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Influence of Asthma Status on Sleep Variability in Overweight/Obese Youth

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Abstract

Objective: Pediatric asthma and overweight/obesity (OV/OB) frequently co-occur and youth with both conditions exhibit poor sleep/bedtime habits. This study assessed differences in week-to-weekend bedtime/wake time variability among OV/OB youth with/without comorbid asthma, and tested whether variability predicted weekday sleep.

Methods: OV/OB youth (n = 142; 28% comorbid asthma; 7–12 years) wore an Accelerometer for 5 days (2 weekend days), providing estimates of week-to-weekend bedtime/wake-time variability, weekday Total Sleep Time (TST), weekday time in bed (TIB), and weekday wake after sleep onset (WASO).

Results: There were no demographic differences between groups beyond lower family income for the OV/OB+asthma group. The OV/OB+asthma group exhibited later weekday (mean OV/OB+asthma=10:39pm, mean OV/OB only=10:30pm) and weekend (mean OV/OB+asthma=11:41pm, mean OV/OB only=11:17pm) bedtimes, earlier weekday waketimes (mean OV/OB+asthma=6:40am, mean OV/OB only=6:51am), and similar weekend waketimes (mean OV/OB+asthma=7:54pm, mean OV/OB only=7:52pm). Univariate MANOVA follow-ups indicated a main effect of asthma group for week-to-weekend bedtime and waketime variability, with the OV/OB+asthma group evidencing approximately 30 minutes greater bedtime (OV/OB+asthma mean = 90 minutes) and waketime (OV/OB+asthma mean = 108 minutes) variability. Within the OV/OB+asthma group, greater waketime variability predicted fewer minutes of weekday TIB and WASO. Within the OV/OB only group, wake time variability predicted fewer minutes of weekday TIB.

Conclusion: Findings suggest that asthma status confers risk for more week-to-weekend variability among currently OV/OB youth, and that greater variability shortens the weekday sleep period. Further research on reasons for greater week-to-weekend sleep variability in asthma is needed.

Keywords

Sleep medicine; Pulmonology; Pediatric; Child

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Declaration of Interest Statement

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Introduction

Pediatric asthma and obesity are the most common chronic health conditions of childhood [1, 2], and are often co-morbid. Youth with asthma have a higher prevalence of overweight/obesity (OV/OB) than their healthy peers [3] and increased weight status is a risk factor for future development of asthma [4, 5]. Comorbid asthma and OV/OB is associated with poorer asthma outcomes, including poorer asthma control [8], more prescribed medications [7], and more frequent emergency department visits [9]. Comorbid status also likely confers synergistic risk for other negative outcomes, including sleep disturbance.

Sleep is integral to the health and development of children [10–12]. Both children with asthma or OV/OB exhibit higher rates of sleep disordered breathing [14, 15], short sleep duration [16, 17], and frequent nighttime arousals [15, 18]. The effects of sleep disruption in asthma are broad, and include increased symptom severity following a night of poor sleep [19], increased incidence of emergency department visits [18–20], and poor psychosocial outcomes, including more frequent school absences, activity limitations, and lower quality of life [20].

Few pediatric studies address sleep difficulties associated with comorbid asthma and OV/OB. In adults, comorbid asthma and obesity is associated with greater sleep difficulties [21]. Two of the few pediatric studies find that youth with comorbid OV/OB+asthma obtain shorter sleep durations than their normal weight peers with asthma [22], as well as their OV/OB peers without asthma [23]. In addition to “biologically”-driven sleep decrements (e.g. sleep apnea), youth with both conditions independently exhibit high rates of problematic, yet modifiable, bedtime habits. For example, youth with asthma exhibit poorer sleep hygiene than healthy counterparts [24], and heavier children exhibit later bedtimes and greater engagement in screen time directly prior to sleep [25].

