

# NIH Public Access

Author Manuscript

Behav Sleep Med. Author manuscript; available in PMC 2015 September 03.

Published in final edited form as:

Behav Sleep Med. 2014 September 3; 12(5): 412-424. doi:10.1080/15402002.2013.825838.

# Advanced Sleep Phase in Adolescents Born Preterm

Anna Maria Hibbs, MD, MSCE<sup>1</sup>, Amy Storfer-Isser, MS<sup>2</sup>, Carol Rosen, MD<sup>1</sup>, Carolyn E. levers-Landis, PhD<sup>1</sup>, Elsie M. Taveras, MD, MPH<sup>3</sup>, and Susan Redline, MD, MPH<sup>4,5</sup>

<sup>1</sup>Department of Pediatrics, Rainbow Babies and Children's Hospital, Case Western Reserve University School of Medicine, Cleveland, OH

<sup>2</sup>Statistical Research Consultants, LLC., Perrysburg, Ohio

<sup>3</sup>Obesity Prevention Program, Department of Population Medicine, Harvard Pilgrim Health Care Institute and Harvard Medical School, Boston, MA

<sup>4</sup>Center for Clinical Investigation and Case Center for Transdisciplinary Research on Energetics and Cancer, Case Comprehensive Cancer Center, Case Western Reserve University School of Medicine, Cleveland, OH

<sup>5</sup>Department of Medicine, Brigham and Women's Hospital and Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA

# Abstract

**Objectives**—To evaluate whether sleep patterns and quality differed between adolescents born preterm and term. To further explore whether differences in sleep patterns were explained by differences in mediating factors, such as mood, behavior, or socioeconomic status.

**Methods**—Five-hundred one 16–19 year old children in the longitudinal Cleveland Children's Sleep and Health Study cohort underwent overnight polysomnography, wore wrist actigraphs, and completed sleep logs for a week. The modified Epworth Sleepiness Scale, the Adolescent Sleep Hygiene Scale, and the Adolescent Sleep Wake Scale were used to further assess sleep.

**Results**—Adolescents born preterm demonstrated significantly (p<0.05) earlier bed and wake times and sleep mid-points (approximately 22 minutes after adjusting for demographic and psychosocial factors) by actigraphy. They also had significantly fewer arousals (by polysomnography), and reported being e more rested and alert in the morning and less sleepiness and fatigue.

**Conclusions**—These findings support a growing body of evidence that perinatal factors may influence sleep phenotypes later in life. These factors may reflect developmental influences, as well as the influence of parenting styles on children's sleep.

# Introduction

Prematurity may have a life-long impact on sleep due to adverse *in utero* exposures or early neonatal stresses, both of which may influence the development of sleep-wake and circadian

The authors have no conflicts of interest to disclose.

control centers.<sup>1</sup> Mechanistically, there is a suggestion in the literature that preterm infants must complete suprachiasmatic nucleus maturation in an abnormal environment, frequently including non-ideal nutrition, stress, and hypoxia, as well as exposure to abnormal light conditions such as the constant light environment of neonatal intensive care units.<sup>1</sup> As shown in animal models and in a few studies of human infants, prenatal exposures, including hypoxia, protein restriction, and stress, may adversely affect the development of the suprachiasmatic nucleus, leading to phase advances.<sup>1–3</sup> Several studies from Finland also show that very low birth weight is associated with reduced sleep efficiency in eight-year-old children and with advanced sleep onset times in young adults.<sup>4, 5</sup> In addition, melatonin rhythmicity may develop more slowly in premature than term infants.<sup>2</sup>

Despite this physiological and clinical evidence supporting an influence of the prenatal and perinatal environment on the development of sleep-wake patterns, only limited research has used objectively measured sleep-wake patterns in large and well characterized samples of children born both at term and pre-term, and no study has yet examined this association in adolescents, a group typically phase-delayed and sleep deprived. In this study, we examined data from the Cleveland Children's Sleep and Health Study to evaluate whether objectively measured sleep patterns and quality differed between adolescents born preterm and term. We hypothesized that adolescents born preterm may be more phase advanced (i.e., routinely experience earlier bedtimes) and would have poorer sleep efficiency compared to their term peers due to the adverse influence of prenatal stresses on circadian and sleep development. We also sought to explore whether any differences in sleep patterns between preterm and term-born adolescents were explained by differences in other mediating factors, such as mood, behavior, or socioeconomic status.

## Methods

Subjects were adolescents participating in a longitudinal cohort study, the Cleveland Children's Sleep and Health Study (CCSHS). The CCSHS is a population-based cohort derived by recruiting a stratified random sample of 490 term and 417 preterm children born between 1988 and 1993 at three Cleveland area hospitals and studied initially between 1998 and 2002, as detailed previously.<sup>6</sup> Preterm infants were born less than 37 weeks gestational age and were admitted to the neonatal intensive care unit (NICU) for at least one week. Term infants were recruited from the normal newborn nursery. This analysis focuses on data collected at a follow-up examination conducted between 2006 and 2010 when the children were ages 16 to 19 years.<sup>7, 8</sup> Of the 517 subjects who participated in this exam. 501 participants did not have sleep apnea on polysomnography (i.e., apnea hypopnea index < 5) and constitute the analytical sample. Pubertal status had been assessed in approximately 70% of the sample (N=350). Over 99% of participants with known pubertal status were tanner stage 5. Institutional review boards at participating hospitals approved the protocol. For participants under age 18, the adolescent's legal guardian provided informed written consent, and the adolescent assented to participation; informed written consent was obtained from participants aged 18 and older.

