Longitudinal Associations of Sleep Duration, Morning and Evening Cortisol, and BMI During Childhood

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Objective: This study aimed to examine associations between sleep duration, BMI, and cortisol levels across childhood.

Methods: Participants included 361 children adopted domestically in the United States. Random-intercept cross-lagged panel models tested for between-person and bidirectional within-person associations of sleep duration, BMI, and morning and evening cortisol at age 4.5 to 9 years.

Results: Sleep duration and BMI were stable during childhood, inversely associated at the between-person level, and unrelated to morning or evening cortisol. BMI at age 6 years predicted longer sleep duration and lower evening cortisol at age 7 years, and lower morning cortisol at age 7 years predicted higher BMI at age 9 years within individuals.

Conclusions: The association between sleep and BMI is more likely a stable between-person phenomenon rather than a unidirectional association that develops within individuals over time.

Introduction

The risk of having overweight or obesity has multiple determinants. The current study focuses on one behavioral risk factor: child sleep duration, defined as the total amount of sleep obtained overnight. Several meta-analyses of cross-sectional studies have indicated that short sleep duration is related to obesity in children (1-3). Recent longitudinal studies have also suggested that short sleep duration predicts higher BMI during childhood (4-6).

The mechanisms that link short sleep duration and BMI during childhood are not well understood. Research conducted with adults has suggested several paths, including the impact of short sleep duration on the regulation of hormones related to hunger and satiation (i.e., ghrelin and leptin) (7,8) and hypothalamic-pituitary-adrenocortical (HPA) axis functioning (6). Yet few longitudinal studies have explored these mechanisms in children. Therefore, questions remain regarding why sleep duration is related to children’s BMI and when such associations emerge.

The current study focuses on HPA dysregulation as a potential explanatory mechanism for associations between short sleep duration and BMI across childhood. The HPA axis is activated by the hypothalamus, which stimulates the pituitary gland to produce adrenocorticotropic hormone, which then signals the adrenal gland to secrete cortisol. Cortisol, in turn, influences multiple bodily functions and is associated with downstream increases in blood glucose levels, insulin resistance, heart rate, and immune system stimulation (9). By middle childhood, children show diurnal cortisol patterns that are similar to those of adults: cortisol levels typically peak in the morning (cortisol awakening response) and decline throughout the day (10). There are individual differences in morning and evening cortisol levels and diurnal patterns that reflect a combination of genetic, physical, behavioral, and environmental factors (9,10). We focus on middle
Sleep duration is a behavioral factor that may affect cortisol levels throughout the day. For example, short sleep duration is associated with higher morning and evening cortisol levels among infants and children (9,11,12). This association may be causal, based on evidence from a controlled trial in adults showing that shorter sleep duration was followed by an elevation in cortisol levels the next evening (13). However, a few studies in children have indicated the reverse: cortisol dysregulation may precede sleep problems (14-16). These studies have suggested that HPA dysregulation and sleep duration are associated, but the direction of effects is not clear and warrants further investigation.

Obesity also is associated with HPA dysregulation; however, the type of dysregulation is not clear, and the direction of effects is also ambiguous. Several primarily cross-sectional studies have found that obesity is associated with increased cortisol production and a blunted diurnal pattern, including higher evening levels (17,18). Other studies have reported that obesity is related to normal to low levels of morning and evening cortisol and increased cortisol clearance throughout the day (17,18). Only a few studies have examined associations between cortisol and obesity or BMI during childhood. Similar to the adult findings, there are cross-sectional reports that, between the ages of 4 and 12 years, children with obesity or overweight show greater overall and morning cortisol production relative to healthy-weight children (15,19). However, other cross-sectional reports in the same age range have found that children with overweight and obesity have lower morning and evening cortisol levels (20,21).

The current longitudinal study examined whether associations between sleep duration and BMI (from ages 4.5 to 9 years) are accounted for by HPA regulation during childhood. We explored whether shorter sleep duration predicts higher levels of cortisol, which, in turn, predict higher BMI over time. However, based on contradictory findings in the literature regarding associations between BMI and cortisol levels and the dearth of longitudinal studies that have explored directional effects of HPA functioning and BMI during childhood, we conducted a full cross-lagged panel model. This enabled us to test for bidirectional associations between sleep duration, cortisol, and BMI. Most previous research on longitudinal associations between BMI and sleep or cortisol have not controlled for between-person stability. As a result, directional paths could be overestimated because they reflect the combined influences of stable characteristics and potentially causal processes. Therefore, we modeled random intercepts and their associations to account for between-person stability for each construct, to attenuate the upward bias of cross-lagged and autoregressive estimates, and to clarify the interpretation of these paths as time-specific within-person associations (22-24).

