# Assessing Sleep in Adolescents Through a Better Understanding of Sleep Physiology 

## Adolescent sleep is complicated by intrinsic as well as external factors.


#### Abstract

OVERVIEW: Adolescents need about nine hours of sleep per night, yet most teens get far less. Inadequate sleep has consequences not only for academic performance but also for mental and physical health; it has been linked to lowered resilience and an increased risk of cardiovascular and metabolic diseases. It's imperative that assessment of sleep become a routine part of adolescent health care. An understanding of sleep physiology is essential to helping nurses better assess and manage sleep deprivation in this population. Sleep assessment involves evaluating the three main aspects of sleep: amount, quality, and architecture. The authors provide an overview of sleep physiology, describe sleep changes that occur during adolescence, and discuss the influence of these changes on adolescent health. They also provide simple questions that nurses can use to assess sleep and risk factors for disrupted sleep, and discuss patient education and other interventions.


Keywords: adolescent, sleep architecture, sleep assessment, sleep hygiene, sleep physiology

Whether American teens get adequate sleep has been a longstanding concern of many clinicians and parents, and rightly so. As early as 1975, writing in the Bulletin of the Psychonomic Society, Webb and Agnew cited research that found that on average, young people between the ages of eight and 17 were getting 1.5 hours less sleep per night than their counterparts did in 1910 and 1911. ${ }^{1}$ Since then, the literature indicates that some degree of disrupted or deficient sleep has become standard for adolescents in this country. ${ }^{2,3}$ According to the National Sleep Foundation, teens need between 8.5 and 9.25 hours of sleep per night, ${ }^{4}$ with some laboratory data indicating that 9.2 hours is optimal ${ }^{3}$; thus sleep deprivation can be defined as anything under about nine hours per night. Yet experts
generally agree that most adolescents get less. After analyzing data from a recent national survey of high school students by the Centers for Disease Control and Prevention, McKnight-Eily and colleagues found that nearly $70 \%$ reported getting less than eight hours of sleep on school nights. ${ }^{5}$ They agreed with other experts that chronic sleep deprivation in adolescents stems from "social, employment, recreational, and academic pressures as well as biologic changes" in the sleep-wake cycle., ${ }^{5,6}$

Contrary to what some believe, sleep needs do not markedly decline after puberty, perhaps because of the increased metabolic expenditures associated with growth during adolescence. ${ }^{7}$ Yet one study of American high school students found that the average amount of sleep reported on school nights declined

from about 7.7 hours for younger students to about seven hours for older ones. ${ }^{3}$ The same study found that $26 \%$ reported that they "usually" sleep less than 6.5 hours per night. ${ }^{3}$ To further complicate matters, most middle and high school students start school earlier in the day than do elementary school children. Not surprisingly, in surveys, $13 \%$ to $24 \%$ of adolescents have reported falling asleep in class at least once. ${ }^{3,8}$ Although one recent study concluded that U.S. children and adolescents do get between nine and 10 hours per night, ${ }^{\text {a }}$ an accompanying editorial cautioned, "We are still far from understanding what a 'normal,' 'ideal,' or 'adequate' sleep duration is for an individual child to promote optimal health and functioning." ${ }^{10}$

Sleep and health. Sleep is, of course, essential to well-being; it is a complex phenomenon with biophysiological, behavioral, and environmental components. Disruptions or alterations in the amount, quality, or architecture of sleep can lead to lowered resilience and an increased risk of disease. Indeed, such alterations have been linked to numerous health concerns, including aggression, ${ }^{11}$ anxiety, ${ }^{12}$ seizures, ${ }^{13,14}$ hypertension, ${ }^{15,16}$ obesity, ${ }^{15}$ and stress, ${ }^{17}$ as well as increased risk of cardiovascular disease, diabetes, and depression. ${ }^{18}$ Although the relationship between compromised neurobehavioral functioning and insufficient or disrupted sleep has been well studied in adults, relevant literature on children and adolescents is limited.

