

Variations in the sleep–wake cycle from childhood to adulthood: chronobiological perspectives

Joanne S Carpenter
Rébecca Robillard
Ian B Hickie

Clinical Research Unit, Brain and Mind Research Institute, University of Sydney, Camperdown, NSW, Australia

Abstract: Changes in the sleep–wake cycle across development from childhood to adulthood, typically involve a steady shortening of the sleep period and a delay of sleep phase, with a period of more rapid change across adolescence. Accompanying these changes is the maturation of neuroendocrine rhythms such as melatonin, cortisol, and pubertal hormones. These endogenous rhythms are closely associated with behavioral changes in rest and activity rhythms, although environmental factors such as light exposure and academic and social demands likely play an interactive role. Other behavioral aspects, such as physical activity and eating behaviors, are also associated with changes in sleep–wake rhythms, and may be mediational factors in the development of physical illnesses. The sleep–wake cycle and related factors are implicated in the development of mental illnesses. There are several potential avenues of future research that may be valuable in terms of improving interventions and treatments for both mental and physical illnesses.

Keywords: circadian rhythm, developmental, adolescence, youth

Introduction

The human 24-hour sleep–wake cycle and underlying biological rhythms undergo several significant changes across development from childhood to adulthood. Adolescence in particular is characterized by substantial changes in the circadian pattern of activity and rest, which occur alongside pubertal maturation and neurodevelopment, including maturation of the neuroendocrine system. Changes in the sleep–wake cycle are also associated with lifestyle and environmental factors such as diet, exercise, and school schedules. Many of these changes take place in systems implicated in the development of mental and physical illnesses and as such it is important to consider sleep–wake and circadian rhythms as potential contributing factors to deviations from healthy development. This review aims to present an overview of the current literature on sleep–wake and circadian changes from childhood to adulthood, focusing on chronobiological aspects of these changes and associated roles in the development of illness.

Changes in the timing of sleep and activity

A consolidated sleep–wake pattern is generally established during the first 6 months of life, with the majority of waking activity occurring during the day and one main nocturnal sleep episode.¹ Across infancy and childhood, this pattern continues to consolidate with a decrease in the frequency and duration of daytime naps.^{2,3} Mean habitual sleep duration also decreases steadily across this period (from 12.8 hours in

Correspondence: Ian B Hickie
Clinical Research Unit, Brain and Mind Research Institute, University of Sydney, Building F, Level 4, 94 Mallett Street, Camperdown, NSW 2050, Australia
Tel +61 2 9351 0810
Fax +61 2 9351 0652
Email ian.hickie@sydney.edu.au

infants to 11.9 hours in 2–5 years old),⁴ and continues to do so throughout childhood (9.2 hours in 6–12 years old),⁴ the adolescent period (8.1 hours in 16 years old),^{2,5} and into adulthood (7.5 hours in 24 years old).⁶

The shortening of sleep duration across childhood and adolescence is accompanied by a delay in the phase of the sleep–wake cycle, particularly characterized by progressively later bedtimes.^{2,7} This delay of sleep onset becomes more pronounced in adolescence, with reports of 5–7 minutes delay

per year from 7 to 9 years old, and 10–17 minutes delay from 9 to 12 years old.³ Wake-up times follow a slightly different pattern, remaining relatively stable across childhood, and beginning to occur later in adolescence, most prominently on weekends.⁷ The adolescent delay in the sleep–wake cycle is one of the most widely reported changes in sleep habits, with consistent reports across a variety of different cultures around the globe.⁵ Typical changes in sleep–wake timing from childhood to adulthood are illustrated in Figure 1.

