



Published in final edited form as:

Pediatr Clin North Am. 2011 June ; 58(3): 637–647. doi:10.1016/j.pcl.2011.03.003.

Sleep in Adolescents: The Perfect Storm

Mary A. Carskadon, PhD[Professor]

Psychiatry and Human Behavior, Warren Alpert Medical School of Brown University and Director of Chronobiology and Sleep Research, E.P. Bradley Hospital

Abstract

The perfect storm metaphor applies to sleep patterns of adolescents in the sense that developmental trajectories of biopsychosocial factors conspire to limit the quantity of sleep for many adolescents resulting in a number of negative consequences. A reduction in sleep amount from late childhood through the second decade has long been known; however, the weight of current evidence holds that sleep need does not decline across this span. Nevertheless, parents, pediatricians, and school teachers, it seems, long assumed that this sleep decline was an inevitable part of growing up and a normative expectation. We shall see below that the loss of sleep through adolescence is not driven by lower need for sleep but arises from a convergence of biological, psychological, and socio-cultural influences.

Keywords

adolescence; circadian rhythms; sleep regulation; homeostatic pressure; melatonin; mood

Sleep Patterns of Adolescents

Adolescent sleep patterns have been surveyed by investigators in many countries from virtually every continent around the world, and a consistent finding is that the timing of bedtime on school nights gets later across the middle school and high school years (roughly ages 11 through 17 years).¹⁻¹⁵ Rise times on school mornings, by contrast, tend to stay relatively consistent except in countries such as the United States where the starting time of school moves to an earlier hour at the transition to high school. Weekend sleep for teenagers tends to delay further, and the difference in amount of sleep reported for school days versus weekends becomes more pronounced as children pass into higher grades (i.e., greater reported sleep on weeknights than school nights).

The most recent US poll of sleep patterns in adolescents was reported by the National Sleep Foundation in 2006, and collected self- and parent-reported sleep patterns from grades 6 through 12.¹⁶ These data serve as a good example of these general trends, as shown in Table 1. The young people interviewed in this telephone poll reported that average bedtime on the nights before school days were approximately 1.5 hours later from grade 6 to grade 12, and reported weekend bedtime delayed from 2231 to 0045 across this same grade span. Sixth graders reported going to bed about an hour later on weekend nights, and for twelfth

© 2011 Elsevier Inc. All rights reserved.

mary_carskadon@brown.edu, Bradley Hospital Sleep Lab, 300 Duncan Drive, Providence, RI 02906, USA, Tel. 401-421-9440, Fax 401-452-2578.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

graders, the weekend bedtime delay was about an hour and 45 minutes. The average reported rise time on school mornings was 0642 in grade 6 and 0631 in grade 12. The reported number of hours slept on school nights declined from 8.4 hours in the sixth grade students to 6.9 hours in the twelfth graders; reported weekend sleep was more consistent from grades 6 through 11—about 9 hours—falling to 8.4 hours in grade 12. The weekend extension of sleep time in this report was nearly an hour in the middle school children (grades 6-8) and approached 2 hours for grade 11 students.

Amounts of sleep reported by adolescents varies across countries and regions; however, the overall patterns of later sleep timing and diminished sleep across adolescence is reported by most investigators. Reports of Korean youth, for example, indicate they begin and end grades 6 to 12 with later school night bedtimes and less sleep than those in the US.¹⁵ Children were assessed with a survey administered in the classroom querying usual sleep schedule. On average, the children reported:

- school night bedtime: grades 5-6 = 2242, grades 7-8 = 2312; grades 9-10 = 0000; grades 11-12 = 0054;
- school morning rise time: grades 5-6 = 0718; grades 7-8 = 0700; grades 9-10- = 0648; grades 11-12 = 0618;
- school night hours slept: grades 5-6 = 8.3; grades 7-8 = 7.6; grades 9-10- = 6.6; grades 11-12 = 5.4;
- weekend night bedtime: grades 5-6 = 2300, grades 7-8 = 2330; grades 9-10 = 2336; grades 11-12 = 2354;
- weekend morning rise time: grades 5-6 = 0806; grades 7-8 = 0854; grades 9-10- = 0930; grades 11-12 = 0918;
- weekend night hours slept: grades 5-6 = 9.0; grades 7-8 = 8.8; grades 9-10- = 9.0; grades 11-12 = 8.4.

