Sleep and Adiposity in Children From 2 to 6 Years of Age

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abstract

OBJECTIVES: To compare sleep in young children at different obesity risks, which were based on parental weight, as well as to explore the longitudinal associations of sleep characteristics with adiposity.

METHODS: In total, 107 children from an obesity prevention project were included, of which 43 had normal-weight parents (low obesity risk) and 64 had overweight and/or obese parents (high obesity risk). Sleep was measured yearly from ages 2 to 6 years by using actigraphy. Five sleep characteristics, that of late sleep, long sleep latency, short sleep duration, low sleep efficiency, and irregular sleep onset, were defined and scored across ages, with a higher score indicating more frequent exposure. The outcome variables, also measured yearly, were BMI *z* score and waist circumference.

RESULTS: There was no difference in sleep patterns among children at different risks. Higher short sleep duration score was associated with a greater increase in BMI *z* score (0.12; 95% confidence interval [CI] 0.01 to 0.25) across ages. Independently of sleep duration, higher late sleep score was associated with greater increases in BMI *z* score (0.16; 95% CI 0.05 to 0.27) and waist circumference (0.60 cm; 95% CI 0.23 to 0.98). Moreover, compared with children at low risk and without habitual late sleep, children at high risk and with habitual late sleep had greater increases in BMI *z* score (0.93; 95% CI 0.40 to 1.45) and waist circumference (3.45 cm; 95% CI 1.78 to 5.12).

CONCLUSIONS: More frequent exposures to late sleep were associated with greater increases in adiposity measures from ages 2 to 6 years, particularly in children with obese parents.

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WHAT'S KNOWN ON THIS SUBJECT: Current evidence, relying heavily on parental reported data, suggests that short sleep increases childhood obesity risk. Limited evidence, using objective sleep measures, explores the associations of other sleep dimensions, such as sleep schedule and quality, with adiposity in young children.

WHAT THIS STUDY ADDS: Beyond sleep duration, more frequent late sleep is also associated with greater increases in adiposity measures from ages 2 to 6 years. The association between late sleep and adiposity was especially evident for children at high family obesity risk.

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An association between short sleep duration and incidence of childhood obesity is indicated by several reviews.[1–3] However, current evidence relies heavily on subjective reports: only 5 of 42 longitudinal studies in the most recent review[2] used objective sleep measures. This might be problematic, considering the relatively low validity of parental sleep reports for young children.[4] Moreover, sleep is a multidimensional structure, including sleep schedules, quantity, and quality.[4] The extant research focusing primarily on sleep duration does not provide enough information on the role of sleep in obesity. Epidemiological studies in school-aged children and adolescents have shown that late sleep and large sleep variability were associated with unhealthy weight, independently of sleep duration.[5,6] Poor sleep quality (eg, subjectively reported sleep disturbances and difficulty initiating sleep) has been associated with obesity in several cross-sectional studies of children and adolescents.[7] However, objective indicators of sleep quality (eg, sleep latency and efficiency) are relatively rarely used, especially in longitudinal designs.

Parental obesity has been identified as a predominant risk factor of childhood obesity.[8] Children of overweight or obese parents have a 3 to 10 times higher risk of developing obesity than children of normal-weight parents.[9] The underlying mechanisms are unclear because established genetic, epigenetic, and psychosocial factors can only partly explain the association.[10–14] Whether poor sleep contributes to the transfer of obesity within families remains less explored. We have previously observed that prolonged sleep-onset latency and more variable weekday-weekend sleep schedules are slightly more common in 2-year-old children with obese parents than those with normal-weight parents.[15] However, determining if sleep development differs over time in children with or without obese parents requires more detailed studies.

Therefore, we explore sleep and the association with adiposity in children at different obesity risks, which were based on parental weight, using objective, repeated sleep measures. We aimed to compare the changes of sleep measures and characteristics in children at different obesity risks from ages 2 to 6 years. In addition, we examined longitudinal associations of different sleep characteristics with adiposity and whether the associations differed with family obesity risk.

