Sleep in Overweight Adolescents: Shorter Sleep, Poorer Sleep Quality, Sleepiness, and Sleep-Disordered Breathing

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Objective To document the sleep of overweight adolescents and to explore the degree to which weight-related sleep pathology might account for diminished psychosocial outcome. **Methods** Sixty children aged 10–16.9 from a weight-management clinic were compared to 22 healthy controls using comprehensive actigraphic, polysomnographic, and parent- and self-report questionnaire assessments. **Results** Overweight participants averaged more symptoms of sleep-disordered breathing, later sleep onset, shorter sleep time, and more disrupted sleep than controls. Although the groups did not differ in self-reported sleep habits, multiple concerns were reported by parents of overweight participants, including daytime sleepiness, parasomnias, and inadequate sleep. Group differences in academic grades and depressive symptoms were at least partially accounted for by short sleep and daytime sleepiness. **Conclusions** Excessive weight is associated with an increased risk of sleep problems. There is a need for further research in this area and for clinicians who work with overweight children to evaluate their sleep.

Key words adolescence; childhood; obesity; overweight; pediatrics; sleep; sleep apnea.

The prevalence of overweight adolescents in the United States has tripled over the past 30 years, with recent estimates falling around 16% (Daniels et al., 2005). Concurrently, there has been a growing interest in identifying behaviors that may be targets for weight-management treatment or prevention of excess weight. Disordered sleep may be one of the many contributors to excessive weight during childhood. Cross-sectional associations between short sleep duration and overweight have been reported (Gupta, Mueller, Chan, & Meininger, 2002; von Kries, Toschke, Wurmser, Sauerwald, & Koletzko, 2002; Sekine et al., 2002), and longitudinal studies have documented that shorter sleep predicts the later emergence of overweight (Agras, Hammer, McNicholas, & Kraemer, 2004; Reilly et al., 2005; Sugimori et al., 2004). Conversely, excessive weight is a risk factor for sleep-disordered breathing, including obstructive sleep apnea (OSA). Between 13 and 33% of overweight children have OSA, which is several times the prevalence in lean children (Chay et al., 2000; Marcus, 1996; Wing et al., 2003). However, research on overweight children has used limited sleep measures (e.g., nonvalidated single-item parent-report scales) or has focused on only selected aspects of sleep (e.g., OSA).

Like excessive weight, OSA and inadequate sleep have been causally linked to insulin resistance (De la Eva, Baur, Donaghue, & Waters, 2002) and cardiovascular disease (Amin et al., 2005). Pediatric OSA and inadequate sleep also have been associated with poor academic performance and depressive symptoms (Fallone, Owens, &

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Deane, 2002). Interestingly, these psychosocial factors are correlates of overweight during early adolescence (Swallen, Reither, Haas, & Meier, 2005), with depressive symptoms more commonly reported than acting out behaviors among treatment-seeking adolescents (Zeller, Saelens, Roehrig, Kirk, & Daniels, 2004) and child mood being an important predictor of dropout from weight-management treatment (Zeller, Kirk et al., 2004). However, little research has examined whether poor sleep quantity or quality mediates the links between childhood overweight and psychosocial outcome.

The primary aim of this study was to provide detailed documentation of the sleep of overweight adolescents as compared with normal-weight controls of similar demographic background. It was hypothesized that the overweight group would display shorter sleep duration, poorer quality sleep, and greater evidence of sleep-disordered breathing than normal-weight controls. The secondary aim was to explore the degree to which short sleep, daytime sleepiness, and OSA symptoms could account for the diminished academic performance and depressive symptoms that have been reported among adolescents who are overweight.

Methods

Sample and Procedures

The overweight group was comprised of 60 participants, aged 10–16.9 years, who were recruited consecutively at the time of intake into a hospital-based multidisciplinary pediatric weight-management clinic. Exclusion criteria included neurological history (e.g., seizures), craniofacial abnormalities, developmental disorders (e.g., Down syndrome), conditions involving daytime hypoxia (e.g., poorly controlled asthma), use of psychiatric or neurological medication (given potential effects on sleep), and treatment for OSA in the past 2 years. The control group was comprised of 22 children, who had been healthy controls in a previous study (Zeller, Ramey, & Allen, 2003), or their siblings. Initial recruitment from that study was from a representative cross-section of public and private school classrooms, using the "revised class play" paradigm (Noll et al., 1999). Parents of controls aged 10–16.9 were invited by mail and telephone to participate in this study. Controls met the same exclusion criteria as overweight participants and were reported by their parents to be of normal weight.

All procedures were approved by the local Institutional Review Board. Parents provided written consent and participants provided oral assent to participate. Families of overweight adolescents were compensated $50 for participating, while those of controls were compensated $70. The difference in compensation was justified by the greater potential for direct benefit to the overweight adolescents; indeed, because of study findings and with family consent, several were referred for clinical follow-up of previously undiagnosed OSA. During the first part of the research, parents and participants completed demographic and sleep questionnaires, and participants underwent a week of objective sleep monitoring with actigraphy (described below). Part two included behavioral questionnaires and an inpatient overnight sleep study.