Methods
Participants
Participants included birth parents, adoptive parents, and adopted children from cohort I (N = 361 families and adopted children; 57% male) of the Early Growth and Development Study (EGDS) (25). Participants were recruited from 33 adoption agencies in 10 states across the United States and were eligible if the adoption was domestic, the child was placed in an adoptive family genetically unrelated to the birth parents or adopted child, the adoption occurred before 3 months of age (mean = 7.11 days; SD = 13.28 days; median = 2 days), the adopted child had no major medical condition, and the birth mother and adoptive parents could read or understand English at an eighth grade level. The research was approved by the institutional review boards of all participating organizations (The George Washington University; Pennsylvania State University; University of California, Davis; University of Minnesota; Oregon Social Learning Center; and University of Oregon). Written informed consent was provided by parents at each assessment, as approved by the institutional review boards (see online Supporting Information Appendix I). Table 1 provides demographic characteristics.

Families in cohort I participated in a series of in-person and telephone interviews (full study procedures have been published previously) (25). For the current study, adopted children were assessed at ages 4.5 years (n = 311), 6 years (n = 315), 7 years (n = 301), and 9 years (n = 266) of age and provided saliva samples for cortisol ascertainment on 3 days at home near the time of the 4.5- (n = 211), 6- (n = 212), and 7-year (n = 203) visits. Additionally, height and weight were assessed during a phone interview at ages 48 months and at 9.5 years to reduce missing data for families that did not complete the full 54-month or 9-year assessment (n = 88 and 44 cases supplemented, respectively).

Measures
Cortisol levels. Cortisol levels were assessed by using average morning and evening levels across the 3 days of assessment at ages 4.5, 6, and 7 years. Children (through their adoptive parents) provided saliva samples via passive drool at 30 minutes after waking and at bedtime (before brushing their teeth) on 3 days. Samples were stored by participants and then mailed to the primary study site, frozen until shipped to the University of Trier Laboratory, and stored frozen (at −20°C) until assayed in duplicate via dissociation-enhanced lanthanide fluorescence immunoassay (26). Average usable values across duplicate assays were used as the level of cortisol in that sample (27). Raw mean (SD) morning values (in micrograms per deciliter) across the 3 days for each assessment ranged from 0.60 (0.34) to 0.64 (0.34) at age 4.5 years, from 0.49 (0.28) to 0.52 (0.28) at age 6 years, and from 0.48 (0.30) to 0.52 (0.30) at age 7 years, respectively. Average evening values (in micrograms per deciliter) ranged from 0.06 (0.09) to 0.08 (0.12) at age 4.5 years, from 0.07 (0.12) to 0.10 (0.24) at age 6 years, and from 0.06 (0.09) to 0.09 (0.20) at age 7 years.

Cortisol data were cleaned as follows: outliers more than 3 SDs above the sample mean were winsORIZED (replaced with 3-SD values; <3.02% of any assessment), and any values that were below the detection limit were set to missing (2-11 values at each assessment). Finally, cortisol values were set to missing if steroid medications were used on that day (8-14 cases per day at each assessment). Morning and evening cortisol values across the 3 days of collection were averaged and then log transformed to normalize distributions.

Sleep duration. Sleep duration was assessed from parent-reported bed and wake times. Specifically, we calculated the average time between bedtime and wake time on the following day between days 1 and 2 and days 2 and 3 of the cortisol collection diary at ages 4.5, 6, and 7 years. At age 9, average sleep duration was calculated from three nights on a daily reports measure (28).
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BMI. Height and weight data were extracted from the children’s medical records and from adoptive parent (P1) (usually the mother) reports at the age 4.5-, 6-, 7-, and 9-year assessments. Because the dates of height and weight measurements from the medical records and P1 reports often varied, we created 6-month age bands (e.g., 7 years ± 3 months) and averaged P1 and medical record reports within each age band. The 4.5-year assessment was supplemented with data from the 4-year age band if the data were missing at age 4.5 years. Likewise, the age 9 assessment was supplemented with the 9.5-year age band if data were missing at age 9 years. Raw BMI is recommended to track changes in BMI over time in children because within-person variability in BMI is unrelated to baseline adiposity (an issue for BMI \( z \) scores) (29). (Analyses using BMI \( z \) scores were highly consistent with findings using BMI raw scores; results are available upon author request).

