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Longitudinal relations between children's sleep and body mass index: the moderating role of socioeconomic risk

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Two separate meta-analyses[1,2] concluded that across nonexperimental studies of children and adolescents, there was consistent evidence of a negative association between short sleep duration and body mass index (BMI). Since 2008, a number of studies have not only provided additional evidence on relations between sleep and BMI but have also pushed the field forward by documenting longitudinal relations and, therefore, direction of effects, between multiple sleep parameters and BMI. Findings from longitudinal work suggest that insufficient and poor-quality sleep may be a contributing factor to the epidemic of youth who are overweight and obese.

In a prospective longitudinal study with nearly 2000 children, Seegers et al[3] found that a 1-hour decrease in parent-reported time in bed at age 10 years was a significant risk factor for obesity at age 13 years. Other longitudinal research[4] found that shorter parent- and child-reported sleep duration at ages 8 to 11 years predicted higher zBMI, but only for boys in the sample. Most recently, El-Sheikh et al[5] included actigraphic-assessed and subjectively reported sleep and used latent growth modeling to examine relations between sleep and BMI from late childhood into early adolescence. Contrary to the findings of Storfer-Isser et al,[4] results of this study were significant only for girls in the sample and suggested that child-reported sleep problems at 9 predicted higher BMI at age 11 years, whereas shorter objectively assessed sleep minutes predicted more growth in BMI over those 2 years.

However, it is also worth noting that not all prior investigations have found significant longitudinal relations between BMI and sleep. Araujo et al[6] examined the association between self-reported sleep duration and zBMI in a sample of more than 1000 adolescents. After accounting for zBMI at age 13 years, sleep duration did not predict zBMI at age 17 years. It is possible that through a more nuanced examination of the conditions under which the influence of sleep on BMI is heightened or buffered, a better understanding of inconsistencies across samples and studies will be gained.

One factor that may moderate the relations between sleep and BMI is socioeconomic and family-level risks. Several individual risk factors have been shown in prior studies to be associated with both poor sleep and obesity risk; thus, it is possible that these same factors could serve to intensify the relations between sleep and BMI. Fewer economic resources or living below the poverty line and low maternal education have been shown to be associated with childhood obesity[7] and children's insufficient sleep.[8] In addition, single parent–headed households have been shown to place children at risk for obesity and overweight[7] and greater sleep problems.[9] Similarly, high levels of maternal stress appear to place children at greater risk for obesity[10] and sleep problems.[11]

Research examining the influence of individual socioeconomic and family-level variables has served as the foundation for our understanding of the influence of risk on development. However, risk factors are often experienced in combination with one another and regression approaches that statistically isolate individual factors might obscure effects of the accumulation of risk. The cumulative risk approach[12] provides a parsimonious framework for understanding how accumulation of risk, rather than any single risk factor alone, might adversely affect developmental outcomes. By dichotomizing risk factors as "present" or "not present," cumulative risk index scores indicate how much risk an individual is exposed to. This approach arguably better represents naturally occurring covariation of stressor exposure. One conceptual explanation for the cumulative risk approach suggests that it is chronic stress, regardless of the individual stressors, which leads to negative outcomes. Potentially, this occurs through dysregulation of the stress response systems and contributes to allostatic load (the "wear and tear" on the physiological systems from repeated demands to respond to stress and maintain homeostasis).[13] Thus, cumulative risk exposure could make children more vulnerable to the negative effects of insufficient sleep on BMI and possibly function as a moderator that would strengthen the relations between poor sleep and greater BMI.

Prior research has shown that greater cumulative risk exposure in childhood leads to larger gains in BMI over 4 years.[14] Yet, to our knowledge, only 1 study has examined the possibility that children whose sleep is short or of low quality in the context of high levels of socioeconomic risk would have higher BMI than their counterparts who are not exposed to socioeconomic risk. This was a cross-sectional study with 10-year-old children using an initial wave of the same data set as the current investigation (the present longitudinal study used the subsequent wave).[15] Specifically, similar to the

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assessment of risk in the present study, risk index scores were created from 4 variables commonly considered as representing socioeconomic and family-level risks (living below poverty line, low maternal education, single parent status, and high levels of maternal stress). Actigraphy-based sleep minutes and efficiency were derived. Findings identified cumulative risk as a moderator of relations between sleep and zBMI. Specifically, the association between either short sleep duration or poor sleep quality and zBMI was evident only for children experiencing cumulative risk. Longitudinal examination of these relations is essential and can shed some light on the role of sleep as a predictor of change in BMI over time.

