# Adolescent Sleep Patterns, Circadian Timing, and Sleepiness at a Transition to Early School Days

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**Study Objectives:** This study examined effects on adolescent sleep patterns, sleepiness, and circadian phase of a school transition requiring an earlier start.
**Design and Setting:** Adolescents were evaluated in 9th and 10th grades; school start time in 9th grade was 0825 and in 10th grade was 0720. Assessments at each point included 2 weeks of actigraphy and sleep diaries at home, followed by a 22-hour laboratory evaluation, including evening saliva samples every 30 minutes in dim light for determination of dim-light salivary melatonin onset phase (DLSMO), overnight sleep monitoring, and multiple sleep latency test (MSLT).
**Participants:** Twenty-five females and 15 males, ages 14 to 16.2 were enrolled; 32 completed the study in 9th grade and 26 completed in 10th grade.
**Interventions:** Participants kept their own schedules, except that laboratory nights were scheduled based upon school-night sleep patterns.
**Measurements and Results:** According to actigraphy, students woke earlier on school days in 10th than in 9th grade, but they did not go to sleep earlier and they slept less. DLSMO phase was later in 10th grade (mean = 2102) than 9th grade (mean = 2024). Sleep latency on MSLT overall was shorter in 10th (mean = 8.5 minutes) than in 9th (mean = 11. 4 minutes), particularly on the first test of the morning at 0830 (5.1 vs 10.9 minutes). Two REM episodes on MSLT occurred in 16% of participants in 10th grade; one REM episode occurred in 48%. When those with REM sleep on one or both morning MSLTs (n=11) were compared to those without morning REM, significant differences included shorter sleep latency on the first test, less slow wave sleep the night before, and later DLSMO phase in those who had morning REM.
**Conclusions:** Early start time was associated with significant sleep deprivation and daytime sleepiness. The occurrence of REM sleep on MSLT indicates that clinicians should exercise caution in interpreting MSLT REM sleep in adolescents evaluated on their “usual” schedules. Psychosocial influences and changes in bioregulatory systems controlling sleep may limit teenagers’ capacities to make adequate adjustments to an early school schedule.
**Key words:** Adolescent; sleep; sleepiness; multiple sleep latency test; melatonin; circadian rhythms

ADOLESCENTS STAY UP LATER than preteens and show a marked delay in spontaneous morning arousal.[1-7] Most reports acknowledge that this pattern is widespread, if not universal, and commonly ascribe it to psychosocial factors that achieve increasing salience in adolescence. For example, with maturation comes an increased desire for independence, greater social opportunities, enhanced response to peer pressure, perhaps more academic responsibilities, more possibilities for extracurricular activities and sports, and—in the US—more time spent working for pay.[8] Many of these phenomena promote a delay in the timing of sleep.

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We have recently begun to examine whether intrinsic biological factors also influence the timing of sleep across adolescent development. Current conceptual models for the control of sleep posit a dynamic interaction of the sleep-wake homeostatic system and the circadian timing system.[9-13] In these models, the homeostatic process accounts for development of sleepiness and accumulation of slow-wave activity with extended waking or with accumulating insufficient sleep. We propose that across adolescence, the daily accumulation of such sleep pressure builds more slowly, and thus facilitates staying awake longer in older than in younger adolescents.[14] Preliminary support for a maturational difference in homeostatic regulation of sleep comes from pilot data showing a reduced amount of slow-wave sleep following sleep deprivation in more mature than in less mature adolescents.[15]

Circadian mechanisms are thought to time the occurrence of sleep[16] and sleepiness,[17] subjective alertness,[18,19] and REM sleep within sleep.[20,21] We have proposed that a delay of circadian phase during adolescent development also contributes to the delay of sleeping times in adolescents.[14] Evidence in support of this second hypothesis comes from a correlation of self reports of pubertal development and circadian phase preference,[22] and—more strongly—from a correlation of physical measurements of puberty[23] with the offset phase of melatonin secretion measured in a constant routine.[24] Maturational changes in other circadian processes may also play a role, including alterations in intrinsic period, phase alignments, or phase response to light.

These findings support the likelihood that adolescent development is associated with psychosocial and biological pressures toward later bedtimes and rising times. Teenagers in the United States, however, confront a powerful social demand in opposition to the phase-delay tendency: school starting time. A common practice in US school districts is to begin school earlier for adolescents than for younger children. This practice is typically implemented by moving the school-day start to earlier times at transitions into new school settings—for example, from 0845 in grammar school to 0800 in middle school or junior high school, and then to 0730 or 0715 or earlier in high school. This advance of the school day is in direct conflict with a putative pubertal/adolescent phase delay.

The present study examined teenagers' responses to an earlier school start time in terms of sleep patterns, daytime sleepiness, and circadian timing. We reasoned that if puberty is associated with a delay, and transition to high school involves a phase advance, then certain teenagers will have difficulty at such a transition, manifesting less sleep and greater daytime sleepiness. On the other hand, teenagers who make appropriate responses to the external demand of early school start will go to bed earlier to maintain sleep amount and will not become sleepier in the daytime. The latter adaptive response would be expected if rising to go to school early exposes the circadian system to light at a phase when light exposure facilitates a phase advance.[25, 26]

Our study examines these issues using field and laboratory measures to evaluate the transition of 9th grade students to 10th grade in a setting that involves a 65-minute phase advance in the starting time of school.

## METHODS

### Participants

Participants were recruited through pamphlets distributed to 9th grade students at two junior high schools in one Rhode Island public school district with starting time of 0825*. Students from these junior high schools progress to 10th grade in a high school with a start time of 0720. Telephone interviews of volunteers and parents were used to exclude students with a personal or family history of sleep disorders, chronic or current illness, or evidence of learning disability, psychopathology, or drug use. The telephone interview also included items to assess morningness/eveningness preference,[27] and participants were selected to favor a broad range of scores on this scale. A description of study procedures was given and informed consent obtained from participants and their parents at an informational meeting in accordance with procedures approved by the E.P. Bradley Hospital Institutional Review Board for the protection of human subjects. Participants received monetary compensation.

Forty volunteers—25 females and 15 males, ages 14 to 16.2 years (mean age 15.0±0.5)—were enrolled in the study. Thirty-two participants completed all portions of the study in the 9th grade, and 26 completed all portions in 10th grade.

### Procedures

Participants began the study in the second half of 9th grade during the spring and concluded the study in the first half of 10th grade in the fall. Most students also completed a partial study during the summer, which is not included in this report. A subgroup of 12 participants repeated parts of the protocol in the second half (spring) of 10th grade.