Measures

Anthropometric Data

To obtain body mass index (BMI; kg/m^2), height was measured with a wall-based stadiometer and weight with a calibrated hospital scale. A BMI *z*-score was then computed based on tables published in 2000 by the U.S. Centers for Disease Control and Prevention. Pubertal status was obtained via a validated self-report questionnaire (Carskadon & Acebo, 1993).

Sleep Measures

A comprehensive sleep evaluation protocol capitalized on the strengths of several measurement techniques. Parent- and self-report questionnaires were used to assess each reporter's perspective on the participant's typical sleep, including unusual sleep behaviors that are not well-quantified by other methods (e.g., parasomnias). However, questionnaires are subject to reporter and recall biases, as well as imprecision with respect to nocturnal arousals and sleep onset and offset times. Complementing questionnaire data, typical sleep patterns also were measured by actigraphy, which uses movement patterns to provide an objective, unobtrusive measure of sleep phase, duration, and continuity over multiple nights in the home (Acebo et al., 1999; Sadeh, Sharkey, & Carskadon, 1994). Finally, overnight polysomnography (PSG) objectively measured sleep-stage distribution and sleep-related respiratory function. PSG was conducted during a single night in a hospital-based sleep laboratory with an examiner-determined bedtime and noninvasive but mildly obtrusive monitoring. As a result, although PSG is considered the gold standard for the diagnosis of OSA, actigraphy and questionnaires may yield better measures of a child's typical sleep habits. By using these complementary measurement techniques, we sought to obtain a more complete and accurate picture of participants' sleep.

Parent-Report Sleep Questionnaire. The Child Sleep Habits Questionnaire (CSHQ) (Owens, Spirito, & McGuinn, 2000) is a broad-band measure of pediatric sleep difficulties. For each of 45 items, parents rate whether a sleep-related behavior occurs rarely, sometimes, or often. The questionnaire has eight subscales: bedtime resistance, sleep onset delay, adequacy of sleep duration, sleep-related anxiety, night wakings, parasomnias (e.g., talks during sleep), sleep-disordered breathing (e.g., snores loudly), and daytime sleepiness. It also includes an item assessing napping frequency, as well as open-ended queries on bedtime, rise time, and sleep duration for weeknights and weekends. Parent-reported sleep duration was computed as the mean of the direct report of the parent and the arithmetic difference between reported bedtime and rise time. Parents also completed the sleepiness and sleep-disordered breathing subscales from the Pediatric Sleep Questionnaire (PSQ) (Chervin, Hedger, Dillon, & Pituch, 2000). All CSHQ and PSQ items and subscales were coded so that high scores reflect increased pathology.

Self-Report Sleep Questionnaires. The Pediatric Daytime Sleepiness Scale (PDSS) (Drake et al., 2003) is a validated scale that asks about the frequency of difficulty waking in the morning and the ability to maintain arousal during the day. The Youth Sleep Habits Survey (YSHS) (Acebo & Carskadon, 2002; Wolfson et al., 2003) is a validated survey measure from which we analyzed five subscales: sleepiness 1 (situations in which sleepiness occurs), sleepiness 2 (general frequency of feeling sleepy), sleep quality (satisfaction with sleep), sleep delay (e.g., staying up past 3:00 a.m.), and 'owl and lark' (time of day preferences for various activities). In addition, the YSHS includes open-ended items relating to bed and rise times and total sleep duration. Self-reported sleep duration was computed as the mean of the direct report of the participants and the arithmetic difference between reported bedtime and rise time.

Polysomnography (PSG). Each participant underwent inpatient full-night PSG, with a parent or guardian present in a separate bed. Participants were not deprived of sleep before the PSG and were not given any sedative. The following were monitored: Electroencephalogram ($C3\text{-}A_2$, $C_4\text{-}A_1$, $O_1\text{-}A_2$, $O_2\text{-}A_1$), electrooculogram, submental and tibial electromyogram, electrocardiography, nasal/oral airflow through nasal pressure transducer or a three-pronged thermistor, end-tidal CO_2 (at the nose), snoring microphone, O_2 saturation by pulse oximeter, oximeter pulse waveform, actigraphy to measure limb movements, infrared video monitoring, and rib cage and abdominal volume (respiratory inductance plethysmograph). Sleep staging and arousals were scored according to standardized criteria (American Sleep Disorders Association, 1992; Rechtschaffen & Kales, 1968). Obstructive apneas (i.e., complete cessation of airflow) were defined as a >80% decline in airflow over two breaths, despite continued chest/abdominal wall movement. Obstructive hypopneas (i.e., partial cessation of airflow) were defined as a decrease of 50–80% in airflow over at least two breaths that was associated with (a) paradoxical respiration, and (b) oxyhemoglobin desaturation (≥4%) or a subsequent arousal. The following variables were coded for analyses: sleep duration, sleep efficiency after sleep onset (percent of the total sleep period spent asleep), percent of sleep in each stage (1–4 and rapid eye movement or REM), lowest oxygen saturation, and percent of sleep spent hypercapnic (end-tidal CO_2 > 50). The obstructive apnea index (AI), apnea + hypopnea index (AHI), and arousal index (ArI) were each computed as the sum of relevant events divided by hours slept.