METHODS

Study Population

This study is embedded in the Early Stockholm Obesity Prevention Project, a longitudinal, randomized controlled obesity prevention project described in detail in the study protocol.[16] Briefly, 238 families in the Stockholm area were randomly recruited to the project between 2010 and 2013 when parents visited a local child health care center for their child's 8-month checkup. Families with at least 1 parent with obesity (BMI ≥30) or 2 parents with overweight (BMI 25–29.9) were recruited to the high obesity risk group (*n* = 181). Families with 2 normal-weight parents (BMI 18–24.9) were recruited to the low obesity risk group (*n* = 57). High-risk families were allocated to either the intervention (*n* = 66) or control group (*n* = 115) through cluster randomization of child health care centers. Inclusion criteria were having a child aged <1 year, parents being able to communicate in Swedish, and the child being without chronic health problems likely to affect growth and development. Ethical approval was given by the Stockholm Regional Ethical Review Board (2009/217–31), and written informed consent was obtained from all families.

Yearly data collection was conducted from child ages 1 to 6 years, whereas sleep measures using accelerometers started at child age 2 years. In this study, only children from the nonintervention groups with at least 2 sets of sleep measures during the study period were included: in total, 107 children remained (64 high-risk controls; 43 low-risk controls). A flowchart of the study population is presented in Supplemental Fig 2.

Sleep Measures and Characteristics

Sleep Measures

From child age 2 years, sleep was measured yearly for 7 consecutive days by using a wrist-worn actigraphy (ActiGraph GT3X+; ActiGraph, LLC, Pensacola, FL), which is a triaxial accelerometer with sensitivity of 0.05 g and sampling rate of 30 to 100 Hz. Activity counts were collected at a 30-Hz sampling rate and integrated to 1-minute epochs by using the manufacturer's software. Each epoch was scored as either "awake" or "asleep" on the basis of the sleep algorithm developed by Sadeh et al,[17] which has been validated in children using polysomnography.[17,18] To reduce overestimation of nighttime awakenings, a secondary algorithm, developed by Sitnick et al[19] and validated with videosomnography and polysomnography,[20] was adopted. The smoothing algorithm required a minimum length of wake after sleep onset (WASO) of 2 minutes. When ≥2 consecutive minutes with activity counts ≥100 were immediately preceded by an activity count >0, that epoch was considered the start of an awakening. An awakening was scored as ending, signifying a return to sleep, at the first of 3 consecutive counts of 0.[19,20] Smoothing was automated by using an Excel formula (Microsoft, Redmond, WA). On actigraphy monitoring days, parents also completed a detailed sleep diary and an actigraphy log. Individual nights were not scored as usable if the actigraphy was off, the diary was not completed properly, or the actigraphy sleep data did not correspond to the sleep period recorded in the diary. At least 4 usable nights of sleep recordings were required for 1 valid sleep measure. In all, 387 valid sleep measures, with 2576 nights of sleep data, were included in the analysis.

Definitions of sleep variables are presented in Table 1. Average values were calculated across full weeks. Weekday-weekend sleep variations were calculated as weekend values minus weekday values, and day-to-day variations were calculated as the means of SDs across full weeks.

