SLEEP AND TRANSITION TO ADOLESCENCE

Sleep and the Transition to Adolescence: A Longitudinal Study

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Study Objectives: To assess the links between sleep and pubertal development using a longitudinal design.

Design: Three consecutive annual assessments of sleep and pubertal development. Sleep was assessed using a week of home actigraphy.

Setting: Naturalistic sleep in the home setting of school children, Tel Aviv Area, Israel.

Participants: A sample of 94 (41 boys) typically developing healthy school-age children (age range at first assessment: 9.9–11.2 years).

Intervention: N/A

Measurements and Results: The Petersen's Pubertal Development Scale (PDS) and Sexual Maturation Scale (SMS) were used to assess pubertal development, and a week of actigraphy served to assess naturalistic sleep patterns. The results reflect expected developmental trends: an increase in signs of pubertal maturation, delayed sleep onset, and shorter sleep time. After controlling for age, significant relationships were found between sleep onset time, true sleep time, and number of night wakings at Time 1 and pubertal ratings at Time 2, and pubertal changes from Time 1 to Time 2. Delayed and disrupted sleep at Time 1 predicted faster pubertal changes from Time 1 to Time 2. These results were supported by structural equation modeling. These findings were similar in boys and girls.

Conclusions: Based on these longitudinal data, it appears that pubertal changes in sleep (delayed sleep phase and disrupted sleep patterns) antedate bodily changes associated with puberty. The underlying mechanisms explaining these predictive links should be further explored.

Keywords: Sleep, development, adolescence, puberty, longitudinal

Citation: Sadeh A; Dahl RE; Shahar G; Rosenblat-Stein S. Sleep and the transition to adolescence: a longitudinal study. SLEEP 2009;32(12):1602-1609.

THE ONSET OF PUBERTY HERALDS A DRAMATIC SET OF PHYSICAL, COGNITIVE, EMOTIONAL, BEHAVIOR-AL, AND SOCIAL CHANGES. THE TRANSITION INTO adolescence also brings on several new health concerns—including increases in risk-taking, sensation-seeking, depression, substance use, and accidents.¹⁻⁴ The onset of adolescence is also a time of both physiological and social changes that affect sleep.¹⁻⁸ Of particular concern from a health perspective is the evidence that many adolescents appear to get insufficient amounts of sleep; this is seen in the context of a growing recognition of the importance of sleep not only for physical health, but also for cognitive and affective function.^{1,3,5,9,10}

This myriad of interrelated changes in early adolescence raises a series of questions about the neurobehavioral underpinnings that impact not only normal development but also the increased vulnerability to clinical disorders that emerges in adolescence, and thus the potential role for sleep to influence healthy and unhealthy trajectories of development Recent studies have shown that the onset of adolescence is associated with unique aspects of brain development. For instance, it has been shown in MRI studies, that around the expected age of the onset of puberty, the volume of gray matter in the frontal and parietal lobes reaches a peak and starts decreasing thereafter. Studies of affective function show puberty-specific changes in neural systems involved in social and emotional processes. Evidence for puberty-specific changes in affective function have implications for understanding how brain/behavior/social

Submitted for publication January, 2009 Submitted in final revised form July, 2009 Accepted for publication September, 2009

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context interactions can lead to healthy or pathological trajectories of development. 16,17

These are high-stakes questions with relevance to a broad range of clinical and social policy issues affecting adolescent health. While it is important to acknowledge rapid progress beginning to occur to advance knowledge in these areas, it is also critical to point out the dearth of *longitudinal studies*. As has been well articulated by Kraemer and colleagues¹⁸ there are several limitations to drawing conclusions about development based on cross-sectional studies—particularly when trying to understand several interrelated processes. The main limitations include methodological issues such, as selection biases of different age samples and mis-specification of external factors, and the inability to identify distinct developmental trajectories. These concerns are highly relevant to understanding the complex biological, behavioral, and social changes in sleep that occur during adolescence.

