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Longer sleep duration during infancy and toddlerhood predicts weight normalization among high birth weight infants

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Abstract

Study Objectives: High birth weight (HBW; ≥ 4000 g) is strongly associated with later overweight, yet little is known about how to disrupt this trajectory. The current study examined sleep practices during infancy and toddlerhood among children born HBW or normal birth weight (NBW; 2500–3999 g).

Methods: Latent growth curve models were used to examine sleep during infancy and toddlerhood among 270 mother–child dyads enrolled in the Health Outcomes and Measures of the Environment Study. Total sleep duration in 24 hr, sleep maintenance, and restlessness/vocalizations were collected at 6 month intervals between ages 6 and 24 months. Height and weight were obtained at ages 24 or 36 months, and normal and overweight BMI were derived. Sleep was examined among children with a normal BMI during the preschool years who were either HBW (HBW-Normal, $n = 36$) or NBW (NBW-Normal, $n = 184$) compared with overweight preschoolers (Overweight, $n = 50$). It was predicted that the Overweight group would have poorer sleep across infancy and toddlerhood compared with HBW-Normal and NBW-Normal.

Results: HBW-Normal had the longest and Overweight had the shortest mean 24 hr sleep duration across all time points with NBW-Normal falling in-between the two groups. Compared with Overweight, HBW-Normal exhibited longer 24 hr sleep duration at age 6 months with this group difference maintained over infancy and toddlerhood. No group difference was found for NBW-Normal.

Conclusions: A longer sleep duration in the first several years of life is associated with development of normal BMI among HBW children. These findings suggest that longer sleep duration may protect HBW children from becoming overweight.

Statement of Significance

High birth weight is strongly associated with later overweight and obesity; however, we know little about how to disrupt this progression. Investigation of potentially modifiable factors during early childhood that predict weight trajectories could open new avenues for prevention of overweight in this high-risk group. The current study examined several aspects of early childhood sleep (24 hr sleep duration, maintenance, and restlessness/vocalizations) among children with a normal body mass index at age 24 or 36 months who had been delivered at either high or normal birth weight, compared with children with overweight at age 24 or 36 months. Findings showed that a longer 24 hr sleep duration was associated with attainment of a normal body mass index among children born at a high birth weight, compared with children who were overweight.

Key words: sleep duration; sleep maintenance; high birth weight; normal birth weight; pediatric overweight; infancy; early childhood

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Introduction

Conventional doctrine suggests that obesity is the product of an imbalance between energy intake and expenditure, but data increasingly show that the origins of obesity begin very early in life, with a diverse array of risk factors present before age 2 [1]. Amongst these early risk factors, high birth weight (HBW), defined as ≥ 4000 g, is a consistent predictor of overweight and obesity across the lifespan [2, 3] and is linked with later medical comorbidities including type 2 diabetes [4, 5], cardiovascular problems [6, 7], and some childhood cancers [8, 9]. The link between heavier birth weight and adverse health outcomes is robust, but little is known about why some HBW children do not develop obesity [10]. Identification of protective factors could point to novel, targeted ways to prevent overweight/obesity and related morbidity in the high-risk group of children born HBW.

Sleep may protect against the development of obesity. Sleep is behaviorally modifiable, and a number of researchers have identified an inverse relationship between sleep duration and overweight/obesity across childhood and adolescence [11–15]. Evidence over the last decade has emerged to suggest that this association may be present as early as infancy [16–19]. Insufficient sleep during infancy and toddlerhood is additionally linked to other obesogenic behaviors including earlier introduction of solids and increased screen time [20], fewer minutes spent in moderate-to-vigorous physical activity [18], and greater intake of energy, fat, and carbohydrates [21]. However, it is not yet clear whether the poor sleep–overweight/obesity association is broadly present, or is particularly relevant for identifiable high-risk subgroups, such as those born at a HBW.

