

ORIGINAL ARTICLE

Sleep duration, timing, variability and measures of adiposity among 8- to 12-year-old children with obesity

M. Zhou¹ , C. Lalani¹, J. A. Banda² and T. N. Robinson³

¹Stanford University School of Medicine, Stanford, CA, USA; ²Department of Health and Kinesiology, Purdue University, West Lafayette, IN, USA; ³Stanford Solutions Science Lab and Division of General Pediatrics, Department of Pediatrics and Stanford Prevention Research Center, Department of Medicine, Stanford University, Stanford, CA, USA.

Received 14 September 2018; revised 23 September 2018; accepted 25 September 2018

Address for correspondence: M Zhou, Stanford Solutions Science Lab, 1265 Welch Road, Medical School Office Building, Room X129, Stanford, CA 94305, USA. E-mail: myzhou@stanford.edu

Summary

Objectives

Sleep disruption in laboratory studies increases adiposity and decreases glucose tolerance. However, few epidemiological studies have used objective measures of sleep. This study aims to assess associations between sleep duration, timing and regularity with measures of adiposity.

Methods

This is a cross-sectional study of 188 children with obesity (age: 10.50 ± 1.39 years; body mass index: 29.24 ± 5.04 kg m⁻²). Nightly sleep duration, bedtime and wake time were measured by multiple-day actigraphy and parent reports. Per cent overweight (per cent over median body mass index for age and sex) was chosen as the primary measure of obesity status. Objective measures of height, weight, waist circumference, blood pressure, fasting blood lipids, glucose, insulin, glycated haemoglobin and C-reactive protein were obtained. Television screen time and total caloric intake were assessed via parent questionnaire.

Results

Each hour later in weekday bedtime was associated with an additional 6.17 per cent overweight (95% confidence interval [CI]: 1.42–10.92). Each hour greater in day-to-day variability in weekday bedtime and weekday wake time was associated with an additional 10.20 (95% CI: 0.50–19.91) and 10.02 (95% CI: 1.55–18.50) per cent overweight, respectively. Associations were similar after controlling for other obesity-related behaviours (television screen time, total caloric intake and physical activity.)

Conclusions

Among children with obesity, later bedtime and greater variability in bedtime and wake time are associated with greater adiposity, independent of other obesity-related behaviours. Early bedtime and wake time and consistent day-to-day sleep timing may be strategies to reduce adiposity in high-risk children.

Keywords: Accelerometer, obesity, paediatrics, sleep.

Introduction

Childhood obesity is considered one of the most serious global public health challenges of the 21st century. Children with obesity are at high risk for prediabetes, dyslipidaemias, hypertension, non-alcoholic fatty liver disease and obstructive sleep apnoea and are more likely to stay obese into adulthood and to develop associated comorbidities at a younger age (1). Improving sleep has

been suggested as one strategy to improve weight status in children with obesity. Short sleep duration and poor sleep quality are associated with higher consumption of carbohydrates (2), energy-rich foods (3) and added sugar and sugar-sweetened beverages (4). A recent meta-analysis of studies on sleep–obesity associations in children and adolescents concluded that sleep timing and variability in sleep patterns contribute to obesity risk beyond sleep duration (5). Late bedtime is associated

with a higher intake of energy-dense, nutrient-poor foods and a higher body mass index (BMI) z-score (6). Moreover, later bedtime has been associated with decreased physical activity during the day (7).

However, there remain important gaps in knowledge about these relationships. Most previous studies included predominantly White/Caucasian populations with high socioeconomic status (8) and have not included racially/ethnically and socioeconomically diverse samples, a vulnerable and less often studied population. Moreover, most prior community-based epidemiology studies have been limited to self-reports or parent-reports of sleep. To limit bias inherent in self-reporting and parent-reporting, recent studies have begun to incorporate objective measures of sleep with wearable devices (accelerometers) to measure sleep duration and sleep timing (bedtime and wake time). While polysomnography is the gold standard for measuring sleep parameters, it is typically used for studying a single night of sleep in the artificial environment of a sleep laboratory in a small sample of children. Accelerometers allow researchers to study children in their natural habitat over multiple days. Furthermore, most prior studies have not included a large sample of children with obesity who may respond differently to prevention strategies than children with normal BMI (9,10). More research is also needed to answer whether sleep timing influences adiposity indices independent of sleep timing's influence on physical activity and dietary intake. Finally, no study has simultaneously studied television screen time and accelerometer-measured sleep timing in the same population. Television viewing has been associated with increased food intake, decreased physical activity and increased adiposity in cross-sectional and longitudinal studies (11–14). Little is known regarding whether television screen time is directly associated with objectively measured sleep parameters. The hypotheses of the present cross-sectional study are that (i) objective accelerometer-measured sleep parameters differ significantly from parent-reported sleep parameters, (ii) shorter sleep duration, delayed sleep timing and greater variability in sleep timing are associated with greater measures of adiposity and (iii) these associations are independent of other obesity-related behaviours (television screen time, dietary energy intake and moderate-to-vigorous physical activity).