Adolescents were invited to participate in an overnight clinical examination in a dedicated clinical research unit (CRU) when free from acute illness. Examinations at the research

center began at approximately 17:00 and ended the following day at 11:00; lights off time was generally 22:00 and lights on at 07:00. The examination included standardized polysomnography (PSG) and physiological and anthropometric assessments were performed using a standardized protocol, as described previously.<sup>7, 8</sup> Percentage of sleep time in each sleep stage was calculated using standard criteria for sleep stage scoring; given the age of the sample, apneas and hypopneas were scored according to 2007 American Academy of Sleep Medicine adult criteria (used for exclusion criteria only).<sup>9</sup>

Within one week of the CRU examination, participants were asked to wear a wrist actigraph and complete a daily sleep log for 5–7 consecutive 24-hour periods. Sleep-wake estimation was completed with wrist actigraphy (Octagonal Sleep Watch 2.01, Ambulatory Monitoring Inc, Ardsley, NY) and analyzed with the Action-W software and the Time-Above-Threshold algorithm.<sup>10</sup> Mean weekday sleep duration was calculated for participants with at least three weekdays of actigraphy data (n=360), and weekend results were included if at least 24 hours were recorded (n=336). Sleep mid-point was calculated as the average clock time between sleep onset and sleep offset on weekdays. Mean sleep efficiency, an objective measure of sleep continuity and quality, was defined as the percentage of time in bed estimated to be asleep (i.e., ((total time estimated to be asleep/total time in bed for the major sleep period) \*100) and was calculated using both actigraphy data (from a minimal of three weekdays) and from a single night of PSG data). Sleep-wake times were also reported by the adolescent in a 7-day sleep diary. Adolescents who were reported to snore loudly at least 1–2 times per week during the past month were categorized as snorers.

Socioeconomic status (SES) measures obtained at the time of adolescent follow-up included parent report of educational level and family income. A digital scale (Health-o-meter, Shelton, CT) and a rigid stadiometer (Holtain Ltd, Pembrokeshire, UK) were used to measure weight and height respectively.<sup>11</sup> BMI was calculated by dividing weight in kilograms by height in meters squared and was then converted into sex- and age-adjusted percentiles.

Questionnaires were used to obtain additional demographic, behavioral, and medical information from both adolescents and their parents. Adolescent daytime sleepiness was assessed with a modified version of the Epworth Sleepiness Scale (ESS).<sup>12</sup> ESS scores range from 0 to 24 with higher scores indicating greater daytime sleepiness; consistent previous studies, <sup>13–15</sup> a cut-off score of 11 or higher was used to identify adolescents with excessive daytime sleepiness.

The Revised Adolescent Sleep Hygiene Scale, which includes 24-items that comprised 6 subscales (physiological, cognitive/emotional, behavioral arousal, sleep environment, daytime sleep, and sleep stability), assesses behavioral practices that may influence the quality and duration of sleep. Subscale scores as well as the total score can range from 1 to 6 with higher scores indicating better sleep hygiene.<sup>16, 17</sup>

The Adolescent Sleep Wake Scale is a 28-item self-report research instrument used to assess adolescent sleep quality using 5 subscales: going to bed, falling asleep, maintaining sleep, reinitiating sleep, and returning to wakefulness. The total score as well as the subscale

scores range from 1 to 6 with higher scores indicating better sleep quality.<sup>16</sup> In addition, because psychosocial factors may relate to sleep, adolescents completed the Rosenberg Self-Esteem scale and profile of mood states (POMS) questionnaire (Educational and Industrial Testing Service, San Diego, CA).<sup>18, 19</sup> Parents completed the Stress Index for Parents of Adolescents (SIPA) scale and the Child Behavior Checklist (CBCL); composite scores indicating internalizing behaviors (withdrawn, anxiety/depression and somatic complaints), externalizing (delinquent and aggressive), and total problems, as well as school competencies, are reported.<sup>20, 21</sup> Using a standardized questionnaire, adolescents reported their daily caffeine consumption, categorized as none, < 1 cup, 1–3 cups, >3 cups.

#### **Statistical Analysis**

Characteristics of adolescents born preterm and term were summarized using medians and the interquartile range for non-normally distributed continuous measures, means and standard deviations for normally distributed variables, and counts and proportions for categorical variables. The two-sample t-test, Wilcoxon rank-sum test, and the Pearson chi-square test were used for bivariate comparisons of term and preterm adolescents, and effect sizes are summarized using Cohen's D,  $\rho$ , and Cramer's V, respectively. Multiple linear regression analyses were also used to assess the relation between term status and weekday sleep midpoint, adjusting for age, sex, race, and household income. Analyses were performed with SAS version 9.2 (SAS Institute, Inc, Cary, North Carolina).

