

Adolescent sleep restriction effects on cognition and mood

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Abstract

Adolescents throughout the world do not obtain adequate sleep. A recent proliferation of experimental and quasi-experimental studies has considerably clarified the relationships between sleep loss and neurobehavioral function suggested by earlier epidemiological and cross-sectional studies. These new studies concur in finding that multiple successive nights of restricted sleep can impair multiple cognitive and affective functions. These effects cumulate from night to night, may not fully recover after weekend recovery sleep and may even be compounded by re-exposure to sleep restriction. An hour long afternoon nap reduces sleepiness in addition to improving vigilance, memory encoding and mood without interfering with nocturnal sleep when the latter is shortened. However, this does not detract from the point that adolescents require approximately 9h of sleep per night for optimal neurobehavioral function, a message that more need to embrace.

Keywords

Adolescent, Sleep, Sleep restriction, Cognition, Mood, Affect, Emotion regulation

1 Introduction

Adolescence is a time of immense developmental change. Sleep evolves dramatically over the teenage years due to the combined effects of sleep regulatory, brain development and psychosocial changes (Carskadon, 2011). Homeostatic sleep pressure builds more slowly in adolescents than in children and the circadian timing system undergoes a puberty-related delay (Carskadon et al., 1980; Jenni et al., 2005; Roenneberg et al., 2004). Combined, these factors are associated with longer periods of wakefulness in the evening and later bedtimes. Adolescence is also a period of

significant psychosocial change, with increased autonomy, greater academic pressure and homework load, more complex social relationships, after school jobs, reduced parental regulation of sleep and pervasive technology use (Bartel et al., 2015; Gangwisch et al., 2010; Hysing et al., 2015; Yang et al., 2005). The result is that adolescents both *want to* and *can* stay up later in the evening, and they have a biology that supports this. Because they are required to wake as early as—if not earlier than—younger children to attend school, this “perfect storm” of factors leads to chronically insufficient sleep (Carskadon, 2011; Crowley et al., 2018).

It is recommended that adolescents obtain between 8 and 10 h’ sleep per night for optimal daytime functioning (Carskadon et al., 1980; Fuligni and Hardway, 2006; Hirshkowitz et al., 2015; Paruthi et al., 2016). However, a recent systematic review and meta-analysis of pediatric total sleep time estimated by actigraphy revealed that adolescents aged 12–18 years average <7 h sleep per night on school nights and <8 h sleep per night on weekends (Galland et al., 2018). When considering the individual study estimates of total sleep time, none had an average total sleep time estimate within the recommended range. The finding that very few children have normative sleep on school nights that falls within the recommended range is a significant barrier for cross-sectional research (Short et al., 2018a). Given there is often a restriction in the range of normative sleep durations on school nights, with very few in the recommended range, the true effect of sleep loss on functioning may be obscured or diminished. As most adolescents are sleep restricted, cross-sectional studies lack data from individuals who obtain sufficient sleep. It is telling that experimental research into sleep loss in teenagers, which typically involve a control condition in which adolescents obtain more sleep than typically obtained at home, will often find causal relationships where cross-sectional studies do not. Accordingly, the present review primarily focuses on experimental and quasi-experimental studies where causal relationships can be tested.

2 Adolescent sleep restriction and cognitive functioning

Multiple studies on sleep curtailment in adolescents have provided clear evidence for increased daytime sleepiness. However, the case for objective cognitive performance degradation following adolescent sleep loss was initially less compelling (Bryant and Gomez, 2015; Kopasz et al., 2010), leading some to suggest that adolescents may be more resistant to sleep loss (Voderholzer et al., 2011). While several observational studies have found that speed of processing, sustained attention, working memory, and executive function are poorer in children and adolescents who report shorter sleep (Gradisar et al., 2008; Sadeh et al., 2002; Steenari et al., 2003), other studies have failed to find a significant relationship between sleep duration and speed of processing (Buckhalt et al., 2007; Sadeh et al., 2002), working memory, or executive function (Anderson et al., 2009; Buckhalt et al., 2007; Sadeh et al., 2002).