A review of the literature that examined longitudinal relations between BMI and sleep[16] concluded that 1 major limitation of the research to date is that few have included multiple parameters of sleep (measures of duration and quality) measured objectively. This limitation may be of particular consequence for studies focusing on children and adolescents because of their reliance on parent-reported sleep. Previous research has shown parental reports to overestimate true sleep time[17]; therefore, objective means of collecting sleep data are less prone to systematic bias.

The aim of the current study is to examine the role of cumulative risk as a moderator of longitudinal relations between sleep (short duration, poor quality, and increased variability in sleep schedule) and zBMI in a community sample of children. We chose to examine indicators of different aspects of sleep (quantity, quality, and variability), as prior research has shown these sleep variables to be related to socioeconomic disadvantage.[18] Toward assessment of the research question, autoregressive effects of zBMI were controlled yielding an explication of change in zBMI over time. Consistent with dual-risk perspectives,[19] it was expected that children with increases in zBMI over 1 year would be those with poor sleep in conjunction with higher levels of socioeconomic and familial risks. Conversely, the association between sleep and zBMI was expected to be attenuated among children exposed to lower levels of cumulative risk. Sleep problems in this study are conceptualized and examined along a continuum and refer to actigraphy-based fewer sleep minutes, lower sleep efficiency (an index of sleep continuity/fragmentation and quality), and increased variability in sleep onset.

Method

Participants

The current investigation is based on 2 study waves (T1 and T2) that occurred between 2010 and 2012 and is part of a larger study (second and third waves of the larger study) focused on relations between biopsychosocial processes and developmental outcomes (Auburn University Sleep Study). Children and their families were recruited from public schools in Alabama. Exclusion criteria included the child having a diagnosed learning disability or sleep disorder. The T1 sample was the same as in Bagley and El-Sheikh.[15]

The current analytical sample included 279 children. Five children who had a chronic illness, including sickle cell (n = 1), diabetes (n = 1), eczema (n = 1), bronchitis (n = 1), and stomach ulcers (n = 1) were excluded. In total, 274 children (123 girls; mean age, 10.40 years; SD, 7.78 months) participated at T1; 64% were European American, and 36% were African American. Based on mothers' reports of pubertal status (1, prepubertal; 2, early pubertal; 3, midpubertal; 4, late pubertal; 5, postpubertal),[20] boys were prepubertal (mean, 1.53; SD, 0.37), and girls were early pubertal on average (mean, 1.98; SD, 0.60).

Families represented a wide range of socioeconomic backgrounds based on the income-to-needs ratio, which is computed using family size and income (US Department of Commerce; www.commerce.gov). Income-to-needs ratio ranged from 0.27 to 4.10 (mean, 1.63; SD, 0.97); a score ≤1.0 is considered poverty. Most children lived with their biological mother (91%); the remaining children lived with another family member. For consistency, we use the term *mother*.

About 1 year later, 256 children (117 girls; 63% European American and 37% African American) returned for T2 (mean duration between T1 and T2, 336 days; SD, 34.74 days). Mean comparison analyses were conducted to determine whether retained and attrited families (from T1 to T2) differed on control and focal study variables; no differences were detected.

Procedures

The study was approved by the university's institutional review board, and consent and assent were obtained. AT T1, children wore actigraphs on their nondominant wrist for 7 consecutive nights. Sleep assessments occurred during the regular school year, excluding holidays, and only data from medication-free nights were included. Parents completed child sleep diary logs to corroborate actigaphy data.[21] Shortly after the actigraph assessment (typically the following day), families visited an on-campus laboratory to complete questionnaires, and children's height and weight were assessed. At T2, children's height and weight were reassessed at the same laboratory with the same scale used at T1. Families were compensated monetarily for their time.