This secondary analysis of data collected from the Health Outcomes and Measures of the Environment (HOME) Study [22] examined the associations between repeated measures of sleep (assessed during infancy and toddlerhood) and early childhood BMI among children born HBW and normal birth weight (NBW). Sleep duration in a 24 hr period, sleep maintenance (difficulties staying asleep or falling back asleep during night-wakings), and restlessness/vocalizations were repeatedly assessed at 6 month intervals between ages 6 and 24 months, and BMI was assessed at ages 24 and/or 36 months. We compared the sleep patterns of children who were born HBW and went on to normalize their BMI by the preschool years (“HBW-Normal”) and those who had NBW and remained normal weight as preschoolers (“NBW-Normal”) against those who were overweight or obese as preschoolers (regardless of birth status; “Overweight”). Because prior research has suggested that both short and disrupted sleep predict overweight/obesity, we predicted that, after controlling for potential confounding factors, the Overweight group would have a history of shorter 24 hr sleep duration, poorer sleep maintenance, and more restlessness/vocalizations across infancy and toddlerhood. To allow for the possibility that children born HBW and NBW might follow different paths to normal BMI in the preschool years (which would have implications for whether sleep might be a uniquely powerful target in children born HBW), we maintained distinct HBW-Normal and NBW-Normal groups in analyses.

Methods

Study sample

The HOME Study is a prospective pregnancy and birth cohort study designed to examine the health effects of common

environmental toxicant exposures. Between March 2003 and January 2006, pregnant women were recruited from nine prenatal clinics associated with three hospitals in the Cincinnati, OH metropolitan area [22]. Women were eligible to participate if they were (1) 16 ± 3 weeks pregnant, (2) ≥ 18 years old, (3) living in a home built on or before 1978, (4) intending to continue prenatal care and deliver at a study-affiliated obstetric clinic and hospital, (5) had no history of HIV infection, (6) were not taking medications for seizures or thyroid disorders, and (7) had no diagnosis of diabetes, bipolar disorder, schizophrenia, or cancer that required radiation or chemotherapy. Longitudinal follow-up of mother–child dyads included home and clinic visits during pregnancy, within 48 hr of delivery, and when children were 4 weeks and 12, 24, and 36 months old. Of the 389 singleton live births with available birth weight information, 24 infants with low birth weight (< 2500 g) were excluded from analyses as they constitute a unique group with a specific pattern of neonatal morbidities and growth outcomes that are distinct from children born at a normal or high weight [23, 24]. From the remaining 365 infants, 95 were missing height and weight information at both 24 and 36 months. A comparison of participants with complete v. incomplete data showed that mother–child dyads with complete data were more likely to be Caucasian, older mothers at delivery, married, and had higher incomes.

The Institutional Review Board of the Cincinnati Children's Hospital Medical Center approved this study, and all participating institutions relied on this as the IRB of record. All mothers provided written informed consent for themselves and their children prior to enrollment.

Sleep questionnaires

Sleep questionnaires were administered by trained research staff at 6 month intervals between ages 6 and 24 months. Face-to-face interviews were conducted during home visits when the child was 12 and 24 months, and phone interviews were conducted when the child was 6 and 18 months. Because there is no validated measure of sleep that spans infancy and early childhood, two questionnaires were developed to assess developmentally distinct domains of sleep in 6- to 18-month-olds (hereafter referred to as the infant questionnaire) and 24-month-olds (referred to as the preschool questionnaire). Items were based on previously validated questionnaires (see Byars et al. [25] for more information), and sleep domains were determined using factor analysis, with psychometrics and specific content described in a previous publication [25]. Item wording for the sleep domains examined across the infant and preschool sleep questionnaires can be found in [Supplementary Table S1](#).

Given the focus of the current study, we selected domains that assessed (1) 24 hr sleep duration, (2) sleep maintenance, and (3) restlessness and vocalizations (which, as previously reported, clustered together) [25]. On the infancy and preschool questionnaires, parents were asked to consider their child's sleep over the past month and past week, respectively. Parents indicated (1) how much time their child spends sleeping each night and napping each day (24 hr sleep duration), (2) frequency of night wakings per week and/or per night (sleep maintenance), and (3) frequency of restlessness/moving and talking/vocalizing during sleep (restlessness/vocalizations). Due to variable response options between the infant and preschool measures, each item response was converted to a common z-score metric

to promote comparability across the infant and preschooler questionnaires. Items were averaged together for domains that contained more than one item. Higher domain z-scores reflect longer 24 hr sleep duration, more sleep wakings, and greater restlessness/vocalizations [25].