An apparent difference in the older Korean children from the 11th and 12th graders in the US poll¹⁶, was that the reported weekend bedtime was earlier than on school nights. The authors note that many students (71.1%) took additional course work in the evening, with classes lasting until midnight or later for 54.5% of the older students. This pattern of late night classes on school days likely also explains why the Korean high school students reported earlier weekend than school-night bedtime on average¹⁵. We note as well that the standard deviation for reported weekend bedtime was 3 to 4 hours, much greater than for other variables.

The issue of determining the 'sleep need' of adolescents is challenging, since the definition of sleep need itself is in dispute. Early work by Carskadon and colleagues⁴ started from a questionnaire assessment of school children in which the discrepancy between school day and weekend reported amount slept was zero, and the average amount was 10 hours a night. Of interest is the parallel finding of Iglowstein and colleagues¹⁷ showing a concurrence of weekend and weekday sleep amounts at about 10 hours from reports by mothers of Swiss children (see Figure 1 in¹⁷). In the longitudinal study of adolescents by Carskadon, time in bed was fixed at 10 hours a night, and the data showed that youngsters across the span of ages 10 to 17 slept about 9 hours and 20 minutes¹⁸. The younger children were more likely to waken spontaneously in the lab, whereas the older children took a bit longer to fall asleep and were rarely spontaneously awake at the end of the 10 hours. Furthermore, assessment with the multiple sleep latency test (MSLT, an objective measure of sleepiness) showed that the younger children were more alert across the day, whereas the pubertal and postpubertal adolescents showed a midday trough of alertness in spite of the same amount of nocturnal

sleep¹⁹. In another study that may bear on the issue of sleep need of adolescents, Carskadon and colleagues²⁰ showed that adolescents (ages 11 to 14.6 years) given a 10-hour sleep schedule for 10 to 14 nights and then studied in the lab for three consecutive 18 hour nights, slept nearly 12.5 hours on average the first night and by night 3 still slept 10.1 hours on average (standard deviation about 1 hour).

Adolescent Development and Sleep Regulation

In describing the biological regulation of sleep, current thinking uses the two-process model, first proposed by Borbély and colleagues²¹. The two processes that comprise the model include a daily (circadian) rhythm of sleep propensity, thought to originate from the suprachiasmatic nucleus of the hypothalamus in mammals, and a sleep-wake 'pressure' (homeostatic) system, for which a neuronanatomical locus has not been identified. In the case of the former process, the signal from the brain's central clock is thought to affect sleep and arousal systems to help gate the timing of sleep. The sleep homeostatic system favors sleep as the hours of wakefulness are extended and favors waking as sleep is prolonged, thus transducing information about the length and amount of prior sleep or wake, rather than the time of day. The role of these two processes can be distinguished independent from one another under certain experimental conditions²², but work interactively to regulate sleep from day to day.

Developmental changes in the circadian timing system were inferred by some from the delay in the timing of sleep onset, and two studies published in 1993 gave initial evidence for the hypothesis that a phase delay occurs in association with puberty. For example, Andrade and colleagues²³ found in a longitudinal study that later sleep times occurred for adolescents at a more mature Tanner stage²⁴ than others. Carskadon and colleagues reported in a cross-sectional study using self reported sleep and pubertal stage that sixth grade girls who rated themselves more mature had a more evening type score on a measure of circadian phase preference²⁵. A subsequent follow-up cross-sectional study in which adolescents' sleep-wake schedules were constrained for two weeks confirmed that circadian phase as evaluated with the objective measure of dim light melatonin onset (DLMO) phase was positively correlated with Tanner stage. In other words, the DLMO was later for participants rated more mature indicating that the onset of the 'biological night' is later for more mature teens.

Several hypotheses have been put forward to explain the features of the circadian timing system that may underlie this developmental change. A review of adolescent changes in sleep and circadian rhythms by Hagenauer and colleagues²⁶ noted that signs of a juvenile phase delay were found in several mammalian species in addition to humans. Table 2 highlights these findings. The occurrence of a circadian delay around the time of puberty for a number of species lends credence to the hypothesis that this phenomenon arises from intrinsic biological processes rather than as a response to social/behavioral exposures. Whether and how the phase delay is linked to reproductive development is unknown.

