HHS Public Access

Author manuscript

Obesity (Silver Spring). Author manuscript; available in PMC 2019 August 28.

Published in final edited form as:

Obesity (Silver Spring). 2019 April ; 27(4): 645–652. doi:10.1002/oby.22420.

Longitudinal associations of sleep duration, morning and evening cortisol, and body mass index during childhood

Kristine Marceau, Ph.D.[1], **Emily A. Abel**[1], **Robert J. Duncan, Ph.D.**[1], **Phillip J. Moore, Ph.D.**[2], **Leslie D. Leve, Ph.D.**[3], **David Reiss, M.D.**[4], **Daniel S. Shaw, Ph.D.**[5], **Misaki Natsuaki, Ph.D.**[6], **Jenae M. Neiderhiser, Ph.D.**[7], and **Jody M. Ganiban, Ph.D.**[2]

[1]Department of Human Development and Family Studies, Purdue University, West Lafayette, IN, USA

[2]Department of Psychology, George Washington University, Washington DC, USA

[3]Department of Counseling Psychology and Human Services, University of Oregon, Eugene, OR, USA

[4]Child Study Center, Yale University, New Haven, CT, USA

[5]Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA

[6]Department of Psychology, University of California, Riverside, Riverside, CA, USA

[7]Department of Psychology, Penn State University, University Park, PA, USA

Abstract

Objective.—The primary objective of this paper was to examine associations between sleep duration, body mass index (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- and bi-directional within-person associations of sleep duration, BMI, and morning and evening cortisol from 4.5 to 9 years of age.

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 6 years predicted longer sleep duration and lower evening cortisol at 7 years, and lower morning cortisol at 7 years predicted higher BMI at 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.

Keywords

sleep duration; morning cortisol; evening cortisol; BMI; adoption

[*]**CONTACT INFO**: Address correspondence to Kristine Marceau, KristineMarceau@purdue.edu, tel: 765-494-9410, 225 Hanley Hall, Purdue University, 1202 West State St., West Lafayette, IN 47907, or to Jody Ganiban, Ganiban@gwu.edu, tel: 202-994-7571, 304 Building GG, George Washington University, 2125 G St NW, Washington, District of Columbia 20052.

DISCLOSURE: The authors declared no conflict of interest.

Introduction

The risk for being overweight or obese 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, 2, 3). Recent longitudinal studies also suggest that short sleep duration predicts higher body mass indexes (BMI) during childhood (4, 5, 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 still 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 start to show diurnal cortisol patterns that are similar to 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 childhood because the HPA axis undergoes change during this period and thus may be particularly influenced by individual differences in behavior.

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 amongst 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, 15, 16). These studies suggest that HPA dysregulation and sleep duration are associated, but the direction of effects are not clear and warrant further investigation.

Obesity also is associated with HPA dysregulation, but the type of dysregulation is not clear and the direction of effects is also ambiguous. A number of 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 report 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 who are obese 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 overweight and obese children had lower morning and evening cortisol levels (20, 21).

Present study

The current, longitudinal study examined whether associations between sleep duration and BMI (from age 4.5 to 9 years) are accounted for by HPA regulation during childhood. We explored whether shorter sleep duration predicted higher levels of cortisol, which, in turn predicted 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 explore 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. Thus, we modeled random intercepts and their associations to account for between-person stability for each construct, attenuate the upward bias of cross-lagged and autoregressive estimates, and clarify the interpretation of these paths as time-specific, within-person associations (22, 23, 24).

Method

Participants

Participants included birth parents, adoptive parents, and adopted children from Cohort I (*N*=361 families, 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 US, 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 (*M*=7.11 days, *SD*=13.28, *Median*=2 days), the adopted child had no major medical condition, and the birth mothers and adoptive parents could read or understand English at an eighth-grade level. The research was approved by the Institutional Review Boards (IRBs) of all participating organizations (George Washington University, The 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 above IRBs (see Appendix 1). See Table 1 for demographic characteristics.

Families in Cohort I participated in a series of in-person and telephone interviews (for full study procedures, see (25)). For the current study, adopted children were assessed at 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 three days at home near the time of the 4.5 (n=211), 6 (n=212), and 7 (n=203) year-old visits. Additionally, height and weight were assessed during a phone interview at 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 were assessed using average morning and evening levels across the three days of assessment at age 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 three days. Samples were stored by participants and then mailed to the primary study site and frozen until shipped to the University of Trier Laboratory and stored frozen (at −20C) until assayed in duplicate via DELFIA immunoassay (26). Average usable values across duplicate assays were used as the level of cortisol in that sample (27). Raw Mean (SD) morning values (μg/dL) across the three days for each assessment ranged from, respectively, .60 (.34) to .64 (.34) at age 4.5 years, .49 (.28) to .52 (.28) at age 6, and . 48 (.30) to .52 (.30) at age 7 years. Average evening values ranged from .06 (.09) to .08 (.12) at age 4.5 years, .07 (.12) to .10 (.24) at age 6, and .06 (.09) to .09 (.20) at age 7 years.

Cortisol data were cleaned as follows: outliers over 3SD above the sample mean were windsorized (replaced with 3SD values, <3.02% of any assessment), and any values that were below the detection limit were set to missing (two to 11 values at each assessments). 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 three days of collection were averaged, and then log transformed to normalize distributions.

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 day 1 and 2, and 2 and 3 of the cortisol collection diary at age 4.5, 6, and 7 years. At age 9 years, average sleep duration was calculated from three nights on a daily reports measure (28).