# Results

Five-hundred seventeen participants in the CCSHS were assessed as adolescents. Compared to the 390 participants in the school-aged CCSHS cohort who were not evaluated as adolescents, the adolescent participants had significantly higher levels of parental education (p=0.03) and higher rates of prematurity (p=0.07). Specifically, 54% of the CCSHS participants born preterm and 56% of those whose parents had finished high school or a GED were assessed as adolescents. Participants and non-participants in the adolescent exam were similar with regards to age, sex, and race..

At the adolescent exam, 501 of the 517 participants did not have evidence of sleep apnea by PSG (obstructive apnea hypopnea index < 5) and therefore met inclusion criteria for this analysis. Of these, 284 (56.7%) were born full-term and 217 (43.3%) were born preterm. The mean  $\pm$  SD birth weight and gestational age for the preterm infants was  $1514 \pm 567$  g and  $31 \pm 3$  weeks, respectively. The preterm and full term infants were similar in regards to sex, race, body mass index, diagnosed attention deficit disorder, smoking and caffeine exposure, and snoring (Table 1). Compared to the full term group, the preterm children were on average 1.2 months younger, were more likely to be from lower-income households with less parental education, and had a higher frequency of asthma than the term group.

Sleep patterns, assessed objectively or by self-report, differed significantly between the term-born and preterm-born groups (Table 2). In particular, for the adolescents who were born preterm compared to term, weekday bedtime by both actigraphy and self-report were approximately 30 minutes earlier, and weekend bedtime was 30 minutes earlier using actigraphy data and 45 minutes earlier based on self-report. Morning wake times were also

approximately 20 minutes earlier in the preterm compared to the full term group. Thus, the adolescents born preterm had comparable total sleep duration as the full term children but were significantly relatively phase-advanced, as noted by the earlier sleep mid-point on weekdays (3:50 vs. 4:12; p=0.02) and weekends (5:17 vs. 5:41; p=0.01). No differences in sleep efficiency were observed. PSG characteristics were also examined and showed that compared to term-born subjects, preterm subjects had significantly fewer EEG arousals but had no difference in sleep stage distributions. Sleep latency tended to be shorter in the group born preterm, but this difference was not statistically significant (Table 2).

Compared to the term group, adolescents born preterm were significantly more likely to report functioning best in the morning (7.4% vs. 15.3%, p<0.01) and significantly less likely to report functioning worst in the morning (63.5% vs. 49.8%), p<0.01). Additionally, compared to the term group, the preterm group reported that it takes them significantly less time to "get going" (become fully alert and functional) after their usual sleep time in the morning. For instance, 27.8% of preterm and 18.2% of term participants reported that it takes them less than 5 minutes to "get going" (p<0.05).

The preterm group reported less daytime sleepiness as measured by the modified Epworth Sleepiness Scale as well as improved "returning to wakefulness" (a measure that includes feeling rested and alert and ease of getting out of bed in the morning) on the Adolescent Sleep Wake Scale (Table 3). Similarly, on the POMS, preterm adolescents reported higher levels of vigor and lower fatigue than the full term adolescents (Table 4). The groups did not differ in reported problems in initiating or maintaining sleep.

We also explored whether any differences in sleep hygiene, behavior and mood might explain an earlier bedtime. Sleep hygiene, as assessed by the Adolescent Sleep Hygiene Scale, showed a lower level of substance use (tobacco and alcohol use after 6pm) in the preterm compared to full term group of adolescents (Table 3). Post-hoc tests of the 2 items comprising the substances scaled showed that the preterm group reported consuming alcohol after 6 pm less often than the full term group, and there were no group differences in terms of using tobacco after 6 pm. There were no differences reported across the other subscales assessing other bedtime behaviors.

On the CBCL, adolescents born preterm scored higher on the internalizing behavior subscale (Table 4). Although, the average t-scores for both groups were in the normal range, the prevalence of clinically elevated internalizing behavior scores was almost twice as high among adolescents born preterm compared to full term (13.7% vs. 7.5%, p=0.03). SIPA scores were similar except for social isolation and withdrawal scores, which were higher in the preterm (48.6 ± 9.4) than the term (46.3 ± 8.5) groups (p=0.004), indicating greater parent report of symptoms of their children seeking less social interaction and preferring to be alone. Consistent with the results from self-reported levels of sleepiness, preterm adolescents (Table 4). No differences were seen between the preterm and term groups in self-esteem as measured by the Rosenberg scale.

To further assess whether the association between sleep timing and term status may have been due to confounding, multiple regression models were fit for actigraphy-based mid-sleep time. The results showed that prematurity was associated with an earlier mid-sleep time (approximately 22 minutes) after adjusting for age, race, sex, maternal education, and household income ( $\beta$ =-22.5, SE=9.2; p=0.02), as well as additionally adjusting for psychosocial factors found to be significantly different between the groups, including internalizing characteristics on the CBCL ( $\beta$ =-22.3, SE=9.3; p=0.02) and substance use on the ASHS scale ( $\beta$ =-21.6, SE=9.2; p=0.02), but resulting only in a 0.9% and 4.0% attenuation of the association of term status and mid-sleep time, respectively.