If the adolescent phase delay is inherent, what mechanisms might explain the phenomenon. One hypothesis is that circadian period (i.e., the internal day length) may become longer during adolescence. Pubertal male rats were found in one study to have longer intrinsic period than adults²⁷, and data from humans show a longer period in teens than in adults²⁸. An alternate explanation is that the circadian timing system manifests an altered sensitivity to phase dependent effects of light; in particular, the pacemaker may become more sensitive to light's phase delaying effects during adolescence than in childhood²⁶. One animal study that offers support for this hypothesis showed an exaggerated delay of rhythms to light occurring at the phase-delaying time in pubertal than in adult mice²⁹. This hypothesis

depends on an underlying biological change that is then affected by behavioral exposures that arise during adolescence (i.e., staying awake later, thus experiencing more evening light). A converse to this hypothesis is that adolescents become less sensitive to phase-advancing (morning) light or experience a change in the shape of the phase response curve. Preliminary data from one study of human adolescents provides a bit of evidence supporting this hypothesis³⁰.

The influence of the sleep-wake homeostasis system is well understood, although specific neuroanatomical or neurochemical constituents are not yet fully established, nor has a link between adolescent maturation and this physiological process been identified. Thus, whether the homeostatic process might be influenced by hormonal input, physical growth, brain maturation, or other factor(s) is unknown. On the other hand, several evaluations of adolescent sleep physiology provide evidence for developmental changes in this sleep regulatory process. The theoretical model of sleep-wake homeostasis has a long history and a significant amount of experimental data supporting the model, based on sleep slow frequency EEG data from animal and human models (see ³¹ for review): As waking is prolonged across a day or longer, slow frequency EEG amplitude and incidence increases; as sleep unfolds across the night, slow frequency EEG amplitude and incidence decays. Several studies have shown that adolescent development is not associated with a change in the decay of sleep homeostatic “pressure” (indexed by EEG slow wave activity in sleep) but remains stable across early adolescent development (ages about 9-10 through ages about 14-15 years)³² and up to age 18 y in another study³³. On the other hand, preliminary data from a recent analyses of sleep EEG slow wave activity in another adolescent cohort (ages 15-16 through about ages 17-19 years) demonstrate a longer time constant for the decay across the night in several brain regions³⁴. One interpretation of the latter findings is that the sleep recovery process is unchanged in early adolescents and that certain parts of the brain may require more sleep to recover in older teens than when younger. This set of findings reinforce the likelihood that sleep need does not decline in adolescents.

Whereas the dissipation of sleep homeostatic pressure may speak to the recovery function of sleep, the other limb of the homeostatic process describes how the ‘pressure’ to sleep or sleep propensity builds across the waking day. Jenni and colleagues³⁵ modeled the accumulation of sleep homeostasis in early adolescent humans (ages 9 to 14 y) who were either pre-/early pubertal or post pubertal. This analysis showed a longer time course for the build up of sleep ‘pressure’ in the more mature group, indicating that the more mature adolescents may find it easier to stay awake longer than the less mature. This hypothesis was confirmed by analyzing speed of falling asleep on the multiple sleep latency test in a group of pre-early pubescent vs. a group of post pubertal adolescents across 36 h of extended waking: the less mature adolescents fell asleep faster than the more mature on the tests occurring 14.5 and 16.5 h after waking³⁶.

In summary, current data on the maturation of the biological regulation of sleep during adolescence show that:

- the circadian timing system undergoes a phase delay;
- the dissipation of sleep homeostatic pressure does not change until late adolescence when it shows evidence of slowing;
- the accumulation of sleep homeostatic pressure slows during puberty.

These findings support the notion that the need for sleep is stable (or increases) across adolescent development and that the delay of sleep timing is supported by the circadian shift as well as the slowing of sleep homeostatic pressure accumulation.