Actigraphy. The actigraphs used in the present study (Mini-Motionlogger, Ambulatory Monitoring, Inc., NY) are the size of a sports wristwatch and were worn on participants' non-dominant wrists during evening and overnight hours. The devices carry a motion detector and internal memory that records movement counts in 1-min epochs. These data were subsequently compared against a self-report sleep diary to screen for potential artifacts, such as removal of the watch. The following variables were then inferred from a validated algorithm (Sadeh et al., 1994): sleep onset and offset times, total sleep period duration (offset minus onset), sleep efficiency, number of awakenings from sleep, number of awakenings from sleep that lasted longer than 5 min, and the longest period of sleep.

Three sets of actigraphy data were generated. The first was comprised of each participant's average scores for all nights studied, provided at least five nights were recorded. This threshold has been established to ensure reliability (Acebo et al., 1999) but does not allow for the differentiation of school versus non-school-nights. As supplemental indexes, the second set of actigraphy data was comprised of each participant's average for school-nights, provided at least three such nights were recorded, and the third was comprised of each participant's average for non-school-nights, provided at least two such nights were recorded.

Academic Grades and Symptoms of Depression

Participants and parents reported the participants' typical academic grades on an 8-point scale (As, As & Bs, Bs,

Bs & Cs, Cs, Cs & Ds, Ds, Ds & Fs). The two reports were highly correlated, $r = .81$, $p < .001$, so they were averaged into a single index. Depressive symptoms were reported by parents via the depression subscale from the Behavioral Assessment System for Children (Reynolds & Kamphaus, 1992), a validated parent-report psychopathology questionnaire. After a mid-study protocol change, 16 overweight and 21 control participants also completed the Children's Depression Inventory (Kovacs, 1992), a validated pediatric self-report questionnaire of depressive symptoms. Because the correlation between parent- and self-report of depressive symptoms was modest ($r = .45$), these scores were analyzed separately.

Preprocessing of Data and Analytic Strategy

On questionnaires, missing data were typically due to omission of one or two items on a multi-item subscale. Consequently, when less than half of a subscale's items were missing, composites were prorated from the remaining items. Several sleep indexes were log-transformed to reduce skew, which included the AI and AHI from the PSG, the PSQ and CSHQ sleep-disordered breathing subscales, the PSQ sleepiness subscale, and the CSHQ sleep onset delay subscale.

Demographic characteristics across groups were compared using chi-square, Fisher's exact tests, or independent sample *t*-tests. These tests also compared cases where there were missing data for a given set of analyses to those with complete data on age, sex, race, family income, maternal education, and study group.

Primary analyses were comprised of eight multivariate analyses of variance (MANOVA), clustered on conceptual and pragmatic grounds. The first compared the overweight group to controls on PSG variables. The second through fourth compared the groups on the three sets of actigraphy data. The fifth compared the groups on parent-reported bedtimes and arousal times, while the sixth compared the groups on the other parent-reported sleep indexes. The seventh and eighth were similar but replaced parent-report questionnaires with self-report.

To increase the clinical utility of these findings, we then compared the prevalence of sleep-disordered breathing (on PSG and per parent report), as well as daytime sleepiness, short sleep duration, and sleep disruption in participants' natural settings (on questionnaires and actigraphy). Unfortunately, few established normative values exit in the pediatric sleep medicine literature. For present purposes, sleep-disordered breathing was defined alternatively as an AHI >1, AHI >5, or a PSQ sleep-disordered breathing score >0.5, indicating endorsement of at least half the items; daytime sleepiness was indexed by a PDSS ≥20 or a PSQ sleepiness score ≥0.5, both indicating reported sleepiness on at least half of the items; short sleep was defined as <8 h; and sleep disruption was defined as post-onset sleep efficiency <80%. Odds ratios were computed with and without adjustments for age, race, gender, income, and maternal education.

