Sleep and Circadian Rhythms in Adolescence

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Abstract
Purpose of Review The purpose of this review is to synthesize the current evidence regarding biological changes to the regulation of sleep-wake behavior in adolescence, summarize the impact of environmental factors (e.g., media use, school start times) on sleep, and discuss the implication of these biological and behavioral changes for adolescent emotional, physical, and cognitive development.
Recent Findings Although our basic understanding of the sleep regulatory process in adolescence has not shifted in recent years, emerging findings highlight the influence of environmental factors (e.g., media use) on sleep behavior. Furthermore, a flurry of recent experimental studies has bolstered our understanding of the influence of short sleep on cognitive and emotional functioning. Despite these advances, longitudinal data elucidating whether there are maturational changes in the impact to sleep loss on adolescent development are largely absent.
Summary A confluence of biological and environmental factors leads to short and ill-timed sleep among adolescents. Given the importance of sleep for the cognitive, emotional, and physical health in adolescence, the high prevalence of sleep loss in this population represents a public health issue.

Keywords Sleep · Circadian rhythms · Adolescent · Cognition · Health · Sleep need

Introduction

Adolescence, the transitional phase between childhood and adulthood, is characterized by rapid physical, emotional, and cognitive maturation. Interacting with and supporting these processes is sleep. Sleep, however, is not a stable phenomenon during adolescence—sleep behavior and circadian rhythms undergo marked changes during this developmental period.

In this review, we give an overview of the changes to sleep behavior and physiology during adolescence with an emphasis on literature published within the last 3 years. We also highlight recent evidence pertaining to the role of sleep in the emotional, cognitive, and physical development of adolescents. Given the focus of this review on literature published in the last 3 years, readers interested in a broader overview of the field are referred to the following two recent reviews [1, 2].

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Sleep Behavior

In their seminal paper, Terman and Hocking (1913) summed up the most prominent developmental progression in sleep patterns of adolescence as follows: "It may be that adolescence tends to change the individual from the vesperal to the matinal type of sleeper." [3]. That is, adolescents become more and more difficult to wake in the morning; they also note that adolescents stay up later in the evening. Terman and Hocking went on to say that "It is more probably due to the fact that…high school pupils…were required to do more evening work than the younger children." [3]. Furthermore, as had others in the late 19th and early twentieth century [4–6], Terman and Hocking identified a reduction in the amount of sleep (consequent to the adolescent delay) and longer sleep on weekends vs. school days. Research in the twenty-first century has not altered the observed patterns of adolescents delaying sleep, reducing sleep, and extending weekend sleep as they mature. Roenneberg, for example, elegantly displayed the delaying pattern across the second decade with his marker of the midsleep time on "free" nights [7]. The logical argument that more evening homework drives the later nights, reduces sleep, and challenges morning arousals has been augmented by findings in the latter years of the twentieth century and early years of this century that identified changes to biological processes as *intrinsic* factors that help account for this developmental change.

Recent studies report a similar pattern of later, less, and irregular sleep for adolescents in samples from many parts of the world. To identify a few, for example, these findings are seen in Saudi Arabian school children [8]; rural Canadian adolescents from farming communities [9]; Nigerian "school-attending" adolescents [10]; adolescent-age students in Delhi, India [11]; and adolescent students in southern Brazil [12]. Of interest are the kinds of external factors that arise as contributing to adolescent sleep. For example, the study of Saudi Arabian teens showed associations of short sleep to gender, socioeconomic status, daytime naps, and screen time [8]. Not all of these associations were in the expected direction; for example, adolescents reporting television or computer (Internet) screen exposure of 2 or more hours per day were less likely than others to report sleep deprivation. This unexpected finding is a mark of the inconsistencies of findings of associated factors.

The two factors receiving most research in recent reports regarding sleep duration and timing are electronic media use and school start time. The review paper of Cain and Gradisar (2010) provided a well-reasoned model for features of media use that may adversely affect adolescent sleep: (1) media use that directly displaces sleep; (2) media use resulting in increased arousal; and (3) a circadian delaying effect of evening light exposure [13]. The various components of this model have been supported in certain reports and not in others and

often are not clearly addressed. That said, Exelmans and Van den Bulck (2017a, 2017b) in two papers address displacement and arousal. As to the issue of displacement, they propose a dual process wherein media use can delay going to bed and further displace sleep by delaying the process of attempting to fall asleep [14]. With regard to presleep arousal, this team compared the impact on sleep of "regular" to "binge" evening TV watching, the latter presumed more arousing and, indeed, showing a more substantial impact on sleep of older adolescents [15]. Finally, Touitou et al. (2016) reviewed the theoretical basis for an impact of evening media use and light exposure on adolescent sleep [16].