Sleep Characteristics

We further focused on 5 characteristics reflecting sleep schedule, quantity, and quality. We classified and scored 3 characteristics at each age using established recommendations with the following cutoff points[21,22]: (1) late sleep (score 1 for average sleep onset >21:00 and 0 for ≤21:00), (2) long sleep latency (score 1 for average sleep latency >45 minutes and 0 for ≤45 minutes), and (3) low sleep efficiency (score 1 for average sleep efficiency <85% and 0 for ≥85%). Because no threshold exists for either objectively measured sleep duration or sleep variation in the pediatric population, lower or upper quartiles of sleep measures in children at each age were used to classify 2 characteristics: (4) short sleep duration (at age 2 years, score 1 for average total sleep <10 hours/day and 0 for ≥10 hours/day; at age 3 years, <9.7 hours/day = 1 and ≥9.7 hours/day = 0; at age 4–5 years, <9.5 hours/day = 1 and ≥9.5 hours/day = 0; at age 6 years, <9.2 hours/day = 1 and ≥9.2 hours/day = 0); and (5) irregular sleep onset (score 1 for day-to-day sleep-onset variation >52 minutes and 0 for ≤52 minutes). Because the upper quartiles of sleep-onset variation at each age were similar, the average value across ages was used. For each characteristic, the range was 0 to 5: 0 indicated never having the characteristic and 5 indicated the maximum prevalence of the characteristic across ages. To analyze the interactive effect between sleep characteristics and family obesity risks, we defined categorical variables of "habitual characteristics" as a child having a certain characteristic (total score ≥3) or not (total score <3).

Anthropometric Measurements

At the family visits, each child's weight, height, and waist circumference were measured by trained research staff following a standard protocol. The mean values of 3 measurements were used in analyses. BMI was calculated on the basis of weight and height, and BMI *z* score was derived from Swedish age- and sex-specific reference values.

Covariates

Demographic variables on child date of birth, sex, parental dates of birth, ethnicity (Nordic or not), and education level were collected by using questionnaires at baseline. Family education level was defined as low if both parents had attended school for ≤12 years and as high if at least 1 parent had postsecondary education. Other background variables, that of having siblings (yes or no), living conditions (apartment or terraced house versus detached house), parental marital status (married or cohabiting versus separated), child screen time (≥2 hours/day or not), and physical activity level (high or low), were collected yearly through questionnaires. A child was classified as having a high physical activity level if he or she spent at least 2 hours/day outdoors playing and was perceived as having a high level of physical activity by the parents; otherwise, he or she was classified as having a low level. The date when sleep was assessed with actigraphy was categorized as summer (April to September) or winter (October to March).

Statistical Analyses

In the included sample (*N* = 107), there were 17 (16%) children at age 2 years, 44 (41%) at age 3 years, 34 (32%) at age 4 years, 33 (31%) at age 5 years, and 20 (19%) at age 6 years with missing sleep data. Only 19

TABLE 1 Definitions of Sleep Variables

Sleep Variable	Definition
Sleep onset, h:min	The clock time for the first of 5 consecutive min scored as asleep after the reported bedtime
Sleep offset, h:min	The clock time for the first of 10 consecutive min scored as awake before the reported rise time
Sleep-onset latency, min	The No. min between parental reported bedtime and sleep onset
Nocturnal sleep duration, h	No. min scored as sleep between nocturnal sleep onset and sleep offset/60 (ie, WASO excluded)
WASO, min	No. min scored as awake between nocturnal sleep onset and sleep offset
Nocturnal sleep period, h	No. min between sleep onset and sleep offset (nocturnal sleep duration + WASO)
Sleep efficiency, %	(Nocturnal sleep duration)/(sleep offset − bedtime) × 100
24-h total sleep duration, h	The total sleep hours during the entire day, including nocturnal sleep duration and daytime naps
Weekday-weekend variation in sleep onset, min	Sleep onset (weekend) − sleep onset (weekday)
Weekday-weekend variation in 24-h sleep duration, min	24-h sleep duration (weekend) − 24-h sleep duration (weekday)
Day-to-day variation in sleep onset, min	The mean of SD of sleep onset across wk
Day-to-day variation in sleep duration, min	The mean of SD of 24-h sleep duration across wk

(18%) children had complete sleep data at all 5 measuring points. The missing data were missing completely at random (MCAR) (Little's MCAR test, $P = .14$). Thus, multiple imputation using 100 imputed data sets was performed on the basis of a scan of the data set. Scoring of sleep characteristics was done after the imputations.

