When Worlds Collide

Adolescent Need for Sleep Versus Societal Demands

By Mary A. Carskadon

Given that the primary focus of education is to maximize human potential, then a new task before us is to ensure that the conditions in which learning takes place address the very biology of our learners, Ms. Carskadon points out.

OUR UNDERSTANDING of the development of sleep patterns in adolescents has advanced considerably in the last 20 years. Along the way, theoretical models of the processes underlying the biological regulation of sleep have improved, and certain assumptions and dogmas have been examined and found wanting. Although the full characterization of teen sleep regulation remains to be accomplished, our current understanding poses a number of challenges for the education system.

The early 1970s found us with a growing awareness that sleep patterns change fundamentally at the transition to adolescence — a phenomenon that is widely acknowledged today. Survey studies clearly showed then and continue to show that the reported timing of sleep begins to shift in early adolescence, with bedtime and rising time both occurring at later hours. This delayed sleep pattern is particularly evident on nonschool nights and days, though the evening delay is obvious on school nights as well. Associated with the delay of sleep is a decline in the amount of sleep obtained and an increase in the discrepancy between school nights and weekend nights. Although the nonschool-night "oversleeping" was acknowledged as recovery from insufficient sleep during the school week, we initially assumed that the amount of sleep required declines with age. This was axiomatic: the older you are, the less sleep you need.

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Assessing the Need for Sleep In the Second Decade

A longitudinal study begun in 1976 at the Stanford University summer sleep camp attempted to examine this axiom.[1] Boys and girls enrolled in this research project at ages 10, 11, or 12 and came to the lab for a 72-hour assessment each year for five or six years. They were asked to keep a fixed schedule, sleeping 10 hours a night for the week before the study, and their sleep was recorded on three consecutive nights from 10 p.m. to 8 a.m. Our hypothesis was that the reduced need for sleep in older children would manifest itself through less sleep within this 10-hour nocturnal window. This hypothesis was *not* confirmed. In fact, regardless of age or developmental stage, the children all slept about 9¼ of the 10 hours. Furthermore, delays in sleep resulted in a reduced likelihood of spontaneous waking before 8 a.m. for all but the youngest participants. One conclusion, therefore, was that the need for sleep does not change across adolescent development.

This study also showed an interesting pattern with respect to waking alertness, which was assessed using a technique called the Multiple Sleep Latency Test (MSLT). The MSLT measures the speed of falling asleep across repeated 20-minute trials in standard conditions. Thus a child who stays awake 20 minutes can be considered alert, faster sleep onsets are a sign of reduced alertness, and a child who falls asleep in five minutes or less is excessively sleepy.[2] The longitudinal study demonstrated that — even though the total amount of sleep was unchanged — alertness declined in association with pubertal development.[3] Figure 1 illustrates the MSLT patterns: under these experimental conditions, more mature adolescents showed signs of reduced alertness even though they slept an equivalent amount at night. One interpretation of these data is that older teenagers may need *more* sleep than when they were younger. On the other hand, the pattern of sleep tendency showing a midafternoon dip may

reflect maturation of a regulated behavioral pattern favoring an afternoon nap or siesta.

Behavioral Factors

The principle that adolescents sleep later and less because of a panoply of psychosocial factors was also axiomatic during the 1970s and the 1980s. The evidence for this included a change in parental involvement in youngsters' sleep schedules as the children age. Thus,until about ages 11 or 12,more children than not reported that they woke spontaneously in the morning and that parents set their bedtimes. Fewer children in their early teens reported that parents still set their bedtimes, and most said that they required an alarm clock or a parent to assist them in waking up.[4]

Other behavioral factors contributing to the changing sleep patterns with age include increased social opportunities and growing academic demands. Another major contributor to changing adolescent sleep patterns is employment. One survey of youngsters in New England in the late 1980s found that two-thirds of high school students had jobs and that nearly 30% worked 20 or more hours in a typical school week.[5] Those high school students who worked 20 hours or more reported later bedtimes, shorter sleep times, more frequent episodes of falling asleep in school, and more frequent oversleeping and arriving late at school.

In addition to changing parental involvement, increasing school and social obligations, and greater participation in the work force, there are a myriad of other phenomena that have not been well explored. Access in the bedroom to computers,televisions, telephones, and so forth probably contributes to the delay of and reduction in sleep.

