

Sleep's role in the development and resolution of adolescent depression

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Abstract | Two adolescent mental health fields — sleep and depression — have advanced largely in parallel until about four years ago. Although sleep problems have been thought to be a symptom of adolescent depression, emerging evidence suggests that sleep difficulties arise before depression does. In this Review, we describe how the combination of adolescent sleep biology and psychology uniquely predispose adolescents to develop depression. We describe multiple pathways and contributors, including a delayed circadian rhythm, restricted sleep duration and greater opportunity for repetitive negative thinking while waiting for sleep. We match each contributor with evidence-based sleep interventions, including bright light therapy, exogenous melatonin and cognitive-behaviour therapy techniques. Such treatments improve sleep and alleviate depression symptoms, highlighting the utility of sleep treatment for comorbid disorders experienced by adolescents.

Adolescents (from pubertal onset to 21 years of age) are the most chronically sleep-restricted subpopulation across human development¹. Data from across the world suggest that adolescents sleep too late and too little². Such meta-analytical and descriptive data show similar patterns of adolescent sleep in Western and Eastern societies^{3–5}. Sleep onset for adolescents becomes later as they age (both on weekdays and weekends)^{3–5}, a phenomenon not observed during any other decade in life¹. A lack of morning commitments allows adolescents to potentially obtain optimal sleep duration (9.0–9.3 hours)⁶ on weekends. However, they are not afforded the same sleep opportunity on weekdays owing to the need to rise earlier than their natural wake time to attend school⁷. This restricted sleep opportunity is especially pronounced in countries that implement an early school start (for example, 7:30 am in the USA)⁷. Increases in sleep duration on school nights occurs when school start times are delayed⁸. Indeed, the quarantine associated with the COVID-19 pandemic eliminated the need for morning travel and therefore provided an extended sleep opportunity for adolescents, who showed increased sleep duration accordingly^{9–11}. However, the return to in-person learning is accompanied by a return to restricted sleep for adolescents¹¹. Although the American Academy of Pediatrics and the US Centers for Disease Control and Prevention have recommended delaying school start times to match adolescents' natural tendency for delayed sleep timing, such policy changes have been largely unsuccessful — and are therefore not the solution¹².

The rapid cascade of events that uniquely affect adolescent sleep begin around the beginning of high school and puberty¹³. This coincides with a period of increased risk for the onset of low mood and depression in adolescents¹⁴. Approximately 2.6% of adolescents experience a depressive disorder at any given time¹⁵, with the cumulative frequency of depression rising to 20% by the end of adolescence¹⁶. Female adolescents are twice as likely as male adolescents to experience depression^{16,17}. Other risk factors include having a parent that has experienced depression^{18,19}, exposure to stressful life events^{20,21} and experiencing bullying²². Experiencing depression in youth can have long-term negative impacts, including an increased risk of further episodes of depression as an adult^{23,24}, and developing other mental health disorders^{25–27}. Adolescence-onset depression has also been associated with educational underachievement, low income levels and unemployment^{28–30}, and is a risk factor for suicidal behaviour³¹.

Depression is characterized by a diverse range of symptoms. From the nine symptom domains identified by the *Diagnostic and Statistical Manual of Mental Disorders* edition 5 (DSM-5; REF.³²), more than 1,000 symptom combinations can be computed that meet the diagnostic criteria³³. Little is known as to whether different combinations of symptoms at presentation are sufficient to form distinct subgroups³⁴. One study suggests that there might be subtypes of depression that are primarily characterized by severity, with less severe groups (for example, fewer symptoms and lower risk) still presenting with high levels of somatic symptoms, such as sleep disturbance³⁵. Among depressed adolescents, sleep problems (92%)

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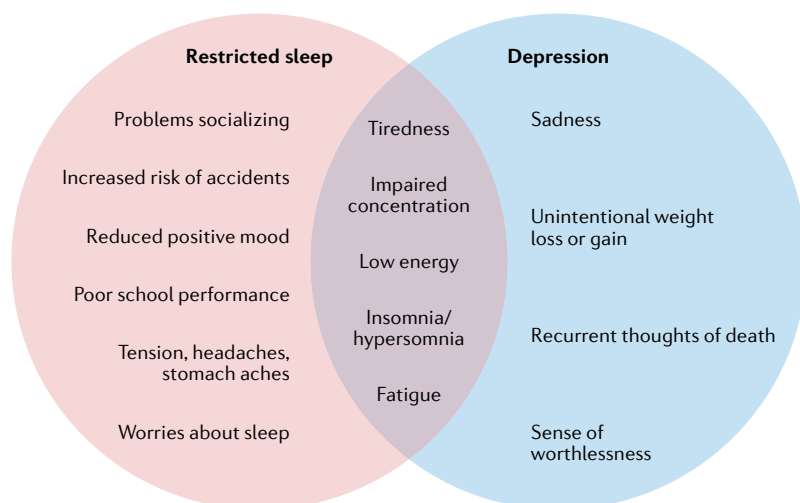


Fig. 1 | Unique and shared symptoms of restricted sleep and depression in adolescents. Symptoms of restricted sleep, depression symptoms and common symptoms according to the DSM-5 (REF.³²) and the International Classification of Sleep Disorders, 3rd edition (ICSD-3) (REF.⁶¹).

are more frequently reported than low mood (84%)³⁶. Indeed, there is overlap between symptoms of depression and those resulting from poor adolescent sleep (FIG. 1). However, rather than follow the notion that sleep problems are a symptom of depression, the scientific evidence suggests the reverse, especially during adolescence³⁷.

In this Review, we consider how adolescent sleep problems might contribute to the emergence of depression during this developmental stage. First, we describe the two main bioregulatory processes that drive sleep delay for adolescents: reduced sleep homeostatic pressure and delayed circadian timing. Next, we discuss how the consequences of these biological sleep characteristics — delayed sleep patterns, restricted sleep on school nights, and a long time taken to fall asleep at night — have biological and psychological implications for depression. Finally, we describe three evidence-based solutions that target the main contributing factors to adolescent sleep problems: bright light therapy, exogenous melatonin and cognitive-behaviour therapy. Although both insomnia and circadian rhythm disorders are present during adolescence³⁸, the scientific literature and clinical trials are weighted towards circadian rhythm disorders. Thus, we focus on delayed sleep–wake phase disorder here.