Independent *t* tests and χ^2 tests were conducted in group comparisons as appropriate. Linear mixed models were used in the longitudinal analysis; this is a powerful and flexible tool for analysis of repeated measures, with advantages of alternative covariance structures and accommodating the time-varying covariates and missing data.[23] The Schwarz-Bayesian criterion was used as a model fit criterion for choosing alternative covariance structures and models. The development of adiposity across ages among children at different obesity risks was compared, with adjustments for child age, sex, family education level, and baseline measures. The changes of sleep measures across ages were compared between groups, with adjustments for factors that have been reported as relating to child sleep, including sex, age, having siblings or not, maternal ethnicity, parental marital status, family education level, living condition, and measuring season. The adjusted associations of sleep characteristics with family obesity risk were analyzed by using generalized linear models, with total scores as continual dependent variables and habitual characteristics as categorical dependent variables, respectively. In addition, the longitudinal associations between sleep characteristics (total scores) and adiposity measures were explored, with adjustments first for child age, sex, family obesity risk, and baseline measures at age 1 (model 1) and additional adjustments for other child and family factors as well as child screen time and physical activity level (model 2). For sleep characteristics with total scores significantly associated with child adiposity, we further assessed the adjusted associations of habitual characteristics with adiposity measures as well as whether the associations varied by obesity family risks. Data were analyzed by using SPSS version 25 (IBM SPSS Statistics, IBM Corporation). All tests were 2 sided, and *P* values of <.05 were considered statistically significant. The Bonferroni correction for multiple testing was used when multiple comparisons were made.

TABLE 2 Baseline Characteristics of Participants by Risk Groups

	Low Risk (*n* = 43)	High Risk (*n* = 64)	*P*
Child			
Sex			
Male	17 (39.5)	35 (54.7)	.12
With siblings			
Yes	19 (44.2)	32 (50.0)	.56
Attending day care			
Yes, full-time	1 (2.3)	4 (6.3)	.35
Mother			
Age	34.3 (4.2)	34.4 (4.5)	.55
BMI	23.0 (2.2)	31.4 (5.7)	<.001
Education level			
<12 y	9 (20.9)	24 (37.5)	.07
Nordic			
No	4 (9.3)	8 (12.5)	.61
Father			
Age	36.7 (4.9)	36.3 (5.0)	.67
BMI	23.3 (1.6)	29.6 (6.6)	<.001
Education level			
<12 y	10 (23.3)	33 (51.6)	.003
Nordic			
No	3 (7.0)	14 (21.9)	.04
Family			
Education level[a]			
Low	4 (9.3)	19 (29.7)	.01
Living conditions			
Apartment	21 (48.8)	24 (37.5)	.24

The baseline characteristics were collected when the child was 1 y old. The marital status of all families included in this study was "married or cohabiting" at baseline. The mean (SD) and number (%) are presented for normally distributed data and categorical data, respectively. *P* value: independent *t* tests were performed on normally distributed data and χ^2 tests were performed on categorical data.

[a] Family education level: low level = neither parent's education was >12 y; high level = at least 1 parent's education was >12 y.

RESULTS

Participant Characteristics and Weight Development

In total, 107 children were included in the analysis, with 43 in the low-risk group and 64 in the high-risk group. Compared with those excluded (*n* = 65), included children were more often from low-risk families, had a mother of lower BMI, lived in a detached house, and had lower BMI *z* score and waist circumference at age 6 (Supplemental Table 6). Among included participants, parents in the high-risk group were heavier than those in the low-risk group, as expected. Fathers in the high-risk group were more likely to have lower education and less likely to be born in Nordic countries. More families in the high-risk group had low education levels (Table 2).

Anthropometric measurements of children by age and risk groups are presented in Supplemental Table 7 and Supplemental Fig 3. Children in the 2 groups had similar BMI *z* scores and waist circumferences at baseline.

From ages 1 to 6 years, children in the high-risk group showed greater increases in BMI z scores (estimated mean difference 0.51; 95% confidence interval [CI] 0.21 to 0.81) and waist circumference (estimated mean difference 1.62 cm; 95% CI 0.68 to 2.56) than children in the low-risk group, adjusted for age, sex, family education level, and baseline measures.