When surveying other aspects of recent literature, most reports are cross-sectional and observational; however, a striking note is the increasing global nature of these investigations examining the association of electronic media use with disrupted sleep and/or daytime sleepiness. A study by Reynolds and colleagues (2019) identified an association in female Australian adolescents of social media use (i.e., email and instant messaging) in the hour before bed with increased reports of insufficient sleep (controlling for age) [17]. An Indonesian report [18] highlighted increased insomnia in association with social network site use, as well as academic stress. From Spain [19], we learn that Internet use time of adolescents mediates the association of sleep quality and academic performance. Arrona-Palacios (2017) from Mexico compared the impact of evening electronic media use in students who were enrolled in a school system with split classroom times, a morning (0730–1330) and an afternoon (1320–1900) group [20]. Although the afternoon group (regardless of media use) went to bed and woke up at later times, the overall association of evening media use on bed time and rise time was observed for all students. Gender and school shift interactions were also found for various types of media use in this interesting study. We note as well papers from Saudi Arabia [8], Nigeria [10], Singapore [21], and South Korea [22, 23] examining sleep behavior in the context of media use. One meta-analysis has appeared in recent years [24], identifying some inconsistencies but overall significant associations of evening electronic device use with shortened and disrupted sleep and daytime sleepiness. A final emerging construct that has been linked to social media usage and adolescent sleep outcomes is the construct of "fear of missing out" (FOMO), which Scott and Woods (2016, 2018) have linked to self-esteem and social anxiety [25, 26].

The second general topic commonly reported as affecting adolescent students' reduced sleep duration is an early start of the school morning, typically earlier than when students were younger. Again, this association has been made in many cohorts from many countries. The USA has produced much of this literature, primarily due to the common use of tiered school bell systems: youngest students scheduled to attend later than oldest students. Recent reports have focused on

efforts to improve adolescent sleep by modifying (delaying) the starting bell for school in this age range. These studies often suffer from a lack of control for other factors, often with a limitation of cross-sectional design, order effect, and lack of randomization, and students' self-report of sleep. One group in the UK attempted to account for such limitations by running a randomized controlled trial [27]. Unfortunately, this study ran into challenges when only two of the 100 schools they approached agreed to participate. The major reason for non-participation was the prospect faced by being randomized to the delayed start time (10:00 am).

Other less ambitious trials have met with better success, though somewhat mixed in terms of impact on sleep. The COMPASS study in Ontario, Canada, that included 49 schools [28] showed that minimal changes in school timing (10 min delay) resulted in reported gains of nearly 25 min in sleep length. By contrast, small advances in earliest school timing (e.g., 8:30 to 8:20 am) resulted in less sleep, but not when school start was later (e.g., 9:00 to 8:50 am). A South Korean study [29] of students' sleep when school start time was delayed until 9:00 am points out a concern voiced by many that delays of the school day will simply result in later bedtimes. Indeed, Rhie and Chae found an initial lengthening of reported sleep but after a year or two, the sleep duration advantage was lost due to students' shifting bedtime later. Most observational studies showing increases in sleep following a delayed start have limited follow-up. An alternative design that examines sleeping patterns in students where the starting times are different, i.e., not following a change per se, shows positive sleep outcomes for those with later timing. For example, the report of Nahmod et al. (2017) showed in data collected from a US national cohort study that students attending schools with earliest start times (7:00–7:30 am) compared with those with the latest (8:30 am or later) reported 46 min less sleep at night [30]. Whether the sleep gains are maintained after changing start times remains somewhat uncertain; however, Lo and colleagues (2018) found that when students, teachers, and parents were in support of the modification of the school schedule, the benefits were sustained at least 9 months later. These data were impressive as well because of the cultural framework in the locale (Singapore) that often favors academic success over the need for sleep [31].

