# Regulation of Adolescent Sleep

## Implications for Behavior

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**ABSTRACT: Adolescent development is accompanied by profound changes in the timing and amounts of sleep and wakefulness. Many aspects of these changes result from altered psychosocial and life-style circumstances that accompany adolescence. The maturation of biological processes regulating sleep/wake systems, however, may be strongly related to the sleep timing and amount during adolescence—either as "compelling" or "permissive" factors. The two-process model of sleep regulation posits a fundamental sleep-wake homeostatic process (process S) working in concert with the circadian biological timing system (process C) as the primary intrinsic regulatory factors. How do these systems change during adolescence? We present data from adolescent participants examining EEG markers of sleep homeostasis to evaluate whether process S shows maturational changes permissive of altered sleep patterns across puberty. Our data indicate that certain aspects of the homeostatic system are unchanged from late childhood to young adulthood, while other features change in a manner that is permissive of later bedtimes in older adolescents. We also show alterations of the circadian timing system indicating a possible circadian substrate for later adolescent sleep timing. The circadian parameters we have assessed include phase, period, melatonin secretory pattern, light sensitivity, and phase relationships, all of which show evidence of changes during pubertal development with potential to alter sleep patterns substantially. However the changes are mediated—whether through process S, process C, or by a combination—many adolescents have too little sleep at the wrong circadian phase. This pattern is associated with increased risks for excessive sleepiness, difficulty with mood regulation, impaired academic performance, learning difficulties, school tardiness and absenteeism, and accidents and injuries.**

**KEYWORDS: circadian rhythms; sleep homeostasis; puberty; melatonin; adolescent humans; MSLT**

### INTRODUCTION

The timing of sleep and wakefulness undergoes one of the most prominent behavioral changes that occur during adolescent development, a change that occurs in a

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majority of young people. Data collected from many countries have confirmed the strong trend for later bedtimes and later rising times during the teen years, for example, USA,[1,2] Canada,[3] Switzerland,[4] Italy,[5] Taiwan,[6,7] Brazil,[8,9] and South Africa.[10] In each instance, the temporal delay of sleep timing manifests most clearly on non-school nights (weekends and vacations); even when the school schedule constrains rising times, the bedtime delay remains apparent on school nights.[11]

Explanations for this developmental pattern are easy to identify in the changing adolescent psychosocial milieu. Such processes as the growing expression of autonomy, the increase in academic obligations and social opportunities, as well as the rising availability of late evening activities offered by access to telephone, television, and Internet—all contribute in a significant way to the behavioral regulation of adolescent sleep patterns.

In addition, however, sleep and waking are under regulatory control by intrinsic brain mechanisms. The most well-known model describing these mechanisms was first expressed by Borbély as the "two-process model,"[12] a model subsequently refined by Borbély and others.[13–16] In straightforward terms, the model posits a central mechanism providing homeostatic sleep regulation that interacts with the circadian timing mechanism. Anatomic, cellular, and molecular properties of the latter system have been described in great detail, as have the organizing principles of its function.[17] The anatomical structure of the homeostatic process is not yet known, however, nor has the specific nature of the neurochemical or neurocellular basis of this process yet been described, though adenosine regulation,[18] thalamocortical brain oscillations,[19,20] and changes in gene expression[21] have been hypothesized as specific features of the system. Nevertheless, the operational outputs of the homeostatic sleep-wake regulatory system such as quantitative measures of the EEG have been well described (for an overview see Ref. 22), and extensive modeling has successfully predicted outcomes.[23]

In brief, the homeostatic sleep-wake dependent process is modeled as "process S," which accumulates while awake and dissipates during sleep (FIG. 1).[12] Electrophysiological markers of process S include (1) stage 4 NREM sleep also known as slow wave sleep or SWS[24] and (2) EEG power density in the low frequency range (0.75–4.5 Hz) also known as slow wave activity or SWA.[25] The time course of process S delineated from SWA exhibits an exponential decay during sleep and an exponential rise during waking. Sleep occurring after a brief episode of waking (as in a daytime nap) shows relatively little stage 4 and low levels of SWA[26,27] as compared to these features during sleep that follows a normal or extended day length.[25,28] Thus, SWA levels during NREM sleep are determined by the duration of prior sleep and wakefulness. In addition, the speed of falling asleep (sleep latency) has been demonstrated to be a marker for sleep homeostasis.[29] For example, sleep restriction over several nights induces a progressive reduction of sleep latency.[30] Thus, faster sleep onsets can indicate greater accumulation of sleep homeostatic drive, familiar to us all, for example, when witnessing sleep deprived students or colleagues nodding off during the day.