Methods

Study sample, eligibility criteria and exclusions

Families with 8- to 12-year-old children with obesity (BMI \geq 95th percentile for age and sex on the 2000

Centers for Disease Control and Prevention growth standards) (15) were recruited for participation in a family-based paediatric weight control study (Stanford CHANGES). Families were recruited via local newspaper advertisements, letters and flyers to local physicians, clinics, school nurses, after school programmes and referrals from past patients. Families were excluded if children were diagnosed with a medical condition affecting growth (for instance, a genetic or metabolic disease/syndrome associated with obesity), were currently taking medications affecting growth or had a developmental or physical disability limiting their participation in the measures. Exclusionary criteria were ascertained by direct measurement (e.g. BMI) and by child and parental report. Parents provided written informed consent for themselves and for their children, and children provided written assent for their participation. The study was approved by the Stanford University Administrative Panel on Human Subjects in Medical Research. Screening and data collection were performed by trained, bilingual (English and Spanish) research assistants following detailed manuals of procedures.

Anthropometric measures, blood pressure and fasting blood measures

Weight and standing height were measured with the participant in light clothing without shoes. BMI was calculated as weight in kilograms divided by the squared height in metres. Per cent overweight (per cent over median BMI for age and sex) was chosen as the primary measure of obesity status because of its preference over BMI z-score or percentiles at the upper extremes of BMI for age and sex (16). Waist circumference was measured with a non-elastic tape measure with children standing, arms at their sides and feet together, at the level of the umbilicus at end expiration. Resting systolic and diastolic blood pressure were measured using an automated blood pressure monitor (Dinamap Pro 100, GE Medical Systems, Wauwatosa, WI, USA) with an appropriately sized and positioned cuff around the right arm supported at heart level. Blood samples were obtained from children by venipuncture after a minimum 8-h fast. All venipuncture, sample handling and assays were performed by the Stanford Hospital and Clinics clinical laboratories. Detailed methodology for anthropometric, blood pressure and fasting blood measures is described elsewhere (17).

Actigraphy sleep measurements

The ActiGraph GT1M monitor, a small single-axis accelerometer (ActiGraph LLC, Pensacola, FL, USA), was used

to assess nightly sleep duration, bedtime and wake time. Children were instructed to wear the accelerometer at their right hip on an adjustable elastic belt around their waist for 24 h per day, except during bathing/showering and water activities. Hip-worn accelerometers have been well validated to measure sleep behaviour in previous studies (18,19). Research assistants provided demonstrations and verbal and written instructions for care and placement of the monitor and belt. Participants were required to have a minimum of at least 3 d of valid data including one weekend day. Accelerometers were initialized to collect data in 1-min epochs and downloaded using ACTILIFE software. Data were processed using a validated algorithm (18,20) to objectively estimate bedtime and wake time. In brief, the ACTILIFE software assigns a binary sleep or wake indicator variable to each epoch of data using the Sadeh method (20). Bedtime was identified as the first 5 consecutive minutes defined as sleep, and wake time was identified as the first 10 consecutive minutes defined as wake after a period of sleep. A detailed description of this algorithm is available elsewhere (18). Data were checked for spurious recording (i.e. high counts more than 20,000 counts per minute). Because time spent in sleep can be confused with non-wear time, presumptive sleep periods longer than 13 consecutive hours and sleep periods that extended beyond 1:30 PM were deemed as non-wear. Results of the algorithm were verified with visual inspections of a random 20% sample of activity count graphs. Similar to previous actigraphy-based sleep studies, weekday nights were classified as starting on Sundays through Thursdays and weekend nights starting on Fridays and Saturdays. Estimates of sleep duration, bedtime and wake time for weekday and weekend nights were derived by averaging those values over all available nights for each individual. Estimates of day-to-day intra-individual variability in sleep duration, bedtime and wake time on weekday and weekend nights were calculated as the standard deviation in these parameters over all available nights for each individual. Sample sleep parameters were similar to those previously reported in the literature (21,22).

Parent-reported sleep measurements

Parents responded to the following questions: (i) 'In a typical week, what time does your child go to sleep on a school night [non-school night]?' and (ii) 'In a typical week, what time does your child wake up in the morning on a school day [non-school day]?' Sleep durations for school days and non-school days were calculated as the time differences between the two questions.

Cardiometabolic risk factor clustering

To create a measure of clustering of cardiometabolic risk factors, a cumulative cardiometabolic risk clustering score was calculated by assigning 1 point each for six risk factors. A continuous risk factor clustering score including multiple risk factor variables is considered superior to dichotomous/categorical approaches (23,24) and associated with atherosclerosis in children and young adults (25). The risk factor clustering measure included the following six risk factors: (i) BMI \geq 97th percentile for age and sex (because all participants were already at or above the 95th percentile), (ii) systolic and/or diastolic blood pressure \geq 90th percentile for age, sex and height percentile (26), (iii) high-density lipoprotein cholesterol < 40 mg dL⁻¹, low-density lipoprotein cholesterol ≥ 130 mg dL⁻¹ and/or total cholesterol ≥ 200 mg dL⁻¹, (iv) serum triglycerides ≥ 100 mg dL⁻¹ if under 10 years old or ≥ 130 mg dL⁻¹ if age 10 or older (27), (v) C-reactive protein in the top quartile of this sample distribution (≥ 3.9 mg L⁻¹) and (vi) at least one marker of insulin resistance: insulin in the top quartile of the sample (≥ 25 g dL⁻¹), glucose ≥ 100 mg dL⁻¹, haemoglobin A1c $\geq 5.7\%$ and/or Homeostatic Model Assessment of Insulin Resistance in the top quartile of this sample (≥ 5.8).