Anthropometrics

Birth weight was abstracted from the child's medical record and coded as NBW (2500–3999 g) or HBW (≥ 4000 g) [2, 3]. At ages 24 and/or 36 months, weight and height measurements were obtained in triplicate and averaged within each time point. We were particularly interested in weight status at 36 months, but to minimize missing data, we capitalized on the high-temporal stability of BMI percentiles ($\rho = .77$, $p < .001$ between 24 and 36 months in our sample) and used the 24 month measurements if those data were missing at age 36 months. The majority of participants had available anthropometric measurements at 36 months ($n = 230$, 85.2%) with the remainder contributing this information at the 24 month assessment ($n = 40$, 14.8%). Weight was measured to the nearest 0.01 kg using a ScaleTronix scale (White Plains, NY) and height was measured to the nearest 0.1 cm with a wall-mounted Ayrton Stadiometer Model S100 (Prior Lake, MN). Weight was measured with the child dressed in undergarments or a dry diaper. Height was measured with the child standing straight, without shoes or head coverings, and with heels positioned against the wall. If the child had a hairstyle that precluded the head board from lying flat across the top of the child's head, we measured height of the hairstyle using a ruler and subtracted it from the average of the three height measurements. If a child was uncooperative during the measurements, we used a sitting scale or length board.

At 24 or 36 months, sex-specific BMI (kg/m^2) z-scores and percentiles were calculated using the 2000 Centers for Disease Control and Prevention growth charts [26]. Children were classified as normal weight (BMI: 5th–<85th percentile) or overweight (BMI $\geq$ 85th percentile). Three groups were calculated based on birth weight and later BMI percentile: (1) HBW with normal BMI percentile in early childhood ("HBW-Normal"), (2) NBW with normal BMI percentile in early childhood ("NBW-Normal"), and (3) overweight or obese in early childhood ("Overweight").

Weight and length were obtained at 1 and 12 months, and examined to gain a better understanding of preceding weight status. Weight-for-length was derived for 1-, 12-, and 24 or 36 months. Although the majority of children who fell below the 85th percentile in weight-for-length at 1 month maintained this weight trajectory, unsurprisingly, we found greater variability in weight-for-length among children ≥ 85 th percentile at 1-month-old. Stronger temporal correlations were observed between weight-for-length at 12 and 24 or 36 months [$r = .74$, $p < .001$] than between 1 and 12 months [$r = .26$, $p < .001$] or 1 and 24 or 36 months [$r = .31$, $p < .001$].

Because ~5.5% ($n = 12$) of all participants were considered underweight (BMI < 5th percentile) in preschool, and all of these children were born at NBW, we conducted separate sensitivity analyses that included v. excluded these children. Results were unchanged in these sensitivity analyses. Consequently, similar to other studies [12, 27], NBW children who met criteria for underweight were retained in the NBW-Normal group.

Potential confounders

To address potential confounding, sociodemographic and maternal variables were selected that plausibly related to either birth weight, weight status, and/or sleep. These included maternal age at delivery, maternal postpartum BMI when child was 1-month-old, household income, parity, marital status, child race/ethnicity, infant sex, mode of delivery, and gestational age. In addition, we adjusted for whether the child was fed breast milk at each survey occasion with the exception of age 24 months given high rates of missing information. The quality of the caregiving environment, as measured by trained research personnel using the semistructured Home Observation for Measurement of the Environment Inventory, was collected when the child was 12 months old during a home visit [28].

