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Associations of Sleep Patterns with Metabolic Syndrome Indices, Body Composition, and Energy Intake in Children and Adolescents

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Abstract

Background: Self-reported short sleep duration is associated with greater risk for metabolic syndrome (MetS), obesity, and higher energy intake (EI). However, studies of these associations in children using objective methods are sparse.

Objectives: To determine the associations for sleep patterns with MetS indices, body composition, and EI using objective measures in children.

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Methods: Free-living sleep and physical activity were measured in 125 children (aged 8–17y, BMI- $z=0.57\pm1.0$, 55% female) using wrist-worn actigraphs for 14 nights. Blood pressure, fasting blood levels of lipids, insulin, glucose, waist circumference, and body composition (DXA) were obtained during outpatient visits. EI was assessed during an *ad libitum* buffet meal.

Results: Later weekday and weekend bedtimes were associated with higher systolic blood pressure ($p<.05$). Sleep duration and bedtime were not significantly associated with other components of MetS, body composition, or EI. Short sleepers (duration <7 h) consumed a greater percentage of carbohydrates than those with adequate (≥ 7 h) sleep ($p<.05$).

Conclusion: Indicators of sleep duration were variably associated with children's eating patterns and risk for chronic disease. Prospective data are needed to determine whether these indicators of sleep quality represent unique or shared risk factors for poor health outcomes.

Abstract

Clinical Trials Identifier: NCT02390765

Keywords

sleep; energy intake; eating; metabolic syndrome; obesity

Introduction

The National Sleep Foundation recommends that school-aged children receive 9–11 hours of sleep and teenagers receive 8–10 hours a night [1]. The Centers for Disease Control reports that many youths are not meeting these guidelines and that more than two-thirds of teenagers report sleeping ≤ 7 hours during school nights [2]. Meta-analyses indicate that self- and parent-reported short sleep in children, defined as ≤ 10 h per night, is associated with a 58–92% increased risk for obesity, with the shortest sleep duration (i.e., < 7 h for children aged 10 years and above) having the highest risk [3, 4]. Other pediatric studies have also found inverse associations for sleep duration with adiposity, waist circumference and body mass index (BMI) [5–7].

Several mechanisms have been proposed to explain the association between short sleep duration and obesity risk in children. Some data suggest that short sleep results in metabolic and endocrine changes that contribute to excess weight gain. Among healthy weight adolescent males, experimental reduction of sleep to 4 hours per night for 3 consecutive nights increased homeostasis model assessment of insulin resistance (HOMA-IR) index by 65% when compared to a long sleep condition (9 hours per night) [8]. Thus, short-term, severe sleep restriction acutely decreased insulin sensitivity, and if sustained, might increase the risk of excess weight gain and abnormalities in glucose homeostasis [9]. Most studies examining endocrine and hormonal changes associated with short sleep have been carried out in adults [10].

An alternate explanation is that short sleep duration provides individuals with more opportunities to eat and increases their desire for high-calorie foods and larger portions [11]. One systematic review using data from 33 studies that measured sleep objectively (via

polysomnography [PSG] or actigraphy) showed an association between short sleep duration and less favorable diet quality in children [12]. In a healthy pediatric sample (27% overweight/obese, 8–11y), children who decreased their sleep by 1.5 hours per night for 1 week reported consuming 134 kcal more per day than children who increased their sleep by the same amount [13]. In separate investigations of children (9–11y), short sleep duration assessed by accelerometry was associated with a higher percentage of energy intake (EI) from carbohydrates and a lower percentage from fats [14], while self-reported shorter sleep duration on weekday nights was associated with more frequent consumption of fast food and sweets and a lower consumption of fruits and vegetables [15].

Sleep timing behavior is also associated with excess weight and poor eating habits in pediatric samples. For instance, children with a delayed bedtime and increased REM sleep measured through PSG reported higher hunger scores the following morning [16]. In a nationally representative sample, parental report of children (3–12y) who slept less or went to bed later at baseline assessment had higher BMIs and were more likely to be overweight at their 5-year follow-up compared to children who slept more than 11 hours a night [17]. Children who demonstrated a tendency to both fall asleep late at night and wake up later in the morning (i.e., late bed-late rise), as measured by actigraphy, also reported less frequent consumption of fruits and vegetables, and more frequent consumption of sweetened beverages compared to children who tended to fall asleep early and wake up early (i.e., early bed-early rise), even after controlling for total sleep duration [18]. In a separate study, youth (9–16y) who were in the late bed-late rise category, through self-report sleep data, had a higher BMI-z score and a lower diet quality, as compared to the early bed-early rise category, despite similar sleep duration [19]. A recent study in young adults shows that individuals who had later bedtimes and wake-up times consumed fewer highly palatable foods during breakfast the following morning but greater consumption of highly palatable foods during dinner [20]. This pattern of EI, characterized by a delayed shift of the consumption of highly palatable foods, may predispose individuals with late-to-bed sleep habits to weight gain and obesity.