Dietary intake

Three randomly timed 24-h dietary recalls were collected by trained registered dietitians, using the University of Minnesota Nutrition Data System for Research. The first recall was collected in person, and the second and third recalls were collected over the telephone. To aid in assessment of portion sizes, data collectors provided children with previously validated posters containing two-dimensional visual representations of food portions (2D Food Portion, Visual, Nutrition Consulting Enterprises, Framingham, MA, USA) (28). The 24-h recall method, including phone interviews, has been previously validated in children (29–31).

Physical activity

Physical activity levels were estimated from the same accelerometer data used to estimate sleep behaviours. Beginning and end of day time points were chosen conservatively to exclude potential sleep times based on the observed sample distribution of sleep times (between 9:00 AM and 8:00 PM on weekdays and 11:00 AM and 9:00 PM on weekend days). Periods of missing data were identified with the Choi *et al.* non-wear algorithm (32), and missing data from other observations were imputed from the same children and times of day using the previously

validated Alhassan and Robinson method (26). The Puyau *et al.* activity cut-points were used to estimate average daily per cent of time spent in moderate-to-vigorous physical activity (33).

Television screen time

Children reported their typical weekend and weekday time spent in the morning and in the afternoon watching television, watching movies or videos on a VCR or DVD on a television and playing video games on a television, like Wii, PlayStation or Xbox. For each media type and half day, responses were reported in intervals of 'none', '15 min or less', '30 min', '1 h', '2 h', '3 h', '4 h', '5 h' or '6 h or more'.

Sociodemographics

Parents/guardians reported their children's age, gender, race/ethnicity, the highest level of parent education completed and annual total household income.

Statistical analyses

Linear regression and non-parametric Spearman rank correlations were used to examine bivariate relationships between sleep parameters and adiposity and other risk parameters. Multivariate linear regression analyses were used to examine relationships between sleep variables and measures of adiposity and cardiometabolic risk parameters while adjusting for age, sex, race/ethnicity and obesity-related behaviours (television screen time, dietary intake and physical activity). All statistical analyses were performed using the SAS Statistical Package 6.1 (SAS Institute Inc, Cary, NC, USA). Statistical significance was defined as $P < 0.05$.

Results

Baseline demographics

Participant characteristics are presented in Table 1. Of 188 children in the sample, 50% (94) were Latino/Hispanic, and 51% (97) lived in a household with an annual total household income less than \$50,000, demonstrating the sociodemographic diversity of the sample. Accelerometer data were available from all 188 participants. The mean \pm standard deviation accelerometer-measured sleep duration was 9.13 ± 0.68 h, which is in line with other studies that objectively measured sleep duration (8,34,35).

Table 1 Participant characteristics

Female, <i>n</i> (%)	108 (57)
Male, <i>n</i> (%)	81 (43)
Age in years (range 8, 12.9), mean (SD)	10.50 \pm 1.39
Race/ethnicity, <i>n</i> (%)	
White	51 (27)
Latino/Hispanic	94 (50)
Asian/Pacific Islander	18 (10)
African-American	5 (2)
Other/mixed	20 (11)
Total household income, <i>n</i> (%)	
\$14,999 or less	33 (17)
\$15,000–24,999	30 (16)
\$25,000–49,999	34 (18)
\$50,000–199,999	33 (18)
Don't know or I prefer not to answer	58 (31)
Parent maximum level of education, <i>n</i> (%)	
Less than high school	34 (18)
High school graduate or some college	15 (8)
Technical or associate's degree	78 (41)
Bachelor's degree [†]	61 (32)
Anthropometric measures [‡] , mean (SD)	
Per cent over 50th percentile (per cent overweight)	71.34 (27.21)
Per cent over 95th percentile	26.55 (20.05)
BMI (kg m^{-2})	29.24 (5.04)
BMI z-score	2.24 (0.31)
BMI percentile	98.38 (1.21)
Waist circumference (cm)	95.63 (12.24)
Triceps skin-fold thickness (mm)	30.26 (4.06)
Total nights of accelerometer data per participant (range 3, 15), mean (SD)	7.0 (1.4)
Weekday only [§]	4.92 (0.99)
Weekend only	2.08 (0.61)
Accelerometer-measured sleep duration, mean (SD)	
Overall (h)	9.13 (0.68)
Weekday night (h)	9.07 (0.76)
Weekend night	9.26 (1.00)
Accelerometer-measured sleep timing, mean (SD)	
Weekday bedtime (h:min)	10:09 PM (0:51)
Weekday wake time (h:min)	7:13 AM (0:49)
Weekend bedtime (h:min)	10:55 PM (1:05)
Weekend wake time (h:min)	8:10 AM (1:12)
Accelerometer-measured sleep variability, mean (SD)	
Weekday sleep duration (h)	0.88 (0.55)
Weekday bedtime (h)	0.69 (0.40)
Weekday wake time (h)	0.63 (0.47)
Weekend sleep duration (h)	1.09 (0.84)
Weekend bedtime (h)	0.79 (0.72)
Weekend wake time (h)	0.80 (0.67)
Parent-reported sleep duration, mean (SD)	
School [¶] night (h)	9.56 (0.73)
Non-school night (h)	9.92 (1.06)

Continues

Parent-reported sleep timing,
mean (SD)

School night bedtime (h:min)	9:19 PM (0:43)
School night wake time (h:min)	6:52 AM (0:27)
Non-school night bedtime (h:min)	10:20 PM (0:53)
Non-school night wake time (h:min)	8:16 AM (1:12)

[†]None of the parents had a master's, professional, or doctoral degree.