Two-thirds of depressed adolescents have at least one comorbid disorder³⁹. After sleep disorders, anxiety is the most common comorbid mental health problem in adolescents with depression^{36,40}. Around 75% of adolescents with depression experience symptoms of anxiety⁴¹ and up to a quarter of depressed adolescents also meet diagnostic criteria for generalized anxiety disorder^{36,42}. Although we focus on depression in this Review, we briefly summarize the interplay between sleep, depression and anxiety in BOX 1.

The biology of adolescent sleep

The timing of when humans sleep is primarily governed by two distinct, yet inter-related, biological processes. Together, these form the ‘two-process model of sleep’⁴³.

Here, we describe these biological sleep processes, how they change over adolescent development, and how a late sleep onset may ensue.

Development of sleep homeostatic pressure. Human physiology strives for sleep–wake equilibrium. The sleep homeostatic process (also called process S⁴³) depends on previous sleep and wake time. Sleep pressure progressively accumulates throughout wakefulness, and dissipates during sleep. Although the neuroanatomical locus of homeostatic regulation is not yet known, several biochemical factors, such as adenosine, have been shown to be involved⁴⁴. The most prominent marker for the sleep homeostatic process is sleep electroencephalogram (EEG) slow-wave activity, which is defined as activity within the EEG frequency range of 0.75–4.5 Hz during non-rapid eye movement sleep. Slow-wave activity demonstrates dose-dependent increases in response to prolonged wakefulness⁴⁵. That is, the greater the amount of time spent awake, the greater the slow-wave activity during sleep, indicating a homeostatic balance between wake and sleep.

The sleep homeostatic process evolves throughout the first two decades of life. Newborn infants have low tolerance for extended wakefulness. The development of an intrinsic bioregulatory process during infancy and young childhood enables multiple sleep periods throughout the 24-hour day to converge into a single, monophasic nocturnal sleep episode⁴⁶. During adolescence, a further ‘developmental leap’ occurs. Slow-wave activity declines by approximately 40%, with the greatest reductions occurring at approximately 12.5 and 13.5 years of age for girls and boys, respectively^{47,48}. This decline in slow-wave activity is associated with synaptic pruning. Some researchers suggest that diminished synaptic activity during wakefulness underlies the diminished need for sleep recuperation during adolescence. Thus, the lower slow-wave activity during adolescent (compared to childhood) sleep reflects a reduced need to recover from extended wakefulness^{49,50}.

Furthermore, the accumulation of sleep pressure during wakefulness undergoes marked developmental changes during adolescence. In a seminal study⁵¹, post-pubertal adolescents showed reduced sleep pressure during sleep deprivation compared to pre-pubertal adolescents. This finding suggests that maturation during adolescence allows individuals to remain awake longer. Sleep propensity (the ability to transition from wakefulness to sleep) is lower in mature versus prepubertal adolescents following 14.5 hours, 16.5 hours and 18.5 hours of sustained wakefulness⁵². Similar patterns have been found in adolescent mice, whereby mature compared to younger individuals made fewer sleep attempts during a sleep deprivation paradigm⁵³. Together, these findings index a growing physiological ability for extended wakefulness in adolescence, which might account for delayed bedtimes.

The rate at which sleep pressure builds during wakefulness decelerates in older adolescents. By contrast, the rate at which sleep pressure dissipates during sleep remains stable across adolescent development^{46,54}. This stability suggests that the need for sleep does not

substantially change throughout adolescence. Indeed, evidence from waking performance (for example, performance on a psychomotor vigilance task^{6,55}), and from self-reports of sleepiness⁶ and mood⁵⁶ assessed as a function of sleep duration, indicate an optimal sleep period of 9.0–9.3 hours per night across adolescence.

Two studies modelling the regulation of sleep homeostatic pressure during chronic sleep restriction (5 hours in bed over five or more nights) in older adolescents (15–17 years old) reveal an increase in slow-wave activity and decrease in slow wave energy (a combination of sleep intensity and duration measured via EEG) following chronic curtailed compared to adequate sleep opportunities^{5,57}. These studies suggest that — like adults⁵⁸ — adolescents do not adapt to continuous sleep loss, and sleep homeostasis remains operative during chronic sleep restriction. Thus, as adolescents mature, they are physiologically prepared to stay awake later. Yet they still require the same amount of sleep as at the onset of puberty, and do not physiologically adjust to chronic sleep loss.

Development of delayed circadian timing. The second biological process involved in regulating sleep is the 24-hour circadian rhythm (also called process C^{43,45,59}). This bioregulatory mechanism interacts with, but is mostly independent of, prior wake time (homeostatic process^{2,60}) in adolescents. The circadian process regulates sleep propensity during the approximately 24-hour day, with diurnal humans falling asleep at some time in the evening. Circadian rhythms can be measured biologically (for example, melatonin levels across the evening)^{61,62}, physiologically (for example, body temperature measured continuously across more than 24 hours)^{63,64} and behaviourally (for example, reaction time measured at intervals across more than 24 hours)⁶⁵.

Although there are a number of biological clocks, the primary circadian clock resides in the suprachiasmatic

nucleus in the anterior hypothalamus of the brain⁶⁶. This master clock helps to regulate daily rhythms via photic input (that is, light and dark signals) from the retinas⁶⁶. The daily oscillations of brain cell processes in the suprachiasmatic nucleus are not precisely 24 hours long. Thus, for most adolescents, the circadian cycle is slightly longer than 24 hours (on average, 24.2 hours)^{67–69}. An adolescent's circadian clock must therefore be reset each day to prevent sleep timing from drifting later and conflicting with school start times. External cues such as ambient light help to stabilize an adolescent's sleep timing, and prevent a conflict between school start times and their natural wake time^{69,70}.