Post-hoc analyses showed that a significantly higher proportion of adolescents born preterm reported a regular bedtime compared to their full-term peers (60.2 vs. 43.6%, p <.001). Among participants with a regular bedtime, the bedtime was more often set by the parent for preterm group compared to full-term group (30.8 vs. 16.3%, p < 0.01).

## Discussion

Adolescents born preterm demonstrated earlier bed and wake times (resulting in an earlier sleep mid-point, approximating a 22 minute phase advance by actigraphy) while monitored at home with actigraphy, fewer arousals overnight while monitored with PSG, reported being more rested and alert in the morning, and reported less sleepiness and fatigue than their term-born peers. While the fewer arousals and less sleepiness, suggesting better sleep quality in the preterm as compared to full term adolescents, did not support our hypothesis that adverse *in-utero* and prenatal exposures would negatively affect sleep quality, the finding of a phase advance is consistent with animal data as well as with limited data in infants and young adults.<sup>1–3, 5</sup> Our findings also suggest that differences in sleep timing were not explained by differences in age, race, or income, evening alcohol and tobacco use, or internalizing behaviors. Increased parental influences on setting regular bedtime routines may explain at least part of this phenomenon.

Phase delay, characterized by late bedtimes and wake times, is characteristic of adolescence, and thought to be due to biological influences on circadian timing associated with puberty as well as to psycho-social influences.<sup>22</sup> When wake times are constrained by early school start times, insufficient sleep, daytime sleepiness and impaired performance may result. In this study, actigraphic records indicated that term children were getting to bed at approximately 00:13 and 1:18 on weekdays and weekends, respectively. However, bedtimes were approximately 30 minutes earlier in preterm adolescents, suggesting relatively less phase delay in these children. Even in analyses adjusted for age, sex, race and caregiver education, preterm adolescents had a sleep period that was shifted approximately 20 minutes earlier than term-born adolescents. Additionally, the preterm adolescents reported being significantly more rested and alert in the morning than the full term adolescents despite similar average sleep duration, supporting that term status is associated with differences in circadian timing in adolescence, with the preterm adolescents more likely to function well in the morning ("larks"). Our study thus provides new data confirmed by actigraphy that sleep timing measured during adolescence varies in association with term status. This observation is consistent with a study from Finland of a sample of young adults (ages 19 to 26 years)

that reported that bedtimes averaged 36 minutes earlier in subjects who were very low birth weight compared to individuals born at term.<sup>5</sup>

One apparent explanation for the observed phase advancement in the preterm adolescents seems to be an increased compliance of this group with parental instructions regarding earlier bedtimes. One might speculate that another factor could be a lesser influence of peer pressure or social factors that contribute to late bedtimes. Children and adolescents born both extremely preterm and late-preterm have been shown to have less risk taking behavior and more internalizing behaviors and social isolation,<sup>23–25</sup> as reflected in this cohort by decreased self-reported evening alcohol use and more parent-reported internalizing symptoms and social withdrawal.

The bases for a relative sleep phase advance among individuals born preterm may also reflect the biological effects of prenatal and post-natal exposures influencing multiple developmental stages and circadian biology. Physiological differences in circadian timing were suggested by differences in not only sleep mid-point, but also the improved morning alertness reported by adolescents who had been born pre-term. In animal models, prenatal conditions frequently encountered by preterm infants, such as hypoxia, stress, and protein restriction, are associated with adult phase advances.<sup>1, 3, 26</sup> Another possible explanation for the earlier sleep and wake times in the preterm group could be a relative immaturity causing a delaying or blunting of the normal sleep phase delay seen in adolescence. There could be a decrement or delay in the decrease in the sleep homeostatic drive that normally occurs in adolescents which contributes to the typical adolescent phase delay.

Sleep quality, measured by actigraphy or polysomnography, did not differ between the preterm and full term children. However, the preterm children had a small but significantly lesser number of nighttime arousals than the full term group. Subjects born preterm also reported better morning alertness, less fatigue and less sleepiness than their full term peers, suggesting overall better sleep quality. The prior literature is inconsistent on whether preterm or low birth weight is associated with altered sleep quality or duration, but has generally suggested poorer sleep quality in preterm patients or in animal models..<sup>6</sup>, 27–2930.312956, 32

The finding of a relative phase advance and improved sleep quality in the adolescents born preterm is of potential clinical importance given prior research showing that even modest differences in sleep behavior impact neurobehavioral functioning in school children.<sup>33</sup> The finding that the preterm adolescents also were significantly less sleepy and scored higher on vigor scores than term-born adolescents, suggest a beneficial effect of earlier bedtimes despite comparability in overall sleep duration. In adolescents, we have found that impaired executive function is more strongly associated with subjective sleepiness than with sleep duration, further underscoring the potential importance of this finding on neurocognitive function.<sup>34</sup> Therefore, despite a lack of difference in sleep duration between adolescents born preterm and term, earlier bedtimes, fewer arousals and less sleepiness may represent important markers of health and functioning. The observation that parents of preterm compared to full term children were more likely to influence bedtimes also indicates the potential for more directive parenting styles to positively influence adolescent sleep.

