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The Characteristics of Sleep and Sleep Loss in Adolescence: A Review

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Abstract

Healthy, adequate sleep is integral to the process of growth and development during adolescence. At puberty, maturational changes in the underlying homeostatic and circadian sleep regulatory mechanisms influence the sleep-wake patterns of adolescents. These changes interact with psychosocial factors, such as increasing academic demands, hours spent in paid employment, electronic media use, and social opportunities, and constrict the time available for adolescents to sleep. Survey studies reveal that adolescents' habitual sleep schedules are associated with cumulative sleep loss. As a consequence, there is growing concern about the effects of insufficient sleep on adolescents' waking function. This review identifies and examines the characteristics of sleep and sleep loss in adolescents. It highlights the need for more research into the effects of chronic partial sleep deprivation in adolescents, and the process of extending sleep on weekends to recover the effects of sleep debt. An understanding of chronic sleep deprivation and recovery sleep in adolescents will facilitate the development of evidence-based sleep guidelines and recommendations for recovery sleep opportunities when habitual sleep times are insufficient.

Keywords: Adolescence, puberty, sleep-wake patterns, sleep need, sleep deprivation, sleep debt, recovery.

1. Introduction

Adolescence is the critical period of transition between childhood and adulthood. Spanning the second decade of life, this sensitive developmental phase emerges from the interaction of neural and behavioural maturation, and results in a sexually mature adult (Sisk and Foster 2004). Healthy, adequate sleep is integral to the process of growth and development during adolescence (Anstead 2000; Dahl and Lewin 2002) Although its specific purpose remains unclear (Siegel 2005), it appears that sleep is particularly important during periods of brain maturation (Dahl 1999). At puberty, changes occur in the way the brain regulates the timing of sleep (Berk 2004), and, across species, increased sleep requirements are observed in maturing versus fully mature individuals (Dahl 1999).

Adequate sleep is also a critical factor in adolescent health and health-related behaviours(Chen, Wang and Jeng 2006). In particular, sleep is important for the control of behaviour, emotion, and attention, which, in turn, has implications for the development of social and academic competence, and psychological health (Dahl et al. 2002). Conversely, inadequate sleep in adolescence is associated with impaired functioning in a broad range of domains, including daytime alertness, motor skills, memory, attention, concentration, motivation, problem solving, emotion regulation, and mood, and is associated with increased risk of accidents and injuries (Carskadon, Acebo and Jenni 2004).

It is of great importance, therefore, to identify and understand the factors that influence the regulation of sleep during this sensitive period of development, as well as the consequences of insufficient sleep. The purpose of the following review is to identify and examine the literature on the characteristics of sleep and sleep loss in adolescents. It focuses on summarising data collected in adolescents; however, due to a scarcity of research on some aspects of adolescent sleep, adult literature will be integrated. Articles cited in this review were accessed from searches conducted through Google Scholar, Psych Articles and Psych Info. Information was also obtained from key textbooks in fields of sleep and sleep medicine (Ferber and Kryger 1995; Carskadon 2002; Lee-Chiong 2006).

2. What Constitutes Normal Sleeping Patterns? Childhood to Adolescence

2.1 Developmental Patterns in Sleep Duration: Birth to Adolescence

Predictable developmental changes occur in the quantity and quality of children's sleep from birth to adolescence (refer Table 1) (Iglowstein, Jenni, Molinari and Largo 2003). At birth, newborns spend 16 to 18 hours per day asleep, and have no clear circadian organization of their sleep-wake patterns (Rosen 2006). At this age, sleep in infants is distributed evenly across the day and night with sleep-wake states alternating in 3- to 4-hour cycles (Anders, Sadeh and Appareddy 1995; Jenni and Carskadon 2005). The ability of newborns to sustain longer episodes of wakefulness gradually increases from a few weeks old as sleep-wake organisation adapts to the light-dark cycle and regular social cues (Anders et al. 1995). By six to nine months of age, most infants have achieved the developmental milestone of 'sleeping through the night' as they become physiologically capable of consolidating 8 hours of sleep without a behavioural awakening (Anders and Keener 1985). Across the first year of life, total sleep duration remains fairly constant, with an average of 14.5 hours a day (Jenni et al. 2005). At this age, sleep periods have shifted to the night-time and waking periods to the day, with the exception of one to two daytime naps (Thoman and Whitney 1989).