Sleep-wake organization appears to undergo significant reorganization during the transition to adolescence. Research on the unique characteristics of sleep in adolescence has been extensive. The main changes in sleep-wake organization can be briefly summarized as follows: (a) a delayed sleep phase (a marked tendency for later bedtimes and rise times), which is associated with the onset of puberty^{7,8,11}; (b) shorter sleep, which is associated with increased levels of daytime sleepiness^{7,11,26,27}; (c) a steep decrease in delta NREM sleep, which is also associated with increased sleepiness¹⁹⁻²³; (d) greater tolerance for sleep deprivation or extended wakefulness with maturation^{6,24}; and (e) development of irregular sleep patterns among many adolescents, with irregular sleep patterns (sleeping very little during weekdays and accumulating sleep debt, and sleeping longer during weekends and partially compensating for their sleep loss).^{7,9,25}

Carskadon, who has been a leader in this field of research, documented with colleagues the role of psychosocial and bio-

logical factors leading to this developmental sleep phase delay, insufficient sleep, and daytime sleepiness in adolescents.^{2,4,9,26} These findings have been replicated and extended by other researchers.^{7,25,27-31} The psychosocial factors that affect the sleep phase delay include academic demands, social activities, afterschool employment, TV, computer, and Internet attractions. The biological mechanisms are not well understood, but the onset of puberty appears to be associated with sleep phase delay (when age is controlled for).^{4,7,31,32} This association has also been established in an animal model.³³

Studies on the links between sleep and puberty—particularly those performed in natural environments—have been mostly cross-sectional and few of these have included objective sleep measures. As mentioned previously, it has been shown that developmental conclusions drawn on the basis of cross-sectional studies can be quite misleading. The purpose of the present study was to explore the links between sleep and puberty, using a longitudinal design and objective sleep measures in natural settings.

METHODS

Procedure

This study was part of a larger study on sleep and neurobehavioral functioning during the transition to puberty. The study was approved by the Institutional Review Board of the university and by the Ministry of Education. The children were recruited from regular classes of 5 different elementary schools in the Tel Aviv area. All children and their parents signed informed consent. Each child completed a number of questionnaires and received an actigraph and a sleep diary for a week of monitoring.

The questionnaires included the Sexual Maturation Scale (SMS)^{43,48} and the Puberty Development Scale (PDS)³⁴ for pubertal development assessment.

The same assessment was repeated, at a similar time of year, for 2 successive years. The average follow-up interval time between Time 1 and Time 2 was 391 days (SD = 28), and the average follow-up interval time between Time 2 and Time 3 was 370 days (SD = 36). The parents completed questionnaires that included family background information, health information, and screening for child behavior problems and psychopathology.

Participants

Ninety-four children (41 boys and 53 girls) completed the first year of the study. At first assessment the age range was 9.9 to 11.2 years (mean age: 10.52). Eighty-two children completed the second year, and 72 completed the third year. All children were reportedly healthy with no chronic medical or psychiatric problems.

Measures

Pubertal Assessment

Two types of self-reports were used to assess pubertal changes. These instruments have been established as relatively reliable methods for assessing pubertal changes. One method is based on line drawings of Tanner stages of pubertal develop-

ment (pubic hair, male genitals, and female breast) and is often referred to as the Sexual Maturation Scale (SMS).^{43,48} A second method is based on a questionnaire, the Puberty Development Scale (PDS), which assesses development on various puberty-related domains.³⁴ This questionnaire, developed and validated by Petersen and colleagues, is particularly suitable for avoiding the need to present explicit pictures or drawings, which may be considered inappropriate in certain settings.³⁴⁻³⁷