Study strengths included the use of objectively measured quantitative sleep indices from actigraphy and polysomnography and the availability of a comprehensive assessment of well-validated and reliable behavioral and psychological measures in a sample of adolescents who were born preterm and term. However, since some of the mediating or confounding factors were assessed by questionnaires, it is possible that misclassification may have reduced the ability to detect some associations. In general however, effect sizes were larger for self-report measures compared to actigraphy-based measures, and smaller effect sizes were observed for PSG-based measures. Information on environmental variables, such as light exposure and specific behaviors such as nighttime TV watching, were not available and may have helped further identify exposure differences between the study groups. In addition, biomarkers of circadian rhythm and validated measures of circadian preference were not obtained Although the sample was moderately large, weekday and weekend actigraphy data were missing or incomplete for 28% and 33% of participants studied at the late adolescent exam. However, a similar relative phase advancement is seen with both actigraphy and self-report in this study. Good agreement among mid-sleep data, morning-eveningness scales, and chronotype questionnaires have previously been demonstrated.<sup>32</sup> Furthermore, only 57% of the original cohort was available and willing to participate in an exam occurring approximately 8 years after initial enrollment, potentially introducing selection biases. While there were no significant differences by age, sex and race between those who did and did not participate at the third exam, adolescents born at term and adolescents with less educated parents were less likely to participate at the third exam. While several analyses were performed, allowing the possibility for spurious positive results, all of our findings were internally consistent, supporting the validity of the findings.

In summary, compared to adolescents born at term, those born preterm experienced less sleep delay, fewer arousals during sleep, improved morning alertness and reported less sleepiness and fatigue. These observations were not explained by the behavioral differences measured in the preterm and full term children and support prior research suggesting the importance of pre- and early post-natal factors influencing the circadian pacemaker. Growing research is focused on the relationship of childhood sleep and health outcomes, such as obesity, and has emphasized the role of interventions aimed at the child's sleep behaviors. Our work supports the idea of a "prenatal origin" for sleep traits and the potential importance of prenatal and early postnatal factors in contributing to pediatric sleep phenotypes, and thus, their role as targets for intervention. Future research is needed to address how the biologic and psychosocial impacts of perinatal stressors influence the development of sleep-wake and circadian processes in children born preterm. This study also points supports a positive influence of parenting style on the sleep of adolescents, and suggests that interventions targeting sleep routines may be useful in improving sleep patterns.

## Acknowledgments

This work was supported by NIH grants: NIH HL07567, HL60957, UL1-RR024989, 1U54CA116867, K23 HD056299, and the Harvard Transdisciplinary Research in Energetics and Cancer Center (1U54CA155626)

# References

- 1. Kennaway DJ. Programming of the fetal suprachiasmatic nucleus and subsequent adult rhythmicity. Trends Endocrinol Metab. 2002; 13:398–402. [PubMed: 12367822]
- Kennaway DJ, Goble FC, Stamp GE. Factors influencing the development of melatonin rhythmicity in humans. J Clin Endocrinol Metab. 1996; 81:1525–32. [PubMed: 8636362]
- Koehl M, Barbazanges A, Le Moal M, Maccari S. Prenatal stress induces a phase advance of circadian corticosterone rhythm in adult rats which is prevented by postnatal stress. Brain Res. 1997; 759:317–20. [PubMed: 9221956]
- 4. Pesonen AK, Raikkonen K, Matthews K, et al. Prenatal origins of poor sleep in children. Sleep. 2009; 32:1086–92. [PubMed: 19725260]
- Strang-Karlsson S, Raikkonen K, Kajantie E, et al. Sleep quality in young adults with very low birth weight--the Helsinki study of very low birth weight adults. J Pediatr Psychol. 2008; 33:387–95. [PubMed: 18077474]
- Rosen CL, Larkin EK, Kirchner HL, et al. Prevalence and risk factors for sleep-disordered breathing in 8- to 11-year-old children: association with race and prematurity. J Pediatr. 2003; 142:383–9. [PubMed: 12712055]
- 7. Javaheri S, Storfer-Isser A, Rosen CL, Redline S. Association of short and long sleep durations with insulin sensitivity in adolescents. J Pediatr. 2011; 158:617–23. [PubMed: 21146189]
- Weiss A, Xu F, Storfer-Isser A, Thomas A, Ievers-Landis CE, Redline S. The association of sleep duration with adolescents' fat and carbohydrate consumption. Sleep. 2010; 33:1201–9. [PubMed: 20857867]
- Rechtschaffen, A.; Kales, A. A manual of standardized techniques and scoring system for sleep stages of human subjects. Washington, D.C: US Government Printing Office; 1968. NIH Publication No. 204
- Johnson NLKH, Rosen CL, Storfer-Isser A, Cartar LN, Ancoli-Israel S, Emancipator JL, Kibler AM, Redline S. Sleep estimation using wrist actigraphy in adolescents with and without sleep disordered breathing: a comparison of three data modes. Sleep. 2007; 30:899–905. [PubMed: 17682661]
- Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. Adv Data. 2000:1–27. [PubMed: 11183293]
- Johns MW. Reliability and factor analysis of the Epworth Sleepiness Scale. Sleep. 1992; 15:376– 81. [PubMed: 1519015]
- Campbell IG, Higgins LM, Trinidad JM, Richardson P, Feinberg I. The increase in longitudinally measured sleepiness across adolescence is related to the maturational decline in low-frequency EEG power. Sleep. 2007; 30:1677–87. [PubMed: 18246977]
- Johnson NL, Kirchner HL, Rosen CL, et al. Sleep estimation using wrist actigraphy in adolescents with and without sleep disordered breathing: a comparison of three data modes. Sleep. 2007; 30:899–905. [PubMed: 17682661]
- Redline S, Storfer-Isser A, Rosen CL, et al. Association between metabolic syndrome and sleepdisordered breathing in adolescents. Am J Respir Crit Care Med. 2007; 176:401–8. [PubMed: 17541017]
- LeBourgeois MK, Giannotti F, Cortesi F, Wolfson AR, Harsh J. The relationship between reported sleep quality and sleep hygiene in Italian and American adolescents. Pediatrics. 2005; 115:257–65. [PubMed: 15866860]
- 17. Storfer-Isser AMKL, Harsh J, Tompsett CJ, Redline S. Psychometric properties of the adolescent sleep hygiene scale (ASHS). Journal of Sleep Research. in press.
- Rosenberg, M. Society and the adolescent self-image. Princeton, NJ: Princeton University Press; 1965.
- Watson D, Clark L, Tellegen A. Development and validation of brief measures of positive and negative affect. Journal of Personality and Social Psychology. 1988; 54(6):1063–70. [PubMed: 3397865]
- 20. Achenbach, T.; Rescorla, L. Manual for the ASEBA School-Age Forms and Profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, and Families; 2001.