Consistent week-to-weekend sleep schedules are a critical aspect of best sleep practices for youth [27], and are often a core component of pediatric behavioral sleep intervention. Greater night-to-night sleep variability is associated with anxiety, depression, and fatigue among adolescents [28] and adverse metabolic outcomes among children [29]. However, no pediatric studies to date have established how variable sleep schedules influence weeknight sleep. In children, variability in bed and wake time (e.g., staying up and waking up later on the weekends) may cause problematic circadian shifts in the sleep cycle. For example, as environmental cues (e.g. sleepovers, later social engagements) push the Friday bedtime later, the brain’s endogenous biological clock, which controls daily rhythms associated with sleep and wake, naturally shifts later in parallel [26]. Thus, when a child rapidly attempts to shift bedtime earlier on Sunday night, the biological rhythms that promote sleepiness (e.g. drop in core body temperature, increases in circulating melatonin) have not yet occurred, creating a biological state that is not conducive to falling asleep.

Given that variable schedules create circadian dysfunction among adults [30], it is likely that greater week-to-weekend variability in sleep patterns create weeknight sleep disturbance among children, as well. Further, inconsistent sleep schedules may be particularly

detrimental among youth with asthma or OV/OB, as both groups appear to be biologically and behaviorally predisposed to sleep problems. Thus, this study examined the impact of asthma status on weekday-to-weekend variability in bed and wake times among a sample of OV/OB youth with and without asthma. The study further examined and how weekday-to-weekend variability influences weekday sleep differentially by group. We hypothesized that asthma status would confer greater risk for poor sleep outcomes, and specifically that the comorbid OV/OB+asthma group would exhibit greater week-to-weekend bedtime and wake time variability than the OV/OB only group. As an exploratory aim, we explored whether weekday-to-weekend variability was predictive of weekday time spent in bed, total sleep time, and minutes spent in wake after first sleep onset.

Method

Participants

Participants were 142 overweight or obese children (male = 77) ages 7–12 years old who lived in [omitted for blind review]. Overweight/obese status was defined as a body mass index (BMI) at or above the 85th percentile for CDC-published age and gender norms. Participants were part of a larger randomized-controlled clinical trial evaluating the efficacy of a behavioral lifestyle intervention to improve child weight status in rural overweight and obese children. Participant demographics of interest included child age, gender, race/ethnicity, family income, and marital status of the primary caregiver, given that children with asthma are more likely to be ethnic minorities [31] and come from low-income households with unmarried parents [32], and older age is associated with changes in sleep [33].

Procedure

Data from this study was taken as part of a larger randomized controlled clinical trial. See [citation omitted for blind review] for a complete description of intervention design and methodology. All data used in this study were collected pre-intervention at the baseline assessment and were part of secondary data analysis. Researchers recruited families through community outreach efforts. Interested families were deemed eligible via phone screener if the child was 8–12 years old at initiation of treatment, resided in a rural county, had a body mass index (BMI) at or above the 85th percentile for CDC-published age and gender norms, and if the parent or legal guardian resided with the child and was younger than age 75. Families were excluded for parent or child contraindicated medical conditions (e.g. hypertension), child contraindicated medications (e.g., antipsychotic agents), extreme child oppositional behaviors or severe cognitive/developmental delays, or if the caretaker or child was participating in another weight management program. Following the phone screening, eligible participants completed an in-person screening, where parents provided oral and written consent to participate in the study. There, researchers measured child height and weight to confirm BMI and parents completed demographic questionnaires. Child height was measured to the nearest 0.1 centimeter using a Harpedon Stadiometer and weight was measured to the nearest 0.1 kilogram using a digital scale. Approximately 1 to 2 months later (1 to 2 weeks prior to treatment), eligible families returned and researchers instructed the children on Accelerometer procedures. Children were instructed to wear the device 24 hours/day for seven consecutive days, with the exception of time spent bathing or

swimming. The Accelerometer provided all sleep data used in these analyses. Children were again measured for height and weight. The [omitted for blind review] Institutional Review Board approved study procedures and all parents or legal guardians provided written informed consent prior to participating.