The circadian timing system is essentially independent of prior waking and sleep.[31] It contributes to the timing of sleep by providing signals (the precise neurophysiological nature of which are unknown) interpreted as "sleep gates" occurring at some times and during other phases as "forbidden zones" for sleep.[13,32] Circadian sleep propensity reaches its maximal levels in the early morning and its trough in the

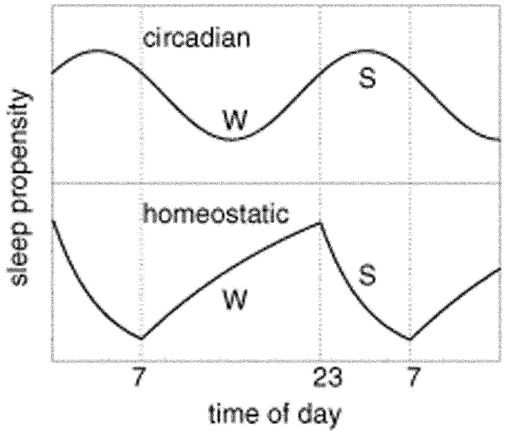


**FIGURE 1.** Schematic of the circadian and homeostatic process underlying sleep regulation. The rise of the homeostatic process yields a longer time constant then the decay reflecting the unbalanced amount of time spent awake and asleep. W, wakefulness; S, sleep. (Redrawn in part from Achermann & Bobély.[23])

evening (FIG. 1).[31] Current models include a concept in which the homeostatic and circadian systems normally operate as opponent processes that enable the consolidation of sleep and wakefulness.[16,31,33,34] In other words, an increasing circadian drive for arousal during the waking day is proposed to counteract the growing homeostatic sleep pressure in order to maintain the waking state.[14,16,33] Conversely, the declining NREMS sleep intensity or pressure during sleep opposes the increase in circadian sleep tendency across the night thereby maintaining consolidated sleep.[34]

Several additional features of the circadian timing system merit introduction (for an overview see Ref. 17). Although we do not have direct access to the clock mechanism itself in human participants, we are able to measure such parameters of the system as phase, period, amplitude, and phase angle through assessing peripheral signals. The timing of melatonin secretion is one of the most reliable of such measures and is eminently accessible in young humans through radioimmunoassay of melatonin from serial saliva samples. Thus, we can assess the phase of the circadian system as the onset, offset, midpoint, or peak phase of melatonin secretion. Phase preference (when it "feels good" to do various activities) can be assessed using standard self-assessment instruments.[35–37] The intrinsic period of the circadian timing system is more difficult to assess. In rodents, period is inferred from the timing of behaviors (usually wheel-running) over the course of many cycles under conditions of environmental isolation and constant darkness—not a testing environment available for human studies, particularly not for young humans. Instead, a method called forced desynchrony is used, wherein participants stay in the lab for a number of weeks under relatively low light conditions with sleep and waking scheduled at a day length that exceeds the range of entrainment of the circadian timing system, such as 20 or 28 hours.[38,39] Under these circumstances, the circadian timing system runs

free from the sleep–wake process, and the intrinsic period of the rhythm can be assessed through the timing of daily phase markers. Coincidentally, this experimental paradigm is useful for analyses to identify the influences of the homeostatic and circadian processes independent from and interacting with one another.[31]