BMI.—Height and weight data were extracted from the children's medical records and from parent (P1, usually mother) reports at EGDS age 4.5, 6, 7, and 9-year assessments. Since 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 report and medical records 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 4.5 years. Likewise, the age 9 assessment was supplemented with the 9.5-year age band if data were missing at 9 years.[1] Raw BMI is recommended to track changes in BMI over time in children as within-person variability in BMI is unrelated to baseline adiposity (an issue for BMI z-scores) (29).

[1]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 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 CDC 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 socio-economic status than adoptive parents. Prenatal influences included pregnancy complications, and weight gain assessed through medical record report—and if prenatal care visits began after 12 weeks gestation—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: *sleep:* age 4.5: 43%; age 6: 43%; age 7: 50%; age 9: 31%; *BMI:* age 4.5: 34%; age 6: 48%; age 7: 42%; age 9: 39%; *morning cortisol;* age 4.5: 43%; age 6: 45%; age 7: 45%; *evening cortisol:* age 4.5: 43%; age 6: 45%; age 7: 45%. 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/ethnicity and age at placement) and study variables/ covariates contributed to participation (yes/no) at the 4.5-year assessment, and whether participants dropped out (yes/no) between the 4.5 and 9-year assessments using a series of Kruskal-Wallace one-way analysis of variance tests. Of 56 tests, four (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 4.5 years but not 9 years experienced more pregnancy complications, were older at the 4.5-year assessment, and had longer age 6 sleep duration. The final analytic sample size was 316 ($n=45$ were missing on all study variables).

Analytic strategy

Longitudinal panel models including a random intercept for BMI and sleep,[2] autoregressive paths for BMI, sleep, and cortisol, and cross-lagged paths between BMI, sleep, and cortisol were fit to the data using Mplus (32). Two sets of panel models—one including morning cortisol, and the second including 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 bi-directionality (22, 23). This model delineates associations attributable to relative stability in constructs (random intercepts, between-person differences), preceding time-point variation

[2]Morning and evening cortisol were initially modeled with a random intercept (as done for BMI and sleep), but model convergence issues emerged due to relatively weak correlations over time within cortisol assessments (r's = .04-.27). Therefore, the relative stability in morning and evening cortisol was only modeled through autoregressive effects and not a random intercept.

within constructs (autoregressive paths), and preceding time-point variation across constructs (cross-lagged paths, within-person effects).

We assessed covariates using correlations (see Table S1). Covariates found to be associated with study variables were included in the hypothesis-testing models, 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, which uses all available data and a sandwich estimator (MLR specification). 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 including morning cortisol, sleep duration, and BMI fit the data well, $\chi^2(100)=115.72$, $p=.13$; RMSEA=.02, CFI=.96, TLI=.95, SRMR=.06. Figure 1 depicts all significant paths; full results are presented in Table 3. 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 auto-regressive pathways in BMI from 6 to 7 and 7 to 9 years. There was consistently strong between-person stability in sleep duration over time, with few autoregressive paths. Further, 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 4.5 to 7 years.

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 6 to 7 years. Second, lower morning cortisol predicted increased BMI from 7 to 9 years. No other concurrent or cross-lagged associations were significant. Two covariate effects were significant: higher BMI at age 2 and early growth predicted the BMI random intercept.

Evening cortisol

The model including evening cortisol, sleep duration, and BMI also fit the data well, $\chi^2(101)=108.29$, $p=.29$; RMSEA=.01, CFI=.98, TLI=.97, SRMR=.06. Figure 2 depicts all significant paths; full results are presented in Table 4. 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 the morning cortisol model.

There was little stability in evening cortisol over time (e.g., no auto-regressive paths were significant). There were few cross-lagged paths: consistent with the prior model, higher BMI

at age 6 predicted increased sleep duration from 6 to 7 years. Additionally, higher BMI at age 6 predicted lower evening cortisol at 7 years.

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 BMIs. 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 6 years predicted *longer* sleep duration at 7 years within individuals; and 4) BMI and cortisol levels were inversely related between 6 and 9 years within individuals.

BMI, sleep, and cortisol stability

BMI showed moderate stability from age 2 through 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 suggests 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 to early childhood. Children undergo an adiposity rebound between approximately ages 4 and 7, resulting in accelerated increases in BMIs (33). Children's rank-order BMI can change during this period because of individual differences in the timing of the adiposity rebound (34), but remain stable thereafter.

Sleep duration was moderately stable across childhood, similar to a previous longitudinal study that assessed children from age 1 to 10 years, which also found moderate intra-individual 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) are 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, as the majority of the literature guiding interpretation of inverse associations of sleep duration and BMI actually reflect this

between-person association rather than the presumed within-person developmental paths often discussed.

Last, 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 suggests 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 BMIs at age 6 showed an increase in sleep duration from age 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 non-significant 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 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 BMIs (40, 41). We may be picking up on a portion of this curve in middle childhood. Or, longer reported sleep duration among children with higher BMIs 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 recent longitudinal studies conducted with older adults have found that changes in BMIs 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/obesity and cortisol levels (16, 21). Consequently, our results suggest reciprocal associations between HPA functioning and children's BMIs, 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 this 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 than 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 naptime sleep and systematically assessing weekend versus weekday sleep is critical. Our measure of BMI is not ideal, and includes error related to how different practices/doctors and parents measure height and weight. Measures of adiposity would better index pathophysiology in future studies. Although we included many potentially relevant confounders, due to 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.