Another factor that has a major influence on adolescent sleep is the school schedule. The starting time of school puts limits on the time available for sleep. This is a nonnegotiable limit established largely without concern for sleep. Most school districts set the earliest starting time for older adolescents and the latest starting time for younger children. District officials commonly acknowledge that the school schedule is determined by the availability of school buses, along with such other factors as time of local sunrise, sports teams' schedules, and so forth. As described in other articles in this special section, concerns about the impact of school schedules on sleep patterns (as well as concerns about after-school teen delinquency) have sparked a reexamination in a number of districts. Our studies indicate that such a reexamination is merited by the difficulties many teenagers experience.

Biological Factors

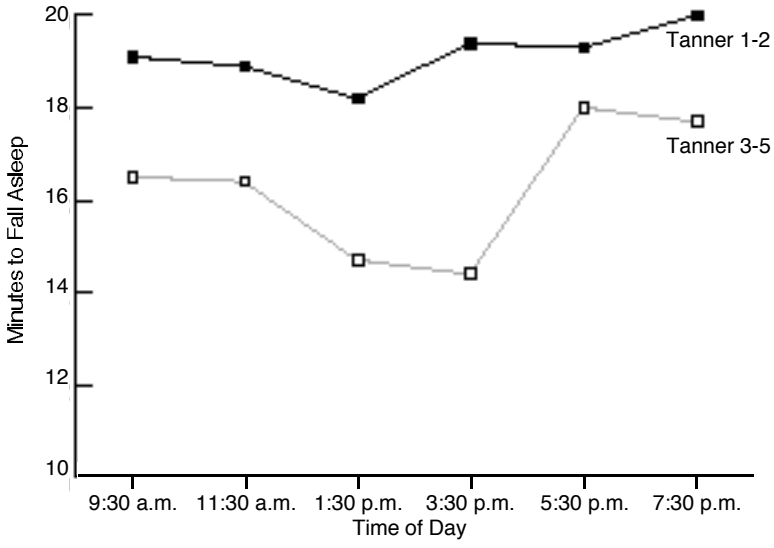
As findings of the tendency for adolescent sleep patterns to be delayed were reported not only in North America but also in South America, Asia, Australia, and Europe, a sense arose that intrinsic developmental changes may also play a role in this phenomenon.[6] At the same time, conceptual models of the underlying internal mechanisms that control the length and timing of sleep began to take shape.

Current models posit three factors that control human sleep patterns. One of these factors is behavior and includes external factors such as those discussed above. The intrinsic factors have been called "sleep/wake homeostasis" and the "circadian timing system," or "process S" and "process C" in one model.[7] Sleep/wake homeostasis more simply stated is that sleep favors wake and wake favors sleep. All other things being equal, therefore, the longer one is awake, the greater the pressure for sleep to occur. Conversely, the closer one is to having slept, the less pressure there is to sleep. This process accounts for the increased need for sleep after staying awake all night and the difficulty of staying awake in general when faced with a chronic pattern of insufficient sleep. Process S can be examined using measures of sleep tendency, such as the MSLT, or measures of EEG (electroencephalogram) slow wave activity (SWA) during sleep. Sleep tendency and SWA increase with insufficient sleep. Both factors also show changes across adolescent development that may be related to the timing of sleep.

Under conditions of optimal sleep ,such as those described in the longitudinal study of sleep ,slow wave sleep declines by 40% from early to late adolescence. This decline may indicate a reduced pressure for sleep with greater maturation. One interpretation of this finding is that the reduced pressure for sleep makes staying up late an eas-

FIGURE 1.
Developmental Change in Daytime Alertness Under Conditions of 'Optimal' Sleep

The upper line, labeled Tanner 1-2, shows that pre- and early-pubescent boys and girls with a 10-hour sleep opportunity are not at all sleepy. The lower line, labeled Tanner 3-5, shows that more physically mature youngsters are sleepier, even though they have the same sleep opportunity.



ier task for older adolescents. Others have interpreted this finding as marking a structural change in the brain (thinning of cortical synaptic density) that is unrelated to sleep/wake homeostasis. The change in sleep tendency — that is, the appearance of a midday trough at midpuberty (Figure 1) — may indicate a reorganization of the sleep/wake homeostatic mechanism to favor daytime napping and an extended late-day waking period, again favoring a later bedtime. These hypotheses are speculative and require additional study.