Our group has begun to apply these principles and methods to examine the development of intrinsic sleep-wake regulatory processes during adolescent development. Our aim is to determine the extent to which these processes undergo predictable changes in association with pubertal development and ultimately to identify how such changes may interact with the behavioral regulation of sleep. Intrinsic changes may either compel or control the adolescent phase delay, or they may be permissive of the phase delay. Thus, for example, a puberty-related delay in circadian phase might prevent older teens from falling asleep early and drive a later time for arousal; or a reduction in the build up rate of process S might permit or ease the way for older teens to stay up late. In addition, an important corollary of these biological systems is that certain aspects of the intrinsic regulatory processes may themselves respond to alterations of sleep and wakefulness associated with behavioral regulation. For example, behaviorally mediated changes in the timing of light–dark exposure directly interact with the phase resetting mechanism of the circadian timing system[40] and can reinforce or strengthen a tendency to phase delay. In this paper, we review findings that address these issues, identify the net effect on sleep patterns, and discuss briefly how the ultimate result of these processes may lay open vulnerable outcomes for adolescents.

## THE HOMEOSTATIC PROCESS (PROCESS S) DURING ADOLESCENCE

The developmental alteration in slow wave sleep during adolescence has been known for a number of years. Feinberg[41] showed that SWS declined across the adolescent years using cross-sectional samples, as did the Williams group.[42,43] Karacan and colleagues[44] showed a pubertal decline in SWS in a longitudinal study where sleep was on the participants "usual" schedules, confirmed by Carskadon[45] in a longitudinal study that held sleep time constant. In the Carskadon report,[45] SWS declined by approximately 40% from Tanner stage 1 (ages 10 to 12 years) to Tanner stage 5 (ages 14 to 16 years). The fundamental question arises now whether the decline of SWS is reflected in altered sleep-wake processes as well. Several groups have presented data examining spectral EEG variables including SWA across adolescent development.[46,47] Gaudreau and colleagues,[47] for example, reported a similar nocturnal decline of SWA between children and adolescents.

Our group has begun to examine pubertal changes in the sleep EEG within the context of the homeostatic model. Sleep EEGs in 6 pre- or early pubertal (Tanner 1 or 2, ages 10.3 to 12.8 years, 5 girls) and 6 postpubertal adolescents (Tanner 5, ages 11.8 to 16 years, 3 girls) have been analyzed using spectral analysis.[48] We showed a significant reduction in EEG power density during NREM sleep at frequencies <2 Hz and 4–6 Hz for the mature versus the prepubertal participants. Total power in the low frequency band <2 Hz was reduced by 64% during NREM sleep. Furthermore, we saw an exponential overnight decay of SWA in both groups, with equal time constants of the decaying function. These findings converge to indicate that the homeostatic process involved in the dissipation of process S across sleep under controlled

sleeping conditions does not manifest a maturational change across pubertal development. The substantially lower amount of SWS and low-frequency EEG power across adolescence may rather reflect changes in underlying brain structure (e.g., declining cortical synaptic density) as noted by Feinberg.[49,50]

The accumulation of process S, however, does appear to differ in its expression across adolescent development, based upon preliminary evidence. A test of this process requires assessment of the homeostatic markers under conditions that involve an alteration of the usual daily sleep-wake schedule, such as napping as suggested previously, or sleep deprivation. We have examined the speed of falling asleep (sleep latency) using standard methods in adolescents during 36 hours of sleep deprivation.[51] These data indicate that sleep latencies during initial hours of extended wakefulness were longer in more mature participants than in prepubertal children, indicating that accumulation of process S across the day may occur at a slower rate in more mature adolescents.

Our preliminary analyses of the EEG in the sleep episode following an extended waking interval of 36 hours showed the expected increase in low frequency EEG power during NREMS sleep in both groups.[48] The prepubertal children, however, manifested a less pronounced (30%) average increase of low frequency power comparing recovery to baseline versus the mature adolescents (70%). These data indicate that the younger child's brain quickly reaches the maximal capacity to generate low frequency activity during sleep. We are currently modeling the spectral EEG data from the nights following baseline and extended wakefulness to determine whether parameters that describe the accumulation of process S change as a function of pubertal development. Although the preliminary analysis of the sleep latency data hints at such a developmental change, we do not yet feel confident in drawing this conclusion.