Measures

Asthma Status.—Study personnel assessed basic demographic data during the initial screening visit via a parent-completed background questionnaire, which included the question: *Has a doctor or health care professional ever said your child has asthma?* This question was used to define asthma status, consistent with methods commonly used among epidemiological studies [34].

Objective Sleep Measure.—Child sleep was objectively assessed using the Sensewear Pro₃ Armband Accelerometer (Bodymedia, Inc., Pittsburgh, PA). The Sensewear Armband is a multi-sensor device that provided minute-by-minute estimations of sleep/wake status based on a proprietary algorithm that incorporates information about demographics (age, gender, height, weight, handedness), motion/positionality (measured via a bi-axial accelerometer), skin temperature, heat dissipation, and the galvanic skin response. Sleep algorithms were developed using overnight polysomnography (PSG) from both healthy and sleep-disordered individuals in which “an artificial neural network was then used to develop sleep prediction algorithms based on the raw data from all the sensors” [35]. Sensewear Armband validation studies against PSG show no systematic biases in the device’s ability to estimate total sleep time, sleep onset latency, or sleep efficiency among children, although the device did estimate approximately 14 minutes greater wake time than the PSG [35].

Children were instructed to wear the device on the upper back of the right arm 24 hours/day for 7 consecutive days. Data was sampled at a frequency of 32 Hz and downloaded in 1-minute epochs, as is limited by the device [35]. Sleep data was included in current analyses only if the child wore the Sensewear Armband for 16+ hours/day for 5+ days (including three week days and two weekend days), consistent with literature that recommends at least five days of Actigraphy sleep data for acceptable reliability and validity [36]. Of 250 children who were ultimately randomized in the intervention post-baseline, 57% ($n = 142$) met data inclusion criteria for this study (e.g. having enough days of sleep data).

Independent t-tests revealed that children who met data-inclusion criteria did not differ significantly from those who did not meet data-inclusion criteria across child BMI z-score, family income, and child age; a 2-way chi-squared test revealed no group differences across child race/ethnicity and child gender.

From the minute-by-minute binary output for sleep/wake status (sleeping = 1, awake = 0), researchers defined the child’s bedtime as the clock time the device algorithm estimated the child had been asleep for three consecutive minutes. Wake time was defined as the clock time in the morning when the child was deemed awake (without falling asleep again afterwards) by the device algorithm. The two sleep variability variables were defined as the absolute value of the difference scores between week bedtime/wake time and weekend bedtime/wake time. Thus, the average weekly value was subtracted from the average

weekend value, such that **bedtime variability** = absolute value |weekend bedtime – weeknight bedtime|, and **wake time variability** = absolute value |weekend wake time – week wake time|. Additional sleep variables included **weekday total sleep time (TST)** = average number of minutes the child slept during the week, **weekday time in bed (TIB)** = average number of minutes the child spent trying to sleep during the week; **weekday wake after sleep onset (WASO)** = average number of minutes the child spent awake in the middle of the night between bedtime and wake time during the week.

Demographics.—Demographics (assessed via demographic questionnaire) included child age, gender, race/ethnicity, family income, and primary caregiver’s marital status. A trained health technician or nurse collected child height and weight, and researchers calculated body mass index as kilograms per meters squared and BMI z-scores using a modified LMS procedure from the Center for Disease Control and Prevention [37].

Data Analysis

Researchers conducted all statistics on SPSS Version 20.0. Covariates considered for all analyses included child age, child race, child gender, and child BMI z-score. In order to address the first study aim, a multivariate analysis of variance was conducted with asthma group predicting bedtime variability and wake time variability (Model 1). All covariates were uncorrelated with outcome variables (bedtime and wake time variability) and thus were not included in Model 1.

To address the second exploratory study aim, a series of within-subjects linear and hierarchical regressions were conducted to assess the relationships between wake time variability and weekday sleep parameters within the OV/OB+asthma and OV/OB only groups, separately. Weekday sleep parameters included total sleep time (TST), time in bed (TIB), and wake after sleep onset (WASO). Within Model 2, child age was significantly correlated with weekday total sleep time and time in bed within both the OV/OB+asthma and OV/OB only groups, and was thus the only covariate included in analyses.