Statistical analytic plan

Descriptive statistics are reported as means (with standard deviations) and frequencies. Comparisons among NBW-Normal, HBW-Normal, and Overweight children were performed using chi-square tests for categorical variables and ANOVA for continuous variables (with post hoc tests using Tukey's HSD). We used latent growth curve models (LGCM) within a structural equation-modeling framework to examine repeated measures (i.e. sleep measured at several time points) as observed indicators of growth over time. Latent variables reflect sleep trajectories including their initial status (intercept) and rate of change (linear and quadratic) and are used to describe average response variable trajectories and model interindividual variations in the trajectory components (e.g. intercept, linear slope, and quadratic change). Two models were estimated. Model 1 was unconditional, contained no predictor variables, and was used to determine the form of change (intercept, linear, or quadratic) for each sleep outcome variable. Model fit indices, as described below, together with statistical tests of trajectory components, were used to select the best form of longitudinal change. Model 2, the conditional model, examined the impact of covariates and group membership on growth model variances specified in Model 1 (i.e. intercept and slope). Model 2 included several confounding variables that could influence birth weight, weight status, and/or sleep including maternal age at delivery, maternal postpartum BMI, household income, parity, marital status, child race/ethnicity, infant sex, mode of delivery, gestational age, Home Observation for Measurement of the Environment Inventory total score, breastfeeding status at 6, 12, and 18 month survey time points, the other two sleep domains, as well as group effects. Group was dummy-coded to compare HBW-Normal v. Overweight (HBW-Normal = 1, Overweight = 0) and NBW-Normal v. Overweight (NBW-Normal = 1, Overweight = 0). Exploratory analyses found no group differences between overweight/obese children who were born HBW v. NBW, supporting our decision to combine these into a single Overweight group in the final analyses presented below. The conditional model is considered the most conservative analysis, as it not only considers potential confounds, but also potential systematic reporter effects [29] on the sleep measures (i.e. significant effects of the sleep outcome variable cannot be accounted for by intentional or unintentional reporter biases).

Model fit indices were used to determine the best fitting unconditional model [30]. Good model fit was indicated by a

nonsignificant χ^2 test, root-mean-square error of approximation (RMSEA) < 0.05, comparative fit index (CFI) > 0.90, and standardized root-mean-square residual (SRMR) < 0.08. Models were tested in Mplus Version 8.1 (Muthén & Muthén, Los Angeles, CA) using maximum likelihood estimation. Empirical standard errors for all parameter estimates were obtained via bootstrap resampling (5000 repetitions).

Mplus Version 8.1 was also used to conduct a power analysis via Monte Carlo simulation that used 5000 replications based on published growth modeling techniques [31, 32] and assumed (1) standardization of all analysis variables, (2) proper missing data handling making $N = 270$ available for analysis, (3) a band-diagonal correlational structure would best capture the relationships among the time-varying covariates (i.e. variables collected at consecutive time points [i.e. 6 and 12 months] correlated at $r = 0.25$; variables separated by one time point [i.e. 6 and 18 months] correlated at $r = 0.30$; and variables separated by two time points [i.e. 6 and 24 months] correlated at $r = 0.06$), (4) time-varying covariates would explain 25%–35% ($R^2 = 0.25$ – 0.35) of repeated measures response variable variance, and (5) time-invariant covariates and group membership would explain 25% ($R^2 = 0.25$) of linear trajectory variance. Results showed power ≥ 0.80 for any standardized “intercept on group” coefficient of $\beta \geq 0.23$ and any linear “slope on group” coefficient of $\beta \geq 0.27$.

Results

As shown in Table 1, several characteristics significantly differed between the Overweight group ($n = 50$) compared with NBW-Normal ($n = 184$) or HBW-Normal ($n = 36$) groups. Consistent

with prior research, a higher rate of children with HBW (28%) compared with NBW (16.4%) went on to develop overweight or obesity. Compared with mothers of Overweight children, mothers of HBW-Normal children were older. Mothers of Overweight children were more likely to have higher BMI at 1 month postpartum compared with NBW-Normal and HBW-Normal. By design, birth weight differed between groups; both normal-weight groups centered near the 50th BMI percentile when assessed at 24 or 36 months, whereas the Overweight group centered above the 90th percentile. A greater proportion of males was found in HBW-Normal compared with Overweight.