In the current study we used both the PDS and the SMS. We used the SMS as our primary measure because of its higher accuracy and the PDS as an additional validation tool. The SMS provides a scale for pubic hair development and a scale for male genitals/female breast development. We allowed children to choose 2 drawings depicting neighboring stages if they were not certain which one fits best. Under such circumstances we averaged the 2 scores. For most analyses we averaged the scores of the pubic hair development with the score of the genitals/breast development. Therefore, we obtained a scale that represents a continuous measure rather than distinct stages. It has been argued that pubertal development is a continuous process and not a single event and such processes are better represented by continuous rather than dichotomized measures. 38,39

Sleep Assessment

Actigraphy and sleep diaries were used to assess sleep-wake patterns. The diary data were used only to detect and remove possible artifacts from the actigraphic data. Actigraphy has been established as a reliable and valid method to assess sleepwake patterns in infants, children, and adults. 52-54 The children were given miniature actigraphs (Mini Motionlogger, Ambulatory Monitoring, Inc) and were instructed to wear these on their non-dominant wrist in the evening when preparing for sleep and remove them in the morning. The actigraph was set to collect data in 1-min epochs and amplifier setting 18, which is the standard mode for sleep-wake scoring. Actigraphic raw data were translated to sleep measures using the actigraphic scoring analysis program (ASA) for an IBM-compatible PC. These sleep measures have been validated against polysomnography with agreement rates for minute-by-minute sleep-wake identification higher than 90%.40

Actigraphic sleep measures included: (1) sleep onset time - ST; (2) true sleep time, sleep time excluding all periods of wakefulness - TSLP; (3) sleep efficiency, percent of true sleep time from total sleep period – SEF; (4) number of night wakings lasting at least 5 minutes – WL. For comparisons of weekdays and Friday (the only day of the week which is not followed by a school day, these measures were averaged across the weekdays and Friday. For all other analyses these measures were averaged across all monitored nights.

The sleep diary included information on sleep schedule and subjective sleep quality (i.e., sleep onset time, rise time, number of night-wakings and their duration).

Data Analysis

The data analysis plan was aimed at assessing (a) the developmental changes in sleep and pubertal manifestations; and (b) the concomitant and predictive links between sleep and puberty. Multivariate analysis of variance (MANOVA) was used to assess developmental changes with Time (Time 1, Time 2,

and Time 3) as the within-subject repeated measures and sleep and puberty variables as the dependent measures. The links between sleep and puberty were assessed using partial Pearson correlations with age partialled out.

Finally, structural equation modeling (SEM) was used to develop a model representing all underlying associations between sleep and puberty. This sophisticated data analytic procedure is particularly well suited for the examination of the direction of the associations involving psychological and medical constructs, particularly as they unfold over time. In latent variable SEM, each construct is assessed as factors, or latent variables, using multiple indicators. This essentially nullifies error measurement (because factors do not include such an error component), consequently increasing psychometric reliability and in turn minimizing type II error. SEM also allows for the examination of effects of multiple "predictors" (exogenous variables) on multiple "outcomes" (endogenous variables), thereby increasing researcher's ability to gain an appreciation of complex relationships among variables as they change in time. A standard SEM analysis is comprised of testing a measurement model, which assesses the associations between the manifest indicators and their respective latent factors, and a structural model, which examines unidirectional (i.e., "causal") associations among the sleep and puberty latent factors was tested (see below).

To succinctly summarize the associations between pubertal development and sleep, as well as to examine the directionality of this association, a cross-lagged analysis of Times 1 and 2 data using SEM⁴¹ was employed. Time 3 data was not used because of the significant participants' drop-out. Our SEM model was based on 2 latent factors, namely, pubertal development and sleep, assessed at both Times 1 and 2. Pubertal development ("Puberty") was measured by means of pubic hair development and genital/breast development. Both measures were derived from the aforementioned SMS. Sleep was assessed via the sleep onset time (which served as the reference variable) and the true sleep time enabled from actigraphy.