Results

Of the 142 children included in analyses, 28% ($n = 40$) met criteria for the comorbid OV/OB +asthma group (detailed below) and the remaining 72% ($n = 102$) met criteria for the OV/OB only group. Independent T-tests revealed that the two groups did not differ significantly by age; however, the two groups did differ significantly in family income ($p = .005$), with the OV/OB+asthma group evidencing a lower family income. Chi-squared tests revealed that children did not differ in race/ethnicity, gender, or parental marital status among the two groups. See Table 1 for participant demographics and a comparison of the two groups by demographic variables.

Model 1: Group Differences in Bedtime and Wake time Variability

Descriptive statistics for week and weekend bed and wake times can be found in Table 2. The OV/OB+asthma group exhibited statistically significantly later weekend bedtimes compared to the OV/OB group ($p = .05$); however, weekday bedtime and weekday and

weekend wake times were relatively similar among groups (see Table 2). Regarding bed and wake time variability, both groups exhibited greater week-to-weekend wake time variability compared to bedtime variability. This seemed to specifically be driven by later wake times and “sleeping in” on the weekends. The OV/OB+asthma group exhibited approximately 30 minutes greater variability in both bedtime and wake variability when compared to their OV/OB only counterparts.

In order to test group differences in sleep variability were statistically significant, a one-way multivariate analysis of variance (MANOVA) was ran with asthma status predicting bedtime variability and wake time variability. Preliminary assumptions of normality, homogeneity of covariance, and equality of error variance were tenable. The final MANOVA revealed a significant omnibus effect ($F(2, 134) = 5.71, p = .004$, Wilk's Lambda = .922) with medium effect size ($\eta_p^2 = .078$) of asthma status on the combined bedtime and wake time variability variables. That is, the differences between groups in bedtime and/or wake time variability were significant and represented a medium effect size. Univariate follow-ups indicated a main effect for asthma group on both bedtime ($p = .013$) and wake time ($p = .003$) variability, with asthma group explaining a small to medium proportion of variance in bedtime and wake time variability, respectively. Specifically, children with comorbid OV/OB +asthma evidenced approximately 26 minutes greater bedtime and 35 minutes greater wake time variability from week to weekend than their OV/OB counterparts. See Table 3 for complete Model 1 univariate outcomes.

Model 2: Association between Wake time Variability and Weekday Sleep

A series of linear and hierarchical regression analyses were then ran separately within the OV/OB+asthma and OV/OB only groups to investigate how wake time variability influenced weekday sleep. Wake time variability predicted a series of independent sleep parameters, including weekday wake after sleep onset (WASO), weekday time in bed (TIB), and weekday total sleep time (TST).

OV/OB+Asthma Group.—A linear regression with covariates predicting outcome variables (TST, WASO, TIB) did not reveal any significant covariates. Because previous literature shows that sleep variables such as TST change developmentally with increasing age, age was included in all analyses as a covariate. Hierarchical multiple regression revealed that age and wake time variability trended towards significantly predicting weekday WASO in the omnibus model ($p = .06$). After controlling for the effect of age, wake time explained 14% greater variance in WASO. Further, each 5 minutes greater variability in weekday-to-weekend wake time corresponded to 1 minute less weekday WASO. Hierarchical multiple regressions showed that age and wake time variability significantly predicted weekday TIB ($p = .001$) and TST ($p = .03$) in the omnibus models. After controlling for age, wake time variability explained significantly greater variance in TIB ($p = .003$) but not TST ($p = .09$). Specifically, each 3 minute increase in wake time variability corresponded to 1 minute less TIB during the week. See Table 4 for complete Model 2 regression output.