Adolescents undergo drastic physiological alterations in the timing of their circadian rhythms and sleep patterns^{2,71}. Relative to children, adolescent circadian rhythms are markedly delayed in their timing⁶⁹ (FIG. 2). Thus, independent of sleep homeostatic pressure, adolescents have a strong tendency to stay up later than children do, owing to a delay in their circadian timing of sleep, and subsequently might experience delayed spontaneous morning arousal^{72,73}. This circadian-driven evening arousal persists until about 20–21 years of age, and then begins to decline across young, middle and older adulthood¹.

When circadian rhythms and sleep-wake patterns become delayed, adolescents have difficulty falling asleep at an earlier, more conventional time, analogous to sleep-onset insomnia^{74,75}. Once asleep, adolescents have objectively and subjectively good-quality sleep⁷⁶. However, owing to the combination of their delayed sleep onset and forced early awakenings to meet school commitments, their sleep becomes restricted across the 5-day school week⁷¹. This accumulation of insufficient sleep leads to daytime impairments (for example, low motivation and low mood) across the week⁷⁷ and across school terms⁷⁸. Although adolescents are afforded two consecutive days where no morning commitments might exist (that is, weekends), adolescents do not fully recover after two consecutive days of recovery sleep (as measured by tests of sustained attention)⁷⁹. Moreover, sleeping in on weekends might exacerbate circadian delay in adolescents because they will lose the resetting benefits of morning bright light^{73,80}. Weekend sleep-ins can therefore lead to further difficulty falling asleep on Sunday night and waking on Monday morning, resetting a weekly cycle⁸⁰. When the delay in sleep timing becomes chronic and extremely late, such that it interferes with important aspects of the adolescent's life (such as attending school), the adolescent might be diagnosed with a condition known as delayed sleep–wake phase disorder⁸¹.

Adolescent sleep and depression

The biological characteristics of adolescent sleep described above lead to sleep loss and delayed circadian timing, both of which have implications for depression. Moreover, the resulting inability to fall asleep has psychological consequences that might also contribute to adolescent depression. In this section, we discuss each of these factors and their contribution to adolescent depression.

Box 1 | Anxiety, depression and sleep

Poor adolescent sleep quality might be linked to the development of anxiety disorders¹⁷⁸. From a neurobiological perspective, poor adolescent sleep is associated with reduced myelination of anterior white matter tracts and fronto-limbic connectivity, both of which are required to evaluate negative emotions¹⁵⁶. In other words, when circadian shifts disrupt adolescent sleep, the brain has less opportunity to create connections in areas essential for processing emotions, and hence anxiety disorders are likely to develop¹⁵⁶. The relationship between decreased sleep and increased anxiety disorders might be bidirectional, with poor sleep influencing daytime emotion regulation and hyperarousal delaying the onset of sleep. However, the evidence suggests that poor sleep is more likely to precede the development of anxiety^{175,178} than the other way around.

Decreased ability to process emotions due to impaired sleep could also contribute to depression¹⁷⁵, especially considering shared psychological processes across anxiety, depression and sleep, such as negative cognitive biases, catastrophizing, rumination and worry^{175,179}.

Biological, psychological and social mechanisms might interact to increase the risk of developing insomnia, anxiety or depression during adolescence (see the review in REF.¹⁷⁵). The high comorbidity rate (between 10–50% in community samples¹⁷⁹) between depression and anxiety in adolescence is therefore unsurprising.

Despite the triadic interplay of anxiety, depression and sleep, poor sleep often precedes these disorders¹⁷⁵. Thus, early intervention of sleep difficulties may prevent future anxiety and/or depression¹⁷⁵, and sleep intervention should be considered as a first approach for prevention and intervention.

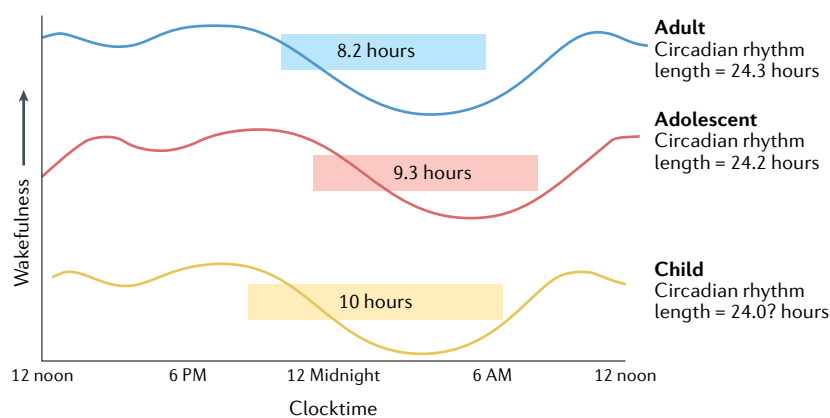


Fig. 2 | Circadian rhythm and optimal sleep duration. Circadian rhythm length, timing relative to clock time and optimal sleep duration (shaded boxes) for adolescents, children and adults. There are small differences in circadian rhythm length but significant differences in the timing of major peaks and troughs across development.

Sleep loss and depression. Logical arguments have been provided for a bidirectional link between adolescent sleep and depression: poor sleep is a risk factor for depression, and depression leads to poor sleep⁸². However, a meta-analysis of prospective and experimental data suggests a greater tendency for poor sleep to lead to depression in adolescents than the other way around³⁷. This meta-analysis also found that all longitudinal studies where sleep problems (specifically, sleep onset latency and wake after sleep onset) predicted depression used the gold standard measurement of sleep (polysomnography). By contrast, all studies that found that depression predicted later sleep problems used self-reports³⁷. Because depressed adolescents might have negative perceptions of their sleep, the direction of effects from depression to sleep problems needs to be verified with objective sleep measures (such as polysomnography).