In the first of a series of quasi-laboratory studies, a moderately severe level of sleep restriction (5 h/TIB \times 7 nights) was compared to a control condition where

adolescents aged 15–19 years of age, were given a 9h/TIB nocturnal sleep opportunity (Lo et al., 2016c). The sleep restricted group showed a cumulative increase in lapses of vigilance with each successive night of sleep restriction. Two nights of 9h/TIB recovery sleep did not fully restore participants vigilance performance to baseline levels or to the level of the control group. Speed of processing, working memory and positive mood were also significantly worse in the sleep restricted adolescents (Fig. 1). Changes in speed of processing manifested as a reduced rate of improvement over time. As in prior work with adults, there was a dissociation between deterioration in subjective sleepiness (which plateaued after several days) and vigilance (Van Dongen et al., 2003), which continued to deteriorate through the week. Of these four indicators, sleepiness, positive mood and speed of processing have shown robust declines following multiple nights of sleep restriction in all four editions of this series of studies.

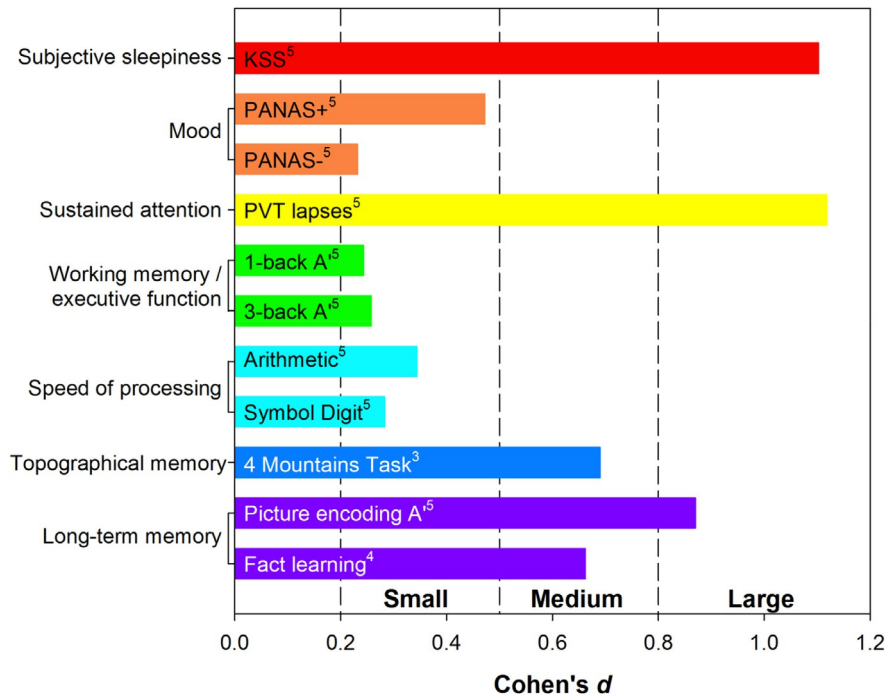


FIG. 1

Relative effect sizes of multi-night sleep restriction on a range of cognitive functions evaluated in several “Need For Sleep” studies conducted on adolescents aged 15–18 years. The effect sizes are meant to be indicative rather than definitive as the duration of sleep restriction varied from 3 to 5 nights and times of testing varied as indicated by a superscript number next to the specific test described in the accompanying text. The level of sleep restriction in all cases was 5h TIB nocturnal sleep opportunity without a daytime nap and performance in the various tasks was compared against that recorded for 9h TIB nocturnal sleep opportunity.