To address the secondary aim, three sets of regression equations were computed, one with the composite academic grade scale as dependent variable and the others with parent- and self-report depressive symptoms as dependent. In each set, the variance accounted for by group membership was first computed as a zero-order association; then analyses were rerun with selected sleep variables as covariates. Potential covariates were selected based on prior research that suggests an association of childhood overweight with short sleep, sleepiness, and OSA: log-transformed AHI, parent-reported OSA symptoms, parent- and self-report of sleepiness and sleep duration on weeknights and weekends, and actigraph-derived sleep duration on weeknights and weekends. To maximize statistical power, only those covariates that predicted each dependent measure in stepwise entry were retained in the final analyses.

Alpha was set at .05 for statistical significance, with Holm's procedure controlling Type I error in primary analyses (see below). Statistical "trends" ($p < .10$) are also reported so that readers can judge their potential relevance in light of the modest sample sizes.

Results

Sample Characteristics

As summarized in Table I, the groups did not significantly differ in age, grade placement, sex, pubertal status, family income, maternal education, or race (all but three non-Caucasian participants—two overweight and one control—were African-American). By design, the groups differed in body mass. All controls fell in the 24th–92nd percentiles of BMI for age and sex; all overweight participants were above the 95th percentile and 65% were above the 99th percentile.

All participants underwent PSG. Eighty-four percent of participants wore the actigraph at least five nights, 62% at least three school nights, and 78% at least two non-school nights. Parent questionnaires yielded all subscales 89% of the time and all bed and arousal times 67% of the time. Ninety-two percent of self-report questionnaires yielded all subscales, while 100% reported sleep onset and offset times. More Caucasians than non-Caucasians had school-night actigraphy data, $p = .001$,

Table I. Demographic Characteristics

	Controls	Overweight	*p*-value
Sample size	22	60	
Body mass index (BMI)	19.4 ± 2.6	37.6 ± 7.5	<.0001
BMI *z*-score for sex and age	0.26 ± 0.61	2.48 ± 0.30	<.0001
Age	12.6 ± 1.7	13.1 ± 1.8	Not significant ($p > .10$)
Grade placement	6.7 ± 1.5	6.9 ± 1.7	Not significant
Family income (in 1000s)[a]	48.7 ± 34.0	39.4 ± 3.2	Not significant
Maternal education (years)[a]	13.4 ± 2.5	13.0 ± 2.1	Not significant
Percentage of females	64	67	Not significant
Percentage of Caucasians	46	40	Not significant
Percentage of Pre- or early pubertal	10	14	Not significant
Percentage of mid-pubertal	38	35	
Percentage of late or post-pubertal	52	52	

Values are presented as mean ± standard deviation or percentage of sample. Significance (*p*) values are based on chi-square for pubertal status, Fisher's exact tests for sex and race, and independent sample *t*-tests for age, grade placement at school, and BMI-related variables.

[a]Family income was not reported on three controls and five overweight children. Maternal education was not reported for three overweight children.

and parent-reported sleep-onset and offset times, $p = .017$. Higher income families more often had school-night actigraphy data, complete parent questionnaire data, and complete child questionnaire data, $p = .001$–$.005$. Otherwise, participants with missing data did not differ in sex, age, race, family income, maternal education, or study group from those with complete data. Moreover, MANOVA findings were essentially unchanged in exploratory analyses that included age, sex, race, income, and maternal education as covariates.

Primary Analyses: Group Differences in Sleep

The overweight and control groups differed on the multivariate PSG analysis, $F(12, 66) = 2.51$, $p = .011$. Follow-up univariate analyses presented in Table II show that this was primarily due to a difference across groups in log-transformed AHI (log-AHI), with statistical trends on log-transformed AI and percent of sleep spent in stage 3.

The groups differed in multivariate actigraphy findings for the entire recording period, $F(8, 60) = 3.69$, $p = .001$, and weekends, $F(8, 52) = 3.96$, $p = .001$, but not weeknights, $F(8, 44) = 1.95$, $p = .105$. Table III presents univariate data. The overweight group on average fell asleep later and had shorter and more disrupted sleep than controls. Although the findings were similar in pattern across school nights and non-school nights, the latter more clearly showed effects.

The groups showed a multivariate difference on parent-report questionnaire subscales, $F(12, 59) = 3.07$,

Table II. Polysomnographic Characteristics

	Controls	Overweight	*p*-value
Sleep duration (min)	394 ± 55	396 ± 60	Not significant ($p > .10$)
Sleep efficiency after onset	89.5 ± 8.6	88.3 ± 8.9	Not significant
Percentage of sleep in stage 1	3.8 ± 2.0	3.5 ± 1.9	Not significant
Percentage of sleep in stage 2	55.8 ± 8.2	55.7 ± 8.0	Not significant
Percentage of sleep in stage 3	2.6 ± 1.1	3.2 ± 1.4	.066
Percentage of sleep in stage 4	21.9 ± 6.1	21.4 ± 8.7	Not significant
Percentage of sleep in REM	15.9 ± 4.5	16.2 ± 4.6	Not significant
Arousal index (events/h)	9.0 ± 3.7	8.2 ± 3.5	Not significant
Apnea index (events/h)[a]	0.27 ± 0.58	0.84 ± 1.85	.056
Apnea + hypopnea index[a]	0.59 ± 1.11	2.17 ± 3.08	.001
Nadir O_2 saturation	92.4 ± 4.0	90.8 ± 4.3	Not significant
Percentage of night with $CO_2 > 50$	10.8 ± 28.0	8.7 ± 19.4	Not significant

Values are presented as mean ± standard deviation. Significance (*p*) values are based on univariate follow-ups to MANOVA.

