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Abstract

Two adolescent mental health fields—sleep and depression—have been advancing largely in parallel until the past 4 years. Although sleep problems have been conceptualised as a symptom of adolescent depression, emerging evidence suggests that sleep difficulties arise before depression. 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.

[H1] Introduction

Adolescents (from pubertal onset to 21 years of age) are the most chronically sleep restricted subpopulation across human development[1]. Data from across the world suggests that adolescents sleep too late and too little[2]. 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 life[1]. A lack of morning commitments allows adolescents to potentially obtain optimal sleep duration (9.0-9.3 hrs)[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-up time to attend school[7]. This restricted sleep opportunity is especially so 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 delayed[8]. 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 will be accompanied by a return to restricted sleep for adolescents[11]. 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 therefore not a solution[12].

The rapid cascade of events that uniquely impact adolescent sleep begin around the onset of high school and puberty[13]. This coincides with a period of increased risk for the onset of low mood and depression in adolescents[14]. Approximately 2.6% of adolescents experience a depressive disorder at any given time[15], with the cumulative frequency of depression rising to 20% by the end of adolescence[16]. 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 live events[20,21] and experiencing bullying[22]. 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].

Adolescent-onset depression has also been associated with educational underachievement, low income levels and unemployment[28-30], and is a risk factor for suicidal behaviour[31].

Depression is characterised by a diverse range of symptoms. From the 9 symptom domains identified by The Diagnostic and Statistical Manual of Mental Disorders – 5[32], more than 1000 symptom combinations can be computed that meet the diagnostic criteria[33]. Little is known as to whether different combinations of symptoms at presentation are sufficient to form distinct subgroups[34]. One study suggests that there might be subtypes of depression that are primarily characterised by severity, with less severe groups (for example, fewer symptoms and lower risk) still presenting with high levels of somatic symptoms, such as sleep disturbance[35]. Amongst depressed adolescents, sleep problems (92%) are more commonly 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 adolescence[37].

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 lengthy time 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 adolescence[38], 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 disorder[39]. 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 anxiety[41] 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 summarise the interplay between sleep, depression and anxiety (Box 1).

[H1] 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.

[H3] Development of sleep homeostatic pressure

Human physiology strives for sleep-wake equilibrium. The sleep homeostatic process (also called Process S[43]) 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 involved[44]. 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 wakefulness[45]. The greater the amount of time spent awake, the greater the slow-wave activity during sleep, which demonstrates the 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-hr day to converge into a single, monophasic nocturnal sleep episode[46]. During adolescence, a further 'developmental leap' occurs. Slow-wave activity declines by approximately 40%, with the greatest reductions occurring at approximately 12.5 to 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 adolescence sleep (compared to childhood) is reflective of a lower 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 study[51], 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, 16.5, and 18.5 hours of sustained wakefulness[52]. Similar patterns have been found in adolescent mice, whereby mature compared to younger individuals made fewer sleep attempts during a sleep deprivation paradigm[53]. 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 sleepiness[6] and mood[56] assessed as a function of sleep duration, all 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 5 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 adults[58] – 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.

[H3] 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 ~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].

Whilst there are a number of biological clocks, the master circadian clock is the primary one and it resides in the suprachiasmatic nucleus in the anterior hypothalamus of the brain[66]. The master clock helps to regulate daily rhythms via photic input (that is, light and dark signals) from the retinas[66]. The daily oscillations of brain cells in the suprachiasmatic nucleus are not precisely 24-hours. Thus, for most adolescents, the circadian cycle is slightly longer than 24 hours (on average, 24.2 hours)[67-69]. Adolescents' 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 stabilise an adolescent's sleep timing, and prevent a conflict between school start times and their natural wake-up time[69,70].

Adolescents undergo drastic physiological alterations in the timing of their circadian rhythms and sleep patterns[2,71]. Relative to children, adolescents' circadian rhythms are markedly delayed in their timing[60] (Fig. 2) Thus, independent of sleep homeostatic pressure, adolescents have a strong tendency to stay up later than children due to a delay in their circadian timing of sleep, and subsequently might experience delayed spontaneous morning arousal[72,73]. This circadian-driven ebenung arousal persists until about 20 to 21 years of age, and then begins to decline across young, middle and older adulthood[1].