A second cycle of sleep restriction to 5 h/TIB after 5 nights of sleep restriction and 2 nights of recovery sleep compounded the effect of sleep restriction on vigilance decline (Lo et al., 2016b). Similar trends were observed for sleepiness, speed of processing, working memory and positive mood suggesting that sustained moderate multi-night sleep restriction has robust and significant effects on basic cognitive processes and positive mood. Tests of vigilance, speed of processing and working memory may be more sensitive to sleep loss than conventional academic testing because responses need to be made within a short time window and without deferment (unlike the self-paced nature of learning and recall of a body of knowledge).

A 60 min afternoon nap at this level of nocturnal sleep restriction significantly improved vigilance, sleepiness, speed of processing and working memory in the first cycle of sleep restriction (Lo et al., 2016b). Lapses in vigilance were suppressed to almost baseline levels of performance in the early- and mid-portions of the first week of sleep restriction but tended to rise in the second cycle pointing to the cumulative effects of multi-night sleep restriction that are not completely “reset” even with a weekend of recovery sleep.

Across the population of adolescents and young adults there is much variation in the proportion of persons who report habitual napping (Ficca et al., 2010; Lovato and Lack, 2010) and this proportion changes depending on a person’s prior sleep history. However, under conditions of sleep restriction to 5 h/TIB all participants were able to do so albeit for varying durations. An independent dataset collected from Singapore supports the notion that shortened nocturnal sleep duration increases napping in adolescents. In this study half of secondary school students napped for 30 min or more during weekdays where the grand average sleep duration was 6.5 h at night (Yeo et al., 2018). Over the weekend when the opportunity to sleep increased and sleep duration rose to 8.8 h, only 38.5% of persons napped. In a subgroup analysis, those who had <7 h nocturnal sleep on weekdays were more likely to report napping (60.2%), compared to those who slept the recommended 8–10 h at night (14.5%). Going back to the experimental data, it is noteworthy that under conditions of sleep restriction, *daily* 1 h naps *did not* interfere with nocturnal sleep (Ong et al., 2017), echoing a point made in a recent review critically evaluating the empirical evidence supporting sleep hygiene recommendations (Irish et al., 2015). As such, making provisions for a 60–90 min mid-afternoon nap is desirable and feasible when students are known to obtain chronically insufficient nocturnal sleep.

Long-term memory performance following sleep restriction is naturally of interest in educational settings. An earlier study found that even 5 nights of restriction to 5 h/TIB did not affect the consolidation of declarative memory for word-pairs or procedural memory for finger-tapped sequences when tested 8–36 days after learning (Voderholzer et al., 2011). The preservation of long-term memory consolidation under such conditions has been attributed the conservation of slow wave sleep in adolescents at the expense of light sleep and REM (Ong et al., 2016; Voderholzer et al., 2011). Extending these findings to ecologically more realistic stimuli, memory for short prose passages was influenced by whether these were prioritized for

subsequent recall, but they were not significantly affected by over a week of sleep restriction after learning (Lo et al., 2016a). Indeed the negative effects of a single night of total sleep deprivation on memories acquired *before* deprivation appear to be negated in young adults if they receive normal sleep on the second and subsequent nights (Schonauer et al., 2015).

Where sleep restriction appears to clearly affect long-term memory in adolescents is during encoding. There is now empirical support for the notion that picture encoding (Cousins et al., 2018) and fact learning are significantly affected by multi-night sleep restriction (Fig. 1). The ability to recognize a computer generated image of four mountains in a way that is point-of-view and time-of-day invariant is hippocampus dependent. Performance in this test is affected even after 3 nights of restriction to 5h/TIB (Cousins et al., 2019) (Fig. 1).

3 Possible mechanisms underlying cognitive impairment

While it seems obvious that a sleepy adolescent with impaired vigilance will not be attentive enough to encode information, there appears to be no significant correlation between PVT lapses and memory encoding. Neuroimaging experiments with sleep deprived young adults have shown that even during epochs when participants are able to respond to visual stimuli, the processing of these is inferior to that occurring in the well-rested state. One is less aware of peripheral information (Kong et al., 2011), the rate of visual information processing is reduced (Kong et al., 2014) and distractor suppression is reduced (Kong et al., 2012) when one is sleep deprived. In addition, pattern separation evident from being able to distinctively encode different exemplars from the same visual category is compromised in sleep deprived persons (Poh and Chee, 2017).