Existing literature regarding the associations for sleep patterns with body composition and EI have overwhelmingly relied on self-report measures. Children are notoriously biased in their reports of their own eating habits [21], and typically overestimate their total sleep duration [22]. Moreover, few studies have examined the link between children's sleep patterns and other indicators of health risk. We sought to analyze the associations for children's sleep patterns with their metabolic syndrome (MetS) indices, body composition, and EI using objective measures. We hypothesized that short sleep duration and later bedtime would be associated with 1) increased serum triglycerides, total cholesterol and LDL-cholesterol, lower HDL-cholesterol, higher diastolic and systolic blood pressure, a larger waist circumference, and higher HOMA-IR; 2) increased BMI-z and adiposity; and 3) greater total EI, a higher percentage of carbohydrates consumed, and a lower percentage of fats and/or protein consumed. Exploratory analyses were also conducted to determine if average sleep duration during the week versus the weekend was differentially associated with MetS indices, body composition, or EI.

Methods

Participants

Healthy boys and girls (8–17 years old) were recruited to participate in the Children's Growth and Behavior Study (Clinical Trials Identifier: NCT02390765), which was designed to understand growth and health behaviors in children. The protocol was approved by the Institutional Review Boards at the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development and the Uniformed Services University of the Health Sciences. All participants were in good general health, with the exception of minor, well-controlled ailments. Exclusion criteria included: 1) history of major illness, brain injury or pregnancy; 2) use of medication known to affect body weight or EI; 3) significant and recent weight loss (>5% body weight); 4) BMI kg/m² < 5th percentile; 5) current and regular use of illicit substances; 6) presence of a full-threshold psychiatric disorder; and 7) full scale intelligence quotient score < 70 [23]. Signed informed consent and assent were obtained from parents/caregivers and children, respectively.

Procedures

First Screening Visit.—Participants completed two screening appointments at the outpatient Pediatric Clinic at the National Institutes of Health Hatfield Clinical Research Center. A physical examination was performed by a physician or nurse practitioner to measure waist circumference [24]. Participants also completed the Children's Depression Inventory (CDI) [25]. Children were instructed to wear accelerometers on their non-dominant wrist for 14 days and nights between the first and second screening visits, removing it only when swimming or participating in contact sports.

Second Screening Visit.—The second screening visit started at approximately 8:00am. Participants were asked to consume nothing but water starting at 10:00pm the night prior to their visit. Height (cm) was measured in triplicate to the nearest millimeter by a calibrated stadiometer, and weight (kg) was measured using a calibrated scale after participants removed shoes and heavy clothing. BMI percentiles and z-scores, adjusted for age and sex, were computed according to the Centers of Disease Control and Prevention growth standards, with overweight being defined as a BMI percentile of 85th - <95th or BMI-z of 1.0–1.64, and obesity defined as BMI percentile of ≥95th or BMI-z >1.64 [26]. Participants had blood drawn while fasting during the second screening visit to determine triglycerides, cholesterol [total cholesterol, LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C)], glucose, and insulin concentrations, measured by the NIH Clinical Center Department of Laboratory Medicine [27]. An index of insulin resistance, HOMA-IR, was calculated by multiplying fasting insulin (mIU/mL) by fasting glucose (mg/dL) and dividing by 405.

At approximately 9:30am, participants were provided with a standardized breakfast shake intended to supply 21% of estimated daily energy needs [28], as determined by measured weight and height and average physical activity level [29]. Body composition was measured using Dual-Energy X-Ray Absorptiometry (DXA; iDXA system, GE Healthcare, Madison WI) to determine total lean mass and fat mass. At 12:30pm, participants were given an *ad libitum* buffet lunch meal to determine EI and macronutrient intake.