Table 1: Average Sleep Duration: Birth-Adolescence

Age	Average Number of Hours of Sleep/Day	Range (h/day)	Naps
Infants (2-12 months)	14.5 h	10-16 h	3
Toddlers (1-3 years)	13.5 h	9-16 h	2
Preschool (3-5 years)	11 h	8-12 h	1
School age, preadolescent (5-13 years)	10 h	8-10 h	0
Adolescent (delay in sleep phase, increase in daytime sleepiness)	9.25 h	8-12 h	0

Adapted from (Rosen 2006)

During the second year of life, toddlers sleep between 11 and 12 hours a night, but daytime naps usually decrease to a single nap a day of one to two hours (Rosen 2006). Between three and six years of age there is usually a decrease of sleep duration as children discontinue their daytime naps without increasing their night-time sleep, which remains at about 11 hours. There is a gradual decrease in night-time sleep duration from 11 hours to 10 hours that occurs between six years of age and the beginning of puberty in adolescence (around 13 years of age) (Rosen 2006). While a clear pattern of decreasing sleep duration is evident in the first 10 years of life, changes in the amount of sleep during adolescence are more difficult to characterise (Dahl and Carskadon 1995). A reduction in sleep duration from late childhood through adolescence is widely documented (Carskadon 1990; Wolfson and Carskadon 1998; Laberge, Petit, Simard, Vitaro, Tremblay and Montplaisir 2001; Iglowstein et al. 2003); however, the weight of current evidence holds that this reduction occurs independently of adolescents' physiological sleep need (Carskadon 2011) (recommended approximately 9.25 hours (Carskadon, Orav and Dement 1983).

2.2 Normal Changes in Sleep Timing in Adolescents

The patterns of sleep and wake that emerge during adolescence are characterised by several unique features that distinguish them from those of other age groups, including preadolescents (Andrade and Menna-Barreto 2002; Carskadon and Acebo 2002). A growing body of literature based on cross-sectional (Strauch and Meier 1988; Carskadon 1990; Andrade, Benedito-Silva, Domenice, Arnhold and Menna-Barreto 1993; Manni, Ratti, Marchioni, Castelnovo, Murelli, Sartori et al. 1997; Wolfson et al. 1998; Giannotti, Cortesi and Carskadon 2002) and longitudinal (Strauch et al. 1988; Laberge et al. 2001; Thorleifsdottir, Björnsson, Benediktsdottir, Gislason and Kristbjarnarson 2002) data from the past two decades reveals several major developmental trends in adolescent sleep-wake patterns.

One of the most consistent trends identified in this body of research with respect to sleep patterns across adolescence is a marked delay in the timing of sleep (Strauch et al. 1988; Carskadon, Vieira and Acebo 1993; Carskadon, Wolfson, Acebo, Tzischinsky and Seifer 1998; Wolfson et al. 1998; Laberge et al. 2001). That is, adolescents tend to go to bed later, and sleep later than preadolescents. The sleep phase delay is particularly evident on weekends and school holidays when the timing of sleep is not restricted by the school schedule (Szymczak, Jasi ska, Pawlak and Zwierzykowska 1993; Hansen, Janssen, Schiff, Zee and Dubocovich 2005); however, a bedtime delay remains apparent on school nights (Carskadon et al. 1998). The trend for delayed sleep timing has been found in adolescents in Australia (Olds et al 2010), Brazil (Andrade et al. 1993; Andrade et al. 2002), Canada (Laberge et al. 2001), China (Chung and Cheung 2008), Germany (Loessl, Valerius, Kopasz, Hornyak,

Riemann and Voderholzer 2008), Iceland , Italy (Giannotti et al. 2002; Tonetti, Fabbri and Natale 2008), Japan (Arakawa, Taira, Tanaka, Yamakawa, Toguchi, Kadekaru et al. 2001; Tagaya, Uchiyama, Ohida, Kamei, Shibui, Ozaki et al. 2004), Korea (Yang, Kim, Patel and Lee 2005), North America (Carskadon et al. 2002), and South Africa (Reid, Maldonado and Baker 2002). The delay, which is associated with pubertal development, occurs one year earlier in females than in males, corresponding to their earlier pubertal onset (Roenneberg, Kuehnle, Pramstaller, Ricken, Havel, Guth et al. 2004).

In addition to the delayed timing of sleep, a second major trend is that the amount of sleep obtained on weekdays decreases steadily across the adolescent span (Anders, Carskadon, Dement and Harvey 1978; Wolfson et al. 1998; Iglowstein et al. 2003; Yang et al. 2005; Loessl et al. 2008; Olds, Maher, Blunden and Matricciani 2010). Corresponding with the reduction in weekday sleep times are increasingly large variations between weeknight and weekend sleep schedules during the school year (Carskadon 1990; Andrade et al. 1993; Hansen et al. 2005; Yang et al. 2005; Wing, Li, Li, Zhang and Kong 2009; Olds, Blunden, Petkov and Forchino 2010). On average, adolescents delay bedtime on weekends by one to two hours, and delay rise time between three and four hours, compared to weekdays (Crowley, Acebo and Carskadon 2007). This results in a weekend sleep extension of between one and three hours (Carskadon 1990; Andrade et al. 1993; Wolfson et al. 1998). It is possible that adolescents extend sleep on the weekend to compensate for insufficient sleep during the school week (Loessl et al. 2008).