In addition to these findings concerning degraded online information processing as a result of sustained wakefulness, the synaptic homeostasis hypothesis (Tononi and Cirelli, 2014) suggests that memory encoding capacity, saturated following sustained wakefulness may be restored by synaptic downscaling that occurs during slow wave sleep (Kuhn et al., 2016). Artificially increasing slow-wave activity (SWA) can increase encoding capacity (Antonenko et al., 2013; Ong et al., 2018), while interrupting SWA reduces it (Van Der Werf et al., 2009).

Slow wave sleep is significantly recovered during afternoon nap opportunities under conditions of sleep restriction (Ong et al., 2017). Performance in the hippocampus weighted Four-Mountains task (Hartley et al., 2007) positively correlated with duration of slow wave sleep during the nap preceding encoding (Cousins et al., 2019). However, this simple correlation was not present with picture encoding or fact learning despite the fact that at the group-level a nap had clear benefit. Further details of long-term memory performance in the setting of naps are presented in the chapter on Sleep and Long-Term Memory.

4 Adolescent sleep restriction and mood

Mood disorders, such as depression and anxiety, can be chronic and lifelong conditions and frequently have their onset in adolescence (Giedd et al., 2008). These conditions reduce quality of life and productivity in affected individuals and pose one of the largest burdens of disease of all physical and mental health conditions (Vigo et al., 2016). Thus, understanding the factors contributing to mood and mood regulatory deficits in adolescents is imperative for timely and effective prevention and intervention.

A recent major study estimated that adolescents need approximately 9 h sleep per night for optimal mood functioning (Fuligni et al., 2017). This estimate was derived by asking adolescents to record their sleep and levels of depressed mood and anxiety each day. Optimal sleep was defined as the duration of sleep at which the lowest level of depressed mood and anxiety were reported. Of interest, sleep need for optimal mood tended to vary depending on mental health symptomology. Specifically, adolescents with clinically meaningful internalizing symptoms, which are common in both anxiety and depressive disorders, needed more sleep than adolescents whose internalizing symptoms were in the normative range (Fuligni et al., 2017). These individual differences in sleep need and vulnerability to sleep loss may help to explain why existing research tends to find stronger relationships between sleep duration and mood in clinical groups than healthy controls (Mullin et al., 2017).

This estimate of sleep need for optimal mood aligns with studies estimating optimal sleep for sustained attention performance and daytime alertness (Carskadon et al., 1980; Short et al., 2018b), however, it is dissimilar to the sleep the majority of adolescents obtain (Galland et al., 2018; Gradisar et al., 2011). In countries such as Singapore, South Korea and China, early school start times, evening classes, intense homework loads and pressure to perform academically combine to restrict sleep even further (Jiang et al., 2015; Lo et al., 2016c; Yang et al., 2005; Yeo et al., 2018). Resultingly, the normative experience for adolescents is chronically restricted sleep. If sleep is important for mood and emotion regulation, then increasing sleep provides one means to reduce the risk of mood and emotion regulation deficits and their more debilitating and sometimes life-threatening counterparts, depression, anxiety and suicidal ideation.

