Sleep in Adolescents

L Tarokh, Brown University, Providence, RI, USA
M A Carskadon, Brown University, Providence, RI, USA; and E. P. Bradley Hospital, Providence, RI, USA

Sleep behavior and sleep physiology manifest dramatic changes across adolescent development. Certain sleep pattern changes arise from psychological and societal sources, such as a growing sense of independence, increasing access to electronic apparatus in the bedroom, greater academic obligations, and more social opportunities. Some have noted that emergent changes in affect regulation and social affiliation, including 'first love,' can have striking influences on sleep patterns.

Certain hormonal changes that herald and accompany puberty are sleep related, such as the pulsatile sleep-dependent release of luteinizing hormone (LH), which leads to subsequent sleep-related release of sex steroids. Whether these hormonal changes have a direct effect on sleep is unknown; that sleep changes substantially in the pubertal transition is well established. As discussed later, maturation of the central nervous system (CNS) affects the phenomenology of sleep as well as the underlying intrinsic regulatory processes.

In addition to CNS changes, adolescent maturation is associated with new physical attributes and reproductive development. A common method to gauge pubertal development in humans is Tanner staging, which relies on identifying benchmarks in secondary sexual characteristics. Thus, in boys genital development and the growth and distribution of pubic hair are evaluated, and in girls breast development and pubic hair growth and distribution are evaluated. Tanner stage 1 is assigned if no signs of development are identified, Tanner stages 2–4 designate intermediate pubertal stages based on achieving developmental criteria, and Tanner stage 5 requires achievement of all the benchmarks identifying full maturation of secondary sexual characteristics. Note that central signs of puberty (e.g., nocturnal LH release and likely CNS changes) occur earlier than the external physical attributes of puberty.

Adolescent Development and the Brain

The behavioral, emotional, and cognitive changes of adolescence are accompanied by large-scale remodeling of the brain that provides a biological basis for many features of adolescent development. *In vivo* longitudinal studies of the developing human brain using magnetic resonance imaging (MRI) show that gray matter volume increases during preadolescence and declines postadolescence. This inverted U-shape change in gray matter volume corresponds on a cellular level with a marked proliferation of axons/synapses in early puberty and synaptic pruning in later adolescence. Positron emission tomography studies have shown a similar inverted U-shape increase and then decrease in waking cerebral glucose metabolic rates. These cross-sectional studies demonstrate a peak in absolute cerebral glucose metabolism at the age of 9 or 10 years followed by a steady decline until the ages of 16–18 years, at which point levels reach adult values. Not surprisingly, there is a strong correspondence between the developmental curve for blood glucose metabolism, gray matter volume, and synaptogenesis. The combined results from these studies provide strong evidence that adolescence is a time of major neural reorganization.

Adolescent Development and the Waking Electroencephalogram

The changes in gray matter volume, glucose metabolism, and synaptogenesis parallel the decline of electroencephalographic (EEG) power across adolescent development. The EEG signal is thought to arise from synchronous synaptic activity at the cortex; therefore, a reduction in the number of cortical synapses would logically result in dampened EEG power. Although gray matter volume and EEG power decline across adolescence, white matter volume – an index of myelination – increases in a linear manner. Adolescent development is also accompanied by greater functional connectivity between distant cortical regions (i.e., connections between frontal and occipital lobes), as inferred from EEG coherence. Cross-sectional studies of waking EEG coherence show a pattern of increasing inter- and intrahemispheric coherence before and during adolescence.

Along with waking EEG power and coherence changes, the frequency of the dominant posterior rhythm (alpha rhythm) increases as the brain matures. This rhythm is associated with a relaxed waking state and increases in amplitude with eye closure. The dominant posterior rhythm is seen during eye closure in infants as young as a few months old and increases in frequency from approximately 4 Hz in infancy to approximately 8 Hz in mid-childhood, reaching adult frequencies (8–12 Hz) by adolescence.

Adolescent Development and the Sleeping EEG

The EEG is a central measure of sleep, and indeed, EEG patterns are a defining characteristic of sleep in mammals. Typically measured along with electrooculogram and electromyogram, the EEG in humans contributes to defining sleep states (nonrapid eye movement (NREM) sleep and rapid eye movement (REM) sleep) and four sleep stages within NREM sleep. As outlined previously, a significant amount is known about the maturation of the waking EEG. Less is known about adolescent development of the sleeping EEG, although a number of characteristic changes have been identified. As with waking, for example, the age-related decrease in EEG power is strikingly apparent during sleep, particularly in the lower frequencies.