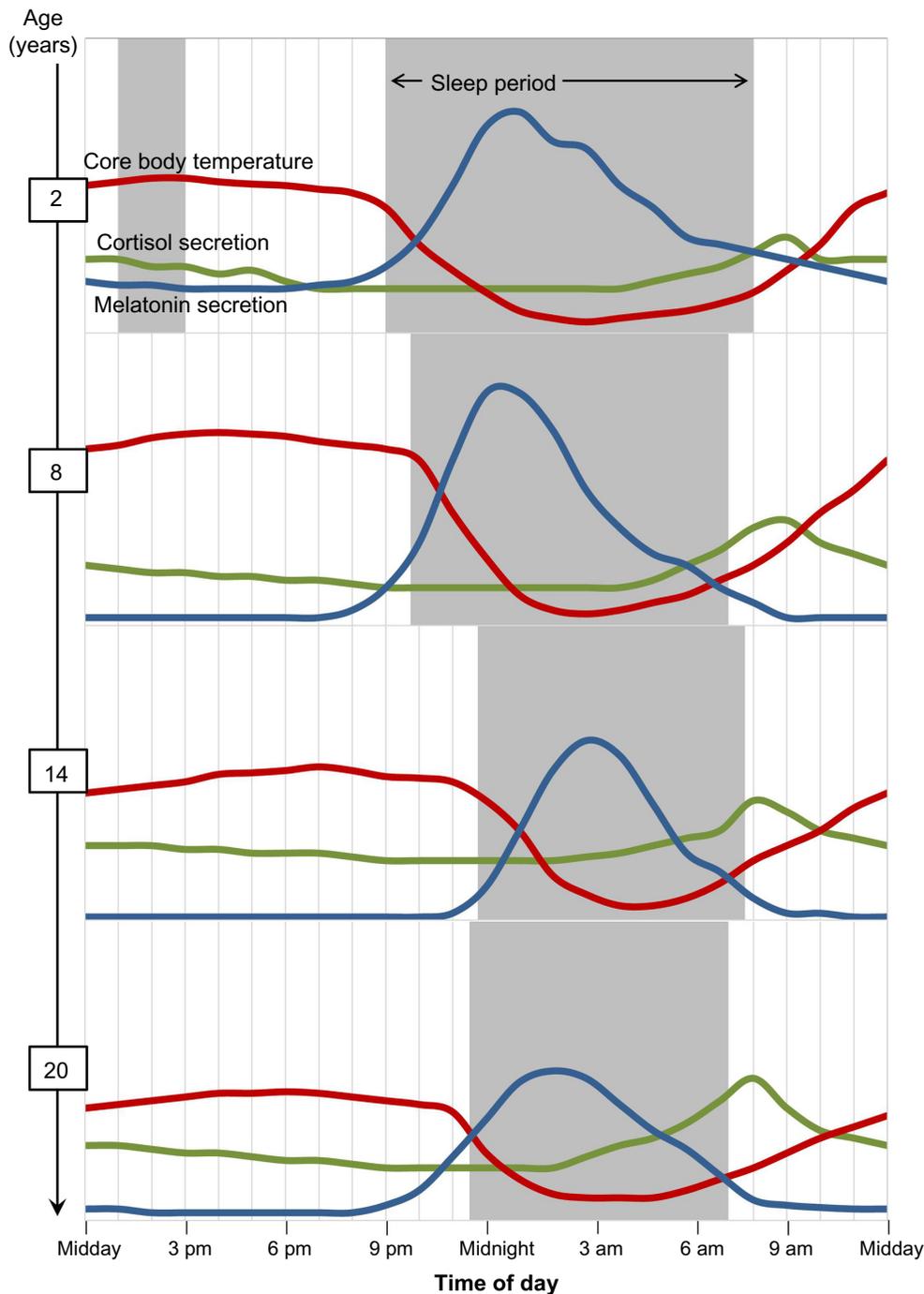


Figure 1 Illustration of changes in 24-hour rhythms of sleep–wake, melatonin secretion, cortisol secretion, and core body temperature from childhood to adulthood.

Accompanying the sleep–wake cycle changes in adolescence is a shift of chronotype toward eveningness,^{8–10} ie, a greater preference for activities to be undertaken later in the day and for later bed and rise times. This shift toward eveningness is estimated to begin at 13 years of age, with a peak at 17–20 years for females and 20–21 years for males.^{11–13} The shift reverses – with a change back toward morningness – across adulthood.^{13,14}

As discussed in the following sections, the sleep–wake cycle changes taking place during childhood and adolescence are influenced by close interactions between a range of neuroendocrine, environmental, occupational, and behavioral factors, and may increase vulnerability to the development of mental and physical illnesses.