Measures

Sleep

At T1, actigraphs (Octagonal Basic Motionloggers; Ambulatory Monitoring, Inc, Ardsley, NY) were used to obtain objective sleep parameters. Motion was measured in 1-minute epochs using zero crossing mode. The Octagonal Motionlogger Interface with Actme software and the analysis software package (Action W2; 2000 Ambulatory Monitoring, Inc) were used. Sadeh's scoring algorithm[22] was used to determine whether children were awake or asleep. The actigraph and analysis software package have established validity for the assessment of children's sleep.[22,23]

Using data from all available nights, 3 frequently used sleep parameters were derived: (*a*) sleep minutes—number of minutes scored as sleep between sleep onset and morning wake time; (*b*) sleep efficiency—percentage of minutes between sleep onset and wake time spent asleep; and (*c*) variability in sleep schedule—variability in sleep onset time across the week of actigraphy, which was computed using the mean-centered coefficient of variance statistic.[24] These parameters index sleep duration, quality, and schedule. On average, participants had 5.97 nights of valid actigraphy data (SD, 1.29; range, 0-7 nights). Reasons for missing data included forgetting to wear the actigraph, discrepancies between sleep logs and actigraphy, and mechanical problems. Researchers have recommended that actigraphy assessments include at least 5 nights,[25] and, thus, sleep data (not cases) for those who had less than 5 valid nights were excluded from analyses (14% had <5 nights). Intraclass correlations indicated adequate night-to-night stability for sleep minutes ($\alpha = .77$) and sleep efficiency ($\alpha = .89$) over the week.

Risk index score

At T1, the risk index score was created using 4 family functioning measures: marital status, maternal education, poverty, and stressful life events. These variables are commonly used to create a cumulative risk index in the family risk literature.[14,26] Marital status, maternal education, and family poverty were treated as dichotomous variables. Marital status was measured by assessing whether primary caregivers in the household were married. Maternal education was

determined by whether the mother or primary caregiver had graduated from high school. Family poverty was measured using income-to-needs ratio and was dichotomized as below the poverty line (income-to-needs ≤1) or above. For stressful life events, mothers reported on the occurrence of common life stressors over the past 12 months, including job loss, housing difficulties, and death of a family member using the well-established 20-item Recent Life Events Scale.[27] Those who scored in the top quartile were coded as experiencing risk; this approach of dichotomizing continuous variables is common in the cumulative risk literature.[26]

To compute the cumulative risk variable, single parent status, maternal educational attainment of less than a high school diploma, family income-to-needs ratio below the poverty line, and scores in the top quartile of stressful life events were individually coded as risk (1, risk; 0, no risk). Next, the risks were summed to calculate the total cumulative risk score for each child, resulting in a possible range of 0-4 (0, 42%; 1, 34%; 2, 18%; 3, 5%; 4, 1%). Because of the small sample size at the 2 highest risk levels, 3 levels of risk were created (1) no risk, (2) 1 risk factor, and (3) 2 or more risk factors. Table 1 provides a summary of risk factors and frequencies.

Weight status

At T1 and T2, children's height (in centimeters) and weight (in kilograms) were assessed with the Tanita wall-mounted stadiometer and digital weight scale (Arlington Heights, IL). Shoes and heavy clothing were removed for the assessment. Height, weight, sex, and age were used to calculate standardized BMI scores (zBMI); conversion was conducted using SAS program provided by the Center for Disease Control and Prevention.[28] Based on Center for Disease Control and Prevention criteria, at T1, 57% of children in our sample were considered having a healthy weight (5th-85th percentile in the United States), 14% were considered as overweight (85th-95th percentile), 26% were classified as obese (>95th percentile), and 3% met criteria for underweight (<5th percentile). These classifications were similar at T2.