Figure 1 illustrates a number of changes of nocturnal sleep that accompany adolescent maturation by displaying the sleep hypnogram of a representative 10-year-old Tanner stage 1 child and a 15-year-old Tanner stage 5 adolescent. When sleep quantity is held constant at approximately 9 or 10 h, adolescent development is accompanied by a profound decline of approximately 40–50% in the quantity of NREM sleep stages 3 and 4, commonly referred to as slow-wave sleep (SWS). The dampening of EEG amplitude consequent to cortical synaptic pruning may account for the developmental reduction of SWS. At the same time, stage 2 NREM sleep, the definition of which involves EEG features unrelated to amplitude (i.e., sleep spindles and K complexes), increases. Another impressive developmental feature of sleep is the advance of REM sleep to an earlier point in sleep, from a latency of approximately 150–180 min to a latency of approximately 100 min in most studies.

Figure 2 illustrates the marked reduction of EEG amplitude for NREM sleep stages 2 and 4 and REM sleep across adolescence. The sleep EEG amplitudes are notably lower in the more mature adolescent. This EEG amplitude decline appears to be non-state-dependent because it is seen in wakefulness and NREM and REM sleep, although definitive longitudinal assessments have not been carried out. Figure 3 emphasizes this point by displaying the absolute EEG power spectra across the entire night for the pre- and postpubertal youngsters. Warmer colors (designating greater power) in the Tanner stage 1 child versus the Tanner stage 5 child are evident across frequencies and states. The differences are especially clear for low frequencies (1–4 Hz), particularly early in the night.