However, there are several other intrinsic and extrinsic factors that have been identified as influencing the abovementioned patterns of sleep that emerge during adolescence and will be described in the following section.

3. Factors Affecting Adolescent Sleep-Wake Patterns
3.1 Extrinsic Factors

Psychosocial Factors

For many years, the delayed sleep phase, and reduced sleep times, observed during adolescence were attributed entirely to psychosocial factors that increase in salience during this developmental period (Carskadon, Acebo and Seifer 2001). These factors include a growing sense of autonomy, increased social opportunities, greater academic demands, extracurricular activities, participation in paid employment, and increased access to drugs and alcohol (Terman and Hocking 1913; Carskadon 1990; Carskadon 2002). Changes in parent-child relationships during this period can also result in less parental regulation of the adolescent's sleep schedules, particularly on school nights. For example, an early survey study examining the sleep patterns of 218 students at the childhood-to-adolescent transition (Carskadon, Division and sciences 1979), demonstrated a linear decline in parental influence over children's sleep patterns, with advancing age. The data showed that children aged 10 and 11 years were significantly more likely to report that parents set their school-night bedtimes (54.3%; 48.3% respectively) than were those aged 12 and 13 years (38.5% and 19.6% respectively). Interestingly, in a recent Australian study that assessed the sleep habits of 385 adolescents (aged 13-18 years), it was demonstrated that adolescents with parent-set bedtimes had earlier bedtimes, obtained more sleep, and experienced improved daytime wakefulness and less fatigue, compared to adolescents without parent-set bedtimes (Short, Gradisar, Wright, Lack, Dohnt and Carskadon 2011).

Another factor widely recognised as influencing adolescent sleep patterns, particularly later bedtimes, is the increasing use of electronic media such as the internet, television, video games, and mobile phones (Van den Bulck 2010). Time-use studies conducted in Western countries demonstrate that children and adolescents spend as much as one-quarter (approximately four hours) of their waking hours using electronic media (Mutz, Roberts and

VUUREN 1993; Olds, Ridley and Dollman 2006), which can displace the timing of sleep. For example, one study found that children with a television in their bedroom sleep around 40 minutes less each night than children without a television in their bedroom (Carskadon, Mindell and Drake 2006).

School schedules are another influential factor. Early school start times place a non-negotiable constraint on adolescents' sleep-wake schedules, and limit the time available for sleep (Carskadon et al. 1998; Hansen et al. 2005). This is particularly problematic in the United States, where it is common practise for school start times to be set earlier for older adolescents than for younger children (Wolfson et al. 1998). Studies examining the consequences of various school start times have demonstrated that earlier start times are associated with a reduction in total sleep time, earlier rise times, and excessive daytime sleepiness (Carskadon et al. 1998; Wahistrom 2002; Dexter, Bijwadia, Schilling and Applebaugh 2003). In a recent Australian longitudinal study examining the school term and holiday sleep patterns of 310 adolescents, it was found that school schedules were associated with insufficient sleep, lowered mood, and reduced overall daytime functioning (Warner, Murray and Meyer 2008). As a consequence, research is beginning to investigate the benefits of delaying school start times (Owens, Belon and Moss 2010). In the United States, Owens and associates (Owens et al. 2010) found that even a modest delay of school start time (from 8.00am to 8.30am) was associated with significant increases in mean school night duration and decreases in daytime sleepiness and depressed mood. Additionally, motivation and class attendance also increased following the intervention.

3.2 Intrinsic Factors

Biological factors

More recently, an accumulating body of evidence demonstrating the universality of the adolescent delayed sleep phase has lead researcher's to hypothesize that maturational changes in the underlying mechanisms regulating sleep-wake behaviour may also play a role in the development of adolescent sleep patterns (Carskadon 1999). According to the two-process model of sleep regulation (Borbély 1982) the timing, intensity, and duration of sleep are determined by the interaction of two independent regulatory processes: a sleep-dependent homeostatic process (Process S) and a sleep-independent circadian process (Process C).

