Prior research in Bohnert et al. (2014) has elaborated on baseline levels of MVPA in this sample and changes in MVPA during programming. In the unstructured context, the sample as a whole exhibited very little MVPA, regardless of zBMI. However, MVPA increased considerably within the context of structured programming, and it is encouraging to find that participants with higher zBMIs were engaging in as much activity during programming as participants with lower zBMIs.

Several interesting relations emerged between sleep, EFs, and zBMI. Consistent with literature documenting relations between poor sleep and limited EF capabilities (Esposito et al., 2013; Goel, Rao, Durmer, & Dinges, 2009), girls who went to bed later reported more daily problems with inhibition before programming began. Inhibition difficulties, such as those experienced in the context of late bedtimes and poor sleep, may be associated with in a limited ability inhibit behavior in response to a cue (i.e., feeling full), greater eating in response to external cues for food, emotional eating, and binge eating (Beebe et al., 2007; Burt et al., 2014; Durmer & Dinges, 2005; Fagundo et al., 2012). Indeed, participants with higher zBMIs in the current study also had more limited inhibition and shifting abilities at both time points, which further substantiates links between EF deficits and obesity (Smith, Hay, Campbell, & Trollor, 2011).

This study is one of the first to test PA and EFs as two independent mediators of relations between objective measures of sleep and zBMI. Contrary to expectation, there were no significant mediational pathways found in this study. However, several limitations of the dataset may have played a role. As previously mentioned, the summer

camp program's start time of 9 AM each day may have truncated possible variation in participant sleep during programming, leading to a limited range of values. Additionally, neither MVPA nor sleep variables were significantly associated with zBMI in the present sample, which likely also contributed to the lack of significant mediation findings in models examining mediation among these variables. Previous research has also failed to find associations between BMI and PA variables among youth (Harrington, 2013), despite research supporting relations (Jiménez-Pavón et al., 2011). Furthermore, though a growing body of literature has suggested that sleep may respond to changes in PA (Delisle et al., 2010; Ekstedt et al., 2013) and influence BMI (Jebb & Moore, 1999; Patel & Hu, 2008), evidence has been mixed and limited, and several studies have failed to demonstrate relations between objectively measured PA and sleep in youth and adolescents (Bénéfice et al., 2004; Hjorth et al., 2013; Soric et al., 2014). However, the theory behind these mediational hypotheses is still supported by researchers in the field (e.g., Baranowski et al, 2014; Patel & Hu, 2008).

Although models were not supported in the current study, there are reasons to further examine the notion that EF may mediate the relation between sleep and zBMI (Spruyt & Gozal, 2012). Results from this study suggest that later sleep onset times were associated with more observed daily problems with inhibition. Additionally, correlations showed that girls with lower inhibitory and shifting abilities tended to have higher zBMIs. This may be due to the possibility that youth with inhibition and shifting deficits may experience more difficulty with inhibitory control related to food and may struggle to inhibit themselves when food is available or shift attention away from food at times

when they are not truly hungry (Fagundo et al., 2012). Though these relations are consistent with hypotheses, the power to detect significant mediational pathways was limited by the small sample size.

Limitations and Future Directions

Despite many contributions of this study, there are several limitations that must be noted. The current study is limited by a lack of a control group and small sample size, which limit generalizability of the work as well as the ability to detect smaller effects. Additionally, the study does not contain the ideal longitudinal data to test the proposed mediation models. The use of change scores is also controversial within the field (Allison, 1990; Husted, Cook, Farewell, & Gladman, 2000; Norman, 1989), although this study benefits from an assessment of objective measures, rather than self-report measures which often observe a regression toward the mean, as well as substantial variability in within-group change over time (Maxwell & Howard, 1981). Further, although waist-worn actigraphy was warranted in this study due to the need to collect both PA and sleep data, the measurement of sleep at the waist may have induced some measurement error, likely over-estimating sleep at times (Hjorth et al., 2012). Future studies of sleep in urban minority youth should be conducted with polysomnography or wrist-worn actigraphy and, ideally, utilize three time points of data to test EF and PA as mediators longitudinally. Additionally, future research would benefit from a comparison of summertime and non-summertime levels sleep, BMI, PA, and EFs among urban minority girls in order to clarify summertime changes in sleep patterns for these youth and the relevance of such changes to other health behaviors and cognitive outcomes. Finally,

research on summertime sleep would benefit greatly from further examination of larger, family-level factors such as SES, access to childcare, family conflict and chaos, and the home environment, all of which may influence children's behaviors to a greater extent when school is not in session.

Acknowledging limitations, this study is nevertheless the first to characterize summertime sleep in a sample of urban minority girls using objective measures, as well as the first to examine differences in sleep between differing minority groups at this time. Given that the sample in the present study slept less than comparable samples of both minority adolescents during the school year and Caucasian adolescents during the summertime, results suggest that both time of year and ethnic or urban status may hold unique relevance for sleep patterns. Moreover, these combined factors may put urban minority youth at high risk for inadequate sleep during the summer. Inadequate sleep, particularly late sleep onset times and early wake times, may relate to lower levels of PA and more EF difficulties, both of which may be associated with greater adiposity. Overall, findings highlight inadequate summertime sleep as a relevant factor that may contribute indirectly to the increased obesity risk experienced disproportionately by low-income, ethnic minority, urban girls during summertime.

In light of this potential accumulation of obesity risk factors that may begin with or be exacerbated by inadequate summertime sleep, results suggest that obesity prevention and intervention programs should promote healthier summertime sleep habits in urban minority youth. A greater focus on sleep duration and sleep schedules during the summer may help to stem the typical trends of accelerated weight gain that occur

during the summer months for minority youth. Though pediatric obesity intervention programs have been widely studied (Waters et al., 2011), these interventions do not typically incorporate sleep hygiene, and to our knowledge, no studies have been published that incorporate promoting healthy sleep hygiene skills into obesity interventions for children. In sum, this study provides an important first step and demonstrates that more work is needed to examine the effects of summertime sleep and benefits of sleep interventions in an effort to reduce obesity among today's youth and adolescents.

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VITA

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