Conclusions

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. But, 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 BMIs. 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.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

FUNDING: This project was supported by grant R01 HD042608 from the Eunice Kennedy Shriver National Institute of Child Health & Human Development and the National Institute on Drug Abuse, NIH, U.S. PHS (PI Years 1–5: David Reiss, MD; PI Years 6–10: Leslie Leve, PhD). This project was also supported by grant R01 MH092118 from the National Institute of Mental Health, NIH, U.S. PHS (PIs: Jenae Neiderhiser, Ph.D. and Leslie Leve, Ph.D.), grant R01 DK090264 from the National Institute of Diabetes and Digestive and Kidney Disease, NIH, U.S. PHS (PI: Jody Ganiban, Ph.D.), grant R01 DA035062 from the National Institute on Drug Abuse, NIH, U.S. PHS (PI: Leslie Leve, Ph.D.), and UG3 OD023389 from the Office of the Director (PIs: Leslie Leve, Ph.D., Jenae Neiderhiser, Ph.D., and Jody Ganiban, Ph.D.). Additional support was provided by the National Institute on Drug Abuse K01 DA039288 (Marceau). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of Child Health & Human Development or the National Institutes of Health.

Appendix 1.

Additional details on consent process and procedures for obtaining height and weight data

Consent.

For this study, we obtained parental consent. Child assent was obtained at age 7 years. We did not, however, request child assent prior to or after age 7 years. Children at and before the 6-year-old assessment were considered to be too young to provide assent. At the 9 year assessment, children were not interviewed directly. Rather, parents provided information about their children. All study procedures, including consent procedures, were reviewed and approved by the GWU Institutional Review Board (IRB). Additional details on consenting procedures can be found in Leve et al., 2013 (25).

Medical Record Procedures.

Prior to accessing children's medical records, the Investigators obtained signed medical record releases from the children's parents. The medical releases asked parents to provide the contact information for their children's medical care providers since birth. The research staff contacted the providers and requested copies of the children's complete medical records. Trained coders reviewed each medical record, and recorded the date of each office visit and every report of the child's height and weight. Height and weight data were collected from well and sick visits.

Height and weight data cleaning.

All implausible values were dropped from the final dataset including: decreases in height over time, extremely low or high weights and heights that deviated substantially from a child's previous or subsequent measurements, and errors associated with unit conversions (n = 260 observations across all assessments, out of 12,964 individual datapoints; 101 were detected in medical records and 159 were detected in parent reports). In addition, several children had duplicate height and weight assessments in their records. Consequently, all assessments that occurred within two months of the target age were averaged. Across all age bands, there were 468 instances in which we had parent and medical record reports of weight, and 399 instances in which we had parent and medical record reports of height. Correlations between parent report and medical record abstraction within each age band were computed. Across all age bands, the average correlation for weight was .92, n = 468 (range across specific age-bands = .88-.96, *n's* ranged from 47-121), and the average correlation for height was .74, n = 399 (range across specific age-bands = .57-.88, *n's* ranged from 36-107). These correlations support combining parent and medical record reports.

Z scored data.

	Height		Weight		BMI	
Age	**Mean**	**Std Dev**	**Mean**	**Std Dev**	**Mean**	**Std Dev**
2 years	0.46	1.19	0.21	1.13	0.03	1.32
4.5 years	0.10	1.63	0.44	1.11	0.63	1.46
6 years	0.28	1.66	0.31	1.13	0.21	1.55
7 years	0.36	1.40	0.30	1.13	0.07	1.36
9 years	0.14	1.32	0.21	1.18	0.12	1.38

Data were z-scored according to the CDC 2000 Growth Charts.

References

1. Cappuccio FP, Taggart FM, Kandala N-B, Currie A, Peile E, Stranges S, et al. Meta-analysis of short sleep duration and obesity in children and adults. Sleep 2008;31: 619–626. [PubMed: 18517032]
2. Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. Obesity 2008;16: 265–274. [PubMed: 18239632]
3. Fatima Y, Doi S, Mamun A. Longitudinal impact of sleep on overweight and obesity in children and adolescents: a systematic review and bias-adjusted meta-analysis. Obesity Reviews 2015;16: 137–149. [PubMed: 25589359]
4. Li L, Zhang S, Huang Y, Chen K. Sleep duration and obesity in children: A systematic review and meta-analysis of prospective cohort studies. Journal of paediatrics and child health 2017;53: 378–385. [PubMed: 28073179]
5. Taveras EM, Gillman MW, Peña M-M, Redline S, Rifas-Shiman SL. Chronic sleep curtailment and adiposity. Pediatrics 2014: peds. 2013–3065.
6. Ruan H, Xun P, Cai W, He K, Tang Q. Habitual sleep duration and risk of childhood obesity: systematic review and dose-response meta-analysis of prospective cohort studies. Scientific reports 2015;5: 16160. [PubMed: 26537869]
7. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Annals of internal medicine 2004;141: 846–850. [PubMed: 15583226]

8. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS medicine 2004;1: e62. [PubMed: 15602591]

9. Hostinar CE, Sullivan RM, Gunnar MR. Psychobiological mechanisms underlying the social buffering of the hypothalamic-pituitary-adrenocortical axis: A review of animal models and human studies across development. Psychological bulletin 2014;140: 256. [PubMed: 23607429]

10. Gunnar MR, Doom JR, Esposito EA. Psychoneuroendocrinology of stress: Normative development and individual differences. 2015.