Factors modulating developmental changes in the sleep–wake cycle

Circadian rhythms and neuroendocrine factors

Changes in endogenous circadian rhythms are a potential driver of the sleep–wake changes seen across development. The mammalian sleep–wake cycle is accompanied by intrinsic circadian rhythms of gene expression, metabolic function, temperature, and hormone production, driven by a complex system of internal pacemakers, environmental inputs, and feedback loops.^{15–19} Measurable outputs of endogenous rhythms such as hormone secretion and body temperature undergo changes across development. Phase delays during adolescence are also displayed in several non-human mammal species,²⁰ indicating that the phenomenon is likely driven by some common feature of mammalian biology. There are several plausible theoretical explanations linking changes in endogenous rhythms with the sleep–wake changes seen across development.

The pineal hormone melatonin plays a key role in the regulation of the sleep–wake cycle and follows a 24-hour pattern of secretion, with an evening onset, a nocturnal peak, and a decrease in the morning.²¹ The daily rhythm of melatonin secretion is not present at birth, but develops in the 1st months of life,^{22–25} is strongest at around 4–7 years of age and then steadily decreases in amplitude across the lifespan.^{26–28} In adolescence, the onset and offset of melatonin secretion have been shown to be delayed with increasing age and throughout pubertal development.²⁹ A later onset of melatonin secretion in the evening is likely to contribute to sleep initiation difficulties and delayed sleep onset. Accordingly, between 7% and 36% of adolescents report difficulty falling

asleep, while 20%–26% report a sleep onset latency greater than 30 minutes.³⁰ In addition, administration of afternoon/evening melatonin to adolescents has been demonstrated to successfully advance the phase of circadian rhythms and sleep timing.³¹

Cortisol secretion also follows a strong circadian rhythm, with high levels upon morning awakening, increasing to a peak about 30 minutes after awakening, then decreasing across the day to the lowest levels around bedtime.^{32,33} The circadian rhythm of cortisol secretion develops during the 1st year of life, and is associated with the establishment of a consolidated sleep–wake cycle.^{34–36} Alongside the decrease in napping across the first 3 years of life, the decrease of cortisol across the day smoothens and follows a slope more similar to that of adults.³² Across adolescence, cortisol levels increase and follow a flatter rhythm with age and pubertal development.^{37,38} A sex difference also emerges across adolescence, with higher morning cortisol levels in girls, likely related to gonadal changes associated with pubertal development.^{37,39,40} A bidirectional relationship between the timing and length of sleep and cortisol patterns in adolescents has been reported with a steeper decline in cortisol across the day associated with longer sleep.⁴¹

A circadian rhythm of core body temperature (CBT) is established in the 1st year of life, showing a peak during the day and a drop to a trough at night between 1 and 6 hours after sleep onset.^{42–45} This rhythm has a greater amplitude in children (between 7 months and 7 years old) than in adults.⁴² Later timing of the CBT rhythm has been associated with eveningness in adolescents and adults,^{46,47} and the rhythm has been reported to shift to an earlier phase across adulthood.⁴⁸ Data on changes in the CBT rhythm across adolescence are lacking, but a report on three case studies suggests that the rhythm may delay across this period.⁴⁹

Another potential influential factor is pubertal maturation, which is reported to be associated with both the adolescent decrease in sleep duration and the delay in sleep onset.⁵⁰ Decreases in sleep duration have also been shown to predict pubertal changes longitudinally.⁵⁰ Thus, it follows that pubertal hormones may be linked to these adolescent sleep–wake and circadian rhythm changes. A recent review by Hagenauer and Lee⁵¹ examines the evidence that the development of adolescent sleep–wake cycles and reproductive hormone secretion are bidirectionally related. Although most of the evidence for this relationship comes from animal studies, there is reason to believe that human circadian and reproductive systems are closely linked, with the biological clock regulating the timing of reproductive events such

as hormone release and ovulation, and gonadal hormones influencing circadian parameters such as photic sensitivity and activity levels and timing. As such, development of one system in adolescence is likely to influence the other to some degree. The chronotype shift toward eveningness in adolescence has also been shown to be related to pubertal development^{14,52} and to increases in testosterone.⁵³