That said, sleep disruptions in adolescents have also been associated with memory and attention deficits and declines in academic performance. ${ }^{3,19}$ McKnightEily and colleagues found that insufficient sleep was associated with many high-risk behaviors; for example, sleep-deprived teens were more likely to engage in physical fighting; seriously consider suicide; and use cigarettes, alcohol, and marijuana. ${ }^{5}$

We found little in the health literature that addressed the connections between sleep physiology and adolescent health and health assessment. In this article, we provide an overview of sleep physiology, describe sleep changes that occur during adolescence, and discuss their influence on adolescent health. We also provide simple questions that nurses can use to assess sleep and risk factors for disrupted sleep in adolescent patients, and discuss patient education and other interventions.

## OVERVIEW OF SLEEP PHYSIOLOGY

Sleep states. The architecture of sleep can be broken down into two main sleep states: rapid eye movement (REM) sleep and non-rapid eye movement (NREM) sleep. (For a glossary of selected sleep terms, see Table 1.) Differences between these two states involve distinct brain wave patterns, the presence or absence of eye movement, and varying degrees of muscle tone. Polysomnography, a diagnostic test that records

## Stage 1





REM Sleep

Figure 1. Examples of waveforms for the three stages of non-rapid eye movement sleep and for rapid eye movement sleep.
physiological responses during sleep, is used to elucidate the two sleep states. Sleep architecture can be further described via recordings of electroencephalographic (EEG) patterns, electrooculographic eye movements, and electromyographic chin muscle activity.

There are three stages of NREM sleep, which differ in terms of the depth of sleep. The first stage, the transition between wakefulness and sleep, is known as stage 1 NREM sleep. This is the lightest stage of sleep, and the sleeper is easily awakened. In stage 2 NREM sleep, the sleeper exhibits frequent bursts of low-voltage, fast-frequency EEG waveforms (sleep spindles) and high-voltage, slow-frequency EEG spikes (K-complexes). ${ }^{7}$ In stage 3 NREM (also called delta or slow-wave) sleep, the sleeper exhibits "more or less continuous high-voltage activity in the slowest $(<2 \mathrm{~Hz})$ frequency range." ${ }^{7}$ (Older sources sometimes refer to stages 3 and 4, but since 2007 these have been combined. ${ }^{20}$ ) (For an example of wave forms for the stages of sleep, see Figure 1.) During all three stages, eye movements are relatively few or absent and the chin muscles show slight to moderate activity. As NREM sleep is dominated by the parasympathetic nervous
system, heart and respiratory rates tend to be slow and regular.

After moving through the three stages of NREM sleep, the sleeper enters REM sleep. REM sleep is characterized by desynchronized EEG activity featuring relatively low voltage and mixed frequencies (also known as paradoxical sleep), bursts of rapid eye movements, and absence of muscle tone. ${ }^{7}$ As REM sleep is dominated by the sympathetic nervous system, heart and respiratory rates tend to be fast and irregular. For a person to be fully rested, it's necessary to cycle through all of these stages several times during the sleep period. The specific functions of each sleep stage are still not well understood.

Regulatory systems. Two systems regulate sleep: the circadian timing system or internal "clock" and the sleep-wake homeostatic system. ${ }^{2,7,21}$ The circadian system regulates events that occur on a roughly 24 hour cycle, including periods of wakefulness and sleep. The homeostatic system strives to balance the needs for sleep and wakefulness, doing so through various mechanisms that prompt an individual to compensate for a lack or surplus of sleep. The two systems interact
to determine the timing, depth, and duration of sleep, although how they do so is not fully understood. ${ }^{2,7}$

Adolescent sleep. During normal development, sleep duration decreases from infancy (mean, 14 hours per day, unconsolidated) through the first 10 years of life and levels off in adolescence (about nine hours' consolidated sleep needed). ${ }^{3,7}$ Several other changes to sleep architecture also occur, of which the most noteworthy are changing proportions of NREM and REM sleep and a delay in sleep phase.