OV/OB Only Group.—A linear regression with covariates predicting outcome variables (TST, WASO, TIB) revealed marital status predicting TIB to be the only significant covariate. As previously stated, age was included in all analyses as a theoretically-relevant covariate. Hierarchical multiple regression revealed that wake time variability did not significantly predict WASO ($p = .10$). In contrast, wake time variability significantly predicted TIB in the omnibus model ($p < .001$). After controlling for any effect of age or marital status, wake time variability explained significantly greater variance in TIB ($p = .003$), and each 3 minute increase in wake time variability corresponded to approximately 1 minute less TIB during the week. Age and wake time variability trended towards significantly predicting weekday TST in the omnibus model ($p = .076$). See Table 4 for complete Model 2 regression output.

Discussion

Findings from this study suggest that children with comorbid OV/OB+asthma evidence approximately 30 minutes greater week-to-weekend bedtime and wake time variability compared to children with only OV/OB. Within the comorbid OV/OB+asthma group, larger discrepancies in wake time variability predicted significantly less time spent in bed during the week and trended towards significantly less wake after sleep onset during the weekday. Interestingly, greater wake time variability was not related to weekday total sleep time over and above the impact of age. Children with comorbid asthma who were more variable in their week and weekend wake times spent less time in bed during the week and less time awake after falling asleep. Thus, it seems that greater variability from week to weekend shortened the sleep period and consolidated sleep during the week. Among the OV/OB only group, greater wake time variability only predicted less time spent in bed, over and above any impact of age or parental marital status.

Results from our study are consistent with previous findings showing children with asthma exhibit poorer bedtime habits than their healthy counterparts [24]. Previous literature finding that adolescents with asthma are more likely to sleep somewhere other than their own bed [38] may be one potential explanation for why children with asthma in our sample evidenced greater variability between weekday and weekend bedtimes, Meltzer and colleagues posit that co-sleeping may be due to parents' desire to monitor their child's breathing overnight. Thus, it is possible that children with asthma are more likely to parallel their parent's sleep patterns on the weekends by staying up and sleeping in later so that their parents may monitor their asthma symptoms as they sleep. Further, 40% of adolescents in Meltzer's 2014 study with severe asthma reported clinically significant insomnia, compared to 20% of adolescents with mild or no asthma. Among adults, variable sleep patterns frequently co-occur with clinically diagnosed insomnia [39, 40]. Results from our study indicated that greater week-to-weekend variability was associated with variables that might put children with OV/OB at risk for future insomnia (e.g. less time spent in bed); however, results also suggested wake time variability could potentially be protective against other symptoms of insomnia, such as greater amounts of wake after sleep onset. When thinking about why children's sleep was shorter and more efficient during the week with greater wake time variability, it is possible that these children experience greater weekday sleep pressure (a propensity to fall asleep quickly) due to being awake longer during the week than weekend.

Future studies would benefit from parsing apart the differential impacts of week-to-weekend variability on homeostatic sleep pressure, the circadian system, and behavioral correlates of healthy asthma management.

Although relatively more studies focus on sleep duration, recent research suggests that circadian aspects of sleep, such as regularity and timing, are important to consider. In the current sample of youth with comorbid OV/OB+asthma, inconsistent weekday-to-weekend wake times predicted sleep quality during the week. This is an example of how behavioral sleep habits may interact with a biological predisposition for poor sleep and ultimately influence disease management and progression. Other examples include the impact of sleep timing; among adolescents, evening-type chronotypes (those preferring to go to bed and rise late) are heavier and less physically active than their morning-type (preferring to go to bed and rise early) counterparts, even after controlling for sleep duration [41]. A recent epidemiological study found that evening-type adults are more likely to carry an asthma diagnosis, be prescribed asthma medication, and exhibit nocturnal asthma symptoms when compared their morning-type counterparts [42].