[‡]Distribution of age-specific and sex-specific body weight based on Centers for Disease Control and Prevention values. One individual, not included in $N = 188$, was missing anthropometric measurements.

[§]Weekday = Sunday night through Thursday night; weekend = Friday night and Saturday night; parent-reported values are reported in the table above as 'school' and 'non-school night' to reflect the way the question was asked in the survey.

^{||}'School' and 'non-school' night correspond to weekday and weekend night, respectively. Similarly, bedtime and wake time correspond to accelerometer-measured sleep onset and sleep offset.

BMI, body mass index; SD, standard deviation.

Comparison of parent reports of children's sleep with accelerometer-measured sleep

Parent reports and accelerometer measures of sleep were moderately correlated but statistically significantly different (Table 2). Parent reports underestimated accelerometer-measured sleep duration by an

Table 2 Correlation and difference between accelerometer and parent-reported measures

		Correlation coefficient (<i>r</i>)	Difference in hours (accelerometer – parent), Mean (SE)
Sleep duration	Weekday	0.43***	-0.48 (0.061)***
	Weekend	0.19*	-0.66 (0.095)***
Sleep timing	Weekday bedtime	0.59***	0.56 (0.069)***
	Weekday wake time	0.54***	-0.091 (0.084)
	Weekend bedtime	0.56***	0.85 (0.056)***
	Weekend wake time	0.47***	0.36 (0.056)***

Two individuals do not have accelerometer-measured weekend sleep values. Student's paired *t*-test was used to test differences in hours between mean accelerometer and parent-reported typical sleep values (accelerometer estimate minus parent estimate) for a given individual. A negative difference in sleep duration indicates that the accelerometer estimate is shorter than the parent estimate. A positive difference in sleep timing indicates that the accelerometer estimate is later than the parent estimate. Mean (SE) of the difference in hours are shown.

* $P < 0.05$.

** $P < 0.01$.

*** $P < 0.001$.

SE, standard error.

average \pm standard error of about 29.04 ± 3.67 (weekday) to 39.30 ± 5.71 min (weekend), underestimated how late children went to sleep by about 50.74 ± 3.34 (weekday) to 33.85 ± 4.12 min (weekend) and underestimated how late children woke up by about 21.70 ± 3.36 min (week-day). Parent-reported wake time on the weekend was not statistically significantly different from accelerometer measures.

Associations of sleep duration and measures of adiposity

In multivariate models adjusted for age, sex and race/ethnicity, parent-reported sleep duration on school nights was significantly associated with per cent overweight, per cent over the 95th percentile and BMI (Table 3). Based on parent reports, every hour of less sleep on school nights was associated with an average additional 5.78 per cent overweight. However, parent-reported sleep duration on non-school nights and all accelerometer-measured sleep durations were not significantly associated with any of the measures of adiposity.

Associations of sleep timing and measures of adiposity

In multivariate models adjusted for age, sex and race/ethnicity, accelerometer-measured weekday bedtime was significantly associated with all four measures of adiposity (Table 3). Every hour later in accelerometer-measured bedtime on weekdays was associated with an average greater 6.17 per cent overweight, 4.60 per cent over the 95th percentile, 1.03 BMI units and 2.06-cm waist circumference. Similar to accelerometer-measured weekday bedtime, an hour later in parent-reported weekday bedtime was associated with an average additional 4.33 per cent over the 95th percentile and 1.01 BMI units. Accelerometer-measured weekday wake time was also significantly associated with BMI and waist circumference.

Associations of sleep variability and measures of adiposity

In multivariate models adjusted for age, sex and race/ethnicity, day-to-day variability in accelerometer-measured weekday sleep duration was significantly associated with all measures of adiposity (Table 3). Every hour greater in day-to-day variability in weekday sleep duration was associated with an average additional 8.62 per cent overweight, 6.31 per cent over the 95th percentile, 1.57 BMI units and 3.16-cm waist circumference.

Table 3 Standardized regression coefficients from multivariate models of sleep dimensions with anthropometric outcomes

