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Sleep-wake patterns in newborns are associated with infant rapid weight gain and incident adiposity in toddlerhood

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Summary

Background: Rapid weight gain (RWG) by 6 months of life is a significant risk factor of childhood overweight (OW)/obesity. Infant sleep patterns are associated with incident OW in childhood, but few have examined its relationship with RWG.

Objective: Examine associations between newborn sleep-wake patterns and incident RWG at 6 months of life and OW at 36 months.

Methods: Low-income Mexican/Mexican-American women with OW/obesity and their infants (n = 126) enrolled in a 1-year randomized controlled trial designed to prevent incident, infant RWG and toddlerhood OW/obesity. Sleep pattern metrics at 1 month were extracted from the Brief Infant Sleep Questionnaire-Revised. Outcome measures included RWG (>0.67 positive change in weight-for-age Z-score) from birth to 6 months and incident OW (body mass index percentile ≥85) at 36 months.

Results: By 6 months, 35.7% (n = 45) of infants experienced RWG, and by 36 months 42.3% (n = 41) of toddlers were OW. Napping ≥5x/day at 1-month was significantly associated with decreased odds for RWG compared to napping <5x (OR = 0.11, 95% CI:0.02, 0.63). Each 1-hour increase in nocturnal vs diurnal sleep was associated with greater odds of incident OW at 36 mos (OR = 1.51, 95%CI:1.13, 2.03).

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CONFLICT OF INTEREST

The authors have no competing interests to report.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

Conclusions: Early-life sleep patterns related to infant nap frequency and nocturnal vs diurnal sleep distribution were associated with obesity outcomes and may be important intervention targets to prevent lasting consequences on infant growth.

Keywords

infant sleep; napping; rapid weight gain; sleep consolidation

1 | INTRODUCTION

Adiposity continues to affect Americans of all ages, with an estimated 18.5% of children aged 2-19 years classified as having obesity in 2015-2016.[1] Among 2-5 year olds, the prevalence of obesity increased from 8.4% in 2011-2012 to 13.9% in 2015-2016.[1] This trend continues to affect children at younger ages, with nearly 10% of infants and toddlers carrying excess weight in the earliest part of life.[2] Obesity later in life may be heralded by events as early as infancy. Rapid weight gain (RWG; greater than 0.67 positive change in weight-for-age Z-score)[3] in the first 6 months of life is a potent indicator of obesity risk across the lifespan.[4-8] RWG during infancy is also associated with comorbidities including hypertension, diabetes and cardiovascular disease later in life.[9-11] Timing of RWG in infancy also appears to be important for adverse metabolic outcomes as excess weight gain within the first 6 months is predictive of poor cardiometabolic outcomes in adulthood.[12]

To date, few studies have identified the modifiable, environmental, social, or behavioural factors that contribute to RWG or obesity risk in infancy and early childhood. Early sleep development may be an important, modifiable health behaviour. Prospective data suggest the important connection of sleep to obesity risk with early exposure to short sleep duration in infancy increasing the risk of increased BMI or obesity during toddlerhood[13] and childhood.[14,15] Systematic reviews and meta-analyses provide support for an association between short sleep duration and overweight/obesity throughout childhood.[15-19] A recent meta-analysis of children from birth to 3 years of age found similar results, such that BMI z-score decreased (−0.02 [95%CI: −0.04,−0.01]) for every 1-hour increase in total sleep.[15] However, the literature remains mixed and limited such that some studies suggest no association between sleep duration and adiposity,[20,21] or that adiposity may precede short sleep duration,[22] and only limited data are available for examining the association between sleep and RWG in early infancy.[23]

Beyond sleep duration, other dimensions of sleep have been infrequently studied and warrant further investigation.[24] Sleep-wake timing and circadian rhythmicity appear to play important roles in obesity risk, as variation in sleep schedules has been found to have a stronger influence on metabolism than total sleep duration.[25] Later bedtimes and greater variability in sleep schedules are predictive of greater changes in weight[26-28] which are also associated with negative metabolic outcomes.[29] Infant growth is also impacted by sleep-wake patterns. Increased nap frequency and longer, overall sleep duration among 23 infants was found to have temporal concordance with subsequent growth in length.[30] In contrast, longer sleep bouts in infancy and greater nap duration in toddlerhood were associated with increased adiposity.[20,30] The influence of sleep-wake patterns extends to preterm infants as

well, where research suggests that more rapid sleep state (ie, active and quiet) development was associated with favourable weight outcomes.[31]