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 sleep[76]. However, due 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 week[71]. This accumulation of insufficient sleep leads to daytime impairments (for example, low motivation and low mood) across the week[77] and across school terms[78]. Although adolescents are afforded two consecutive days where no morning commitments might exist (that is, weekends), adolescents do not fully recover from 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 Sunday night and waking Monday morning, resetting a weekly cycle[80]. When the delay in sleep timing becomes chronic and extremely late, such that it interferes with important aspect of the adolescent's life (such as attending school), the adolescent might be diagnosed with a condition known as Delayed Sleep-Wake Phase Disorder [81].

[H1] 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.

[H3] 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 sleep[82]. 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 around[37]. 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-reports[37]. Depressed adolescents' perception of their sleep may be viewed negatively, thus the direction of effect from depression to sleep problems needs verification with objective measures of sleep (for example, polysomnography).

There is a longitudinal association between insufficient sleep and subsequent depressed mood in non-clinical groups of adolescents[83]. 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 mood[56]. 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 mood[56]. The impact of sleep loss was felt more keenly among adolescents experiencing clinically significant internalising symptoms,

who needed more sleep than adolescents below the clinical range[56]. 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 impacts several risk factors for depression, including mood valence[84-86]. Multiple studies find large effects of insufficient sleep on adolescents' ability to experience positive affective states, such as happiness, enthusiasm and excitement (for review see[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 deficits[87]. 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 sequelae[88]. Experimental studies have found that adolescents' ability to regulate their emotions varies according to prior sleep[84-86]. A physiological mechanism may be at play, with the limbic system, which is involved in emotion regulation, functionally impacted by sleep loss. Thus, sleep loss impacts adolescents' ability to regulate emotions at a physiological level[89].

[H3] Delayed circadian timing and depression

In addition to restricted sleep duration, adolescent's delayed sleep timing also contributes to depression[90]. 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-up times) and, finally, evening versus morning preference for daily activities (for example, their preference for when they choose to eat meals, study and/or exercise)[90]. When measured simultaneously, those who report an evening preference, delayed chronotype, and show a delayed circadian phase also report more symptoms of depression[91]. Similarly, independent studies have found that adolescents and young adults with elevated symptoms of depression concurrently report delayed circadian timing[92], later chronotype[93], and a preference for evening activities[94,95]. Together, these studies suggest consistnt 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 evening-types 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 preference [99]. A bi-directional 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 time[100].

Taken to the extreme, an adolescent with altered circadian timing would sleep during the day and be awake at night. But even less dramatic alterations mean that the adolescents' body clock is dyssynchronous with the environment and society, with consequences including school tardiness, and missing morning classes or entire school days[101]. An adolescent who is asleep during the day will miss opportunities to be exposed to the circadian resetting properties of bright light[73], be physically active, and socialise 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 reported during holiday periods, when delayed sleep timing is more pronounced as adolescents stay up later and sleep-in when not attending school[105]. Delaying sleep onset well into the night—when family and friends are asleep—might create a mental space for rumination[106], which is a powerful risk factor for depression[107]. Indeed, people tend to ruminate more later in the day[107,108]. Thus, dyssynchronisation 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 regulation[100]. For example, there is evidence for lower melatonin amplitude in depressed individuals, which means melatonin levels do not rise or decline sufficiently to regulate the alternation of sleepiness and wakefulness during the day and night[109]. Moreover, melatonin secretion may be delayed in adolescents or even unsynchronised 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 impact sleep regulation and daily variations in energy levels, alertness, and mood typically seen in depression[110]. 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.

[H3] The psychological contribution of adolescent sleep to depression

The changes to bioregulatory sleep processes during adolescence can lead to many sleep difficulties[111]. 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 asleep[112], which in adults would qualify as sleep-onset insomnia[113]. However, adolescents with depression take even longer to fall asleep compared to both anxious and non-depressed counterparts[114]. The amount of time spent awake before falling asleep predicts future depression in adolescents[37].