[a]Raw apnea and apnea + hypopnea index scores are presented to aid interpretability, although both indexes were log-transformed in analyses to reduce marked skew.

Table III. Actigraphy Findings

	Whole week		School nights		Non-school nights	
	Controls	Overweight	Controls	Overweight	Controls	Overweight
Sleep onset time	23:02 ± 0:43	23:43 ± 1:12**	22:33 ± 0:44	22:56 ± 0:59	23:48 ± 1:17	00:22 ± 1:17
Sleep offset time	7:27 ± 0:58	7:30 ± 1:08	6:27 ± 0:46	6:27 ± 0:37	8:48 ± 1:19	8:30 ± 1:16
Sleep period (min)	507 ± 49	469 ± 50***	475 ± 61	452 ± 58	535 ± 46	489 ± 73***
Sleep efficiency post-onset (%)	86.8 ± 7.2	80.2 ± 12.2**	87.1 ± 8.8	84.4 ± 10.3	88.3 ± 6.2	78.3 ± 12.5****
Motionless sleep (%)	57.7 ± 11.4	53.2 ± 15.0	59.7 ± 11.4	60.1 ± 13.1	59.2 ± 11.6	51.1 ± 15.3****
Number of arousals	18.0 ± 9.3	16.0 ± 5.2	13.6 ± 6.4	14.3 ± 5.7	19.7 ± 9.4	17.3 ± 5.9
Number of arousals >5 min	3.9 ± 2.3	4.9 ± 2.6	3.6 ± 2.4	3.9 ± 2.6	3.9 ± 2.4	5.6 ± 2.5**
Longest continuous sleep (min)	147 ± 71	109 ± 47*	154 ± 68	118 ± 53*	151 ± 86	109 ± 50**

Significance values based on univariate follow-ups to MANOVA.
*$p < .10$, **$p < .05$, ***$p < .01$, ****$p < .005$.

Table IV. Questionnaire Results

	Controls	Overweight	*p*-value
Parent report			
School night asleep time	21:41 ± 0:34	21:54 ± 0:50	Not significant ($p > .10$)
Non-school night asleep time	23:07 ± 1:02	23:33 ± 1:00	Not significant
School night arousal time	6:25 ± 0:49	7:00 ± 1:18	Not significant
Non-school night arousal time	9:29 ± 1:35	9:29 ± 1:47	Not significant
School night sleep period (min)	523 ± 45	522 ± 55	Not significant
Non-school night sleep period	583 ± 46	570 ± 82	Not significant
PSQ sleep-disordered breathing[a]	.05 ± .10	.35 ± .32	<.001
CSHQ sleep-disordered breath[a]	3.1 ± 0.4	4.6 ± 1.6	<.001
PSQ sleepiness[a]	.07 ± .18	.26 ± .31	.042
CSHQ sleepiness	10.7 ± 2.5	13.2 ± 3.7	.009
CSHQ bedtime resistance	6.7 ± 0.7	7.3 ± 1.4	.037
CSHQ sleep onset delay[a]	1.2 ± 0.4	1.5 ± 0.6	.029
CSHQ sleep duration	3.7 ± 1.2	4.6 ± 1.6	.018
CSHQ night wakings	3.8 ± 0.9	3.9 ± 1.0	Not significant
CSHQ parasomnias	8.0 ± 1.1	8.9 ± 1.3	.007
CSHQ naps item	1.4 ± 0.5	1.4 ± 0.6	Not significant
Self-report			
School night asleep time	21:51 ± 0:50	22:06 ± 0:55	Not significant ($p > .10$)
Non-school night asleep time	22:57 ± 2:42	23:55 ± 2:00	.084
School night arousal time	6:15 ± 1:35	6:04 ± 1:46	Not significant
Non-school night arousal time	9:46 ± 3:49	9:14 ± 3:17	Not significant
School night sleep period (min)	528 ± 60	499 ± 55	.047
Non-school night sleep period	600 ± 61	570 ± 90	Not significant
PDSS	12.2 ± 6.7	14.1 ± 6.6	Not significant
YSHQ sleepiness scale 1	14.2 ± 3.7	14.1 ± 3.2	Not significant
YSHQ sleepiness scale 2	9.3 ± 4.0	9.8 ± 4.5	Not significant
YSHQ phase delay behaviors	12.5 ± 5.8	13.9 ± 5.5	Not significant
YSHQ sleep quality	7.4 ± 2.0	7.8 ± 2.1	Not significant
YSHQ owl vs. lark (higher = lark)	28.4 ± 5.6	27.0 ± 5.3	Not significant