	Per cent overweight	Per cent over 95th percentile	Body mass index	Waist circumference	Cardiometabolic risk score
	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
Accelerometer-measured sleep duration					
Weekday (h)	-1.84 (-6.97 to 3.29)	-1.49 (-5.30 to 2.32)	-0.28 (-1.15 to 0.60)	-0.20 (-2.17 to 1.76)	0.15 (-0.10 to 0.40)
Weekend (h)	0.15 (-3.92 to 4.21)	0.17 (-2.85 to 3.20)	0.0034 (-0.69 to 0.70)	-0.083 (-1.64 to 1.47)	0.11 (-0.081 to 0.31)
Accelerometer-measured sleep timing					
Weekday bedtime (h)	6.17 (1.42 to 10.92)*	4.60 (1.07 to 8.13)*	1.03 (0.22 to 1.85)*	2.06 (0.23 to 3.88)*	0.030 (-0.21 to 0.27)
Weekday wake time (h)	4.90 (-0.077 to 9.87)	3.54 (-0.16 to 7.24)	0.85 (0.00061 to 1.70)*	2.03 (0.13 to 3.93)*	0.17 (-0.070 to 0.42)
Weekend bedtime (h)	2.19 (-1.63 to 6.01)	1.62 (-1.22 to 4.46)	0.37 (-0.29 to 1.03)	0.65 (-0.81 to 2.11)	0.032 (-0.15 to 0.22)
Weekend wake time (h)	1.87 (-1.56 to 5.29)	1.43 (-1.11 to 3.97)	0.30 (-0.29 to 0.89)	0.46 (-0.85 to 1.78)	0.11 (-0.058 to 0.27)
Accelerometer-measured sleep variability					
Weekday duration (h)	8.62 (1.34 to 15.89)*	6.31 (0.90 to 11.72)*	1.57 (0.33 to 2.82)*	3.16 (0.37 to 5.95)*	0.12 (-0.24 to 0.48)
Weekday bedtime (h)	10.20 (0.50 to 19.91)*	7.72 (0.51 to 14.93)*	1.78 (0.12 to 3.44)*	3.09 (-0.64 to 6.82)	0.090 (-0.39 to 0.57)
Weekday wake time (h)	10.02 (1.55 to 18.50)*	7.28 (0.98 to 13.58)*	1.85 (0.40 to 3.29)*	2.98 (-0.29 to 6.24)	0.20 (-0.22 to 0.62)
Weekend duration (h)	4.11 (-0.73 to 8.95)	3.11 (-0.47 to 6.69)	0.72 (-0.12 to 1.55)	1.26 (-0.63 to 3.16)	0.025 (-0.23 to 0.28)
Weekend bedtime (h)	4.33 (-1.49 to 10.14)	3.30 (-0.99 to 7.60)	0.72 (-0.29 to 1.72)	1.38 (-0.88 to 3.65)	0.18 (-0.12 to 0.48)
Weekend wake time (h)	0.098 (-6.00 to 6.02)	0.13 (-4.31 to 4.57)	0.020 (-1.02 to 1.06)	0.98 (-1.36 to 3.31)	-0.042 (-0.35 to 0.27)
Parent-reported sleep duration					
School (h)	-5.78 (-11.42 to -0.15)*	-4.43 (-8.61 to -0.24)*	-1.05 (-2.01 to -0.086)*	-1.68 (-3.84 to 0.49)	0.20 (-0.079 to 0.47)
Non-school (h)	-2.46 (-6.29 to 1.36)	-1.85 (-4.70 to 0.99)	-0.41 (-1.06 to 0.25)	-0.58 (-2.04 to 0.89)	0.15 (-0.038 to 0.33)
Parent reported sleep timing					
School bedtime (h)	5.69 (-0.089 to 11.47)	4.33 (0.038 to 8.63)*	1.01 (0.021 to 2.00)*	1.64 (-0.59 to 3.86)	-0.17 (-0.45 to 0.12)
School wake time (h)	-0.90 (-9.74 to 7.95)	-0.74 (-7.31 to 5.83)	-0.21 (-1.73 to 1.30)	-0.30 (-3.68 to 3.09)	0.097 (-0.33 to 0.53)
Non-school bedtime (h)	4.22 (-0.36 to 8.81)	3.15 (-0.25 to 6.56)	0.73 (-0.058 to 1.51)	1.01 (-0.75 to 2.78)	-0.13 (-0.36 to 0.092)
Non-school wake time (h)	0.39 (-3.19 to 3.97)	0.28 (-2.38 to 2.94)	0.080 (-0.53 to 0.69)	0.11 (-1.26 to 1.48)	0.049 (-0.13 to 0.22)

Per cent overweight indicates child's BMI relative to the 50th percentile for age and sex as a percentage ($100 \times [\text{participant BMI} - \text{median BMI for age and sex}] \div \text{median BMI for age and sex}$). Similarly, per cent over 95th percentile indicates child's BMI relative to the 95th percentile for age and sex as a percentage ($100 \times [\text{participant BMI} - 95\text{th percentile BMI for age and sex}] \div 95\text{th percentile BMI for age and sex}$). Results are adjusted for age, sex and race/ethnicity. Results remained consistent while adjusting for sleep duration (data not shown). Sleep analyses were done in hours. For instance, every hour later in accelerometer-measured bedtime on weekdays was associated with an average 6.2 greater per cent overweight (e.g. the difference between 77.5 per cent overweight and 71.3 per cent overweight). Every hour greater in day-to-day variability in time of weekday bedtime was associated with an average 10.2 greater per cent overweight.

* $P < 0.05$.** $P < 0.01$.*** $P < 0.001$. β , standardized regression coefficient; CI, confidence interval.

Day-to-day variability of accelerometer-measured weekday bedtime and wake time was significantly associated with all BMI-related measures of adiposity. Every hour greater in day-to-day variability in weekday bedtime and weekday wake time was associated with an average additional 10.20 and 10.02 per cent overweight, 7.72 and 7.28 per cent over the 95th percentile and 1.78 and 1.85 BMI units, respectively.

Associations of sleep measures and cardiometabolic risk factor clustering

None of the individual parent or accelerometer-measured sleep parameters were statistically significantly associated with the cardiometabolic risk factor clustering score. Additional exploratory analysis of associations of all sleep measures with each individual cardiometabolic risk factor measure included in the clustering measure, treated as continuous variables, produced just a small number of significant relationships with inconsistent patterns (data not shown).

