2. Factors Influencing Sleep Patterns of Adolescents

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> How I hated her method of waking me. My adolescent sleeps were long, dark and sullen. Never once in all those years did I wake of my own accord. It was Margaret, always, knocking on my door like some rodent trapped behind a wall. This would bring me to a rage of wakefulness and I would stomp into the bathroom, bad-tempered and clearly in the wrong, while Margaret, who had been up and gone to six o'clock Mass, would watch me with a silent and superior reproach. That would increase my fury; it is impossible to feel the equal of someone who's been awake longer than you.
>
> Mary Gordon, *Final Payments*

Sleep patterns in humans emerge from a complex interplay of several distinct processes: maturation and development, behavioral phenomena, and intrinsic sleep and circadian regulatory mechanisms. Each factor likely plays an important role during the transition from childhood to adulthood, a time when significant changes in sleep patterns occur. Sleep also affects many facets of waking human life, although a definitive explanation of sleep's function(s) remains undiscovered. Unquestioned, however, is the obligatory nature of sleep and our commonsense intuition that sleep fulfills some vital role in our waking lives, a role that enhances our abilities to think, perform, feel, and interact.

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The patterns of sleep that unfold during adolescence differ markedly from those of preadolescents. Our sense is that many adolescents in the United States obtain insufficient and ill-timed sleep and that daytime functioning suffers as a consequence. This review will focus on a number of major factors that affect sleep patterns of adolescents, summarize a recent study that examines several factors in an operational setting, and speculate on major consequences of these changes.

Although large-scale epidemiologic studies of broadly generalizable samples are not available, our group is reasonably certain that many adolescents do not obtain adequate sleep, based upon self-reported sleep-wake patterns of children and adolescents investigated by a number of groups, primarily using cross-sectional sleep habits surveys (Strauch, Dubral, & Strucholz, 1973; Webb & Agnew, 1973, 1975; Zepelin, Hamilton, & Wanzie, 1977; Anders, Carskadon, Dement, & Harvey, 1978; Price, Coates, Thoresen, & Grinstead, 1978; Carskadon, 1979; White, Hahn, & Mitler, 1980; Klackenberg, 1982; Petta, Carskadon, & Dement, 1984; Bearpark & Michie, 1987; Billiard, Alperovitch, Perot, & Jammes, 1987; Henschel & Lack, 1987; Strauch & Meier, 1988; Carskadon, 1990a,b; Andrade, Benedito-Silva, & Menna-Barreto, 1992; Gau & Soong, 1995; Saarenpaaheikkila, Rintahaka, Laippala, & Koivikko, 1995; Wolfson & Carskadon, 1998). A few longitudinal survey studies have also been done (Klackenberg, 1982; Strauch & Meier, 1988; Andrade et al., 1992). Several major trends emerge from such data:

- Older teenagers sleep less than younger teenagers.
- The timing of sleep is delayed in older versus younger teenagers.
- With age, teenagers show an increasingly large discrepancy between school night and weekend sleep schedules.

This chapter focuses on the first two of these trends; the third is examined by Acebo and Carskadon (Chapter 13 in this volume).

Behavioral Phenomena

Physiological processes play an important role in regulating sleep and wakefulness. Yet, human sleep patterns are also determined by choices, often rooted in psychosocial phenomena. Such phenomena include, for example, delaying bedtime to socialize or to finish reading a good book, advancing bedtime in anticipation of an early rising, truncating sleep

length with an alarm clock, and so forth. Behavioral contributions to sleep patterns are strong in both children and adolescents; however, a rapidly changing psychosocial milieu during adolescence contributes to marked alterations in the behavioral phenomena affecting sleep patterns.

Parents

One of our first studies of sleep patterns at the childhood-to-adolescent transition (Carskadon, 1979) showed a change in the influence of parents on children's sleep patterns, particularly on school days. Among other items, this sleep habits survey of 218 children asked students to describe the reasons they had for going to bed at night and waking up in the morning. Children aged 10 and 11 years were significantly more likely to report that parents set their school-night bedtimes (age 10 = 54.3%; age 11 = 48.3%) than were the 12- and 13-year-old children (age 12 = 38.5%; age 13 = 19.6%); conversely, the 12- and 13-year-olds reported more frequently (age 12 = 73.1%; age 13 = 70.2%) that parents or alarm clocks provided the morning stimulus to wake up on school mornings than did the younger children (age 10 = 45.7%; age 11 = 37.9%).

Our subsequent studies of high school students have shown that older adolescents report much later bedtimes and give such reasons for staying up late as watching television, finishing homework, and socializing. For example, our group recently undertook a survey of approximately 3,000 9th through 12th grade students from four Rhode Island school districts (Acebo & Carskadon, 1997; Wolfson & Carskadon, 1998; referred to here as "the high school survey") using an eight-page anonymously administered self-report form (reproduced at http://www.sleepforscience.org). Only 5.1% of these older teens had a school-night bedtime set by parents; 32.7% went to bed when homework (13.1%), TV viewing (8.7%), or socializing (10.9%) was finished for the day, and 44.1% reported that bedtime was set by the time they feel sleepy. Furthermore, an even higher percentage of high school than primary school students reported relying on an external source for a school-morning wake-up cue. Our high school survey data show that 87% of older teens use an alarm (59.9%) or parent (27.1%) for waking them up on school days. As summarized in Figure 2.1, these data indicate strong developmental trends: parents are more likely to set bedtimes for younger adolescents, more likely to assist with waking up older adolescents, and the younger adolescents are