Sleep Measures and Characteristics

The sleep measures in children across ages are presented in Supplemental Table 8. After adjustment for child and family factors, family obesity risk was not associated with any sleep variable across ages (at the Bonferroni-corrected α level of 0.004). Child age was related to most sleep measures. From ages 2 to 6 years, children tended to get later sleep onset (7 minutes/year; $P < .001$), shorter total sleep duration (14 minutes/year; $P < .001$), and better sleep efficiency (1.4% per year; $P < .001$). Weekday-weekend variation in both sleep onset (8 minutes/year; $P < .001$) and sleep duration (4 minutes/year; $P = .007$) increased with age, whereas the day-to-day variation in sleep onset was relatively constant across ages.

The prevalence of sleep characteristics at each age, total scores, and the prevalence of habitual characteristics in children at different risks are presented in Table 3. The prevalence of late sleep increased with age, and the prevalence of low sleep efficiency decreased with age, indicating that sleep schedules grew later, whereas sleep quality improved from ages 2 to 6 years. The prevalence rates of short sleep duration, long sleep latency, and irregular sleep onset were relatively stable over time. Around 20% of children had habitual late sleep and habitual irregular sleep onset from 2 to 6 years old, respectively. Only 2% had habitual low sleep efficiency. After adjustment for other child and family factors, obesity risk was not associated with either total scores or prevalence of habitual characteristics (at the Bonferroni-corrected α level of 0.005).

Association of Sleep Characteristics and Measures of Adiposity

The associations of sleep characteristic scores with adiposity are presented in Table 4. When only adjusted for child age, sex, and family obesity risk (model 1), scores of late sleep and short sleep duration were both positively associated with changes in BMI z score and waist circumference across ages (at the α level of 0.05). After further adjustment for other child and family factors (model 2), the association between the short sleep duration score and waist circumference attenuated to no significance. However, at the Bonferroni-corrected α level of 0.005, only association between the late sleep score and waist circumference was significant. The late sleep score was still associated with waist circumference (0.58 cm; 95% CI 0.21 to 0.95; $P = .003$) after further adjustment for sleep duration, indicating that the relationship between more exposure to late sleep and weight development was independent of sleep duration. No relationship could be detected between other sleep characteristic scores and adiposity.

The associations of habitual late sleep and habitual short sleep duration with adiposity were further examined, respectively, as was the possible interactive effect with family obesity risk. After adjustment for other factors, children with habitual late sleep had greater increases in both BMI z score (estimated mean difference 0.39; 95% CI 0.03 to 0.75) and waist circumference (estimated mean difference 1.50 cm; 95% CI 0.30 to 2.70) from ages 2 to 6 years relative to those without. A significant combined effect was detected between high family obesity risk and habitual late sleep. More specifically, for children at low obesity risk, having habitual late sleep did not significantly increase the risk of gaining more weight across ages. Meanwhile, for children at high obesity risk, having habitual late sleep significantly related to greater increase in both BMI z score and waist circumference across ages (Table 5, Fig 1). Habitual short sleep duration was not associated with either BMI z score (estimated mean difference 0.27; 95% CI −0.16 to 0.70) or waist circumference (estimated mean difference 0.71 cm; 95% CI −0.69 to 2.12).

DISCUSSION

This study indicated that the development of sleep patterns was similar in children at different obesity risks based on parental weight. The higher prevalence of late sleep was associated with greater increases in adiposity measures between ages 2 and 6 years. Furthermore, children who were at high obesity risk and had habitual late sleep had significantly greater increases in adiposity measures, indicating a combined effect of late sleep and parental obesity on child adiposity.