Measures

Sleep and Physical Activity

Wrist-mounted triaxial accelerometers (ActiGraph LLC, Pensacola, FL, model GT3X+) were initialized to record at a 30Hz sampling frequency. While wearing the actigraph on non-dominant wrists, participants were asked to fill out a paper sleep diary twice a day for 14 days (wake-up time and bedtime) and make notes related to any events that might have significantly influenced their sleep (e.g., severe weather). The raw tri-axial accelerometer data were downloaded from the devices and subsequently filtered and aggregated into 1-minute activity counts with Actilife software version 6.13.3 (Actigraph, Pensacola, FL, USA). The Sadeh sleep detection algorithm validated for adolescents [30] was applied in Actilife software, and auto-detected bed- and rise-times were adjusted based on visual inspection and participant sleep diaries. Bedtime (first minute of a 5-minute period in which the activity counts were all zero), wake-up time (first minute of a 5-minute period in which the activity counts were all greater than zero), and total sleep time (time in bed minus sleep onset latency minus minutes of wake after sleep onset) were calculated. If a nap occurred <60 minutes from the wake-up time, the nap was included as part of the total sleep time. Only participants with 3 valid week nights and 1 weekend night of sleep, determined by visual inspection, were included in the final analysis.

Total physical activity was expressed as 3D vector magnitude from the filtered accelerometry data. Vector magnitude was calculated by taking the square root of the sum of the square of acceleration for each of the three axes and averaged over minutes of valid wear time (> 600). This measure of total physical activity is significantly correlated with activity energy expenditure [31].

Energy Intake

Total EI (kcal) and macronutrient composition (% of total intake) were measured during a validated lunchtime laboratory test meal ($>10,000$ kcal; ~12% protein, 32% fat, 56% carbohydrate) [32]. Participants were individually studied in a room without other food-related stimuli and given a tape-recorded instruction to “Let yourself go and eat as much as you want. Take as much time as you need, and open the door when you are done.” Amounts of food and beverage consumed were calculated by weighing each food or beverage item once before and once after the meal to the nearest tenth of a gram. Foods consumed were determined by subtracting the food weights after the participant’s meal from initial weights. Food EI and macronutrient content of the consumed meal were calculated using ProNutra (Viocare Technologies, Princeton, NJ). Food items on the array included: various breads/rolls, sandwich meats, condiments, and cheeses; chicken nuggets; fruits and vegetables; cookies and candies; chips and pretzels; and milk, juice, and water.

Data Analytic Plan

All analyses were conducted using SPSS for Windows version 24 (SPSS, INC., Chicago, IL). Data were screened for normality. Arcsine square root transformations were conducted for percentage of macronutrient content intake, percentage fat, and percentage lean mass to enhance normality. Z-scores adjusted for age and sex were calculated for waist

circumference [33], diastolic blood pressure, systolic blood pressure [34], total cholesterol, LDL-C, HDL-C, and triglycerides [35]. Age-adjusted residuals for sleep were created for sleep duration and bedtime (weekday, weekend, and total) to avoid concerns with multicollinearity.

Multiple linear regressions were conducted to examine the main effects of total sleep time and bedtime on the dependent variables of MetS indices (waist circumference, blood pressure, cholesterol, triglycerides, and HOMA-IR), body composition, total EI (kcal), and macronutrient content intake (% protein, % fat, % carbohydrate). To examine the effects of short sleep on MetS indices, body composition, and EI, analyses of covariances were conducted with short sleep (<7 hours of sleep) and adequate sleep (≥ 7 hours of sleep) as the independent variables. Given the links for depressive symptoms with sleep, MetS indices, and EI in children [36–38], CDI total score was considered as a covariate in all models. As bedtimes differed between youth who wore an actigraph during the school year versus the summer break ($p<.001$), time of assessment was also considered as a covariate in all models. Depressive symptoms and time of assessment were retained in each model only if they were significant. Analyses related to MetS indices adjusted for race (0=Non-Hispanic white, 1=other), sex, fat mass (kg), lean mass (%), and physical activity [39]. Analyses related to body composition adjusted for race, sex, and physical activity. Analyses related to EI and macronutrient intake adjusted for race [40], sex, lean mass (kg), fat mass (%), and physical activity. Height was considered as a covariate, but it was found to be highly correlated with lean mass ($r=.90$); as such, only lean mass was retained in the relevant models. For these exploratory analyses, $p<.05$ was considered significant.

Results

Participants

The study sample included 147 children; 12 youths were excluded due to non-compliance and 10 were excluded due to device malfunction. Those with missing actigraphy data did not differ from the studied sample significantly in sex, race, age, or BMI-z ($ps>.22$). A total of 125 participants (12.4 ± 2.6 y; 55% female; 46% Non-Hispanic white) were included in the final analyses (Table 1). Missing data in this final sample were minimal. Three children did not have test meal data (e.g., food measurements not able to be obtained); 1 participant's lipid levels were not able to be calculated by the laboratory medicine team; and 1 participant's waist circumference measurement was not taken during the physical exam. Individuals with these missing data were excluded from relevant analyses.