The homeostatic sleep–wake process plays out in another manner when the adolescent delay in the timing of sleep produces chronic insufficient sleep in many youngsters. Chronic sleep restriction becomes manifest when we examine speed of falling asleep.[29] For example, we showed that tenth-grade students with a "first bell" at 7:20 AM were able to fall asleep in fewer than 5 minutes on morning tests of sleep latency, never rising above about 10 minutes; indeed, many showed REM sleep in brief morning naps, coinciding with the time of their second-period class.[11] These data illustrate a powerful effect of homeostatic sleep regulation, as well as the impact of the circadian timing system (vis-à-vis the REM sleep finding). To the extent that the bedtime delay in older adolescents is mediated by intrinsic processes, the requirement for early rising to attend school inevitably results in inadequate sleep.

## THE CIRCADIAN TIMING SYSTEM (PROCESS C) DURING ADOLESCENCE

Our interest in the possibility that changes in the intrinsic circadian timing system accompany puberty stemmed from a self-report assessment of pubertal development and circadian phase preference in sixth graders.[37] These data showed that those children (particularly the sixth grade girls) who rated themselves as more physically mature also rated themselves as more "evening" type in their phase preference. We subsequently confirmed this circadian phase delay tendency in a laboratory study in

which Tanner stage was assessed by physician evaluations and circadian phase was measured by salivary melatonin[a] levels. Again, pubertal stage correlated with the circadian phase marker, such that more mature children showed a later phase of melatonin secretion offset.[52] We here also report that melatonin onset phase measured in 27 adolescents after controlled sleep-wake schedules was positively correlated with Tanner stage ($r = .54$; $P = .003$). These findings provide convergent evidence that circadian phase undergoes a delay in association with puberty, even under conditions controlling for psychosocial influences on sleep-wake patterns.

We should note that an alteration of the amplitude of the daily melatonin secretory patterns across pubertal development has been known for many years. A decline in nocturnal plasma melatonin levels across puberty was shown in cross sectional samples.[53,54] This decline was initially thought to be part of the hormonal cascade initiating pubertal development. In fact, the earliest reports of the reduction of melatonin secretion in pubertal children led to a speculation that melatonin was a "trigger" for puberty,[55] which was disputed strongly by others (cf. Ref. 56). Other reports, however, indicated that no change occurred in total excretion of the melatonin urinary metabolite, 6-hydroxymelatonin sulfate (6-OHMS), across puberty. The pubertal change in plasma melatonin levels was attributed to a change in the distribution of secreted melatonin in the larger body mass of the more mature adolescents with no change in the production of melatonin per se.[57] Recently, Griefahn and colleagues[58] examined urinary 6-OHMS excretion in a longitudinal sample. Their data lead to the interpretation that body size is the mediating factor for the pubertal decline in plasma melatonin levels, not falling melatonin secretion. No studies are available that have assessed plasma and urinary concentrations of melatonin simultaneously.

We recently analyzed data from a cross-sectional sample of adolescents in whom salivary melatonin was collected at 30-minute intervals across 18 hours during a "constant routine" protocol.[59] The salivary melatonin profiles of 14 participants (ages 9.6 to 12.9 years, 7 girls) at Tanner stage 1 and 12 participants (ages 11.8 to 14.4 years, 6 girls) at Tanner stage 5 were collected. Linear regression analysis of melatonin levels (area under the curve) and amplitude (maximum) including age, body mass index, phase preference, and Tanner stage, showed a significant contribution only for Tanner stage. Furthermore, we found (as have others[60–62]) large individual differences in melatonin levels. Our analyses, based on data collected under very controlled conditions of sleep timing, again raise the possibility that pineal secretion of melatonin declines during pubertal development. We can only speculate that this developmental change—whether mediated centrally or by secondary developmental characteristics such as body size—may signal a pubertal reduction in feedback of melatonin to the circadian timing system, which could alter the circadian signal to the sleep-wake system.[63] Shanahan[64] hypothesized, for example, that low melatonin

[a]Pineal melatonin secretion is controlled by the circadian timing mechanism (and feeds back on this system) to rise during the brain's nighttime and nearly cease during the daytime of the brain. Hence, melatonin has been dubbed the "hormone of darkness." Melatonin secretion can be suppressed by light, and such suppression is thought to be one indicator of the extent to which light affects the timing system. We use melatonin levels to mark internal time as the "hands" of the intrinsic circadian clock.