Overall, research on which sleep-wake patterns in early infancy are related to adiposity during infancy and later in childhood is limited. Further, most studies that have conducted sleep-wake assessments were completed later in infancy or during the toddler years. The first half, particularly the first 3 months, of infancy is an important period for establishing sleep-wake circadian rhythms,[32,33] and may be a target for intervention to prevent RWG and subsequent obesity. Though at least one ongoing study[23] is currently investigating how variation in sleep-wake patterns during early infancy impact RWG,[34-36] an important early indicator of risk for childhood and adulthood adiposity, we know of no published findings on this relationship. The aims of the present exploratory study were to address these gaps by examining associations between sleep-wake patterns in early infancy and RWG during the first 6 months, and incident overweight (OW) at 36 months of age among infants enrolled in a randomized clinical trial that recruited mothers with overweight or obesity. We hypothesized that adequate sleep duration (ie, 14-17 hours among newborns according to the National Sleep Foundation recommendations[37]), and more consolidated nighttime sleep would be associated with lower odds for RWG and incident OW.

2 | METHODS

2.1 | Study design

Pregnant, low income Latina women with preconception overweight or obesity were recruited from the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) program to take part in a randomized controlled trial designed to provide education on infant feeding and development to prevent incident overweight. This trial was approved by the following institutional review boards (IRB): Arizona State University IRB, the Texas Department of State Health Services IRB, and the City of Houston Health and Human Services research committee. More details about this study can be found elsewhere.[38,39] In brief, expectant mothers were enrolled in the third trimester of their pregnancies, and their infants were enrolled within 1 week postpartum. The mothers provided written informed consent during the prenatal visit. Mothers and infants were excluded from participation if they were not discharged to home together after delivery, or if the mother or infant experienced significant postpartum complications or illness. The intervention was delivered by trained community health workers in participants' homes starting at 36 weeks of pregnancy followed by visits at 3 days after birth, 2 weeks, and 2, 4, 6, 9, 12, 18, 24 and 30 months with follow-up at 36 months. The intervention focused on delivering education and feedback on Institute of Medicine recommendations on growth monitoring, feeding (eg, support exclusive breastfeeding), parenting (eg, recognizing hunger and satiety cues), activity (eg, promoting active play), and sleep (eg, 10-12 hours of sleep per day[40]). At 1, 6, 12, 18, 24, 30 and 36 months of age, trained and blinded research assistants measured infant weight and length, and at 1 and 6 months, mothers retrospectively reported on their babies' sleep-wake patterns. To evaluate the aims of this sub-study, we examined the sleep measures at the 1-month visit, and anthropometric measures at the 6-, and 36-month visits as these time points best address how weight gain in the first 6 months of life impacts risk for obesity

as a child. We also sought to replicate prior literature reporting a relationship between RWG in the first 6 months of life and increased risk for childhood OW. Lastly, as a sensitivity analysis, to examine the importance of sleep in early infancy compared sleep in mid-infancy on increased risk for childhood OW, we explored the association between sleep measures at 6 months with incident OW at 36 months.

2.2 | Analytic sample

Full-term (37-42 weeks) infants with normal birth weight of 2500 to 4000 g were eligible for the present study (n = 150). There were no statistically significant effects of the intervention on infant RWG at 6 months and OW at 36 months (data not presented), therefore both groups were eligible to be included in the present analytic sample. Infants with incomplete anthropometric data at birth (n = 9) and sleep data at the 1-month visit were excluded (n = 1). A further 13 infants with incomplete covariate or anthropometric data at 6-month follow-up were excluded from subsequent analyses with RWG as the outcome, yielding a final analytic sample size of 126. For analyses examining incident OW at the 36-month follow-up visit, 31 toddlers without anthropometric measures due to loss to follow-up were excluded, yielding a final analytic sample size of 96.