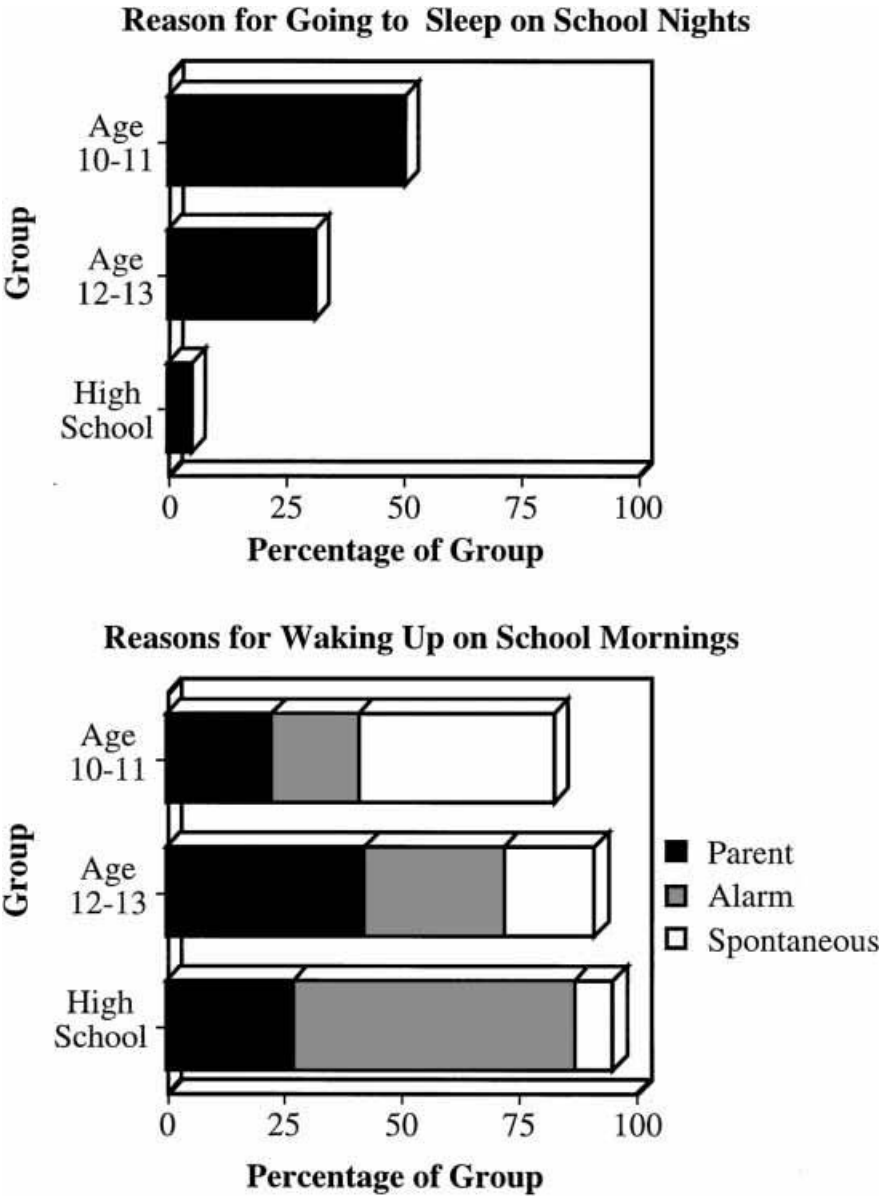

Figure 2.1. Self-report data from sleep habits surveys of younger (ages 10 and 11 years; 12 and 13 years) and older adolescents (high school students ages 14 to 18 years). The top figure shows the percentage of students who reported that their parents set their bedtimes on school nights. In the lower panel, data are similarly summarized for three of the reasons students reported for factors that determine what time they wake up on school mornings, parents (black), alarm clock (gray), or spontaneous (white, i.e., "I don't know. I just woke up") arousals.

significantly more likely than older teens to wake spontaneously on school mornings.

Peers

Although a commonly remarked feature of adolescent development is the increasing influence of peer group on behavior, we are aware of no data that directly assay this factor with regard to sleep patterns. Our recent high school survey data indicate a minor influence of school on one or two sleep variables; however, this effect may be more a result of the school schedules per se than of peer influences. The data show that evening "socializing" is a factor influencing school-night bedtimes in only 10.9%, although a case might be made that TV watching (associated with bedtimes in 8.7% of students) may have a peer-group component. These adolescents reported a significantly greater influence of social activities to account for bedtime on weekend nights, with 40.9% reporting this factor as the chief reason they choose to go to bed. One conclusion, therefore, is that the older adolescents have more social opportunities or greater access to evening social activities, and these activities have a greater influence on sleep patterns during the weekends than on school nights.

Academics

Academic obligations are often mentioned by adolescents when asked about factors affecting their sleep patterns. Our high school survey data show evidence that homework is a significant factor influencing sleep patterns for only a limited number of students. Thus, approximately 13% of the 9th through 12th grade students reported that their school-night bedtime is set according to the time they finish their homework. This relationship does not seem to reflect a developmental change, because about 15.2% of 12- and 13-year-old students in our earlier study reported staying up until homework was finished (Carskadon, 1979). Our high school survey data also indicate a rather low mean number of hours these students reported studying in the last week, on average about an hour a day (6.7 ± 5.9 hours per week). Data from students in another educational system – Taipei, Taiwan – showed that the students in more academically challenging programs reported less sleep and lower levels of alertness than those students in the less challenging program (Gau & Soong, 1995). In the United States, as well, those students on

the academic fast track are likely to sleep less, although data confirming this trend are not available.

Extracurricular Club Activities and Sports

Other activities that may influence sleep patterns of adolescents include extracurricular club activities, such as chorus, band, orchestra, and scouting, as well as after school sports. Our recent high school survey examined these factors by asking students to describe the nature of these obligations. Only about one-quarter of the sample reported participating in extracurricular club activities during the preceding week, and 90% of these students took part fewer than 12 hours per week. Students in this survey reported somewhat more participation in sports, with about one-third of students involved in organized athletics in the past week, 80% of whom reported participating 12 or fewer hours per week. For the majority of the students, therefore, extracurricular club activities or after school sports were not a major factor determining sleep patterns. Future analyses will examine these issues more closely, particularly to identify students whose commitments span many activities, in which case sleep may be affected more significantly. One group most likely to experience significant sleep loss includes those students with multiple commitments who also work.

Employment

As we have indicated elsewhere (Carskadon, Mancuso, & Rosekind, 1989; Carskadon, 1990a,b), a major influence on sleep patterns of U.S. high school students is the number of hours they spend working for pay. Thus, we have previously noted that students who report working 20 or more hours per week (about 28% of our earlier high school sample) report having later bedtimes, sleeping fewer hours per night, and falling asleep in school and oversleeping more frequently than do those who either do not work or who work fewer than 20 hours per week. Our more recent high school survey, which asked students to report hours worked in the last week, shows similar findings. About half the students reported working, and the average number of hours worked was 19.5 (median = 18 hours). As further explicated in Chapter 12, the association of hours spent working with sleep parameters and other outcome variables is also similar to our previous findings. For example, number of hours worked across the week reported by the new high school sample

is correlated with school-night total sleep time ($r = -.235$; $p < .001$) and school-night bedtime ($r = .345$; $p < .001$).

The developmental psychologist Laurence Steinberg and his colleagues Bradford Brown and Sanford Dornbusch (1996) make the point that the rather impressive amount of time adolescents in the United States spend working for pay is a relatively new phenomenon, appearing only in the second half of the 20th century. Furthermore, they note that the typical adolescent is neither working to save for college education or to supplement family income nor serving in a true apprenticeship position to learn valuable job skills but rather is earning money to spend on personal consumables by working as largely unskilled laborers. Hours of work are not confined to weekends but extend significantly into the school week. According to Steinberg (1996), "by the time they are seniors in high school, many students spend more time on the job than they do in the classroom" (p. 169).