Covariates. Adopted child characteristic covariates included sex, birth weight, growth during infancy, BMI at age 2 years, and exact age and adoption openness at the 4.5-year assessment (25). Adoption openness is included to attenuate bias in adoptive parents’ ratings of child BMI based on birth parent obesity status. Birth weight was drawn from medical records (30). Growth rate during early infancy (early growth) has been associated with obesity (31) and was computed by subtracting children’s sex- and age- based weight \( z \) score (based on Centers for Disease Control and Prevention norms) at birth from their weight \( z \) score at 9 months. Weight was assessed via parental reports and, when available, by medical record reports.

Birth parent characteristics were included to control for genetic and prenatal confounding of cross-lagged associations as well as effects of a relatively lower socioeconomic status than that of adoptive parents. Prenatal influences included pregnancy complications and weight gain assessed primarily through medical record report. If prenatal care visits began after 12 weeks’ gestation, medical record reports were supplemented with birth mothers’ self-reported weight gain (30). Indicators of genetic risk for BMI included birth mothers’ and fathers’ average BMI across 9 years.

Missing data

Percentages of missing data from the initial sample of 361 at each assessment were as follows: sleep: 43% at age 4.5 years, 43% at age 6 years, 50% at age 7 years, and 31% at age 9 years; BMI: 34% at age 4.5 years, 48% at age 6 years, 42% at age 7 years, and 39% at age 9 years; morning cortisol: 43% at age 4.5 years, 45% at age 6 years, and 45% at age 7 years; and evening cortisol: 43% at age 4.5 years, 45% at age 6 years, and 45% at age 7 years. We tested whether a series of demographic (P1 education and household income, P1 and birth mothers’ age when the child was born, and child’s race and/or ethnicity and age at placement) and study variables and/or covariates contributed to participation (yes or no) at the 4.5-year assessment and whether participants dropped out (yes or no) between the 4.5- and 9-year assessments using a series of Kruskal-Wallis one-way analysis of variance (ANOVA) tests. Of 56 tests, 4 (7%) reached significance at \( P < 0.05 \). Children who did not participate at the 4.5-year assessment experienced more pregnancy complications. Participants who were in the study at age 4.5 years but not at age 9 years experienced more pregnancy complications, were older at the 4.5-year assessment, and had a longer sleep duration at age 6. The final analytic sample size was 316 (\( n = 45 \) missing on all study variables).

### Table 1 Sample demographic characteristics

| Variable                              | Birth mothers | Birth fathers | Adoptive parent 1 | Adoptive parent 2 |
|---------------------------------------|---------------|---------------|-------------------|-------------------|
| Age at child birth, y, mean (SD)      | 24.12 (6.89)  | 25.43 (7.18)  | 37.78 (5.50)      | 38.14 (5.77)      |
| Race and/or ethnicity, %              |               |               |                   |                   |
| White                                 | 71.1          | 74.6          | 91.4              | 90.2              |
| African American                      | 11.4          | 8.7           | 3.6               | 5.0               |
| Hispanic or Latino                    | 6.7           | 8.7           | 2.5               | 1.7               |
| Multiethnic                           | 5.0           | 4.8           | 1.1               | 1.1               |
| Other                                 | 5.8           | 3.2           | 1.4               | 2.0               |
| Median education level                | High school   | High school   | 4-y college       | 4-y college       |
| Median annual income, $               | 25,001-40,000 | 25,001-40,000 | 100,001-150,000   | 100,001-150,000  |
| Employment, %                         |               |               |                   |                   |
| Full-time                             | 43.1          | 60.3          | 36.1              | 75.3              |
| Part-time                             | 16.1          | 7.9           | 18.9              | 2.0               |
| Unemployed but looking for work       | 12.8          | 11.9          | 0.8               | 0.9               |
| Full-time homemaker                   | 9.2           | 0.8           | 23.1              | 1.1               |
| Self-employed                         | 4.0           | 4.4           | 13.9              | 17.0              |
| Other                                 | 14.0          | 14.7          | 7.2               | 3.7               |
| Marital status, %                     |               |               |                   |                   |
| Single, never married                 | 33.6          | 34.1          | 0.8               | 0.3               |
| Married                               | 24.4          | 33.3          | 83.1              | 83.9              |
| Living in a committed relationship    | 28.3          | 26.2          | 2.2               | 1.1               |
| Other                                 | 13.7          | 6.4           | 13.9              | 14.7              |