Changing proportions of NREM to REM sleep. In the first few months of life, sleep is divided about equally between NREM and REM sleep; thereafter the proportions change. ${ }^{7}$ Compared with younger children, adolescents spend increasingly more time in stage 2 NREM sleep and less time in stage 3 NREM sleep. Between the ages of 12 and 14 , the amount of time spent in stage 3 NREM sleep drops $40 \%$ to $60 \%$ and continues to decline, albeit more gradually, throughout life. ${ }^{7,8}$ Time spent in REM sleep decreases throughout childhood until adolescence, when it levels off at about $20 \%$ to $25 \%$ of total sleep. ${ }^{7}$

Each full cycle through the sleep stages lasts about 50 minutes in infants and lengthens gradually to a "mature period length" of 90 to 110 minutes by the time a child starts school. ${ }^{7}$ Adolescents typically cycle five to six times per night. ${ }^{7}$ NREM sleep, particularly stage 3 sleep, predominates in the first half of the total sleep period, with periods of REM sleep increasing in frequency and duration during the second half. ${ }^{21}$ Thus, for adolescents whose total sleep time is shortened, one likely consequence is insufficient REM sleep.

The less REM sleep one gets, the greater the degree of REM rebound; that is, the first REM sleep stage occurs earlier in the sleep cycle, and the sleeper spends a higher percentage of time in REM sleep, with REM periods lasting longer and occurring more frequently with an increase in REM density. Some have argued that short REM sleep latencies indicate a misalignment of the two regulatory systems-homeostatic and circadian. ${ }^{7}$

Delayed sleep phase. The circadian timing system stabilizes at around six months of age and remains relatively unchanged until puberty. ${ }^{2}$ The onset of puberty is associated with a natural delay in sleep phase: adolescents tend to stay up later at night and to sleep later in the morning than do younger children. ${ }^{3,7}$ Some experts posit that this delay may be explained by three possible mechanisms: intrinsic circadian changes, pubertal hormonal changes, and increased light sensitivity. ${ }^{2,7}$ For example, pubertal hormonal changes affect the hypothalamic-pituitary-adrenal axis, a major component in energy regulation and stress response, which in turn can affect sleep. And there is some evidence that adolescents experience increased sensitivity to evening light. ${ }^{2}$

The sleep-wake pattern is also affected by external factors such as early school start times, resulting
in insufficient sleep during the school week and the use of weekends to catch up on sleep. ${ }^{6,7}$ It can be further complicated for adolescents who work, have curfews, or must abide by parentally set bedtimes that are contrary to their circadian rhythm. And there is much evidence that sleep in children and adolescents is vulnerable to psychosocial stressors such as natural disasters and child abuse. ${ }^{22}$

Genetics and sleep. Although sleep genetics is an emerging field, it's evident that sleep is a genetically complex phenomenon. ${ }^{23}$ The genetic basis of sleep has been studied extensively in insects and small mammals but not in humans. Most of the genetic studies in humans have been conducted in twins. ${ }^{24}$ The most heritable factors identified in humans thus far are the distribution of gray matter in the cerebral cortex and EEG activity. ${ }^{24}$

One recent study, conducted among more than 4,000 Europeans, reportedly found evidence linking the $A B C C 9$ gene to regulation of sleep duration. ${ }^{25}$ (It's worth noting that the same gene has been associated with heart disease and diabetes.) In a smaller human study, Retey and colleagues found that functional variants of the $A D A$ gene were associated with individual differences in sleep architecture and EEG activity; people with one genotype had longer stage 3 NREM sleep periods and slept more deeply than people with another genotype. ${ }^{26}$

Although genetic factors have been identified in several sleep disorders, most of these disorders have complex causes. ${ }^{23}$ Toh and colleagues found that a behavioral trait known as familial advanced sleep phase syndrome (FASPS) -characterized by "a striking fourhour advance" of the sleep-wake pattern-was associated with an alteration in hPer 2 , a gene involved in regulating circadian rhythms. ${ }^{27}$ But other researchers have noted that most cases of FASPS are not caused by hPer 2 mutations, which suggests an underlying "genetic heterogeneity" involving other circadian-related genes. ${ }^{23}$ Narcolepsy cases are rarely familial, so genetic