Much of the contemporary excitement about adolescent sleep comes from studies of the circadian timing mechanism, which independently and interactively exerts influences on sleep through processes that favor or inhibit sleep according to the dictates of an internal biological "clock." Several features of the human circadian timing system and its interactions with sleep and wakefulness are relevant here.

- Circadian rhythms are biological oscillations with periods of about 24 hours.
- Circadian rhythms are synchronized to the 24-hour day chiefly by light signals.
- The chief circadian oscillator in mammals is located deep within the brain in the suprachiasmatic nuclei (SCN) of the hypothalamus.
- Circadian rhythms can be assessed by measuring the timing of biological events.
- Circadian rhythms are thought to control the timing of "sleep gates" and "forbidden zones" for sleep.
- Circadian rhythms control the timing of REM (rapid eye movement) sleep within the sleep period.

A first attempt to examine whether the circadian timing system undergoes developmental changes during adolescent maturation involved a survey of sixth-grade girls. In this survey, one series of questions allowed us to estimate physical development and another series gave a measure of circadian phase preference. Phase preference refers to an individual's tendency to favor activities in the morning or evening, i.e., morningness/eveningness. In these 275 sixth-grade girls, the puberty score and circadian phase preference score showed a significant relationship: less mature girls favored earlier hours, and more mature girls favored later hours.[8] These data were the first to implicate a biological process in the later timing of adolescent sleep; however, the measures were indirect and self-reported.

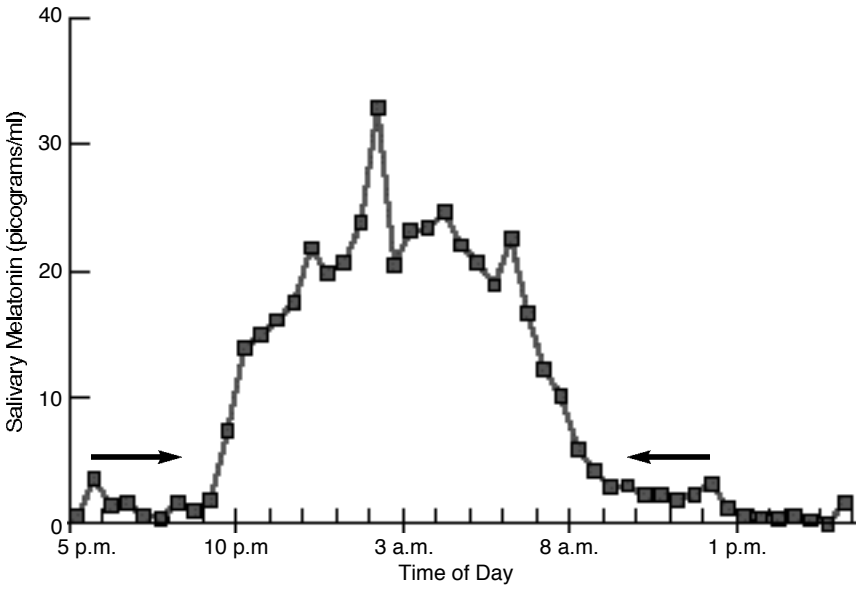
Our subsequent studies have attempted to confirm a pubertally mediated phase delay in adolescents using more precise measures. For example, one of the best ways to identify time in the intrinsic biological clock in humans is to examine melatonin secretion.[9] Melatonin is a hormone that is produced by the pineal gland and regulated by the circadian timing system. Melatonin secretion occurs during nocturnal hours in both day-active species, like humans, and night-active species. Melatonin can be measured from saliva samples collected in dim lighting conditions. The normal melatonin curve in dim light provides a very robust signal,as shown in Figure 2.