[36x384]BMI. Height and weight data were extracted from the children’s medical records and from adoptive parent (P1) (usually the mother) reports at the age 4.5-, 6-, 7-, and 9-year assessments. Because the dates of height and weight measurements from the medical records and P1 reports often varied, we created 6-month age bands (e.g., 7 years ± 3 months) and averaged P1 and medical record reports within each age band. The 4.5-year assessment was supplemented with data from the 4-year age band if the data were missing at age 4.5 years. Likewise, the age 9 assessment was supplemented with the 9.5-year age band if data were missing at age 9 years. Raw BMI is recommended to track changes in BMI over time in children because within-person variability in BMI is unrelated to baseline adiposity (an issue for BMI \( z \) scores) (29). (Analyses using BMI \( z \) scores were highly consistent with findings using BMI raw scores; results are available upon author request).

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Analytic strategy
Longitudinal panel models that included a random intercept for BMI and sleep; autoregressive paths for BMI, sleep, and cortisol; and cross-lagged paths between BMI, sleep, and cortisol were fit to the data by using Mplus (Muthén & Muthén (32). Morning and evening cortisol were initially modeled with a random intercept (as done for BMI and sleep), but model convergence issues emerged because of relatively weak correlations over time within cortisol assessments ($r = 0.04-0.27$). Therefore, the relative stability in morning and evening cortisol was modeled only through autoregressive effects and not a random intercept.

Two sets of panel models (one that included morning cortisol and the second that included evening cortisol) were tested. The rationale for including random intercepts with the cross-lagged panel model approach stems from recent critiques of the applicability of cross-lagged panel models for drawing inferences about bidirectionality (22,23). This model delineates associations attributable to relative stability in constructs (random intercepts; between-person differences), preceding time-point variation within constructs (autoregressive paths), and preceding time-point variation across constructs (cross-lagged paths; within-person effects).

We assessed covariates using correlations (Supporting Information Table S1). Covariates found to be associated with study variables were included in the hypothesis-testing models and regressed on the random intercept for BMI and sleep and on the 4.5-year assessment of morning or evening cortisol. All models used full information maximum likelihood estimation with robust standard errors (SE), which uses all available data and a sandwich estimator (MLR specification in Mplus). This limits the bias attributable to listwise deletion of missing data and provides a more conservative estimate of statistical significance by accounting for any non-normality in the dependent variable distributions.

Results
Sample descriptive statistics for study variables are presented in Table 2.

Morning cortisol
The model that included morning cortisol, sleep duration, and BMI fit the data well ($\chi^2_{100} = 115.72; P = 0.13$; root-mean-square error of approximation [RMSEA] = 0.02; confirmatory fit index [CFI] = 0.96; Tucker-Lewis index [TLI] = 0.95; standardized root-mean-square residual [SRMR] = 0.06). Figure 1 depicts all significant paths; full results are presented in Supporting Information Table S2. There was strong but decreasing between-person stability in BMI, as indicated by decreasing standardized estimates of the loadings on the BMI random intercept. This between-person stability in BMI transitioned to significant autoregressive pathways in BMI at ages 6 to 7 years and at ages 7 to 9 years. There was consistently strong between-person stability in sleep duration over time, with few autoregressive paths. In addition, there was a strong association of the random intercepts such that shorter sleep duration was associated with higher BMI at the between-person level. There was some autoregressive stability in morning cortisol from ages 4.5 to 7.

Overall, there were very few significant cross-lagged paths (within-person, time-specific effects after accounting for the large between-person association of sleep and BMI). First, higher BMI at age 6 predicted increased sleep duration from ages 6 to 7 years. Second, lower morning cortisol predicted increased BMI from ages 7 to 9. No other concurrent or cross-lagged associations were significant. Two covariate effects were significant: higher BMI at age 2 and early growth predicted increased sleep duration from ages 6 to 7 years. First, higher BMI at age 2 and early growth predicted increased BMI from ages 7 to 9. No significant cross-lagged paths were observed. Second, higher BMI predicted increased sleep duration from ages 6 to 7 years. First, higher BMI at age 2 and early growth predicted increased sleep duration from ages 6 to 7 years. Second, lower morning cortisol predicted increased BMI from ages 7 to 9. No other concurrent or cross-lagged associations were significant. Two covariate effects were significant: higher BMI at age 2 and early growth predicted increased BMI from ages 6 to 7 years.