There is a longitudinal association between insufficient sleep and subsequent depressed mood in non-clinical groups of adolescents⁸³. Analysis of the temporal relationship between sleep and next-day mood in clinical and non-clinical groups of adolescents showed that 9 hours of sleep per night was needed for optimal next-day mood⁵⁶. The relationship between sleep duration and mood was U-shaped, with sleep durations longer and shorter than 9 hours associated with worse next-day depressed mood⁵⁶. The impact of sleep loss was felt more keenly among adolescents experiencing clinically significant internalizing symptoms, who needed more sleep than adolescents below the clinical range⁵⁶. This result suggests a differential vulnerability to the effects of sleep loss among adolescents, with those experiencing symptoms of psychopathology more at risk of experiencing insufficient sleep.

Insufficient sleep causally affects several risk factors for depression, including mood valence^{84–86}. Multiple studies find large effects of insufficient sleep on the ability of adolescents to experience positive affective states, such as happiness, enthusiasm and excitement (for a review see REF.⁸⁷). A meta-analytic review of 74 studies of 361,505 adolescents found that less sleep was associated with a 55% increase in the likelihood of emotional deficits⁸⁷. A decrease in positive mood

showed the largest effect, followed by increases in anger, depression, negative affect and anxiety.

Another mechanism by which sleep loss can lead to depression is through its effect on emotion regulation. Emotion regulation is a key transdiagnostic risk factor for a range of negative psychological sequelae⁸⁸. Experimental studies have found that the ability of adolescents to regulate their emotions varies according to prior sleep^{84–86}. A physiological mechanism may be at play: the limbic system, which is involved in emotion regulation, might be functionally affected by sleep loss. Thus, sleep loss affects the ability of adolescents to regulate emotions at a physiological level⁸⁹.

Delayed circadian timing and depression. In addition to restricted sleep duration, the delayed sleep timing of adolescents also contributes to depression⁹⁰. Studies investigating the association between circadian rhythm and depression have focused on biological, behavioural and social aspects. Biological markers, such as the timing of melatonin production, are more precise measures of circadian timing, followed by self-reported chronotype (a measure of actual sleep behaviour, that is, the midpoint of sleep calculated from adolescents' reports of their sleep onset and wake times) and, finally, evening versus morning preference for daily activities (for example, their preference for when they choose to eat meals, study and exercise)⁹⁰. When measured simultaneously, those who report an evening preference, delayed chronotype and show a delayed circadian phase also report more symptoms of depression⁹¹. Similarly, independent studies have found that adolescents and young adults with elevated symptoms of depression concurrently report delayed circadian timing⁹², later chronotype⁹³ and a preference for evening activities^{94,95}. Together, these studies suggest consistent links between different indicators of altered circadian timing and depression.

Beyond concurrent associations, longitudinal studies point to a reciprocal association between delayed circadian timing and symptoms of depression. Several studies have shown that adolescents who preferred evening activities were more likely to subsequently report depressive symptoms^{96–98}, and one study found that depressed adolescents were more likely to subsequently develop an evening preference⁹⁹. A bidirectional association between circadian timing and depression is not surprising, given that both share similar biological and psychological underpinnings that might maintain and exacerbate each other over time¹⁰⁰.

Taken to the extreme, an adolescent with altered circadian timing would sleep during the day and be awake at night. But even much less dramatic alterations mean that an adolescent's body clock is not synchronous with the environment and society, with consequences such as school tardiness, and missing morning classes or entire school days¹⁰¹. An adolescent who is asleep during the day will miss opportunities to be exposed to the circadian-rhythm-resetting properties of bright light⁷³, to be physically active and to socialize with friends and family. Bright light, physical activity and connectedness with people are closely related to a positive mood^{102–104}. A slight elevation of depressed mood has also been

Polysomnography

A comprehensive test used to diagnose sleep disorders that includes measures of brain waves, blood oxygen level, heart rate, breathing and eye and leg movements.

reported during holiday periods, when delayed sleep timing is more pronounced as adolescents stay up later and sleep in when not attending school¹⁰⁵. Delaying sleep onset well into the night — when family and friends are asleep — might create a mental space for rumination¹⁰⁶, which is a powerful risk factor for depression¹⁰⁷. Indeed, people tend to ruminate more later in the day^{107,108}. Thus, dyssynchronization with dark-and-light cycles and society is one possible explanation for the overlap between delayed circadian timing and depression.

Alterations in the regulation of hormones, body temperature, and neurotransmitter release might also explain deficits in mood regulation¹⁰⁰. For example, there is evidence for lower melatonin amplitude in depressed individuals, which means that melatonin levels do not rise or decline sufficiently to regulate the alternation of sleepiness and wakefulness during the day and night¹⁰⁹. Moreover, melatonin secretion might be delayed in adolescents or even unsynchronized with other circadian processes (such as cortisol levels and body temperature) that regulate the sleep–wake cycle (for a review, see REF.¹¹⁰). These alterations in circadian processes affect the sleep regulation and the daily variations in energy levels, alertness and mood typically seen in depression¹¹⁰. Therefore, both biological and psychosocial processes, or an interaction between the two, might explain why adolescents with altered circadian timing report higher rates of depression.

The psychological contribution of adolescent sleep to depression. The changes to bioregulatory sleep processes during adolescence can lead to many sleep difficulties¹¹¹. The most common sleep problem is difficulty falling asleep, measured by sleep onset latency (the number of minutes from the sleep attempt to sleep onset)⁴. The majority of adolescents take more than 30 minutes to fall asleep¹¹², which in adults would qualify as sleep-onset insomnia¹¹³. However, adolescents with depression take even longer to fall asleep compared to both anxious and non-depressed counterparts¹¹⁴. The amount of time spent awake before falling asleep predicts future depression in adolescents³⁷.

Adolescents and their families are generally unaware that the combined effect of a delayed circadian rhythm and reduced sleep homeostatic pressure might result in a delayed sleep onset¹¹¹. Instead, adolescents tend to choose a bedtime based on the best opportunity to gain sufficient sleep (9 h in bed), rather than based on their perceived level of sleepiness¹¹⁵. Consequently, adolescents attempt sleep at a time of near peak-circadian alertness¹⁰¹, and therefore experience difficulty falling asleep (that is, sleep onset latency greater than 30 minutes). One mechanism proposed to link the time taken to fall asleep and future depression are pre-sleep cognitions (the thoughts that occur before sleep onset)³⁷.