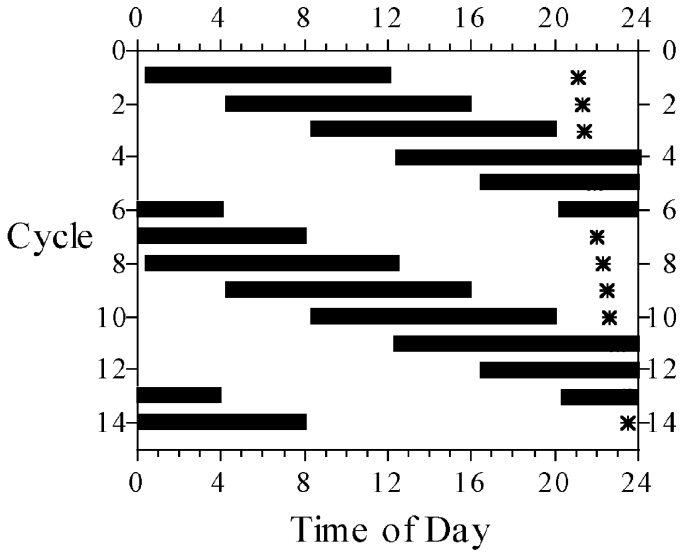


**FIGURE 2.** Schematic of the forced desynchrony protocol. Scheduled sleep episodes (*black bars*) are shown for a 28-hour day length for 14 consecutive cycles. Participants live in the laboratory under dim light conditions (~15 lux) so that dim-light melatonin onset (DLMO) can be determined from serial saliva samples. A hypothetical pattern of DLMO is shown by the *star symbols*, indicating that the circadian timing system is running free of the 28-hour day length at a period of about 24.2 hours.

amplitude reflects a diminished amplitude of the light-sensitive endogenous circadian pacemaker. Perhaps decreased melatonin amplitude in older adolescents plays a role in their tendency to manifest a delayed sleep phase.

Another mechanism predicted by circadian rhythms models is that a delay of circadian phase may be related to a lengthening of the period of the circadian clock, that is, a longer internal day length.[65] Under normal day-to-day circumstances, features of the environment that have a 24-hour period, particularly the light-dark cycle, entrain the internal clock to 24 hours. The phase angle with which the internal clock time aligns with the external day, however, is determined in part by the intrinsic circadian period: the phase angle of entrainment is delayed in parallel with the extent to which the internal day length exceeds 24 hours. Thus, we predicted that period of the circadian timing system may lengthen during puberty.

As mentioned previously, the method for measuring period is a bit arduous, involving prolonged laboratory stays under carefully controlled conditions. FIGURE 2 illustrates an experimental protocol we have used to assess period in adolescents by collecting serial measures of salivary melatonin onset and offset across 12 cycles on a 28-hour day( i.e., forced desynchrony). Our initial analysis of intrinsic period in adolescents showed that period appeared longer than reported by others in young adults, but we did not have adequate numbers of subjects to test the pubertal hypothesis.[66] We present here data from 27 participants who have completed the forced de-

synchrony protocol. We found no evidence of a pubertal change in period in this still rather small cross-sectional sample, but hope to acquire longitudinal data in order to re-examine this hypothesis. One of the major limitations of studying this phenomenon is the difficulty in making the measure in truly prepubertal participants because of the lengthy commitment to the laboratory stay. We compared the distribution of intrinsic period in our sample with samples of adults in whom period was also derived from melatonin phase markers in a 28-hour forced desynchrony paradigm.[67,68] The mean period of the circadian clock in adolescent participants (24.27 h) is significantly longer than in the adult samples (24.12 h). These comparisons are not conclusive, rather they are suggestive that longer internal day lengths may emerge in certain adolescents.