Results

Statistical analysis plan

Path models were fit to examine cumulative risk at T1 as a moderator of relations between sleep at T1 (sleep minutes, sleep efficiency, and variability in sleep onset) and zBMI at T2. To account for autoregressive effects, T1 zBMI was allowed to predict zBMI at T2. Controlling for autoregressive effects helps to eliminate bias in parameter estimates, permits for conclusions regarding change in the predicted variable, and provides information about the directionality of effects.[29] In each model, variables known to influence sleep or BMI were controlled including child sex, ethnicity, age, pubertal status, and asthma (36 children had asthma). The covariates were allowed to correlate with the T1 sleep variable, cumulative risk at T1, zBMI at T1, and the interaction term (sleep × cumulative risk) at T1 and to predict zBMI at T2. To reduce outlier effects, data points that exceeded 3 SDs among study variables were removed. Specifically, 4 values were removed for T1 sleep minutes; 6 values, for T1 sleep efficiency; 2 values, for T1 variability in sleep onset; 1 value, for T1 zBMI; and 1 value, for T2 zBMI.

Table 1
Frequencies of risk factors.

Measure	Definition of risk	Prevalence of risk (%)
Income-to-needs ratio	1 or below (below poverty)	33%
Maternal education	Did not complete high school	8%
Family structure	Primary caregiver was unmarried	32%
Recent life events	Top quartile	25%
Cumulative risk index	0	42%
	1	33%
	2 or more	25%

Table 2
Descriptive statistics and bivariate correlations among primary study variables.

	1.	2.	3.	4.	5.	6.
1. Sleep minutes T1	–					
2. Sleep efficiency T1	0.67**	–				
3. Variability in sleep onset T1	−0.11	−0.04	–			
4. Cumulative risk T1	−0.11	−0.04	0.08	–		
5. zBMI T1	−0.20*	−0.10	−0.03	0.17*	–	
6. zBMI T2	−0.19*	−0.04	0.01	0.20*	0.93**	–
Mean	447 min	88.95	0.04	0.76	0.64	0.77
SD	43 min	6.20	0.02	0.78	1.13	1.17

Note: For sleep minutes, 447 minutes translates into 7 hours and 27 minutes. The 3 sleep measures were derived from wrist actigraphy.
* $P < .01$.
** $P < .001$.

Because of power considerations, separate models were fit for each sleep parameter (a total of 3 path models were fit). Following well-established practices, interactions were plotted at high (+1 SD) and low (−1 SD) levels of sleep and at the 3 different levels of cumulative risk.[30] The interaction utility of Preacher et al[31] was used to plot interactions.

Analyses were conducted using Amos 22. Full information maximum likelihood was used to handle missing data.[32] Nonsignificant covariances among exogenous variables were omitted from each model to increase degrees of freedom. Acceptable model fit was based on satisfying at least 2 of the 3 following criteria: $\chi^2/df < 3$, comparative fit index (CFI) > 0.90, and root mean square error approximation (RMSEA) ≤ 0.08[33]; each model satisfied these criteria.

Preliminary analyses

Descriptive statistics and bivariate correlations among study variables are presented in Table 2.

Cumulative risk at T1 as a moderator of relations between sleep at T1 and change in zBMI at T2

Sleep minutes

The path model fit to examine cumulative risk at T1 as a moderator of relations between T1 sleep minutes and T2 zBMI was a good fit: $\chi^2 = 14.39$ ns, $df = 25$; $\chi^2/df = 0.57$; CFI = 0.99; RMSEA = 0.02 ns (Fig. 1). In total, the model explained 87% of the variance in zBMI at T2. Asthma at T1 was related to zBMI at T2, such that those with asthma had decreased zBMI. Furthermore, zBMI at T1 and T2 was strongly related. Cumulative risk at T1 interacted with T1 sleep minutes to predict zBMI at T2 ($\Delta R^2 = 0.03$). At high levels of sleep minutes, zBMI was rather similar for all children regardless of cumulative risk (Fig. 3A). However, at low levels of sleep minutes, children with 2 or more risk factors had greater zBMI than those with fewer risk factors.

Sleep efficiency

The path model fit to examine cumulative risk at T1 as a moderator of relations between T1 sleep efficiency and T2 zBMI was a good fit: $\chi^2 = 23.15$ ns, $df = 24$; $\chi^2/df = 0.04$; CFI = 0.99; RMSEA = 0.00 ns (model not shown for brevity). However, cumulative risk at T1 did not function as a moderator of the relation between sleep efficiency at T1 and zBMI at T2.