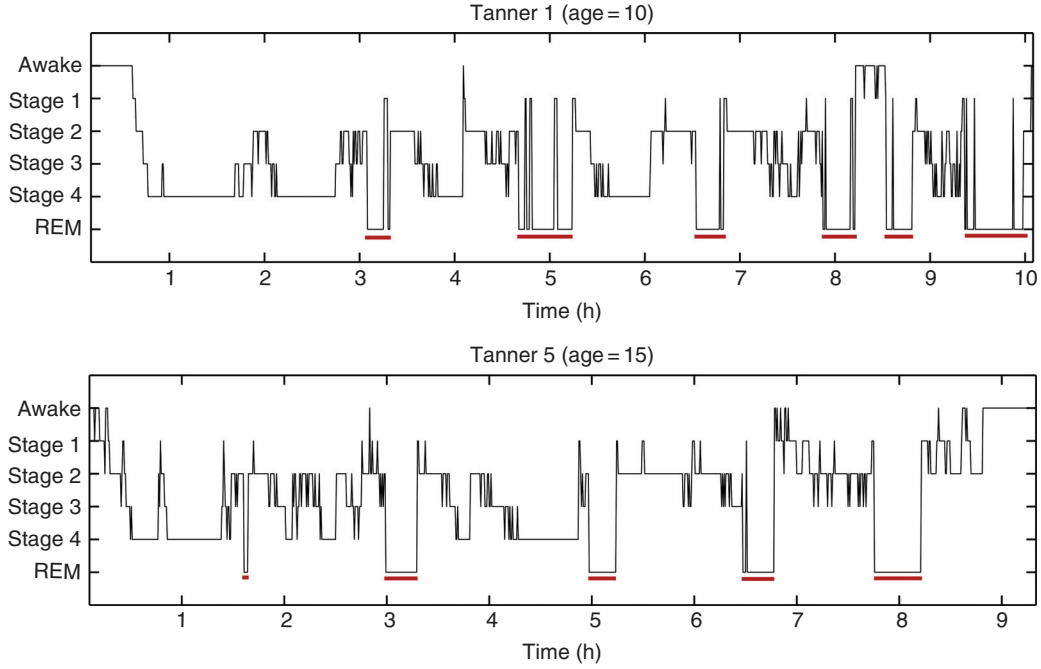

Figure 1 Sleep hypnograms of two boys, one at pubertal Tanner stage 1 (top) and the other at Tanner stage 5 (bottom). The *y*-axis designates stage/state, and the *x*-axis indicates elapsed time from lights out. REM episodes are underlined with a red bar. Stages were determined by visual scoring of 30 s epochs according to standard staging criteria. Significantly greater stage 4 sleep is typical of prepubertal adolescents compared to postpubertal adolescents. The decreased stage 4 sleep in more mature adolescents is accompanied by increased stage 2 sleep. Note also that REM sleep episodes increase in duration over the course of the night. Of note is the delayed onset of the first REM episode in the Tanner 1 preadolescent. Indeed, the first REM episode is often 'skipped' in Tanner 1 preadolescents.

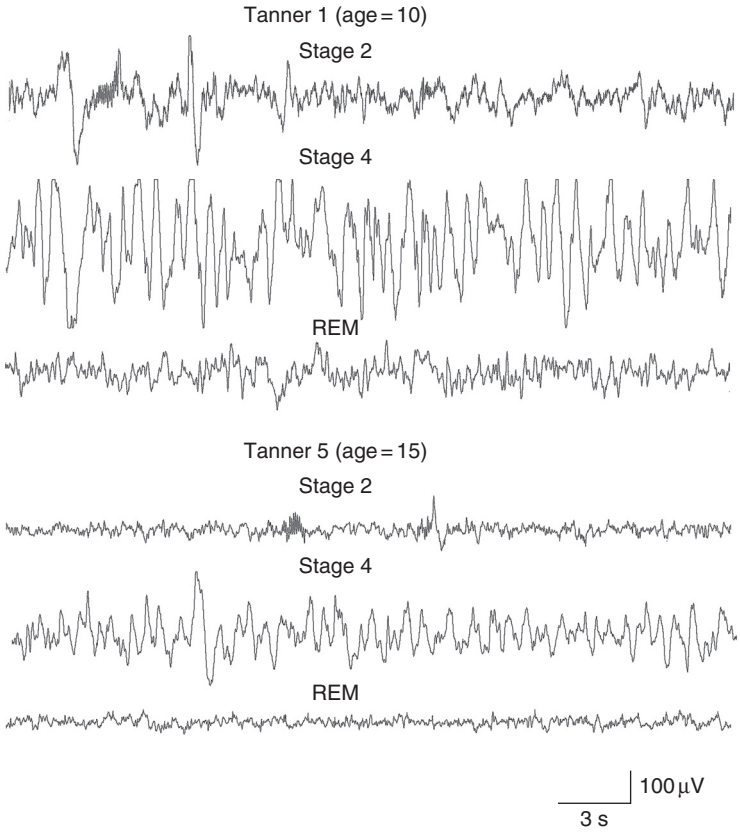

Figure 2 Comparison of Tanner stage 1 (top) and Tanner stage 5 (bottom) EEG tracings of NREM sleep stage 2, stage 4, and REM sleep from the same children and nights as in Figure 1. The calibrations designate the voltage and timescale of all tracings. The diminished EEG amplitude in the more mature adolescent is obvious from these tracings and is most evident in stage 4 sleep; however, this developmental change is not stage specific and occurs in all sleep stages and in the waking EEG. The reduction of EEG amplitude during early adolescence is believed to result from cortical synaptic pruning that occurs during this developmental stage.

This EEG balance of power toward the less mature brain is also clear for the sigma frequencies (approximately 14 Hz). Note that the sigma 'stripe' in these plots is evident only for NREM sleep and disappears during REM episodes.

Sleep Regulation and Adolescent Development

Homeostatic Process (Process S)

In the two-process model of sleep regulation, independent homeostatic (process S) and circadian (process C) processes operate interactively to regulate the amount and timing of sleep. Process S, which can be characterized colloquially as sleep pressure, has been modeled using the NREM sleep EEG slow-wave activity (0.5–4.5 Hz; SWA). As waking is extended, NREM EEG SWA power increases during subsequent sleep and is modeled using a saturating exponential function. With sleep, NREM EEG SWA spectral power manifests an exponential decay across the sleep episode.