Sleep measures and physical activity, television screen time and caloric intake

Accelerometer-measured weekday bedtime remained significantly associated with per cent overweight after adjusting for obesity-related behaviours (moderate-to-vigorous physical activity, television screen time and total dietary energy intake). Accelerometer-measured weekday wake time and variability in weekday bedtime and wake time were no longer significant after adjusting for obesity-related behaviours. Later accelerometer-measured weekday bedtime was significantly associated with greater total weekly television screen time by an average \pm standard error of 3.19 ± 1.42 h. Sleep timing was not significantly associated with total caloric intake or with moderate-to-vigorous physical activity.

Discussion

The present study extends past research on sleep and adiposity to a racially/ethnically and socioeconomically diverse sample of children with obesity, using both parent report and objective accelerometer measures of sleep duration and timing. This study found that parent reports and accelerometer-derived estimates of sleep duration and timing are correlated but not equivalent. Parents reported significantly earlier typical weekday and weekend bedtime, earlier weekday wake times and longer sleep durations than those measured objectively with accelerometers. Previous studies have also demonstrated that parent reports overestimate children's total sleep duration

and how early children fall asleep and wake up (36). These results provide further evidence that accelerometer-measured and parental reports of sleep parameters are different and not interchangeable.

Accelerometer-measured weekday sleep timing and variability in weekday sleep duration and sleep timing were most consistently associated with anthropometric measures of adiposity. Sleep timing and regularity may be critical for making recommendations for children with obesity. Children who went to sleep later on weekdays were also more likely to go to sleep later on the weekend and wake up later on weekdays and on the weekend (Table 4). These results are consistent with recent evidence that obesity status is heavily influenced by sleep timing (7,37,38) and variability in sleep schedules (35). Other possibilities for the association between delayed sleep timing and irregular sleep patterns with higher adiposity measures are that children voluntarily adjust their sleep-wake cycles to accommodate personal activity interests and physical activity and eating behaviours or that individuals have an inherent psychological or genetic disposition regarding their sleep behaviours. Previous studies have shown that delays in melatonin onset and offset can alter eating behaviour (39). External factors may also predispose children to a particular sleep/wake pattern, such as parents' work schedules, siblings' sleep/wake patterns, school start times and household rules around bedtime. In the present study, weekday sleep measures were more consistently associated with measures of adiposity than weekend measures. The fewer number of weekend measurements relative to weekday measurements may have underpowered the study in estimating the true weekend sleep parameters.

Sleep duration, timing and variability were not found to be significantly associated with cardiometabolic risk factor clustering in this sample. Clustering of risk factors was expected to increase statistical power to detect associations and has been validated in previous studies (40). Because this sample consisted of all children with obesity who are already at elevated risk, this may have limited power to detect associations, to some extent, due to less variability in cardiometabolic risk across the sample.

These results suggest that bedtime influences adiposity indices independently from dietary energy intake, amount of television screen time and amount of moderate-to-vigorous physical activity. Thus, delayed bedtime may be an independent modifiable risk factor for obesity. Unsurprisingly, later bedtime was associated with greater television screen time. Bedtime was not significantly associated with total energy intake or moderate-to-vigorous physical activity (MVPA), which was likely because dietary intake and physical activity are influenced

Table 4 Within-child correlations between sleep measures

	Accelerometer-measured sleep duration		Accelerometer-measured sleep timing				Parent-reported sleep timing			
	Weekday	Weekend	Weekday bedtime	Weekday wake time	Weekend bedtime	Weekend wake time	Weekday bedtime	Weekday wake time	Weekend bedtime	Weekend wake time
Accelerometer-measured sleep duration	Weekday	0.27***	Weekday	-0.54***	Weekday	-0.22**	Weekday	-0.31***	Weekday	-0.27***
	Weekend		Weekend	-0.08	Weekend	-0.30***	Weekend	0.06	Weekend	-0.04
Accelerometer-measured sleep timing	Weekday bedtime		Weekday wake time	0.21**	Weekday bedtime	0.48***	Weekday wake time	0.14	Weekday bedtime	0.14
	Weekend bedtime		Weekend wake time	0.52***	Weekend bedtime	0.49***	Weekend wake time	0.25***	Weekend bedtime	0.63***
	Weekend bedtime		Weekend bedtime	0.44***	Weekend wake time	0.55***	Weekend bedtime	0.32***	Weekend wake time	0.41***
	Weekend wake time		Weekend wake time	0.64***	Weekend wake time	0.64***	Weekend wake time	0.37***	Weekend wake time	0.59***
Parent-reported sleep timing	Weekday bedtime		Weekday bedtime	0.34***	Weekday bedtime	0.48***	Weekday bedtime	0.38***	Weekday bedtime	0.48***
	Weekday wake time		Weekday wake time	0.26***	Weekday wake time	0.67***	Weekday wake time	0.26***	Weekday wake time	0.67***
	Weekend bedtime		Weekend bedtime		Weekend bedtime	0.26***	Weekend bedtime		Weekend bedtime	0.26***
	Weekend wake time		Weekend wake time		Weekend wake time	0.51***	Weekend wake time		Weekend wake time	0.51***

Two individuals do not have accelerometer-measured weekend sleep values. Statistically significant relationships are shaded for ease in viewing. Correlation coefficients (*r*-values) are shown.