Because the intrinsic circadian timing system is synchronized to the 24-hour cycle principally by light (even in humans), careful evaluation of the relationship between the endogenous cycle and the developmental phase must control for or eliminate behavioral differences, such as later bedtimes,that affect the timing of light the internal clock receives. Hence, we developed an experimental protocol to incorporate such controls by placing adolescents on identical light/dark schedules. Under strictly controlled conditions,we found a significant correlation between pubertal development (which can be evaluated by trained physicians or nurses using markers of secondary sexual characteristics[10]) and circadian timing: more mature adolescents had a later timing of the termination of melatonin secretion.[11]

Current and planned investigations need to examine mechanisms for this pattern and to determine more clearly how the circadian timing system is linked to the sleep/wake system during development. One approach to this inquiry uses an elegant experimental design in which youngsters are placed on a sleep/wake schedule that is outside the range of values capable of synchronizing the internal clock. In this experiment, bedtime and rising time are scheduled four hours later from one day to the next,effecting a 28-hour day. Because the internal clock cannot accommodate to this demand, it runs free at its own intrinsic day length. This measure itself can provide crucial evidence about adolescent timing in the 24-hour world. According to circadian rhythms theory, adolescents with long in-

FIGURE 2.
Pattern of Melatonin Secretion (Measured in Saliva Samples)

Melatonin secretion has a sharp onset and relatively sharp offset. The arrows indicate times at which onset and offset of melatonin secretion occur in adolescents who keep a very strict schedule, going to bed at 10 p.m and waking at 8 a.m. The secretion of melatonin is controlled by the circadian timing system and is an excellent measure for determining internal clock time.



ternal day lengths will synchronize to the external day with a later alignment than adolescents with shorter internal day lengths.

In such a study, we can also evaluate sleeping and waking measures occurring at every phase of the circadian cycle and at every time since the participant fell asleep or woke up. In other words, we are able to examine the independent influences of the circadian timing system and the sleep/wake homeostatic process and their interactions. When we evaluated the sleep tendency of adolescents using the MSLT measured in the 28-hour day, we found that the circadian pressure to sleep — regardless of how long an individual had been awake — was greatest right as the melatonin secretion was about to turn off, about an hour before "normal" waking up time; the circadian pressure to stay awake was greatest right before melatonin secretion was about to begin, about an hour before "normal" bedtime.[12] Taken alone, this is a curious finding, because one ordinarily experiences sleep pressure to be greatest at bedtime and least on waking up.

On the other hand, when we examined sleep tendency as a function of how long the participants had been awake — regardless of circadian time — the lowest sleep pressure occurred close to rising time and the greatest sleep pressure at the end of the waking period. When examined in combination, these two processes provide an explanation for humans' ability to experience an extended wakeful period. When viewed as opposing processes,[13] sleep/wake homeostasis provides for alertness early in the day, when the circadian timing mechanism favors sleep,and the circadian timing system props up alertness late in the day, when the homeostatic process favors sleep. In the context of adolescent development, if there is a situation in which sleep pressure builds more slowly or circadian timing is delayed or both, adolescents will encounter a "forbidden zone" for sleep later in the day.

Other facets of the circadian timing system that may influence the later timing of sleep behavior in adolescents include a potentially greater sensitivity to low light levels in the evening. Such a sensitivity could affect the circadian timing system by pushing the forbidden zone for sleep to a later time. The mechanism for such an effect is described in the phase response curve, in which light to the internal clock late in the evening pushes the clock to a later time and light to the internal clock early in the morning pushes the clock to an earlier time. The characteristics of this circadian phase resetting mechanism may also change during adolescent maturation.

One other important finding from our studies is that the circadian timing system can be reset if light exposure is carefully controlled. In many of our studies, we require adolescents to keep a specific sleep schedule (for example, 10 p.m. to 8 a.m.) and to wear eyeshades to exclude light during these hours. In fact, we pay adolescents to keep this schedule! When we measure melatonin secretion before the students go on the new schedule (when they are still on their self-selected routine) and again after 10 or 11 nights on the new schedule, we find that the melatonin secretion has moved significantly toward a common time: those who were early melatonin secretors move to a later time, and those who were late secretors move earlier.[14] Thus we know that the system is not immutable; with time, effort, *and* money, we can get adolescents to realign their rhythms!

Let us summarize what we now know about the developmental trends in adolescent sleep behavior and adolescents' sleep/wake and circadian systems.