The first regulatory process, Process S, represents the homeostatic drive for sleep, or sleep pressure, which builds cumulatively as a function of prior wakefulness and dissipates across periods of sleep (Daan, Beersma and Borbély 1984). Experimental evidence for the homeostatic process emerges from data on sleep architecture which demonstrates that the amount of slow wave sleep (SWS) and electroencephalogram (EEG) slow-wave activity (SWA) during sleep depends on the length of prior wakefulness increasing as a function of previous wakefulness and decreasing during sleep (Borbély, Baumann, Brandeis, Strauch and Lehmann 1981).

Working simultaneously with Process S is Process C, the circadian process. The circadian process is an endogenous circadian rhythm that varies the drive for sleep throughout the 24 hour day/night cycle. The circadian drive for sleep is lowest in the morning and highest towards the end of the day (Van Dongen, Rogers and Dinges 2003). After the onset of melatonin secretion (a biological marker and trigger of the circadian rhythm) this drive increases rapidly. In humans, the suprachiasmatic nuclei (SCN) of the anterior hypothalamus appear to be the site of an endogenous circadian pacemaker, or 'body clock'(Rosenthal 2006). Circadian timing is also influenced by exogenous factors, the dominant exogenous influence being environmental light (Dorrian and Dinges 2005). The retinohypothalamic tract links the retina directly to the SCN, conveying photic information that facilitates synchronization to the light-dark cycle (Rosenthal 2006). In humans, the intrinsic period of the circadian pacemaker

averages 24.2 hours (Czeisler, Duffy, Shanahan, Brown, Mitchell, Rimmer et al. 1999). According to the two process model (Borbély 1982), sleep will begin and end where the homeostatic and circadian processes intercept (see Figure 1).

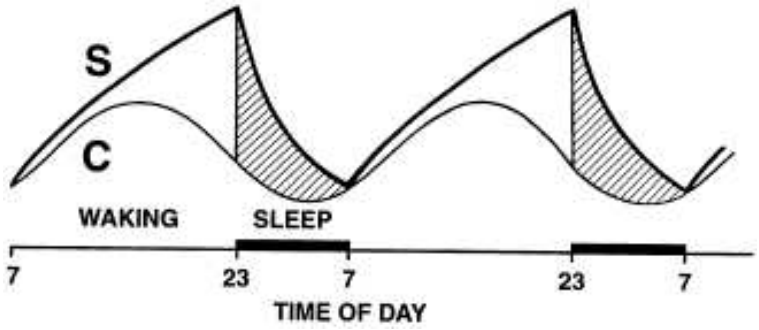

Figure 1- The two-process model of sleep regulation. Process S represents the homeostatic drive for sleep, which increases during wakefulness, and dissipates during sleep. Process C represents the circadian system which varies the drive for sleep throughout the 24 hour period. The shaded area represents the optimal time for sleep (Borbely and Achermann 1999).

Based on the two-process model of sleep, Carskadon (2008) has recently proposed a theoretical model of adolescent delayed sleep phase that incorporates developmental changes in both the homeostatic (process S) and circadian (process C) processes regulating sleep.According to this model, adolescents develop a resistance to sleep pressure that enables them to stay awake longer. At the same time, their circadian phase becomes relatively delayed, with a delay in melatonin secretion, which provides them with a drive to stay up later at night and to sleep later in the morning (Carskadon 2008).

3.2.1 Adolescent Changes in the Homeostatic Drive for Sleep

A small number of cross-sectional (Carskadon et al. 2001; Jenni, Achermann and Carskadon 2005; Taylor, Jenni, Acebo and Carskadon 2005) and longitudinal (Carskadon et al. 1983) studies have examined developmental changes in the homeostatic regulation of sleep in adolescents. For example, one study (Jenni et al. 2005) examined the effects of 36-hours of extended wakefulness on sleep and sleep homeostasis in early-pubertal children (mean age 11.9 years) and mature adolescents (14.2 years). Pubertal status was determined by a brief physical examination using the standardized Tanner assessment scales (Tanner 1962). Differences in the homeostatic drive for sleep (Process S) between the two developmental groups were modelled according to the two-process model (Borbély 1982) and were estimated on the basis of EEG slow wave activity (SWA) data, obtained with polysomnography. The findings demonstrated that the build-up of homeostatic sleep pressure (Process S) during wakefulness was slower in mature adolescents compared with early-pubertal children.

These findings are consistent with Taylor and colleagues (Taylor et al. 2005) who, using a comparable protocol, found that sleep tendency after 14.5, 16.5, and 18.5 hours awake was lower in mature compared to prepubertal children. Overall, these findings provide evidence that indicates developmental changes in the physiological regulation of sleep occur during puberty, and that these changes may contribute to differences in the timing of sleep across developmental stages. Moreover, they suggest that mature adolescents develop a sensitivity to extended wakefulness that promotes later bedtimes and may underlie the delayed sleep phase observed in adolescents (Jenni et al. 2005).