Each phase of the project included 2 weeks of actigraph recording (Mini-Act Actigraph, AMI, Ardsley, NY) coupled with daily sleep diary completion. Actigraphy is widely accepted as a relatively noninvasive means to estimate sleep patterns under nonlaboratory conditions,[28] and

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* Throughout this paper, all times are given in 24-hour clock time in US eastern daylight savings zone.

we are confident of these estimates when paired with a behavioral indicator of sleep, such as sleep diary self-report.[29,30] Participants were instructed in the use of the actigraphs and how to complete sleep diaries, but no constraints were placed on their sleep schedules. Participants were also asked to telephone the laboratory's answering machine each day upon arising. The latter procedure was implemented as an aid to compliance in completing the diary and also as a secondary check on daily reports of sleep schedule used in analyzing the actigraph data.

Actigraph data were analyzed for "sleep" and "wake" by applying a validated algorithm[30] to the portions of the records identified as sleep periods by the combination of sleep diary and telephone log. Questionable records were examined by a group and consensus judgments made regarding acceptability of the data. Nights were excluded if they did not show reasonable concordance between self-report and actigraph record (eg, reported bedtime of 2230 but continuous sleep scored from 2100), and if the adolescent was sick, the actigraph was off or not working for all or part of a night, or the actigraph record included unusual external motion, such as sleeping in a car. On average, 8.9 (of 10 possible) school nights were scored for students in the 9th grade and 9.4 in the 10th grade, representing less than one unscorable night per student overall. Only 8 nights in the entire data set were lost due to a lack of concordance between self-report and actigraph record. Nights were coded as school nights (if the participant indicated attendance at school the next day) or nonschool nights (which included weekends, school holidays, and other days when the students did not attend school). Actigraph records were used to assess students' sleep patterns, not sleep quality per se. Hence, three actigraph variables were derived for each night: sleep-onset time was defined as the start of the first 3 consecutive minutes scored as sleep by the algorithm; sleep-offset time was the end of the last 5 consecutive minutes scored as sleep; and total sleep time was the amount of sleep scored by the algorithm from sleep onset to sleep offset time.

Participants came to the laboratory on a Friday or Saturday evening at the end of the 2 weeks of actigraphy to complete the in-lab portion of the study. For those participants whose studies occurred on Saturday night, Friday night bedtime and wake-up time on Saturday morning were fixed at the mean actigraphically defined school-night sleep times determined from the first week's actigraph record. Thus, participants were assessed after 5 or 6 nights on their school-night schedule.

Participants' in-lab schedules were set according to school-night actigraph records of week 1. In-lab bedtime and rising time were set at the quarter hour earlier than (bedtime) and later than (rise time) the mean school-night sleep-onset and sleep-offset times. Participants were scheduled to arrive at the lab approximately 5 hours before the mean school-night sleep onset time for collection of saliva samples to determine melatonin secretory onset phase. Melatonin is a hormone secreted nocturnally by the pineal gland under direct control of the intrinsic circadian pacemaker.[31] Melatonin is detectable in plasma and saliva, and its metabolite, 6-sulfatoxymelatonin, is detectable in urine. Suppression of melatonin secretion occurs in the presence of moderately bright light.[32] The time of the evening onset of melatonin secretion under dim light conditions—dim-light melatonin onset, or DLMO—has been used as a marker for the phase position of the circadian oscillator, usually from plasma samples obtained at frequent (about 30-minute) intervals.[33] A relatively noninvasive method of obtaining this measure is to collect saliva at similarly frequent intervals, providing a measure we call the dim-light salivary melatonin onset or DLSMO phase. We use DLSMO to mark the phase of the intrinsic circadian timing system in teenagers.[24]

Participants were kept in dim light (≤50 lux) until bedtime for saliva collection, which was stimulated by chewing Parafilm®. Seven subjects in 9th grade provided 2 ml of saliva at 60-minute intervals, while the remaining 9th and 10th grade samples were provided at 30-minute intervals. Samples were frozen (-20°C) within 4 hours, and vials of frozen saliva were shipped in dry ice to Elias, Inc. (Osceola, Wis) for melatonin radioimmunoassay. [The detection limit of the assay was .75 pg/ml, and the intra-assay coefficients of variation (n=11) were 12.1%, 5.7%, and 9.8% at mean concentrations of 16.5 pg/ml, 68.7 pg/ml, and 162.7 pg/ml, respectively. The interassay coefficients of variation (n=10) were 13.2%, 8.4%, and 9.2% at mean concentrations of 17.3 pg/ml, 69.0 pg/ml, and 164.7 pg/ml, respectively.]

Deacon and Arendt[34] have shown a direct correlation between plasma and salivary melatonin levels, with salivary melatonin levels approximately 40% (±6.9%) of concurrent plasma levels. Because 10 pg/ml is the threshold commonly used to determine dim-light melatonin onset from plasma melatonin levels,[35] we chose 4 pg/ml from salivary samples to denote salivary melatonin onset as a marker of circadian phase. Melatonin onset phase (DLSMO) was calculated as the linearly interpolated time of the first sample above 4 pg/ml that was followed by a higher value. When data did not permit application of this algorithm, a conservative estimate was used. To wit, in several cases when the threshold value was exceeded on the first evening sample, the time of that sample was used as the DLSMO phase (n=3); conversely, if 4 pg/ml was not achieved by the final sample, the midpoint between the final sample and the time the next sample would have occurred (eg, 30 minutes later) was used as the DLSMO phase (n=6). These estimates were made to enable us to use

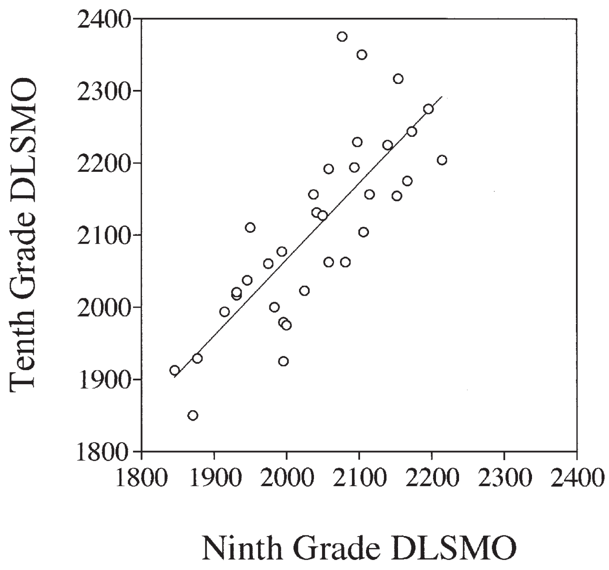

**Figure 1.—**The phase of dim-light salivary melatonin onset (DLSMO) is displayed for students in 9th and 10th grade assessments. Although the measures remained stable within the group (r= .80), this marker of circadian phase position showed a significant delay in the 10th grade as compared to 9th. A simple linear regression line is also plotted.

the entire participating sample for our analyses; they are conservative because they do not exaggerate either early or late melatonin onset values. As such, they preserve the power of our DLSMO analyses. Without these values, most results are unchanged, though in some instances reduction of statistical power compromised statistical significance.