The substantial decline in absolute SWA EEG power across adolescence raises the additional question of whether the homeostatic regulatory process is altered and perhaps secondarily accounts for certain expressed sleep behaviors in adolescents. The Brown University group examined this issue in several ways using cross-sectional data sets. First, they examined the dynamics of the decay of SWA across the sleep episode of Tanner stage 1 and Tanner stage 5 adolescents. As Figure 4 illustrates, the exponential fits were overlapping functions, and the mean decay time constant for Tanner stage 1 subjects was 143.2 min (range, 87.0–195.5) and that for Tanner stage 5 subjects was 128.2 min (range, 80.4–176.9). This finding indicates that the nocturnal recovery process is similar for adolescents across this developmental phase, with an implication that neither sleep intensity nor sleep need change with adolescence.

On the other hand, when the accumulating function of process S is examined in the context of extended wakefulness, the data indicate the presence of a maturational change. One approach examined sleep pressure by assessing the speed of falling asleep

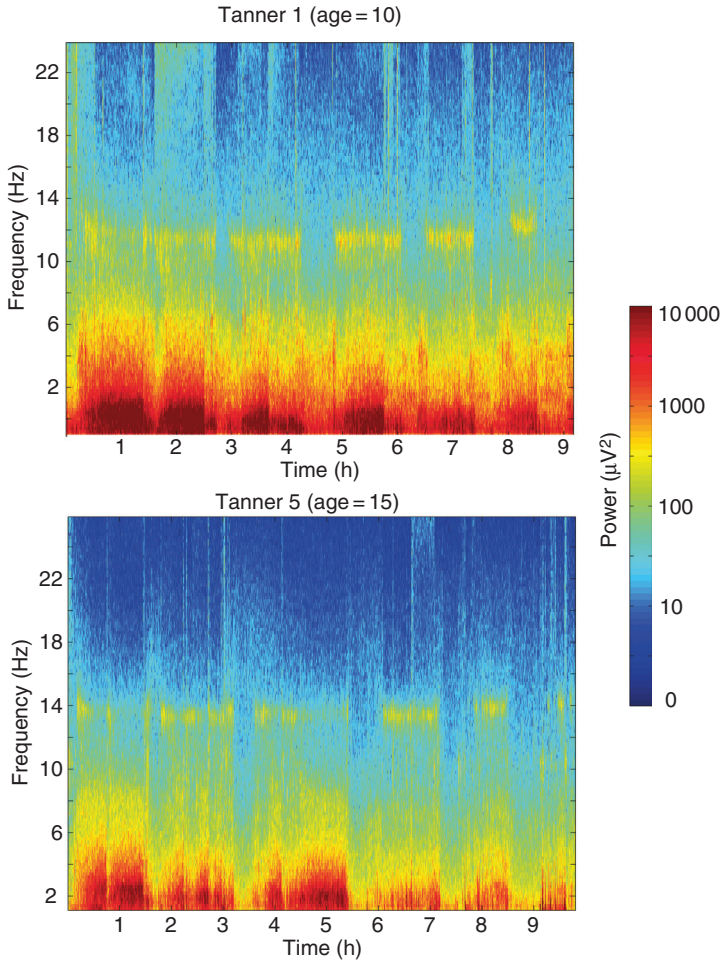

Figure 3 Time–frequency plot of the same nights and children depicted in the hypnograms displayed in Figure 1. The figure is derived from fast Fourier transform analysis of the C3/A2 EEG signal. The plots depict how the frequency content of the EEG signal changes over the course of the night. The *y*-axis is the frequency and the *x*-axis shows hours since lights out. EEG power is indicated by the color, in which warmer colors (reds and yellows) indicate higher power and cooler colors (aqua and blue) indicate lower power. During NREM sleep, the power of the signal at frequencies below 4 Hz is maximal, particularly early in the night, ebbing in strength across the sleep episode. Note also that the power in these slow frequencies is markedly greater for the younger boy. NREM episodes are also marked by high power in the spindle frequency range (12–16 Hz). In contrast, during REM episodes, the power in the lower frequency and spindle bins is diminished. The characteristic cycle-by-cycle nature of the sleep EEG is apparent in these plots. Overall EEG power differences between the Tanner 1 and Tanner 5 participants are readily apparent at all frequency bins.

in adolescents at Tanner stage 1 and Tanner stage 5. This study found that after approximately 14.5, 16.5, and 18.5 h awake, the less mature youngsters fell asleep significantly faster than the more mature group. A second approach modeled process S using SWA in a sleep deprivation study and found fitted functions for the exponential saturating function with a time constant of 15.4 ± 2.5 h for the mature Tanner stage 5 adolescents compared to 8.9 ± 1.2 h for a group of pre-/early pubertal (Tanner stages 1/2) children. These two findings indicate that the more mature adolescents, although they exhibit the same sleep recovery pattern, may find it easier to remain awake longer than the less mature adolescents. This developmental alteration may contribute to the later timing of sleep that emerges across adolescence.