We highlight two additional studies where implementation of delayed school start times resulted in changes to sleep as well as changes to academic performance. The 2018 paper of Dunster and colleagues showed that the delay of the school bell resulted in more sleep and better grades [32]. One of the important components of this study was that sleep was measured pre- and post-change with actigraphy. A gain of over 30 min of sleep with a start time delay of 55 min (7:50–8:45 am) was found. Furthermore, the median grades and school attendance were improved a year after the change. One study from the UK [33••] examining several academic

outcomes was remarkable for several reasons. First, the start time was delayed to 10:00 am; second, the follow-up was for 2 years; and third, the start time was reversed after the second year and the follow-up continued. Also of interest was the comparison "group," i.e., nationwide outcomes. This study showed sustained positive outcomes for school absences attributed to illness and school-level academic performance versus the national average. Both trends reverse trajectory following the return to an earlier school start schedule. We note that one of the most fraught issues for school districts in the USA that are attempting to change the starting times is the expense of changing the transportation system to accommodate the schedules. A recent report from a group at the MIT in Boston [34••] is unique in developing an algorithm that would address the school bell scheduling and bus route planning together to derive the most economically viable solution.

The Two Process Model of Sleep Regulation in Adolescence

Understanding the unique sleep behaviors of adolescents requires an understanding of how sleep regulatory systems change across this period. According to our current understanding, two processes, a homeostatic (Process S) and circadian (Process C), regulate the timing and duration of sleep. The homeostatic process builds in a saturating exponential manner during waking and dissipates during sleep. This system favors waking after long periods of sleep and sleep after prolonged waking. The circadian system on the other hand oscillates with a period close to 24 h independent of prior sleep, and favors sleep at certain times of day. The interaction of these two systems determines sleep propensity at any given time. Changes to these two processes during adolescent development provide the context within which sleep behavior occurs during this maturational phase.

Process S

The sleep homeostatic process is reflected in sleep EEG slow-wave activity (SWA; typically defined as low-frequency activity in the range below 4.6 Hz). SWA is sensitive to prior sleep/wake history, accumulating during the waking day and dissipating during sleep. Much of our knowledge about changes to the homeostatic system in adolescence comes from a seminal study conducted in 2005 [35]. In this study, the rate at which sleep pressure built up and was dissipated was modeled using a sleep deprivation (32 h time awake) protocol. The authors observed that sleep pressure builds more slowly in post-pubertal as compared with pre- or early-pubertal adolescents. The maturational slowing of the build-up of sleep pressure led the authors to hypothesize that this change in the homeostatic system allowed more mature adolescents to delay bedtimes as compared with younger adolescents. This

hypothesis was supported by a later study which found that younger adolescents (pre- /early-pubertal adolescents; mean age = 11.1 years) fell asleep faster than more mature teens (post-pubertal adolescents; mean age = 13.9 years) after 14.5 and 16.5 h of waking [36]. In line with the idea that sleep pressure builds more quickly in younger animals, a study of adolescent mice found that during a 4-h sleep deprivation phase, younger mice required more frequent intervention (21 times) to stay awake than more mature adolescent mice (7 times) [37].

In the past few years, studies have increasingly used sleep restriction/deprivation protocols in adolescents to further query the sleep homeostatic process. One study in adolescents between the ages of 9.9 and 14 years by Campbell and colleagues examined the impact of four nights consisting of three different doses of sleep (7, 8.5, and 10 h) on SWA [38]. These authors found no differences in SWA among the three sleep opportunity conditions when using the same amount of NREM sleep in the analysis. Another study in somewhat older adolescents (15–19 years) of five nights of 5 h time in bed— markedly less sleep than the Campbell study—found a substantial increase in slow-wave energy, a metric that takes into account the cumulative amount of SWA across the entire sleep period [39]. Whether the differences in findings between the two studies are due to differences in the experimental paradigm (i.e., five nights of 5 h versus four nights of 7 h), or the analysis method (use of SWE versus SWA, comparison to baseline [39] versus comparison across sleep doses [38]) is unclear. Another factor that may account for the disparate findings is the age of the participants studied. In a study of adolescent mice, only those mice at the midpoint of puberty or older showed an increase in SWA after acute sleep restriction (4 h), whereas younger mice did not [37]. This may be due to a ceiling effect that may prohibit the increase in SWA in younger brains, which already exhibit high SWA under well-slept conditions.

In contrast to the build-up of sleep pressure which undergoes a maturational shift during adolescence, evidence from longitudinal [40] and cross-sectional studies [35, 41, 42] to date suggests that the dissipation of sleep pressure does not change across adolescence. The implication of this finding is that sleep need does not change across adolescent development. This conjecture is supported by a series of experimental studies reviewed below in the "Adolescent Sleep Need" section.