Experimental research using sleep loss paradigms in adolescents has shown that sleep loss reduces positive affect (Baum et al., 2014; Lo et al., 2016c; McMakin et al., 2016; Reddy et al., 2017; Short and Louca, 2015). A recent study exposed 57 adolescents, aged 15–19 years, to either 12 successive nights with 9 h TIB, or to one of two sleep restriction paradigms (Lo et al., 2016b). Both sleep restriction conditions included 5 h TIB for 5 nights, then 2 nights of recovery sleep with 9 h TIB, followed by a further 3 nights of 5 h TIB; however, half of these sleep restricted adolescents also had a 1-h nap each sleep restriction day. This protocol mimics the typical adolescent cycle of alternating restricted sleep on school nights with catch-up sleep on weekends and provides the opportunity to observe whether weekend “catch

up” sleep is enough to ameliorate the deficits accrued across weekdays. This study found that positive affect reported by the no-nap sleep restriction group deteriorated across the first 5 nights of sleep restriction. While improvement in positive affect was observed with recovery sleep, exposure to the second cycle of sleep restriction increased the rate at which positive affect deteriorated. Napping attenuated, but did not eliminate, this effect. These findings suggest that deficits in positive affect accumulate, not only across each week of sleep restriction, but also across successive cycles of sleep restriction, with important implications for the normative sleep behavior of adolescents over successive weeks of the school term. Of interest, negative affect was not similarly affected, which is consistent with most, but not all, prior research (Lo et al., 2016c; McMakin et al., 2016; Reddy et al., 2017).

The notion that sleep loss does not affect mood states uniformly has received greater recognition in recent times (Shen et al., 2018; Short and Louca, 2015). While much previous research on the relationship between sleep loss and mood has focused on negative mood states, recent research suggests that positive mood states may be more susceptible to sleep-related deficits (Lo et al., 2016c; Shen et al., 2018). Extant studies of both sleep restriction and total sleep deprivation have found that sleep loss increased reports of anxiety, confusion, and fatigue more than depressed mood and anger (Baum et al., 2014; Short and Louca, 2015). The tendency to focus on negative mood states is understandable given that heightened negative moods, such as depressed mood, sadness, and anxiety, are among the diagnostic criteria for many mental health conditions (American Psychiatric Association, 2013). However, it is important to note that anhedonia, or reduced ability to gain pleasure from typically pleasurable activities, is both clinically relevant and a symptom that diminishes the well-being and functioning of affected individuals (Bryant et al., 2017). Thus, present research suggests that one mechanism by which sleep loss impacts clinically relevant mood symptomology is through heightened anhedonia (Fig. 2).

The Positive and Negative Affect Schedule (PANAS) is the most widely used tool to assess positive and negative affect (Watson et al., 1988). The PANAS consists of two 10-item scales. Each scale contains a list of 10 words describing either positive or negative affect, to which participants rate how each word describes how they feel right now on a 5-point Likert-type scale ranging from “Very slightly or not at all” to “Extremely.” Some argue that the PANAS is not sensitive to sleep-related deficits to negative affect as it is susceptible to a floor effect on many of the negative affect items (Franzen et al., 2008; Lo et al., 2016b). Indeed, in the study of successive cycles of sleep loss, researchers note that many participants reported that some of the items on the PANAS, such as guilty, scared, and afraid, were irrelevant to them (Lo et al., 2016b). As such, more fine-grained assessment of mood states, such as unipolar visual analog scales, may help to elucidate which moods are affected by sleep loss and to what extent.

The effect of sleep loss on mood may vary depending on an individuals’ ability to regulate their emotional experiences. However, while reduced emotion regulation capacity is frequently discussed as one of the deleterious consequences of insufficient sleep among adolescents, very few studies have directly assessed this, with