School Start Time

In most U.S. school districts, the start of the school day is progressively earlier as students move from grade school to middle school to high school (Allen, 1991, 1992). Thus, adolescents are required to rise earlier in the morning than preteens in order to get to school on time. We have hypothesized that older adolescents do not adjust appropriately to these demands. As with adolescent employment, historical trends may play a role in the issue of early school start time for older teens. Preliminary data, for example, show that the starting times for U.S. high schools have moved to an earlier hour across the past 20 years (Carskadon & Acebo, 1997). Other countries are not immune to this problem, as noted in Israel, where the "zero hour" (i.e., 7:00 A.M.) for school start time has become a recent concern (Epstein, Chillag, & Lavie, 1995). Clearly for most teenagers, the school bell is a major nonnegotiable factor that mandates the termination – often premature – of nocturnal sleep.

One other important consequence of earlier school start times unrelated to sleep patterns is the amount of largely self-supervised time adolescents have when school release times are also moved earlier. Increasingly, investigators (and legislators, at least in Minnesota) are noting this phenomenon with concern. For example, Richardson, Radziszewska, Dent, and Flay (1993) note an association with after school "self-care" in adolescents and substance abuse, risk taking, depressed mood, and lower academic grades.

Intrinsic-Biological Processes

While the behavioral and psychosocial processes and exposures that adolescents undergo clearly have a marked influence on developing sleep patterns, biological processes may also contribute. The notion that physical changes associated with adolescent development may contribute to sleep patterns is relatively new. In fact, we and others had assumed for many years that the entire scope of the broad behavioral changes in sleep patterns associated with adolescence (e.g., reduced and delayed sleep) could be entirely accounted for by behavioral factors. A gradual accumulation of small pieces of evidence has led us to speculate more strongly about the influence of biological processes, which we categorize under two principal regulatory systems: the sleep mechanisms and the circadian timing system.

Intrinsic Sleep Mechanisms

Commonly referred to as sleep homeostatic mechanisms, intrinsic sleep mechanisms strongly influence the distribution and patterning of sleeping and waking. In simplest terms, the longer one goes without sleep or with minimal sleep, the greater the rebound of sleep. Data on sleep infrastructure from many groups clearly indicate that the amount of slow wave (stages 3 and 4) sleep (cf. Berger & Oswald, 1962; Williams, Hammach, Daly, Dement, & Lubin, 1964; Moses, Johnson, Naitoh, & Lubin, 1975; Webb & Agnew, 1975) and electroencephalographic (EEG) delta power during sleep (Borbély, Bauman, Brandeis, Strauch, & Lehmann, 1981; Feinberg, Fein, & Floyd, 1982) depend on the length of prior wakefulness, providing experimental evidence for and a physiological marker of the homeostatic process. We have previously shown that, like adults, children and adolescents respond to sleep restriction and sleep deprivation with increased physiological sleepiness, as well as an increase in stages 3 and 4 non–rapid eye movement (NREM) sleep during recovery (Carskadon, 1979; Carskadon, Harvey, & Dement, 1981a,b,c). Experimental support for this phenomenon was recently reemphasized in a report by Rosenthal, Roehrs, Rosen, and Roth (1993) demonstrating that graded restriction of nocturnal sleep produced graduated increases of recovery sleep during the subsequent 24 hours, as well as daytime sleepiness on the multiple sleep latency test (MSLT).[1]

[1] The MSLT is a test of sleep tendency or "physiological sleepiness" developed in the 1970s by the Stanford group (Carskadon & Dement, 1977b; Richardson, Carskadon,

Few data exist to indicate whether homeostatic mechanisms change at the childhood-to-adolescent transition or across adolescent development. Could adolescents find it easier to stay awake later because the sleep homeostatic mechanism undergoes developmental alterations? Known developmental changes in sleep physiology that occur in adolescence are rather subtle, largely involving the infrastructure of sleep and the pattern of sleepiness-alertness. For example, when youngsters are permitted to sleep 10 hours a night (i.e., when their sleep is not constrained to their "usual" amount), a marked linear decline in slow wave sleep occurs across adolescence, although total nocturnal sleep length is unchanged at about 9.25 hours (Carskadon, 1982). (The latter finding is often cited as evidence that the need for sleep does not decline across the teenage years.)

Studies have also shown that REM sleep latency at night is generally shorter in adolescents than in prepubertal children (Karacan, Anch, Thornby, Okawa, & Williams, 1975; Carskadon, 1982; Coble, Kupfer, Taska, & Kane, 1984). Furthermore, midpuberty is accompanied by a clear-cut augmentation of daytime sleepiness measured using the MSLT (Carskadon, Harvey, Duke, Anders, Litt, & Dement, 1980), even when sleep amount is unchanged. (This finding has been interpreted by some to indicate that teenagers may actually have an increasing need for sleep.)

One interpretation of these developmental findings is that they indicate a reduction in the intensity of sleeping (reduced slow wave sleep) and waking (increased physiological sleepiness on MSLT) processes coincident with adolescence, in other words suggesting a developmental decline in the strength of homeostatic control. The slow-wave sleep decline across adolescence, on the other hand, may simply represent an epiphenomenon of cortical maturation (dendritic pruning), with no fundamental relationship to sleep mechanisms (Feinberg, 1983).

Our previous studies of acute sleep restriction and sleep loss in adolescents and young adults showed no marked age-related differences

Flagg, van den Hoed, Dement, & Mitler, 1978). The measure involves assessment of the speed of falling asleep at 2-hour intervals across a day under controlled conditions. Useful in the diagnosis of narcolepsy because of the occurrence of REM sleep during the brief naps (Mitler et al., 1979), sleep latency scores on the MSLT have been categorized by the *International Classification of Sleep Disorders* (1990) as indicating severe sleepiness (< 5 minutes), moderate (5 to 10 minutes), and mild (10 to 15 minutes). Behavioral studies indicate significant performance decrements and episodes of unintended sleep in association with the severe level of sleepiness (Roth, Roehrs, Carskadon, & Dement, 1994).

either in the "sleepiness response" to these procedures or in the recovery process, although these issues were not expressly addressed in experimental design and analyses. On the other hand, using a matched comparison of prepubescent and pubertal adolescents in a carefully monitored sleep-loss study, we noted a tendency for younger children to respond with somewhat more frequent "unintentional" sleep episodes (Carskadon, Littell, & Dement, 1985). This finding also seems to indicate a reduction in the intensity of the homeostatic sleep-wake control as a function of adolescent development. Nevertheless, the question remains open whether pubertal maturation impacts on the homeostatic mechanisms controlling sleep and its consequences for waking behavior.

These intrinsic homeostatic mechanisms also provide tools to examine the neurophysiologic responses to insufficient sleep. Thus, we can use the MSLT to examine the response of the waking brain and slow-wave sleep data to assess the response of the sleeping brain. Ongoing experiments in our laboratory address these mechanisms directly. One preliminary finding (Carskadon, Acebo, & Seifer, 2001) indicates that the slow-wave sleep response to sleep deprivation in pubertal teenagers is reduced versus prepubertal youngsters. Our most current model integrates developmental changes in the organization of the circadian timing system with sleep-wake homeostasis in ways that favor evening alertness in pubertal adolescents.