Psychosocial Factors Affecting Adolescent Sleep

Many psychosocial factors affect sleep patterns in adolescents and contribute to the phase delay and the decline of time slept. We consider a few examples below and examine how they interact with these biological regulatory processes. A primary psychodevelopmental task of adolescents is to achieve independence in many areas of their lives. One area where this striving for autonomy is displayed is the decision of when to go to sleep. One sleep habits surveys from the early 1980s showed that a significantly higher percentage of children ages 12 or 13 y reported setting their own bedtime on school nights (e.g., 19% of 13 y olds) than did younger children (e.g., 50% of 10 y olds) 10- to 12-y old children and that reported bedtimes were later for the older children³⁷. The other end of school-night sleep—waking up—showed an opposite trend: more older children reported needing a parent or alarm to wake them than did the younger children, who were likelier to report that they “just wake up.” Thus, the adolescents who actualize autonomy by staying up late seem to “ride the wave” of the circadian phase delay and to avail themselves of the relative emancipation from rapid sleep pressure accumulation. Yet, they were sleeping less and rising early required external intervention to wake up.

Two recent studies highlight the importance of parental-set bedtimes. Gangwisch and colleagues³⁸ examined from a large epidemiologic data set gathered from adolescents (grades 7 to 12) in the US during the mid-1990s. The analysis showed that young people whose parent set their bedtime at midnight or after relative to those with bedtimes set at 2200 or earlier, were significantly more likely to suffer from depression or suicidal ideation. The authors also found that this association was mediated by total sleep time; thus, those with earlier bedtimes reported sleeping more and were less likely to be depressed or experience suicidal ideation. A smaller, focused study of South Australian adolescents found that adolescents who reported a parental-set bedtime *per se* versus those without a set bedtime reported earlier bedtimes, more sleep, and less daytime fatigue experienced³⁹. These two studies provide evidence that 1) parental intervention by setting a bedtime results in extended sleep; 2) the combination of earlier bedtime and extended sleep contribute to more positive outcomes for depression, sleepiness, and fatigue. These findings may also indicate that such behavioral interventions as having a fixed, earlier bedtime may help mitigate the circadian phase delay associated with adolescent maturation, likely through preventing light to the circadian timing system at the delay-sensitive evening phase.

Screen time, technology use, and social engagement in the evening become more available as youngsters pass through adolescence. The US National Sleep Foundation poll of adolescents¹⁶ reported that electronic devices (i.e., electronic music devices, television, electronic/video games, cell phone, telephone, computer, Internet access) in the bedroom become more common from grade 6 to grade 12 students: the median number of such devices in bedrooms of younger adolescents was two versus a median of four in the older teens. A recent review by Cain and Gradisar⁴⁰ notes that the preponderance of studies report shorter, later, and/or more disrupted sleep, as well as such daytime consequences as sleepiness or disruptive behavior, for children and adolescents as TV watching, computer/Internet/electronic games use, or mobile phone use in the evening before bedtime is greater. These activities are arousing in and of themselves and usually more easily accessed by the older adolescents, taking advantage of increased accessibility of technology and of the changes to the sleep regulatory systems that make it easier to stay away later. Indeed, to the extent that the activities involve light exposure—perhaps particularly blue-spectrum light exposure to which the circadian clock may have greater sensitivity^{41,42}—evening light has the phase-specific effect of delaying circadian rhythms, thus pushing sleep timing later⁴³.

The impact of school schedules of adolescents is most often to restrict sleep by requiring an early rising time. As noted above, however, school schedules can delay sleep further, such as described for Korean adolescents enrolled in evening classes to improve preparation for academic placement tests¹⁵. Evening homework assignments often carry a similar challenge for adolescents in the US. More attention has been directed toward school starting times because of the tendency for many school systems in the US to start the school day earlier as children get older, thus requiring teens to arise at an early hour relative to their typical bedtimes, circadian phases, and need for sleep.