Adolescents and their families are unaware that the combined effect of a delayed circadian rhythm and reduced sleep homeostatic pressure might result in a delayed sleep onset[111]. Instead, adolescents tend to choose a bedtime based on the best opportunity to gain sufficient sleep (9 hours in bed), rather than based on their perceived level of sleepiness[115]. Consequently, adolescents attempt sleep at a time of near peak-circadian alertness[101], 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 alertness[101], rather than the constant hyperarousal observed with 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 concerned with their education and friendships[119]. Furthermore, adolescents report such pre-sleep cognitions on both school nights and weekends, despite shorter sleep latencies on weekends due to later bedtimes[121]. Whilst 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 differ between these developmental stages.

Adolescents with depression negatively interpret events and information[122], 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 thoughts[123]. For example, detailed pre-sleep cognitions captured during focus group interviews with adolescents suggest that pre-sleep cognitions mirror typical negative thoughts reported during depression[115]. In another study, adolescents were surveyed about difficulty initiating sleep, repetitive negative thinking (specifically worry and rumination) and depression[124]. The results showed that repetitive negative thinking fully mediated the relationship between difficulty initiating sleep and depressed mood[124]. 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, are likely to play a role 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).

[H1] 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).

[H3] Bright light therapy

Light is one of the most potent time-givers for the human circadian system. Bright light exposure (~5,000 lux) close to an adolescent's natural wake-up time might advance the timing of their circadian rhythm[125]. Consequently, bright light therapy is commonly used to treat a delayed circadian rhythm experienced by adolescents[126]. 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 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, experiences 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 light[134], and short-wavelength light (for example, blue light) should be more effective than long-wavelength light (for example, red light)[135], randomised 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 components[136]. The addition of cognitive behavioural therapy has been shown to confer benefits beyond sleep improvements to adolescents', for example, 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 impact on 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 inpatients[137]. Short-wavelength light (10,000 lux) administered via portable light glasses was equally effective at reducing depression symptoms as broad-spectrum white light (10,000 lux) administered via a light lamp[137]. It is possible that light therapy indirectly improves depressed mood via improvements in sleep. For example, reducing sleep onset latency and associated repetitive negative thinking[138], or reducing the misalignment in biological circadian timing with one's social timing, might mitigate depression symptoms[90]. 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 mood[128].

Finally, in contrast to the depressogenic outcomes from chronic sleep loss over weeks and months that has described thus far, acute sleep deprivation over a short number of days (sometimes referred to as wake 'therapy') actually shows an anti-depressant effect[139]. 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 alone[139]. Similarly, bright light therapy alone appears 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.

[H3] Exogenous Melatonin

Melatonin is a neurohormone that aids the onset of sleep[141]. Endogenous melotonin 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, thus following a 24-hour circadian rhythm[142]. 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 times[145]. To correct this delay, exogenous melatonin delivered in the evening can produce a 'pulling' effect such that a person's circadian rhythm timing is gradually advanced 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 hours before expected sleep onset, and in doses of up to 3mg[143,147]. Melatonin use can typically cease once the individual reaches their desired sleep time, and is therefore not a long-term treatment[127].

The use of melatonin as a paediatric sleep treatment has increased in popularity over the past two decades[150-152 146,148,149]. 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 randomised controlled trials[148]. 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 adolescents[150]. 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 latency[147]. Other studies have found improvements for sleep onset latency[144,151], but not total sleep time[144]. 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 timing[130]. Furthermore, the benefits of combining exogenous melatonin and bright light therapy might be maintained longer compared to either treatment alone[130]. 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 Disorder[126].

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 medications[152]. However, exogenous melatonin can have a sleep-inducing in addition to circadian benefits[153] in children, leading to increased popularity for parents helping their children 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 systems[154]. 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 studies[155]. There is general agreement that more long-term studies with paediatric populations are needed to reach a consensus regarding safety[155]. 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 placebo[150].

[H3] Cognitive and behavioural techniques

As previously discussed, a long sleep latency provides more opportunities for adolescents to worry and ruminate[128]. This is confirmed by a review of neuroimaging studies which suggested that sleep latency predicted future worry during adverse events [156]. 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 adolescents[157]. 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 these additional sleep issues and depression[158]. Indeed, a meta-analysis of cognitive-behavioural sleep interventions demonstrated clear subjective improvements to sleep (sleep duration, sleep latency, sleep efficiency, and wake after sleep onset), objective improvements (sleep latency and efficiency) and decreased depression and anxiety symptoms[159].