Process C

The circadian timing system (Process C) is the second process of the model that works together with the homeostatic sleep system to regulate sleep timing. The approximate 24-h (circadian) timing system, the center of which has been localized to the suprachiasmatic nucleus (SCN) of the hypothalamus, is a self-sustaining, genetically-regulated system. Among many physiological and behavioral rhythms, the circadian system signals fluctuations of more or less sleep propensity across the 24-h day regardless of prior sleep/ wake duration. Early studies suggested that adolescents develop more evening-like tendencies (in sleep/wake behavior and peak performance) as they become older and progress through puberty. In now a seminal finding, Carskadon and colleagues confirmed that the central circadian clock measured objectively with salivary melatonin is later in adolescents who are at a more mature puberty stage compared with their less mature peers, despite similar sleep/wake timing conditions [43]. Similar findings are seen in other non-human mammals; the most recent report by Melo and colleagues [44] observed delayed activity rhythms in juvenile marmosets relative to their adult parents.

The mechanisms underlying this puberty-related phase delay shift in the circadian timing system are still unclear, though works within the past few years have provided some key insights. A long-standing hypothesis posits that the endogenous circadian period (internal day length) lengthens as adolescents transition through puberty. In humans, the endogenous circadian period is not exactly 24 h, and usually runs a little longer than 24 h. A circadian period longer than 24 h favors more evening tendencies, whereas a circadian period shorter than 24 h favors morningness. Therefore, a lengthening of the endogenous circadian period as adolescents mature would be consistent with the evening-like tendencies that are also occurring during this time. Data from animal (McGinnis, Lumia, Tetel, Molenda-Figueira, and Possidente 2007) and human (Carskadon and Acebo 2005) studies provided initial support for this hypothesis showing longer circadian period in adolescents compared with adults. A recent study, however, does not support this hypothesis [45]. When run in the same laboratory protocol during the same season, late- and post-pubertal adolescents (Tanner stage 4 or 5; 14.3–17.8 years) showed similar free-running circadian periods compared to adults (30.8– 45.8 years). Both age groups showed an average circadian period of about 24.2 h. Ancestry differences were noted; free-running circadian period was shorter in African-American participants compared with those of other ancestries (mostly White), but this difference was primarily driven by the adult group. Trends for sex differences in both age groups were also reported. These results suggest that late- and post-pubertal adolescents have adult-like circadian periods, and do not provide support for free-running circadian period changing concurrently with sleep behavior during late adolescence. Changes to free-running circadian period may be occurring earlier in development [46], though analyses that consider ancestry and sex differences in these earlier developmental patterns may be needed.

A second area of investigation that has received attention in the past few years is the sensitivity of the adolescent circadian

system to light. The circadian system can shift earlier (advance) or later (delay) in response to light exposure, but the direction of shift depends on the time of day that the light is received by the eye. In general, light exposure in the evening or first part of habitual sleep shifts the circadian system later (phase delay) and light exposure in the second half of habitual sleep or shortly after waking shifts circadian rhythms earlier (phase advance). These responses are illustrated as a phase response curve (PRC) to light. The light sensitivity hypothesis posits that a delayed circadian system as seen in older adolescents could develop as a result of a blunted response to morning phase advancing light, an exaggerated response to evening phase delaying light, or a combination of both. A few recent studies have set out to test this hypothesis. In a group of adolescents aged 9.1–15.9 years who spanned the pubertal transition [47], Crowley and colleagues assessed melatonin suppression responses to 0, 15, 150, or 500 lx of light delivered in the evening (between 23:00 and midnight) or the morning (03:00–04:00). Unexpectedly, early- to mid-pubertal adolescents (Tanner stages 1–3) showed *greater* sensitivity to light compared with the late- and post-pubertal adolescents (Tanner stages 4 and 5) in the evening. These findings contradict the original hypothesis and in fact, the younger group was so sensitive to evening light that melatonin was suppressed in light levels (~ 15 lx) far less than normal room lighting in most homes (~ 100 lx). In the morning, 500 lx of light-suppressed melatonin more in the early- to mid-pubertal group compared with the late- and post-pubertal group, but this difference was only a trend ($p = 0.06$) and it was not sustained across the 1-h light exposure. Taken together with other studies of circadian sensitivity to light in young children [48] and school-aged children [49], it is more likely that light sensitivity decreases with age and is not related to puberty.