The changes observed in the sleep–wake cycle, hormone secretion, and temperature rhythms may be explained by a variety of other factors. For instance, a lengthening of the intrinsic circadian phase would drive later timing of hormone secretion, which may in turn promote later sleep and wake times. In support of this, a study by Carskadon et al⁵⁴ in which participants were maintained on a 28-hour day (forced desynchrony protocol) suggested that the intrinsic circadian period length in adolescents was approximately 24.3 hours, which is longer than some, but not all, reported intrinsic phases for adults. These results should be taken as preliminary given the small sample size (five boys and five girls), and no replication has yet been done. In addition, behavioral changes and other environmental influences (such as light exposure) may feed back into the internal timing system to elicit a shift in the internal pacemaker. More complex interactions between internal and external factors may also play a role, for example, changes in sensitivity of the internal pacemaker to light has been suggested as a possible mechanism for the phase delay in adolescence.⁵⁵

In conjunction with the chronobiological and pubertal maturation perspectives, a recent homeostasis model proposed by Hagenauer and Lee²⁰ provides another possible account of delays in sleep–wake timing and shortening of sleep length. According to the model, a decrease in the buildup of sleep pressure across the day and in its dissipation across the night predicts the observed delay in sleep–wake timing and shortening of duration.²⁰ The findings of Jenni et al⁵⁶ provide support for a slower buildup of sleep pressure based on changes in waking slow wave activity across adolescence; however, no changes were found in slow wave activity during sleep. Another theoretical model proposed by Phillips et al⁵⁷ integrates several parameters to represent influences of circadian and homeostatic processes on neuronal firing and resultant circadian preference. This model explains the shift to eveningness in adolescence by a reduced strength of the homeostatic drive relative to the circadian drive.

Environmental factors

Human circadian rhythms are entrained to a 24-hour cycle by various environmental cues, the most influential of which

is light.^{58,59} Exposure to morning light typically advances circadian phase, whereas exposure to evening light typically results in a delay.^{60,61} Thus, increased evening light exposure and decreased morning light exposure may contribute to the phase delays experienced in adolescence. In support of this, evening light exposure has been shown to correlate with eveningness in adolescents,⁶² and adolescents without electric lighting have been shown to have earlier sleep onset times.⁶³ Although other factors may be at play (eg, cultural and occupational differences, absence of technology in those without electric lighting), this provides preliminary evidence for a role of light exposure in adolescent phase delays. On the other hand, changes in endogenous circadian rhythms may drive behavior leading to altered light exposure patterns. For example, a delay in endogenous phase may lead to later bedtimes as well as encouraging later light schedules. An alternative hypothesis is that adolescents experience an increased sensitivity to evening light and a decreased sensitivity to morning light.⁵⁵

Various other environmental and behavioral factors have been proposed as contributors to sleep–wake and circadian changes in adolescence. Increasing academic and social demands are thought to be implicated in the extension of waking activity into the evening and subsequent delay and shortening of the sleep period.^{8,64,65} Increased use of electronic media seems to have a similar effect.^{66,67} The decrease in parentally set bedtimes has a noteworthy effect, with significantly later bedtime and shorter sleep duration in those without parent-set bedtimes.⁶⁸ Another contributing factor is working status, with adolescents who engage in employment displaying shorter sleep durations and increased daytime sleepiness compared to their non-working peers.^{69–71} Fischer et al⁷¹ also found shorter adolescent sleep duration to be related to increased smoking and drinking behavior. Many of these environmental and behavioral factors relate to social and emotional maturation and increased independence, and are likely to interact with the biological factors described above in influencing sleep–wake and circadian changes. In addition, several environmental factors are known to influence chronotype, including photoperiod at birth, geographical location, and daylight savings time.^{72,73} As such, age-related chronotype and sleep–wake changes may be differentially influenced by multiple factors, with potentially complex interactions.