One study examined sleep, sleepiness, and circadian rhythms in teens for whom the transition from ninth grade to tenth grade involved an advance of the school start time from 0825 to 0720⁴⁴. Table 3 highlights findings from this study. Actigraphically monitored sleep data across two weeks confirmed that, although the students woke up significantly earlier on school nights in tenth than ninth grade, they did not go to sleep earlier and consequently experienced less sleep on average. The general level of sleepiness in these students is shown with the daytime sleep latency data, showing a moderate level of sleepiness in ninth grade, and a significant decline in tenth grade to a level considered severely sleepy for the sleep latency at 0830. The 0830 assessment was also associated with the occurrence of REM sleep within 10 or 15 minutes of sleep onset, (a finding often used to aid in the diagnosis of narcolepsy) in about one half of the participants. The study also showed that the time of the dim light melatonin onset (DLMO) was nearly 30 m later on average in the students when assessed in tenth grade; indeed, the DLMO time in the tenth graders was significantly later (2146 vs. 2036) for those with the short sleep latency and REM sleep in the morning nap, interpreted as evidence that the circadian timing system of these teens was phase delayed relative to the early start time and favored sleep during the morning hours they were scheduled to attend school⁴⁴. The association of a circadian phase delay, excessive morning sleep tendency, and REM sleep in morning trials for one-half of the adolescents studied is remarkable and raises the level of concern for early morning classes.

The Perfect Storm: Sleep Behavior and Troubling Outcomes

Figure 1 illustrates the confluence of factors that combine for adolescents in the 21st century to reduce time spent sleeping on school nights below a healthful amount, with waking and expected school performance timed to occur at an inappropriate circadian phase. As reviewed above, bioregulatory and psychosocial forces collude to push sleep onset later, yet schools are timed to begin earlier across adolescence, and sleep time is compressed as a consequence. The list of negative outcomes associated with insufficient sleep is lengthy and ranges from sleepiness and mood disturbances noted above, inattention, poor grades, behavior problems, substance use, driving crashes, overweight, and immune system compromise. For some adolescents, the issue can present as sleep-onset insomnia that may be associated with the circadian phase delay; when combined with lack of motivation, depressed mood, and fatigue, a depressive disorder is often the initial assumption. An approach that targets sleep timing and phase adjustment is likely to be ameliorative.

At the end of the day, the pediatrician can have positive impact by intervening in any of a number of levels:

- encourage your local school districts to consider starting the school day later for adolescents;
- recommend that the local school district also limit late-evening activities;
- galvanize schools to provide instructional information about sleep and circadian rhythms;

- support and encourage parents to identify and set an appropriate bedtime;
- encourage teens to avoid light and stimulating activities in the evening and to get light exposure in the morning;
- empower adolescents to make informed choices about their sleep schedules;
- remind families of the utility of a relaxing pre-sleep ritual.