3.2.2 Adolescent Changes in the Circadian Regulation of Sleep

Several lines of evidence indicate that changes in the endogenous circadian mechanism also influence the delay of sleep timing during adolescence. In general, studies investigating the sleep patterns of adolescents have shown in different cultures the sleep phase in adolescents starts to delay/shift from the age of 12-13 years parallel to the onset of puberty(Carskadon et al. 1993; Park, Matsumoto, Seo, Shinkoda and Park 1997; Laberge et al. 2001; Gau, Shang, Merikangas, Chiu, Soong and Cheng 2007; RussoP.M., Bruni, Lucidi, Ferri and Violani 2007; Tonetti et al. 2008), which suggests a primary role of biological factors. This shift comes to an end at around 20 years of age, and it has been proposed that the end of this process could be taken as a biological marker of the end of adolescence (Roenneberg et al. 2004).

Studies of nocturnal melatonin secretion in adolescents also provide evidence indicating a relationship between pubertal development and circadian phase. In one study, Carskadon and colleagues (Carskadon, Acebo, Richardson, Tate and Seifer 1997) measured nocturnal melatonin secretion to study the parameters of circadian rhythms in adolescents. Consistent with other studies (Cavallo 1991; Cavallo 1993), they demonstrated that normal pubertal developmental was associated with a delay in nocturnal melatonin secretion. It is not clear from this research, however whether this relationship represents changes in the circadian pacemaker itself or in the environmental factors regulating the circadian phase. For example, changes in behaviour and the environment have been shown to produce long-lasting changes in circadian oscillations (e.g., periods of active melatonin secretion) that subsequently control the durations of daily rhythms, including sleep (Wehr 1991; Wehr, Moul, Barbato, Giesen, Seidel, Barker et al. 1993).

Taken together, the reviewed evidence indicates that developmental changes in both the homeostatic and intrinsic circadian mechanisms influence the regulation of sleep during adolescence. Overall, it appears that adolescents may have difficulty falling asleep early enough to obtain the optimal amount of sleep needed for their developmental age before they are forced to rise early for school.

4. How much Sleep do Adolescents Need?

4.1 Defining Sleep Need

The notion of sleep ‘need’ itself is a contested issue that has attracted much scientific debate (Van Dongen, Maislin, Mullington and Dinges 2003). In 1913, researchers Terman and Hocking argued that the key question was, ‘What is the optimal amount of sleep for physical and mental efficiency?’(Terman et al. 1913). Almost 100 years later this question remains largely unanswered. Given the complex and unique characteristics of adolescent sleep it is particularly difficult to establish an optimal amount of sleep for adolescents. It is widely reported that adolescents need around 9.25 hours sleep each night (Carskadon, Harvey, Duke, Anders, Litt and Dement 1980; Wolfson et al. 1998), however, there is debate about whether this amount is necessary or just optimal (Matricciani, Olds and Williams 2011). For example, some authors suggest that 9.25 hours is optimal for adolescents, while 8.5 hours may be considered adequate (Wolfson et al. 1998).

The recommendation of 9.25 hours was based on findings from a single classic study conducted by Carskadon and colleagues (Carskadon, Keenan and Dement 1987) at Stanford University between 1975 and 1985. As part of the longitudinal study, children, aged 10 to 17 years, returned to sleep camp each summer with the total amount of time in bed held constant each year. Sleep ‘need’ was operationally defined as the amount of sleep obtained in the sleep opportunities for each assessment (Carskadon et al. 2002). The study found that total nocturnal sleep length remained unchanged at about 9.25 hours across the adolescent span

when adolescents were provided a 10-hour sleep opportunity, and sleep was not constrained to their habitual schedule (Carskadon 1982; Carskadon et al. 2002).

Interestingly, the study further demonstrated that even when the participants obtained the same amount of nocturnal sleep as in previous summers, they showed increased objective sleepiness (measured using the multiple sleep latency test) as pubertal development advanced. What these data suggest is that daytime sleepiness increases in adolescence in the absence of marked changes in total sleep time across yearly assessments (Carskadon et al. 1983). Moreover, the findings have been supported by more recent studies which also demonstrate that objective sleepiness increases with puberty (Carskadon et al. 1998). Taken together, these findings are often cited as evidence that (a) 9.25 hours may be considered the physiological optimal sleep need duration of adolescents, and (b) the need for sleep does not decline across the adolescent span (Carskadon 2002), and may actually increase (Dahl 1999). It should be noted that the recommendations for adequate sleep in adolescents has been based on these small amount of data. However, as will be discussed below, an alternative interpretation of these findings cannot be ruled out.