- 21. Sheras, P.; Abidin, R.; Konold, T. Stress Index for Parents of Adolescents:Professional Manual. Lutz, FL: Psychological Assessment Resources; 1998.
- Carskadon MA, Vieira C, Acebo C. Association between puberty and delayed phase preference. Sleep. 1993; 16:258–62. [PubMed: 8506460]
- Hack M, Cartar L, Schluchter M, Klein N, Forrest CB. Self-perceived health, functioning and wellbeing of very low birth weight infants at age 20 years. J Pediatr. 2007; 151:635–41. 41, e1–2. [PubMed: 18035144]
- 24. Hack M, Klein N. Young adult attainments of preterm infants. JAMA. 2006; 295:695–6. [PubMed: 16467241]
- Talge NM, Holzman C, Wang J, Lucia V, Gardiner J, Breslau N. Late-preterm birth and its association with cognitive and socioemotional outcomes at 6 years of age. Pediatrics. 126:1124– 31. [PubMed: 21098151]
- 26. Koehl M, Darnaudery M, Dulluc J, Van Reeth O, Le Moal M, Maccari S. Prenatal stress alters circadian activity of hypothalamo-pituitary-adrenal axis and hippocampal corticosteroid receptors in adult rats of both gender. J Neurobiol. 1999; 40:302–15. [PubMed: 10440731]
- Gossel-Symank R, Grimmer I, Korte J, Siegmund R. Actigraphic monitoring of the activity-rest behavior of preterm and full-term infants at 20 months of age. Chronobiol Int. 2004; 21:661–71. [PubMed: 15470961]
- Iglowstein I, Latal Hajnal B, Molinari L, Largo RH, Jenni OG. Sleep behaviour in preterm children from birth to age 10 years: a longitudinal study. Acta Paediatr. 2006; 95:1691–3. [PubMed: 17129987]
- Wolke D, Sohne B, Riegel K, Ohrt B, Osterlund K. An epidemiologic longitudinal study of sleeping problems and feeding experience of preterm and term children in southern Finland: comparison with a southern German population sample. J Pediatr. 1998; 133:224–31. [PubMed: 9709710]
- Dugovic C, Maccari S, Weibel L, Turek FW, Van Reeth O. High corticosterone levels in prenatally stressed rats predict persistent paradoxical sleep alterations. J Neurosci. 1999; 19:8656–64. [PubMed: 10493766]
- Asaka Y, Takada S. Activity-based assessment of the sleep behaviors of VLBW preterm infants and full-term infants at around 12 months of age. Brain Dev. 32:150–5. [PubMed: 19136227]
- Paavonen EJ, Strang-Karlsson S, Raikkonen K, et al. Very low birth weight increases risk for sleep-disordered breathing in young adulthood: the Helsinki Study of Very Low Birth Weight Adults. Pediatrics. 2007; 120:778–84. [PubMed: 17908765]
- 33. Sadeh A, Gruber R, Raviv A. The effects of sleep restriction and extension on school-age children: what a difference an hour makes. Child Dev. 2003; 74:444–55. [PubMed: 12705565]
- Anderson B, Storfer-Isser A, Taylor HG, Rosen CL, Redline S. Associations of executive function with sleepiness and sleep duration in adolescents. Pediatrics. 2009; 123:e701–7. [PubMed: 19336360]