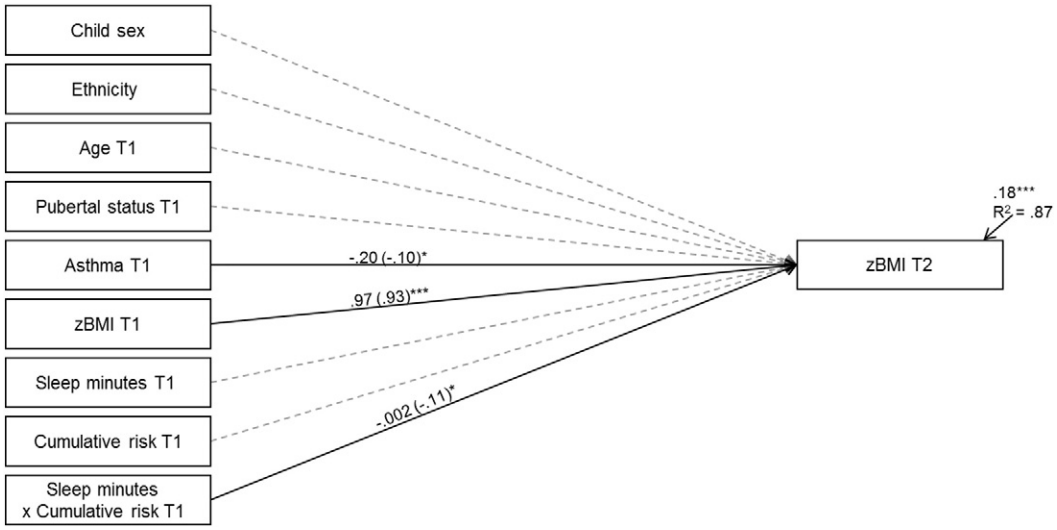

Fig. 1. Examination of cumulative risk at T1 as a moderator of relations between sleep minutes at T1 and zBMI at T2. Nonsignificant covariances among exogenous variables were omitted from the model. Unstandardized and standardized coefficients (in parentheses) are provided. For ease of interpretation, statistically significant lines are solid, whereas nonsignificant lines are dotted. Child sex was dummy coded such that 1 = boys and 0 = girls. Ethnicity was dummy coded such that 1 = African American and 0 = European American. Asthma was dummy coded such that 1 = asthma and 0 = no asthma. T1, data collected at T1; T2, data collected at T2. Model fit: $\chi^2 = 14.39$ ns, $df = 25$; $\chi^2/df = 0.57$; CFI = 0.99; RMSEA = 0.02 ns.

Variability in sleep onset

The path model fit to examine cumulative risk at T1 as a moderator of relations between T1 variability in sleep onset and T2 zBMI was a good fit: $\chi^2 = 17.30$ ns, $df = 24$; $\chi^2/df = 0.72$; CFI = 0.99; RMSEA = 0.00 ns (Fig. 2). In total, the model explained 88% of the variance in zBMI at T2. Cumulative risk at T1 moderated relations between variability in sleep onset at T1 and increased zBMI at T2 ($\Delta R^2 = 0.03$). Specifically, greater variability in sleep onset predicted an increase in zBMI 1 year later, but only for children who experienced 2 or more risk factors at T1. Variability in sleep onset was not related to an increase in change in zBMI over time for children who experienced 1 risk factor or zero risk factors (Fig. 3B). In initial analyses, variability in wake time was considered, but no effects emerged possibly because all children woke up early during the school week.