The SEM model included synchronous (cross-sectional), stability (autoregressive) and cross-lagged paths. ⁴² To ensure measurement invariance across time, loadings of the manifest variables onto their respective latent factors were constrained to equality. As well, to account for shared method variance across time, autocorrelations were specified between the errors of the manifest variables (i.e., the part of the variance not related to the latent factor). Analyses were conducted using the AMOS 7.0 software, based on the maximum likelihood iteration procedure. AMOS 7.0 enabled a sophisticated imputation of missing data based on the full information maximum likelihood procedure. ⁴³

RESULTS

Sample Characteristics

The demographic characteristics of the sample are presented in Table 1. By Israeli standards (according to the level of education and family rooms per members) the sample consists of mostly upper-middle class families. Most of the children lived with both biological parents and most of the parents held full-time jobs.

Comparison of the initial data (at Time 1) obtained from children who dropped out (at Time 2 or Time 3) and those who

Table 1—Sample Characteristics

	Range	Mean ± SD		
Child's age	9.9-11.2	10.52 ± 0.32		
Mother's age	30–56	41.89 ± 5.51		
Father's age	31–62	45.11 ± 5.99		
Mother's education (y)	10–22	15.54 ± 2.86		
Father's education (y)	10–30	15.58 ± 4.05		
No. of children in family	1–5	2.48 ± 0.87		
No. of rooms at home	2–8	4.34 ± 1.09		
Birth order	Firstborn-43.18%; Latest-40.91%			
Employment (full time)	Fathers-84.34%; Mothers-51.65%			
Family structure	Two biological parents: 80.85% One-parent family: 14.89%			

completed the study revealed no significant difference on any of the demographic, sleep or puberty measures.

Development of Pubertal Status and Sleep

To identify developmental trajectories in pubertal status, the 2 scales, the PDS (questionnaire-based scale)³⁴ and the SMS (drawing-based scale),⁴⁴ were analyzed. The correlations between the SMS and PDS scores in our sample were 0.44, 0.53 and 0.66 for Time 1, Time 2, and Time 3, respectively.

A MANOVA with time as a repeated independent measure and sex as a between subject independent measure was used with the SMS and PDS scores as the dependent variables. The analysis of the PDS scale revealed 2 statistically significant effects (see Figure 1). Time was evinced as a statistically significant effect, $F_{2,65} = 29.07$, P < 0.0001, but this effect was embedded in a statistically significant time by sex interaction, $F_{2,65} = 9.03$, P < 0.0005. These results, presented in Figure 1, indicate that girls show more advanced changes in pubertal rating during Time 2 and Time 3.

On the SMS, a similar developmental trajectory was identified, such that time exerted a statistically significant main effect, $F_{2,65} = 107.26$, P < 0.0001, embedded in a time by sex statistically significant interaction, $F_{2,65} = 3.37$, P < 0.05. High stability across time has been demonstrated for the puberty measures over the 3 time points (Table 2).

To assess developmental changes in sleep we used MANO-VA with time as a repeated independent measure and sex as a between subject independent measure and the sleep measures as the dependent variables.

Actigraphic sleep measures reflected significant delay in sleep onset time with age (Figure 1). The main effect of time was statistically significant, $F_{2,65} = 56.97$, P < 0.0001, embedded in a statistically significant sex by time interaction $F_{2,65} = 3.36$, P < 0.05. A statistically significant main effect of time was also found for the true sleep time measure, $F_{2,132} = 33.81$, P < 0.0001. True sleep time was reduced with age. No developmental trajectories were found for any of the sleep quality measures.

Statistically significant sex differences were found for the sleep quality measures. Girls had higher sleep efficiency, $F_{1,66} = 4.56$, P < 0.05. Girls had fewer subjectively reported night wakings, $F_{1,66} = 4.56$, P < 0.05. High stability across time has been demonstrated for the actigraphic sleep measures over the 3 time points (Table 2).