Overnight monitoring included continuous recording of electroencephalogram ($C_3/A_2$ or $C_4/A_1$ and $O_2/A_1$ or $O_1/A_2$), electrooculogram (right and left outer canthus), and electromyogram (mentalis/submentalis), according to standard techniques,[36] and electrocardiogram from modified lead II. The multiple sleep latency test (MSLT) was monitored four times across the day according to standard guidelines,[37] with the exception that all tests lasted 20 minutes regardless of whether sleep occurred. The MSLT is an accepted means of assessing sleep tendency under standard conditions[37] through repeated assessments of the speed of falling asleep. This measure has been used extensively in adolescents and adults. The MSLT also marks daytime sleep structure anomalies, such as the occurrence of REM sleep within 10 or 15 minutes of sleep onset, which is commonly used to aid in the diagnosis of narcolepsy.[38,39] All nocturnal sleep recordings and MSLTs were scored visually in 30-second epochs according to standard criteria.[36]

Repeated-measures analysis of variance was used to analyze dependent variables across sessions (9th and 10th grades) with sex as a between-subjects factor. For subgroups at a single time point, groups were compared using

**Table 1.—**Mean (standard deviation) for sleep variables from 2 weeks of actigraph monitoring in 9th and 10th grades

| | 9th Grade | 10th Grade | Significant effects |
|---|---|---|---|
| School Nights | | | |
| Sleep onset time | | | |
| Boys (n=13) | 2247 (31) | 2245 (31) | |
| Girls (n=22) | 2239 (39) | 2234 (51) | |
| Overall | 2242 (36) | 2238 (44) | |
| Sleep offset time | | | |
| Boys (n=13) | 0620 (32) | 0605 (20) | Session (F= 33.25, p<001) |
| Girls (n=22) | 0630 (24) | 0558 (19) | Sex by session (F=4.39, p=.044) |
| Overall | 0626 (28) | 0601 (19) | |
| Total sleep time | | | |
| Boys (n=13) | 402 (40) | 390 (33) | Sex (F=8.80, p=.006) |
| Girls (n=22) | 446 (43) | 422 (42) | Session (F=9.07, p=.005) |
| Overall | 429 (47) | 410 (42) | |
| Nonschool nights | | | |
| Sleep onset time | | | |
| Boys (n=11) | 2325 (43) | 2343 (50) | Sex by session (F=5.13, p=.032) |
| Girls (n=18) | 2347 (56) | 2345 (57) | |
| Overall | 2338 (52) | 2344 (53) | |
| Sleep offset time | | | |
| Boys (n=11) | 0755 (51) | 0803 (55) | Sex (F=4.87, p=.036) |
| Girls (n=18) | 0849 (72) | 0851 (67) | |
| Overall | 0828 (69) | 0832 (66) | |
| Total sleep tme | | | |
| Boys (n=11) | 439 (46) | 426 (37) | Sex (F=25.35, p<.001) |
| Girls (n=18) | 515 (42) | 512 (60) | |
| Overall | 485 (57) | 478 (67) | |

**Table 2.—**Mean (standard deviation) time of the dim-light salivary melatonin onset (DLSMO) in 9th and 10th grades

| | 9th grade | 10th grade | Significant effects |
|---|---|---|---|
| Boys (n = 13) | 2057 (42) | 2134 (67) | Session (F=21.66, p<.001) |
| Girls (n = 22) | 2005 (58) | 2044 (77) | Sex (F=5.86, p=.021) |
| Overall | 2024 (57) | 2102 (77) | |
| | 10th Grade Fall | 10th Grade Spring | |
| Overall (n = 12) | 2011 (57) | 2026 (67) | |

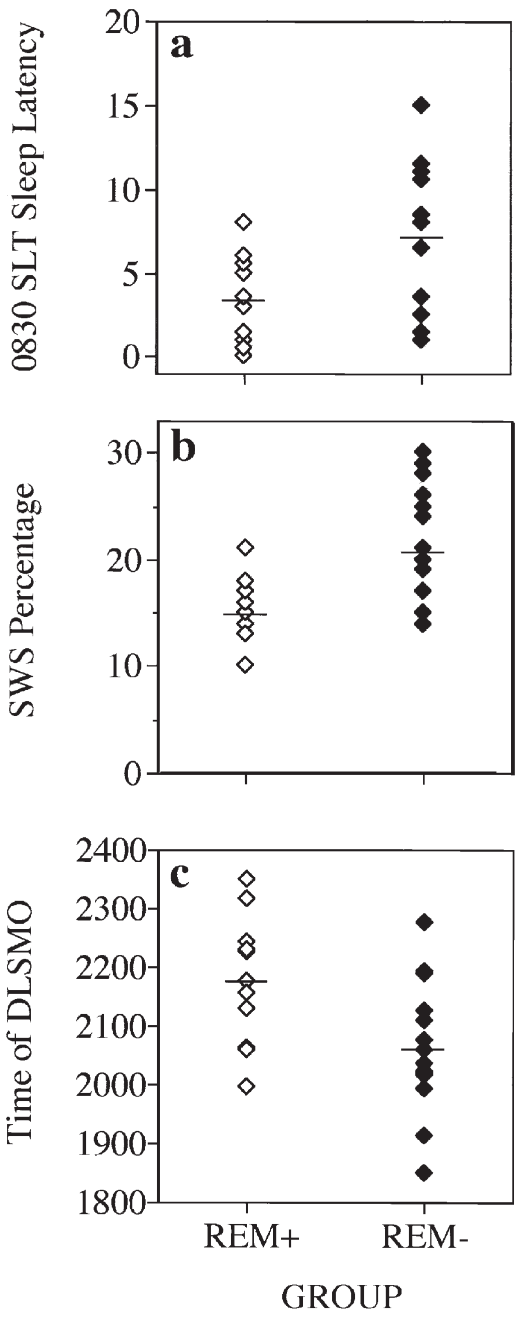

**Figure 2.—**Participants who did (REM+) and did not (REM-) have REM sleep on morning MSLTs are compared on three parameters in these plots. (a ) Sleep latency on the 0830 MSLT. (b) Percentage of total sleep spent in slow wave (stages 3-4) sleep on the prior night. (c) DLSMO phase. Horizontal lines indicate group mean values.

*t* tests for independent groups. Single logistic regression was used to determine odds ratios for significant factors relating to group designations. Pearson correlations were calculated as appropriate. An alpha of .05 was used to determine statistical significance.

## RESULTS

No effects of age were identified for variables investigated in this project.