Discussion

The results of this study extend prior cross-sectional research showing that, in the context of socioeconomic and familial risk, the effects of short sleep duration on growth in zBMI are amplified. In addition, novel findings show that variability in sleep onset interacted with cumulative risk to predict increases in zBMI over 1 year. The findings are notable because the moderating effects were seen despite the highly stable nature of zBMI over the course of a year and the fact that we controlled for T1 zBMI when predicting zBMI at T2. Overall, results indicate that not all children with poor sleep are at risk for higher zBMI. Rather, explicating vulnerability and protection in the context of poor sleep, children with poor sleep (shorter duration and variable schedule) in conjunction with higher levels of

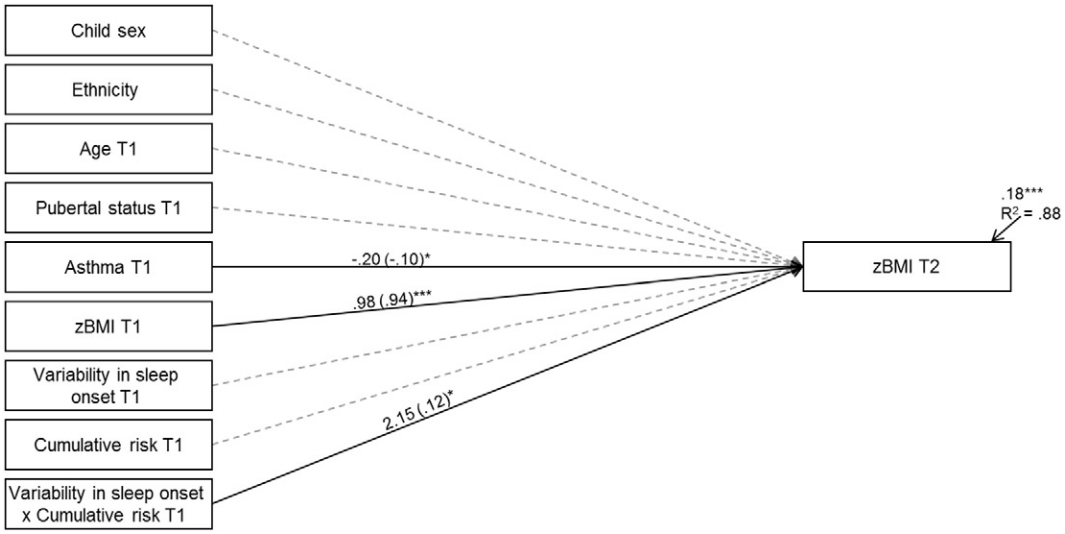

Fig. 2. Examination of cumulative risk at T1 as a moderator of relations between variability in sleep onset at T1 and zBMI at T2. Nonsignificant covariances among exogenous variables were omitted from the model. Unstandardized and standardized coefficients (in parentheses) are provided. For ease of interpretation, statistically significant lines are solid, whereas nonsignificant lines are dotted. Child sex was dummy coded such that 1 = boys and 0 = girls. Ethnicity was dummy coded such that 1 = African American and 0 = European American. Asthma was dummy coded such that 1 = asthma and 0 = no asthma. T1, data collected at T1; T2, data collected at T2. Model fit: $\chi^2 = 17.30$ ns, $df = 24$; $\chi^2/df = 0.72$; CFI = 0.99; RMSEA = 0.00 ns.