#### Table 1

#### Participant Characteristics<sup>†</sup>

Subject Characteristics	Full-term (n=284)	Preterm (n=217)	<i>p</i> -value
Age	$17.8\pm0.4$	$17.7\pm0.4$	0.004
Male sex	138 (48.6%)	108 (49.8%)	0.79
African-American Race	101 (35.6%)	83 (38.3%)	0.54
BMI z-score	$0.62 \pm 1.03$	$0.52 \pm 1.05$	0.31
Habitual loud snoring			
No	199 (70.1%)	153 (70.5%)	
Yes	59 (20.8%)	48 (22.1%)	0.75
Unknown	26 (9.2%)	16 (7.4%)	0.75
Current diagnosis of asthma	37 (13.0%)	49 (22.8%)	0.004
Current ADHD	34 (12.0%)	19 (8.8%)	0.26
Current smoking	22 (7.8%)	16 (7.4%)	0.88
Caffeine intake (cups per day)			
None	33 (11.7%)	28 (13.0%)	
< 1 cup	93 (33.0%)	55 (25.6%)	
1 cup	56 (19.9%)	41 (19.1%)	
1–3 cups	71 (25.2%)	64 (29.8%)	0.41
>3 cups	29 (10.3%)	27 (12.6%)	
Parent/Family characteristics			
Household income			
<\$30k	52 (18.3%)	50 (23.0%)	
\$30 - \$49k	26 (9.1%)	33 (15.2%)	
\$50 - \$74k	42 (14.8%)	45 (20.7%)	
\$75 – \$99k	36 (12.7%)	29 (13.4%)	
\$100 - \$149k	52 (18.3%)	23 (10.6%)	-0.001
\$150k	46 (16.2%)	12 (5.5%)	<0.001
Missing / no answer	30 (10.6%)	25 (11.5%)	
Parent education			
Less than high school	14 (5.2%)	15 (7.3%)	
High school / GED	37 (13.8%)	38 (18.5%)	
Vocational school / Associates	33 (12.3%)	48 (23.4%)	
Some college courses towards a 4-yr degree	41 (15.2%)	36 (17.6%)	-0.001
4-yr college degree (BA, BS)	64 (23.8%)	38 (18.5%)	<0.001
Beyond college	80 (29.7%)	30 (14.6%)	

 $^{\dagger}$  Mean  $\pm$  SD shown for normally distributed measures; N (%) shown for categorical measures.

#### Table 2

# Sleep Characteristics by Term Status $^{\dagger}$

	Full-term (n=284)	Preterm (n=217)	<i>p</i> -value	Effect Size
Self-Reported Weekday Sleep	Duration			
Weekday sleep duration	$469\pm105$	$486\pm98$	0.06	0.17
Weekday bedtime	$23{:}39\pm1{:}18$	$23{:}06\pm1{:}19$	< 0.001	0.42
Weekday waketime	$7{:}28 \pm 1{:}55$	$7{:}12\pm1{:}56$	0.14	0.14
Weekday mid-sleep	$3:33 \pm 1:23$	$3{:}10\pm1{:}25$	0.003	0.27
Self-Reported Weekend Sleep	Duration			
Weekend sleep duration	$547 \pm 102$	$564\pm104$	0.08	0.16
Weekend bedtime	$1{:}07\pm1{:}24$	$00{:}24\pm1{:}16$	< 0.001	0.53
Weekend wake time	$10{:}14\pm1{:}53$	$9{:}48 \pm 1{:}46$	0.01	0.24
Weekend mid-sleep	$5{:}43 \pm 1{:}35$	$5{:}06\pm1{:}17$	< 0.001	0.42
Actigraphy Weekday Sleep*				
Weekday sleep duration	$449\pm73$	$451\pm 63$	0.74	0.04
Weekday bedtime	$00{:}13\pm1{:}20$	$23{:}54\pm1{:}22$	0.05	0.23
Weekday wake time	$8{:}05\pm1{:}38$	$7{:}44 \pm 1{:}36$	0.04	0.22
Weekday mid-sleep	$4{:}12\pm1{:}21$	$3:\!50\pm1:\!23$	0.02	0.26
Weekday sleep duration				
< 6.5 hours	36 (17.8%)	27 (17.1%)		
6.5 - 8.49 hours	129 (63.9%)	104 (65.8%)	0.93	0.02
8.5 hours	37 (18.3%)	27 (17.1%)		
Weekday sleep efficiency	97.0 (94.3, 98.3)	97.1 (94.5, 98.2)	0.79	0.01
Actigraphy Weekend Sleep**	ĸ			
Weekend sleep duration	$480\pm104$	$498\pm89$	0.09	0.19
Weekend bedtime	$1{:}18\pm1{:}41$	$0{:}46\pm1{:}37$	0.004	0.32
Weekend wake time	$9:52 \pm 1:50$	$9{:}35\pm1{:}39$	0.14	0.16
Weekend mid-sleep	5:41 ± 1:34	$5:17 \pm 1:26$	0.01	0.27
Weekend sleep efficiency	96.8 (93.1, 98.4)	97.0 (94.1, 98.6)	0.49	0.04
PSG Sleep Architecture				
Sleep efficiency	89.1 (82.8, 93.7)	89.9 (82.8, 94.1)	0.40	0.04
Sleep onset latency				
< 5 min	109 (38.4%)	99 (46.0%)		
5–19 min	63 (22.2%)	52 (24.2%)	0.08	0.10
20 min	112 (39.4%)	64 (29.8%)		
Arousal index	8.3 (6.4, 10.9)	7.4 (5.9, 9.6)	0.006	0.12
Total sleep time (min)	$461\pm71$	$472\pm 66$	0.08	0.16
% time stage 1	$4.1\pm2.6$	$4.1\pm2.5$	0.76	0.03
% time stage 2	$51.8\pm7.2$	$52.1\pm7.6$	0.64	0.04
% time stage 3–4	$23.3\pm8.0$	$23.2\pm8.0$	0.82	0.02
% time REM	$20.7\pm5.4$	$20.7\pm5.1$	0.87	0.02