**P* < 0.05.

***P* < 0.01.

****P* < 0.001.

by other factors besides sleep timing that were not included in the model, including access to snacks, portion size during mealtimes and individual choice of extracurricular activities.

The primary limitation of this study is the cross-sectional design that is unable to determine whether the associations identified are temporally or causally related. While experimental animal and lab studies suggest that adiposity is impacted by sleep behaviours (41), the reverse could also be true, or both might occur together. An important strength of this study is the use of accelerometers, an objective measure of sleep, in addition to parent reports. While accelerometers are not a direct measure of sleep, the gold standard measure of polysomnography is impractical for use in a large, multi-day study of children sleeping in their natural environments outside of a sleep laboratory. The sample of children with obesity is a strength because this is a high-risk group that was previously understudied with regard to sleep and adiposity relationships. It is also a potential disadvantage, however, if it limited power due to less variability in weight status, cardiometabolic risk, dietary energy, screen time and physical activity. Moreover, this sample consisted of children and families who enrolled in a clinical research study and who may not be representative of the general population of children with obesity who do not all seek care. Overall, despite these limitations, the many strengths of the methods and consistency of the findings, internally and with other past relevant research, give confidence in these results that later bedtime and wake time and irregular sleep patterns are associated with higher adiposity indices in children with obesity.

Conclusions

Among this racially/ethnically and socioeconomically diverse cohort of children with obesity, later bedtime, later wake times and more variable day-to-day sleep duration and timing were associated with greater measures of adiposity. The association between late bedtime and greater adiposity was independent of other obesity-related behaviours (dietary intake, physical activity and television screen time.) Future prospective and experimental intervention research are recommended to investigate the impact of an early bedtime on measures of adiposity in children with obesity and the potential mediators between sleep and adiposity, eating, screen time and physical activity behaviours.

Conflict of Interest Statement

The authors have no conflicts of interest to report.

Acknowledgements

We thank the study participants and their families for making this study possible.

Funding

This study was supported in part by the Stanford University School of Medicine Medical Scholars Research Program. This study was also supported in part by the Stanford Child Health Research Institute and the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (NIH), award number R01HL096015. The funders had no role in the design of the study, data collection and analysis, decision to publish or preparation of the manuscript.

References

1. Dietz WH, Robinson TN. Clinical practice. Overweight children and adolescents. *N Engl J Med* 2005; **352**: 2100–2109.
2. Firouzi S, Poh BK, Ismail MN, Sadeghilar A. Sleep habits, food intake, and physical activity levels in normal and overweight and obese Malaysian children. *Obes Res Clin Pract* 2014; **8**: e70–e78.
3. Westerlund L, Ray C, Roos E. Associations between sleeping habits and food consumption patterns among 10–11-year-old children in Finland. *Br J Nutr* 2009; **102**: 1531–1537.
4. Kjeldsen JS, Hjorth MF, Andersen R, et al. Short sleep duration and large variability in sleep duration are independently associated with dietary risk factors for obesity in Danish school children. *Int J Obes (Lond)* 2014; **38**: 32–39.
5. Miller AL, Lumeng JC, LeBourgeois MK. Sleep patterns and obesity in childhood. *Curr Opin Endocrinol Diabetes Obes* 2015; **22**: 41–47.
6. Golley RK, Maher CA, Matricciani L, Olds TS. Sleep duration or bedtime? Exploring the association between sleep timing behaviour, diet and BMI in children and adolescents. *Int J Obes (Lond)* 2013; **37**: 546–551.
7. Olds TS, Maher CA, Matricciani L. Sleep duration or bedtime? Exploring the relationship between sleep habits and weight status and activity patterns. *Sleep* 2011; **34**: 1299–1307.
8. McNeil J, Tremblay MS, Leduc G, et al. Objectively-measured sleep and its association with adiposity and physical activity in a sample of Canadian children. *J Sleep Res* 2015; **24**: 131–139.
9. Miller MA, Kruisbrink M, Wallace J, Ji C, Cappuccio FP. Sleep duration and incidence of obesity in infants, children, and adolescents: a systematic review and meta-analysis of prospective studies. *Sleep* 2018; **41**.
10. Hart CN, Carskadon MA, Considine RV, et al. Changes in children's sleep duration on food intake, weight, and leptin. *Pediatrics* 2013; **132**: e1473–e1480.
11. Robinson TN. Television viewing and childhood obesity. *Pediatr Clin North Am* 2001; **48**: 1017–1025.
12. Robinson TN, Matheson DM, Kraemer HC, et al. A randomized controlled trial of culturally tailored dance and reducing screen time to prevent weight gain in low-income African American girls: Stanford GEMS. *Arch Pediatr Adolesc Med* 2010; **164**: 995–1004.