Table 1. Glossary of Selected Terms

| Sleep architecture | The structure of sleep, in particular the cyclical <br> pattern of NREM and REM sleep stages |
| :--- | :--- |
| Sleep duration | Total time spent asleep |
| Sleep efficiency | Percentage of time spent sleeping based on <br> total time in bed |
| Sleep latency | Length of time it takes to fall asleep after lights out <br> Circadian rhythm |
| Biologic changes that follow an approximate <br> 24-hour cycle and occur primarily in response to <br> light and darkness in an organism's environment |  |
| Homeostatic | Regulatory mechanisms that are involved in <br> maintenance of a constant environment |

REM = rapid eye movement; NREM = non-rapid eye movement.

## Assessing for Risk Factors for Disrupted or Insufficient Sleep

## Questions to ask might include the following.

- Do you work?
- What are your work hours?
- How late at night do you use the computer?
- Is the computer in your bedroom?
- Is the computer left on with the screen lit up at night?
- Do you use an e-reader at night? Does it have a backlight?
- Do you have a television in your bedroom?
- How late at night do you watch television?
- Is the television left on at night?
- Do you fall asleep while watching television?
- What are your bedtime habits?
- How dark is your room at night when you go to bed?
-When do you usually do your homework?
- How often do you exercise in a given week?
- How much alcohol, caffeine (coffee, tea, "energy" drinks), and nicotine do you normally consume?
- What are your habits when you awaken?
susceptibility probably plays a minor role; but up to $80 \%$ of cases of parasomnias (sleepwalking and night terrors) have been found to be familial in nature. ${ }^{23}$ Sleep apnea syndrome cases are often familial, and many of the risk factors (such as body fat distribution and metabolism resulting in upper-body obesity) are largely genetically determined. ${ }^{23}$ In short, as the body of knowledge grows, sleep genetics will help us to better understand the molecular basis of sleep and sleep disorders; but much more research is needed.


## ASSESSMENT OF ADOLESCENT SLEEP

High-quality sleep is characterized by effortless transitions between wakefulness and sleep as well as between the sleep stages, and there should be no interruption of sleep. Assessment of sleep involves assessment of its three main aspects-amount, quality, and architecture-and of sleep behaviors. Such assessment can help clinicians to develop a better picture of the degree to which sleep may be disturbed or insufficient, and to how this might be affecting an adolescent's health.

There are several questionnaires that have been used in research to assess sleep quality. The School Sleep Habits Survey ${ }^{3}$ (http://bit.ly/YW6opq), the Sleep-Wake Diary, ${ }^{28}$ and the Pittsburgh Sleep Quality Index ${ }^{29}$ are among the research tools used to assess adolescent sleep. These tools have been found to have adequate reliability and validity, but can be cumbersome to use in clinical practice; some are lengthy and require substantial time to administer, while others have complicated scoring methods. The Epworth Sleepiness Scale (http://bit.ly/LfbMlM) can be used to determine the average level of sleepiness a person
feels in daily life. Research indicates that it has adequate reliability and validity, ${ }^{30,31}$ and with only eight items, it's easy to use in clinical situations.

An assessment of the three main aspects of sleep can be done fairly easily through a series of direct questions. We have developed several bulleted lists as guides. Assessment of the amount of sleep involves asking the following questions:

- What time do you usually feel tired?
- When do you actually go to bed?
- How long does it take you to fall asleep?
- What time do you awaken on school days?
- What time would you get up if left to awaken on your own?
- How many hours of sleep do you get on most nights of the week?
Assessment of the architecture of sleep involves asking questions about sleep patterns, including:
- Do you have a set bedtime?
- During the school week, do you sleep more or less than you do on weekends?
- On weekends, do you sleep more or less than you do during the school week?
- What activities do you participate in and when are they scheduled?
- Do your activities change the time you awaken or your bedtime?
Assessment of the quality of sleep involves asking the following questions:
- Do you ever have problems falling asleep? How many times in the last month have you had such problems?
- How often do you wake up in the middle of the night or early morning?
- How often do you get up to use the bathroom, answer a text message, or answer your cell phone after you've gone to bed?
- How would you rate your overall sleep quality on a scale of 1 to 5 , with 1 being "very good" and 5 being "very bad"?
Assessment of risk factors for disrupted or insufficient sleep involves assessing activities, habits, and environmental elements that can influence sleep. These include work and exercise habits, exposure to and use of electronic screens, and alcohol and caffeine use, among others. (See Assessing for Risk Factors for Disrupted or Insufficient Sleep.)

Finally, since there is evidence that many sleep disorders have a genetic component, ${ }^{23}$ attention should be paid to any family history of such disorders.

A thorough understanding of adolescent sleep physiology will allow the clinician to better gauge the implications of the patient's responses. This knowledge then allows for more appropriate interventions.

## INTERVENTIONS IN BRIEF

By addressing poor sleep in adolescents, nurses can potentially increase their physiological resilience and
lower their risk of cardiovascular and metabolic diseases, among others. First, it's important for adolescents to understand the need to establish good sleep habits. Nurses can help foster such habits by providing education to adolescent patients and their families. Adolescents must learn how to unwind from the day's activities, go to sleep and wake up at the same time each day, and get the minimum recommended hours of sleep per night. Establishing nighttime rituals such as listening to calming music, meditating, and laying out clothes for the next day can help in making the transition to sleep. Given that adolescents may be more sensitive to evening light, it's essential to ensure that the room is dark and quiet and that no electronic devices (computers, cell phones, televisions) are left on with their screens shining light. Adolescents should also avoid activities that might disturb or impede sleep, such as using alcohol, caffeine, or nicotine; studying just before bedtime; and engaging in arguments. And they may benefit from opening window blinds or curtains immediately upon awakening and from "catch-up" sleep on weekends. (However, advancing weekend bedtimes and rise times by more than two to three hours can dysregulate the circadian cycle. ${ }^{2}$ )

If the assessment yields responses that are of concern to the clinician-for example, if the adolescent indicates having no set bedtime, frequently awakening during the night, and often getting up to answer text messages-the next step might be to have her or him keep a Sleep-Wake Diary, which will help in eliciting more detailed information. Instructions and a sample form are free online from Bradley Hospital/ Brown University's Sleep for Science Research Lab (http://bit.ly/128I3Dk). Adolescents whose sleep problems are not helped by these measures may benefit from referral to a sleep disorders clinic for a more indepth evaluation.

For 57 continuing nursing education articles on pediatric topics, go to www.nursingcenter.com/ce.

Nancy M. George is an assistant professor of nursing and the assistant director for the DNP program, and Jean E. Davis is the associate dean of academic and clinical affairs, at the College of Nursing, Wayne State University, Detroit. Contact author: Nancy M. George, nancy.george@wayne.edu. The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