The potential impacts of inconsistent sleep patterns may be particularly far-reaching for children with asthma, as those with persistent subtypes require consistent and daily medication adherence. Indeed, consistency is related to medication adherence among youth with asthma [43] whereas less structured family environments are associated with increased risk for poor adherence, poorly controlled asthma, and increased lifetime hospitalizations [44–46]. Billows and colleagues [47] find that cohesive, organized family environments correlate with longer child sleep duration, shorter sleep onset latency, decreased daytime sleepiness, and better sleep hygiene among healthy youth. Among youth with asthma, families who follow consistent daily medication routines show better medication adherence [48, 49]. Therefore, although speculative, inconsistency in bed and wake times may negatively impact a family's ability to maintain routines in medication administration and asthma management.

Taken together, these findings suggest the importance of assessing sleep patterns and consistency among children with asthma who are overweight. Importantly, it appears that asthma status confers even greater risk for potentially deleterious sleeping patterns than simply being overweight. This is significant, given that being overweight or obese is already associated with serious sleep morbidities. Youth with asthma are clearly predisposed towards biological sleep problems, and these sleep problems are clearly linked to their disease state and management. Thus, health providers would likely glean useful information by asking questions about sleeping patterns across the week among asthmatic youth, as well as how week-to-weekend variability influences things such as medication adherence.

Findings from this study should be taken in light of several limitations. Although other studies have also assessed asthma status via a one-item parent-report response, there are certainly downsides to classifying asthma subjectively in this way. Children with any history of asthma diagnosis were classified into the comorbid OV/OB+asthma group, thus it is possible that children who no longer exhibit symptoms were included in these analyses. Asthma was not objectively confirmed in this study, nor was severity measured. Given that

our study showed that subjectively-reported asthma status confers additional risk for poor sleep outcomes over and above OV/OB status, future studies should attempt to replicate these findings using objective measures of asthma. Future studies should also explore any potential mediating roles of asthma severity on sleep. Additionally, sample size was relatively small with unbalanced numbers of children with comorbid OV/OB+asthma ($n = 40$) and OV/OB only ($n = 102$). Further, group differences in bedtimes/wake times and bedtime and wake time variability were relatively small. Future studies should assess whether these differences are clinically meaningful. This was a cross-sectional study, and study results cannot be interpreted as more than time-invariant associations. Further, although the Sensewear Armband is well-validated for measuring energy expenditure [33], it is relatively less commonly used to measure child sleep. Finally, participants included were relatively poor-sleeping overweight/obese children residing in rural counties in [omitted for blind review]. Thus, results may not be widely generalizable to children across the weight spectrum who reside in other geographical areas.

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Table 1 –**Participant Demographics by Group**

Demographic	Total Group		OV/OB Only		OV/OB +Asthma		p
	M	SD	M	SD	M	SD	
Child age (7–12 years)	9.8	1.4	10.0	1.5	9.8	1.4	.4
Family Income (see below for numeric values)	2.9	1.5	3.1	1.5	2.4	1.2	.005
Demographic	N	%	N	%	N	%	p
Child Gender							.55
Female	66	46.5%	49	48.0%	17	42.5%	
Male	76	53.5%	53	52.0%	23	57.5%	
Total	142	100.0%	102	100.0%	40	100.0%	
Child Race/Ethnicity							.86
Caucasian	99	69.7%	73	71.6%	26	65.0%	
African American	18	12.7%	11	10.8%	7	17.5%	
Biracial	15	10.6%	10	9.8%	5	12.5%	
Other	2	1.4%	2	1.4%	0	0.0%	
No Response	8	5.6%	6	5.9%	2	5.0%	
Total	142	100.0%	102	100.0%	40	100.0%	
Family Income							.005
1) < \$19,000	22	15.5%	11	10.8%	11	27.5%	
2) \$20,000–\$39,999	46	32.4%	31	30.4%	15	37.5%	
3) \$40,000–\$59,999	31	21.8%	25	24.5%	6	15.0%	
4) \$60,000–\$79,999	19	13.4%	14	13.7%	5	12.5%	
5) \$80,000–\$99,999	10	7.0%	7	6.9%	3	7.5%	
6) > \$100,000	12	8.5%	12	11.8%	0	0.0%	
No response	2	1.4%	2	2.0%	0	0.0%	
Total	142	100.0%	102	100.0%	40	100.0%	
Parent Marital Status							.11
Never married	15	10.6%	8	7.8%	7	17.5%	
Divorced/Separated	20	14.1%	11	10.8%	9	22.5%	
Widowed	3	2.1%	2	2.0%	1	2.5%	
Presently Married	98	69.0%	77	75.5%	21	52.5%	
Living with partner	6	4.2%	4	3.9%	2	5.0%	
Total	142	100.0%	102	100.0%	40	100.0%	