\* n=202 full-term and n=158 preterm participants;

# \*\* n=190 full term and n=146 preterm participants

<sup> $\dagger$ </sup>Mean ± SD and Cohen's *D* effect size shown for normally distributed measures; median (25<sup>th</sup>, 75<sup>th</sup> percentiles) and  $\rho$  effect size shown for non-normally distributed measures; N (%) and Cramer's *V* effect size shown for categorical measures.

#### Page 14

#### Table 3

Subjective Sleepiness, Sleep Habits and Sleep Hygiene by Term Status<sup>†</sup>

	Full-term (n=284)	Preterm (n=217)	<i>p</i> -value	Effect Size
Subjective Sleepiness				
Modified ESS	$8.2\pm3.7$	$6.8\pm3.9$	< 0.001	0.39
ESS > 10	90 (31.8%)	37 (17.1%)	< 0.001	0.17
Adolescent Sleep Wake Scale	* e			
Total sleep quality	$4.39\pm0.63$	$4.44\pm0.61$	0.34	0.09
Going to bed	$4.17 \pm 1.03$	$4.12 \pm 1.02$	0.62	0.05
Falling asleep	$4.53\pm0.84$	$4.46\pm0.86$	0.35	0.09
Maintaining sleep	$4.79\pm0.87$	$4.83\pm0.76$	0.54	0.06
Reinitiating sleep	$5.15\pm0.63$	$5.18 \pm 0.64$	0.54	0.06
Returning to wakefulness	$3.30 \pm 1.08$	$3.61 \pm 1.08$	0.002	0.29
Adolescent Sleep Hygiene Sc	cale *			
Total sleep hygiene	$4.30\pm0.70$	$4.32\pm0.58$	0.81	0.02
Physiological	$4.70\pm0.86$	$4.76\pm0.79$	0.40	0.08
Cognitive/Emotional	$4.36\pm0.94$	$4.36\pm0.97$	0.99	0.00
Behavioral Arousal	$3.47 \pm 1.19$	$3.40 \pm 1.23$	0.56	0.05
Sleep Environment	$5.06\pm0.81$	$5.04\pm0.77$	0.78	0.03
Daytime Sleep	$4.71 \pm 1.22$	$4.84 \pm 1.06$	0.21	0.11
Sleep Stability	$3.52 \pm 1.17$	$3.50\pm1.06$	0.83	0.02

\* higher scores are *better* 

<sup>†</sup>Normally distributed measures: mean ± SD and Cohen's D effect size shown; Categorical measures: N (%) and Cramer's V effect size reported.

#### Table 4

#### Mood and Behavioral Measures by Term Status $^{\dagger}$

	Full-term (n=284)	Preterm (n=217)	<i>p</i> -value	Effect Size
Mood: POMS t-scores *				
Tension	$45.1\pm6.7$	$44.7\pm7.4$	0.51	0.06
Depression	$45.4\pm6.5$	$45.4\pm7.4$	0.96	0.00
Anger	$44.2\pm7.1$	$44.0\pm7.2$	0.73	0.03
Vigor (higher is better)	$38.5\pm8.0$	$40.9\pm9.0$	0.002	0.29
Fatigue	$45.2\pm8.0$	$42.8\pm7.3$	< 0.001	0.31
Confusion	$48.6\pm7.0$	$47.5\pm7.3$	0.10	0.15
Total mood disturbance	$48.1\pm 6.3$	$46.9\pm7.1$	0.04	0.19
Behavior: CBCL t-scores *				
Total Competence (higher is better)	47.9 + 11.0	46.0 + 11.1	0.08	0.17
Total Problems	$45.3 \pm 11.4$	46.7 + 11.4	0.20	0.12
Externalizing	$46.6\pm10.2$	46.6 + 10.3	0.98	0.00
Internalizing	$46.6\pm10.4$	$48.7 \pm 11.0$	0.03	0.20

\* higher scores are worse unless otherwise indicated

 $^{\dagger}$ Mean ± SD and Cohen's *D* effect size shown.