**9th vs 10th Grade.—**Table 1 displays means and standard deviations for variables estimated from actigraphy obtained from school nights and nonschool nights based on the 2-week recordings. These data show no significant change of school-night sleep-onset time with the transition to 10th grade; however, both sleep-offset time and total sleep time demonstrated significant modifications. Sleep offset was approximately 25 minutes earlier on average in 10th than 9th grade, with a significant session-by-sex interaction: for boys, the sleep offset time was about 15 minutes earlier in 10th grade, whereas girls woke up about 30 minutes earlier in 10th grade than in 9th grade. These changes are also reflected by lower school-night total sleep time (about 20 minutes) in 10th than in 9th grade. Boys slept less than girls on both sessions.

Actigraphic estimates of sleep from nonschool nights showed no significant main effect of session; however, a session-by-sex interaction was found for sleep-onset time, with boys manifesting a delay of sleep onset in 10th grade. Nonschool-night sleep-offset times and total sleep times were unaffected by session; however, a significant effect of sex was found for both measures—boys woke earlier and slept less than girls at both time points.

The time of the DLSMO (Table 2) changed significantly from 9th to 10th grade, and a main effect of sex was also statistically significant. Thus, boys in general had later times of melatonin secretory onset than girls by about 50 minutes on average. Overall, a delay of melatonin onset time of about 40 minutes occurred across the transition from 9th to 10th grades. Even with this mean overall change, the adolescents' relative ranks on DLSMO remained highly stable. Figure 1 illustrates the 9th and 10th grade DLSMO values, reflecting the stability ($r=.80$, $p<001$) of scores across this interval. Because we were concerned that students might not have adjusted fully to the school schedule in the 10th grade assessment (which occurred after 3 to 8 weeks of school), we reassessed 12 students (9 girls, 3 boys) approximately 5 months later. As shown in the lower portion of Table 2, the DLSMO times in this subgroup did not show a statistically significant difference (paired *t* test; $t=.59$, ns) between the two 10th grade assessments.

Data from the laboratory assessments are presented in Tables 3 and 4. We note that eight participants were not recorded on one session due to construction in the laboratory, and seven were not able to schedule the overnight in either the 9th (n=1) or 10th (n=6) grades. In the analysis of nocturnal sleep recording data (Table 3) from the 25 youngsters recorded on both sessions, no significant sex differences or interactions were found; therefore, means and

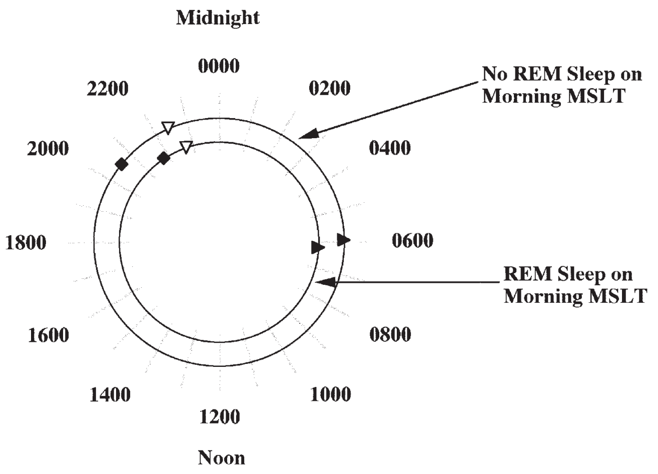

**Figure 3.**—The average time of actigraphically estimated sleep onset (∇) and sleep offset (►) on school nights and the time of DLSMO (◆) phase for participants who did (inner arc) and did not (outer arc) have REM sleep on morning MSLTs are displayed on this 24-hour clock.

standard deviations are presented for boys and girls combined. Three sleep parameters showed statistically significant changes from 9th to 10th grade. Total dark time decreased in 10th grade, as the laboratory schedule reflected the participants' school-night sleep schedule changes; yet total sleep time was not significantly shorter in 10th than in 9th grade. Sleep consolidation improved somewhat (increased 10th grade sleep efficiency, F=3.92, p=.06), though this effect was not statistically significant. The reduction in stage 1 percentage may also reflect greater sleep consolidation in the 10th than in the 9th grade. Finally, REM latency changed significantly, overall approximately 25 minutes shorter in 10th grade.

The MSLT data are shown in Table 4. No significant main or interaction effects of sex occurred. Overall, the daily mean MSLT was lower in 10th than in 9th grade, principally due to significantly shorter sleep latency on the first (0830) test. The lower portion of Table 4 indicates the number of participants with REM sleep during each MSLT and overall. Four of the 32 participants who took the MSLT in 9th grade and 12 of the 25 who took the MSLT in 10th grade manifested REM sleep on at least one test. (The four with REM sleep in 9th grade also showed REM sleep in 10th grade.) In 9th grade, three students had REM on the 0830 and 1030 SLTs, and one had REM only on the 0830 SLT. In the 10th grade, four students had two or more REM episodes on MSLT, seven had a single episode at either 0830 (n=5) or 1030 (n=2), and only one student showed a single REM episode during an afternoon (1230) test. Eleven students (5 boys, 6 girls), therefore, had REM sleep in 10th grade on either or both of the morning sleep latency tests. Although REM episodes on the MSLT are often an indication of narcolepsy, no student had family history or complaints—other than sleepiness—of that sleep disorder.

**Morning REM vs noREM or only afternoon REM.**—Because of the high number of REM episodes on MSLT, particularly in the morning, we wished to examine whether those subjects in whom morning REM occurred differed from those without REM. Therefore, we selected a series of variables that might predict or affect the MSLT REM sleep structure. These variables included those marking increased sleepiness, typically associated with REM sleep on MSLT in narcolepsy and in studies of normals assessed on short sleep periods[40,41]—mean daily MSLT sleep latency, sleep latency on the morning MSLTs, and nocturnal sleep latency to stage 1. We also included variables that might predict or cause greater daytime sleepiness—nocturnal total sleep time, stage 1 sleep percentage, and slow-wave sleep (SWS) percentage. Two parameters were selected because they might indicate REM "pressure" in general—nocturnal REM sleep percentage and REM sleep latency. Finally, three measures were chosen as indicators of sleep timing or circadian phase, which has a known relationship to REM sleep timing[20,21]—mean sleep-onset and sleep-offset times estimated from actigraphy on the 5 nights preceding the in-lab night and time of DLSMO from the night preceding MSLT.

Table 5 shows the means and standard deviations for these variables in subjects who did and did not have REM sleep on either or both of the morning SLTs in 10th grade. Only three variables showed a statistically significant difference between groups: sleep latency on the first sleep latency test, nocturnal SWS percentage, and time of the DLSMO. These results are displayed in Fig. 2. Additional analysis showed that the phase angle (time difference) between DLSMO and sleep onset was significantly (t=2.88, p=.008) shorter in the group with REM sleep on morning MSLT (46±52 minutes) vs those without REM sleep on MSLT (113±61 minutes). Figure 3 illustrates phase relationships in these two groups among in-lab sleep onset, sleep offset, and melatonin onset.