While melatonin suppression provides an index of whether the circadian system can "see" the light stimulus, it does not provide the functional outcome of phase shift—a change in timing of the central circadian clock—to test whether older adolescents delay more to evening light exposure, advance less to morning light exposure, or both. Crowley and Eastman (2017) tested this hypothesis by constructing a phase response curve (PRC) to light in late- and post-pubertal (Tanner stages 4 and 5) adolescents aged 14.3 to 17.8 years [50]. The adolescent phase response curve to bright light showed a predictable pattern with the largest delay shifts occurring in the hours around habitual bedtime, and the largest advance shifts occurring around habitual wake-up time. Unexpectedly, the amplitude of the delay responses and the advance responses were symmetrical, suggesting that late- to post-pubertal adolescents do not show large delay shifts and small advance shifts in response to bright light as was predicted. Also, inconsistent with previous animal data, the older adolescent phase response curve does not differ from a phase response curve constructed in adults (30–45 years) using the

same protocol (Crowley, unpublished). Nagare and colleagues also reported no age-related differences in melatonin suppression between adolescents (13–18 years) and individuals who were older (24–55 years) when equalizing characteristics of the light source, like dominant wavelength [51]. Whether phase shift responses of older adolescents differ from younger pre- or early-pubertal adolescents is still unclear.

These recent studies begin to discount the hypothesis that modulation of light sensitivity underlies the puberty-related delay in circadian phase and thus delay of sleep timing. What is more likely is the opportunity for light exposure in the evening increases as adolescents get older. Older adolescents (15–18 years) fall asleep later into their biological night marked by the onset of melatonin compared with younger adolescents (9–13 years) [52]. This difference is likely due to a slowed accumulation of waking homeostatic sleep pressure allowing older adolescents to stay awake later into their biological night. These changes to sleep physiology propping up evening alertness increases the likelihood of light exposure later into the evening, a time when the circadian system is particularly sensitive to phase delaying light [50]. As described in an updated version of Carskadon's "Perfect Storm" model [1], exposure to light at this time could potentially feedback and reinforce evening alertness and delayed sleep onset in older adolescents.

Adolescent Sleep Need

The "Perfect Storm" model provides a comprehensive overview of factors that limit sleep duration of adolescents, the idea being that there is an optimal amount of sleep—or sleep need—that is curtailed by these intrinsic and extrinsic forces. While sleep need recommendations for adolescents have been offered for generations, empirical research in support of such recommendations has, until recently, been scant [53]. In 1913, Terman and Hocking summarized sleep need recommendations for children and adolescents (see Table 1). They note the "…large number of estimates based upon opinion and loose observation, but no answer based on data of scientific validity" (p. 138) [3]. It would be another 60 years before one

Table 1 Estimates of sleep need in hour from Terman and Hocking, 1913 [3]

	Age (years)					
	13	14	15	16	17	18
Dukes (1899)	10	10	9.5	9	9	8.5
Bernhard (1908)	10	10	9.5			
Hertel (1885)	9.5	9	9	8.75		
Claparède (1905)	9.5	9	9			
Manaceïne (1897)	8.5	8.5	8	7–8		

of the earliest studies to address this research question would be conducted [54]. Carskadon and colleagues conducted a longitudinal study whereby 19 adolescents spent 3 consecutive days and nights in a sleep laboratory every year for 3 years and were given 10-h sleep opportunities each night. This approach assumes that the amount of sleep obtained in conditions of ample opportunity indicates biological sleep need. Results revealed that adolescents obtained an average of just over 9 h of sleep per night and that this duration did not change across adolescence.

Despite the importance of having a solid evidence-base for sleep recommendations, relevant research directly addressing this question has been largely absent until the last 4 years, during which several divergent approaches have been taken to address the gap in the literature. Expert panels from the American Academy of Sleep Medicine (AASM) and the National Sleep Foundation (NSF) both used the RAND appropriateness method [55] to derive sleep recommendations [56, 57]. The panels reviewed the extant literature on sleep duration and various outcomes and voted on the sleep durations that they deemed as appropriate or not appropriate across the lifespan. Both panels deemed that 8 to 10 h sleep per night was optimal for adolescents aged 13 to 18 years. This approach, however, is subject to a range of potential biases. Further, most of the studies included did not directly assess optimal sleep duration for relevant outcomes, but rather examined linear, cross-sectional relationships in which causation cannot be inferred and "optimal" sleep is not estimated.