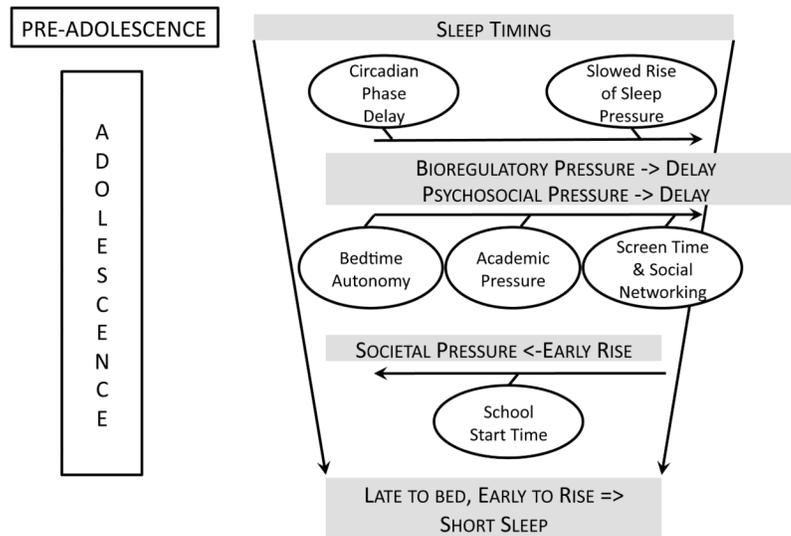
References

1. Andrade, M.; Menna-Baretto, L. Sleep patterns of high school students living in Sao Paulo, Brazil. In: Carskadon, MA., editor. *Adolescent Sleep Patterns: Biological, Social, and Psychological Factors*. Cambridge University Press; New York: 2002. p. 118-31.
2. Arakawa M, Taira K, Tanaka H, et al. A survey of junior high school students' sleep habit and lifestyle in Okinawa. *Psychiatry and clinical neurosciences*. 2001; 55(3):211-2. [PubMed: 11422845]
3. Bearpark HM, Michie PT. Prevalence of sleep/wake disturbances in Sidney adolescents. *Sleep Research*. 1987; 16:304.
4. Carskadon MA. Patterns of sleep and sleepiness in adolescents. *Pediatrician*. 1990; 17(1):5-12. [PubMed: 2315238]
5. Dorofaeff TF, Denny S. Sleep and adolescence. Do New Zealand teenagers get enough? *Journal of paediatrics and child health*. 2006; 42(9):515-20. [PubMed: 16925537]
6. Gau SF, Soong WT. The transition of sleep-wake patterns in early adolescence. *Sleep*. 2003; 26(4): 449-54. [PubMed: 12841371]
7. Gibson ES, Powles AC, Thabane L, et al. "Sleepiness" is serious in adolescence: two surveys of 3235 Canadian students. *BMC public health*. 2006; 6:116. [PubMed: 16670019]
8. Laberge L, Petit D, Simard C, et al. Development of sleep patterns in early adolescence. *J Sleep Res*. 2001; 10(1):59-67. [PubMed: 11285056]
9. Park YM, Matsumoto K, Seo YJ, et al. Changes of sleep or waking habits by age and sex in Japanese. *Perceptual and motor skills*. 2002; 94(3 Pt 2):1199-213. [PubMed: 12186242]
10. Reid A, Maldonado CC, Baker FC. Sleep behavior of South African adolescents. *Sleep*. 2002; 25(4):423-7. [PubMed: 12071543]
11. Saarenpaa-Heikkila OA, Rintahaka PJ, Laippala PJ, et al. Sleep habits and disorders in Finnish schoolchildren. *J Sleep Res*. 1995; 4(3):173-82. [PubMed: 10607156]
12. Strauch I, Meier B. Sleep need in adolescents: a longitudinal approach. *Sleep*. 1988; 11(4):378-86. [PubMed: 3206056]
13. Thorleifsdottir B, Bjornsson JK, Benediktsdottir B, et al. Sleep and sleep habits from childhood to young adulthood over a 10-year period. *Journal of psychosomatic research*. 2002; 53(1):529-37. [PubMed: 12127168]
14. Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. *Child development*. 1998; 69(4):875-87. [PubMed: 9768476]
15. Yang CK, Kim JK, Patel SR, et al. Age-related changes in sleep/wake patterns among Korean teenagers. *Pediatrics*. 2005; 115(1 Suppl):250-6. [PubMed: 15866859]
16. National Sleep Foundation. 2006 Sleep In America Poll Summary Findings. 2006. at http://www.sleepfoundation.org/site/c.huIXKjM0Ix/b.2419037/k.1466/2006_Sleep_in_America_Poll.htm
17. Iglowstein I, Jenni OG, Molinari L, et al. Sleep duration from infancy to adolescence: reference values and generational trends. *Pediatrics*. 2003; 111(2):302-7. [PubMed: 12563055]
18. Carskadon, MA.; Orav, EJ.; Dement, WC. Evolution of sleep and daytime sleepiness in adolescents. In: Guilleminault, C.; Lugaresi, E., editors. *Sleep/wake disorders: Natural history, epidemiology, and long-term evolution*. Raven Press; New York: 1983. p. 201-16.
19. Carskadon MA, Harvey K, Duke P, et al. Pubertal changes in daytime sleepiness. *Sleep*. 1980; 2:453-460. [PubMed: 7403744]