We examined the predictive value of each individual statistically significant effect using logistic regression to obtain odds ratios. For DLSMO, the odds ratio was 2.77 (p<.05), indicating that for each delay of melatonin onset of 1 hour, the odds of showing REM sleep on a morning MSLT increased almost threefold. Also, 73% of this sample could be correctly classified as to their MSLT-REM status based on knowledge of DLSMO alone. The odds ratio for predicting REM on MSLT from MSLT nap 1 latency was 1.28 and for SWS percentage was 1.37.

Finally, because of our concerns about the relation of circadian phase to sleep patterns, we computed correlation coefficients between DLSMO and actigraphically estimat-

**Table 3.—**Laboratory sleep variables for boys (n=10) and girls (n=15) recorded in both 9th and 10th grades—mean (standard deviation)

| Variables | 9th grade | 10th grade | Significant effects |
|---|---|---|---|
| Sleep-onset time | 2246 (29) | 2231 (38) | |
| Minutes total dark time | 477 (37) | 460 (40) | Session (F=5.47, p=.029) |
| Minutes total sleep time | 448 (36) | 440 (42) | |
| Latency to stage 1 sleep (min.) | 11.5 (7.5) | 9.3 (10.3) | |
| REM sleep latency (min.) | 155 (53) | 120 (43) | Session (F=7.35, p=.012) |
| Percentage stage 1 sleep | 6.5 (3.6) | 4.2 (1.8) | Session (F=6.62, p=.017) |
| Percentage stage 2 sleep | 57.0 (5.3) | 58.0 (5.8) | |
| Percentage stage 3 sleep | 7.7 (2.3) | 8.0 (2.3) | |
| Percentage stage 4 sleep | 11.0 (4.3) | 10.2 (5.3) | |
| Percentage slow-wave sleep | 18.7 (4.0) | 18.2 (5.7) | |
| Percentage REM sleep | 17.5 (4.5) | 19.5 (5.5) | |
| Sleep efficiency | 94.0 (3.8) | 95.6 (2.6) | |

Total dark time was fixed based upon average school-night sleep onset and sleep offset times from actigraphic recordings of 1-week before each in-lab session. See text for detail.
Percentages are computed based on total sleep time [eg, (stage 1 time/total sleep time) x 100]
Sleep efficiency = (minutes total sleep time/minutes total dark time) x 100

**Table 4.—**Multiple sleep latency test results from boys (n=10) and girls (n=15) evaluated in 9th and 10th grades—mean (standard deviation)

| Time of sleep latency test | Sleep latency in 9th grade | Sleep latency in 10th grade | Significant effects |
|---|---|---|---|
| 0830 | 10.9 (6.5) | 5.1 (4.1) | Session (F=17.29, p<.001) |
| 1030 | 10.7 (5.9) | 9.2 (6.4) | |
| 1230 | 11.0 (5.5) | 9.8 (5.5) | |
| 1430 | 11.5 (6.1) | 11.0 (7.2) | |
| Daily mean | 11.4 (4.6) | 8.5 (4.5) | Session (F=10.45, p=.004) |
| | Number with REM Sleep in 9th Grade | Number with REM Sleep in 10th Grade | |
| 0830 | 4 | 9 | |
| 1030 | 3 | 3 | |
| 1230 | 0 | 3 | |
| 1430 | 0 | 2 | |
| Overall with REM | 4 | 12 | |

ed sleep patterns from the preceding 2 weeks. In the 9th grade, DLSMO was correlated with school-night sleep-onset time (r=.39, p=.013, n=29) and total sleep time (r=-.40, p=.012, n=39). In the 10th grade, DLSMO was correlated with these two variables (school-night sleep-onset time r=.51, p=.002, n=34; school-night total sleep time r=-.50, p=.003, n=34), as well as school-day sleep-offset time (r=.50, p=.003, n=34). In neither 9th nor 10th grades were weekend actigraphically recorded sleep patterns related to DLSMO.

## DISCUSSION

### Sleep Pattern

This study demonstrates the difficulty faced by adolescents in the US if they are to obtain adequate sleep, particularly when confronting an early school day. Previous research indicates that *optimal* alertness in adolescents requires over 9 hours of sleep nightly,[42,43] a sleep quantity achieved on school nights by only one 9th grade student in the present study. According to actigraph records, only 62% of the students in 9th grade and fewer than half the students (15 of 32) in 10th grade obtained an average of even 7 hours of sleep on school nights. Most participants in this study did not make up sleep by napping; however, sleep extension occurred on nonschool nights for 85% both in 9th and 10th grades. Even on the nonschool nights, however, students were sleeping considerably less than 9 hours. In general, these students were conspicuously sleep-deprived throughout the school week, a pattern not unique to this sample. In a recent survey of approximately 3,000 9th to 12th grade students,[44] for example, we found the average self-reported school-night total sleep time was 7 hours and 20 minutes.

The pattern of sleep deprivation in the present sample was more pronounced in boys than in girls, and the actigraphically recorded data showed that boys were less likely to extend sleep on nonschool nights. Although school nights showed no sex differences in actigraphically estimated sleep-onset and sleep-offset times, total amount of sleep was significantly less in boys than in girls. We have examined the actigraph records and note a considerably greater level of overall activity during the sleep period in boys than in girls.

**In-lab Sleep.—**The nocturnal sleep recordings made

**Table 5.—**Comparison between those with and without REM sleep on morning multiple sleep latency tests in 10th grade—mean (standard deviation)

| Variable | Without morning REM | With morning REM | Significant effects |
|---|---|---|---|
| Daily mean MSLT sleep latency | 9.4 (4.9) | 7.9 (4.1) | |
| Sleep latency 0830 SLT | 7.1 (4.9) | 3.4 (2.5) | t = 2.27, p = .033 |
| Sleep latency 1030 SLT | 9.3 (6.2) | 9.1 (6.6) | |
| Nocturnal stage 1 sleep latency | 7.8 (10.9) | 11.1 (9.0) | |
| Nocturnal total sleep time | 441 (38) | 439 (47) | |
| Nocturnal stage 1 sleep percentage | 4.0 (2.0) | 5.1 (1.7) | |
| Nocturnal SWS percentage | 20.9 (5.8) | 14.9 (3.1) | t = 3.12, p = .005 |
| Nocturnal REM sleep percentage | 18.8 (6.3) | 20.3 (4.0) | |
| Nocturnal REM sleep latency | 129 (34) | 111 (51) | |
| Actigraphy sleep onset time | 2224 (46) | 2246 (27) | |
| Actigraphy sleep offset time | 0558 (20) | 0615 (22) | |
| DLSMO time | 2036 (65.2) | 2146 (65.7) | t = 2.69, p = .013 |

during these students' usual school-night sleeping hours showed several significant changes following the transition to the earlier school starting times. Although the recording (dark) time decreased significantly—reflecting shorter sleeping hours in 10th grade—total sleep time was not significantly affected. The in-lab sleep changes seen from 9th to 10th grade likely resulted from adjustment to the laboratory, rather than from any change in sleep structure due to the school schedule change. Thus, the somewhat lower sleep efficiency, longer REM sleep latency, and greater stage 1 sleep percentage in the 9th grade in-lab sleep are consistent with a "first-night effect"[45] that was absent in 10th grade. An alternate explanation is that the between-session in-lab sleep differences resulted from greater sleepiness in the 10th grade. The actigraphic assessments, by contrast, clearly showed a reduction in school-night sleeping time across the transition.