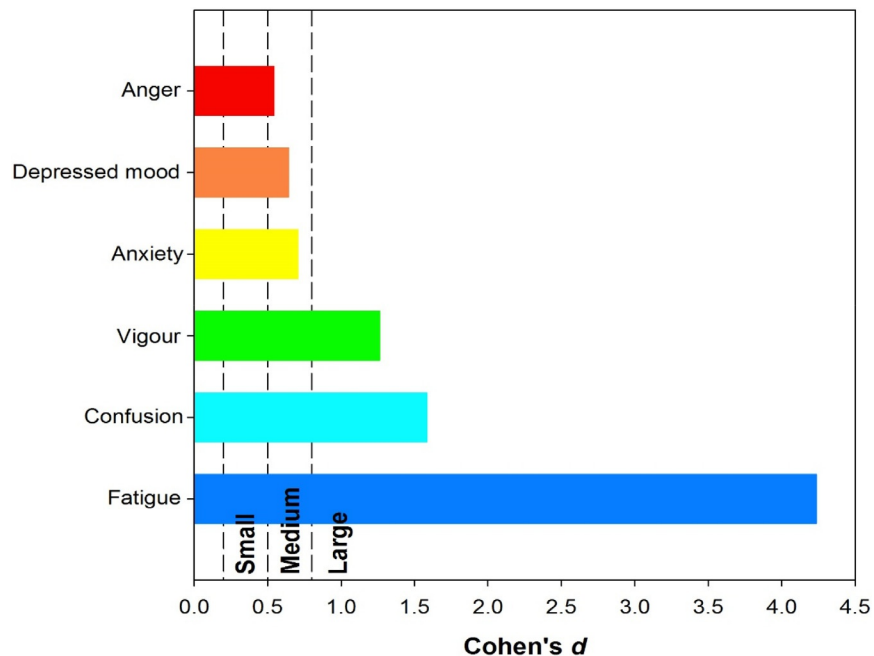


FIG. 2

Relative effect sizes of 1 night of total sleep deprivation on subjective indicators in a sample of adolescents aged 14–18 years.

most inferring emotion dysregulation from observed mood deficits. Emotion regulation is the ability to identify, monitor, evaluate and modulate one's emotional responses to function adaptively in different contexts and to achieve goals (McLaughlin et al., 2011). It encompasses different strategies, such as cognitive reappraisal, whereby an individual manages their emotional response to a stimulus by reinterpreting the meaning they initially ascribed to it, and expressive suppression, which involves suppressing the verbal and non-verbal expression of emotion to reduce one's subjective emotional experience (Niedenthal et al., 2006). The flexible use of different emotion regulation strategies assists with managing emotion in different environments (Cai et al., 2018). When people experience emotion dysregulation, their risk of psychopathology increases, with emotion dysregulation being an important transdiagnostic risk factor across many psychological conditions (McLaughlin et al., 2011).

Experimental studies support the notion that adolescents become more emotionally dysregulated with insufficient sleep (Baum et al., 2014; Talbot et al., 2010; Vriend et al., 2013). One randomized, counterbalanced, cross-over experimental design exposed 50 adolescents aged 14–17 years, to 5 nights of sleep restricted to 6.5 h' time in bed per night for 5 nights and 5 nights of healthy sleep, with 10h' time in bed per nights for 5 nights. As well as deficits in mood and oppositionality, both adolescents and parents reported worse emotion regulation during sleep restriction

(Baum et al., 2014). Another experimental protocol involving more acute sleep loss compared the affective functioning of adolescents aged 10–16 years after 2 nights of 7–8 h sleep for 2 nights and following 2 nights of sleep restriction, with 6.5 h sleep on the first night and 2 h sleep on the second. When sleep restricted, adolescents experienced greater anxiety during a catastrophising task and rated the likelihood of catastrophes as higher (Talbot et al., 2010).