Sleep and Metabolic Syndrome Indices

After adjusting for race, sex, fat mass, percentage lean mass, physical activity, depressive symptoms, and time of assessment, sleep duration (total, weekday, or weekend) was not significantly associated with markers of MetS ($ps>.051$). Later bedtimes, both during the week and weekend, were significantly associated with higher systolic blood pressure z-scores ($ps<.05$; Table 2, Figure 1A and 1B) but were not associated with waist circumference z-score ($ps>.06$) or other measures of MetS ($ps>.11$). There were no group

differences between children with short sleep and adequate sleep for any MetS component ($p>.37$).

Sleep and Body Composition

After adjusting for race, sex, physical activity, depressive symptoms, and time of assessment, sleep duration (total, weekday, or weekend) was not significantly associated with BMI-z ($p>.28$) or fat mass ($p>.22$). Bedtime (total, weekday, or weekend) was also not significantly associated with BMI-z ($p>.18$) or fat mass ($p>.30$). There were no significant differences between children with short versus adequate sleep duration for body composition ($p>.71$; Table 1).

Sleep and Energy Intake

Neither sleep duration nor bedtime (total, weekday, or weekend) were associated with total EI ($p>.71$) or macronutrient intake ($p>.06$) after adjusting for race, sex, lean mass, fat mass, and physical activity. Yet, children with short (<7h) versus adequate (≥ 7 h) sleep consumed a greater percentage of calories from carbohydrates during the test meal [Table 1; $F(1, 109) = 4.18$, $\eta^2_p = .04$, $p<.05$]. There were no significant differences between the two groups in total energy intake ($p=.58$), or percentage of consumption from protein ($p=.47$) or fat ($p=.06$).

Discussion

The current study utilized objective methods to evaluate the associations for children's sleep duration and bedtime with their metabolic functioning, body composition and EI. Contrary to our hypotheses, the large majority of these associations were non-significant. However, children who slept less than 7 hours per night consumed a greater percentage of carbohydrates during a lunch buffet test meal compared to their peers who slept more than 7 hours, consistent with previous findings [14]. A previous study has shown that sleep-restricted adolescents increase their consumption of foods with a high glycemic index, such as desserts and sweets, and trend towards greater consumption of carbohydrates [41]. Greater consumption of carbohydrates associated with short sleep duration may be a primary mechanism through which these children experience increased risk for obesity and obesity-related diseases such as type 2 diabetes [3].

There was also a significant positive association for later bedtimes (during both the week and weekend) and systolic blood pressure. Studies suggest that long term exposure to circadian disruption is a risk factor for the development of MetS components [42]. Of particular interest, disruption in sleep timing can lead to disturbances in the circadian rhythmicity of blood pressure [43]. Blood pressure varies diurnally, rising during the day and dipping at night. The loss of this pattern due to disrupted circadian rhythm is correlated with insulin resistance and cardiovascular disease [44, 45]. It is unclear why the current study found a significant association only between sleep timing and systolic blood pressure. Larger replication studies with a greater proportion of children with obesity are needed to determine possible associations between sleep timing and other MetS components. There were non-significant trends for later bedtime and greater waist circumference z-scores ($p=$

056), as well as for less weekend sleep duration and higher serum triglycerides ($p=.051$). These findings may be the result of limited variability in MetS values and/or limited power due to sample size.

Previous studies have also found a significant association between short sleep duration and MetS components [6, 8] and a U-shaped association between sleep duration and BMI in children, indicating that the highest level of risk are among those with very low and very high amounts of sleep [46]. In the current sample of healthy boys and girls, average sleep duration of the 14 nights was approximately 7 hours, with a range of 4.9 – 8.7 hours. It is possible that the present sample did not include enough participants on the “extreme” ends of sleep duration (e.g. extreme short sleepers or long sleepers), thereby attenuating our ability to detect significant associations with body composition or MetS components.

The findings that sleep duration and bedtime were not significantly associated with other MetS indices, body composition, total EI, or macronutrient intake were unexpected. It is certainly possible that 14-day data do not capture chronic sleep patterns that could influence body composition and BMI longitudinally. Previous studies have shown significant associations for children with shorter sleep duration and greater EI, poorer diet quality, and greater frequency of eating in response to external cues for food [13, 47]. However, these studies have relied on self-report measures for food intake that may not accurately represent children’s eating behaviors. The current study captured EI during a lunch meal, but the length of sleep during the prior night may only demonstrate effects on food choices during breakfast and/or dinner [48].