11. Fernandez-Mendoza J, Vgontzas AN, Calhoun SL, Vgontzas A, Tsaoussoglou M, Gaines J, et al. Insomnia symptoms, objective sleep duration and hypothalamic-pituitary-adrenal activity in children. European journal of clinical investigation 2014;44: 493–500. [PubMed: 24635035]

12. Hatzinger M, Brand S, Perren S, Stadelmann S, von Wyl A, von Klitzing K, et al. Sleep actigraphy pattern and behavioral/emotional difficulties in kindergarten children: association with hypothalamic-pituitary-adrenocortical (HPA) activity. Journal of psychiatric research 2010;44: 253–261. [PubMed: 19762039]

13. Leproult R, Copinschi G, Buxton O, Van Cauter E. Sleep loss results in an elevation of cortisol levels the next evening. Sleep 1997;20: 865–870. [PubMed: 9415946]

14. Noppe G, van den Akker ELT, de Rijke YB, Koper JW, Jaddoe VW, van Rossum EFC. Long-term glucocorticoid concentrations as a risk factor for childhood obesity and adverse body-fat distribution. International Journal Of Obesity 2016;40: 1503. [PubMed: 27339603]

15. Raikkonen K, Matthews KA, Pesonen A-K, Pyhala R, Paavonen EJ, Feldt K, et al. Poor sleep and altered hypothalamic-pituitary-adrenocortical and sympatho-adrenal-medullary system activity in children. The Journal of Clinical Endocrinology & Metabolism 2010;95: 2254–2261. [PubMed: 20194713]

16. Saridjan NS, Kocevska D, Luijk MP, Jaddoe VW, Verhulst FC, Tiemeier H. The Prospective Association of the Diurnal Cortisol Rhythm With Sleep Duration and Perceived Sleeping Problems in Preschoolers: The Generation R Study. Psychosomatic medicine 2017;79: 557–564. [PubMed: 28570434]

17. Rodriguez ACI, Epel ES, White ML, Standen EC, Seckl JR, Tomiyama AJ. Hypothalamic-pituitary-adrenal axis dysregulation and cortisol activity in obesity: a systematic review. Psychoneuroendocrinology 2015;62: 301–318. [PubMed: 26356039]

18. Roelfsema F, Pereira AM, Veldhuis JD. Impact of Adiposity and Fat Distribution on the Dynamics of Adrenocorticotropin and Cortisol Rhythms. Current Obesity Reports 2014;3: 387–395. [PubMed: 26626915]

19. Chu L, Sheng K, Liu P, Ye K, Wang Y, Li C, et al. Increased Cortisol and Cortisone Levels in Overweight Children. Medical Science Monitor Basic Research 2017;23: 25–30. [PubMed: 28179618]

20. Kjolhede EA, Gustafsson P, Gustafsson P, Nelson N. Overweight and obese children have lower cortisol levels than normal weight children. Acta Paediatrica 2014;103: 295–299. [PubMed: 24205857]

21. Lumeng JC, Miller A, Peterson KE, Kaciroti N, Sturza J, Rosenblum K, et al. Diurnal cortisol pattern, eating behaviors and overweight in low-income preschool-aged children. Appetite 2014;73: 65–72. [PubMed: 24177439]

22. Berry D, Willoughby MT. On the practical interpretability of cross-lagged panel models: Rethinking a developmental workhorse. Child development 2017;88: 1186–1206. [PubMed: 27878996]

23. Hamaker EL, Kuiper RM, Grasman RP. A critique of the cross-lagged panel model. Psychological methods 2015;20: 102. [PubMed: 25822208]

24. Prenoveau JM. Specifying and interpreting latent state–trait models with autoregression: An illustration. Structural Equation Modeling: A Multidisciplinary Journal 2016;23: 731–749.

25. Leve LD, Neiderhiser JM, Shaw DS, Ganiban J, Natsuaki MN, Reiss D. The Early Growth and Development Study: a prospective adoption study from birth through middle childhood. Twin research and human genetics : the official journal of the International Society for Twin Studies 2013;16: 412–423. [PubMed: 23218244]