2.3 | Measures

2.3.1 | Sleep—Infant sleep patterns were assessed with the Brief Infant Sleep Questionnaire - Revised (BISQ-R). The BISQ-R is a validated questionnaire that measures infant sleep-wake patterns and disturbances in the context of the sleep environment and sleep-related parental interventions.[41] Only items from the BISQ-R that are most relevant to sleep patterns at 1 and 6 months of age were examined including bedtime ("What time do you put your child to bed at night? (time of turning off the light)," measured in 30-minute intervals of clock time), nap frequency per day ("how many naps does your child take during a typical DAY (between 8:00 AM and 7:00 PM)?", measured continuously and by quartiles), longest nocturnal sleep bout ("on a typical night, what is the longest stretch of time that your child is asleep during the night without waking up?" measured in hours and minutes and analysed as number of minutes and by quartiles), nocturnal total sleep time (TST; "how much total time does your child spend sleeping during the NIGHT (between 7:00 PM and 8:00 AM?", in hours), diurnal TST ("how much total time does your child spend sleeping during the DAY (between 8:00 AM and 7:00 PM)?", in hours), 24-hours TST (sum of nocturnal and diurnal TST measured continuously and by cutoffs based on recommendations for sleep duration among newborns),[37] and the difference between nocturnal and diurnal TST. Diurnal sleep was subtracted from nocturnal TST such that a positive difference indicates greater nocturnal than diurnal TST, and a negative difference indicates greater diurnal than nocturnal TST. Napping frequency and longest nocturnal sleep bout values were polytimized into quartiles because of their positively-skewed, leptokurtic distributions. Later, the top quartiles of napping frequency and longest nocturnal sleep bout were compared to all other quartiles. High variability in sleep-wake patterns is common among newborns with the most rapid consolidation of sleep occurring within the first 4 months.[42-44] For newborns, there are currently no guidelines or recommendations for sleep metrics beyond 24-hour sleep duration due to either low quality or scarce availability of evidence tying these sleep metrics

to health outcomes.[37,45-47] Thus, with the exception of 24-hour TST, a data-driven, exploratory approach was elected to examine their potential relevance.

2.3.2 | Adiposity—At 6-month visit, infant weight was measured (in kg, to the nearest 0.1 kg) recumbent on an electronic digital portable scale (Seca model 19-17-05-224) with the infant wearing only a dry diaper. Recumbent length at 6 months was measured to the nearest cm with a portable Tanita length board holding the head and feet steady with legs extended. At the 36-month visit, child's standing weight and height were measured to the nearest 0.1 kg and cm, respectively. Birth weight was parent-reported and recorded at the first in-home visit. Outcome measures included RWG (>0.67 positive change in weight-for-age Z-score) from birth to 6 months,[3] and incident OW (body mass index [BMI] percentile ≥85) at 36 months using World Health Organization growth charts from birth to 2 years and the Centers for Disease Control and Prevention growth charts for children ages two and older.[48]

2.3.3 | Covariates—Several prenatal, post-birth, and interventional variables were selected as covariates either because of prior literature indicating their influence on growth or due to methodological considerations. During the prenatal period, mothers self-reported their weight and height prior to pregnancy (used to calculate prenatal maternal BMI),[49] parity,[50] and educational attainment (high-school degree or greater vs middle-school or some high-school).[51] Because the parent study was a randomized trial, group assignment was included as a covariate in all analyses. At post-birth, birth weights were reported,[35] and mothers also self-reported their gestational weight gain.[52] Mothers also were asked to report any prenatal smoking or pregnancy complications they experienced including gestational diabetes, gestational hypertension, preeclampsia, eclampsia, and anaemia. None of the mothers reported prenatal smoking, and 15 of 126 mothers (11.9%) did not report information about pregnancy complications. Of those that did, presence of pregnancy complications was not associated with RWG (X^2 [1] = 0.06, P = .81) or OW status (X^2 [1] = 0.98, P = .32). Thus, due to missingness and a lack of association of those that did report, we elected to not include this variable in our models. Each postpartum month during the trial period, mothers reported breastfeeding status from which was calculated the duration of any breastfeeding up to 12 months.[49,50]

2.4 | Statistical analysis

Descriptive characteristics of each sleep variable were quantified and checked for normality. Proportions meeting the definitions of RWG by 6 months and OW by 36 months were determined. Demographic, anthropometric and sleep characteristics of participants with OW status data at 36 months were compared to those of participants lost to follow-up. First, the association of each sleep variable with each outcome (RWG status at 6 months [yes/no] and OW status at 36 months [yes/no]) was analysed in logistic regression models while adjusting for weight-for-age Z score at birth (Model 1). Sleep variables categorized into quartiles were entered into the models as dichotomous variables comparing the top quartile of the distribution to all other quartiles. Then, in Model 2, the associations of each sleep variable with each outcome were further adjusted for duration of any breastfeeding in months (across the first 6 months for RWG analyses, and then up to 12 months for the OW analyses),