## Circadian Phase Adjustment

Exacerbation of sleep loss in 10th grade occurred in part because subjects' schedules did not adjust adequately to optimize sleep in response to the earlier school day. The students were confronted with a 65-minute-earlier non-negotiable start for their school days, and they woke earlier to accommodate this external demand; however, neither the time they fell asleep nor DLSMO manifested a phase advance. In fact, the onset time of melatonin secretion delayed significantly in 10th grade. This response would not be predicted by circadian physiology. If the early rising time extended daylight hours through early-morning light exposure, a phase advance should have occurred. Why, then, did these students phase delay? Does the pubertally associated phase delay[22,24] continue in these older, generally late-pubertal adolescents? Are the psychosocial factors that foster later sleep times[14] gaining strength? Is the adolescent circadian timing system insensitive to morning light's phase advancing properties and more sensitive to evening light's phase delay? These questions point to issues requiring further study. On the other hand, we note that circadian phase in adolescents is not immutable: in other studies, we have demonstrated that controlled light-dark exposure can shift an adolescent's circadian phase.[24] The significant correlations between sleep patterns and DLSMO also indicate that the two phenomena are linked.

The sex difference in melatonin onset phase is difficult to explain, given the modest differences in sleep-onset and sleep-offset times in the boys and girls. Lacking adequate measures of light exposure in these adolescents, we make certain assumptions about light exposure based upon sleep patterns, since sleep provides a lengthy "dark" signal. We found no major sex-linked schedule differences in this sample, and a simple comparison of DLSMO time in youngsters who did and did not report after-school activities (eg, sports, band practice) revealed no significant differences based on schedule. Our data do not support the hypothesis that DLSMO sex difference results from maturational differences, since Tanner stages were similar in girls and boys. Furthermore, our data from adolescents studied under a fixed light-dark schedule show no sex differences in melatonin secretory phase (unpublished data).

## Sleepiness and REM Sleep on MSLT

The sleep deprivation experienced by adolescents taking part in this study was clearly reflected in their MSLT scores, particularly in 10th grade and especially in the morning. As a group, the students bordered on pathologically sleepy[38] in 10th grade, with a mean MSLT of 8.5 minutes. Ten of the 10th graders (38%) had an MSLT average under 6 minutes; the lowest value was 1.8 minutes. Sleep latency in 10th grade was lowest in the first morning test—which for these students coincided with the time of the second class period on school days—and was highest on the last afternoon test, coincident with the end of the school day.

The most notable finding, however, was the extent to which these young people manifested REM sleep on the MSLT. The rate of REM sleep occurrence in 9th grade (4 of 32 subjects = 12.5%) was unexpectedly high for a group of adolescents without narcoleptic symptoms or immediate family history. In 10th grade, however, the rate was exceptional: 12 of 25 (48%) had at least one REM episode, and 4 (16%) had two REM sleep episodes. For comparison, we examined data from MSLTs in normal adolescents sleeping

8 or more hours per night in our previous studies,[43] in which fewer than 7% had even a single REM episode on MSLT.

The adult sleep literature has recently marked the occurrence of a high frequency of REM sleep episodes on MSLT in healthy control subjects. For example, one report[46] noted two MSLT REM sleep onsets in 17% (24 of 139) young adults, and 23% had one or more REM sleep episodes (32 of 139). In the Bishop et al study, the distribution of MSLT REM sleep episodes did not differ across the day, with testing schedules beginning at 0930 or 1000. Explanations for this high rate of daytime REM sleep episodes have included sleep deprivation, sleep fragmentation, and incipient narcolepsy, though none has been supported. The etiology of REM sleep during MSLTs in noncomplaining normals is of growing concern,[47,48] and the data from these teenagers may help explain certain instances.

## What Factors Relate to REM Sleep on MSLT?

Most of the REM episodes in our adolescent subjects occurred during the morning MSLT sessions; only one had REM solely on an afternoon test. This pattern of occurrence suggested a possible explanation for the high REM incidence related to the known circadian regulation of REM sleep propensity. Czeisler[20] and Zulley[21] independently showed that peak REM sleep propensity in human adults is linked to the trough of the circadian rhythm of core body temperature. The temperature trough of young adult humans under entrained conditions typically occurs approximately 1.5 to 2 hours before usual rising time.[49,50] Studies of brief sleep episodes also confirm that the likelihood of REM sleep occurring during a brief sleep episode is increased at the trough of the body temperature cycle.[51]

The marker of the circadian timing system used in the current study was the onset of melatonin secretion, which was significantly later in the group of youngsters who had REM sleep episodes on morning MSLTs, indicating a phase delay of about 1.16 hours. In young adults and adolescents under entrained controlled lighting conditions, the interval between the onset of melatonin secretion and the minimum of core body temperature is 6.5 to 7 hours.[50,52] If adolescents in naturalistic conditions have the same phase relationship, then school-day wake-up time for youngsters in the group with morning REM sleep on MSLT occurred at a circadian phase promoting REM sleep even several hours after rising time. Thus, those students with the phase delay experienced pressure from the circadian timing system to have REM sleep on morning MSLTs.