Circadian Timing System

Another factor with considerable consequences for the patterning of sleeping and waking is the circadian timing system, which affects both the infrastructure of sleep as well as the timing of sleep and waking behavior. For example, studies that involve temporal isolation or multiple sleep opportunities across a day (e.g., 90-minute or 180-minute day) show that the timing of sleep onset (Weitzman et al., 1974; Carskadon & Dement, 1977a; Zulley, Wever, & Aschoff, 1981), the length of sleep (Czeisler, Weitzman, Moore-Ede, Zimmerman, & Kronauer, 1980; Strogatz, 1986), and the timing of REM sleep (Carskadon & Dement, 1980; Czeisler, Zimmerman, Ronda, Moore-Ede, & Weitzman, 1980; Zulley, 1980) vary as a function of the phase of the circadian timing system. In most human studies, the output of the circadian oscillator is marked principally by the pattern of core body temperature, which typically rises across the day to the late afternoon or early evening and

then falls across the nighttime, rising again in the early morning hours. The trough of this temperature cycle marks the peak phase for REM sleep, which seems to be tightly coupled to the circadian timing system. Human studies have also identified the predictable occurrence of daily "forbidden zones" when it is difficult to fall asleep (Strogatz, 1986) and "gates" when sleep comes most easily (Lavie, 1985). Animal studies also indicate that the circadian oscillator provides a major signal for daily activity onset (Edgar, Dement, & Fuller, 1993).

Another variable that provides access to the "hands" on the circadian clock involves the measurement of the melatonin secretory cycle. The circadian timing system controls the timing of the secretion of this hormone by the pineal gland. The melatonin secretory phase occurs during the nocturnal hours in both diurnal and nocturnal animals, and it is thus sometimes referred to as the hormone of darkness. If an animal is exposed to a bright light source during the melatonin secretory phase, the hormone's secretion is temporarily terminated. Thus, measurement of melatonin must take place in dim lighting conditions. Melatonin is available from plasma samples as well as from saliva. The onset of the melatonin secretory phase measured in dim light has been called the dim-light melatonin onset (DLMO) measure and is used by Lewy and Sack (1989) and others (e.g., Van Cauter et al., 1994) to assess the circadian system. In our laboratory, we have successfully measured a component of the circadian timing system with salivary melatonin. Figure 2.2 illustrates data obtained using this method in adolescents evaluated on two occasions separated by about 4 months.

A number of investigators have noted alterations in melatonin secretion in association with pubertal development: in general, the level of serum or salivary melatonin secretion declines during puberty. Some have speculated that melatonin may be involved in inhibiting the onset of puberty. These speculations were raised in conjunction with findings that a "dilution" of circulating nocturnal melatonin levels is associated with physical growth. Furthermore, an inverse relationship between circulating melatonin and leuteinizing hormone (LH) levels occurs across pubertal development (Waldhauser & Steger, 1986). Others have failed to confirm a direct causal association between melatonin and puberty, however, even with extensive trials (e.g., Wilson, Lackey, Chikazawa, & Gordon, 1993). These concerns are not germane in the context of our experimental protocols, in which melatonin is used to mark the phase of the circadian timing system.

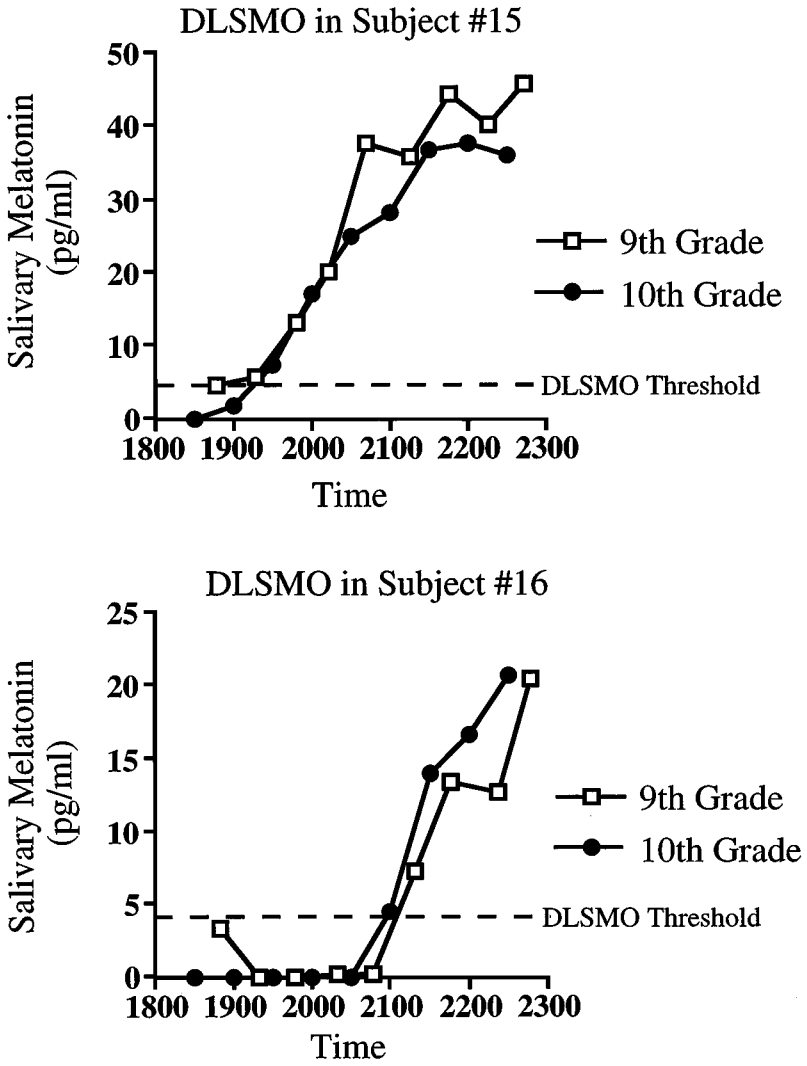

Figure 2.2. Dim-light salivary melatonin levels obtained from 30-minute evening samples obtained in dim light (< 50 lux) in two adolescent volunteers on occasions approximately 4 months apart. These adolescents were participants in the school transition project. Subject 15 is a girl who was age 14.8 years on the first (9th grade) assessment; subject 16 is a girl who was age 15.1 years on the first assessment.

Many studies have noted a clear circadian component to the timing of a variety of human behaviors. Thus, in addition to sleep and wakefulness, performance, memory function, and mood fluctuate with a circadian periodicity (cf. Monk, Fookson, Moline, & Pollack, 1985; Boivin et al., 1997). Such behaviors are also influenced by sleep deprivation (cf. Dinges & Kribbs, 1991). Thus sleep deprivation and circadian timing may both relate to vulnerability in these behavioral domains, either independently (through insufficient sleep or ill-timed behavior) or simultaneously.