Mother–child dyads contributed 1002–1012 repeated sleep measurements (mean observations per dyad = 3.7); approximately 79% of dyads had complete sleep information at all four assessments (6, 12, 18, and 24 months) and 94% had sleep information at three or more time points. Children slept a mean (SD) of 12.7 (2.2), 12.8 (1.8), 12.4 (1.6), and 12.5 (1.5) hr per 24 hr period at ages 6, 12, 18, and 24 months, respectively. Per 24 hr day, HBW-Normal, NBW-Normal, and Overweight slept 13.5 (1.8), 12.7 (2.2), and 12.2 (2.0) hr at age 6 months; 13.0 (1.7), 12.8 (1.8), and 12.5 (1.7) at 12 months; 12.8 (1.6), 12.5 (1.6), and 11.9 (1.7) at 18 months; and 12.7 (1.3), 12.6 (1.5) and 12.2 (1.7) at 24 months. Categorization of children as short sleepers at each age (i.e. short sleep, <12 hr per day at 6 months; <11 hr per day at 12, 18, and 24 months) based on sleep duration recommendations [33] revealed that a higher frequency of short sleepers was found in the Overweight group at 6 months (44.4%) compared with NBW-Normal [26.2%; $\chi^2(1, n = 209) = 5.6, p < .05$] and HBW-Normal [9.1%; $\chi^2(1, n = 78) = 11.4, p < .001$], and marginally significant differences at 18 months for Overweight (25%) relative to the

Table 1. Mother–child dyadic characteristics categorized by group

Variable	N	NBW-Normal	HBW-Normal	Overweight	P
Mother					
Age, years*	270	29.8 ± 0.4	32.4 ± 0.9	29.9 ± 0.7	.02
Postpartum BMI*†	266	27.0 ± 0.4	27.9 ± 0.7	31.6 ± 1.2	.00
Income	270	63940 ± 3126	65000 ± 5429	59350 ± 5782	.75
Cesarean delivery, n (%)	253	34 (19.8)	11 (31.4)	14 (30.4)	.15
Nulliparity, n (%)	270	81 (44.0)	11 (33.3)	22 (44.0)	.48
Married, n (%)	270	131 (71.2)	31 (86.1)	33 (66.0)	.10
HOME Inventory Total Score	270	39.8 ± 0.4	40.6 ± 0.6	40.2 ± 0.6	.59
Caucasian, n (%)	270	124 (67.4)	31 (86.1)	34 (68.0)	.08
African American, n (%)	270	48 (26.1)	4 (11.1)	13 (26.0)	.15
Hispanic, n (%)	270	3 (1.6)	0 (0.0)	2 (4.0)	.37
Asian/Pacific Islander, n (%)	270	6 (3.3)	0 (0.0)	0 (0.0)	.24
American Indian, n (%)	270	3 (1.6)	1 (2.8)	1 (2.0)	.89
Child					
Birth weight, grams*†	270	3260.7 ± 27.9	4260.3 ± 35.7	3734.9 ± 86.7	.00
Infant sex, n (%) male*	270	77 (41.9)	24 (66.7)	21 (42.0)	.02
Gestational age, weeks	270	39.1 ± 0.1	39.8 ± 0.2	39.5 ± 0.2	.00
Preschool BMI %ile*†	270	40.8 ± 1.8	54.7 ± 3.7	91.9 ± 0.6	.00
Caucasian, %	270	120 (65.2)	29 (80.6)	34 (68.0)	.20
African American, n (%)	270	52 (28.3)	4 (11.1)	13 (26.0)	.10
Hispanic, n (%)	270	3 (1.6)	1 (2.8)	2 (4.0)	.58
Asian/Pacific Islander, n (%)	270	7 (3.8)	0 (0.0)	0 (0.0)	.19
American Indian, n (%)	270	2 (1.1)	2 (5.6)	1 (2.0)	.19

Data are means ± SD or n (%). P-values are ANOVA or chi-square test, and significant ANOVA effects were followed by Tukey's HSD tests.

NBW-Normal = Normal birth weight with a normal body mass index percentile in preschool; HBW-Normal = High birth weight with a normal body mass index percentile in preschool; Overweight = Child with overweight or obesity in preschool; HOME = Home Observation for Measurement of the Environment Inventory.

* $p < .05$ between HBW-Normal and Overweight.