We also emphasize that these young people in general carried a significant chronic sleep debt. Chronic insufficient sleep may also play a role in the appearance of REM sleep on MSLT, and this phenomenon may be modulated by developmental or maturational processes. For example, the subjects in the study of Bishop et al[46] who had REM during MSLT were significantly younger (27.9±9.5 vs 34.6±12.2 years) than the group without REM. Thus, even though both groups in that study reported comparable sleeping times on weekdays and weekends, the younger group may in general carry a higher biological sleep need, and thus accumulate a greater sleep debt with comparable sleeping times. Sleep extension alone can decrease the occurrence of REM sleep on MSLT.[43]

## What About Slow-wave Sleep?

The group of adolescents in our sample with REM episodes on MSLT had reduced SWS compared to those without REM sleep on MSLT. We consider it unlikely that reduced SWS was causally related to the occurrence of REM sleep on MSLT; rather, we attribute the SWS difference to the circadian phase disruption, specifically to the smaller phase angle between DLSMO and sleep onset in the group with REM episodes (see Fig. 3). Considerable evidence links melatonin to a decrease in core body temperature,[53] and the slope of the nocturnal core body temperature fall has been linked to the potentiation of slow-wave sleep.[54] Furthermore, a small but significant circadian modulation of slow waves[12] has been reported in adults evaluated in a forced-desynchrony paradigm. This circadian effect may be related to the link between melatonin secretion and temperature decline. Two adolescents examined during forced desynchrony[55] displayed a prominent circadian component to the occurrence of SWS (unpublished data). Thus, the circadian relationships may be a stronger feature of slow-wave sleep control in adolescents, who in general have greater slow-wave sleep than adults.[56] If the altered phase relationship between melatonin onset and sleep onset indicates that these students go to bed at an inappropriate circadian phase (to rise for an early school start time), then slow-wave sleep may be affected. Thus, the early school start time may have an exaggerated negative impact: going to bed at an inappropriate phase adversely affects nocturnal sleep, and waking up at an inappropriate circadian phase adversely affects daytime alertness.

## Implications for Clinical Practice

Many teenagers do not get adequate sleep and are sleepy. Furthermore, the typical clinical practice of studying individuals on their "usual" schedules may introduce interpretational difficulties. As this study shows, schedules combining insufficient sleep and early rise times are associated, in a high percentage of youngsters, with chronically altered sleeping patterns, realignment or misalignment of circadian phase relationships affecting sleep and wakefulness, and consequent alterations of "normal" patterns on the MSLT. Thus, one's "usual" schedule is not necessarily the appropriate or optimal schedule for clinical assessments. Young adults and other groups may encounter similar phase mismatches when their intrinsic biological propensities are confronted daily by conflicting external demands or lifestyle choices. Clinicians need to exercise caution in interpreting REM sleep on MSLT in adolescents studied on their "usual" schedule when that schedule involves early rising enforced by alarm clocks, parents, or both. Collection of serial saliva samples in dim light during the hours before habitual bedtime to obtain an estimate of circadian phase with DLSMO may also be useful in evaluating such patients.

## CONCLUSION

In summary, along with strong psychosocial influences, changes in bioregulatory systems controlling sleep may limit teenagers' capacities to make adequate adjustments to an early school schedule. Imposition of an early school start time may require unrealistic—if not unattainable—bedtimes to provide adequate time for sleeping. Our study clearly showed that early school start times for adolescents were associated with significant sleep deprivation. The consequences of insufficient sleep in adolescents are substantial. Excessive sleepiness of the degree documented here can be associated with performance decrements, memory lapses, and mood changes,[57-59] as well as behavior problems. In susceptible young people, this pattern may lead to academic, behavioral, and psychological problems,[44] as well as increased risk for accidents and injuries, particularly for teenaged automobile drivers.[60]

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## REFERENCES

**1.** Andrade MMM, Benedito-Silva AA, Menna-Barreto L. Correlations between Morningness-Eveningness character, sleep habits and temperature rhythm in adolescents. Braz J Med Biol Res 1992;25:835-9.

**2.** Carskadon MA. Patterns of sleep and sleepiness in adolescents. Pediatrician 1990; 17:5-12.

**3.** Bearpark HM, Michie PT. Prevalence of sleep/wake disturbances in Sidney adolescents. Sleep Res 1987;16:304.

**4.** Epstein R, Chillag N, Lavie P. Sleep habits of children and adolescents in Israel: The influence of starting time of school. Sleep Res 1995;24a:432.

**5.** Ishihara K, Honma Y, Miyake S. Investigation of the children's version of the morningness-eveningness questionnaire with primary and junior high school pupils in Japan. Percept Mot Skills 1990;71:1353-4.

**6.** Strauch I, Meier B. Sleep need in adolescents: A longitudinal approach. Sleep1988; 11:378-86.

**7.** Saarenpaa-heikkila OA, Rintahaka PJ, Laippala PJ, Koivikko MJ. Sleep habits and disorders in Finnish schoolchildren. J Sleep Res 1995;4:173-82.

**8.** Steinberg L. Beyond the Classroom: Why School Reform Has Failed and What Parents Need To Do. New York, Simon & Shuster, 1996.

**9.** Borbély AA. A two process model of sleep regulation. Hum Neurobiol 1982; 1: 195-204.

**10.** Borbély AA, Achermann P, Trachsel L, Tobler I. Sleep initiation and initial sleep intensity: interactions of homeostatic and circadian mechanisms. J Biol Rhythm 1989;4:149-60.

**11.** Dijk DJ, Czeisler CA. Paradoxical timing of the circadian rhythm of sleep propensity serves to consolidate sleep and wakefulness in humans. Neurosci Lett 1994; 166:63-8.

**12.** Dijk DJ, Czeisler CA. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. J Neurosci 1995; 15:3526-38.

**13.** Edgar DM, Dement WC, Fuller CA. Effect of SCN Lesions on Sleep in squirrel monkeys—evidence for opponent processes in sleep-wake regulation. J Neurosci 1993; 13:1065-79.

**14.** Carskadon MA. Factors influencing sleep patterns of adolescents. In: Carskadon MA ed. Adolescent Sleep Patterns: Biological, Social, and Psychological Influences. New York: Cambridge University Press, in press.

**15.** Carskadon MA, Acebo C, Seifer R. Adolescent sleep on long nights with and without prior sleep deprivation. Sleep 1998;21Supp:241.

**16.** Czeisler CA, Weitzman ED, Moore-Ede MC, Zimmerman JC, Kronauer RS. Human sleep: its duration and organization depend on its circadian phase. Science 1980;210:1264-7.

**17.** Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation of sleep tendency in elderly and young adult subjects. Sleep 1982;5Supp:82-94.

**18.** Johnson MP, Duffy JF, Dijk DJ, Ronda JM, Dyal CM, Czeisler CA. Short-term memory, alertness and performance: a reappraisal of their relationship to body temperature. J Sleep Res 1992;1:24-9.

**19.** Åkerstedt T, Folkard S. Validation of the s and c components of the three-process model of alertness regulation. Sleep 1995; 18:1-6.

**20.** Czeisler CA, Zimmerman JC, Ronda JM, Moore-Ede MC, Weitzman ED. Timing of REM sleep is coupled to the circadian rhythm of body temperature in man. Sleep 1980;2:329-46.

**21.** Zulley J. Distribution of REM sleep in entrained 24 hour and free-running sleep-wake cycles. Sleep 1980;2:377-89.