13. Caroli M, Argentieri L, Cardone M, Masi A. Role of television in childhood obesity prevention. *Int J Obes Relat Metab Disord* 2004; **28**: S104–S108.
14. Ghobadi S, Hassanzadeh-Rostami Z, Salehi-Marzjarani M, et al. Association of eating while television viewing and overweight/obesity among children and adolescents: a systematic review and meta-analysis of observational studies. *Obes Rev* 2018; **19**: 313–320.
15. Kuczmarski RJOC, Grummer-Strawn LM, Flegal KM, et al. *CDC Growth Charts: United States. Advance Data from Vital and Health Statistics; no 314*. National Center for Health Statistics: Hyattsville, Maryland, 2000.
16. Dietz WH. Time to adopt new measures of severe obesity in children and adolescents. *Pediatrics* 2017; **140**: e20172148.
17. Robinson TN, Matheson D, Desai M, et al. Family, community and clinic collaboration to treat overweight and obese children: Stanford GOALS—a randomized controlled trial of a three-year, multi-component, multi-level, multi-setting intervention. *Contemp Clin Trials* 2013; **36**: 421–435.
18. Tudor-Locke C, Barreira TV, Schuna JM Jr, Mire EF, Katzmarzyk PT. Fully automated waist-worn accelerometer algorithm for detecting children's sleep-period time separate from 24-h physical activity or sedentary behaviors. *Appl Physiol Nutr Metab* 2014; **39**: 53–57.
19. Barreira TV, Schuna JM Jr, Mire EF, et al. Identifying children's nocturnal sleep using 24-h waist accelerometry. *Med Sci Sports Exerc* 2015; **47**: 937–943.
20. Sadeh A, Sharkey KM, Carskadon MA. Activity-based sleep-wake identification: an empirical test of methodological issues. *Sleep* 1994; **17**: 201–207.
21. Martinez SM, Greenspan LC, Butte NF, et al. Mother-reported sleep, accelerometer-estimated sleep and weight status in Mexican American children: sleep duration is associated with increased adiposity and risk for overweight/obese status. *J Sleep Res* 2014; **23**: 326–334.
22. Wong WW, Ortiz CL, Lathan D, et al. Sleep duration of underserved minority children in a cross-sectional study. *BMC Public Health* 2013; **13**: 648.
23. Eisenmann JC. On the use of a continuous metabolic syndrome score in pediatric research. *Cardiovasc Diabetol* 2008; **7**: 17.
24. Raitakari OT, Porkka KV, Rasanen L, Ronnema T, Viikari JS. Clustering and six year cluster-tracking of serum total cholesterol, HDL-cholesterol and diastolic blood pressure in children and young adults. The Cardiovascular Risk in Young Finns Study. *J Clin Epidemiol* 1994; **47**: 1085–1093.
25. Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med* 1998; **338**: 1650–1656.
26. Alhassan S, Robinson TN. Defining accelerometer thresholds for physical activity in girls using ROC analysis. *J Phys Act Health* 2010; **7**: 45–53.
27. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics* 2011; **128**: S213–S256.
28. Posner BM, Smigelski C, Duggal A, Morgan JL, Cobb J, Cupples LA. Validation of two-dimensional models for estimation of portion size in nutrition research. *J Am Diet Assoc* 1992; **92**: 738–741.
29. Van Horn LV, Gernhofer N, Moag-Stahlberg A, et al. Dietary assessment in children using electronic methods: telephones and tape recorders. *J Am Diet Assoc* 1990; **90**: 412–416.
30. van Horn LV, Stumbo P, Moag-Stahlberg A, et al. The Dietary Intervention Study in Children (DISC): dietary assessment methods for 8- to 10-year-olds. *J Am Diet Assoc* 1993; **93**: 1396–1403.
31. Lytle LA, Nichaman MZ, Obarzanek E, et al. Validation of 24-hour recalls assisted by food records in third-grade children. The CATCH Collaborative Group. *J Am Diet Assoc* 1993; **93**: 1431–1436.
32. Choi L, Liu Z, Matthews CE, Buchowski MS. Validation of accelerometer wear and nonwear time classification algorithm. *Med Sci Sports Exerc* 2011; **43**: 357–364.
33. Puyau MR, Adolph AL, Vohra FA, Butte NF. Validation and calibration of physical activity monitors in children. *Obes Res* 2002; **10**: 150–157.
34. Harrington SA. Relationships of objectively measured physical activity and sleep with BMI and academic outcomes in 8-year-old children. *Appl Nurs Res* 2013; **26**: 63–70.
35. Spruyt K, Molfese DL, Gozal D. Sleep duration, sleep regularity, body weight, and metabolic homeostasis in school-aged children. *Pediatrics* 2011; **127**: e345–e352.
36. Iwasaki M, Iwata S, Iemura A, et al. Utility of subjective sleep assessment tools for healthy preschool children: a comparative study between sleep logs, questionnaires, and actigraphy. *J Epidemiol* 2010; **20**: 143–149.
37. Carskadon MA, Acebo C, Jenni OG. Regulation of adolescent sleep: implications for behavior. *Ann N Y Acad Sci* 2004; **1021**: 276–291.
38. Garaulet M, Ordovas JM, Madrid JA. The chronobiology, etiology and pathophysiology of obesity. *Int J Obes* 2010; **34**: 1667–1683.
39. Arble DM, Bass J, Behn CD, et al. Impact of sleep and circadian disruption on energy balance and diabetes: a summary of workshop discussions. *Sleep* 2015; **38**: 1849–1860.
40. Berenson GS, Srinivasan SR, Xu JH, Chen W. Adiposity and cardiovascular risk factor variables in childhood are associated with premature death from coronary heart disease in adults: The Bogalusa Heart Study. *Am J Med Sci* 2016; **352**: 448–454.
41. Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. *Obesity (Silver Spring)* 2008; **16**: 643–653.