Although pre-sleep cognitions also occur in adult insomnia^{116–118}, two key distinctions make adolescent pre-sleep cognitions unique. First, as described above, adolescents' pre-sleep cognitions occur during a period of arousal driven by the circadian peak of alertness¹⁰¹, rather than the constant hyperarousal observed in adults with insomnia^{116–118}. Second, the content of

pre-sleep cognitions differs between adults and adolescents. For example, 'rehearsal and planning' (that is, thinking about the past day, past experiences, next day, things to do, planning things and forthcoming events) is a common pre-sleep cognition theme for both adults with insomnia and adolescents diagnosed with delayed sleep–wake phase disorder^{119,120}. However, for adolescents these rehearsal and planning pre-sleep cognitions are more likely to be related to their education and friendships¹¹⁹. Furthermore, adolescents report such pre-sleep cognitions on both school nights and weekends, despite shorter sleep latencies on weekends due to later bedtimes¹²¹. Although the immediate implications of pre-sleep cognitions are similar between adolescents and adults (a longer time taken to fall asleep), it is the content of such pre-sleep cognitions that differs between these developmental stages.

Adolescents with depression negatively interpret events and information¹²², and become fixated with negative self-perceptions (for example, describing themselves as "useless", "unimportant" and "worthless")¹²³. Night-time ruminative thinking overlaps with and amplifies these depressogenic thoughts¹²³. For example, detailed pre-sleep cognitions captured during focus group interviews with adolescents suggest that pre-sleep cognitions mirror typical negative thoughts reported during depression¹¹⁵. In another study, adolescents were surveyed about difficulty initiating sleep, repetitive negative thinking (specifically worry and rumination) and depression¹²⁴. The results showed that repetitive negative thinking fully mediated the relationship between difficulty initiating sleep and depressed mood¹²⁴. Although further replication and more empirical support for the role of specific pre-sleep cognitions is needed, the initial evidence suggests that increased sleep onset latency, and subsequent pre-sleep cognitions, probably play a part in the relationship between sleep disturbance and depression in adolescents. Future studies are also needed to confirm whether such processes occur when adolescents wake during the night for extended periods of time, as this phenomenon is relatively rare in this population (compared to a long sleep latency).

Adolescent sleep interventions

Given the prevalence of adolescent sleep difficulties and their maladaptive consequences, it is clear that interventions are necessary. In this section, we outline three evidence-based interventions to resolve sleep issues that are applicable to adolescents, and for which there is evidence for reductions in depression symptoms (TABLE 1).

Bright light therapy. Light is one of the most potent time-givers for the human circadian system. Bright light exposure (around 5,000 lux) close to an adolescent's natural wake time might advance the timing of their circadian rhythm¹²⁵. Consequently, bright light therapy is usually used to treat a delayed circadian rhythm experienced by adolescents¹²⁶. Bright light therapy involves post-awakening light exposure, usually via natural sunlight or bright-light-emitting devices (lamps or portable light glasses)¹²⁷. Bed and wake times, and

Table 1 | Summary findings of key sleep intervention studies and meta-analyses

Sleep treatment	Contributing factor targeted	Key paper(s)	Design	Sample	Key findings
Bright light therapy	Advances the timing of an adolescent's delayed circadian rhythm; reduces sleep onset latency; reduces the opportunity for repetitive negative thinking	Richardson et al. (2018) ¹²⁹	Randomized controlled trial	Clinical (n=60)	Bright light therapy reduces adolescents' sleep onset latency by about 50 minutes by 3-month follow-up
		Richardson and Gradisar (2021) ¹²⁸	Randomized controlled trial	Clinical (n=63)	Bright light therapy decreased repetitive negative thinking ($d = 0.39$) and depression symptoms ($d = 0.83$) in adolescents at 3-month follow-up
Exogenous melatonin	Advances the timing of an adolescent's delayed circadian rhythm; reduces sleep onset latency; reduces the opportunity for repetitive negative thinking.	Wei et al. (2020) ¹⁵⁰	meta-analysis	Clinical (n=387)	Compared to placebo, exogenous melatonin decreased sleep onset latency by 21 minutes in children and adolescents
Cognitive-behaviour therapy for insomnia (CBT-I)	Reduces sleep onset latency; reduces the opportunity for repetitive negative thinking	Bootzin and Stevens (2005) ¹⁶¹	Single-arm study	Clinical (n=55)	Adolescents that completed treatment reduced sleep onset latency by 19 minutes, and reported significantly less worry
		de Bruin et al. (2015) ¹⁶⁷	Randomized controlled trial	Clinical (n=116)	Adolescents provided with CBT-I via group or internet format reduced their sleep onset latency by 21 minutes and 29 minutes, respectively
		Blake et al. (2017) ¹⁵⁹	Meta-analysis	Clinical (n=221)	Within-person meta-analysis found sleep onset latency decreased by 21 minutes and depression decreased ($d = 1.22$)
Mindfulness body scan	Reduces sleep onset latency; reduces the opportunity for repetitive negative thinking	Bartel et al. (2018) ¹⁶⁹	Randomized controlled trial	School-based (n=65)	Relative to control, listening to a 15-minute body scan at bedtime for 2 weeks reduced sleep onset latency by 8 minutes for adolescents with a baseline sleep onset latency greater than 30 minutes

d is Cohen's *d*, where small effect >0.20, moderate effect >0.50, large effect >0.80.

post-awakening light exposure, are then scheduled to become gradually earlier until the desired sleep timing is achieved. Bright light therapy effectively treats the symptoms of delayed sleep-wake phase disorder, including depression symptoms, experienced by adolescents^{101,128}.