Cognitive Behaviour Therapy for insomnia (CBT-I) was originally developed for adults experiencing insomnia[160]. 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, yet providing enough opportunity for restorative sleep, and stimulus control therapy that increases the odds of falling asleep associated with discrete stimuli (for example, lying down in bed)[161]. Relaxation training is also used to reduce physiological arousal, as well as sleep hygiene which improves the adolescent's bedroom environment (for example, dark, quiet bedroom and comfortable bedding) and pre-sleep behaviours (for example, reducing stimulating activites before going to bed)[161]. Cognitive techniques address adolescents' unhelpful thoughts when attempting sleep by critically evaluating them and generating alternative helpful thoughts[161]). CBT-I typically involves 4 to 6 sessions, and effectively decreases sleep latency following the intervention, although there is mixed evidence as to whether this improvement is maintained[163,164]. These combined cognitive and behavioural approaches for insomnia mentioned above show promise for

improving depression, evidenced by meaningful declines in self-reported depression scores by the end of CBT-I[164,165]. Indeed, a meta-analysis found that treating adolescent sleep issues reduces depression symptoms[166]. Moreover, a pilot study in which adolescents presenting with insomnia and co-morbid anxiety, depression and/or pain were treated with CBT-I for six weeks found pre-to-post treatment improvements in insomnia, depression and anxiety symptoms[167].

Unlike bright light therapy and exogenous melatonin, CBT-I is not a singular treatment, and it might take longer to implement[101]. CBT-I is considered a 'modular approach' (that is, each behavioural and cognitive technique is considered a 'module'), and thus we have a limited understanding of which techniques of CBT-I for adolescent sleep problems are more potent in improving sleep and depression[168,169]. Furthermore, the greater number of sessions needed to implement CBT-I (compared to bright light therapy and exogenouse melatonin) means 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 randomised 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 condition[170]. 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)[170]. 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 week[171]. 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 min per night[172]. We can only infer that reducing sleep latency, or increasing total sleep time, would decrease depression symptoms, as these studies testing a single therapeutic technique did not simultaneously measure levels of depression.

[H1] Summary and future directions

The combination of developmental changes to the bioregulatory systems controlling sleep and wake during adolescence provides unique pathways to depression. First, reduced sleep homeostatic pressure delays the onset of sleep in mature adolescents[52]. 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 reductions in positive mood states occur following sleep loss, which in turn increases depression symptoms[87]. Second, there is a gradual delay in sleep timing (both sleep onset and offset) due to a delay in circadian rhythm timing across adolescent development[71]. Delayed circadian rhythm exacerbates delayed sleep onset, and is consistently associated with increased depression symptoms[90]. 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[173-175] (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 cognitions[124]. This opportunity for repetitive negative thinking is linked to higher levels of depression in adolescents[124].

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 advancement of bright light exposure in the morning (bright light therapy) and the gradual advancement of evening exogenous melatonin can advance the timing of the circadian rhythm[129,130,133]. Doing so advances sleep onset earlier in the evening, shortening sleep onset latency. These chronotherapies not only shorten adolescents' sleep onset latency, but also decrease depressive symptomology via reduced negative pre-sleep cognitions when attempting sleep[128] . 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 they should be used in the first instance when adolescents experience co-morbid depression and sleep problems[166].

Due to the array of potential mechanisms linking sleep and depression in adolescents[176], further research is needed to explore the potential of bright light therapy for

adolescent depression, as the testing of multiple mechanistic pathways in the same sample has only been tested by one study to date[126]. 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 may 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 like bright light therapy or concurrent cognitive behavioural therapy. However, this needs to be tested empirically.