† $p < .05$ between NBW-Normal and Overweight.

Table 2. Model fit statistics for the sleep outcome variables using unconditional latent growth curve models

	Model fit indices					
	df	χ^2	P	CFI	RMSEA	SRMR
24 hr sleep duration						
Intercept	10	14.08	.17	0.98	0.04	0.05
Sleep maintenance						
Quadratic	1	0.04	.85	1.00	0.00	0.00
Restlessness/vocalizations						
Intercept	8	8.60	.38	1.00	0.02	0.05

df = degrees of freedom; CFI = comparative fit index; RMSEA = root-mean-square error of approximation; SRMR = standardized root-mean-square residual.

other two groups [NBW-Normal, 11.9%; $\chi^2(1, n = 170) = 3.9, p = .05$; HBW-Normal, 8.8%; $\chi^2(1, n = 70) = 3.2, p = .07$]. A marginal group difference was found at 24 months for Overweight (16.7%) and NBW-Normal [7.6%; $\chi^2(1, n = 201) = 3.2, p = .07$].

Model fit statistics for the unconditional models are presented in Table 2. No change, intercept-only provided the best fit for the 24 hr sleep duration and restlessness/vocalizations outcomes, although a quadratic model provided the best fit for sleep maintenance. The negative linear term (mean slope = $-0.03, p < .05$) indicated that sleep maintenance decreased over time but also evidenced a leveling off over time (mean quadratic = $0.00, p < .01$). Intercept variance was significant for all three sleep outcome variables (24 hr sleep duration: mean variance = $0.23, p < .001$; sleep maintenance: mean variance = $0.34, p < .05$; restlessness/vocalizations: mean variance = $0.20, p < .001$), indicating substantial heterogeneity at the initial start value. For the sleep maintenance model, the quadratic (mean variance = $0.00, p < .05$) but not the slope variance (mean variance = $0.01, p > .05$) was significantly different from zero.

A conditional model that included covariates and group as predictors was then tested (Table 3). HBW-Normal evidenced a significantly longer 24 hr sleep duration at age 6 months compared with Overweight (mean intercept: $b = 0.27, p < .01$; $\beta = 0.20, p < .01$; Cohen's $d = 0.72$). This group difference remained stable across infancy and toddlerhood (Figure 1). There was no effect of intercept on NBW-Normal for 24 hr sleep duration, nor was there an effect of intercept or slope on group (NBW-Normal or HBW-Normal) for sleep maintenance. HBW-Normal exhibited greater restlessness/vocalizations compared with Overweight (mean intercept: $b = 0.29, p < .05$; $\beta = 0.24, p < .05$; $d = 0.50$) but no difference was found for NBW-Normal.

Although outside of our stated hypotheses, a trend towards significance was found for the effect of intercept on NBW-Normal (reference group: HBW-Normal) for 24 hr sleep duration: NBW-Normal had lower initial 24 hr sleep duration at age 6 months (mean intercept: $b = -0.14, p = .11$; $\beta = -0.15, p = .11$; $d = .40$). NBW-Normal evidenced lower restlessness/vocalizations at age 6 months compared with HBW-Normal (mean intercept: $b = -0.33, p < .001$; $\beta = -0.38, p < .001$; $d = 0.50$).

Discussion

Consistent with our hypotheses, overweight children had shorter parent-reported 24 hr sleep duration during infancy and toddlerhood compared with HBW-Normal; this effect was present even after controlling for important confounders (e.g. quality of the home environment, sociodemographic characteristics, and breastfeeding status) and potential reporter

biases. In general, these findings suggest that longer 24 hr sleep duration early in life may buffer against overweight in a sample of HBW children, suggesting a potential intervention for this high-risk group. Although our findings fit within a broader literature implicating short sleep as a risk factor for overweight/obesity [11–13, 16–19], they also suggest that this relationship may be particularly strong in subgroups with high overweight/obesity risks, such as those born at a HBW.