Occupational constraints: school schedules

There is evidence to suggest that the restrictions imposed by school schedules may be a key driver in the shortening

of sleep duration across childhood and adolescence. If it is assumed that the steady delay in sleep onset is part of a delay in the whole 24-hour sleep–wake cycle, it is reasonable to believe that forced wake-up times may be cutting short the sleep period. In support of this, studies comparing weekend sleep durations across adolescence have found no differences with increasing age;^{74,75} thus, the shortened duration is likely to be specific to factors present only on weekdays. Further, in countries with earlier school starting times, adolescent sleep duration is reported to be shorter.⁵ This influence of school schedule on sleep duration is supported by one meta-analysis of objective measures (polysomnography and actigraphy) that found total sleep time decreased across adolescence only when the measures were taken on school days.⁷⁶ In contrast, a meta-analysis of subjective measures (self- or proxy-report based studies) noted a significant decline in both school and non-school day sleep duration with increasing age, although the school day decline was significantly steeper than the non-school day decline.⁵

On weekends, both bed and wake times are reported to become later with increasing age across adolescence.⁷⁷ This is accompanied by a greater disparity between sleep duration on school and non-school days, resulting in lengthier sleep episodes on weekends (a 25-minute difference at age 9 increasing to an 86-minute difference at age 18).^{5,78–80} This pattern becomes significant at age 9,⁷ and is related to pubertal development.⁸¹ This difference between weekday and weekend sleep duration does not appear to be present during vacation from school, and overall sleep duration is longer in these vacation periods.⁸² As such, it is likely that during the school week, adolescents obtain inadequate amounts of sleep, driving them to “catch-up” on weekends and vacation periods. This is accompanied by increased daytime sleepiness across adolescence with increasing age,^{83–85} and increasing pubertal development.⁸⁶ The increase in sleepiness has been related to later bedtimes and shorter sleep times;⁷ however, it is also reported to occur in the absence of sleep deprivation.^{86,87} Thus, it remains to be established whether this increase in sleepiness is a direct consequence of inadequate sleep or related to some other factor such as changes in the sleep homeostatic system. Adolescents also experience an increase in naps,^{3,88} which may form part of the catch-up sleep phenomenon. In adults with evening chronotypes, an analogous weekend catch-up pattern is observed in response to work schedules.^{89,90}

The adolescent delayed sleep–wake pattern on weekends appears to be accompanied by a delay in endogenous

circadian rhythms (as measured by dim light melatonin onset).⁸⁰ This indicates that the behavioral shift of sleep–wake phase may occur in conjunction with a neurobiological shift, making it more difficult for the students to return to an earlier phase upon their return to school the following week. The resulting misalignment between internal circadian phase and occupational schedule is termed “social jet lag”, and is associated with elevated risk for negative mental and physical health outcomes.⁹¹ For most individuals, phase advances (shifts to earlier times) are more difficult to achieve than phase delays (shifts to later times);^{58,92} thus, the weekend catch-up sleep phenomenon may be detrimental to students’ functioning. In line with this, both shorter school night sleep duration and greater disparity between weekday and weekend sleep times have been associated with poorer academic performance, increased daytime sleepiness, greater emotional distress, more risk taking behavior, and increased depressive symptoms.^{93–96} Further, experimental extension of sleep duration in adolescents has been shown to improve aspects of cognitive performance.⁹⁷ In addition, eveningness is associated with higher cognitive abilities, yet is also related to poorer academic achievement,^{98,99} indicating that the scheduling of current educational activities may not be conducive to maximizing potential.

Educational programs designed to improve sleep in adolescents by encouraging better sleep habits have generally failed to produce sustainable behavioral changes.¹⁰⁰ However, delays in school start times have been reported to result in increased sleep duration, increased school attendance, improved motivation, decreased daytime sleepiness, and fewer mood symptoms.^{101,102} This evidence provides a promising avenue for policy change to adjust school scheduling in accordance with neurodevelopmental changes in the sleep–wake cycle and improve adolescent health and functional outcomes.