Furthermore, we lack the data tracking 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 pinpoint when prevention studies for adolescent sleep problems—which are currently lacking[177]—should be conducted. Future research is also needed to convert efficacy trials into broader effectiveness studies performed in health care, school and digital settings. Health professionals' paediatric sleep literacy has much room for improvement[178], suggesting 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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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 ~50min by 3-month follow-up.
		Richardson & 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 min in children and adolescents.
Cognitive-Behaviour Therapy for	Reduces sleep onset latency	Bootzin & Stevens (2005)[161]	Single-Arm Study	Clinical (N=55)	Adolescents that completed treatment reduced sleep

Insomnia (CBT-I)	Reduces the opportunity for repetitive negative thinking.				onset latency by 19 min, and reported significantly less worry.
		de Bruin et al. (2015)[168]	Randomized controlled trial	Clinical (N=116)	Adolescents provided with CBT-I via group or internet format reduced their sleep onset latency by 21 min and 29 min, respectively.
		Blake et al. (2017)[164]	Meta-analysis	Clinical (N=221)	Within-person meta-analysis found sleep onset latency decreased by 21 min and depression decreased (*d*=1.22).
Mindfulness body scan	Reduces sleep onset latency Reduces the opportunity for repetitive negative thinking.	Bartel et al. (2018)[170]	Randomized controlled trial	School-based (N=65)	Relative to control, listening to a 15-min 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 min.

Note: *d*= Cohen's d, where small effect >0.20, moderate effect >0.50, large effect >.80.

Figure Captions

Figure 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 and ICD-3.

Figure 2: Circadian rhythm and optimal sleep duration. Circadian rhythm length, timing relative to clock time, and optimal sleep duration (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.

Box 1: Anxiety, Depression and Sleep

Poor adolescent sleep quality might be linked to the development of anxiety disorders[179]. 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 emotions[156]. In other words, when circadian shifts disrupt adolescent sleep, the brain has less opportunity to create connections in areas essential for processing emotions, hence anxiety disorders are likely to develop[156]. The relationship between decreased sleep and increased anxiety disorders might be bidirectional, where poor sleep can affect daytime emotion regulation and hyperarousal can delay the onset of sleep. However, the evidence suggests that poor sleep is more likely to precede the development of anxiety[176,179] rather than the other way around.

Decreased ability to process emotions due to impaired sleep could also contribute to depression[176], especially considering shared psychological processes across the anxiety, depression and sleep, such as negative cognitive biases, catastrophising, rumination and worry[176,180].

Biological, psychological and social mechanisms might interact to increase the risk of developing insomnia, anxiety or depression during adolescence (see[176] for a review). The high comorbidity rate (between 10-50% in community samples[180]) between depression and anxiety in adolescence is therefore unsurprising.

Despite the triadic interplay of anxiety, depression and sleep, poor sleep often precedes these disorders[176]. Thus, early intervention of sleep difficulties may prevent future anxiety and/or depression[176], and sleep intervention should be considered as a first approach for prevention and intervention.

Box 2: Evening technology use and adolescent sleep

Multiple pathways connecting evening technology use and sleep in adolescents have been proposed[173,174]. First, technology use might increase arousal that extends the onset of sleep. Second, the blue light emitted from LED screens might promote alertness and extend sleep onset. Finally, according to the displacement hypothesis adolescents might continue using technology past their usual sleep onset time and thereby restrict their sleep[175].

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 min) versus moderate (150 min) doses of violent videogames before sleep. Differences in the time taken to fall asleep between conditions in these experiments have been minimal (mean differences < 5 min)[181-184].

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-blocking glasses[185], and apps that decrease blue light emissions (for example, *f.lux* and Apple's nightshift mode)[186,187]. Mean differences in the time taken to fall asleep between these blue light-reducing conditions and control are again minimal (mean differences < 10 min)[185-188].

By contrast, tests of the displacement hypothesis have yielded more substantial effects (for example, delaying bedtime by 1.5 hrs)[189]. 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[189-191]. 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, the Internet, and

video gaming) and sleep onset latency (all weighted correlation coefficients r<.01)[192]. 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)[192]. Moreover, all factors combined only explained roughly a quarter of the variance in adolescents' sleep duration[192]. 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[193,194]. Adolescents with sleep-onset difficulties report substantial negative pre-sleep cognitions[119]. When surveyed, the majority of adolescents report that using technological devices might aid in the onset of sleep[195]. 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 cognitions[196]. Cross-sectional associations have been found between using social media to cope with negative feelings and sleep-onset difficulties in adolescents[197]. 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.

Glossary

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.