To the best of our knowledge, only one study has examined sleep practices among infants with distinct obesity risk. Ekstedt et al. [34] examined the sleep of 12-month-old infants with high- vs. low-obesity risk based on parent weight. High-risk infants, based on high parental weight, had significantly later bedtimes and wake times and a longer sleep onset latency compared with offspring of normal-weight parents. Later bedtime was also weakly associated with higher BMI after adjustment for family obesity risk [34]. Findings from that study converge with findings of the current study indicating that sleep patterns vary early in life and characterize children with distinct vulnerabilities to overweight and obesity.

Although the current study examined modifiable, protective influences that are associated with development of normal BMI among HBW children, it was not designed to identify the mechanisms for this effect. Prior authors have suggested that insufficient sleep may result in fatigue that leads to increased sedentary behaviors [17, 18, 20] or an increase in caloric intake [21, 35, 36]; an imbalance in energy intake and expenditure may therefore lead to excess weight gain. There may also be a relationship between sleep and fluctuations in appetite-regulating hormones such as ghrelin and leptin, though this remains unclear in childhood [37–40]. Prior cross-sectional work has found that, although HBW and NBW infants have similar parent-reported eating behaviors and feeding practices, HBW infants who remain high weight-for-length at age 7–8 months have a poorer response to internal cues of satiety than those whose weight normalizes [41]. Since reduced responsiveness to satiety is linked with later weight gain [42], one testable mechanism for current findings might be that short-sleep duration is linked to poor responsiveness to satiety, particularly for children born HBW.

It may also be that dysregulated appetite and poor sleep better reflect general self-regulation difficulties (e.g. related to feeding, appetite, sleeping, and soothing) and that the parental reaction to distress may maintain specific behavioral patterns that have implications for later weight status [43]. Children with prolonged parent contact at bedtime or during the night (e.g. tendency to immediately attend to crying child in the night, provide a feed to settle, and bed share) experience poorer sleep

Table 3. Unstandardized and standardized estimates^a and standard errors for conditional latent growth curve models

Predictor	24 hr sleep duration ^a				Sleep maintenance ^b				Restlessness/vocalizations ^c			
	b	SE b	β	SE β	b	SE b	β	SE β	b	SE	β	SE β
HBW-Normal												
Intercept	0.27**	0.11	0.20*	0.08	0.19	0.20	0.11	0.11	0.29*	0.12	0.24*	0.10
Slope					-0.02	0.01	-0.21	0.17				
NBW-Normal												
Intercept	0.13	0.08	0.13	0.09	0.24	0.14	0.18	0.11	-0.04	0.09	-0.04	0.10
Slope					-0.02	0.01	-0.25	0.18				

* $p < .05$; ** $p < .01$.

b = Unstandardized beta weight; SE = Standard error; β = Standardized beta weight.

^aModels adjusted for maternal age at delivery, maternal postpartum BMI, method of delivery, gestational age, infant sex, marital status, parity, income, breastfeeding status, and Home Observation for Measurement of the Environment Inventory.

^bModel adjusted for sleep maintenance and restlessness/vocalizations z-scores.

^cModel adjusted for 24 hr sleep duration and restlessness/vocalizations z-scores.

^dModel adjusted for 24 hr sleep duration and maintenance z-scores.w

Figure 1. Predicted mean 24 hr sleep duration z-score from ages 6 to 24 months by group membership.

quality [44–47]. Moreover, problematic infant sleep predicts future (lower) parental tolerance for crying and greater efforts towards nighttime soothing [48]. It is possible that children with consistent parent intervention are not given the opportunity to self-settle or soothe and return to sleep on their own, and therefore become reliant upon or demand greater parental attention throughout the night; this may result in greater sleep fragmentation for both parties and poorer mental health for mothers [45]. Although there exists a relationship between the quality of maternal–infant interactions and adiposity or weight [49], little is known about these associations as they pertain to nighttime dyadic behaviors. Indeed, both parents and young children mutually influence each other's sleep behaviors—which in combination with birth weight—may shape weight development.