20. Carskadon MA, Acebo C, Seifer R. Extended nights, sleep loss, and recovery sleep in adolescents. *Arch Ital Biol.* 2001; 139(3):301–12. [PubMed: 11330207]
21. Borbély AA. A two process model of sleep regulation. *Hum Neurobiol.* 1982; 1(3):195–204. [PubMed: 7185792]
22. Czeisler, FD.; Czeisler, CA.; Allan, JS.; Kronauer, RE. A method for assaying the effects of therapeutic agents on the period of the endogenous circadian pacemaker in man. In: Montplaisir, J.; Godbout, R., editors. *Sleep and biological rhythms: basic mechanisms and applications to psychiatry.* Oxford University Press; New York: 1990. p. 87-98.chapter
23. Andrade MM, Benedito-Silva AA, Domenice S, et al. Sleep characteristics of Adolescents: A longitudinal study. *J Adol Health.* 1993; 14:401–6.
24. Tanner, JM. *Growth at Adolescence.* 2nd ed.. Blackwell; Oxford: 1962.
25. Carskadon MA, Vieira C, Acebo C. Association between puberty and delayed phase preference. *Sleep.* 1993; 16(3):258–62. [PubMed: 8506460]
26. Hagenauer MH, Perryman JI, Lee TM, Carskadon MA. Adolescent changes in the homeostatic and circadian regulation of sleep. *Dev Neurosci.* 2009; 31(4):276–84.Fe. [PubMed: 19546564]
27. McGinnis MY, Lumia AR, Tetel MJ, et al. Effects of anabolic androgenic steroids on the development and expression of running wheel activity and circadian rhythms in male rats. *Physiol Behav.* 2007; 92:1010–18.
28. Carskadon MA, Labyak SE, Acebo C, et al. Intrinsic circadian period of adolescent humans measured in conditions of forced desynchrony. *Neuroscience Letters.* 1999; 260(2):129–32. [PubMed: 10025716]
29. Weinert D, Eimert H, Erkert HG, Schneyer U. Resynchronizatin of the circadian corticosterone rhythm after a light/dark shift in juvenile and adult mice. *Chronobiol Int.* 1994; 11:222–31. [PubMed: 7954905]
30. Carskadon MA, Acebo C, Arnedt JT, et al. Melatonin sensitivity to light in adolescents: preliminary results. *Sleep.* 2001; 24(Suppl.):A190–A1.
31. Borbély, AA.; Achermann, P. Sleep Homeostasis and Models of Sleep Regulation. In: Kryger, MH.; Roth, T.; Dement, WC., editors. *Principles and Practice of Sleep Medicine.* 3 ed.. W.B. Saunders Co.; Philadelphia: 2000. p. 377-90.
32. Jenni OG, Carskadon MA. Spectral analysis of the sleep electroencephalogram during adolescence. *Sleep.* 2004; 27(4):774–83. [PubMed: 15283014]
33. Campell IG, Darchia N, Higgins LM, et al. Adolescent changes in homeostatic regulation of EEG activity in the delta and theta frequency bands during NREM sleep. *Sleep.* 2011; 34(1):83–91. [PubMed: 21203377]
34. Tarokh L, Carskadon MA, Rusterholz T, Achermann P. Homeostatic sleep regulation in adolescents: Longitudinal perspectives. *Sleep.* in press.
35. Jenni OG, Achermann P, Carskadon MA. Homeostatic sleep regulation in adolescents. *Sleep.* 2005; 28(11):1446–54. [PubMed: 16335485]
36. Taylor DJ, Jenni OG, Acebo C, et al. Sleep tendency during extended wakefulness: insights into adolescent sleep regulation and behavior. *J Sleep Res.* 2005; 14(3):239–44. [PubMed: 16120098]
37. Carskadon, MA. The second decade. In: Guilleminault, C., editor. *Sleep and waking disorders: Indications and techniques.* Addison Wesley; Menlo Park, CA: 1982. p. 99-125.
38. Gangwisch JE, Babiss LA, Malaspina D, et al. Earlier parental set bedtimes as a protective factor against depression and suicidal ideation. *Sleep.* 2010; 33(1):97–106. [PubMed: 20120626]
39. Short MA, Gradisar M, Wright H, Lack LC, Dohnt H, Carskadon MA. Time for bed: Parent-set bedtimes associated with improved sleep and daytime functioning in adolescents. *Sleep.* in press.
40. Cain N, Gradisar M. Electronic media use and sleep in school-aged children and adolescents. *Sleep Med.* 2010; 11(8):735–42. [PubMed: 20673649]
41. Brainard GC, Hanifin JP, Greenson JM, et al. Action spectrum for melatonin regulation in humans: evidence for a novel circadian photoreceptor. *J Neurosci.* 2001; 21(16):6405–12. [PubMed: 11487664]