intervention group, maternal education, prenatal BMI, parity, and gestational weight gain. For models examining OW status as outcome, Model 2 was further adjusted for infant RWG status at 6 months. For any sleep variables other than 24-hours TST that were significantly associated with RWG or OW, sensitivity analyses were conducted by adding 24-hours TST to Model 2 to assess the unique contribution of the sleep variable in question above and beyond the influence of sleep duration. In an additional sensitivity analysis, status of any breastfeeding whether exclusive or mixed at 1 month (yes/no) was adjusted for in Model 2 to account for its influence on sleep-wake patterns at 1-month. Lastly, the association between the sleep measures at 6 months and incident OW at 36 months were conducted in a sensitivity analysis. IBM SPSS v. 25 was used to perform all statistical tests and significance was set at $P < .05$.

3 | RESULTS

3.1 | Sample characteristics

By 6 months, 45 (35.7%) of 126 infants experienced RWG, and by 36 months 41 (42.3%) of 96 toddlers were OW. See Table 1. In the overall sample, about half of the mothers did not graduate high school and a quarter had three or more children prior to giving birth to the infant included in the study. Mothers breastfed their infants on average 4 to 5 months. Infants who experienced RWG tended to have lower weight for age Z scores at birth ($t[124] = 4.07$, $P < .001$), and were more likely to be born to mothers who had no prior children ($t[124] = 2.11$, $P = .04$). There was also a trend towards fewer months of breastfeeding among infants with RWG ($t[124] = 1.93$, $P = .06$). Infants who experienced incident OW by 36 months were more likely to be born to mothers who had not graduated from high school ($\chi^2[1] = 9.23$, $P = .002$). Compared to participants lost to follow-up, participants with weight status data at 36 months of age were more likely to be male, to have greater nocturnal and 24-hours TST at 1 month, and to be breastfed longer (data not presented, all $P < .05$). RWG at 6 months of life was associated with increased odds of OW at 36 months ($\chi^2[1, N = 96] = 4.05$, $P = .04$; OR = 2.35, 95%CI: 1.02, 5.42).

Table 2 displays average sleep characteristics at 1 month of age in the overall sample and by RWG and OW statuses. Overall, mean bedtime was slightly earlier than 10:00 PM. Infants napped on average about four times per day, and the average longest sleep bout at night was 4 hours and 36 minutes. Mean nocturnal TST, diurnal TST, and 24-hour TST were about 9, 7 and 16 hours, respectively. Infants, on average, slept 2 hours more at night than during the day. Univariate associations indicated that the top quartile of nap frequency, compared to all other quartiles, was significantly associated with RWG status ($P = .003$). Longer nocturnal TST ($P = .037$), and greater differences in nocturnal and diurnal sleep ($P = .013$) were significantly associated with OW status.

3.2 | Sleep at 1 month and incident RWG at 6 months

Table 3 displays the birth weight z score adjusted (Model 1) and fully-adjusted (Model 2) odds for RWG by 6 months predicted by each sleep variable. In Model 1, only napping frequency (measured in quartiles) was significantly related to RWG status. Compared to infants napping less than five times per day, infants who napped more (top quartile) were at

significantly decreased odds for incident RWG (OR: 0.12, 95%CI: 0.03, 0.58). After further adjustment (Model 2), this association remained significant (OR = 0.11, 95%CI: 0.02, 0.63, $P = .013$), as did the overall model fit (χ^2 [8, $N = 126$] = 41.4, $P < .001$). The Cox and Snell R Square and Nagelkerke R Square for the model were 28.0% and 38.4%, respectively, and correctly classified 73.8% of cases. To account for the potential confounding due to the positive correlation between napping frequency and 24-hour TST ($r = .32$, $P < .001$), and to gauge the unique contribution of napping frequency, in a sensitivity analysis, 24-hour TST was added to Model 2. Frequent napping remained significantly associated with decreased odds of RWG (OR: 0.09, 95%CI: 0.02, 0.56, $P = .009$). In addition to 24-hours TST and breastfeeding at 1-month was also added as a sensitivity analysis to Model 2 for its potential influence on the sleep assessment; the association between frequent napping and decreased odds of RWG remained (OR: 0.09, 95%CI: 0.01, 0.53, $P = .008$).