Behavioral factors: physical activity and eating behaviors

Physical activity peaks in mid-childhood and decreases across adolescence.^{103–105} In particular, time spent engaging in moderate and vigorous physical activity is reported to decrease across childhood and early adolescence in both cross-sectional and longitudinal studies.^{106–108} The decline across adolescence (from 11 to 16 years) has been found to be steeper than that of childhood (from 6 to 9 years) and is accompanied by an increase in sedentary time.¹⁰⁶ Evidence suggests that levels of physical activity remain low, or decrease further, into adulthood.^{105,106,108,109}

Increased sedentary behavior and decreased physical activity has been associated with poorer fitness,¹¹⁰ increased risk for obesity, diabetes, and cardiovascular disease,^{111–114} and increased incidence of psychiatric disorders,^{115,116} making this an area of critical health concern.

The timing of the adolescent decline in physical activity corresponds to that of the sleep–wake cycle changes and as such may form part of an overall change in 24-hour sleep and activity patterns. Several lines of evidence support close relations between sleep changes and activity changes across this developmental period. In one study, greater sedentary time was found in adolescents with sleep durations of less than 8 hours.¹¹⁷ In another study, 10–12 years old who slept less than 9 hours were less active than those sleeping 10 or more hours, and in addition, those engaging in weekend catch-up sleep were reported to be less active.¹¹⁸ In a third study, the frequency and duration of exercise bouts in 9–11 years old was found to be greater on weekdays than weekend days.¹¹⁹ These findings may reflect interacting disruptions in the regularity of sleep, wake, and activity patterns, which may have detrimental effects on health and functioning. A clear example of the effect of activity levels on sleep and functioning parameters comes from Brand et al,¹²⁰ who compared adolescent athletes to controls and found that the athletes (who spent an average of over 17 hours per week exercising vs less than 5 hours in the controls) reported higher sleep quality, shorter sleep onset latency, fewer awakenings after sleep onset, less daytime tiredness, and increased concentration, as well as fewer anxiety and depressive symptoms.

Later timing of sleep and wake, as well as greater eveningness has also been associated with decreased physical activity and increased screen time (computer/television use) in adolescence, as well as poorer attitudes toward physical activity.^{121–123} There is some evidence for a continuation of this relationship between sleep timing, chronotype, and physical activity into adulthood, with lower levels of physical activity in adults who have later sleep times¹²⁴ and a propensity for morningness in adult athletes.¹²⁵ It is thus important to consider both daytime activity patterns and sleep patterns as part of an overall 24-hour cycle and contributions of this to adverse health outcomes.

The sleep–wake cycle changes in adolescence are also associated with changes in diet and related health factors. Adolescents with shorter sleep durations present with unhealthy dieting behaviors¹²⁶ and are less likely to eat adequate fruit, vegetables, and fish.¹¹⁷ Short sleep duration is also associated with higher body mass index, body fat, and waist and hip circumferences in adolescents,¹¹⁷ and with

an increased risk for obesity in both children and adults.¹²⁷ Experimentally restricted sleep duration in adolescents has been shown to result in consumption of foods with higher glycemic indices,¹²⁸ indicating that shortened sleep may contribute to poorer food choices. Obese children and adolescents are also reported to have a greater incidence of sleep disordered breathing,^{129–131} which may cause fragmented sleep and increased daytime sleepiness, thus disruption of the rest–activity cycle. Children and adolescents with late bed and wake times have also been shown to have poorer diet quality, a higher intake of energy-dense, nutrient poor foods, and reduced consumption of fruit and vegetables independent of sleep duration.¹³² There also appear to be similar effects of chronotype, with reports of increased consumption of fast food¹³³ and longitudinal increases in body mass index¹³⁴ in adolescents with evening chronotypes. The negative associations between sleep–wake factors and eating behaviors continue into adulthood with less fruit and vegetable consumption and more calories consumed after 8 pm in those with later bed and wake times.¹³⁵

Further to behavioral changes in diet with increasing age, changes in the internal circadian timing system can have effects on metabolic processes, such as the timing of glucose metabolism and cardiovascular function.^{16,136} Thus, circadian misalignment, as seen in social jetlag, can have detrimental effects on health, with increased risk of obesity, insulin resistance, and hypertension.^{16,136} Conversely, the timing of food consumption feeds back into the internal timing system and can itself promote changes in endogenous circadian rhythms.¹³⁶ This reinforces the importance of maintaining regularity in sleep–wake behaviors, and eating behaviors, as well as maintaining a schedule in line with internal circadian phase.