Table 2 –

Descriptive Statistics of Outcome Variables

Outcome Variable	Group	M	SD	<i>p</i>
Bedtime (week)	1. OV/OB Only	10:30 PM	77 mins	.85
	2. OV/OB+Asthma	10:39 PM	65 mins	-
Bedtime (weekend)	1. OV/OB Only	11:17 PM	88 mins	.05
	2. OV/OB+Asthma	11:41 PM	78 mins	-
Wake time (week)	1. OV/OB Only	6:51 AM	70 mins	.88
	2. OV/OB+Asthma	6:40 AM	71 mins	-
Wake time (weekend)	1. OV/OB Only	7:52 AM	87 mins	.11
	2. OV/OB+Asthma	7:54 AM	66 mins	-

Table 3 –

Univariate Effects in Outcome Variables by Group

Outcome Variable	Group	M	SD	SE	F	df	p
Bedtime Variability (weekend-week)	1. OV/OB Only	63.14	51.68	5.43	-	-	-
	2. OV/OB+Asthma	89.13	59.77	8.76	6.36	1, 135	0.013
Wake time Variability (weekend-week)	1. OV/OB Only	72.80	58.11	6.01	-	-	-
	2. OV/OB+Asthma	107.92	64.10	9.70	9.47	1, 135	0.003

Table 4 –
Linear and Hierarchical Linear Regression Predicting Weekday TST, WASO, and TIB

OV/OB + Asthma Group					
Outcome Variable	b (<i>p-value</i>)	Cumulative R ²	R ² change for step	Cumulative F (<i>p-value</i>)	F change for step (<i>p-value</i>)
Weekday TST					
Age	-12.37 (.04)	.10	.10	4.36 (.04)	4.36 (.04)
Wake time Variability	-.25 (.09)	.17	.07	3.87 (.03)	3.14 (.09)
Weekday WASO					
Age	-.70 (.89)	.001	.001	.04 (.85)	.04 (.85)
Wake time Variability	-.20 (.02)	.14	.14	3.03 (.06)	6.02 (.02)
Weekday TIB					
Age	-12.91 (.02)	.13	.13	5.63 (.02)	5.63 (.02)
Wake time Variability	-.37 (.003)	.32	.19	8.55 (.001)	10.11 (.003)
OV/OB Only Group					
Outcome Variable	b (<i>p-value</i>)	Cumulative R ²	R ² change for step	Cumulative F (<i>p-value</i>)	F change for step (<i>p-value</i>)
Weekday TST					
Age	-7.47 (.14)	.02	.02	2.17 (.14)	2.17 (.14)
Wake time Variability	-.21 (.08)	.05	.03	2.72 (.07)	3.21 (.08)
Weekday WASO					
Age	-3.41 (.21)	.02	.02	1.59	1.59 (.21)
Wake time Variability	-.11 (.08)	.05	.03	2.41	3.18 (.08)
Weekday TIB					
Age	-14.56 (.001)	.15	.15	8.66 (.001)	8.66 (.001)
Parental Marital Status	10.97 (.05)	.15	.15	8.66 (.001)	8.66 (.001)
Wake time Variability	-.28 (.003)	.23	.08	9.36 (.001)	9.29 (.003)

Note: TST = total sleep time; WASO = wake after sleep onset; TIB = time in bed. Significant findings bolded.