3.3 | Sleep at 1 month and incident OW at 36 months

Table 3 displays the weight-for-age adjusted (Model 1) and fully-adjusted (Model 2) odds for OW by 36 months predicted by each sleep variable. In Model 1, each 1-hour increase in nocturnal TST (OR: 1.42, 95%CI: 1.02, 1.96), as well as each 1 hour increase in the difference between nocturnal and diurnal TSTs (OR: 1.41, 95%CI: 1.10, 1.83) were associated with increased odds for incident OW. After further adjustment, greater nocturnal TST was no longer significantly associated with incident OW (OR: 1.41, 95%CI: 0.99, 2.03). After further adjustment (Model 2), the model including the difference between nocturnal and diurnal TSTs remained statistically significant χ^2 (9, $N = 96$) = 27.8, $P = .001$. The Cox and Snell R Square and Nagelkerke R Square for the model were 25.2% and 33.8%, respectively, and correctly classified 75.0% of cases. Each 1-hour increase in the difference between nocturnal and diurnal sleep amount (in favour of proportionately more nocturnal sleep) was significantly associated with increased risk for incident OW (OR = 1.51, 95%CI: 1.13, 2.03, $P = .005$).To account for the potential confounding from the negative correlation between the difference in hours between nocturnal and diurnal TST and 24-hour TST ($r = -.24$, $P = .007$), and to gauge the unique contribution of the proportion of night to daytime sleep, in a sensitivity analysis, 24-hours TST was added to Model 2. Greater difference in hours between nocturnal and diurnal sleep amount remained significantly associated with increased odds of OW (OR: 1.53, 95%CI: 1.13, 2.07, $P < .005$). In addition to 24-hours TST, any breastfeeding at 1-month was also added as a sensitivity analysis to Model 2 for its potential influence on the sleep assessment, and the association remained (OR: 1.53, 95%CI: 1.14, 2.07, $P = .005$). Supplementary Table 1 displays the weight-for-age adjusted odds for OW by 36 months predicted by each sleep variable reported at six months of age. There were no significant associations identified.

4 | DISCUSSION

In a high-risk, Mexican or Mexican-American maternal-child sample, 1-month old infants obtaining five or more naps per day had 89% lower odds of experiencing RWG by 6 months of life, an important early predictor of childhood obesity as found in the literature, as well as the present sample. Sleep at 1 month of age was also associated with OW at 3 years of age, such that each 1-hour increase in nocturnal vs diurnal sleep over 24 hours was associated

with a 1.51 greater odds of incident OW. These data suggest the importance of napping frequency and allowing the opportunity and surrounding circumstances for sleep episodes throughout both the day and night independent of the total sleep amount to avoid adverse weight outcomes in infancy that may persist into early childhood.

RWG in the first 6 months of infancy increases the odds of obesity[4,5,7,8] and related comorbidities across the lifecourse.[9-11] In this study we observed RWG in nearly 36% of infants. Experiencing RWG in the early months of infancy increased the likelihood of overweight in toddlerhood, which corresponds with previous literature linking early weight gain with childhood obesity.[3,6,53] The literature linking RWG with subsequent obesity is consistent across diverse cohorts of mixed race/ethnicity, demographics and socio-economic status indicating that RWG is a robust indicator of obesity risk even after adjusting for confounding perinatal factors including maternal weight status, infant birth weight, etc. Despite this link, few studies have investigated the modifiable environmental, social, and behavioural factors that predict whether infants will experience RWG.

We found that napping five or more times during the day at 1 month of age was associated with a decreased likelihood of RWG. Increased nap frequency has previously been linked to anabolic growth characterized by increased length growth and more favourable weight outcomes.[30] Assessments of napping frequency and body weight outcomes remain sparse during infancy. Among toddlers, findings regarding napping effects (frequency and duration) on BMI percentiles remain mixed with some studies observing no association,[27,54,55] while others report an inverse relationship.[56] Despite the limited data in infants and conflicting results from toddler studies, increased nap frequency in this cohort suggests the importance of this aspect of sleep-wake patterns for childhood obesity risk.