26. Dressendorfer RA, Kirschbaum C, Rohde W, Stahl F, Strasburger CJ. Synthesis of a cortisol-biotin conjugate and evaluation as a tracer in an immunoassay for salivary cortisol measurement. The Journal of steroid biochemistry and molecular biology 1992;43: 683–692. [PubMed: 1472460]
27. Laurent HK, Leve LD, Neiderhiser JM, Natsuaki MN, Shaw DS, Harold GT, et al. Effects of Prenatal and Postnatal Parent Depressive Symptoms on Adopted Child HPA Regulation: Independent and Moderated Influences. Developmental Psychology 2012: No Pagination Specified.
28. Chamberlain P, Reid JB. Parent observation and report of child symptoms. Behavioral Assessment 1987;9: 97–109.
29. Cole TJ, Faith MS, Pietrobelli A, Heo M. What is the best measure of adiposity change in growing children: BMI, BMI%, BMI z-score or BMI centile?. European journal of clinical nutrition 2005;59: 419. [PubMed: 15674315]
30. Marceau K, De Arujo-Greecher M, Miller ES, Massey SH, Mayes LC, Ganiban JM, et al. The Perinatal Risk Index: Early risks experienced by domestic adoptees in the united states. PloS one 2016.
31. Woo Baidal JA, Locks LM, Cheng ER, Blake-Lamb TL, Perkins ME, Taveras EM. Risk Factors for Childhood Obesity in the First 1,000 Days. American Journal of Preventive Medicine 2016;50: 761–779. [PubMed: 26916261]
32. Muthén LK, Muthén BO. Mplus User's Guide: Statistical Analysis with Latent Variables: User'ss Guide. Muthén & Muthén, 2010.
33. Rolland-Cachera MF, Deheeger M, Maillot M, Bellisle F. Early adiposity rebound: causes and consequences for obesity in children and adults. International Journal Of Obesity 2006;30: S11. [PubMed: 17133230]
34. Koyama S, Ichikawa G, Kojima M, Shimura N, Sairenchi T, Arisaka O. Adiposity Rebound and the Development of Metabolic Syndrome. Pediatrics 2014;133: e114–e119. [PubMed: 24366997]
35. Jenni OG, Molinari L, Caflisch JA, Largo RH. Sleep Duration From Ages 1 to 10 Years: Variability and Stability in Comparison With Growth. Pediatrics 2007;120: e769–e776. [PubMed: 17908734]
36. Saper CB, Scammell TE, Lu J. Hypothalamic regulation of sleep and circadian rhythms. Nature 2005;437: 1257. [PubMed: 16251950]
37. Allebrandt KV, Teder-Laving M, Akyol M, Pichler I, Müller-Myhsok B, Pramstaller P, et al. CLOCK gene variants associate with sleep duration in two independent populations. Biological psychiatry 2010;67: 1040–1047. [PubMed: 20149345]
38. Ross KM, Murphy ML, Adam EK, Chen E, Miller GE. How stable are diurnal cortisol activity indices in healthy individuals? Evidence from three multi-wave studies. Psychoneuroendocrinology 2014;39: 184–193. [PubMed: 24119668]
39. Carter PJ, Taylor BJ, Williams SM, Taylor RW. Longitudinal analysis of sleep in relation to BMI and body fat in children: the FLAME study. BMJ (Clinical research ed) 2011;342.
40. Grandner MA, Schopfer EA, Sands-Lincoln M, Jackson N, Malhotra A. Relationship between sleep duration and body mass index depends on age. Obesity 2015;23: 2491–2498. [PubMed: 26727118]
41. Wu J, Wu H, Wang J, Guo L, Deng X, Lu C. Associations between Sleep Duration and Overweight/Obesity: Results from 66,817 Chinese Adolescents. Scientific Reports 2015;5: 16686. [PubMed: 26568253]
42. Joseph JJ, Wang X, Diez Roux AV, Sanchez BN, Seeman TE, Needham BL, et al. Antecedent longitudinal changes in body mass index are associated with diurnal cortisol curve features: The multi-ethnic study of atherosclerosis. Metabolism - Clinical and Experimental 2017;68: 95–107. [PubMed: 28183457]
43. Travison TG, O'Donnell AB, Araujo AB, Matsumoto AM, McKinlay JB. Cortisol levels and measures of body composition in middle-aged and older men. Clinical endocrinology 2007;67: 71–77. [PubMed: 17466009]

Key Questions:

What is already known about this subject?

- There is a well-established association between shorter sleep duration and BMI.

What does your study add?

- We show that this association between shorter sleep and BMI occurs in children at the trait level, but not that a child who sleeps less develops higher BMI in later years or vice versa.
- Longer sleep duration may predict higher BMI at specific times during middle childhood.
- Cortisol does not appear to mediate associations between sleep duration and BMI, but higher BMI is associated with lower cortisol levels between ages 6 and 9 years.