Another important feature of the circadian timing system is the phase-dependent sensitivity to light intrinsic to the clock resetting process.[69] Light occurring in the early part of the circadian night (evening and early nighttime) produces a delay resetting response in the clock, whereas light signals in the late night/early morning result in an advance resetting response (cf. Ref. 70). One marker of the effects of light on the circadian timing system is the suppression of melatonin levels by light.[40] We have hypothesized that the sensitivity of the circadian system to light may change during pubertal development in a manner that accentuates the tendency for a phase delay and have tested the hypothesis using melatonin suppression.[71] In brief, we suggest that a heightened sensitivity to evening light or a decreased sensitivity to morning light across pubertal development could result in the pubertal delay of sleep timing. An assessment of this hypothesis in 66 participants who received 4 levels of light on consecutive nights either in the late evening or early morning showed greater suppression to a low level of light administered in the morning for pre- and early pubertal participants than for late or postpubertal participants, and no differences in response to late night light.[72] These findings provide suggestive support for the hypothesis.

On the other hand, the most relevant assessment of the circadian response to light is actual phase realignment response to light signals. Thus, to what extent does the circadian timing mechanism actually reset its phase when an adequate light signal is given? We are currently evaluating this process by exposing adolescents who are Tanner stage 1 or Tanner stage 5 to a 2-hour light signal of 3,000 lux on two consecutive days. We administer light at times where the phase response curve predicts either a phase resetting delay or a phase resetting advance. Ultimately, we propose to acquire data across the entire phase response curve (PRC) for pre- and postpubertal adolescents, hypothesizing that the amplitude of the phase-resetting responses will be greater in the delaying direction or smaller in the advancing direction for the more mature group. If such developmental changes in PRC are operational, the permissive/responsive interaction of the behavioral and biological processes could set up a synergistic increase in pressure for adolescents to phase delay.

## INTERACTION OF THE HOMEOSTATIC AND CIRCADIAN PROCESSES DURING ADOLESCENT DEVELOPMENT

A number of findings predict that a critical developmental outcome during adolescence may be a change in the phase angle of the homeostatic to the circadian tim-

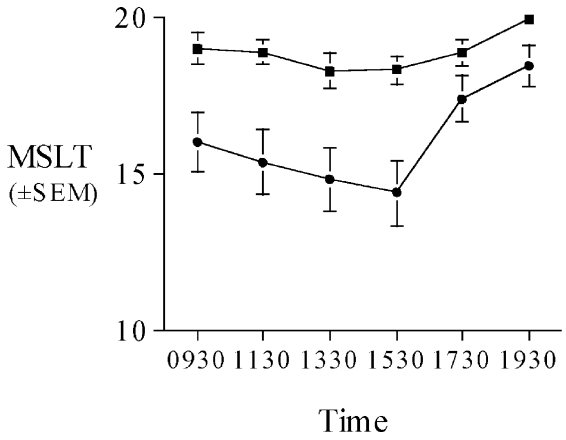


**FIGURE 3.** Sleep latency from Multiple Sleep Latency Tests (MSLT) in 25 pre-/early pubertal (Tanner stages 1 or 2; *squares*) and 27 mid-/late pubertal (Tanner stages 3, 4, or 5; *circles*) participants in a longitudinal sleep study.[29] Mean (± standard error) of sleep onset latencies indicate speed of falling asleep on tests scheduled at 2-hour intervals after 10-h nocturnal sleep episodes. More-mature participants fell asleep faster on the tests at 1530 and 1730, indicating augmented midday sleep tendency in spite of sleeping the same amount at night.[30]