Our interest was initially drawn to a possible association between the circadian timing system and adolescent development by the patterns of behavioral change revealed by surveys. As noted, sleep time shortens across the adolescent span, and this reduction of sleep time under weekday constraints is related to a delay in the phase of sleep onset and an advance in the *enforced* time of sleep offset. On weekends, sleep onset is later, as is sleep offset – if released from *enforced* arousal – in adolescents. These patterns are also associated with a change in teenagers' perceived phase preference (Andrade et al., 1992; Carskadon, Vieira, & Acebo, 1993) – that is, the degree to which youngsters recognize in themselves an increasing "evening" versus "morning" capacity for activities. An important corollary may be found in other primates, as noted Golub and her colleagues, who find a delay in the timing of activity rhythms occurring in association with puberty in female rhesus monkeys (Golub, 1996; Chapter 5 in this volume). We have also obtained data from our "long nights" studies of human adolescents indicating that a phase delay in the circadian timing system may occur in association with puberty (Carskadon, Acebo, Richardson, Tate, & Seifer, 1997). The offset phase of melatonin secretion was correlated with age ($r = .62$, $df = 12$, $p = .018$) and Tanner stage ($r = .62$, $df = 12$, $p = .02$).[2] This finding indicates that the intrinsic circadian timing system changes during adolescent development in a phase delay direction. If so, then strategies for interventions to improve adolescent sleep patterns will need to take this change into consideration.

These findings highlight the usefulness of monitoring circadian timing when assessing adolescent sleep patterns and their consequences. Certain adolescents may be profoundly affected by changes in the

[2] Tanner staging is a means of identifying pubertal status by physical examination of secondary sexual characteristics, in which stage 1 is prepubertal and stage 5 is mature (Tanner, 1962).

circadian timing system and others may experience a more modest effect. The project described in the next section utilized sleep and circadian measures to evaluate the response of adolescents to a behavioral event: the transition to an earlier school start time.

School Transition Study

The chief goal of this study (Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998) was to examine the effects of a transition from 9th grade, where school started at 8:25 A.M., to 10th grade, where school started at 7:20 A.M. Evaluations were scheduled at three time points: spring (9th grade), summer vacation, and fall (10th grade); a subgroup was also evaluated in March of 10th grade. Participants wore small wrist monitors to measure sleeping and waking activity levels, and they also completed sleep diaries for two weeks. Saliva samples were collected on the last evening under dim light conditions, and this was followed by an overnight polysomnographic sleep study and subsequent daytime sleepiness testing using the MSLT. Forty 9th grade students were enrolled in the project. They included 15 males and 25 females, ages 14 to 16.2 (mean = 15.0 ± .47) years at the time of the first assessment. Tanner staging was performed in 34 of the students in 9th grade, and most were well advanced in pubertal development: 3 were Tanner stage 3, 12 were Tanner stage 4, and 19 were Tanner stage 5.

Sleep data were derived from the actigraphically estimated values averaged for the school nights of the week immediately preceding the laboratory study. Our measure of the circadian timing system was the phase of the dim-light salivary melatonin onset (DLSMO), derived from saliva samples obtained in dim light (approximately 20 to 30 lux) on the evening of the in-lab study and computed as the interpolated time of salivary melatonin rising above a threshold of 4 picograms per milliliter. The MSLT measures included the mean value for speed of falling asleep across the day of testing (four sleep latency tests) and the type of sleep that occurred.

Several important changes occurred across the 9th-to-10th-grade transition in these youngsters. In the 10th grade, students showed:

- Less sleep on school nights.
- Earlier rising times on school mornings.
- No change in sleep onset times on school nights.
- Later time of the DLSMO phase.

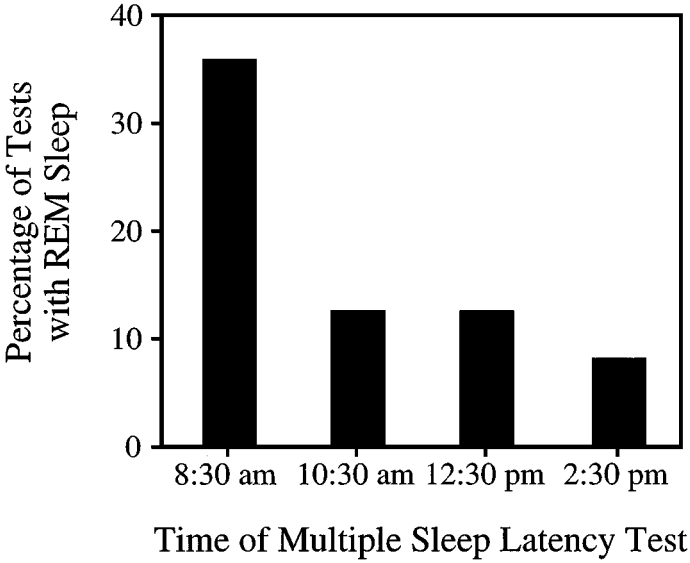

Figure 2.3. The percentage of MSLTs with REM sleep during the 10th-grade assessment of the school transition project. The likelihood of REM sleep episodes was significantly greater in the morning than in the afternoon.

- Greater sleepiness on MSLT.
- REM sleep on MSLT in 12 of 25 students evaluated, REM occurring chiefly on morning MSLTs (see Figure 2.3) and related to a delay in the DLSMO phase (Carskadon et al., 1998).

The latter finding was striking and of particular importance: REM sleep episodes do not frequently occur on MSLTs in normal humans under normal sleep-wake conditions, but such episodes are significant for the diagnosis of narcolepsy (Richardson et al., 1978; Mitler et al., 1979; Carskadon, Dement, Mitler, Roth, Westbrook, & Keenan, 1986). In patients with narcolepsy, REM sleep occurs during daytime sleep episodes with equal likelihood at any time of day (Mitler, 1982). In our many studies of normal children, adolescents, and young adults, we have seen REM sleep rarely on MSLTs, with an incidence of less than 7% for even one REM episode in subjects studied when on schedules providing at least 8 (young adults) or 10 (children and adolescents) hours of nocturnal sleep. Rosenthal and his colleagues recently (1995) reported an REM sleep incidence of 23% on MSLTs in nonnarcoleptic adults, more commonly in males than females and with a relatively consistent distribution across the day on naps given at 10:00 A.M., 12:00 A.M., 2:00 P.M., and 4:00 P.M.

The implications of these findings are striking. If a delay in circadian phase is related to adolescent development, then requiring older adolescents to attend school and attempt to take part in intellectually meaningful endeavors in the early morning may be biologically inappropriate. Furthermore, rapid sleep onset and early transitions to REM sleep are more indicative of a brain ready to sleep than to be awake. Data from our long-nights study showed a mean melatonin secretory duration of 634 minutes in adolescents. Thus, it is not at all unlikely that teenagers are being asked to be awake when the circadian system is in its nocturnal mode. The students may be in school, but their brains are at home on their pillows.