CSHQ, Child Sleep Habits Questionnaire; PDSS, Pediatric Daytime Sleepiness Scale; PSQ, Pediatric Sleep Questionnaire; YSHQ, Youth Sleep Habits Questionnaire.
Values are presented as mean ± standard deviation. Significance (*p*) values are based on univariate follow-ups to MANOVA.
[a]Raw scores on the marked scales are presented to aid interpretability, but were log-transformed in analyses to reduce marked skew.

$p = .002$, but not bedtimes and rise times, $F(4, 51) = 1.01$, $p = .41$. Table IV presents univariate data. The greatest differences were due to greater parent-reported symptoms of sleep-disordered breathing, sleepiness, and parasomnias in the overweight group, though several other subscales differed to a measurable degree across

groups. In contrast, there was not a multivariate difference between the overweight and control groups in self-report subscales, $F(8, 66) = 1.31$, $p = .25$, or sleep onset and offset times, $F(4, 77) = 0.88$, $p = .48$.

Given that eight MANOVAs were run, we used Holm's sequential procedure to guard against Type 1 errors. This procedure has improved power over the Bonferroni correction without sacrificing control of Type I error (Aickin & Gensler, 1996). Under this procedure, the most stringent Bonferroni alpha cut-off (.05/8 or .006 in this case) is applied only to the most statistically significant finding in the family of analyses. If this threshold is crossed, then the next best significance level obtained in the family is compared to a slightly less stringent cut-off (.05/7 or .007 in this case), and so on. Using this procedure, the multivariate findings for PSG, actigraphy for the total recording period and for weekends, and parent-report questionnaire subscales all exceeded significance thresholds, whereas the others fell far short.

Table V summarizes follow-up analyses on the prevalence of sleep-disordered breathing, short sleep, sleep disruption, and daytime sleepiness. These categorical findings mirror those from analyses in which sleep was treated continuously. Sleep problems were often several times more likely among overweight participants than controls. Indeed, some odds ratios could not be computed because no controls met cut-offs for pathology.

Secondary Analyses: Does Sleep Mediate Group Effects on Grades and Depressive Symptoms?

Study group accounted for 4.2% of the variance in academic grades, $p = .033$, 6.0% of the variance in parent-report depressive symptoms, $p = .013$, and 24.8% of the variance in self-report depressive symptoms, $p = .001$. The overweight group averaged one letter grade poorer (B/C vs. A/B) and was one standard deviation higher on depressive symptoms (parent $T = 53$ vs. 45, self $T = 55$ vs. 45) than controls.

Several potential covariates correlated with academic grades in bivariate analyses: log-AHI, $r = .34$, $p = .002$; actigraph-determined sleep duration for all recorded nights and weeknights, $r = -.23$ and $-.28$, $p = .045$ and .043, respectively; log-PSQ sleepiness, $r = .25$, $p = .023$; parent-reported weeknight sleep, $r = -.33$, $p = .003$; and PDSS, $r = .39$, $p < .001$. In stepwise entry into the prediction of academic grades, only the PDSS and parent-reported and self-reported weeknight sleep were unique predictors, together accounting for 24% of the variance in grades, $p = .003$. Consequently, a regression equation was constructed in which academic grades were predicted by these three covariates, followed by study group. The amount of variance

Table V. Frequencies and Odds Ratios for Selected Sleep Measures Across Groups

	Controls	Overweight	Raw OR	Adjusted OR (95% CI)
PSG				
AHI >5	0%	13%	n/a	n/a
AHI >1	14	50	6.3***	6.6** (1.4–31.0)
Actigraphy				
Total recording sleep <8 h	26%	56%	3.5**	11.5*** (1.8–74.0)
Weeknight sleep <8 h	40	68	3.1*	5.9* (0.9–38.8)
Weekend sleep <8 h	12	46	6.3**	13.1** (1.8–94.6)
Total recording efficiency <80%	21	42	2.7	4.3* (0.8–23.3)
Weeknight efficiency <80%	20	29	1.7	7.5 (0.5–109.8)
Weekend efficiency <80%	12	50	7.5**	10.3** (1.4–76.9)
Parent questionnaire				
Weeknight sleep <8 h	23%	27%	1.2	1.7 (0.4–7.7)
Weekend sleep <8 h	5	13	3.1	47.9(0.6–3722.2)
PSQ sleep-disordered breath ≥0.5	0	39	n/a	n/a
PSQ sleepiness ≥0.5	14	39	4.0**	5.4** (1.0–28.4)
Child questionnaire				
Weeknight sleep <8 h	32%	49%	2.1	5.1** (1.2–22.0)
Weekend sleep <8 h	0	18	n/a	n/a
PDSS	14	23	1.9	2.6 (0.5–13.8)

AHI, apnea + hypopnea index; OR, odds ratio; adjusted OR covaried for age, sex, race, maternal education, and family income, with 95% confidence interval in parentheses; PDSS, Pediatric Daytime Sleepiness Scale; n/a, not able to compute odds ratio because controls had 0% prevalence (in each case, Fisher's exact test $p < .05$).
*$p < .10$, **$p < .05$, ***$p \leq .01$.

uniquely predicted by study group was thereby cut to 2.3%, $p = .14$.