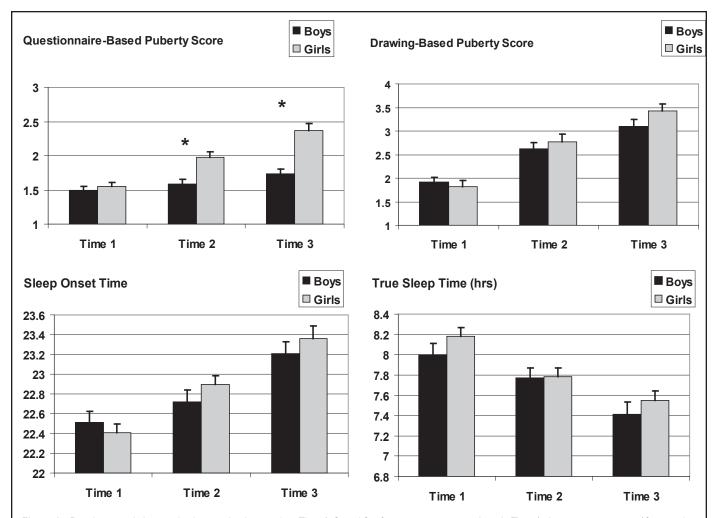


Figure 1—Developmental changes in sleep and puberty rating. Time 1, 2, and 3 refer to measurement points. At Time 1, the average age was 10.5 y and at Time 2 and Time 3, 11.5 and 12.5 y, respectively. The left upper figure is based on the PDS and the right upper figure is based on the SMS. Sleep measures are based on actigraphy. Post hoc analysis revealed that all discrete differences between Time 1, Time 2, and Time 3 are statistically significant. Significant gender differences are marked by *.

There were no significant associations between puberty measures and the Friday-weekdays sleep changes.

Sleep During Weekend Versus Weekdays

In Israel there is a 6-day school week, and only Friday night is followed by a non-school day (Saturday). Therefore sleep measures on Friday nights were compared to those on week-days (Figure 2).

ANOVA with Time (1st, 2nd, and 3rd administrations) and Night (Friday versus weekdays) as independent variables and sleep variables as the dependent measures revealed significant main Time and Night main effects, but no interactions. The significant Time effects have been reported earlier. Significant Night effect was found for sleep onset time, $F_{1,145} = 125.41$, P < 0.0001; true sleep time, $F_{1,145} = 12.04$, P < 0.001; sleep percent $F_{1,145} = 9.40$, P < 0.005; and number of night wakings $F_{1,145} = 22.31$, P < 0.0001.

Relationship Between Pubertal Development and Sleep

The Pearson correlations between sleep and pubertal development are presented in Table 3. Statistically significant correlations were found between sleep and puberty measures,

particularly during the early stage of the study (the first and second year). The correlations are mostly predictive in nature, such that sleep appears to predict puberty but not vice versa. Figure 3 describes 2 of the statistically significant correlations and the similarity between boys and girls in the association patterns.

To summarize the associations between pubertal development and sleep, as well as to examine the directionality of this association, we used a cross-lagged SEM analysis of Times 1 and 2 data.

The measurement model, which examines the associations between the manifest indicators and their respective latent factors, was established prior to the examination of the cross-lagged paths.⁴⁵ An excellent model fit was evinced ($\chi^2_{[df=12]} = 7.40$, p = 0.82; $\chi^2/df = 0.61$; NNFI = 1.06; CFI = 1.00; RMSEA = 0.00). Loadings of manifest variables onto their respective latent factors were strong and statistically significant (Range: |0.56| to |0.78|, P < 0.01).

Next, a structural model, examining unidirectional (i.e., "causal") associations among the sleep and puberty latent factors was tested. Results of the structural model are presented in Figure 4. As shown, a very clear-cut pattern emerged, whereby Time 1 levels of sleep predicted an increase in pubertal devel-

Table 2—Stability of Puberty and Sleep Measure Over Time—Correlations between Different Time Points

	Time 1 - Time 2	Time 2 - Time 3	Time 1 - Time 3
Puberty Measure			
SMS Score	0.66	0.75	0.46
PDS Score	0.48	0.67	0.29
Sleep Measure			
Sleep onset time	0.66	0.67	0.53
True sleep time	0.60	0.49	0.53
Night wakings	0.70	0.61	0.60
Sleep percent	0.68	0.67	0.58
Quiet sleep percent	0.72	0.77	0.69

opment over time (β = 0.45, P < 0.005), but not vice versa. These findings emerged even after controlling for participants' sex and age.