- As they mature, adolescents tend to go to bed later and to wake up later (given the opportunity).
- Adolescents also tend to sleep less as they mature.
- The difference between the amount and timing of sleep on weekend nights versus school nights grows during adolescence.
- These trends are apparent in adolescents both in North America and in industrialized countries on other continents.
- Sleep requirements do not decline during adolescent development.
- Daytime sleep tendency is augmented during puberty.
- The timing of events controlled by the circadian timing system is delayed during puberty.

We propose that the delay of sleep during adolescent development is favored by behavioral and intrinsic processes and that the reduction of sleep experienced by adolescents is largely driven by a collision between the intrinsic processes and the expectations and demands of the adult world. The study described in the following section illustrates this point.

School Transition Project

Our school transition project took a look at what happened to sleep and circadian rhythms in a group of youngsters for whom the transition from junior high school to senior high school required a change in the starting time for school from 8:25 a.m. to 7:20 a.m. Twenty-five youngsters completed our study at two time points, in the spring of the ninth grade and in the autumn of the 10th grade.[15] These boys and girls were all well beyond the beginning changes of puberty; some were physically mature. They were enrolled in the study with instructions simply to keep their usual schedules, to wear small activity monitors on their wrists, and to keep diaries of their activities and sleep schedule for two consecutive weeks. At the end of the two weeks,participants came to the sleep laboratory for assessment of the onset phase of melatonin secretion, overnight sleep study, and daytime testing with the MSLT. The laboratory sleep schedule was fixed to each student's average school-night schedule based on the data from the wrist monitor (actigraph).

As predicted, the actigraph data showed that students woke up earlier when confronted with the 7:20 a.m. start time, although rising time was on average only about 25 minutes earlier (6:26 a.m. to 6:01 a.m.), not the 65 minutes represented by the school schedule change. Sleep onset times did not change, averaging about 10:40 p.m. in both grades. The average amount of sleep on school nights fell from seven hours and nine minutes to six hours and 50 minutes, a statistically significant amount and probably a meaningful amount when considered as producing an ever cumulating sleep deficit.

The amount of sleep these students obtained in ninth grade was below the amount we feel is required for optimal alertness, and the further decline in 10th grade had added impact. One way to examine the impact is to look at the MSLT data from tests that occurred at 9:30 a.m., 10:30 a.m., 12:30 p.m., and 2:30 p.m. If we look at comparable MSLT data from Figure 1, we find an average score of 18.9 minutes for the early pubertal children and 15.5 minutes for the mid- to late pubertal adolescents sleeping on the optimizing 10-hour schedule. The ninth-grade students in this more naturalistic study, by contrast, had an average MSLT speed of falling asleep of 11.4 minutes, and in 10th grade the sleep score was 8.5 minutes. In clinical terms, these students were in a borderline zone

for daytime sleepiness, well below the alert range and below the "normal" range, yet not in the "pathological" range.

A closer look at the MSLT test results shows that the students in 10th grade were in the pathological range when tested at 8:30 a.m. (MSLT score = 5.1 minutes). Furthermore, nearly 50% of these 10th-graders showed a reversed sleep pattern on the morning MSLT tests that is similar to the pattern seen in patients with the sleep disorder called narcolepsy — that is, REM sleep occurs before non-REM sleep. The 12 students who showed this "narcoleptic"pattern fell asleep in an average of 3.4 minutes when tested at 8:30 a.m. These students did not have narcolepsy; what they did have was a significant mismatch between their circadian rhythms and the necessity to get up and go to school. The evidence for this mismatch was a later time for the onset of melatonin secretion compared with those who did not have the "narcoleptic"pattern: 9:46 p.m. versus 8:36 p.m. This marker of the circadian timing system indicates that 1) the students' natural time to fall asleep is about 11 p.m. or later (on average) and 2) the abnormally short time to sleep onset on the 8:30 a.m. MSLT and the abnormal occurrence of REM sleep took place because the students were tested at the very nadir of their circadian day. In other words, at 8:30 a.m., these students' brains were far better suited to be asleep than awake!