In bivariate analyses, the CSHQ sleepiness subscale and the log-PSQ sleepiness subscale each correlated with parent-reported depressive symptoms, $r = .33$ and .27, $p = .003$ and .017, respectively, but only the former survived stepwise regression entry. A regression equation consequently predicted parent-reported depressive symptoms by CSHQ sleepiness, followed by study group. The variance uniquely predicted by study group was thereby cut to 2.2%, $p = .17$.

Self-reported depressive symptoms correlated with log-PSQ sleepiness, $r = .51$, $p = .001$; parent report of weekend sleep, $r = -.33$, $p = .050$; PDSS, $r = .54$, $p = .001$; self-report on the YSHS Sleepiness 1 subscale, $r = .34$, $p = .001$; and self-report of weeknight sleep, $r = -.40$, $p = .017$. Only the PDSS and parent- and self-reports of weeknight sleep duration survived stepwise entry into the prediction of self-report depressive symptoms, together accounting for 57% of the variance, $p < .001$. A regression equation entering these three covariates, followed by study group, found that the variance in self-reported depressive symptoms that was uniquely predicted by study group was cut to 7.1%, $p = .022$.

Taken together, these data suggest that sleep duration on weeknights and daytime sleepiness may have mediated the difference between the groups in academic grades and may have at least partially mediated group differences in depressive symptoms.

Discussion

Consistent with our hypotheses, overweight adolescents were at elevated risk for sleep-disordered breathing. In addition, actigraphy data collected in the child's normal sleep environment found that, compared to demographically similar normal-weight controls, those who were overweight averaged shorter sleep time—due largely to a later sleep onset—and less efficient and more disrupted sleep. Although the groups did not differ in self-reported sleep habits, multiple sleep concerns were reported by parents of overweight participants, including daytime sleepiness, parasomnias (e.g., sleepwalking), bedtime resistance, delayed sleep onset, and dissatisfaction with the adolescent's sleep duration.

Because of the cross-sectional research design, it is impossible from these data to infer the direction of causality between sleep problems and high body mass. However, the research literature suggests that short sleep duration may contribute to excessive weight gain (Agras et al., 2004; Reilly et al., 2005; Sugimori et al., 2004). The mechanism for this is not known, but sleep deprivation may cause dietary cravings and hormonal and metabolic shifts that foster weight gain (Spiegel, Tasali, Penev, & van Cauter, 2004). Further, sleep deprivation hampers attention, impulse control, and higher level problem solving (Durmer & Dinges, 2005), providing a second route by which dietary choices might be undermined. Finally, sleepiness may contribute to physical inactivity, although activity level has not been found to statistically mediate the link between short sleep and overweight (von Kries et al., 2002; Sekine et al., 2002).

On the other hand, OSA is more likely to be an effect, rather than a cause, of overweight. In adults, OSA is particularly predicted by fat deposition centrally and around the upper airway (Gami, Caples, & Somers, 2003). Weight loss among overweight adolescents and adults often results in improvement of OSA (Dixon, Schachter, & O'Brien, 2005; Sugerman et al., 2003). Overweight children also are at elevated risk for residual or recurrent OSA following adenotonsillectomy, the most common treatment (Mitchell & Kelly, 2004; Morton et al., 2001). Interestingly, even when effective, adenotonsillectomy may be followed by weight gain, not loss, among lean and overweight children alike (Soultan, Wadowski, Rao, & Kravath, 1999).

The excessive daytime sleepiness reported by parents of overweight participants may result from short sleep time, poor sleep quality, increased respiratory effort during sleep, or excessive weight itself. Each has been linked to systemic inflammation that may promote sleepiness (Mills & Dimsdale, 2004). Of note, our overweight adolescents appeared to be going to bed later than their lean counterparts despite reported sleepiness.