*All correlations are significant at P < 0.0001 except for r = 0.29, P < 0.05

DISCUSSION

This is, to the best of our knowledge, the first longitudinal study focusing on sleep and puberty that employed objective sleep measures obtained in natural home environments amidst the social complexities of adolescents' real lives. The findings reflect several of the anticipated developmental processes in sleep and pubertal changes. Over the course of 2 years of development (from Time 1 to Time 3), sleep onset was significantly delayed (by an average of 50 min) and true sleep time was significantly shortened (by an average of 37 min). There were no significant changes associated with measures of sleep quality (i.e., night wakings or sleep efficiency). These findings are similar to those obtained in a cross-sectional actigraphic sleep study in children in similar ages. 46 Interestingly, in both these cross-sectional and longitudinal studies of development, a significant reduction in sleep time was noted with no compensatory improvement in sleep quality. However, in a sleep restriction/extension study in children in similar age range, when sleep time was manipulated for 3 nights by similar doses (i.e., 40 min), a compensatory improvement of sleep quality was noted in response to sleep restriction.⁴⁷ These results may be indicative of a decreased drive toward deep sleep and or reduced process "S" (rather than simply a delay in timing of bedtime leading to relative sleep restriction). This may reflect a shift in sleep physiology, not simply circadian or social changes associated with development. In addition to the expected changes in sleep patterns, a relatively strong stability for sleep measures over time was documented. This finding suggests that in spite of numerous developmental changes the sleep-wake patterns continue to be relatively stable and robust individual characteristics over time.

As expected, significant differences were found between sleep on Friday versus sleep on weekdays. On Fridays, sleep onset was delayed, true sleep time was extended, and sleep quality was poorer in comparison to weekdays. However, contrary to other reports,⁷ this weekend-weekday difference was not in interaction with the year of administration and was not associ-

Table 3—Pearson Correlations between Sleep and Puberty Measures with Age and Sex Partialled Out

	Pub1	Pub2	Pub3	Pub1-2	Pub2-3
Time 1					
ST	-0.07	0.23*	0.20	0.36***	-0.14
TSLP	-0.01	-0.32***	-0.30*	-0.42#	0.11
SEF	-0.02	-0.29*	-0.17	-0.37***	0.16
WL	0.01	0.28*	0.15	0.35***	-0.16
Time 2					
ST	-0.07	0.11	0.02	0.20	-0.19
TSLP	-0.06	-0.22	-0.17	-0.21	0.04
SEF	-0.03	-0.19	-0.10	-0.21	0.01
WL	0.05	0.19	0.11	0.20	-0.01
Time 3					
ST	0.00	0.17	80.0	0.19	-0.15
TSLP	-0.03	-0.01	-0.03	0.00	0.01
SEF1	0.02	-0.02	-0.02	-0.06	-0.12
WL1	0.05	0.11	0.08	0.09	0.08

*P <0.05; **P <0.01; ***P <0.005; #P <0.0005

Pub1 = Puberty rating at Time 1; Pub2 = Puberty rating at Time 2; Pub3 = Puberty rating at Time 3; Pub1-2 = the increase in puberty rating from Time 1 to Time 2; Pub2-3 = the increase in puberty rating from Time 2 to Time 3; ST = Sleep onset time; TLSP = True sleep time; SEF = Sleep efficiency; WL = Number of night wakings. All puberty measures are based on the Sexual Maturation Scale.

ated with puberty status or gender, suggesting that perhaps this tendency for weekend compensatory sleep is relatively steady over the period of early adolescence at least within the structure of the 6-day school week in Israel. This issue should be further explored within different school-week structures and cultural settings using objective sleep measures.