We observed child sleep between ages 2 and 6 years, taking family obesity risks based on parental weight into consideration. No difference in either sleep measures or sleep characteristics was detected between obesity risk groups across ages. This finding is not consistent with our previous observations among children age 1 to 2 years, in which long sleep latency, low sleep efficiency, as well as variable sleep schedules between weekdays and weekends were more common in children at high obesity risk.[15,24] We hypothesized that children in families with obese parents were more exposed to unfavorable sleep hygiene because it has been reported that adults with obesity tend to have

TABLE 3 Sleep Characteristics in Children at Different Obesity Risks, Including the Prevalence at Each Age, the Total Scores, and the Prevalence of Habitual Characteristics

Sleep Characteristic	Age 2, %	Age 3, %	Age 4, %	Age 5, %	Age 6, %	Total Scores, Mean (SD)	Habitual Characteristic,[a] %
Late sleep							
High risk	20.3	31.3	28.1	37.5	45.3	1.63 (1.1)	21.9
Low risk	23.3	34.9	30.2	37.2	34.9	1.58 (1.3)	25.6
Short sleep duration							
High risk	19.1	23.4	25.0	21.9	31.3	1.17 (1.1)	13.2
Low risk	38.6	18.6	27.9	23.3	13.9	1.16 (1.1)	13.2
Long sleep latency							
High risk	23.4	31.2	28.1	21.9	29.7	1.22 (1.1)	14.1
Low risk	27.9	30.2	25.6	20.9	16.3	1.23 (1.1)	14.0
Low sleep efficiency							
High risk	37.5	15.6	5.0	2.0	7.8	0.69 (0.8)	2.0
Low risk	25.6	7.0	5.0	7.0	2.3	0.53 (0.7)	2.5
Irregular sleep onset							
High risk	26.2	30.9	33.4	25.0	30.3	1.41 (1.3)	21.7
Low risk	24.1	34.4	25.6	22.0	16.7	1.23 (1.4)	17.4

Results are pooled results from all imputation data sets.
[a] Habitual characteristics: the total score of the characteristic was >3.

poorer and more irregular sleep than normal-weight individuals.[25,26] However, we could not confirm this hypothesis in the follow-up data using objective measures. These inconsistencies may be due to the previous studies primarily using parental reports, which have lower validity in measures of sleep quality.

We observed that frequent short sleep exposure was significantly related to greater increases in BMI *z* score from 2 to 6 years old. Although the association was not significant at the Bonferroni-corrected α level of 0.005, this finding is in line with other studies using repeated sleep measures, relating short sleep to higher BMI at different ages in early childhood.[27,28] Moreover, we objectively confirmed that children with more frequent exposure to late sleep (sleep onset after 21:00) from 2 to 6 years old had greater weight gain over time; this association was independent of sleep duration. Using reported data, late sleep (after 21:00) in children at age 4 to 5 years has been associated with higher BMI at the same ages[29] and with higher risk of obesity in adolescents.[30] Authors of another study among 4-year-old children reported that the association between short sleep and higher BMI was mediated by late sleep.[31] The underlying mechanism is unclear. In adolescents and adults, late sleep has been associated with greater total energy intake, greater energy intake at night, higher consumption of fast food, lower consumption of vegetables, and more screen time.[32–34] However, a study of children at age 33 months revealed no relationship between late sleep and greater energy intake.[35]

TABLE 4 Associations of 5 Sleep Characteristic Scores With Adiposity in Children From Ages 2 to 6 Years

Sleep Characteristic Scores[a]	Adiposity	
	BMI *z* Score	Waist Circumference
Late sleep		
Model 1	0.12 (0.02 to 0.23)*	0.45 (0.10 to 0.80)**
Model 2	0.16 (0.05 to 0.27)*	0.60 (0.23 to 0.98)**
Short sleep duration		
Model 1	0.12 (0.01 to 0.24)*	0.47 (0.10 to 0.84)*
Model 2	0.12 (0.01 to 0.25)*	0.39 (−0.01 to 0.80)
Long sleep latency		
Model 1	0.11 (−0.03 to 0.25)	0.25 (−0.17 to 0.67)
Model 2	0.12 (−0.06 to 0.18)	0.23 (−0.25 to 0.71)
Low sleep efficiency		
Model 1	0.10 (−0.08 to 0.29)	0.20 (−0.38 to 0.77)
Model 2	0.16 (−0.03 to 0.34)	0.20 (−0.42 to 0.82)
Irregular sleep onset		
Model 1	0.01 (−0.14 to 0.89)	0.14 (−0.23 to 0.51)
Model 2	0.04 (−0.08 to 0.15)	0.26 (−0.13 to 0.64)