Circadian Timing (Process C)

Maturational changes in the circadian timing system (CTS) also appear to contribute to the behavioral changes in adolescent sleep patterns. The master 'clock' in mammals has been localized to a small paired nucleus in the hypothalamus, the suprachiasmatic nuclei (SCN). A direct pathway from the retina, particularly the light-sensitive melanopsin-containing retinal ganglion cells, carries light input directly to the SCN, where the light signals affect the molecular machinery of the CTS within SCN neurons.

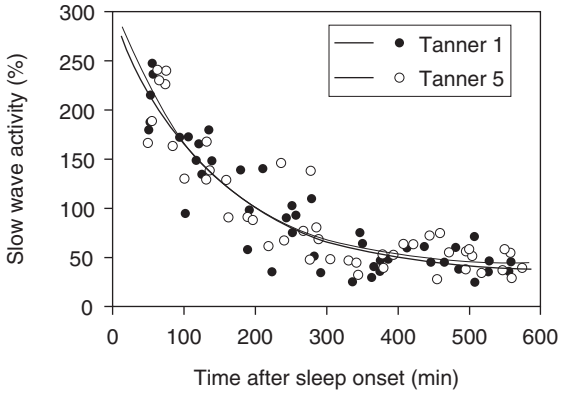

Figure 4 Dynamics of slow-wave activity (SWA; 0.6–4.6 Hz) across the sleep episode in eight prepubertal (solid circles) and eight mature (open circles) adolescents. SWA power for each NREM sleep cycle is shown as a percentage of the individual child's average overnight NREM sleep, and each symbol represents this value plotted at the midpoint of the time relative to the onset of sleep. The lines show exponential functions fitted to the Tanner 1 (thick line) and Tanner 5 (thin line) data. (Both fits achieved r^2 values of 0.83.) These data indicate that the dissipation of SWA, and inferentially of process S, does not change with pubertal development, although the power of the EEG SWA signal is markedly changed as shown in **Figure 3**. Reproduced from Jenni OG and Carskadon MA (2004) Spectral analysis of the sleep electroencephalogram during adolescence. *Sleep* 27: 774–783.

One of the first findings implicating the CTS in the adolescent delay of sleep timing was an association between self-reports of phase preference (e.g., morningness vs. eveningness) and puberty in sixth-grade girls: Girls who rated themselves more mature also rated themselves with greater evening-type phase preference. Subsequent studies examined these phenomena using better measures, such as Tanner staging and circadian phase measured using the dim light melatonin onset (DLMO) phase. DLMO is a reliable and noninvasive assessment of circadian phase acquired from serial plasma or saliva samples collected under dim light (less than approximately 40 lux) conditions. Because melatonin levels are low during the biological daytime and rise during the biological night, the circadian phase can be measured by assessing the onset phase of melatonin release. In adolescents, saliva is collected approximately every 30 min in dimly lit conditions during the participants' usual evening hours.

A major feature of the CTS is its sensitivity to environmental light. Effects of light on the CTS are phase dependent; to wit, light impinging on the clock in the early nighttime produces phase delays (later timing) and light occurring in the early morning produces phase advances (earlier timing). Because of this phase-dependent light sensitivity, mature adolescents living in their normal circumstances will inevitably express later circadian phase positions because their delayed sleep pattern exposes them to light at a later time. Therefore, to determine whether the internal process undergoes developmental change, light exposure needs to be controlled, for example, by controlling sleep (dark) schedules. In such controlled circumstances, the DLMO phase is positively correlated with Tanner stage, whereby more mature adolescents have a later DLMO phase. This circadian phase delay across adolescence may affect the ability of older adolescents to fall asleep easily in the evening since the CTS favors wakefulness in the early circadian night, sometimes called the forbidden zone for sleep. In addition to humans, a pubertal delay of sleep behavior has been reported in rhesus monkeys and in the octodon degus, a small diurnal mammal with a relatively protracted developmental course from birth to reproductive maturity.