**22.** Carskadon MA, Vieira C, Acebo C. Association between puberty and delayed phase preference. Sleep 1993;16:258-62.

**23.** Tanner JM. Growth at Adolescence. Oxford: Blackwell, 1962.

**24.** Carskadon MA, Acebo C, Richardson GS, Tate BA, Seifer R. An approach to studying circadian rhythms of adolescent humans. J Biol Rhythm 1997;12:278-89.

**25.** Czeisler CA. The effect of light on the human circadian pacemaker. In: Chadwick DJ, Ackrill K ed. Circadian Clocks and Their Adjustment. Baffins Lane, Chichester, England P019 7UD: John Wiley & Sons Ltd, 1995: 254-90.

**26.** DeCoursey PJ, Menon SA. Circadian photo-entrainment in a nocturnal rodent quantitative measurement of light-sampling activity. Anim Behav 1991;41:781-5.

**27.** Smith CS, Reilly D, Midkiff K. Evaluation of three circadian rhythm questionnaires with suggestions for an improved measure of morningness. J Appl Psychol 1989;74:728-38.

**28.** Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. Sleep 1995;18:288-302.

**29.** Acebo C, Sadeh A, Seifer R, et al. Estimating sleep patterns with activity monitoring in children and adolescents: How many nights are necessary for reliable measures? Sleep, in press.

**30.** Sadeh A, Sharkey KM, Carskadon MA. Activity-based sleep-wake identification: an empirical test of methodological issues. Sleep 1994; 17:201-7.

**31.** Cassone VM, Natesan AK. Time and time again: the phylogeny of melatonin as a transducer of biological time. J Biol Rhythm 1997; 12:489-97.

**32.** Lewy AJ, Wehr TA, Goodwin FK, Newsome DA, Markey SP. Light suppresses melatonin secretion in humans. Science 1980;310:1267-9.

**33.** Lewy AJ, Sack RL. The dim light melatonin onset as a marker for circadian phase position. Chronobiol Int 1989;6:93-102.

**34.** Deacon S, Arendt J. Posture influences melatonin concentrations in plasma and saliva in humans. Neurosci Lett 1994; 167:191-4.

**35.** Lewy AJ, Ahmed S, Jackson JML, Sack RL. Melatonin shifts human circadian rhythms according to a phase- response curve. Chronobiol Int 1992;9:380-92.

**36.** Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects. Brain Information Service/Brain Research Institute, UCLA: Los Angeles, 1968.

**37.** Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook P, Keenan S. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. Sleep 1986;9:519-24.

**38.** Mitler MM, van den Hoed J, Carskadon MA, et al. REM sleep episodes during the multiple sleep latency test in narcoleptic patients. Electroencephalogr Clin Neurophysiol 1979;46:479-81.

**39.** ASDA. International Classification of Sleep Disorders: Diagnostic and Coding Manual. Diagnostic Classification Steering Committee, Thorpy MJ, Chairman ed. Rochester, MN, American Sleep Disorders Association, 1990.

**40.** Richardson GS, Carskadon MA, Flagg W, van den Hoed J, Dement WC, Mitler MM. Excessive daytime sleepiness in man: multiple sleep latency measurement in narcoleptic and control subjects. Electroencephalogr Clin Neurophysiol 1978;45:621-7.

**41.** Carskadon MA, Dement WC. Sleepiness and sleep state on a 90-minute schedule. Psychophysiology 1977;14:127-33.

**42.** Carskadon MA, Harvey K, Duke P, Anders TF, Litt IF, Dement WC. Pubertal changes in daytime sleepiness. Sleep 1980;2:453-60.

**43.** Carskadon MA. The second decade. In: Guilleminault C ed. Sleeping and Waking Disorders: Indications and Techniques. Menlo Park, CA: Addison Wesley, 1982:99-125.

**44.** Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. Child Development, 1998;69:875-87.

**45.** Agnew HW, Webb WB, Williams RL. The first-night effect: An EEG study. Psychophysiology 1966;2:263-6.

**46.** Bishop C, Rosenthal L, Helmus T, Roehrs T, Roth T. The frequency of multiple sleep onset REM periods among subjects with no excessive daytime sleepiness. Sleep 1996; 19:727-30.

**47.** Rosenthal L, Bishop C, Helmus T, Roehrs T, Roth T. Frequency of multiple sleep onset REM periods in "control" subjects - response. Sleep 1997;20:814.

**48.** Rye DB, Bliwise DL. Frequency of multiple sleep onset REM periods in "control" subjects. Sleep 1997;20:813.

**49.** Czeisler CA, Dumont M, Duffy JF, et al. Association of sleep-wake habits in older people with changes in output of circadian pacemaker. Lancet 1992;340:933-6.

**50.** VanCauter E, Sturis J, Byrne MM, et al. Demonstration of rapid light-induced advances and delays of the human circadian clock using hormonal phase markers. Am J Physiol 1994;266:E953-E63.

**51.** Carskadon MA, Dement WC. Distribution of REM sleep on a 90-minute sleep-wake schedule. Sleep 1980;2:309-17.

**52.** Labyak SE, Acebo C, Carskadon MA. Circadian phase markers in entrained adolescents. Paper presented at the annual meeting of the Society for Research on Biological Rhythms, 1998, Amelia Island Plantations.

**53.** Cagnacci A, Krauchi K, Wirz-Justice A, Volpe A. Homeostatic versus circadian effects of melatonin on core body temperature in humans. J Biol Rhythm 1997; 12:509-17.

**54.** Campbell SS, Broughton RJ. Rapid decline in body temperature before sleep: fluffing the physiological pillow? Chronobiol Int 1994; 11: 126-31.

**55.** Carskadon MA, Acebo C, Tate BA, Richardson GS. MSLT during forced desynchrony in two adolescents. Sleep Res 1997;26:706.

**56.** Williams RL, Karacan I, Hursch CJ. Electroencephalography (EEG) of Human Sleep: Clinical Applications. New York: John Wiley and Sons, 1974.

**57.** Dinges DF, Kribbs NB. Performing while sleepy: Effects of experimentally-induced sleepiness. In: Monk TH ed., Sleep, Sleepiness and Performance. Chichester, United Kingdom: John Wiley and Sons, Ltd., 1991:97-128.

**58.** Carskadon MA, Rosekind MR, Galli J, Sohn J, Herman KB, Davis SS. Adolescent sleepiness during sleep restriction in the natural environment. Sleep Res 1989;18:115.

**59.** Leotta C, Carskadon MA, Acebo C, Seifer R, Quinn B. Effects of acute sleep restriction on affective response in adolescents: Preliminary results. Sleep Res 1997;26:201.

**60.** Pack AI, Pack AM, Rodgman D, Cucchiara A, Dinges DF, Schwab CW. Characteristics of crashes attributed to the driver having fallen asleep. Accident Anal Prev 1995;27:769-75.