Results are pooled for all imputation data sets. Application of the Bonferroni correction for multiple comparisons indicates statistical significance at $P < .005$. Values are estimated parameters for fixed effects of sleep characteristics: total scores (95% CI). Model 1: adjusted for age, sex, obesity risk group, and baseline BMI *z* score or waist circumference. Model 2: model 1 additionally adjusted for other confounders, including maternal ethnicity, family education level, parental marital status, living condition, having siblings or not, screen time, and physical activity level.
[a] Total score ranged from 0 to 5, with higher scores indicating higher prevalence of late sleep onset, short sleep duration, long sleep latency, low sleep efficiency, and greater day-to-day sleep-onset variation, respectively, across ages.
* $P < .05$; ** $P < .005$.

Subjectively assessed poor sleep quality has been related to obesity in children and adolescents,[7] whereas studies using objective indicators have yielded inconsistent results.[36,37] One study in school-aged children

TABLE 5 Adjusted Associations of Habitual Late Sleep With Adiposity by Obesity Risks

	Low Obesity Risk		High Obesity Risk	
	Estimate (95% CI)	*P*	Estimate (95% CI)	*P*
BMI *z* score				
No habitual late sleep	Reference	—	0.39 (0.05 to 0.73)	.023
Habitual late sleep	0.15 (−0.41 to 0.70)	.60	0.93 (0.40 to 1.45)	<.001
Waist circumference				
No habitual late sleep	Reference	—	1.64 (0.56 to 2.71)	.003
Habitual late sleep	0.91 (−0.79 to 2.61)	.29	3.45 (1.78 to 5.12)	<.001

Habitual late sleep: total score of late sleep ≥3. No habitual late sleep: total score of late sleep <3. Values are estimated parameters for effects of having or not having habitual late sleep by obesity risks (95% CI). All models were adjusted for child age, sex, maternal ethnicity, family education level, parental marital status, living condition, having siblings or not, screen time, physical activity level, total sleep duration, and baseline BMI *z* score or waist circumference. —, not applicable.

revealed no associations of objectively assessed sleep-onset latency and sleep efficiency with obesity after 2 years' follow-up.[38] No association between sleep quality and adiposity was detected in our study. Moreover, whereas numerous cross-sectional studies reveal that variable sleep schedules are related to higher weight in school-aged children and adolescents,[5,39,40] no longitudinal association between irregular sleep onset and weight development was observed in this young population.

We know of no study investigating the interactive effect between sleep and family obesity risk on child adiposity. Because both child sleep and weight develop in the family, family contextual conditions should be considered in linking sleep and adiposity. Authors of a previous study reported that the association between poor sleep and child BMI was more pronounced in families with low parental education, poverty, and more stressful life events.[36] In this study, we focused on parental obesity, which has been identified as the most important family contextual factor for childhood obesity.[41] Although the prevalence of late sleep did not differ between children at high or low obesity risk, habitual late sleep significantly amplified the relations between family obesity risk and child adiposity. This may indicate that generational transfer of obesity was stronger in children with habitual late sleep. It may also suggest that other unmeasured genetic or environmental factors in high-risk families (eg, child-rearing practices and diet) affect both child sleep and weight development.