Attempts have been made to identify the underlying mechanism for this adolescent phase delay. One hypothesis holds that the intrinsic period of the circadian timing system may slow from prepubertal to postpubertal adolescence, thus delaying circadian phase. Studies that permit assessment of internal circadian period are both difficult and time-consuming and have not established the validity of this hypothesis. One study, however, has shown that the period is longer on average in adolescents than in adults. Another theory for the developmental change in circadian phase posits that the phase-dependent sensitivity to light's phase shifting effects may favor phase delays either through strengthening the delay response to evening light or through weakening the phase advance to morning light. Attempts to demonstrate this phenomenon have not been successful to date, although none has used the most circadian-sensitive shortwave (blue) light sources to examine the possibility of a maturational change in light sensitivity.

Thus, the circadian timing system does manifest a phase delay associated with pubertal/adolescent development; however, the mechanism for the delay is unknown. Unlike the sleep system in which the adolescent decline in EEG amplitude can be attributed directly to cortical synaptic pruning, no molecular, cellular, or systems basis has been identified to explain the maturation of the CTS across adolescence.

Interaction of Process S and Process C

As noted previously, the circadian timing system favors waking in the biological evening. Indeed, the strength of the arousing signal from the circadian timing system increases from the usual time of morning arousal until about the time of usual bedtime and

decreases across the typical nighttime window. On the other hand, the homeostatic sleep–wake process builds as waking is extended, only to dissipate during sleep. Either process on its own is untenable for supporting our usual sleeping and waking patterns; however, the interaction provides an arousing circadian signal later in the day to support alertness when the homeostatic pressure would otherwise favor sleep. Conversely, the circadian arousing signal wanes during the night. As morning comes, with circadian arousal at its nadir, wakefulness is underwritten by the restorative function of sleep.

In the case of adolescent development, this interaction plays out in several important ways. When daytime alertness is evaluated in children keeping an optimizing sleep schedule (10 h per night from 10:00 p.m. to 8:00 a.m.), the interaction of processes S and C manifests as maximal alertness from rising until bedtime. With the onset of puberty, however, the pattern shifts to one that augments midday sleepiness. In the experimental paradigm that showed this phenomenon, young people were followed annually across several years with summertime assessments on the optimized sleep schedule and sleepiness measured with the Multiple Sleep Latency Test (MSLT), a standardized measure of the speed of falling asleep (sleep propensity) at 2 h intervals across the day. In prepuberty, the youngsters maintained alertness near the ceiling of this measure throughout the day; at midpuberty, the pattern changed to exhibit faster sleep onsets on tests given in the middle of the afternoon. Subsequent evaluation of these data indicated that the phase delay of pubertal development resulted in a reorganization of the phase relationship between sleep and the circadian timing system. With this reorganization, the circadian buffering of accumulating sleep pressure across the waking day did not occur until a later phase, and thus midday sleepiness emerged.

In normal circumstances, however, adolescents do not live with optimal sleep. As outlined below, sleep occurs later and is shorter in older adolescents. This pattern results in chronic insufficient sleep on school nights that is not recuperated fully by nighttime sleep. In these circumstances, morning alertness specifically is impaired since adolescents wake with elevated homeostatic pressure and also at a circadian phase that does not support alertness.

One study examined the consequences of this mismatch in students attending the 10th grade of high school in the United States. These students were required to attend school beginning at 7:20 a.m. When followed on their normal sleep schedule for 2 weeks, the students obtained approximately 7.5 h of sleep each night, going to bed on average at approximately 10:30 p.m. and waking at approximately 6:00 a.m. When their alertness was measured using the MSLT, these adolescents were sleepiest at 8:30 a.m. (average speed of falling asleep was 5 min), and alertness improved in the late morning and early afternoon, presumably due to the alerting effects of the circadian timing system. Thus, the speed of falling asleep at 2:30 p.m. was 11 min. Of greater note in this study, however, was the appearance of an abnormal pattern of sleep in nearly 50% of the young people on the MSLTs: They had at least one test (usually at 8:30 a.m.) in which they fell directly into REM sleep rather than NREM sleep (i.e., sleep-onset REM episodes). Furthermore, the speed of falling asleep in this group was even faster at 8:30 a.m. – 3.4 min on average. A distinguishing feature of this group was that the phase of DLMO was later, and the investigators concluded that they woke at a much earlier circadian phase than the other students, at a time favoring sleeping over waking and REM sleep over NREM sleep. The MSLT results in these young people looked more like those of patients with a serious sleep disorder – narcolepsy – than healthy teens.