In 2016, Ojio and colleagues estimated the sleep duration needed for optimal mood using cross-sectional data from 15,637 Japanese adolescents aged 12 to 18 years [58]. They calculated that sleep durations ≥ 8.5 h were optimal for males, while sleep durations ≥ 7.5 h were optimal for females, with adolescents sleeping significantly more or significantly less than these amounts reporting heightened depressed mood and anxiety. This study extended existing knowledge by evaluating non-linear relationships between sleep duration and mood and by using these data to estimate sleep need associated with lowest risk of mood deficits. This approach also has inherent limitations, including the inability to infer causation and the tendency associated with this approach whereby the points of lowest risk tend to approximate the norm for that population [59]. Thus, being an outlier within your own culture seems to be associated with heightened risk [59]. The fact that the sleep durations that define "outliers" vary so widely across cultures is problematic when considering the ability of this method to accurately estimate biological sleep need in a generalizable fashion.

Fuligni and colleagues similarly estimated sleep durations needed for optimal mood functioning [60••]. They examined nightly sleep duration and next-day distress (the sum of depression and anxiety items) for 2 weeks in a group of 419 Mexican American adolescents. Findings supported a non-linear association between sleep duration and mood. The sleep duration associated with lowest levels of next-day distress was 9 h per night, with both too much and too little sleep associated with heightened next-day distress. They also reported that sleep need was higher in younger adolescents and in adolescents with elevated psychopathology.

More recently, Short and colleagues [61••] utilized an experimental design to estimate sleep need using two approaches. First, they estimated sleep need from extended sleep opportunities. They also used a dose-response design from which the amount of sleep adolescents would need to obtain to sustain optimal attention performance across wake was estimated [61••]. These different approaches converged in their findings. Given extended sleep opportunities, adolescents obtained approximately 9 h of sleep per night, consistent with earlier findings [54], while the amount of sleep needed to maintain optimal sustained attention performance was estimated to be an average of 9.35 h of sleep per night.

Overall, the field has made significant advances in our understanding of, and evidence for, adolescent sleep need over recent years (Table 1). Current evidence-based estimates largely concur with those summarized by Terman and Hocking over 100 years prior that were based on "…opinion and loose observation…" [3]. Taken together, these divergent studies support sleep durations of 8 to 10 h per night for adolescents. Estimating sleep need, and the related goal of defining long and short sleep in adolescents, is fraught with challenges, not the least of which is accounting for inter-individual variability [62]. Relying upon naturally occurring sleep durations to estimate sleep need for optimal functioning, or even to examine the relationship between sleep duration and functioning, is problematic because of the paucity of adolescents who regularly obtain sleep in the recommended range [63]. The relative absence of adolescents whose normative sleep falls within the recommended range means that there is often a restriction in the range of sleep durations which potentially limits these studies from accurately estimating optimal sleep. Chronic sleep restriction in the absence of causal medical or psychological conditions is so widespread it has become the "norm" for most adolescents, while long sleep is relatively rare [64]. In normative samples, it is unclear whether long sleep causes poor outcomes or if it is a consequence of poor outcomes. Further dose-response studies including a range of sleep durations are needed to estimate optimal sleep for a range of different outcomes and thus provide a rigorous base on which to provide sleep duration recommendations.

Implications of Adolescent Sleep Restriction

The chronic sleep restriction that many adolescents experience summarized in the above sections negatively impacts multiple domains of functioning. In the below section, we summarize areas where research efforts have focused; however, we note

that the impact of sleep loss is far reaching and that many disorders influence and are influenced by sleep loss.

Sleep and Cognition

Adolescence is an important developmental period with regard to the acquisition of cognitive abilities [65] since academic performance in high school influences future career opportunities [66]. Current evidence suggests that sleep supports multiple cognitive domains in adolescents, from boosting attention to promoting long-term memory consolidation and having a positive influence on the development of semantic schemata [67]. Experimental and observational studies have shown that the chronic sleep restriction that is characteristic of adolescent sleep has serious consequences for cognitive function [68], negatively impacting processing speed [69–71], sustained attention [68, 69], working memory [68, 69, 72], long-term memory consolidation [67, 68], psychomotor vigilance [68], executive function [68, 69], inhibition [72], and cognitive flexibility [72].