Deviation from normal developmental changes

Delayed sleep phase disorder

A potential deviation from normal developmental changes of the sleep–wake cycle can be seen in delayed sleep phase disorder (DSPD). This condition is characterized by severely delayed timing of sleep onset and offset, increased daytime tiredness, and consequent impairment in daytime functioning, and has typical onset in childhood or adolescence.^{137,138} The disorder may represent an exaggerated case of the delay that occurs across development, and as such may be a malfunction of the normal maturational development of sleep and activity patterns. DSPD is also linked to abnormalities in endogenous rhythms, with a delay of melatonin secretion and

CBT accompanying the delay in sleep–wake patterns.^{139–144} Additionally, evening exogenous melatonin administration has been demonstrated as an effective treatment for this disorder.^{145–147} DSPD is associated with various functional difficulties (such as failure to attend school)^{137,148} as well as increased incidence of depressive symptoms.^{137,149} As such, screening to identify individuals with exaggerated or abnormal changes in sleep–wake cycles during childhood and adolescence may prove useful in early intervention and prevention for DSPD.

Mental illness

Deviations from normal developmental sleep–wake changes may also form risk factors for adverse mental health outcomes. In one instance, shortened sleep duration is found in several neurodevelopmental and mental disorders including mood disorders, anxiety disorders, attention deficit hyperactivity disorder (ADHD), schizophrenia, and alcohol dependence.^{150–153} Similarly, a delay in timing of the sleep–wake cycle (including delayed sleep onset and offset) has been associated with anxiety disorders, depression, bipolar disorder, seasonal affective disorder, and psychotic disorders.^{154–158} Various disturbances in sleep are found in mental disorders: increased sleep latency, awakenings, limb movement disorders, and sleep disordered breathing in ADHD;^{159,160} increased sleep latency, awakenings, nightmares, and night time panic attacks in anxiety disorders;^{161–163} increased sleep latency and awakenings in affective disorders;^{162,164,165} irregular and fragmented sleep in psychotic disorders;¹⁶⁶ and increased sleep latency, awakenings, and sleep disordered breathing in alcoholism.^{167,168} Increased daytime sleepiness is also found in ADHD and major depression.^{160,169,170} There is a period of increased onset of many of these mental illnesses during adolescence,^{171,172} which coincides with the period of increased change in sleep–wake patterns outlined above. An increased incidence of depressive symptoms is also found in children and adults with sleep disordered breathing^{173–175} which may reflect, among other possible pathophysiological mechanisms, an influence of disruption in the sleep–wake cycle on the development of mental illness. Sleep–wake changes have been indicated as prodromal symptoms for several mental disorders^{164,176–178} and have been shown to worsen in more severe and persistent illness phases.^{179,180} Thus, anomalous changes in sleep and activity patterns across childhood and adolescence may form part of the etiology of these conditions and should be considered as a potential risk factor.

In conjunction with changes in the timing and length of sleep, irregularities of 24-hour sleep and activity patterns have been implicated as risk or causal factors for mental illness. For example, seasonal affective disorder and non-seasonal depression have been associated with a lower amplitude of circadian activity rhythms in children.^{181,182} Irregularity of these patterns has also been demonstrated in both bipolar disorder¹⁸³ and ADHD.¹⁸⁴ These two disorders are characterized by abnormally elevated activity levels, which may be reflective of failure to develop a regular pattern of sleep and activity. There is also evidence of increased variability and reduced stability of sleep–wake cycles in those at high risk for bipolar disorder,¹⁷⁶ as well as longitudinal evidence that irregularity of sleep–wake patterns in childhood is associated with increased behavioral difficulties,¹⁸⁵ and incidence of depressive and anxious disorders in adolescence and adulthood.¹⁸⁶ Symptoms themselves show time of day variations, including hyperactivity and inattention in ADHD,¹⁶⁰ as well as mood in both healthy and mood disordered patients.^{187–189}