Our study has several strengths. The repeated measurements of sleep using actigraphy and longitudinal design provide objective evidence on the associations of sleep characteristics with adiposity outside of only sleep duration. Moreover, we assessed the association between sleep and obesity in the context of different family obesity risks. Some limitations should be addressed. First, collecting actigraphy data in the longitudinal study was challenging, particularly in children from high-risk families because they were allocated to the control group and probably felt less motivated. Strategies need be considered to improve cooperation. Fortunately, the missing data were MCAR, suggesting that the analysis remained unbiased. We also conducted multiple imputation, which is useful for handling missing data and results in a valid statistical inference.[42] Second, there are no thresholds available for actigraphy-assessed sleep duration and sleep

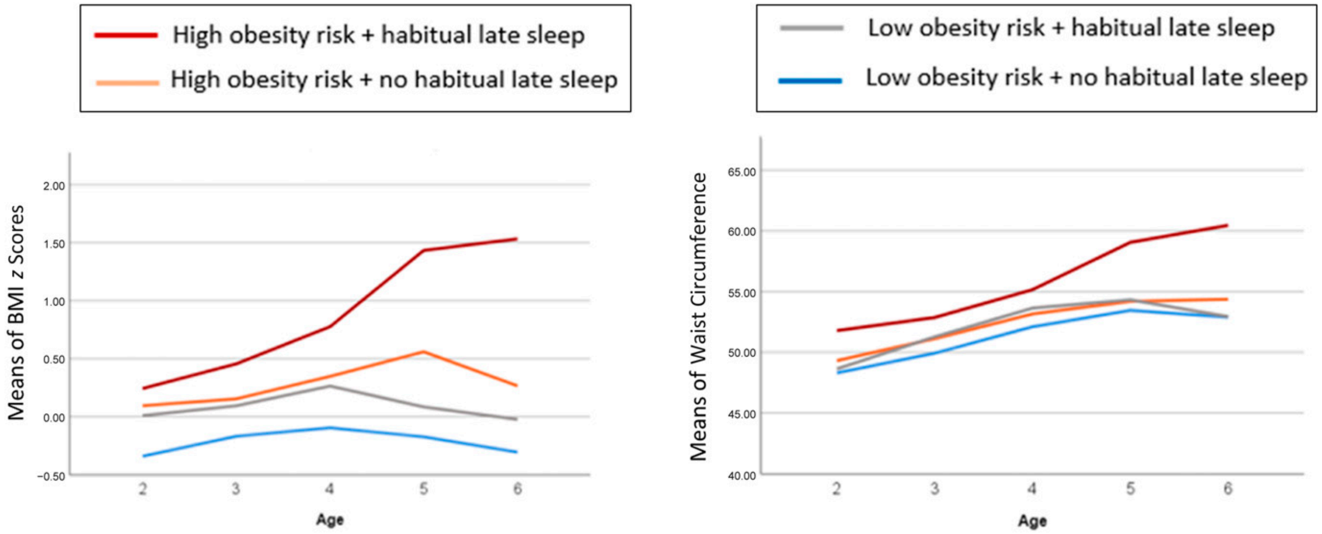

FIGURE 1
BMI *z* score and waist circumference in children with or without habitual late sleep by different family obesity risks, ages 2 to 6 years.

variation. Sleep recommendations from the National Sleep Foundation and the American Academy of Pediatrics are based on subjective sleep data,[43,44] which are unsuitable for objective data. Therefore, quartiles were used to classify these 2 characteristics. This may be somewhat arbitrary and increase capitalization on chance. Moreover, factors not included in the study (eg, dietary patterns and objectively measured physical activity) may differ between children at different obesity risks. Finally, the small sample size may limit the statistical power to detect small differences in sleep development.

CONCLUSIONS

More frequent exposure to late sleep was associated with greater increases in adiposity measures from ages 2 to 6 years, particularly in children at high obesity risk based on parental obesity. Late sleep is probably involved in familial vulnerability to obesity development and needs more attention in prevention efforts.

ABBREVIATIONS

CI: confidence interval
MCAR: missing completely at random
WASO: wake after sleep onset

and all authors approved the final manuscript as submitted and agreed to be accountable for all aspects of the work.

This trial has been registered at www.clinicaltrials.gov (identifier NCT01198847).

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Sleep and Adiposity in Children From 2 to 6 Years of Age

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