Sleep Habits and Adolescent Development

As mentioned previously, sleep patterns undergo major alterations across adolescent development – changes that have been confirmed in many surveys of young people on every continent (except Antarctica). In each case, the timing of sleep is later overall in older adolescents. In societal circumstances requiring early morning awakening – for example, with early starting time for school as found in many communities in the United States – sleep quantity also is reduced, at least on school days. Indeed, the difference between school day and weekend schedules becomes greater during adolescence. At age 9 or 10 years, for example, children may delay weekend bedtime slightly but tend not to delay or extend sleep as much on weekend mornings. By age 14 or 15 years, on the other hand, bedtime is much later on school nights, delayed by 1–3 h on weekends, and weekend sleep can be several hours longer than on school days. These weekday–weekend differences express both the pressure for sleep to delay during adolescence and the compensatory need for sleep on weekends due to insufficient sleep on school nights in the older adolescents.

The 2006 Sleep in America poll of the National Sleep Foundation surveyed approximately 1600 adolescents and caregivers. Data from this poll provide a contemporary perspective of adolescent sleep. Although this article has emphasized the biological bases of sleep pattern alterations across adolescence,

the influence of environmental factors is undeniable. Furthermore, the interactions among sleep regulatory changes and social/environmental influences and constraints can lead to troubling outcomes in susceptible youngsters. As the Sleep Foundation poll data show, school night sleep time reported by sixth-grade students in the United States is approximately 8.4 h and it is only 6.9 h in 12th-grade students, which by general consensus is significantly below the sleep need of teens.

The starting time for school plays an important role in setting the stage for short sleep in many adolescents. Thus, although teens' bedtimes delay significantly across adolescence, rising time on school days does not delay and indeed may actually become earlier (Figure 5). Later bedtimes fit well with the intrinsic maturational changes; earlier rising times, however, do not arise from a natural tendency on the part of adolescents but in direct response to waking in time for school. The result is truncated sleep time. In cases in which school begins later in the day, as shown by a number of ongoing studies of students in Brazil, Croatia, and the United States, teens sleep later and longer. One US study that examined sleep patterns of high school students before and after imposition of a later school schedule found that bedtimes did not change significantly; however, rising times were later and, consequently, youngsters slept more.

A newly emerging consensus also credits access to contemporary technologies as contributing to adolescent sleep patterns. For example, Belgian adolescents who report using electronic media to help them fall asleep in fact sleep less than other teens and report themselves as more tired. Furthermore, the National Sleep Foundation's 2006 Sleep in America Poll found