Why were these 12 students so different from the others? We were unable to identify a specific cause. None of the 25 students made an optimal adjustment to the new schedule; none was sleeping even as much as 8¼ hours on school nights, a value we suggest elsewhere might be adequate if not optimal for high school students.[16] A few students maintained a "normal" level of alertness, others were borderline, and still others were in the pathological range. The 12 students whose circadian timing systems moved to a much later timing in 10th grade, however, showed signs associated with marked impairment, particularly in the morning hours.

Consequences, Concerns, and Countermeasures

Among the known consequences of insufficient sleep are memory lapses, attentional deficits,depressed mood, and slowed reaction time. Sleep deprivation studies have shown that divergent thinking suffers with inadequate sleep. A few surveys have noted poorer grades in students with inadequate sleep. Many important issues have not yet been well studied. For example, little is known about the consequences of insufficient sleep for relationship formation and maintenance, emotion regulation,delinquency, drug use, and violent behavior. Long-term consequences of insufficient sleep — particularly at critical developmental stages — are utterly unknown.

The problem of inadequate sleep affects more segments of our society than adolescents; however, adolescents appear to be particularly vulnerable and face difficult challenges for obtaining sufficient sleep. Even without the pressure of biological changes, if we combine an early school starting time — say 7:30 a.m., which, with a modest commute, makes 6:15 a.m. a viable rising time — with our knowledge that optimal sleep need is 9¼ hours, we are asking that 16-year-olds go to bed at 9 p.m. Rare is the teenager of the 1990s who will keep such a schedule. School work, sports practices, clubs, volunteer work, and paid employment take precedence. When biological changes are factored in, the ability even to have merely "adequate" sleep is lost. As a consequence, sleepy teens demand that parents provide an extreme form of reveille, challenge teachers to offer maximal classroom entertainment and creativity just to keep them awake, and suffer the consequences of disaffection from school and dissatisfaction with themselves.

Can these problems be solved by delaying the starting time for school as adolescents move into the pubertal years? Not entirely. Moving the opening bell to a later time may help many teens with the mismatch between biological time and scholastic time, but it will not provide more hours in the day. It is not difficult to project that a large number of students see a later starting time as permission to stay up later at night studying, working, surfing the net, watching television,and so forth. Today's teens know little about their sleep needs or about the biological timing system. Interestingly, students do know they are sleepy, but they do not have skills to cope with the issue, and many assume — just as adults do — that they are expected to function with an inadequate amount of sleep. This assumption is a physiological fallacy:sleep is not optional. Sleep is biologically obligatory. If students learn about sleep, they have a basis to use a changed school starting time to best advantage. Adding information about sleep to the school curriculum can certainly help.

As with other fields of scientific investigation, the knowledge base, the scientific opportunities, and the level of pure excitement in sleep and biological rhythms research have never been greater. This knowledge and excitement can be shared with students at every academic level. Furthermore, sleep and biological rhythms are natural gateways to learning because students are drawn to the topics. Thus, as grammar school students learn about the nutrition pyramid, so too could they learn about the body's sleep requirements and how the biological timing system makes humans day-active rather than night-active. (Did you know that,if you put your hamster in a box with lights that turn on at night and off in the daytime, it will start running on its wheel during the day?)

As middle school students are leaming about comparative biology, they can be sharing in the excitement of where, when, and how animals sleep. (Did you know that certain dolphins can be half asleep . . . literally? One half of the brain sleeps while the other half is awake! Did you know that mammals stop regulating body temperature in REM sleep? Did you know that you are paralyzed in REM sleep?)

High school students can share the excitement in the discoveries about genes that control the biological clock,about the brain mechanisms that control dreaming, about the way sleep creates breathing problems, and about sleep disorders that may affect their family members. (Did you know that snoring may be a sign of a serious sleep disorder afflicting as many as 5% of adults? Did you know that some people act out their dreams at night? Did you know that genes controlling the biological clock in mice and fruit flies are nearly identical?)

Challenges and an Opportunity

The challenges are great,and solutions do not come easily. School scheduling is incredibly complex, and accounting for youngsters' sleep needs and biological propensities adds to the complexity. Yet we cannot assume that the system is immutable. Given that the primary focus of education is to maximize human potential, then a new task before us is to ensure that the conditions in which learning takes place address the very biology of our learners.