Both sleep extension and increased objective sleepiness are physiological indicators of accumulated sleep debt (Monk, Buysse, Rose, Hall and Kupfer 2000; Ferrara and De Gennaro 2001; Van Dongen et al. 2003). It is, therefore, possible that sleep times remained long and objective sleepiness increased during each consecutive summer in the Stanford study (Carskadon and Dement 1987) as participants may have been carrying a residual sleep debt from nights prior to the study sessions. Although participants were asked to maintain the scheduled study bed times for 10 days prior to each session, sleep diaries generally revealed that the children, particularly the older adolescents, failed to comply (Carskadon et al. 1983). Thus, defining adolescent sleep 'need' as the amount of sleep obtained in the sleep opportunities provided (Carskadon et al. 2002) may have yielded inaccurate results or influenced the conclusions drawn. It may be more accurate to interpret adolescents sleep durations as an interaction between their basal sleep need and their accumulated sleep debt.

4.2 Interaction between Basal Sleep Need and Sleep Debt

Sleep debt can be defined as the cumulative hours of sleep loss relative to an individual's daily need for sleep (Van Dongen et al. 2003), while basal sleep need is operationalised as an individual's habitual sleep duration in the absence of pre-existing sleep debt (Van Dongen et al. 2003). At any point in time, an individual's sleep duration is determined by the interaction of these two factors, making it difficult for researcher's to ascertain an individual's optimal sleep over a limited number of experimental observations.

Several laboratory-based studies have found evidence of pre-existing sleep debt in their participants. For example, in adults, Klerman and Dijk(2005) found that when provided with 16 hour sleep opportunities, participants slept an average of 4.9 hours more than their habitual sleep duration. This amount decreased across the first three days of the study; however, individuals with habitually shorter sleep durations continued to sleep more than those with habitually longer durations. Similarly, Wehr (Wehr 1991) found that adult participants extended their sleep from 7.7 to 11 hours when provided with 14-hour nights. Sleep debt can also be measured by the propensity to fall asleep rapidly (Bonnet and Arand 1995). Klerman and Dijk (2005) found that individuals with shorter habitual sleep times fell asleep more quickly and frequently during the multiple sleep latency test, compared to those with longer habitual sleep times. These findings have been replicated in adolescent samples, even when the participants were pre-treated with 10h sleep opportunities for a week prior to experimental observation (Carskadon et al. 2001). As such, total sleep times obtained in laboratory studies where sleep opportunities are extended beyond participants' "usual" amounts may reflect

their attempts to recover a residual sleep debt (i.e., by extended their sleep) and not their physiological sleep 'need'.

4.3 Sleep Habits Surveys: What do Adolescents say about their Sleep?

Survey studies regularly report that adolescent's want more sleep than they obtain (Andrade et al. 1993). Consistent with laboratory data, the self-reported sleep need of adolescents is frequently greater than 9 hours, indicating a disparity between what adolescents say they need and what they actually obtain. In a large survey study (Wolfson et al. 1998) that examined the sleep patterns and preferences of American adolescents, approximately 87% of adolescents reported needing more sleep than they obtained on weeknights. Those who indicated needing more sleep reported an ideal sleep time of nine hours, about two hours more than they were getting. Similarly, adolescents in Germany (Mercer, Merritt and Cowell 1998) stated their subjective sleep need to be around nine hours. Several other studies have also found that adolescents frequently report their sleep time to be insufficient (Lazaratou, Dikeos, Anagnostopoulos, Sbokou and Soldatos 2005) and endorse a wish for sleep (Strauch et al. 1988). Although the actual physiological sleep need of adolescents remains unknown and may be quite changeable, the primary conclusion to be drawn from the available literature is that many adolescents do not get sufficient sleep.

5. Insufficient Sleep and its Consequences

5.1 Sleep Deprivation

Sleep deprivation is common in modern society (Bonnet and Arand 2003) and can occur in a number of ways (Dorrian et al. 2005). Sleep deprivation frequently results from not sleeping for a long enough duration to ensure normal daytime functioning without sleepiness. It can also occur when sleep is fragmented or disturbed due to an untreated sleep or health disorder or lifestyle factors. It can be acute (lasting one to two nights) or chronic (lasting multiple days, weeks, months, or years), and can occur voluntarily or involuntarily. However it occurs sleep deprivation has detrimental effects on waking neurobehavioural functions (Dorrian et al. 2005). When compared to the effects of alcohol consumption, the psychomotor impairments produced by sleep deprivation have been shown to be equivalent to those induced by alcohol consumption at or above the legal limit (Dawson and Reid 1997; Arnedt, Wilde, Munt and MacLean 2001).