Disruption of endogenous biological rhythms likely contributes to behavioral abnormalities in sleep and activity patterns, and may also play a role in the development of mental illnesses. Elevated cortisol levels in adolescence have been implicated in the development of several mental illnesses including anxiety,¹⁹⁰ schizophrenia,¹⁹¹ and affective disorders.¹⁹² Major depression has been shown to be associated with elevated cortisol levels in the evening.^{193,194} Bipolar disorder has also been associated with abnormalities in cortisol secretion, with elevated levels in manic, depressive, and euthymic states.¹⁹⁵ Abnormalities in melatonin secretion are found across several diagnostic subgroups of psychiatric patients, including those with mood,^{196–199} anxiety,²⁰⁰ and psychotic^{201–203} disorders. There is also some evidence to suggest a desynchrony between endogenous melatonin rhythms and rest–activity cycles in young people with mood disorders.^{91,199} Further, interventions that target the melatonin system have shown promise in treatment of mood disorders.^{204–206} In ADHD, there is some evidence for a delay of melatonin rhythms in children and adults with comorbid insomnia, and abnormal patterns of cortisol secretion have been reported in children.¹⁶⁰ However, results are mixed on the direction of deviations and roles of comorbid factors, and a specific role of endogenous rhythm disruptions in ADHD remains to be established.¹⁶⁰ Circadian gene polymorphisms and circadian gene expression have been implicated in the development of anxiety disorders, affective disorders, psychotic disorders, ADHD, and alcoholism,^{207–216} further

supporting a role of circadian systems in neurodevelopmental and mental disorders.

Conclusion

The developmental period between childhood and adulthood involves changes in the sleep–wake cycle, and the circadian rhythm of rest and activity, most notably a delay in sleep onset and shortening of sleep duration. Several environmental and biological factors appear to contribute to these changes, including the maturation of neuroendocrine systems, school schedules, and light exposure. There is potential to improve adolescent functioning and academic performance with informed adjustments of school schedules. Changes in the sleep–wake cycle and circadian rhythms are also associated with various aspects of physical activity, eating behaviors, and physical and mental health, and thus represent important factors to consider in investigations of the etiology and treatment of mental and physical illnesses. Current understanding of these areas would benefit from longitudinal research into specific causal relationships between rest and activity patterns and the development of mental and physical illnesses. The efficacy of treatments for mood disorders targeting the melatonin system illustrates the value of considering sleep–wake and circadian systems in the development of novel treatments; further expansion of this area will be of considerable interest. Future research should also consider the potential utility of screening to identify individuals with abnormal development of sleep–wake cycles and the possible effectiveness of sleep and circadian-based prevention and intervention strategies in relation to health outcomes.

Disclosure

The authors would like to declare the following conflicts of interest: IBH is a Commissioner in Australia's new National Mental Health Commission from 2012. He was a director of headspace: the national youth mental health foundation until January 2012. He was previously the chief executive officer (till 2003) and clinical adviser (till 2006) of beyond blue, an Australian National Depression Initiative. He is the executive director of the Brain and Mind Research Institute, which operates two early-intervention youth services under contract to headspace. He has led a range of community-based and pharmaceutical industry-supported depression awareness and education and training programs. He has led projects for health professionals and the community supported by governmental, community agency and pharmaceutical industry partners (Wyeth, Eli Lilly, Servier, Pfizer, and AstraZeneca) for the identification and

management of depression and anxiety. He has received honoraria for presentations of his own work at educational seminars supported by a number of non-government organizations and the pharmaceutical industry (including Servier, Pfizer, AstraZeneca, and Eli Lilly). He is a member of the Medical Advisory Panel for Medibank Private and also a Board Member of Psychosis Australia Trust. He leads an investigator-initiated study of the effects of agomelatine on circadian parameters (supported in part by Servier) and has participated in a multicenter clinical trial of the effects of agomelatine on sleep architecture in depression and a Servier-supported study of major depression and sleep disturbance in primary care settings.

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