The assessment of sexual maturation is complex because it includes a variety of physical changes that usually overlap, but represent separate physiological processes including gonadarche, adrenarche, and rapid physical growth. 43-46 Furthermore, various methods developed for the assessment of puberty (e.g., physical exam, self-report, parental report) have yielded agreement levels that range from low to excellent in different studies.44,46-49 In the current study, we used the self-report based on the line drawings of Tanner stages of pubertal development the SMS, 43,48 and the PDS which assesses development on various puberty-related domains.³⁴ The correlations between the SMS and PDS scores in our sample were in the modest-to-high range, suggesting that they do indeed tap into the same pubertal process, albeit with important differences in focus. As expected, a significant increase in pubertal status was found on both the SMS and PDS instruments. On the PDS, girls advanced faster than boys in reporting on pubertal signs consistent with the well-established finding that girls mature earlier than boys. In addition to these development changes, a strong stability over time was noted for the scores on both of these instruments suggesting that during this age period the reports on pubertal signs represent a stable individual characteristic.

The analysis of the relationship between sleep and physical signs of puberty revealed an interesting but complex picture.

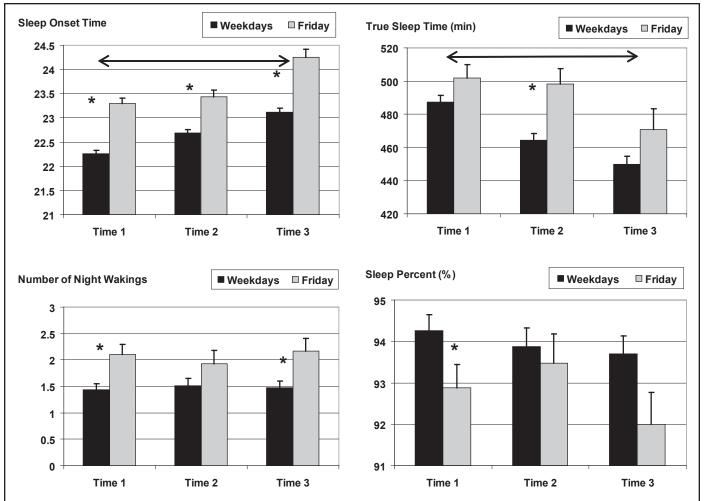


Figure 2—Sleep measures on weekdays versus Friday. Time 1, 2, and 3 refer to measurement points. At Time 1, the average age was 10.5 y and at Time 2 and Time 3, 11.5 and 12.5 y, respectively. Friday night is the only night that is not followed by a school day. Arrows between Time 1 and Time 3 indicate that all discrete post hoc analyses of the differences between Time 1, Time 2, and Time 3 were statistically significant.

There were no significant links between sleep measures and the PDS score. However, while controlling for age, significant correlations were found between the sleep measures and SMS scores during Time 1, Time 2 and the increase in SMS score from Time 1 to Time 2. These correlations indicate that reporting a higher level of pubertal ratings or an increase in pubertal ratings over time was associated with delayed sleep onset, reduced true sleep time, increased number of night wakings, and reduced sleep efficiency. Delayed sleep onset and reduced sleep time and sleep quality predicted more pubertal development from Time 1 to Time 2. This was true for the entire sample as well as within each sex. The SEM analysis focusing on Time 1 and Time 2 provided additional evidence that during this early period of pubertal development maturational changes in the sleep-wake patterns predicted pubertal changes, whereas there was no similar prediction in the opposite direction. These findings suggest that adolescent changes in the sleep-wake system were evident before the bodily manifestations of puberty.