## REFERENCES

1. Webb WB, Agnew HW. Are we chronically sleep deprived? Bull Psychon Soc 1975;6(1):47-8.
2. Crowley SJ, et al. Sleep, circadian rhythms, and delayed phase in adolescence. Sleep Med 2007;8(6):602-12.
3. Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. Child Dev 1998;69(4):875-87.
4. National Sleep Foundation. Teens and sleep. n.d. http:// www.sleepfoundation.org/article/sleep-topics/teens-and-sleep.
5. McKnight-Eily LR, et al. Relationships between hours of sleep and health-risk behaviors in US adolescent students. Prev Med 2011;53(4-5):271-3.
6. Carskadon MA. Factors influencing sleep patterns of adolescents. In: Carskadon MA, editor. Adolescent sleep patterns: biological, social, and psychological influences. Cambridge, UK; New York: Cambridge University Press; 2002. p. 4-26.
7. Jenni OG, Carskadon MA. Sleep behavior and sleep regulation from infancy through adolescence: normative aspects. Sleep Med Clin 2007;2(3):321-9.
8. Carskadon MA. The second decade. In: Gulleminault C, editor. Sleeping and waking disorders-indications and techniques. Menlo Park, CA: Addison-Wesley; 1982. p. 99-125.
9. Williams JA, et al. Norms and trends of sleep time among US children and adolescents. JAMA Pediatr 2013;167(1):55-60.
10. Jenni OG. How much sleep is "normal" in children and adolescents? JAMA Pediatr 2013;167(1):91-2.
11. Ireland JL, Culpin V. The relationship between sleeping problems and aggression, anger, and impulsivity in a population of juvenile and young offenders. J Adolesc Health 2006;38(6):649-55.
12. Talbot LS, et al. Sleep deprivation in adolescents and adults: changes in affect. Emotion 2010;10(6):831-41.
13. Colten HR, et al., editors. Sleep disorders and sleep deprivation: an unmet public health problem. Washington, DC: National Academies Press; 2006. https://www.ncbi.nlm.nih.gov/ books/NBK19960.
14. Kothare SV, Kaleyias J. Sleep and epilepsy in children and adolescents. Sleep Med 2010;11(7):674-85.
15. Enright PL, et al. Blood pressure elevation associated with sleep-related breathing disorder in a community sample of white and Hispanic children: the Tucson Children's Assessment of Sleep Apnea study. Arch Pediatr Adolesc Med 2003;157(9):901-4
16. Javaheri $S$, et al. Sleep quality and elevated blood pressure in adolescents. Circulation 2008;118(10):1034-40.
17. Hamilton NA, et al. Sleep and the affective response to stress and pain. Health Psychol 2007;26(3):288-95.
18. National Heart, Lung and Blood Institute. What are sleep deprivation and deficiency? National Institutes of Health. 2012. http://www.nhlbi.nih.gov/health/health-topics/topics/sdd.
19. Alhola P, Polo-Kantola P. Sleep deprivation: impact on cognitive performance. Neuropsychiatr Dis Treat 2007;3(5):554-67.
20. Iber C, et al. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester, IL: American Academy of Sleep Medicine; 2007. http://www.nswo.nl/userfiles/files/AASM\% 20-\%20Manual\%20for \% 20the \% 20Scoring \% 20ofSleep \%20and\%20Associted \% 20Events\%20-\%2005-2007_2.pdf.
21. Wyatt JK, et al. Circadian temperature and melatonin rhythms, sleep, and neurobehavioral function in humans living on a $20-\mathrm{h}$ day. Am J Physiol 1999;277(4 Pt 2):R1152-R1163.
22. Sadeh A. Stress, trauma and sleep in children. Child Adolesc Psychiatr Clin N Am 1996;5(3):685-700.
23. Dauvilliers Y, et al. Genetics of normal and pathological sleep in humans. Sleep Med Rev 2005;9(2):91-100.
24. Andretic R, et al. Genetics of sleep. Annu Rev Genet 2008;42:361-88.
25. Ludwig-Maximilians-Universitaet Muenchen (LMU). The ABCC 9 of sleep: a genetic factor regulates how long we sleep. ScienceDaily 2011 Nov 25. http://www.sciencedaily. com/releases/2011/11/111124150237.htm.
26. Retey JV, et al. A functional genetic variation of adenosine deaminase affects the duration and intensity of deep sleep in humans. Proc Natl Acad Sci U S A 2005;102(43):15676-81.
27. Toh KL, et al. An hPer2 phosphorylation site mutation in familial advanced sleep phase syndrome. Science 2001;291(5506):1040-3.
28. Manber R, et al. The effects of regularizing sleep-wake schedules on daytime sleepiness. Sleep 1996;19(5):432-41.
29. Buysse DJ, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28(2):193-213.
30. Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 1991;14(6):540-5.
31. Johns MW. Sleepiness in different situations measured by the Epworth Sleepiness Scale. Sleep 1994;17(8):703-10.