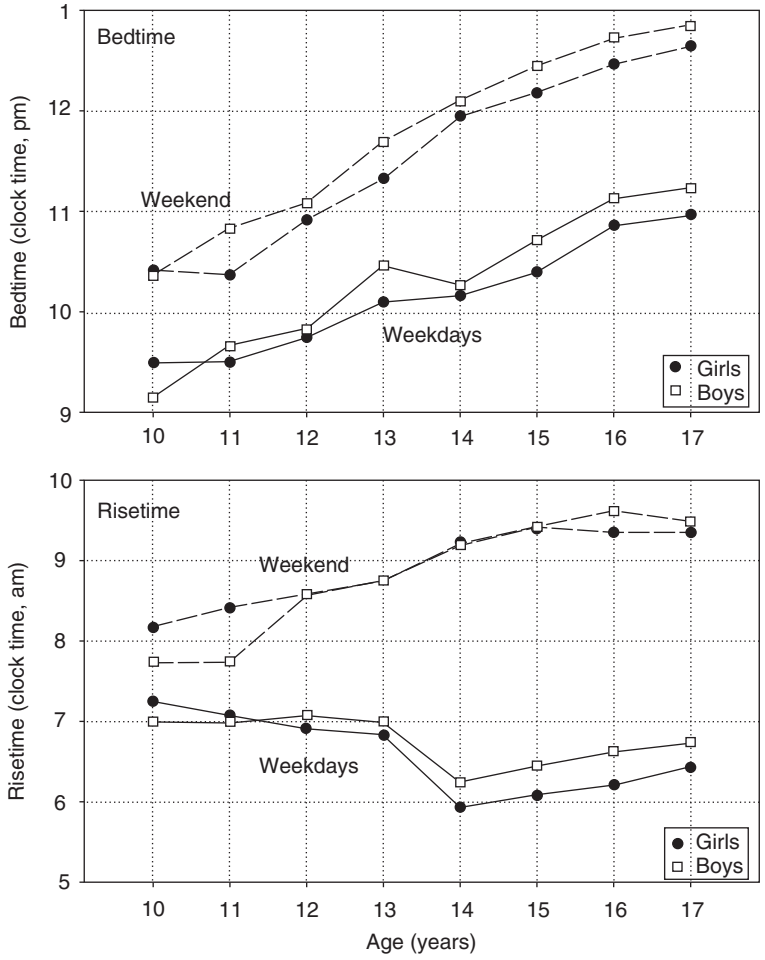

Figure 5 Adolescent self-reported sleep patterns taken from surveys performed in the 1990s with children living in the United States. Girls are represented by closed symbols and boys by solid symbols. Weekend bedtimes (top) and rise times (bottom) are shown with dotted lines; weekday (school day) schedules are indicated by solid lines. The sample included children ages 10–17 years who were enrolled in school at the time of the survey. Times on the *y*-axes are indicated using 24 h clock hours. Reproduced from Jenni OJ and Carskadon MA (2005) Infants to adolescents. In: *SRS Basics of Sleep Guide*, pp. 11–20. Westchester, IL: Sleep Research Society.

that compared to younger teens, older teens have increased numbers of technological devices (e.g., telephone, instant messaging, MP3 player, electronic games, television, computer, and Internet connection) accessible to them in their bedrooms. In addition, teens who reported having four or more such devices in the bedroom also reported sleeping 30 min less on average each night than those with fewer devices.

Consequences of Chronic Insufficient and Ill-Timed Sleep in Adolescents

The association of maturational changes of the intrinsic regulatory systems with the seductive nocturnal lifestyle options available to teenagers makes for a synergistic drive toward later timing of sleeping and waking for most teens. When confronted with the inevitability of an early school bell, a pattern emerges that comprises late, short, and irregular sleep. As noted previously, for some teens the combination results in extreme morning sleepiness and sleep-onset REM episodes. For others, the combination can be deadly. For example, in car crashes attributed to the driver having fallen asleep, the age of the driver is 16–25 years in more than 50% of cases.

Consequences of insufficient, irregular, and ill-timed sleep also include potential problems with mood regulation and depression, learning and memory, weight regulation, substance use or abuse, school tardiness and absenteeism, along with impulsivity and risk taking. Many of these behavioral tendencies are thought to be endemic problems of adolescence, yet the contributions of poor sleep may be involved. Serious consideration and scientific examination of these issues is needed. Trends point to continued deterioration of adolescent sleep patterns and at younger ages. Thus, whether adolescent sleep may affect the maturation of neurons, neural circuitry, or neurochemistry is in urgent need of study.

See also: Circadian Oscillations in the Suprachiasmatic Nucleus; Circadian Regulation by the Suprachiasmatic Nucleus; Circadian Genes and the Sleep–Wake Cycle; Circadian Function and Therapeutic Potential of Melatonin in Humans; Circadian Rhythms: Influence of Light in Humans; Circadian Rhythms in Sleepiness, Alertness, and Performance; Endocrine Function During Sleep and Sleep Deprivation; Entrainment of Circadian Rhythms by Light; Narcolepsy; Photoreceptors and Circadian Clocks; Sleep and Sleep States: Phylogeny and Ontogeny; Sleep Architecture; Sleep and Sleep States: PET Activation Patterns; Sleep Deprivation: Neurobehavioral Changes; Sleep Deprivation and Brain Function; Sleep: Development and Circadian Control; Sleep-Dependent Memory Processing.

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