At face value, these data indicate that many students are not adjusting adequately to the school start time change and that nearly one-half of the students are perhaps attending school at a circadian phase that favors sleep over waking. In our protocol, the first MSLT occurred at 8:30 A.M., which places it during the time of the second school class period. At this time, students were able to fall asleep in less than 5 minutes and were likely to have REM sleep onsets. In summary, our school transition project provides compelling evidence calling into question early school start times in the absence of any positive steps to assist students to make an appropriate adjustment to such schedules. In general, the adolescents with early school start times did not obtain sufficient sleep, and many were excessively sleepy and attending school at a biologically inappropriate time.

Vulnerable Systems

We have noted two principal alterations of sleep-wake patterns during adolescence: a change in the timing of sleep, which is strongly influenced by psychosocial factors that likely occur in combination with changes in the biological sleep and circadian timing systems, all of which produce a delay in the timing of sleep onset; and a widespread pattern of insufficient sleep, which is influenced greatly by the sleep delay compounded by the nonnegotiable necessity to terminate sleep prematurely in order to attend school. The association of reduced nocturnal sleep with increased daytime sleepiness is well established. Sleepiness has also been associated with a number of behavioral consequences in adults, principally in the realm of performance decrements (cf. Dinges & Kribbs, 1991). Academic performance difficulties have been reported in adolescents (Kowalksi & Allen, 1995; Link & Ancoli-Israel, 1995; Wolfson & Carskadon, 1998). Another area recently identified is the relationship

between sleep loss and the immune system, with several animal and human studies providing preliminary, as yet tentative evidence that the processes are linked (Dinges, Douglas, Hamarman, Zaugg, & Kapoor, 1995; Everson, 1995). Direct evidence of increased incidence of illness in sleep-deprived populations is not available. Danner (1993) was among the first to note that physical injury may be associated with inadequate sleep in adolescents, and Pack and his colleagues (1995) clearly indicate that teenagers and young adults are at highest risk for automobile accidents. Our recent high school survey data also indicate a small but significant association between injuries and sleep patterns in high school students (Acebo & Carskadon, 1997). Other important issues, such as relationship formation and maintenance, truancy and delinquency, and especially emotion regulation, have been inadequately studied in association with sleep patterns and insufficient sleep. Animal research, however, has found that sleep loss – in particular, REM sleep loss – is associated with marked increases in aggressive behavior and violence (cf. Hicks, Moore, Hayes, Phillips, & Hawkins, 1979; Peder, Elomaa, & Johansson, 1986; Vogel, Minter, & Woolwine, 1986).

Concern over insufficient sleep in our society has been rising, yet this concern is based principally on "disasters" that occur in sleep-deprived adults resulting largely from performance deficits – the nuclear accidents at Three Mile Island and Chernobyl, highway crashes, and so forth (e.g., Mitler, Carskadon, Czeisler, Dinges, Graeber, & Dement, 1988; Dinges, Graeber, Carskadon, Czeisler, & Dement, 1989). Our previous research has highlighted particular risks for adolescents in whom sleep need competes for time in the face of increasing social and academic pressures (Carskadon, 1990a,b). Hard data are sparse and rarely include youngsters who may confront the greatest threats to safe and adequate sleep and attendant risk factors.

Research is needed to fill these gaps and help us begin to understand more completely the role sleep plays in adolescents' daily lives. For example, if as we suspect, sleep helps to regulate emotions, may there be lasting consequences? We can only speculate about emotional consequences of insufficient sleep on a societal level. If the hypotheses of our research are supported, we will have evidence that sleepiness is related to negative emotional expression and higher levels of emotional dysregulation. An important connection may exist between the growing level of violent behavior among young people (perhaps in part a reflection of failure to regulate emotions) and our society's increasing failure to attend to our bioregulatory needs. Even if the consequences are less

overt, our research may expose a more subtle risk – that insufficient sleep alters cognitive perception, literally coloring life blue. Does excessive sleepiness constitute a dark cloud hanging over adolescents (e.g., Tanz & Charrow, 1993)? Our research postulates that sleep patterns are related to successful waking adaptability and that problems with sleep jeopardize behavioral development. These issues attain increasing importance as adolescents and families are confronted by life-style changes that themselves impinge upon sleep patterns.

Because of the core nature of sleep in the human behavioral repertoire, we believe that sleep difficulties in a large segment of a population can indicate dysregulation on a broader societal level. To the extent that adolescents in our society are not able to obtain sufficient sleep at the appropriate time to facilitate their chief developmental task – that is, to become educated members of society by participating effectively in the learning process – this phenomenon marks a potentially very serious problem. Laurence Steinberg's recent treatise (1996) on the plight of adolescent education speaks eloquently about factors affecting poor school performance. Steinberg bases his conclusions on a large, longitudinal study of American schoolchildren. He concludes that "No curricular overhaul, no instructional innovation, no change in school organization, no toughening of standards, no rethinking or teacher training or compensation will succeed if students do not come to school interested in, and committed to learning" (p. 194). Steinberg cites many factors affecting students' lack of interest in schooling, including that they are falling asleep in the classroom. Our attention is obviously drawn to the sleep issue, and based on our data regarding insufficient and ill-timed sleep in adolescents, we believe that these factors may have a powerful impact on student disaffection with school and may also influence their affinity for after school jobs.

REFERENCES

Acebo C, Carskadon MA (1997). Relations among self-reported sleep patterns, health, and injuries in adolescents. *Sleep Research* 26:149.

Allen RP (1991). School week sleep lag: Sleep problems with earlier starting of senior high schools. *Sleep Research* 20:198.

(1992). Social factors associated with the amount of school week sleep lag for seniors in an early starting suburban high school. *Sleep Research* 21:114.

Anders TF, Carskadon MA, Dement WC, Harvey K (1978). Sleep habits of children and the identification of pathologically sleepy children. *Child Psychiatry and Human Development* 9:56–63.

Andrade MMM, Benedito-Silva AA, Menna-Barreto L (1992). Correlations between morningness-eveningness, character, sleep habits and temperature rhythm in adolescents. *Brazilian Journal of Medical and Biological Research* 28:835–839.

Bearpark HM, Michie PT (1987). Prevalence of sleep/wake disturbances in Sidney adolescents. *Sleep Research* 16:304.

Berger RJ, Oswald I (1962). Effects of sleep deprivation on behavior, subsequent sleep, and dreaming. *Journal of Mental Science* 108:457–465.

Billiard M, Alperovitch A, Perot C, Jammes A (1987). Excessive daytime somnolence in young men: Prevalence and contributing factors. *Sleep* 10:297–305.