Bright light therapy alone^{129,130} or in combination with cognitive behavioural therapy^{101,131} improves a range of sleep parameters (advances sleep timing, shortens sleep onset latency and increases sleep duration) and daytime functioning (decreases sleepiness and fatigue and increases cognitive functioning)^{132,133} in adolescents with delayed sleep-wake phase disorder. Despite experimental evidence with adults that bright light should be more effective than dim light¹³⁴, and short-wavelength light (for example, blue light) should be more effective than long-wavelength light (for example, red light)¹³⁵, randomized controlled trials with adolescents have failed to replicate these effects^{129,130}. These results suggest that evening light restriction and sleep scheduling might be important treatment components¹³⁶. The addition of cognitive behavioural therapy to bright light therapy has been shown to provide benefits to adolescents beyond

sleep improvements, such as decreased depression symptoms^{101,126,131}.

Although the mechanism for linking bright light therapy to improved sleep outcomes is clear (light advances circadian timing and therefore advances sleep timing), the mechanisms for linking bright light to improvements in depression are less so. One possibility is that doses of bright light directly affect mood. A feasibility study found that 30 minutes of post-awakening light exposure improved depressive symptoms and sleep quality in moderately-to-severely depressed adolescents who were hospital inpatients¹³⁷. Short-wavelength light (10,000 lux) administered via portable light glasses was just as effective at reducing depression symptoms as broad-spectrum white light (10,000 lux) administered via a light lamp¹³⁷. It is possible that light therapy indirectly improves depressed mood via improvements in sleep. For example, reducing sleep onset latency and associated repetitive negative thinking¹³⁸, or reducing the misalignment in biological circadian timing with one's social timing, might mitigate depression symptoms⁹⁰. Indeed, data from a clinical trial suggests

that bright light therapy for adolescents diagnosed with delayed sleep–wake phase disorder reduces sleep onset difficulties, repetitive negative thinking (both sleep- and non-sleep-related) and depressed mood¹²⁸.

Finally, in contrast to the depressogenic outcomes from chronic sleep loss over weeks and months described thus far, acute sleep deprivation over a short number of days (sometimes referred to as wake ‘therapy’) actually shows an anti-depressant effect¹³⁹. The combined effect of bright light therapy and wake therapy has been explored in depressed adolescent inpatients. Results suggest that a combined approach is no more effective than bright light therapy alone¹³⁹. Similarly, bright light therapy alone appears to be as effective as bright light therapy plus medication (fluoxetine) for adult depression (fluoxetine alone was no more effective than placebo)¹⁴⁰. The effect of combined bright light therapy and medication has not yet been explored in adolescent populations.

Exogenous melatonin. Melatonin is a neurohormone that aids the onset of sleep¹⁴¹. Endogenous melatonin is naturally produced by the pineal gland in the absence of light in the evening, and is suppressed during the day with re-exposure to light, thereby following a 24-hour circadian rhythm¹⁴². Exogenous melatonin is taken as a dietary supplement to complement melatonin produced by the body^{143,144}. As children develop into adolescents, their endogenous melatonin is produced later in the night, contributing to later sleep times¹⁴⁵. To correct this delay, exogenous melatonin delivered in the evening can produce a ‘pulling’ effect such that a person’s circadian rhythm timing gradually becomes earlier^{127,146}. The timing and dosage of exogenous melatonin administration is often individually tailored. However, exogenous melatonin is most effective when delivered in immediate-release form, at least 1–3 h before expected sleep onset, and in doses of up to 3 mg (REFS^{143,147}). Melatonin use can typically cease once the individual reaches their desired sleep time, and is therefore not a long-term treatment¹²⁷.

The use of melatonin as a paediatric sleep treatment has increased in popularity over the past two decades^{146,148–152}. Consequently, many studies have emerged investigating its efficacy. A systematic review of sleep treatments for children younger than 18 years old showed that exogenous melatonin significantly improved sleep latency, sleep duration and night-time awakenings across 19 randomized controlled trials¹⁴⁸. Furthermore, a meta-analysis found that exogenous melatonin improved both sleep onset latency and total sleep time compared to placebo across seven trials with 387 children and adolescents¹⁵⁰. Exogenous melatonin successfully advances circadian timing in adolescents with delayed circadian phase^{130,133,149}. However, there have been other mixed findings. Some research suggests that exogenous melatonin improves sleep timing but not sleep onset latency¹⁴⁷. Other studies have found improvements for sleep onset latency^{144,151}, but not total sleep time¹⁴⁴. On balance, the overall body of evidence suggests that exogenous melatonin is effective as a treatment for at least some sleep difficulties^{148,150}.

Exogenous melatonin has the potential to improve symptoms of both sleeplessness and depression because it reduces time awake in bed (by shortening sleep latency)¹⁵¹, and phase-advances circadian rhythm timing^{130,133,147}. Additionally, exogenous melatonin has shown efficacy in combination with other evidence-based treatments. For example, sleep interventions that combine bright light therapy with exogenous melatonin have shown improvements from baseline in sleep onset latency, total sleep time and circadian timing¹³⁰. Furthermore, the benefits of combining exogenous melatonin and bright light therapy might be maintained longer compared to either treatment alone¹³⁰. A combination of exogenous melatonin and bright light therapy might therefore be particularly effective at treating comorbid depression and sleep difficulties. Indeed, based on meta-analytic results a task force of sleep experts recommended exogenous melatonin to treat both sleep and comorbid psychiatric conditions in children and adolescents with delayed sleep–wake phase disorder¹²⁶.