Despite being more prevalent in modern society (Belenky, Wesensten, Thorne, Thomas, Sing, Redmond et al. 2003), partial sleep deprivation (PSD) has been less extensively studied than total sleep deprivation (TSD). TSD is characterised by the complete absence of sleep during a 24h period and occurs less frequently than PSD (Bonnet et al. 2003). In contrast, PSD, also referred to as sleep restriction, occurs when an individual fails to obtain their usual amount of sleep within a 24h period (Webb and Agnew 1969). The cumulative hours of sleep lost across consecutive days of PSD is commonly referred to as 'sleep debt' (Van Dongen et al. 2003). In general, studies indicate that the effects of PSD are qualitatively similar to those observed after TSD, but differ in their magnitude (Bonnet et al. 2003). Moreover, following consecutive days of chronic sleep restriction, significant cumulative, dose-dependent deficits in neurobehavioural functioning are evident (Dinges, Pack, Williams, Gillen, Powell, Ott et al. 1997; Belenky et al. 2003; Van Dongen et al. 2003), and appear to be directly related to the accumulation of sleep debt across days of sleep restriction (Van Dongen et al. 2003).
In a seminal study, Dinges and associates (Dinges et al. 1997) conducted a laboratory study where 16 healthy young adults had their sleep restricted to an average of 5 h per night (33% below participants habitual sleep duration) for seven consecutive nights. Cognitive performance was assessed using the psychomotor vigilance performance test (PVT). The results showed that subjective sleepiness and mood were elevated, while lapses in cognitive

performance and daytime sleepiness increased across days of sleep restriction. Moreover, elevated sleepiness and performance deficits continued beyond day 7 of restriction (Dinges et al. 1997).

Van Dongen and colleagues (Van Dongen et al. 2003) extended previous findings by performing a chronic sleep restriction experiment that involved randomization to a 4h, 6h, or 8h time in bed per night dose, which were maintained for 14 days. The results of this study revealed that restriction to either 4h or 6h per night over 14 consecutive days resulted in significant cumulative, dose-dependent deficits in cognitive performance on all neurobehavioural tasks relative to the 8 h sleep period. Importantly, the findings demonstrated that sleep restriction-induced deficits continued to accumulate beyond 7 nights of restriction used in other studies, (Dinges et al. 1997; Belenky et al. 2003) with performance deficits still increasing at day 14 of the restriction protocol.

Taken together, the findings of these studies demonstrate that, in adults, chronic restriction of sleep to 6h or less per night produces neurobehavioural dysfunction comparable to one to two nights of total sleep deprivation (Banks and Dinges 2007). Moreover, the findings suggest that even relatively moderate sleep restriction can impair waking neurobehavioural functions in healthy adults. Future studies in adolescent samples, are needed to establish whether these findings extrapolate to adolescents.

5.2 Sleep Deprivation in Adolescents

In contrast to the growing body of literature investigating sleep deprivationin adults, there is a paucity of well-controlled, experimental studies that have examined the specific consequences of sleep deprivation in children and adolescents. Despite this, the available data suggests that the consequences of restricted sleep in adolescents are comparable to those observed in adults, and manifest across multiple functional domains, such as behaviour, mood, and cognitive function (Fallone, Acebo, Arnedt, Seifer and Carskadon 2001).

Longitudinal data indicates that, in general, young people have difficulty adapting to the sharp declines in sleep time across adolescence (Strauch et al. 1988), and experience excessive levels of daytime sleepiness as a consequence of insufficient sleep (Fallone et al. 2001). Moreover, survey data, although limited by its self-report nature, demonstrates that inadequate sleep is associated with reduced academic performance, lower school attendance, increase risk of alcohol- and drug-related injuries (Wolfson et al. 1998; Acebo and Carskadon 2002; Fallone, Acebo, Seifer and Carskadon 2005), increased emotional problems, such as worries, anxiety and depression, lower self-esteem (Manni et al. 1997; Fredriksen, Rhodes, Reddy and Way 2004; Gau et al. 2007), and higher Body Mass Index (BMI) (Wing et al. 2009).