Boivin DB, Czeisler CA, Dijk DJ, Duffy JF, Folkard S, Minors DS, Totterdell P, Waterhouse JM (1997). Complex interaction of the sleep-wake cycle and circadian phase modulates mood in healthy subjects. *Archives of General Psychiatry* 54(2):145–152.

Borbély AA, Bauman F, Brandeis P, Strauch I, Lehmann D (1981). Sleep-deprivation: Effect on sleep stages and EEG power density in man. *Clinical Neurophysiology* 51:483–493.

Carskadon MA (1979). Determinants of daytime sleepiness: Adolescent development, extended and restricted nocturnal sleep. Ph.D. dissertation, Stanford University.

(1982). The second decade. In C. Guilleminault, ed., *Sleeping and Waking Disorders: Indications and Techniques*, pp. 99–125. Menlo Park, CA: Addison Wesley.

(1990a). Adolescent sleepiness: Increased risk in a high-risk population. *Alcohol, Drugs and Driving* 5–6:317–328.

(1990b). Patterns of sleep and sleepiness in adolescents. *Pediatrician* 17(1):5–12.

Carskadon MA, Acebo C (1997). Historical view of high school start time: Preliminary results. *Sleep Research* 26:184.

Carskadon MA, Acebo C, Richardson GS, Tate BA, Seifer R (1997). An approach to studying circadian rhythms of adolescent humans. *Journal of Biological Rhythms* 12(3):278–289.

Carskadon MA, Acebo C, Seifer R (2001). Extended nights, sleep loss, and recovery sleep in adolescents. *Archives of Italian Biology* 139:301–312.

Carskadon MA, Dement WC (1977a). Sleepiness and sleep state on a 90-minute schedule. *Psychophysiology* 14:127–133.

(1977b). Sleep tendency: An objective measure of sleep loss. *Sleep Research* 6:200.

(1980). Distribution of REM sleep on a 90-minute sleep-wake schedule. *Sleep* 2:309–317.

Carskadon MA, Dement WC, Mitler MM, Roth T, Westbrook P, Keenan S (1986). Guidelines for the multiple sleep latency test (MSLT): A standard measure of sleepiness. *Sleep* 9:519–524.

Carskadon MA, Harvey K, Dement WC (1981a). Acute restriction of nocturnal sleep in children. *Perceptual Motor Skills* 53:103–112.

(1981b). Multiple sleep latency tests in the development of narcolepsy. *Western Journal of Medicine* 135:414–418.

(1981c). Sleep loss in young adolescents. *Sleep* 4:299–312.
Carskadon MA, Harvey K, Duke P, Anders TF, Litt IF, Dement WC (1980). Pubertal changes in daytime sleepiness. *Sleep* 2:453–460.
Carskadon MA, Littell WP, Dement WC (1985). Constant routine: Alertness, oral body temperature, and performance. *Sleep Research* 14:293.
Carskadon MA, Mancuso J, Rosekind MR (1989). Impact of part-time employment on adolescent sleep patterns. *Sleep Research* 18:114.
Carskadon MA, Vieira C, Acebo C (1993). Association between puberty and a circadian phase delay. *Sleep* 16(3):258–262.
Carskadon MA, Wolfson AR, Acebo C, Tzischinsky O, Seifer R (1998). Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. *Sleep* 21(8):871–881.
Coble PA, Kupfer DJ, Taska LS, Kane J (1984). EEG sleep of normal healthy children. Part 1: Findings using standard measurement methods. *Sleep* 7:289–303.
Czeisler CA, Weitzman ED, Moore-Ede MC, Zimmerman JC, Kronauer RS (1980). Human sleep: Its duration and organization depend on its circadian phase. *Science* 210:264–1267.
Czeisler CA, Zimmerman JC, Ronda JM, Moore-Ede MC, Weitzman ED (1980). Timing of REM sleep is coupled to the circadian rhythm of body temperature in man. *Sleep* 2:329–346.
Danner F (1993). Sleep patterns and health during adolescence. *Sleep Research* 22:79.
Dinges DF, Douglas SD, Hamarman S, Zaugg L, Kapoor S (1995). Sleep deprivation and human immune function. *Advances in Neuroimmunology* 5(2): 97–110.
Dinges, DF, Graeber RC, Carskadon MA, Czeisler CA, Dement WC (1989). Attending to inattention. *Science* 245:342.
Dinges DF, Kribbs NB (1991). Performing while sleepy: Effects of experimentally-induced sleepiness. In T. Monk, ed., *Sleep, Sleepiness and Performance*, pp. 97–128. New York: John Wiley & Sons.
Edgar DM, Dement WC, Fuller CA (1993). Effect of SCN lesions on sleep in squirrel monkeys: Evidence for opponent processes in sleep-wake regulation. *Journal of Neuroscience* 13(3):1065–1079.
Epstein R, Chillag N, Lavie P (1995). Sleep habits of children and adolescents in Israel: The influence of starting time of schools. *Sleep Research* 24A:432.
Everson C (1995). Functional consequences of sustained sleep deprivation in the rat. *Behavioural Brain Research* 69:43–54.
Feinberg I (1983). Schizophrenia: Caused by a fault in programmed synaptic elimination during adolescence? *Journal of Psychiatric Research* 17(4): 319–334.
Feinberg I, Fein G, Floyd TC (1982). Computer-detected patterns of electroencephalographic delta activity during and after extended sleep. *Science* 215:1131–1133.
Gau S-F, Soong WT (1995). Sleep problems of junior high school students in Taipei. *Sleep* 18(8):667–673.

Golub MS (1996). Changes in diurnal rest-activity patterns of rhesus monkeys during adolescence. Paper presented at NIH symposium on Neurobiology of Adolescent Depression, Bethesda, MD, March 28–29.

Henschel A, Lack L (1987). Do many adolescents sleep poorly or just too late? *Sleep Research* 16:354.

Hicks RA, Moore JD, Hayes C, Phillips N, Hawkins J (1979). REM sleep deprivation increases aggressiveness in male rats. *Physiology and Behavior* 22(6):1097–1100.

International Classification of Sleep Disorders: Diagnostic and Coding Manual (1990). Diagnostic classification steering committee, MJ Thorpy, Chairman, Rochester, Minnesota, American Sleep Disorders Association.

Karacan I, Anch M, Thornby JI, Okawa M, Williams RL (1975). Longitudinal sleep patterns during pubertal growth: Four-year follow-up. *Pediatric Research* 9:842–846.

Klackenberg G (1982). Sleep behaviour studied longitudinally. *Acta Paediatrica Scandinavica* 71:501–506.

Kowalski N, Allen R (1995). School sleep lag is less but persists with a very late starting high school. *Sleep Research* 24:124.

Lavie, P (1985). Ultradian rhythms: Gates of sleep and wakefulness. *Experimental Brain Research* (suppl. 12):149–164.