The association between puberty and the delayed sleep phase and decrease in sleep time has been documented in previous studies.^{8,11,40-42} However, the association between the puberty and sleep quality has not been well established before. Previous research has documented significant decrease in slow wave

sleep (SWS) and an associated increase in sleepiness during adolescence, ²⁸⁻³² findings that may be related to the decrease in sleep quality detected by actigraphy in our study. However, it is important to note that on the basis of their mixed cross-sectional/ longitudinal study, Feinberg and colleagues argued that the decline in SWS is associated with age and sex rather than with pubertal stage. ²³

The fact that changes in the sleep-wake system predicted pubertal development suggests that neurobehavioral changes associated with adrenarche and/or gonadarche^{14-18,32} may be evident earlier in measures of sleep organization and only later are these manifest in the bodily changes associated with puberty. The relatively early timing of the significant correlations in our study (Time 1 – Time 2 as opposed to Time 2 – Time 3) suggests that changes in the sleep-wake system associated with the transition into adolescence is linked to the *onset* of puberty in most of the children, around the ages of 11–12, which is roughly the timing of the dramatic changes seen in SWS and the sex differentiation in SWS.²³

It is important to address the limitations of our study. Pubertal assessment was based on self-reports without external objective validation. However, we used two pubertal assessment tools which showed reasonable agreement and stability over

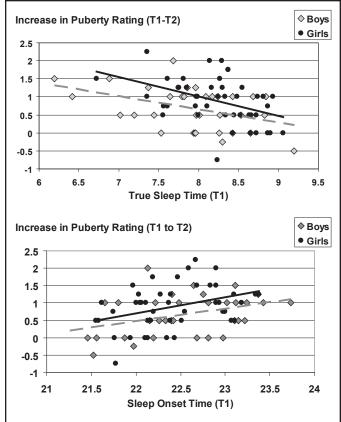


Figure 3—Correlations between sleep measures (sleep onset time and true sleep time) at Time 1 and the increase from Time1 to Time 2 in puberty rating on the SMS. Regression lines are plotted in black for girls and in broken gray line for boys. No significant sex differences were found.

time. Another limitation is the relatively large age range (9.9 to 11.2 years) at the time 1 assessment; to control for this, age variance was controlled in all analyses.

Considering the limitations of the study and the fact that some of the findings have not been reported before, additional research, starting at an earlier age and collecting hormonal data as well as other measures of puberty, is needed for validation and to explore underlying mechanisms.³³ Future studies could potentially disentangle the specific pubertal changes that are associated with maturational changes in sleep (which might explain why, for example, our findings show strong links between sleep and puberty as measured by the SMS but not by the PDS). Ultimately, a deeper understanding of the normal maturational changes regarding sleep and pubertal maturation—and the specific neural and hormonal underpinnings of these changes—may provide new insights into clinically relevant questions about the emergence of new vulnerabilities regarding behavioral and emotional health in early adolescence. Given the high rates of sleep problems and growing evidence of the importance of sleep in health, a better understanding of the interrelationships (and sequences of developmental changes) should help to inform opportunities for prevention and early intervention.

ACKNOWLEDGMENTS

The research was supported by the Israel Science Foundation (Grant # 334/04). We would like to thank all the participat-

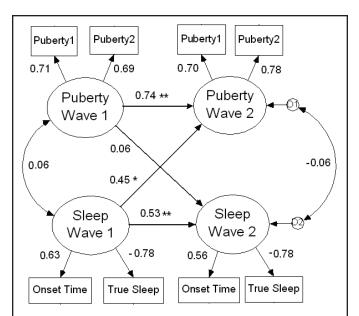


Figure 4—Cross-lagged structural equation model for the relationships between sleep and puberty rating (Time 1 and Time 2). Puberty1 = SMS puberty score on pubic hair development. Puberty2 = SMS puberty score on genital development in boys and breast development in girls. D1 and D2 represent "disturbances," namely, the part of the variable of an endogenous ("dependent") latent variable that is not accounted for by predictors in the SEM model. * P < 0.005, ** P < 0.001.

ing families and the students who served as research assistants. Special thanks to Ornit Arbel who coordinated all the logistics of this study and to Jodi Mindell for her valuable comments.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

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