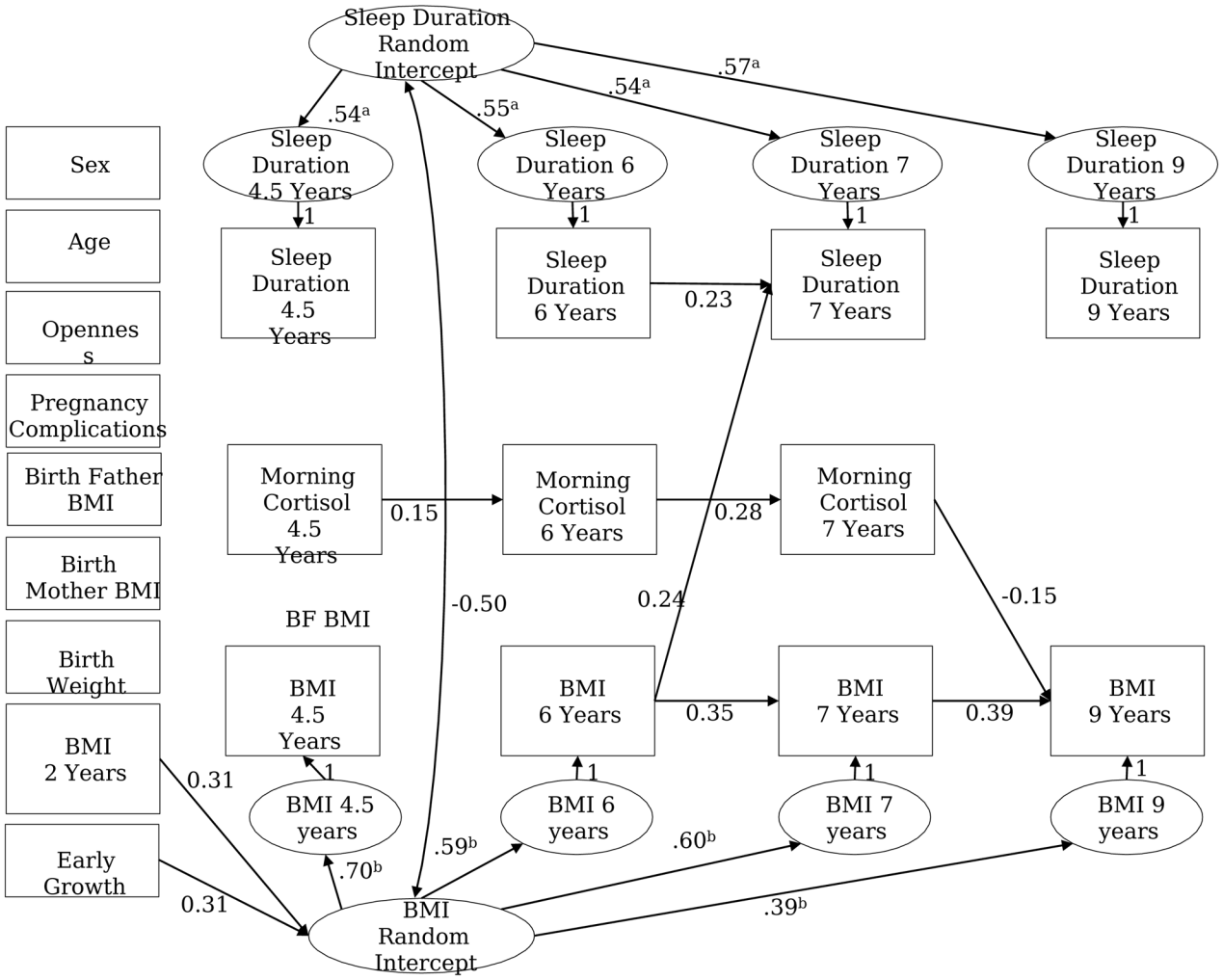

Figure 1. Standardized estimates for statistically significant paths: Morning Cortisol

Standardized path estimates are presented. All paths denoted by [a] are constrained to equality (in the unstandardized estimates). All paths denoted by [b] are constrained to equality (in the unstandardized estimates). Significant associations among covariates are excluded from the Figure (see Table 3).

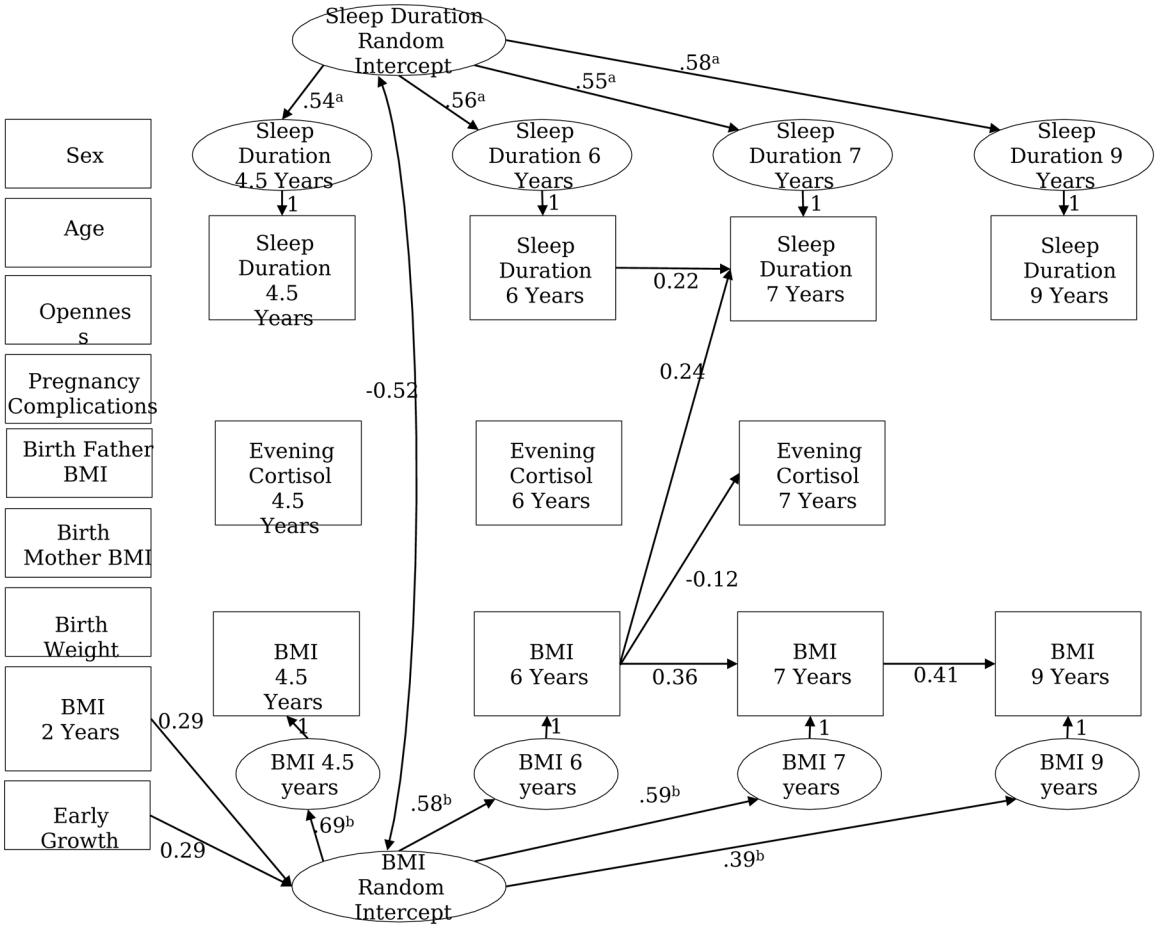

Figure 2. Standardized estimates for statistically significant paths: Evening Cortisol

Standardized path estimates are presented. All paths denoted by [a] are constrained to equality (in the unstandardized estimates). All paths denoted by [b] are constrained to equality (in the unstandardized estimates). Significant associations among covariates are excluded from the Figure (see Table 4).