Insufficient sleep is a risk factor for suicidal ideation and suicide attempts in adolescents (Liu and Buysse, 2006). In a study that examined the association between sleep duration and suicidality in over 8000 Taiwanese adolescents (Yen et al., 2010), short sleepers, who averaged <6 h sleep per night, were compared to long sleepers, who averaged >8 h sleep per night, and average sleepers, who obtained between 6 h and 8 h sleep per night. Results suggested that longer sleep durations conferred a benefit in reducing suicidality, with long sleepers reporting the lowest levels of suicidality, followed by the average sleepers, while the shortest sleepers reported suicidality that was higher than both other groups. After controlling for moderating factors of depression and sociodemographic characteristics, short sleep was still significantly associated with heightened suicidality. Of interest, however, subgroup analyses indicated that this relationship only was only present in short sleepers who were not depressed. Whether this association occurs due to the impact of sleep loss on mood, impulsivity, or through other mechanisms, such as perfectionism (Boergers et al., 1998), nighttime rumination (Lovato and Gradisar, 2014; Miranda and Nolen-Hoeksema, 2007), lack of positive expectations for the future (Chou et al., 2013), or failing to reach personal and familial academic expectations (Lee et al., 2006), warrants future research attention. Another study investigated sleep and suicidal behavior in 1362 Chinese adolescents aged 12–18 years (Liu, 2004). Nearly 1 in 5 adolescents reported suicidal ideation and 1 in 10 reported a suicide attempt in the past 6 months. After controlling for depressive symptoms and demographic variables, sleep duration was not significantly associated with suicidal ideation, however, sleeping <8 h per night increased the likelihood that an adolescent would attempt suicide by nearly three times.

5 Mechanisms underlying affective effects of sleep restriction

There are many mechanisms posited to explain the effect of sleep loss on mood and emotion regulation. The effect that sleep loss has on brain function is the common theoretical framework overarching these mechanisms. Sleep loss is known to result in diminished activity in prefrontal regions implicated in planning, decision making, and moderating social behavior, as well as a functional disconnection between prefrontal and limbic brain regions (Yoo et al., 2007). This exacerbates the existing biological propensity during adolescence for increased limbic activity, which biases behavior toward sensation-seeking, reward and emotion, and relatively underdeveloped prefrontal cortex functioning, which is needed to temper these

impulses with reasoning and consideration of long-term goals and consequences (Casey et al., 2008). Sleep loss heightens the already elevated physiological propensity for emotional instability (Astill et al., 2012).

Sleep loss leads to reduced paradoxical or rapid eye movement (REM) sleep in adolescents in exchange for preserved SWS (Ong et al., 2016). REM sleep is implicated in the processing of emotional memories. Two central theories describe the role of REM sleep in emotional memory processing. While both argue that REM helps with the processing of emotional memories via the reactivation of brain networks involved in the acquisition of emotional memories, one posits that REM sleep helps to separate the emotional tone from declarative memories by strengthening the declarative memory component while diminishing the emotional memory component (Goldstein and Walker, 2014; Walker and van Der Helm, 2009), while the other theory holds that REM sleep strengthens the relationship between an event and its emotional salience (Baran et al., 2012; Werner et al., 2015). Just as the flexible use of emotion regulation strategies is argued to be adaptive, it is possible that there is a role for both of these processes, with strengthened declarative-affective relationships important in threat-related situations and weakened declarative-affective relationships occurring for less emotionally salient events (Palmer and Alfano, 2017).

The impact that sleep loss has on cognitive functioning may also contribute to diminished affective functioning, as aspects of affective reasoning and emotion regulation, such as identifying and monitoring emotion, deciding on whether regulation is needed, selecting an emotion regulation strategy and implementing it, all rely upon cognitive processing (Palmer and Alfano, 2017). In addition to emotion regulation, there are myriad facets of cognition that both effect and are affected by mood. For example, negative memory bias is a thought to contribute to the development and maintenance of depression. REM sleep is argued to selectively consolidate emotional memories that have a negative tone. As REM sleep tends to be greater in both duration and density in individuals with depression, it is plausible that these REM alterations may contribute to the development of negative memory bias in susceptible individuals (Harrington et al., 2018). Another facet of mood-related cognition is repetitive negative thinking (RNT). RNT is the persistent focus of attention to the negative aspects of one's experiences (Nota and Coles, 2015). RNT is a transdiagnostic risk factor for several psychological disorders, including mood disorders and insomnia (Harvey, 2002; Klemanski et al., 2017). Sleep loss is argued to affect RNT by reducing an individual's ability to shift attention away from negatively valenced emotional information (Nota and Coles, 2018).