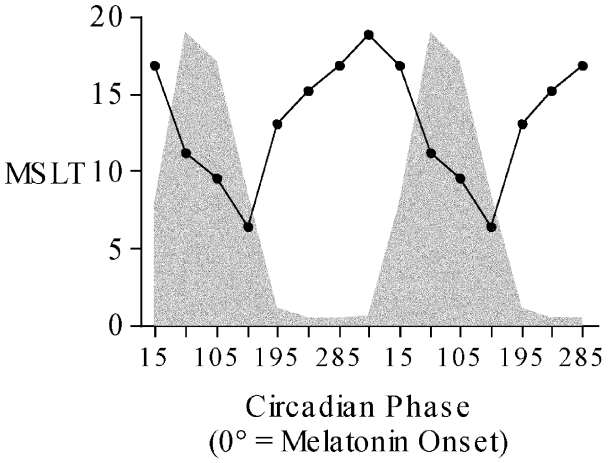


**FIGURE 4.** Circadian rhythm of sleep tendency as measured by MSLT on the background of averaged melatonin secretion (*gray shading*) from 10 adolescents studied in a 28-hour FD. The pattern displayed here is derived by averaging data from particular circadian phases (determined by the melatonin phase marker) irrespective of time awake.[74] As indicated in the text, the pattern appears paradoxical, since shortest sleep latency occurs at the end of the circadian night and long sleep latencies at the beginning of the circadian night. The rising latencies in the circadian day are conceptualized as indicating a clock-dependent alerting signal.

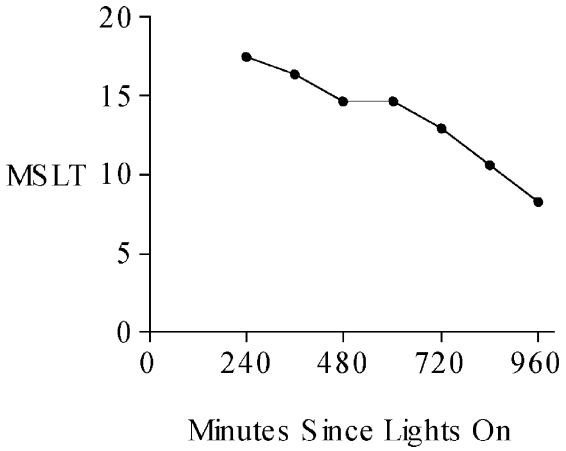


**FIGURE 5.** The MSLT sleep tendency pattern from the study described in the legend for FIG. 4 was derived by the average of sleep latencies in reference to the length of time a participant was awake regardless of circadian phase.[74] This curve is conceptualized to indicate the homeostatic process accumulating as a growing sleep propensity across the waking day. As described in the text, the pattern of sleep propensity across an individual's waking day will depend upon the phase alignment of sleep/wake to the circadian timing system. Alterations of this phase alignment can facilitate late bedtimes when clock-dependent alerting rises late in the day. On the flip side, the phase angle of entrainment favoring late bedtime also is associated with difficulty waking in the morning.[74]

ing system. One of our earliest findings regarding sleep tendency during puberty[73] demonstrated a paradoxical increase in sleep tendency in adolescence at mid- to late puberty compared to pre- or early puberty. FIGURE 3 illustrates this finding, which was initially interpreted as an increase in sleep pressure, even though total sleep time had not changed. We also speculated that the changed pattern of diurnal sleep tendency might indicate a reorganization of central behavioral organization, for example, to favor a "siesta" in more mature humans.[73]

Recent conceptual advances in combination with data acquired in the context of forced desynchrony allow us to reinterpret this finding as a pubertal change in the phase angle of entrainment of sleep and waking to the circadian timing system.[74] We have shown, for example, that each of the two systems—when examined in isolation—provides counterintuitive findings with regard to circadian timing. As FIGURE 4 illustrates, the circadian signal demonstrates fastest sleep onsets (greatest sleep tendency) at the end of the circadian night (inferred from the melatonin secretory pattern) and slowest at the start of the circadian night. Taken at face value, however, these data are paradoxical, since most would assume that humans should fall asleep fastest at the beginning of the night rather than at night's end. The homeostatic process when examined separately from the circadian rhythm, builds to greater sleep tendency across time, thus faster sleep onsets occur the longer one is awake (FIGURE 5). Alignment of these two processes, however, as occurs during normally entrained

conditions, solves the paradox: the circadian system, through "clock-dependent alerting," offsets the growing homeostatic pressure across the waking day. This association describes graphically the competing nature of these processes as described earlier.[74]