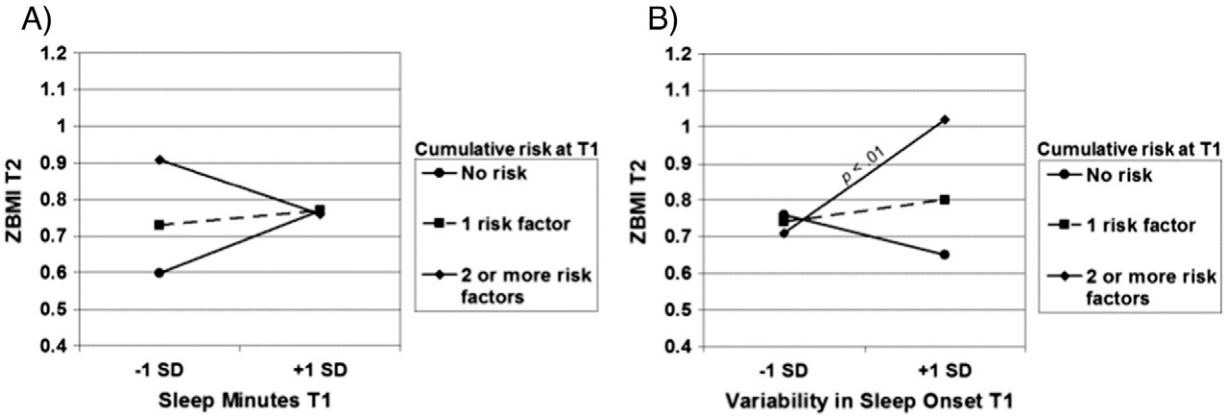

Fig. 3. Cumulative risk at T1 as a moderator of relations between sleep at T1 and change in zBMI at T2. (A) Relations between sleep minutes at T1 and zBMI at T2 are plotted at high (+1 SD) and low (−1 SD) levels of sleep minutes for children who were exposed to no risk, 1 risk factor, or 2 or more risk factors. (B) Relations between variability in sleep onset at T1 and zBMI at T2 are plotted at high and low levels of variability in sleep onset for children who were exposed to no risk, 1 risk factor, or 2 or more risk factors. For the slope that differed from zero, the *P* value is presented next to the slope. T1, data collected at T1; T2, data collected at T2.

socioeconomic and familial risks are those most likely to exhibit increases in zBMI over time.

The use of objective sleep assessment at 2 time points across an important developmental period and the participation of a diverse, community-based sample with a relatively wide range of zBMI and socioeconomic status are study features that strengthened the assessment of our research questions. The sample was drawn from Alabama with comparatively high rates of obesity and poverty. However, research could benefit from examination of sleep stages through polysomnography that could reveal more nuanced relations between sleep, cumulative risk, and BMI. The risk factors chosen to be included in the familial risk index were consistent with prior literature that has examined cumulative risk and health in children. Nevertheless, it is likely that there are other relevant indicators of risk that were not examined and might have enhanced the assessment of the research questions. Finally, although having longitudinal data helps infer directionality of effects, the study is observational and, thus, limited to the extent that causation can be inferred. Furthermore, future consideration of change in zBMI over longer periods or across different developmental periods is warranted.

The findings of the current study have implications for prevention programs and future research. First, health care providers could target children from higher risk families for more in-depth assessment of sleep given the influence it may have on childhood obesity. It is worth noting that, in preliminary analyses, cumulative risk did not moderate relations between child-reported sleep problems and changes in zBMI. This suggests that simply asking children about sleep may not be sufficient to rule out problems with sleep that lead to increases in zBMI, particularly in high-risk contexts. Additional research into the reasons for shorter, more variable sleep patterns in higher risk homes is vital. The factors that lead to poor sleep in children from higher risk families might be different from those that explain poor sleep in lower risk homes, and understanding possible differences might shed light on underlying processes linking sleep to BMI.

A growing literature has examined links between cumulative risk exposure in childhood and health, and this prior work may provide insights into the mechanisms at work in the current study. As mentioned earlier, cumulative risk may indicate exposure to chronic stress and greater allostatic load, which, in turn, may have negative effects on metabolism.[13] Relatedly, McEwen[34] has argued that sleep deprivation may serve as a physiological stressor. Taken together, it is possible that children who are exposed to higher levels of cumulative risk and are not receiving sufficient sleep have the greater burden of allostatic load leading to weight gain. It is possible that there are additional behavioral and contextual differences related to familial risk (eg, daytime sedentary behavior, television watching, or access to calorie dense/nutrition poor foods) that could explain the observed moderating effect. Cumulative risk has also been shown to have detrimental effects on self-regulation[35] and may contribute to weight gain through behavioral pathways. In fact, Evans et al[14] found that early childhood cumulative risk predicted increases in BMI in adolescence and that these relations were mediated by lower self-regulatory abilities, as assessed with delay of gratification tasks. Deficiencies in self-regulatory abilities may also be a consequence of insufficient sleep[36]; thus as our study suggests, poor sleep and high risk together could lead to higher chances of weight gain. Similarly, reciprocal relations between sleep and BMI are not unlikely and should be considered especially with data sets that include multiple assessments across several years. Future research aimed at understanding why the influence of sleep on zBMI is stronger for children from families with more risk may be particularly fruitful for obesity prevention.

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