Study strengths include a racially/ethnically diverse sample, and the inclusion of healthy boys and girls who had a broad range of ages and weights. Additionally, the use of objective measures of children’s physical activity, sleep and eating patterns provide precise measurements of these notoriously difficult to quantify variables [21]. Limitations of the study include the use of cross-sectional data, and thus, no causal or mechanistic conclusions can be drawn from the findings. Moreover, while we excluded children taking medications known to affect weight and appetite, we did not exclude those taking medications that may affect sleep. Given the exploratory nature of the current study, additional studies with larger samples with more diversity in sleep duration are needed and may provide greater insight into the associations for sleep patterns with MetS indices and food intake among those at highest risk for obesity and its comorbidities. Relatedly, it is important for future research to consider the quality of children’s sleep. Children’s perception of the quality of their sleep is associated with obesity, independent of duration [49]. Similarly, poor sleep quality, as measured by PSG, has been linked to insulin resistance in a small sample of children [50]. Finally, inflammation is also a marker of disease risk and is associated with shorter sleep duration in adolescence [51]. Future studies should further investigate the associations between children’s sleep patterns and subclinical inflammation.

While previous research has found an association between short sleep duration and metabolic functioning in children, our study indicated that specific indicators of sleep duration are variably associated with children’s eating patterns and risk for chronic disease.

Prospective data are needed to determine whether these indicators represent unique or shared risk factors for poor health outcomes.

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Figure 1:

The association between bedtime and systolic blood pressure was significant for both weekday (Figure A, $p < .05$) and weekend nights (Figure B, $p < .05$). All analyses adjusted for age, sex, race, fat mass, percentage lean mass, physical activity, depressive symptoms and time of assessment. Bedtimes was converted to clock time to ease interpretation.

Table 1.

Demographic, sleep duration, energy intake, and physiological data for total sample, short sleep, and adequate sleep

Characteristics ^a	Total Sample (N=125)	Short Sleep (<7h; n=43)	Adequate Sleep (≥7h; n=82)
Age (y)	12.4±2.6 (8.0–17.0)	13.4±2.4 (9.0–17.0)*	11.8±2.5 (8.0–16.0)
Race/Ethnicity			
Non-Hispanic White (%)	45.6	25.6	56.1
Non-Hispanic Black (%)	28.0	37.2	23.2
Non-Hispanic Other (%)	19.2	25.6	15.8
Sex			
Female (%)	55.2	44.2	61.0
Physical Activity	245.7±57.4 (124.5–417.8)	229.4±44.9 (124.5–320.9)*	254.3±61.5 (153.5–417.8)
Total Sleep Duration (minutes)	434.3±46.8 (294.0–525.9)	384.9±32.9 (294.0–417.6)	460.3±28.6 (420.4–525.9)
Weekday Sleep Duration (minutes)	427.6±54.6 (204.1–519.2)	373.0±45.5 (204.1–433.2)	456.2±33.0 (383.9–519.2)
Weekend Sleep Duration (minutes)	450.5±61.5 (317.0–637.5)	413.6±67.4 (317.0–637.5)	469.9±48.3 (353.0–575.3)
Total Week Bedtime ^b	11:33PM±79.9 (8:51PM–3:30AM)	12:18AM±75.9 (8:51PM–2:55AM)	11:09PM±71.3 (8:51PM–3:30AM)
Weekday Bedtime ^b	11:20PM±82.1 (8:29PM–3:48AM)	12:03AM±78.2 (8:50PM–2:42AM)	10:58PM±75.4 (8:29PM–3:48AM)
Weekend Bedtime ^b	12:04AM±84.9 (8:40PM–4:32AM)	12:54AM±83.0 (8:53PM–4:32AM)	11:37PM±73.6 (8:40PM–3:47AM)
BMI ^c z-Score	0.57±1.0 (–1.6–2.8)	0.57±1.8 (–1.1–2.5)	0.56±1.2 (–1.6–2.8)
Lean Mass (%)	68.2±8.7 (43.0–85.2)	69.0±14.5 (43.0–84.6)	68.4±10.0 (44.1–85.2)
Fat Mass (%)	28.1±9.2 (10.0–54.8)	27.3±15.6 (10.8–54.8)	28.0±11.0 (10.0–53.6)
HOMA-IR	3.0±2.3 (0.4–14.5)	3.1±3.0 (0.4–14.5)	2.9±2.1 (0.5–11.5)
Waist Circumference z-score	0.30±0.68 (–0.60–2.78)	0.25±1.2 (–0.57–2.18)	0.32±0.86 (–0.60–2.78)
Systolic Blood Pressure z-score	0.22±0.85 (–1.99–2.64)	0.23±1.6 (–1.21–1.37)	0.22±1.1 (–1.99–2.64)
Diastolic Blood Pressure z-score	–0.20±0.71 (–2.06–1.69)	–0.27±1.3 (–1.74–1.01)	–0.17±0.88 (–2.06–1.69)
Triglycerides z-Score	–0.44±0.85 (–1.56–2.73)	–0.32±1.6 (–1.54–1.97)	–0.48±1.1 (–1.56–2.73)
Total Cholesterol z-Score	–0.28±0.56 (–1.70–1.63)	–0.20±1.1 (–1.70–0.90)	–0.21±0.75 (–1.56–1.63)
HDL-C ^d z-Score	1.34±3.16 (–5.89–10.96)	1.21±5.3 (–4.24–10.96)	1.27±3.6 (–5.89–10.14)
LDL-C ^e z-Score	–0.44±0.95 (–2.04–2.81)	–0.47±1.7 (–2.04–1.98)	–0.43±1.1 (–2.03–2.81)
Total Energy Intake (kcal)	956.7±421.6 (141.8–2535.4)	972.1±701.2 (358.3–2017.9)	927.1±496.5 (141.8–2535.4)