Multicomponent interventions have been created to improve caregiver response to infant distress in an effort to improve self-regulation abilities and growth patterns [50, 51]. In a randomized controlled trial that did not explicitly target nutrition or obesity, families were assigned to a responsive parenting intervention that targeted self-regulation difficulties and infant sleep hygiene versus an attentional control condition [52]. Compared with the control group, infants in the responsive parenting intervention group had earlier bed times and longer nighttime sleep duration [51], as well as slower weight gain and reduced prevalence of overweight at age 12 months [52]. The potentially important role of infant sleep was highlighted in another randomized controlled trial that found a reduced prevalence of obesity among children

whose mothers had received an infant sleep intervention, both compared with a no-intervention control condition and a condition that offered guidance to mothers on infant nutrition and physical activity [53]. Importantly, these studies have applied intervention techniques to a broad range of infants, but it remains to be seen how these interventions fare amongst a targeted sample of infants and toddlers at high risk for overweight/obesity. Moreover, the current study found the most pronounced group (HBW-Normal v Overweight) difference in 24 hr sleep duration occurred at age 6 months and remained constant across infancy and toddlerhood, indicating that these differences may persist into childhood. And although a difference was found in 24 hr sleep duration at 6 months for HBW-Normal and NBW-Normal, it was only at trend level and the two groups appeared to converge at later time points (ages 18–24 months). Future studies should further examine these relationships and use the findings to capitalize and implement behavioral sleep interventions during this early time frame to reduce risk for overweight/obesity.

Although this study has several strengths including the LGCM approach, repeated assessment of sleep, and adjustment for a large number of covariates and reporter biases, our results are subject to limitations that are inherent to longitudinal, observational research. First, sleep questionnaires are widely used, cost-effective [54], and many are considered well-validated, promising, or well-established assessment tools [55] (including items adapted for use in the current study) [56–63]; however, there remain substantial concerns related to subjectivity and measurement imprecision. Although we do not have a compelling reason to expect systematic reporter bias, we cannot rule out that possibility and acknowledge that parent-report may be an indirect and imprecise measure of sleep quantity and quality. It should be noted, however, that the variability (standard deviation) in sleep duration decreased as child age increased and the greatest between-group differences were found at these younger ages, suggesting a lack of precision may have influenced our findings. However, HBW-Normal evidenced the lowest standard deviation in 24 hr sleep duration at 6 months, indicating perhaps lower variability in this group. In future studies, it will be important to mitigate the effects of recall biases by including objective measures of sleep (e.g. polysomnography) to examine the relationships between birth weight, weight, and sleep. Second, although the sleep constructs we measured were equivalent over time, developmental shifts

necessitated different wording of sleep questions at 24 months from those when the children were younger. Prior work with this approach offers psychometric support for doing so (see Ref. 25), and we found no evidence of a shift in effects at the questionnaire transition point (i.e. between the infant and preschool questionnaires), but the potential for nonequivalent measurement over time is important to acknowledge. Third, although we included many relevant covariates, there are potentially unmeasured child, family, and community-level factors (e.g. child screen time, activity level, child temperament, and environment) that could affect findings. It is plausible that 24 hr sleep duration is a marker for other parenting or family factors (e.g. maternal responsivity at night) that influence long-term weight status. Fourth, as is common in longitudinal work, approximately 26% of participants were excluded due to missing anthropometric data. Exploratory analyses suggested that retention in the study may be related to patterns that influence adiposity (e.g. higher incomes) [64], though it is not immediately clear whether or how that might affect the relationships between sleep and adiposity reported here. Future studies using larger sample sizes to examine these relationships will be needed, given that the HOME Study was not specifically designed or powered to examine these hypotheses. Finally, these data are more than 15 years old and although 24 hr sleep duration has decreased [65] and obesity rates have steadily increased over time [66]; additional contemporary analysis is needed to enhance understanding of these pathways.

Although HBW is strongly associated with later overweight and obesity, that progression is not universal. Current findings suggest that longer 24 hr sleep duration during infancy and toddlerhood may be a protective factor, setting children born HBW on a trajectory towards weight normalization. Replication of these associations using larger sample sizes and objective sleep measurement tools is needed before engaging in the design and evaluation of behavioral sleep prevention programs to reduce overweight/obesity in this high-risk group of HBW children.

Supplementary Material

Supplementary material is available at SLEEP online.

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