Contrary to the lay person’s beliefs, exogenous melatonin does not cause feelings of sleepiness in most adolescents and adults, and therefore is not akin to other sedative medications¹⁵². However, exogenous melatonin can have a sleep-inducing effect in addition to circadian benefits¹⁵³ in children, leading to increased popularity for parents who wish to improve their children’s sleep. But, there is also controversy in the medical community about the use of melatonin^{146,149}. Specifically, there is debate regarding the safety of melatonin use in paediatric populations^{143,154}. Safety concerns stem largely from animal models suggesting that exogenous melatonin impairs reproduction and other physiological systems¹⁵⁴. Opposing arguments are based on the fact that exogenous melatonin in paediatrics is generally safe, and is not associated with adverse side effects, even in long-term studies¹⁵⁵. There is general agreement that more long-term studies with paediatric populations are needed to reach a consensus regarding safety¹⁵⁵. Nonetheless, in adolescents exogenous melatonin is most often used to improve circadian timing and therefore is recommended for short-term use (a few weeks)¹⁴⁷, not long-term use. Indeed, a meta-analysis of studies investigating short-term paediatric melatonin use revealed little to no difference in side effects in exogenous melatonin users compared to placebo¹⁵⁰.

Cognitive and behavioural techniques. As previously discussed, a long sleep latency provides more opportunities for adolescents to worry and ruminate¹²⁸. This is confirmed by a review of neuroimaging studies that suggested that sleep latency predicted future worry during adverse events¹⁵⁶. Bright light therapy and/or exogenous melatonin can reduce this opportunity for repetitive negative thinking by advancing sleep onset. However, these interventions might not sufficiently reduce negative pre-sleep cognitions for some adolescents¹⁵⁷. Furthermore, an adolescent’s long sleep latency might be due to other factors, such as insomnia, rather than delayed circadian rhythm alone. Cognitive and behavioural sleep interventions can simultaneously address

Box 2 | Evening technology use and adolescent sleep

Multiple pathways connecting evening technology use and sleep in adolescents have been proposed^{172,173}. First, technology use might increase arousal that delays the onset of sleep. Second, the blue light emitted from LED screens might promote alertness and delay sleep onset. Finally, according to the displacement hypothesis, adolescents might continue using technology past their usual sleep onset time and thereby restrict their sleep¹⁷⁴.

Tests of the arousal mechanism usually compare conditions in which adolescents participate in highly interactive technology use before bed (for example, playing a violent videogame) versus engage with less interactive technology (for example, watching TV) or they compare small (50 minutes) versus moderate (150 minutes) periods of violent videogames before sleep. Differences in the time taken to fall asleep between conditions in these experiments have been minimal (mean differences <5 minutes)^{180–183}.

Owing to a lack of support for the arousal mechanism, researchers turned towards testing the influence of blue light from LED screens. Methods for reducing these blue light emissions include wearing blue-light-blocking glasses¹⁸⁴ and apps that decrease blue light emissions (for example, *f.lux* and Apple's nightshift mode)^{185,186}. Mean differences in the time taken to fall asleep between these blue light-reducing conditions and control are again minimal (mean differences <10 minutes)^{184–187}.

By contrast, tests of the displacement hypothesis have yielded more substantial effects (for example, delaying bedtime by 1.5 hours)¹⁸⁸. However, bedtime delays only occur when there is an interaction between personality characteristics (such as risk-taking, flow state, self-control and bedtime procrastination) and the type of technology consumed before sleep^{188–190}. Although empirical support is more substantive for the bedtime displacement mechanism, the emerging evidence suggests that technology use does not influence all adolescents in the same way.

'Social media and networking' is a component in one of the most cited explanatory models of adolescent sleep^{2,71}. However, a meta-analysis of all risk and protective factors for adolescent sleep identified in the published literature found no significant relationship between any form of technology (TVs, phones, internet and video gaming) and sleep onset latency (all weighted correlation coefficients $r < 0.01$)¹⁹¹. Overall, the influence of technology use on sleep was lower than other factors, including tobacco and alcohol use and family factors (such as parent-set bedtimes)¹⁹¹. Moreover, all factors combined explained only roughly a quarter of the variance in adolescents' sleep duration¹⁹¹. Thus, the evidence to date casts doubt on the existence of a significant influence of evening technology use on adolescents' sleep.

Instead, longitudinal studies suggest that an increase in poor sleep is followed by an increase in technology use^{192,193}. Adolescents with sleep-onset difficulties report substantial negative pre-sleep cognitions¹¹⁹. When surveyed, the majority of adolescents report that using technological devices might aid in the onset of sleep¹⁹⁴. This finding suggests that using evening technology might benefit adolescents. Specifically, using devices while waiting for sleep onset might be a form of cognitive distraction from negative pre-sleep cognitions¹⁹⁵. Cross-sectional associations have been found between using social media to cope with negative feelings and sleep-onset difficulties in adolescents¹⁹⁶. In the context of the abovementioned studies this finding could be interpreted as the use of evening technology as a coping mechanism for a delayed sleep onset.

these additional sleep issues and depression¹⁵⁸. Indeed, a meta-analysis of cognitive-behavioural sleep interventions demonstrated clear subjective improvements to sleep (sleep duration, sleep latency, sleep efficiency and waking after sleep onset), objective improvements to sleep (sleep latency and efficiency) and decreased depression and anxiety symptoms¹⁵⁹.

Cognitive behaviour therapy for insomnia (CBT-I) was originally developed for adults experiencing insomnia¹⁶⁰. However, its potential for use in adolescents has been demonstrated over the past 15 years^{161,162}. CBT-I targets both the adolescent's sleep behaviours and cognitions. Behavioural techniques include calibrating an adolescent's time in bed to reduce wakefulness in bed while ensuring sufficient opportunity for restorative sleep, and stimulus control therapy that increases the odds of falling asleep through associations with discrete stimuli (for example, lying down in bed)¹⁶¹.

Relaxation training is also used to reduce physiological arousal, and sleep hygiene improves the adolescent's bedroom environment (for example, a dark, quiet bedroom and comfortable bedding) and pre-sleep behaviours (for example, reducing stimulating activities before going to bed)¹⁶¹. Cognitive techniques address adolescents' unhelpful thoughts when attempting sleep by critically evaluating them and generating alternative helpful thoughts¹⁶¹. CBT-I typically involves four to six sessions, and effectively decreases sleep latency following the intervention, although there is mixed evidence as to whether this improvement is maintained^{159,163}. These combined cognitive and behavioural approaches for insomnia show promise for improving depression, as evidenced by meaningful declines in self-reported depression scores by the end of CBT-I^{159,164}. Indeed, a meta-analysis found that treating adolescent sleep issues reduces depression symptoms¹⁶⁵. Moreover, a pilot study in which adolescents presenting with insomnia and co-morbid anxiety, depression or pain were treated with CBT-I for six weeks found pre-to-post-treatment improvements in insomnia, depression and anxiety symptoms¹⁶⁶.