Characteristics ^a	Total Sample (N=125)	Short Sleep (<7h; n=43)	Adequate Sleep (≥7h; n=82)
Protein Consumed (%)	14.2±3.7 (6.4–34.5)	13.6±6.8 (7.9–23.3)	14.5±4.8 (6.4–34.5)
Fat Consumed (%)	35.9±7.0 (17.2–49.6)	33.9±12.6 (17.2–49.6)	36.7±8.9 (20.6–48.2)
Carbohydrate Consumed (%)	50.0±7.6 (32.1–69.8)	52.5±13.6 (36.2–68.2)*	48.9±9.7 (32.1–69.8)

^aValues presented are unadjusted mean ± standard deviation (range), unless otherwise noted.

^bBedtime ± standard deviation, presented in minutes.

^cBMI = body mass index;

^dHDL-C = High-density lipoprotein cholesterol;

^eLDL-C = Low-density lipoprotein cholesterol; MetS data are adjusted for race, sex, fat mass, percentage lean mass, and physical activity; Body composition data are adjusted for race, sex, and physical activity; Energy Intake data are adjusted for race, sex, lean mass, percentage fat mass, and physical activity; All sleep, body composition, and MetS data are adjusted for depressive symptoms and time of assessment and retained only if significant; Data presented are non-transformed for ease of interpretation;

* $p < .05$.

Table 2.

Linear Regressions for Metabolic Syndrome Indices and Sleep Patterns

Systolic Blood Pressure z-score	b^a	$SE(b)$	β^b	p -value	R^2^c
Race	-0.08	0.15	-0.05	.59	
Fat Mass	-0.01	0.02	-0.12	.52	
Percentage Lean Mass	-1.95	1.55	-0.22	.21	
Physical Activity	0.001	0.002	0.07	.50	.04
Total Week Bedtime	3.56	1.51	0.22	.02 *	.08 *
Systolic Blood Pressure z-score	b^a	$SE(b)$	β^b	p -value	R^2^c
Race	-0.08	0.15	-0.05	.59	
Fat Mass	-0.01	0.02	-0.12	.52	
Percentage Lean Mass	-1.95	1.55	-0.22	.21	
Physical Activity	0.001	0.002	0.069	.50	.04
Weekday Bedtime	3.32	1.45	0.21	.02 *	.08 *
Systolic Blood Pressure z-score	b^a	$SE(b)$	β^b	p -value	R^2^c
Race	-0.08	0.15	-0.05	.59	
Fat Mass	-0.01	0.02	-0.17	.52	
Percentage Lean Mass	-1.95	1.55	-0.22	.21	
Physical Activity	0.001	0.002	0.069	.50	.04
Weekend Bedtime	3.44	1.41	0.23	.02 *	.08 *

^a b = unstandardized regression coefficient;^b β = standardized coefficient Beta;^c R^2 = proportion of variability in the dependent variable accounted for by model;* $p < .05$