Table 1.

Sample Demographic Characteristics

Variable	Birth Mothers	Birth Fathers	Adoptive Parent 1	Adoptive Parent 2
Age at child birth	24.12 (5.89)	25.43 (7.18)	37.78 (5.50)	38.14 (5.77)
Race/Ethnicity				
Caucasian	71.1%	74.6%	91.4%	90.2%
African-American	11.4%	8.7%	3.6%	5.0%
Hispanic/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-year college	4-year college
Median Annual Income	$25,001-40,000	$25,001-40,000	$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%

Table 2.

Study Variables Descriptive Statistics

		N	Mean	SD
BMI	Age 2	247	16.66	1.68
	Age 4	239	16.67	2.25
	Age 6	186	16.24	2.22
	Age 7	172	16.18	2.07
	Age 9	217	17.49	3.30
Sleep Duration, hours	Age 4	207	10.60	0.73
	Age 6	205	10.62	0.70
	Age 7	180	10.43	0.73
	Age 9	249	10.17	0.68
Morning Cortisol, μg/dL	Age 4	204	0.63	0.22
	Age 6	200	0.50	0.19
	Age 7	197	0.50	0.21
Evening Cortisol, μg/dL	Age 4	205	0.07	0.06
	Age 6	200	0.07	0.08
	Age 7	197	0.06	0.06
Potential Covariates	Age at 4 year assessment	297	4.62	0.16
	Adoption Openness (Age 4)	259	0.01	0.96
	Birth Weight	323	3.28	0.51
	Early Growth	319	0.59	1.12
	Pregnancy Complications	351	5.23	3.66
	Obstetric Complications	351	9.87	6.76
	Birth Mother BMI	360	28.01	6.46
	Birth Father BMI	124	26.46	4.72
	Birth Mother Pre-pregnancy BMI	335	25.06	6.15
	Pregnancy Weight Gain	332	33.82	14.85

Note. All BMI values reflect raw body mass index values. Sleep is assessed in hours per night. Cortisol values are presented after cleaning and averaging across days, but before log transforming: units = μg/dL. Age is assessed in years. Adoption openness is on a scale of 1 (very closed) to 7 (very open). Birth weight is assessed in *kg*. Early growth is a z-score for weight gain between birth and either 6 or 9 months of age. Pregnancy and obstetric complications are weighted sum scores of multiple complications based on the presumed severity of effects on the fetus; see Marceau et al., (2016) for details. Higher numbers indicate more, and more severe complications. Birth mother and Father BMI is averaged across 6 assessments from 4 months to 9 years post-partum. Pregnancy weight gain is assessed in *lbs*.

Table 3.

Unstandardized Path Estimates for the Full Model including Morning Cortisol

Random Intercept Loadings	BMI random intercept	Sleep random intercept
Age 4	1.10 *** (0.18)	0.36 *** (0.05)
Age 6	1.10 *** (0.18)	0.36 *** (0.05)
Age 7	1.10 *** (0.18)	0.36 *** (0.05)
Age 9	1.10 *** (0.18)	0.36 *** (0.05)
Association of Random Intercepts		
Sleep Random Intercept	−0.50 ** (0.16)	

Cross-lagged and autoregressive paths

	Age 6			Age 7			Age 9	
	BMI	**Sleep**	**Cortisol**	**BMI**	**Sleep**	**Cortisol**	**BMI**	**Sleep**
BMI (T-1)	0.10 (0.17)	0.01 (0.04)	0.01 (0.03)	0.34 *** (0.09)	0.08 ** (0.03)	−0.01 (0.01)	0.60 *** (0.10)	0.04 (0.03)
Sleep (T-1)	0.09 (0.29)	0.19 + (0.29)	0.002 (0.04)	0.32 (0.22)	0.23 * (0.09)	0.05 (0.03)	−0.21 (0.29)	0.05 (0.11)
Cortisol (T-1)	−0.08 (0.61)	0.12 (0.12)	0.13 * (0.06)	−0.39 (0.51)	0.07 (0.14)	0.31 *** (0.08)	−1.34 * (0.48)	−0.01 (0.14)