Evening cortisol
The model that included evening cortisol, sleep duration, and BMI also fit the data well ($\chi^2_{100} = 108.29; P = 0.29$; RMSEA = 0.01; CFI = 0.98; TLI = 0.97; SRMR = 0.06). Figure 2
depicts all significant paths; full results are presented in Supporting Information Table S3. Consistent with the prior model, there was between-person stability in BMI and in sleep duration over time and a strong inverse association between the sleep duration and BMI random intercepts. Covariate effects were identical to those of the morning cortisol model.

There was little stability in evening cortisol over time (e.g., no autoregressive paths were significant). There were few cross-lagged paths; consistent with the prior model, higher BMI at age 6 predicted increased sleep duration from ages 6 to 7 years. Additionally, higher BMI at age 6 predicted lower evening cortisol at age 7.

**Discussion**

The primary objective of this study was to rigorously examine the associations between sleep duration, BMI, and cortisol levels from early to late childhood. We initially hypothesized that HPA dysregulation would mediate associations between short sleep duration and higher BMI. Our analyses yielded four key findings: (1) sleep duration and BMI were moderately stable during childhood, whereas morning and evening cortisol levels were not; (2) sleep duration and BMI random intercepts were inversely associated at the between-person level, and neither was related to morning or evening cortisol; (3) higher BMI at age 6 years predicted longer sleep duration at age 7 years within individuals; and (4) BMI and cortisol levels were inversely related between ages 6 and 9 within individuals.

BMI, sleep, and cortisol stability

BMI showed moderate stability from ages 2 to 9 years, which could be related to genetic, prenatal, or early postnatal factors, such as rapid growth during early infancy (31). However, the BMI factor accounted for less of the total variance in BMI as children aged, and within-person autoregressive paths became more important. These findings suggest that between-person differences in BMI are important, especially in early childhood, but that BMI follows an iterative developmental trajectory during middle childhood: BMI is stable over shorter time periods during middle childhood compared with early childhood. Children undergo an adiposity rebound between approximately ages 4 and 7, resulting in accelerated increases in BMI (33). Children’s rank-order BMI can change during this period because of individual differences in the timing of the adiposity rebound (34), but they remain stable thereafter.
Sleep duration was moderately stable across childhood, similar to a previous longitudinal study that assessed children from ages 1 to 10 years and also found moderate intraindividual stability (35). This stability could be driven by a combination of biological and genetic factors (36,37) or stable environmental influences. However, morning and evening cortisol demonstrated little stability over time, to the extent that a random intercept could not be estimated, consistent with some prior research (38). There are few longitudinal studies of cortisol, and we were unable to find another study that used a similar panel model.

Between-person associations of sleep duration and BMI
Sleep duration and BMI were moderately and inversely associated between persons. Inverse associations between sleep duration and BMI within cross-sectional and longitudinal studies (which typically confound between-person differences and within-person changes) have been well established (1,5). The current study indicates that this association is a stable between-person phenomenon rather than an association that develops slowly over time via a canalizing process. This finding is a critical insight for the field because the majority of the literature guiding interpretation of inverse associations of sleep duration and BMI actually reflects this between-person association rather than the presumed within-person developmental paths often discussed.

Lastly, contrary to expectations, BMI and sleep factors were not related to children’s morning or evening cortisol levels at any age. This finding suggests that associations between childhood BMI and sleep duration are independent of diurnal HPA activity. Other biological processes may mediate associations between BMI and sleep duration. For example, some research has suggested that short sleep duration is associated with the hormones leptin and ghrelin, which are more directly related to hunger and satiation (7,8).