Sleep duration has previously been identified as an important indicator of childhood overweight or obesity, as reported in multiple systematic reviews and meta-analyses.[16,17,19,25,57] Although we did not observe significant associations between sleep duration and incident overweight in our cohort, this relationship has been reported by others in the 0-3 year age range.[15] The influence of sleep duration on obesity risk may not be independent of other aspects of sleep-wake patterns. For example, sleep-wake distribution and timing (ie, bedtime and sleep-wake scheduling consistency) in low-income preschoolers has been found to moderate the relationship between nocturnal sleep duration and BMI z-scores.[27] Our data corroborate the importance of sleep-wake distribution as the difference between nocturnal and diurnal sleep over 24 hours in the first month of life emerged as an important predictor of early childhood obesity. Contrary to our hypothesis that greater indication of nighttime sleep consolidation would be associated with favourable metabolic outcomes, we found that for each 1-hour increase in nocturnal sleep, at the expense of daytime sleep, the odds of incident OW at 3 years of age was increased by 1.51. Again, data in infants are limited making comparison difficult. However, the literature available for preschool-aged children supports our findings regarding sleep-wake distribution across the 24 hours. Both later bedtimes and variable sleep schedules are believed to predict greater changes in weight[26,27] and poor metabolic outcomes including alterations in insulin, low-density lipoprotein, and inflammatory markers.[29] Previous work suggests that aspects of sleep-wake distribution including timing and variability in sleep length and scheduling may have stronger influences

on metabolism than sleep duration,[25] which further emphasizes the importance of our finding that certain sleep-wake distributions as early as 1 month of age may have lasting impacts on weight status through early childhood.

Sleep-wake distribution across the 24 hours at 1 month of age, whether reflected in the number of daytime sleep opportunities or in the duration of sleep at night relative to during the day, appear to be important for future adiposity. Development of the 24-hour circadian sleep-wake rhythm is typically established around 12 weeks of age.[32] Prior to that milestone, most infants experience ultradian or irregular sleep-wake rhythms that gradually develop into an entrained 24-hour rhythm over this time period.[33] Signs of entrainment are displayed, on average, as early as 3 to 5 weeks of life,[32,33] which matches the 1-month assessment period in the present study. Evidence suggests that entrainment of the 24-hour circadian sleep-wake rhythm is highly influenced by mother-infant interaction that even begins in the womb and becomes more synchronous with the mother's circadian sleep-wake rhythm over the first 12 weeks.[32,58] Mother-infant interaction may have an independent and perhaps greater influence than light exposure on entrainment.[32,59,60] Other behavioural factors that contribute to healthy sleep development may include reducing noise in the sleep environment, infant massage and skin-to-skin contact.[60] Thus, maternal patterns and interactions with their infants likely have a strong influence on when, how often, and for how long newborns are afforded opportunities and circumstances to sleep. Mothers may unintentionally contribute to inconsistent sleep-wake routines and non-sleep promoting environments. Our data suggest early sleep-wake patterns that allow for multiple sleep opportunities during the day to prevent overcompensation during the night may be protective metabolically, and thus may be a potential target for intervention. In other words, a “sleep-on-demand” approach in the early weeks of life may be beneficial, but further research is needed to confirm this observation.

The chronobiology underpinning sleep-wake consolidation and rhythmicity has substantial crosstalk with the neuroendocrine system and metabolic regulation that affect physical growth.[30,61] For example, growth hormone secretion is substantial in the first few months of life, and mostly occurs soon after sleep onset. However, little is known about the impact of growth hormone on sleep-wake consolidation and rhythmicity in healthy, developing infants.[61] Another example demonstrates that duration of sleep bouts tend to increase as body size increases suggesting that metabolic rate may regulate sleep organisation.[62,63] However, investigations examining dysregulation of sleep-wake consolidation and rhythmicity, the neuroendocrine system, and metabolism in infancy remain rare. Thus, the mechanisms linking sleep-wake distribution to infant adiposity and growth are poorly understood.

Strengths of this study include the study of a high-risk minority population that has largely been ignored in the scientific literature. Evaluation of sleep in relation to RWG during infancy is an important contribution as the impact of sleep on overweight and obesity risk has not been well-studied in infancy. Our careful consideration of other covariates in each statistical model also contribute important information to the literature by demonstrating that sleep influences obesity risk in early life independent of other known risk factors. While the sample size was relatively large for studies of vulnerable and underrepresented women and infants, generalizability may still be limited to the geographical location and characteristics

of this study cohort. Other limitations include the retrospective, self-reported sleep data available from screening tools rather than prospective data using objective measurements such as actigraphy. Objective measures would have allowed for granular scrutiny of sleep-wake patterns across the 24-hours and precluded limitations inherent to the BISQ. For example, according to the BISQ, nocturnal and diurnal sleep is defined as occurring between the hours of 7 PM to 8 AM and 8 AM to 7 PM, respectively. Thus, we could not account for infants with nocturnal sleep schedules that extended past 8 AM.