42. Thapan K, Arendt J, Skene DJ. An action spectrum for melatonin suppression: evidence for a novel non-rod, non-cone photoreceptor system in humans. *The Journal of physiology*. 2001; 535(Pt 1): 261–7. [PubMed: 11507175]
43. Khalsa SB, Jewett ME, Cajochen C, Czeisler CA. A phase response curve to single bright light pulses in human subjects. *J Physiol*. Jun 15; 2003 549(Pt 3):945–52. [PubMed: 12717008]
44. Carskadon MA, Wolfson AR, Acebo C, Tzischinsky O, Seifer R. Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. *Sleep*. Dec 15; 1998 21(8): 871–81. [PubMed: 9871949]

Adolescent Development & Sleep: The Perfect Storm

**Figure 1.**

This figure illustrates the timing of sleep from preadolescence through adolescent development highlighting the factors that affect sleep as described in the text. Thus, sleep is relatively long and timed at an early hour for preadolescents, but maturational changes to intrinsic bioregulatory factors—the circadian phase delay arising from the circadian timing system and a slowed rise of sleep pressure stemming from sleep-wake homeostasis—push for a delay of the timing of sleep. Such psychosocial factors as self-selected bedtimes, response to academic pressure, and the availability and use of technology and social networking in the evening also push for a delay in the timing of sleep. Note that the length of sleep is not affected by these processes. Societal pressures that push for an early rise time—most notably an early start to the school day—are the forces that limit amount of time available for sleep. As a consequence, adolescents sleep too little and are asked to be awake at an inappropriate circadian phase.

Table 1

Sleep Patterns Reported by Adolescent School Children: National Sleep Foundation 2006 Sleep in America Poll¹⁶

Sleep Variable	Grade in School												
	Sixth	Seventh	Eighth	Ninth	Tenth	Eleventh	Twelfth						
School Nights													
Bedtime (24-Hour)	2124	2152	2153	2215	2232	2251	2302						
Risetime (24-Hour)	0642	0635	0636	0628	0623	0623	0631						
Average Sleep (hours)	8.4	8.1	8.1	7.6	7.3	7.0	6.9						
Weekend Nights													
Bedtime (24-Hour)	2231	2305	2326	2353	0003	0025	0045						
Risetime (24-Hour)	0853	0912	0921	0954	0954	1006	0951						
Average Sleep (hours)	9.2	8.9	9.0	8.8	8.9	8.8	8.4						
School Night – Weekend Hours Slept Difference	0.8	0.8	0.9	1.2	1.6	1.9	1.5						

Table 2Mammals with a Juvenile Phase Delay¹

Species	Amount of Delay	Rhythms Delayed	Sex Difference
Human (<i>Homo sapiens</i>)	1 – 3 h	Sleep, melatonin	Males > females
Rhesus monkey (<i>Macaca mulatta</i>)	2 h	Activity	Only females studied
Degu (<i>Octodon degus</i>)	3-5 h	Activity, sleep(?)	Males > females
Laboratory rat (<i>Rattus norvegicus</i>)	1-4 h	Activity	Males > females
Laboratory mouse (<i>Mus musculus</i>)	1 h (?)	Activity, corticosterone	Only females studied
Fat sand rat (<i>Psammomys obesus</i>)	0-3 h to 10-14 h (photoperiod dependent)	Oxygen consumption, temperature	Sex unspecified

¹ See Hagenauer et al.²⁶ for more information about these adolescent phase delays.

Table 3

Sleep, Sleepiness, and Circadian Rhythms at a Transition to Early School Days [mean (standard deviation)]

Actigraph Sleep Data	School Nights		Weekend Nights	
	9 th Grade	10 th Grade	9 th Grade	10 th Grade
Sleep Onset	2242 (36)	2238 (44)	2338 (56)	2344 (53)
Sleep Offset	0626 (28)	0601 (19)*	0828 (69)	0832 (66)
Time Slept	429 (47)	410 (42)*	485 (57)	478 (67)
Daytime Sleep Latency				
0830	10.9 (6.5)	5.1 (4.1)*		
1030	10.7 (5.9)	9.2 (6.4)		
1230	11.0 (5.5)	9.8 (5.5)		
1430	11.5 (6.1)	11.0 (7.2)		
Melatonin Onset Time	2024 (57)	2102 (77)*		

* Statistically significant difference between 9th and 10th grade⁴