BMI at 6 years predicts longer sleep duration at 7 years
After accounting for sleep duration and BMI stability, we explored cross-lagged associations between sleep duration, BMI, and cortisol levels. Contrary to our expectations, a positive (albeit modest) cross-lagged path between BMI and sleep emerged: children with higher BMI at age 6 showed an increase in sleep duration from ages 6 to 7 years. We were able to find only one study that examined within-person associations of poor sleep phenotypes and BMI and found small...
or nonsignificant effects in 3- to 7-year-old children (39), providing some support of this observation. Because we included between-person effects (stability in sleep duration and BMI) and cross-lagged effects in the same model, the cross-lagged paths between sleep and BMI can be interpreted as time-specific within-person associations (23,24). Previous research has also found that within- and between-person effects do not necessarily occur in the same direction (24). Although the underlying explanation for a positive association between child BMI at age 6 and increased sleep duration at age 7 is unclear, there is some evidence of U-shaped associations between BMI and sleep duration in adolescents and middle-aged populations such that both shorter and longer sleep duration are associated with higher BMI (40,41). We may be picking up on a portion of this curve in middle childhood. Alternatively, longer reported sleep duration among children with higher BMI may also reflect more time in bed (rather than more or better sleep). Future research is needed to replicate these findings and explore these potential explanations.

BMI and cortisol levels are related between 6 and 9 years

Contrary to expectations, higher BMI at age 6 predicted decreased evening cortisol at age 7, and lower morning cortisol at age 7 predicted increased BMI at age 9, although morning and evening cortisol levels at age 7 were not correlated. This pattern suggests an evolving reciprocal relationship between cortisol and BMI during childhood, which may eventually contribute to lower cortisol levels and higher BMI. These findings are broadly consistent with research linking obesity with lower cortisol awakening response and increased cortisol clearance throughout the day among adults (17,18) and low cortisol levels in children (20,21). Additionally, two longitudinal studies conducted with older adults found that changes in BMI predicted subsequent decreases in morning cortisol over a period of years, although changes in cortisol did not predict changes in BMI (42,43). This association between obesity and low cortisol may reflect a bidirectional inverse relationship between HPA activity and adipocyte metabolic processes (19). Higher abdominal obesity is associated with increased conversion of inactive cortisone to cortisol within adipose tissue, which may lead to lower HPA cortisol production. Yet it is important to note that much of the previously discussed research focused on adult populations and that several studies have reported positive associations between child overweight and obesity and cortisol levels (16,21). Consequently, our results suggest reciprocal associations between HPA functioning and children’s BMI, although further research is needed.

Strengths and limitations

Although bivariate associations between these constructs have been explored previously, the associations between all three have remained largely unexamined. Our longitudinal design enabled us to assess the associations between all three constructs across childhood and differentiate between- and within-person effects to examine both stable and time-specific associations. Although we conducted a rigorous analysis of a unique sample to test a mediation hypothesis based on a strong body of literature, there were also limitations. We had large amounts of missing data. Although we accommodated these missing data using full information maximum likelihood, inferences would be strengthened by utilizing a sample with more complete data or a larger sample size. The measurement of cortisol was limited to home-based collection, which is subject to more measurement error compared with tightly controlled laboratory settings. We also did not have a measure of cortisol at age 9 years, restricting the number of time points that cortisol could contribute to the panel model. Subjective estimates of sleep are subject to reporting bias. In the future, better measures of sleep, including information about nap time sleep and systematically assessing weekend versus weekday sleep, are critical. Our measure of BMI is not ideal and includes error related to how different practices and/or doctors and parents measure height and weight. Measures of adiposity would better index pathophysiology in future studies. Although we included many potentially relevant confounders, because of data constraints, we were unable to adequately assess diet, physical activity, and seasonality as covariates. Finally, although these models improve upon cross-sectional and unidirectional longitudinal studies, findings should not be interpreted in terms of causal inferences.

Conclusion

Based on previous research, we hypothesized that higher morning and evening cortisol levels would link short sleep duration and higher childhood BMI. Our findings indicate that there is a stable, inverse, between-person association between sleep duration and BMI across childhood. However, there may also be time-specific associations in the opposite direction; namely, at some ages, BMI and sleep duration may be positively related, and BMI may affect sleep rather than the reverse. Contrary to our expectations, HPA activity did not mediate associations between sleep duration and BMI across childhood. However, we found evidence of time-specific associations between low cortisol levels and higher BMI. These latter findings suggest an emergent association between low cortisol and child BMI during middle childhood that may parallel recent research on BMI change and lower cortisol levels in adults (42).

Future research should continue to examine the factors that predict between-person differences in sleep duration and BMI that manifest early in life, with interventions needed for the most convincing causal evidence for associations between BMI and sleep. More longitudinal work that separates between-person differences from the within-person processes in directional associations between BMI and sleep is also needed. This research should include larger samples, multiple measures, hypothesized predictors and mediators, and differing time lags between observations to better understand the development and consequences of associations of sleep and BMI.

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