Covariate Effects

	BMI random intercept	Sleep random intercept	Age 4 Cortisol	Sex	Age	Openness	Birth Weight	Early Growth	Pregnancy Complications	BMI 2 Years	BM BMI
Sex	0.17 (0.16)	0.18 (0.17)	0.04 (0.06)	1							
Age	0.86 (0.66)	−0.93 (0.65)	−0.22 (0.25)	−0.010 * (0.004)	1						
Openness	−0.03 (0.10)	0.11 (0.10)	0.03 (0.03)	−0.03 (0.03)	−0.03 ** (0.01)	1					
Birth Weight	0.35 + (0.18)	0.16 (0.20)	0.01 (0.06)	−0.02 (0.01)	−0.001 (0.004)	0.04 (0.03)	1				
Early Growth	0.30 ** (0.09)	0.01 (0.10)	0.02 (0.04)	−0.01 (0.03)	0.002 (0.01)	−0.08 (0.06)	−0.29 *** (0.04)	1			
Pregnancy Complications	0.01 (0.03)	0.01 (0.03)	0.01 (0.01)	0.002 (0.10)	0.07 * (0.03)	−0.14 (0.22)	−0.04 (0.11)	0.38 (0.24)	1		
BMI 2 Years	0.23 * (0.10)	−0.08 (0.07)	−0.02 (0.03)	−0.14 ** (0.05)	0.02 (0.01)	−0.13 (0.14)	0.04 (0.06)	0.61 *** (0.14)	1.60 *** (0.39)	1	
BM BMI	0.03 + (0.01)	−0.02 (0.01)	0.01 (0.01)	−0.30 + (0.17)	−0.01 (0.05)	−0.70 + (0.40)	0.61 ** (0.20)	−0.50 (0.40)	2.32 + (1.23)	1.65 * (0.56)	1

Random Intercept Loadings	BMI random intercept	Sleep random intercept									
BF BMI	−0.003 (0.03)	−0.04 (0.03)	0.01 (0.01)	−0.12 (0.20)	−0.09 (0.08)	0.21 (0.41)	−0.08 (0.20)	0.41 (0.36)	1.24 (1.63)	1.08 (0.69)	7.60 *(3.41)

Note. Values in table refer to unstandardized β, followed by (SE).

$^{+}p < .10$

$^{*}p < .05$

$^{**}p < .01$

$^{***}p < .001$

Table 4.

Unstandardized Path Estimates for the Full Model including Evening Cortisol

Random Intercept Loadings	BMI random intercept	Sleeprandom intercept
Age 4	1.07 *** (0.18)	0.37 *** (.05)
Age 6	1.07 *** (0.18)	0.37 *** (.05)
Age 7	1.07 *** (0.18)	0.37 *** (.05)
Age 9	1.07 *** (0.18)	0.37 *** (.05)
Association of Random Intercepts		
Sleep Random Intercept	−0.52 ** (0.17)	

Cross-lagged and autoregressive paths

	Age 6			Age 7			Age 9	
	BMI	**Sleep**	**Cortisol**	**BMI**	**Sleep**	**Cortisol**	**BMI**	**Sleep**
BMI (T-1)	0.13 (0.16)	0.02 (0.04)	0.003 (0.01)	0.36 *** (0.097)	0.08 ** (0.03)	−0.01 * (0.003)	0.63 *** (0.11)	0.04 + (0.03)
Sleep (T-1)	0.17 (0.28)	0.19 + (0.10)	0.02 (0.02)	0.38 + (0.22)	0.233 * (0.09)	−0.02 (0.01)	−0.16 (0.30)	0.04 (0.11)
Cortisol (T-1)	−2.48 + (1.33)	−0.05 (0.31)	0.04 (0.11)	0.95 (0.72)	−0.18 (0.30)	0.06 (0.06)	−0.52 (1.53)	0.51 (0.48)

Covariate Effects

	BMI random intercept	Sleep random intercept	Age 4 Cortisol	Sex	Age	Openness	Birth Weight	Early Growth	Pregnancy Complications	BMI 2 Years	BM BMI
Sex	NE	0.24 (0.16)	0.003 (0.02)	1							
Age	0.82 (0.66)	−0.88 (0.63)	−0.06 (0.08)	−0.01 * (0.004)	1						
Openness	−0.04 (0.10)	0.12 (0.09)	0.01 (0.01)	−0.03 (0.03)	−0.03 ** (0.01)	1					
Birth Weight	0.34 + (0.18)	0.17 (0.20)	−0.01 (0.02)	−0.02 (0.01)	−0.002 (.004)	0.004 (0.03)	1				
Early Growth	0.30 ** (0.09)	0.004 (0.10)	−0.002 (0.01)	−0.01 (0.03)	0.002 (0.010)	−0.08 (0.06)	−0.29 *** (0.04)	1			
Pregnancy Complications	0.01 (0.03)	0.01 (0.03)	−0.001 (0.003)	0.002 (0.10)	0.07 * (0.03)	−0.14 (0.22)	−0.04 (0.11)	0.38 (0.24)	1		
BMI 2 Years	0.21 * (0.09)	−0.08 (0.07)	0.003 (0.01)	−0.13 * (0.05)	0.02 (0.01)	−0.12 (0.14)	0.05 (0.06)	0.60 *** (0.14)	1.60 *** (0.40)	1	
BM BMI	0.03 + (0.01)	−0.02 (0.01)	0.000 (0.002)	−0.30 + (0.17)	−0.02 (0.05)	−0.71 + (0.40)	0.61 ** (0.20)	−0.50 (0.40)	2.33 + (1.23)	1.64 ** (0.57)	1

Obesity (Silver Spring). Author manuscript; available in PMC 2019 August 28.

Random Intercept Loadings	**BMI random intercept**	**Sleeprandom intercept**									
BF BMI	0.003 (0.03)	−0.04 (0.03)	0.001 (0.002)	−0.11 (0.22)	−0.08 (0.08)	0.09 (0.41)	−0.12 (0.21)	0.42 (0.36)	1.24 (1.65)	0.87 (0.68)	7.94 *(3.41)

Note. Values in table refer to unstandardized β, followed by (SE).

+ p < .10

* *p* < .05

** *p* < .01

*** *p* < .001.

Due to convergence problems, the effect of sex on BMI random intercept could not be included in this model: this effect did not approach significance in the model including morning cortisol (Table 3).