Although the supportive role of sleep with regard to cognitive function has been extensively studied and established in adults, only recently have experimental studies begun to systematically address the impact of sleep restriction and deprivation on cognitive performance in adolescence. These studies attempt to replicate what happens in the "real world" and typically compare multiple nights (4 to 7 nights) of sleep restriction (5 h to 6.5 h TIB) to adequate sleep (9 h to 10.5 h TIB) in order to study the impact of sleep loss on adolescent cognitive functioning. The results of these studies are clear—a few hours of sleep loss over several days significantly impact multiple domains of cognitive functioning, including information processing speed [70, 71], sustained attention [69], working memory [69], executive function [69], declarative memory [73–75], and spatial memory [73].

Interestingly, not all domains of cognitive function are equally impacted by sleep loss. In a series of studies using experimental manipulation of sleep combined with measurement of several cognitive domains, the largest effect sizes were found for sustained attention (psychomotor vigilance task; PVT) and processing speed, as compared with higher order cognitive functions, such as executive functions [69–71]. Furthermore, higher order cognitive functions may more quickly return to baseline levels after recovery sleep. For example, following 1 week of significant sleep restriction (5 h TIB), two nights of recovery sleep (9 h TIB) were insufficient to achieve a complete recovery in processing speed [69], while performance on working memory and executive function task returned to near-baseline levels.

Napping can in part ameliorate the detrimental effects of sleep restriction on processing speed [71]. In the aforementioned studies, participants were randomly assigned to either a 9-h, 6.5-h, or 5-h TIB combined with a 1.5-h afternoon nap.

Thus, total sleep opportunity was the same in the 6.5 h and 5 h + 1.5 h nap conditions. Participants who were given a nap opportunity had better sustained attention [76], working memory and executive function [69], faster speed of processing [71], and declarative memory [73] compared with those whose sleep opportunity was confined to nocturnal sleep. We note that these effects were strongest in the afternoon and evening following the nap as compared to morning performance prior to the nap.

More recent evidence suggests that chronotype may play just as important a role in academic performance as sleep duration. A study comparing school performance in early and late chronotypes found that students with late chronotypes had worse grades [66]. Surprisingly, this study found that chronotype was a better predictor of academic performance than sleep duration, suggesting that the association between these factors may be driven by daytime somnolence or a mismatch between environmental demands and biological predisposition [66]. Along the same lines, a study by Philipps and colleagues which used a novel metric, sleep regularity index (SRI), to quantify the regularity of sleep timing (bed and rise times) using sleep diaries in 61 undergraduate students for 30 days [77] found a positive correlation between sleep regularity and academic performance [77]. In other words, a pattern of more regular sleeping was associated with better academic performance [77]. Therefore, other measures such as chronotype and sleep regularity may also be important in sustaining and supporting academic and cognitive performance.

Sleep and Mental Health

Adolescence is a sensitive period for the emergence of sleep and mood problems [78] in non-psychiatric samples. Previous research has suggested a bidirectional relationship between sleep and mental health, where poor sleep predicts later mental health problems, while disrupted sleep can exacerbate an existing disorder. More recent epidemiological as well as experimental studies continue to show that adolescents who do not get sufficient sleep suffer from diminished mental well-being [79–81]. For example, in a study of 27,939 adolescents, 1-h less of weekday sleep was associated with greater odds of feelings of hopelessness, suicidal ideation, and substance use [82].

Not only is sleep duration an important factor with regard to mental health, but self- /parent-reported [81–85] and objectively measured [78, 86] sleep quality is also associated with mood [81, 83, 84, 86], mental well-being [78], behavioral difficulties [78, 82, 87], difficulties regulating emotion [2, 78, 88, 89], psychiatric disorders [79, 81, 82, 90, 91], and aggression [89]. Thus, the higher the sleep quality adolescents experience, the better their emotional, social, and behavioral performance [88].

Chronotype appears to be another important factor when it comes to adolescent mental health. For example, an observational study including 29,635 students between the ages of 10 and 18 years investigated the role of chronotype on mental health, finding that a later chronotype was associated with worse mental health, independent of sleep duration and across internalizing (e.g., anxiety, depression) and externalizing (e.g., aggressive behavior, delinquency) mental health domains [92]. Other studies examining circadian preference found that eveningness is linked to a higher probability of rule-breaking behavior [93] as well as conduct problems [93], lower self-regulation [94], attention deficit/hyperactivity problems [93], affective problems [93], and somatic complaints [93], when compared with a circadian preference towards morningness [93, 94].