Lewy AJ, Sack, RL (1989). The dim light melatonin onset as a marker for circadian phase position. *Chronobiology International* 6(1):993–1002.

Link SC, Ancoli-Israel S (1995). Sleep and the teenager. *Sleep Research* 24a:184.

Mitler MM (1982). The multiple sleep latency test as an evaluation for excessive somnolence. In C. Guilleminault, ed., *Sleeping and Waking Disorders: Indications and Techniques*, pp. 145–153. Menlo Park, CA: Addison-Wesley.

Mitler MM, Carskadon MA, Czeisler CA, Dinges D, Graeber RC, Dement WC (1988). Catastrophes, sleep, and public policy: Consensus report. *Sleep* 11:100–109.

Mitler MM, van den Hoed J, Carskadon MA, Richardson GS, Park R, Guilleminault C, Dement WC (1979). REM sleep episodes during the multiple sleep latency test in narcoleptic patients. *Electroencephalography and Clinical Neurophysiology* 46:479–481.

Monk TH, Fookson JE, Moline ML, Pollack CP (1985). Diurnal variation in mood and performance in a time-isolated environment. *Chronobiology International* 2:185–193.

Moses JM, Johnson LC, Naitoh P, Lubin A (1975). Sleep stage deprivation and total sleep loss: Effects on sleep behavior. *Psychophysiology* 12(2): 141–146.

Pack AI, Pack AM, Rodgman D, Cucchiara A, Dinges DF, Schwab CW (1995). Characteristics of crashes attributed to the driver having fallen asleep. *Accident Analysis and Prevention* 27:769–775.

Peder M, Elomaa E, Johansson G (1986). Increased aggression after rapid eye movement sleep deprivation in Wistar rats is not influenced by reduction of dimensions of enclosure. *Behavioral and Neural Biology* 45(3):287–291.

Petta D, Carskadon MA, Dement WC (1984). Sleep habits in children aged 7–13 years. *Sleep Research* 13:86.

Price VA, Coates TJ, Thoresen CE, Grinstead OA (1978). Prevalence and correlates of poor sleep among adolescents. *American Journal of Diseases of Children* 132:583–586.

Richardson GS, Carskadon MA, Flagg W, van den Hoed J, Dement WC, Mitler MM (1978). Excessive daytime sleepiness in man: Multiple sleep latency measurement in narcoleptic and control subjects. *Electroencephalography and Clinical Neurophysiology* 45:621–627.

Richardson JL, Radziszewska B, Dent CW, Flay BR (1993). Relationship between after-school care of adolescents and substance use, risk taking, depressed mood, and academic achievement. *Pediatrics* 92(1):32–38.

Rosenthal L, Bishop C, Helmus T, Roehrs TA, Brouillard L, Roth T (1995). The frequency of multiple sleep onset REM periods among subjects with no EDS. *Sleep Research* 24:331.

Rosenthal L, Roehrs TA, Rosen A, Roth T (1993). Level of sleepiness and total sleep time following various time in bed conditions. *Sleep* 16(3):226–232.

Roth T, Roehrs TA, Carskadon MA, Dement WC (1994). Daytime sleepiness and alertness. In M. H. Kryger, T. Roth, & W. C. Dement, eds., *Principles and Practice of Sleep Medicine* (2d ed.), pp. 40–49. Philadelphia: W. B. Saunders.

Saarenpaaheikkila OA, Rintahaka PJ, Laippala PJ, Koivikko MJ (1995). Sleep habits and disorders in Finnish school children. *Journal of Sleep Research* 4(3):173–182.

Steinberg L with Brown B, Dornbusch S (1996). *Beyond the Classroom: Why School Reform Has Failed and What Parents Need to Do*. New York: Simon & Schuster.

Strauch I, Dubral I, Struchholz C (1973). Sleep behavior in adolescents in relation to personality variables. In U. J. Jovanovic, ed., *The Nature of Sleep*, pp. 121–123. Stuttgart: Gustav Fischer.

Strauch I, Meier B (1988). Sleep need in adolescents: A longitudinal approach. *Sleep* 11:378–386.

Strogatz SH (1986). *The Mathematical Structure of the Human Sleep-Wake Cycle*. New York: Springer-Verlag.

Tanner JM (1962). *Growth at Adolescence*. Oxford: Blackwell.

Tanz RR, Charrow J (1993). Black clouds – work load, sleep and resident reputation. *American Journal of Diseases of Children* 147(5):579–584.

Van Cauter E, Sturis J, Byrne MM, Blackman JD, Leproult R, Ofek G, L'Hermite-Baleriaux M, Refetoff S, Turek FW, Van Reeth O (1994). Demonstration of rapid light-induced advances and delays of the human circadian clock using hormonal phase markers. *American Journal of Physiology* 166 (Endocrinology and Metabolism 29):E953–E963.

Vogel GW, Minter K, Woolwine B (1986). Effects of chronically administered antidepressant drugs on animal behavior. *Physiology and Behavior* 36(4): 659–666.

Waldhauser F, Steger H (1986). Changes in melatonin secretion with age and pubescence. *Journal of Neural Transmission* 21:183–197.

Webb WB, Agnew HW (1973). *Sleep and Dreams.* Dubuque, Iowa: Wm. C. Brown Company.

(1975). The effects on subsequent sleep of an acute restriction of sleep length. *Psychophysiology* 12:367–370.

Weitzman ED, Nogeire C, Perlow M, Fukushima D, Sassin J, McGregor P, Gallagher T, Hellman L (1974). Effects of a prolonged 3-hour sleep-wakefulness cycle on sleep stages, plasma cortisol, growth hormone and body temperature in man. *Journal of Clinical Endocrinology and Metabolism* 38:1018–1030.

White L, Hahn PM, Mitler MM (1980). Sleep questionnaire in adolescents. *Sleep Research* 9:108.

Williams HL, Hammack JT, Daly RL, Dement WC, Lubin A (1964). Responses to auditory stimulation, sleep loss and the EEG stages of sleep. *Electroencephalography and Clinical Neurophysiology* 16:269–279.

Wilson ME, Lackey S, Chikazawa K, Gordon TP (1993). The amplitude of nocturnal melatonin concentrations is not decreased by oestradiol and does not alter reproductive function in adolescent or adult female rhesus monkeys. *Journal of Endocrinology* 137(2):299–309.

Wolfson A, Carskadon MA (1998). Sleep schedules and daytime functioning in adolescents. *Child Development* 69(4):875–887.

Zepelin H, Hamilton P, Wanzie FJ (1977). Sleep disturbance in early adolescence. *Sleep Research* 6:183.

Zulley J (1980). Distribution of REM sleep in entrained 24 hour and free-running sleep-wake cycles. *Sleep* 2(4):377–389.

Zulley J, Wever R, Aschoff J (1981). The dependence of onset and duration of sleep on the circadian rhythm of rectal temperature. *Pflügers Archiv. European Journal of Physiology* 391:314–318.