1.Mary A. Carskadon, "Determinants of Daytime Sleepiness: Adolescent Development, Extended and Restricted Nocturnal Sleep"(Doctoral dissertation, Stanford University, 1979); idem, "The Second Decade," in Christian Guilleminault, ed., *Sleeping and Waking Disorders:Indications and Techniques* (Menlo Park,Calif.:Addison Wesley, 1982),pp. 99-125; and Mary A. Carskadon,E. John Orav, and William C. Dement, "Evolution of Sleep and Daytime Sleepiness in Adolescents," in Christian Guilleminault and Elio Lugaresi,eds., *Sleep/Wake Disorders:Natural History, Epidemiology, and Long-Term Evolution* (New York: Raven Press, 1983), pp. 201-16.

2.Mary A. Carskadon and William C. Dement,"The Multiple Sleep Latency Test: What Does It Measure?," *Sleep*, vol. 5, 1982, pp. 67-72.

3.Mary A. Carskadon et al., "Pubertal Changes in Daytime Sleepiness," *Sleep*, vol. 2,1980, pp. 453-60.

4.Carskadon, "Determinants of Daytime Sleepiness."

5.Mary A. Carskadon,"Patterns of Sleep and Sleepiness in Adolescents," *Pediatrician*, vol. 17, 1990, pp. 5-12.

6.Mirian M. M. Andrade and Luiz Menna-Barreto, "Sleep Patterns of High School Students Living in Sao Paulo,Brazil," in Mary A. Carskadon,ed., *Adolescent Sleep Patterns:Biological,Social,and Psychological Influences* (New York: Cambridge University Press, forthcoming); Kaneyoshi Ishihara, Yukako Honma, and Susumu Miyake, "Investigation of the Children's Version of the Morningness-Eveningness Questionnaire with Primary and Junior High School Pupils in Japan," *Perceptual and Motor Skills*, vol. 71, 1990, pp. 1353-54; Helen M. Bearpark and Patricia T. Michie, "Prevalence of Sleep/Wake Disturbances in Sidney Adolescents," *Sleep Research*, vol. 16, 1987, p. 304; and Inge Strauch and Barbara Meier, "Sleep Need in Adolescents: A Longitudinal Approach," *Sleep*, vol. 11, 1988, pp. 378-86.

7.Alexander A. Borbély, "A Two Process Model of Sleep Regulation," *Human Neurobiology*, vol. 1, 1982, pp. 195-204.

8.Mary A. Carskadon,Cecilia Vieira,and Christine Acebo, "Association Between Puberty and Delayed Phase Preference," *Sleep*, vol. 16,1993,pp. 258-62.

9.Alfred J. Lewy and Robert L. Sack, "The Dim Light Melatonin Onset as a Marker for Circadian Phase Position," *Chronobiology International*, vol. 6, 1989, pp. 93-102.

10. J. M. Tanner, *Growth at Adolescence*, 2nd ed. (Oxford: Blackwell, 1962).

11.Mary A. Carskadon et al., "An Approach to Studying Circadian Rhythms of Adolescent Humans," *Journal of Biological Rhythms*, vol. 12, 1997, pp. 278-89.

12.Mary A. Carskadon et al.,"Circadian and Homeostatic Influences on Sleep Latency in Adolescents," paper presented at the sixth meeting of the Society for Research on Biological Rhythms,Amelia Island Plantations, 1998.

13.Dale M. Edgar, William C. Dement,and Charles A. Fuller,"Effect of SCN Lesions on Sleep in Squirrel Monkeys — Evidence for Opponent Processes in Sleep-Wake Regulation," *Journal of Neuroscience*, vol. 13, 1993, pp. 1065-79.

14.Carskadon et al., "An Approach to Studying Circadian Rhythms."

15.Mary A. Carskadon et al., "Adolescent Sleep Patterns, Circadian Timing, and Sleepiness at a Transition to Early School Days," *Sleep*, in press.

16.Amy R. Wolfson and Mary A. Carskadon,"Sleep Schedules and Daytime Functioning in Adolescents," *Child Development*, vol. 69, 1998, pp. 875-87.

Mary A. Carskadon, When Worlds Collide: Adolescent Need for Sleep Versus Societal Demands, Phi Delta Kappan, Vol. 80, No. 05, January 1999, pp. 348-353.