5 | CONCLUSION

Few daytime naps of may lead to disruptions in circadian sleep-wake development that may have links to future adiposity. Specifically, limiting the number of daytime sleep episodes as early as 1 month of age may increase the risk for RWG in infants, while an imbalance in nocturnal and daytime sleep at 1 month may be an important indicator of OW through early childhood. These findings indicate the importance of early life sleep needs for obesity prevention in the context of other risk factors inherent to high-risk ethnic populations. Future work should be aimed at understanding how infant sleep recommendations may need to be modified to address challenges experienced by low-income minority women in the United States including family dynamics, shift-work, socioeconomic status, acculturation and maternal health status (eg, elevated body mass). Continued understanding and more rigorous assessment of how sleep influences the risk for RWG and whether other behavioural factors can reverse the adverse health consequences associated with RWG by 6 months of age will also be important for identifying best practices for intervention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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TABLE 1

Maternal and infant sample characteristics by rapid weight gain (RWG) (N = 126) and overweight (OW) status (n = 96)

		RWG status				OW status			
Variable	**Overall (N = 126)**	**RWG (n = 45)**	**No RWG (n = 81)**	**X^2/t**	***P***	**OW (n = 41)**	**No OW (n = 55)**	**X^2/t**	***P***
Infant sex; female, n (%)	55 (43.7)	20 (36.4)	25 (35.2)	0.02	.89	13 (31.7)	24 (43.6)	1.41	.24
Infant birth weight-for-age Z score M (SD)	0.42 (0.92)	0.01 (0.79)	0.66 (0.91)	4.07	<.001	0.51 (0.86)	0.33 (0.88)	−0.99	.33
Maternal education n (%)				1.13	.29			9.23	.002
<High school graduate	64 (50.8)	20 (44.4)	44 (54.3)			27 (65.9)	19 (34.5)		
≥High school graduate	62 (49.2)	25 (55.6)	37 (45.7)			14 (34.1)	36 (65.5)		
Maternal parity, n (%)				2.11	.04			−1.36	.18
0	32 (25.4)	15 (33.3)	17 (21.0)			9 (22.0)	13 (23.6)		
1	35 (27.8)	13 (28.9)	22 (27.2)			8 (19.5)	18 (32.7)		
2	27 (21.4)	10 (22.2)	17 (21.0)			13 (31.7)	11 (20.0)		
≥3	32 (25.5)	7 (15.5)	25 (30.9)			11 (26.8)	13 (23.6)		
Maternal prenatal BMI, M (SD)	35.9 (5.2)	36.8 (5.4)	35.4 (5.0)	−1.45	.15	36.6 (5.4)	35.1 (5.3)	−1.40	.17
Gestational weight gain (lb), M (SD)	25.3 (11.1)	23.1 (10.6)	26.5 (11.3)	1.69	.09	25.3 (10.6)	25.4 (11.3)	0.08	.94
Months breastfed, M (SD)	4.4 (4.8)	3.3 (4.7)	4.99 (4.7)	1.93	.06	4.9 (5.2)	4.8 (4.8)	−0.11	.92
Randomized to active intervention, n (%)	64 (50.8)	18 (40.0)	46 (56.8)	3.26	.07	20 (48.8)	28 (50.9)	0.04	.84

Abbreviations: BMI, body mass index; OW, overweight by 36 mo; RWG, rapid weight gain by 6 mo.