Even if daytime sleepiness and short sleep are the consequence, rather than the cause, of overweight, they may mediate at least some of the link between pediatric overweight and psychosocial morbidity. In this study, short sleep and daytime sleepiness together statistically accounted for much of the difference between the groups in academic grades and self-reported depressive symptoms. Daytime sleepiness also accounted for much of the difference between groups in parent-reported depressive symptoms. It is important to note that these findings were based on exploratory analyses and should be considered preliminary. Moreover, the symptoms of sleepiness and those of depression can be difficult to disentangle, though it may be helpful to limit item overlap on questionnaires. For example, the BASC depression subscale focuses on mood and social functioning, rather than somatic symptoms, whereas items on the PDSS and the sleepiness subscales from the CSHQ and PSQ focus

almost exclusively upon drowsiness or sleep need (not mood or social functioning). While the behavioral manifestations of tiredness and depressed mood share some features, the minimal item overlap in our measures lends some confidence that associations between parent-reported sleepiness and depressive symptoms found in this sample were not simple artifacts. Further, exploratory analyses suggest that the several nonsomatic subscales on the CDI correlated with diminished weeknight sleep in this sample (data not shown), lending further confidence to our conclusions. These preliminary findings highlight the need for further investigation of the potential role of sleep-related variables as mediators of adverse psychosocial outcomes among overweight children.

Current data also suggest that research on the sleep of overweight children extend beyond OSA, sleep duration, and daytime sleepiness. In this sample, overweight was associated with parasomnias and diminished sleep quality, neither of which has received much research attention in this population. Sleep-disordered breathing may contribute to increased restlessness and to partial arousal parasomnias (Guilleminault, Palombini, Pelayo, & Chervin, 2003). While actigraphy can neither differentiate partial from complete arousals nor determine the nature of sleep disruption, our findings do suggest the presence of disruptions in sleep continuity.

Despite evidence of differences in sleep functioning across groups based on parent-report and two objective sleep measures, self-report measures failed to yield significant differences. To some degree, this may relate to the relative inaccessibility of key sleep symptoms to self-observation. For example, symptoms of OSA and parasomnias are almost always more apparent to others than to the person who experiences them. It is also worth noting that self-report effects were generally in the same direction as those found via parent-report and actigraphy, but analyses may have been underpowered.

These findings have important clinical implications. Clinicians working with overweight adolescents should ask about symptoms associated with OSA, including loud snoring, labored breathing, or breathing pauses during sleep. When there is a suspicion of OSA, patients should be referred for formal sleep evaluation. The American Academy of Sleep Medicine and American Board of Sleep Medicine (http://www.sleepcenters.org, http://www.absm.org) list accredited sleep centers and board-certified sleep specialists. Clinicians should also ask about sleep habits. Current recommendations are that most adolescents need about 9¼ hours of sleep each night for optimal functioning (Mindell & Owens, 2003). Based on actigraphy, our control group came close to that only for non-school nights. Worse still, our overweight children averaged over an hour below recommendations on non-school nights and nearly 2 h below on school nights, suggesting the presence of a chronic sleep deficit that may have physiological and psychosocial consequences. Because this sleep deficit was largely due to delayed sleep onset, clinicians should attend to potential obstacles to establishing an appropriate bedtime. Current research cannot yet support the assertion that better sleep will contribute to weight loss, but successful interventions for sleep problems have been associated with subsequent gains in academic, behavioral, and emotional functioning (Gozal, 1998; Minde, Faucon, & Falkner, 1994).

Before concluding, it is important to highlight key limitations of this study. Although the overweight participants were not referred because of sleep problems, the fact that they were participating in a comprehensive pediatric weight-management program may make them unique relative to the larger population of overweight individuals. Missing data may also affect how representative findings are of the general population. Present findings generally agree with those from epidemiological studies that had more limited sleep assessments, but some caution is recommended in applying the present findings to the general population, especially to minorities and low income families who disproportionately had missing data in this study, or to younger children. In addition, though the CSHQ has been validated with grade-school children, subscale findings in the current sample should be considered tentative; to our knowledge, no broad-band parent-report sleep questionnaire has published validity evidence for adolescents. On a similar measurement issue, although parent- and child-reported grades correlated strongly, we did not have access to actual school records. Finally, the modest size of the sample (especially for self-reported depression) precluded investigation of more complex mediation and moderation models, such as developmental and gender effects, and whether sleep disorders can account for a wider array of weight-linked medical (e.g., insulin resistance) and behavioral morbidity.

Even so, this study provides the most comprehensive assessment of sleep to date in a sample of overweight adolescents, drawing upon the strengths of both objective and subjective sleep assessment methods. Present data add to a growing body of literature that links short sleep to overweight, confirm previous findings of increased risk for OSA among overweight children, and further suggest the presence of a wider range

of sleep pathology, including parasomnias and poor sleep quality, among overweight adolescents. These data also suggest that at least some of the negative psychosocial factors known to be associated with excessive weight are also associated with sleep disturbance, with sleep acting as a potential mediator. By carefully considering children's sleep, pediatric psychologists are in a unique position to develop novel approaches to the prevention of childhood overweight, behavioral weight-management treatment, and prevention of weight-related morbidity.

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