What is also apparent is that the alignment of these two processes will affect the balance of the two processes across the day. Thus, we now interpret our earlier findings of augmented midday sleep tendency in pubertal adolescents[73] to indicate a reorganization of the phase relationship of these processes. The mechanism underlying this realignment is not known, but it may be determined by the factors outlined above, such as alterations of intrinsic period or phase resetting properties of the clock mechanism. If the phase angle becomes realigned during late puberty, then the clock-dependent alerting later in the day can facilitate late sleep onset. Similarly, this phase realignment may manifest as a significant increase in the difficulty of waking in the morning. As more is learned about these underlying mechanisms and their association to the multiple behavioral influences on adolescent sleep timing, we may gain insights into opportunities for intervention.

## CONSEQUENCES OF THE SLEEP DELAY FOR ADOLESCENT BEHAVIOR

In the United States in particular, as well as in a few other industrialized societies, the changing adolescent sleep–wake system exists in the context of a relatively unforgiving educational structure demanding earlier school attendance in older than younger children. Whether the delay of the timing of sleep in adolescents is exclusively attributable to the psychosocial milieu or receives contributions from changes in the intrinsic regulatory processes, we observe inadequate and ill-timed sleep in a large number, if not a majority of young people. Our group has found, for example, that the estimated amount of sleep accumulated during the school week in youngsters in grade 6 averages 500 minutes, in grade 8 is 473,[75] and in grade 10, the average is 452 and grade 12, 420 minutes based upon objective monitoring in the field. Laboratory data indicates that the sleep need in these youngsters is closer to 555 minutes per night.[45,76]

As we learn more about the effects of chronic insufficient sleep, concerns grow about the potential for negative impacts on adolescents. For example, rates of automobile crashes attributed to falling asleep while driving are markedly higher for the youngest drivers. Indeed, a retrospective analysis of over 4000 such occurrences showed that the drivers' ages in just over 50% of crashes were 16 to 25 years.[77] Growing evidence also indicates that adequate sleep plays an important role in memory consolidation and learning processes,[78–80] though research specific to adolescents is scarce. The preponderance of evidence from a variety of studies examining the association of sleep patterns with academic performance indicates that too little and poorly timed sleep has a negative impact in children, adolescents, and young adults.[81] Furthermore, tardiness, absenteeism, and high school graduation rates in adolescent students have been linked to sleep schedules and early school starting times.[82]

Mood regulation also suffers with inadequate sleep. Time and again, our studies—whether observational or experimental—have found depressed mood at greater

rates in young people whose sleep is compromised (see Ref. 2, for example). Behavior disruption has also been noted as a concomitant of disturbed sleep in children with sleep disorders [83–86]; less is known about this association in adolescents.

Substance use, including caffeine, alcohol, and tobacco, is also greater in teens who sleep less.[87,88] Recent data in adults show that sleep is not simply for the mind, but affects metabolic processes as well.[89] Indeed, one epidemiologic study linked adolescent obesity with poor sleep patterns.[90] As sleep patterns are examined along with other lifestyle or medical outcomes, we can anticipate more negative associations of poor sleep to become apparent.

## CONCLUSION

The robust tendency for the timing of sleep to delay during adolescent development is undeniably associated with the changed psychosocial environment of the developing teen and may also rely on developmental changes in fundamental regulatory processes. For many teens, this sleep delaying pattern cascades into a chronic pattern of insufficient school-day sleep, forced arousals at a biologically inappropriate time, and resulting negative impacts on adolescent performance, behavior, mood, and other processes. Countermeasures that are implemented on a personal, family, community, or societal level need to acknowledge all the factors contributing to the issue. We feel that primary among the countermeasures is an acknowledgement of a positive priority for sleep and a need for a better understanding of the sleep-wake regulatory process through education and research.

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