While chronotype is largely genetically determined in adolescence [95], there is evidence that environmental factors may mediate this relationship. For example, later school start times were found to be associated with greater psychological health in adolescents [96–99], although causality could not be inferred. In line with this idea, another study demonstrated that school start times may serve as a moderator in models of adolescent sleep and their daily functioning [98]. In an observational study, 146 adolescents (mean age 16.2; SD = 1.0) wore an actigraph that assessed their bedtime, time in bed, sleep onset latency, and rise time. The measurements took place during a 15-day vacation and offered relatively unconstrained sleep opportunity [86]. In the aforementioned study, more variable time in bed as well as more variable sleep onset latency were found to be linked to poorer self-reported sleep quality, which was in turn associated with more negative mood [86]. This effect was mediated by poorer perceived sleep quality [86]. These findings suggest that objective intra-individual sleep variability is relevant to how adolescents perceive their sleep and their mood [86]. The authors posit that reducing variability in sleep timing and duration and in turn variability of sleep onset latency may decrease sleep complaints and improve mood in adolescents [86].

To summarize, current evidence suggests that sleep duration, quality, and chronotype all impact mental health and mood in non-psychiatric populations. While, the relative contribution of each factor is unclear and somewhat difficult to disentangle, current evidence suggests that each may confer its own unique risk.

Sleep and Physical Health

Although most research has focused on how sleep supports brain function, it is becoming increasingly clear that sleep also benefits the body. During adolescence, a period of rapid bodily growth, sleep supports physical developmental and wellbeing. For example, negative associations between short sleep duration and obesity [100–103], adiposity [80],

cardiometabolic biomarkers [80], type 1 diabetes mellitus [104], asthma [105], and headache as well as migraine [106] and menstrual problems [107] have been found. We note that while our review focuses on the associations between physical health and sleep in the general population, most medical disorders (e.g., migraine) are accompanied by disruptions in sleep, which may in turn exacerbate the underlying disorder.

Worldwide, obesity rates are increasing, with estimates that approximately one in five children and adolescents are overweight or obese [108]. The chronic sleep restriction observed in adolescents may be one of many factors that contributes to this rising trend. In a systematic review of the relationships between objectively and subjectively measured sleep duration and health indicators in children and youth between 5 and 17 years of age, longer sleep duration was associated with lower obesity levels [80]. Furthermore, a week long cross-sectional actigraphy study in 528 (mean age 14.4; SD = 2.1 years) Mexican youth found that adolescents with sufficient sleep had lower BMIs than those with insufficient sleep duration [109]. Furthermore, among the insufficient sleepers, those with regular sleep timing (e.g., stable sleep) had slightly lower BMI as compared with adolescents with irregular sleep timing.

Along the same lines, chronotype has been found to be linked to physical health (e.g., more headaches, stomach aches, back aches, dizziness, and worse self-related health) [110]. A later chronotype was related to worse physical health and also unhealthy behavior (e.g., daily soft drink consumption, smoking, screen time) in Canadian adolescents aged from 10 to 18 years [110].

Further studies are required to examine the circumstances under which physical health is particularly vulnerable to sleep deprivation and vice versa as well as to deepen knowledge about sleep quality and chronotype and their influence on physical health. To assess physical health problems as well as the sleep complaints of adolescents is crucial and may serve as an important domain for clinical interventions.

Conclusion

During adolescence, sleep duration and timing are constrained by environmental factors, resulting in sleepy teens. While mounting evidence suggests a stable need for about 9 h of sleep, changes to the bioregulatory processes of the homeostatic and circadian timing system during adolescence push sleep later, while school start times pull sleep earlier. This tug of war between biological and societal demands results in a pattern of insufficient and irregular sleep, with consequences for cognitive, mental, and physical health. Though the field has made rapid progress in the past 3 years, many open questions remain. One looming challenge is to make use of longitudinal data to map developmental trajectories and

identifying factors that make individuals vulnerable or resilient to irregular or short sleep. Another is to communicate research findings to stakeholders, such as parents, educators, and policy makers, to improve the sleep of adolescents.

Compliance with Ethical Standards

Conflict of Interest   The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent   This article does not contain any studies with human or animal subjects performed by the author.

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