Unlike bright light therapy and exogenous melatonin, CBT-I is not a singular treatment, and it might take longer to implement¹⁰¹. CBT-I is considered a 'modular approach' (that is, each behavioural and cognitive technique is considered a 'module'), and we therefore have a limited understanding of which specific CBT-I techniques for adolescent sleep problems are more potent in improving sleep and depression^{167,168}. Furthermore, the greater number of sessions needed to implement CBT-I (compared to bright light therapy and exogenous melatonin) means that it is a less cost-effective therapy for adolescents with sleep problems.

These issues have led some researchers to explore the effect of single stand-alone cognitive or behavioural techniques on adolescents' sleep. For example, a randomized trial conducted in schools found that constructive worry (a cognitive technique where adolescents wrote down their worries and solutions before bed) did not influence sleep latency relative to a control condition¹⁶⁹. By contrast, a mindfulness body scan technique at bedtime that aimed to focus thoughts on the present moment reduced the time taken to fall asleep by more than 50% for adolescents experiencing sleep-onset insomnia (that is, a sleep latency greater than 30 minutes)¹⁶⁹. A mindfulness body scan, combined with instructions for good sleep hygiene (including a comfortable sleep environment, consistent bed and wake times, relaxing before bed and avoiding pre-bed stimulation), also decreased sleep latency in high-level junior tennis players during a tournament week¹⁷⁰. Although not a standard behavioural technique for treating insomnia, one study found that restricting adolescents' smartphone use before bed did not influence the time taken to fall asleep, but increased total sleep time by 19 minutes per night¹⁷¹. We can only infer that reducing sleep latency, or increasing total sleep time, would decrease depression symptoms, as these studies testing single therapeutic techniques did not simultaneously measure levels of depression.

Summary and future directions

The combination of developmental changes to the bioregulatory systems controlling sleeping and waking during adolescence provides unique pathways to depression. First, reduced sleep homeostatic pressure delays the onset of sleep in mature adolescents⁵². This delayed sleep onset restricts sleep opportunity on school nights, making it difficult for adolescents to achieve the optimal 9.3 hours of sleep. Empirical evidence shows that reductions in positive mood states occur following sleep loss, which in turn increases depression symptoms⁸⁷. Second, there is a gradual delay in sleep timing (both sleep onset and offset) due to a delay in circadian rhythm timing across adolescent development⁷¹. Delayed circadian rhythm exacerbates delayed sleep onset, and is consistently associated with increased depression symptoms⁹⁰. These biological underpinnings are unique to adolescent development, and cast doubt on models that posit that evening technology use is a cause for poor adolescent sleep^{172–174} (BOX 2). The physiologically driven delay in sleep onset provides the opportunity for a third pathway to depression that is more psychological. More time between an adolescent’s bedtime and sleep onset increases the probability of experiencing negative pre-sleep cognitions¹²⁴. This opportunity for repetitive negative thinking is linked to higher levels of depression in adolescents¹²⁴.

Identifying these unique biological and psychological contributors that work in concert to produce delayed sleeping patterns is important because they can be matched to evidence-based techniques to reverse poor sleep and depressed mood. The gradual advance of bright light exposure in the morning (bright light therapy) and the gradual advance of evening exogenous melatonin can advance the timing of the circadian rhythm^{129,130,133}. Doing so advances sleep onset to earlier in the evening, shortening sleep onset latency. These chronotherapies not only shorten adolescent sleep onset latency, but also decrease depressive symptomology via reduced negative pre-sleep cognitions when attempting sleep¹²⁸. However, these treatments do not fully resolve sleep issues and depression symptoms. Thus, cognitive therapy techniques can be used to target residual sleep difficulties and depressed mood. Together, the clinical and meta-analytic evidence, and the cost-effectiveness

of sleep interventions, suggest that they should be used in the first instance when adolescents experience co-morbid depression and sleep problems¹⁶⁵.

An array of potential mechanisms link sleep and depression in adolescents¹⁷⁵. Thus, further research is needed to explore the potential of bright light therapy for adolescent depression, given that only one study to date has tested multiple mechanistic pathways in the same sample¹²⁶. Future research should also explore potential benefits of deploying interventions sequentially. For examples, it might be useful for adolescents who experience significant depression alongside sleep problems to start with exogenous melatonin treatment. Taking exogenous melatonin is less energy-intensive than other sleep interventions, which might increase the uptake of sleep interventions by adolescents who are fatigued by co-morbid depression and sleep problems. The adolescent might then be better able to engage in more energy-taxing interventions such as bright light therapy or concurrent cognitive behavioural therapy. However, this needs to be tested empirically.

Furthermore, we lack data that track the natural course and development of sleep problems due to evolving homeostatic and circadian changes over adolescent development. Longitudinal studies are needed to determine at what ages underlying biological sleep changes and depression symptoms occur. This will help to pinpoint when prevention studies for adolescent sleep problems — which are currently lacking¹⁷⁶ — should be conducted. Future research is also needed to convert efficacy trials into broader effectiveness studies performed in health care, school and digital settings. The paediatric sleep literacy (that is, the sleep knowledge, practices and attitudes) of health professionals has much room for improvement¹⁷⁷, suggesting that substantial efforts are needed to translate clinical research evidence to adolescent mental health practice. Given the worldwide prevalence of sleep problems, extrapolating this clinical evidence to a stepped-care model (for example, sleep prevention and intervention in high schools, intensive sleep interventions for adolescents on mental health service waiting lists, and accessible digital sleep interventions) for adolescents is needed.

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