TABLE 2

Sleep characteristics at 1 mo of age by RWG and OW status

	Overall	RWG				OW			
Sleep variable	**M (SD) or n(%)**	**RWG (n = 45)**	**No RWG (n = 81)**	**X^2/t**	***P***	**OW (n = 41)**	**No OW (n = 55)**	**X^2/t**	***P***
Bedtime (hh:mm)	9:52 (0:57)	9:55 (0:56)	9:55 (0:57)	−0.48	.63	9:44 (1:02)	9:51 (0:51)	0.55	.59
Nap frequency	3.8 (1.3)	3.6 (1.0)	4.0 (1.4)	1.96	.05	4.1 (1.3)	3.8 (1.1)	−1.18	.24
Nap frequency (top quartile: ≥5)	23 (18.3)	**2 (4.4)**	**21 (25.9)**	**9.0**	**.003**	9 (22.0)	9 (16.4)	0.48	.49
Longest nocturnal sleep bout (h)	4.6 (1.7)	4.6 (1.5)	4.5 (1.8)	−0.48	.63	4.6 (1.6)	4.5 (1.6)	−0.36	.72
Longest nocturnal sleep bout (top quartile: ≥5)	47 (37.3)	19 (42.2)	28 (34.6)	0.73	.40	15 (36.6)	20 (36.4)	0.00	.98
Nocturnal TST (h)	9.1 (1.4)	9.2 ± 1.2	9.1 (1.4)	−0.13	.90	**9.6 (1.3)**	**9.1 (1.3)**	**−2.11**	**.037**
Diurnal TST (h)	7.1 (1.7)	7.1 (1.71)	7.2 (1.8)	0.33	.74	7.2 (1.7)	7.5 (1.5)	1.01	.32
24-h TST (h)	16.3 (2.4)	16.2 (2.3)	16.3 (2.5)	0.15	.87	16.8 (2.5)	16.5 (2.2)	−0.53	.60
24-h TST categories[a]				1.0	.61			3.71	.16
<14	21 (16.7)	6 (13.3)	15 (18.5)				6 (14.6)		
14-17	63 (50.0)	25 (55.6)	38 (46.9)				15 (36.6)		
>17	42 (33.3)	14 (31.1)	28 (34.6)				20 (48.8)		
Difference in Nocturnal and Diurnal TST (h)	2.0 (1.9)	2.1 (1.9)	2.0 (2.0)	−0.38	.70	**2.5 (1.7)**	**1.6 (1.8)**	**−2.52**	**.013**

Abbreviation: TST, total sleep time.

[a]Based upon National Sleep Foundation recommendations.[31]

TABLE 3

Associations between sleep predictors at 1 mo with incident RWG from birth to 6 mo, and incident OW at 36 mo

	RWG				OW			
	Model 1[a]		Model 2[b]		Model 1[a]		Model 2[c]	
Sleep variable	**OR**	**95%CI**	**OR**	**95%CI**	**OR**	**95%CI**	**OR**	**95%CI**
Bedtime (30-min bins)	1.07	0.87, 1.32	1.09	0.86, 1.37	0.94	0.75, 1.17	0.91	0.70, 1.19
Nap frequency (no.)	0.74	0.53, 1.05	0.77	0.55, 1.09	1.23	0.86, 1.75	1.37	0.90, 2.09
Nap frequency (Ref. <5)[d]	**0.12**	**0.03, 0.58**	**0.11**	**0.02, 0.63**	1.39	0.49, 3.92	2.16	0.63, 7.46
Longest nocturnal sleep bout (h)	1.15	0.91, 1.45	1.19	0.91, 1.55	1.05	0.81, 1.36	0.99	0.74, 1.33
Longest nocturnal sleep bout (h) (Ref. <5) [d]	1.47	0.65, 3.29	1.48	0.62, 3.55	1.09	0.46, 2.57	1.08	0.41, 2.84
Nocturnal TST (h)	1.00	0.75, 1.34	1.03	0.75, 1.43	**1.42**	**1.02, 1.96**	1.41	0.99, 2.03
Diurnal TST (h)	0.99	0.79, 1.23	1.08	0.84, 1.37	**0.85**	**0.65, 1.11**	0.77	0.57, 1.06
24 h TST	0.99	0.85, 1.17	1.05	0.88, 1.25	1.04	0.87, 1.24	1.00	0.82, 1.23
24 h TST (Ref. 14-17 h)								
<14	0.53	0.17, 1.67	0.29	0.08, 1.01	2.32	0.62, 8.64	4.30	0.95, 19.47
>17	0.77	0.32, 1.81	0.63	0.23, 1.75	2.26	0.93, 5.51	2.74	0.94, 7.99
Night TST–Day TST (h)	1.01	0.83, 1.24	0.96	0.77, 1.19	**1.41**	**1.10, 1.83**	**1.51**	**1.13, 2.03**

Abbreviations: OW, overweight by 36 mo; RWG, rapid weight gain by 6 mo; TST, total sleep time.

[a] Adjusted for weight-for-age Z score at birth.

[b] Adjusted for weight-for-age Z score at birth, months breastfed (measured until 6 mo for RWG and 12 mo for OW), intervention assignment group, and maternal parity, prenatal body mass index, gestational weight gain, and education attainment.

[c] Adjusted for weight-for-age Z score at birth, RWG status at 6 mo of age, months breastfed (measured until 6 mo for RWG and 12 mo for OW), intervention assignment group, and maternal parity, prenatal body mass index, gestational weight gain, and education attainment.

[d] Dichotomized into the top quartile compared to all other quartiles.