Recent years have seen an increase in the number of well-controlled experimental and quasi-experimental studies examining the impact of sleep loss on adolescent functioning. These have been a much-needed addition to the literature, which had been largely populated with cross-sectional studies. These experimental studies provide evidence that the effect that sleep loss has on cognitive and affective functioning is *causal*, and not related to confounding factors, such as socioeconomic status or family environment.

6 Gaps in the literature and future research directions

Substantial developments have been made in our understanding of how sleep loss affects cognitive and affective functioning in adolescents but the significant disconnect between knowledge and action calls for concurrent research into countermeasures that on the one hand are scientifically sound and on the other, practically implementable.

Afternoon naps have been shown to be historically feasible and accepted but their adoption in a faster paced world is an opportunity for fresh research. For example, while we know that a 1 h nap has neurobehavioral benefits, it is unclear if shorter naps may have similar benefits in adolescents. Even if naps alleviate neurobehavioral deficits, how does napping in the setting of shortened nocturnal sleep affect a teen's overall health? Relatedly, naps have traditionally been studied as an "add on" to adequate nocturnal sleep. However, an increasing number of persons will choose to segment their total 24 h sleep allocation instead of obtaining one continuous stretch of nocturnal sleep. Under such conditions, to what extent is continuous nocturnal sleep preferable (or not) to one major sleep episode at night and a shorter daytime nap? What combination of nocturnal and daytime napping is most beneficial? How important to cognition is it to have regular sleep? In this regard, the widespread adoption of wearable sleep-proxy monitors is providing an unprecedented avenue for data collection to examine the impact of different sleep patterns and their interaction with interindividual differences in sleep need across different cognitive domains.

The cognitive and affective deficits that result from insufficient sleep do not occur in isolation but are often bidirectional in that many neuropsychiatric disorders affect sleep, cognition, mood and well-being. As such, the field would profit by taking an integrated approach in considering how cognition and affective functioning are affected by sleep loss, and how they affect one another as dynamic processes triggered by sleep loss likely cause a cascade of negative and interacting effects across both domains. Indeed, it may be impossible to isolate how sleep loss affects cognitive functioning without factoring in how affect might bias cognition and vice versa. With evidence for binary causal relationships between adolescent sleep restriction and deficits to cognition and mood established, it is timely that the field expands to take a broader approach.

An important area for future research is to determine the mechanisms by which sleep loss affects the onset, maintenance and recurrence of psychological disorders. Insufficient sleep is likely a transdiagnostic risk factor for disorders such as depression, anxiety, social anxiety disorder, bulimia, binge eating disorder, and obsessive-compulsive disorder. Resultingly, consideration must be given to existing disorder-specific conceptual models of psychological disorders. Disorder-specific models explain the development and maintenance of a psychological disorder. They remain an under-utilized tool from which to identify specific cognitive and affective processes likely affected by sleep, and thus guide both research and clinical practice.

For example, Beck's unified model of depression highlights multiple mechanisms through which sleep loss may negatively act, including mood, anhedonia, depressogenic beliefs, negative cognitive appraisal, negative automatic thoughts, suicidal ideation, cognitive restructuring, rumination (or RNT), problem solving, avoidance, interpersonal conflict and social support seeking (Beck and Bredemeier, 2016). Chronic sleep loss may act both directly and indirectly on many, if not most, of these factors proposed to explain depression across both cognitive and affective levels, yet the research is lacking.

7 Concluding remarks

While sleep has long been recognized as one of the pillars of health, gaining traction to improve the sleep of adolescents remains challenging. The current review highlights the importance of sufficient sleep for core aspects of adolescent functioning, with chronic insufficiency negatively impacting thinking, learning and well-being. While optimal nocturnal sleep is the current ideal, enacting the changes that would enable this, will require the combined efforts of individuals, families, educators and community leaders. While this process must continue, urgent action is required to mitigate existing effects of pervasive sleep reduction.

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