Although sparse, experimental studies generally support the findings of correlational studies, and go further to indicate that chronic partial sleep deprivation causes significant neurobehavioural impairments in adolescents (Beebe, Fallone, Godiwala, Flanigan, Martin, Schaffner et al. 2008). Studies have shown that one to two days of sleep restriction leads to decrements in psychomotor and cognitive performance (Randazzo, Muehlbach, Schweitzer and Walsh 1998; Curcio, Ferrara and De Gennaro 2006), dysphoric changes in positive and negative mood (Beebe et al. 2008; Talbot, McGlinchey, Kaplan, Dahl and Harvey 2010), increased subjective sleepiness and inattentive behaviours (Fallone et al. 2005; Talbot et al. 2010), and shorter daytime sleep latency (Carskadon et al. 2001). In contrast, less-complex cognitive functions, including learning, verbal creativity, memory consolidation, and sustained attention appear to be less impaired following acute sleep restriction, possibly because motivation may overcome sleepiness-related impairment in these domains (Randazzo

et al. 1998; Kopasz, Loessl, Valerius, Koenig, Matthaeas, Hornyak et al. 2010; Voderholzer, Piosczyk, Holz, Landmann, Feige, Loessl et al. 2011).

Overall, these findings demonstrate that even short-term sleep restriction has detrimental effects on the waking neurobehavioural functions of adolescents. However, as the majority of experimental studies have utilised acute (1-2 days) sleep restriction protocols, it is not clear whether the effects of partial sleep deprivation accumulate across days in adolescents, in a manner similar to that observed in adults. There is a great need for further studies utilising multi-night sleep restriction protocols to investigate the dynamics of chronic partial sleep deprivation in adolescent samples.

6. Recovery from Sleep Deprivation

In addition to the restriction of sleep across the working week, one of the most ubiquitous features of sleep-wake patterns in the modern world is the extension of sleep on the weekend (Dinges 2005). This pattern is especially typical for adolescents who sleep significantly longer on weekends, possibly to recover an accumulated school week sleep debt (Wolfson et al. 1998). Despite this, studies examining the recovery of neurobehavioural functions from chronic sleep deprivation are sparse (Belenky et al. 2003; Van Dongen et al. 2003; Banks, Van Dongen, Maislin and Dinges 2010).

In general, the available data suggests that neurobehavioural impairment resulting from accumulated sleep debt can be effectively reversed, or 'recovered' in healthy individuals whose sleep time is extended (Roehrs, Timms, Zwyghuizendoorenbos and Roth 1989; Roehrs, Shore, Papineau, Rosenthal and Roth 1996). It is assumed that acute recovery from chronic sleep restriction results from the increased sleep pressure evident in both increased slow wave energy and total sleep time (Banks et al. 2010).

Several studies investigating the recovery of neurobehavioural functioning following experimental sleep restriction or total sleep deprivation demonstrate that when recovery opportunity is greater than eight hours, impairments in neurobehavioural function recover to baseline levels after one to two days of extended sleep (Carskadon, Harvey and Dement 1981; Reynolds, Kupfer, Hoch and Stack 1986; Jay 2007; Banks et al. 2010). In contrast to these findings, other studies have found that complete recovery from sleep debt may take more than three days of extended sleep (Wehr 1991; Belenky et al. 2003).

These findings suggest that an individual who habitually restricts sleep time for five nights a week to meet academic or work demands could at least partially recover their lost sleep with one to two nights of extended sleep on the weekend (Carskadon and Dement 1981). Future research should focus on examining whether such an individual would become more vulnerable to the effects of chronic sleep restrcition over time (Carskadon et al. 1981).
Despite the widely reported variance between adolescent weekday and weekend sleep durations, and speculation that the observed weekend extension reflects an attempt to recover sleep lost during the week, studies examining the nature and dynamics of sleep recovery in adolescents are absent.

7. Summary, Conclusions and Directions for Future Research

Several conclusions can be drawn from the current review. Firstly, the unique sleep patterns that emerge during adolescence are common across cultures, and appear to reflect interacting developmental changes in the circadian and homeostatic systems (Hagenauer, Perryman, Lee and Carskadon 2009). Current evidence suggest that despite the sharp declines in sleep duration from late childhood through adolescence, the physiological sleep need of adolescents does not decline across the adolescent span (Carskadon 2011). On the basis of the available

literature, it appears that the loss of sleep throughout adolescence results from the combination of the adolescent delayed sleep phase, which promotes later bedtimes, and unchanged early school start times which restrict the amount of time available for adolescents to sleep.

It is widely accepted that healthy, adequate sleep is integral to the process of normal development throughout adolescence, however, the physiological sleep need of adolescents remains unclear. Laboratory data indicates that physiological sleep need of adolescents is 9.25h, however, it remains unclear whether this amount is necessary or just optimal and whether this guideline is valid (Matricciani, Olds et al 2011). Future research needs to take into account the interaction between basal sleep need and pre-existing sleep debt, when attempting to establish the physiological sleep need of adolescents.

Notwithstanding the sleep need debate, adolescents themselves frequently report a disparity between how much sleep they obtain and how much sleep they need, with many teenagers reporting an ideal sleep time of 9h. Additionally, although research examining the specific consequences of sleep deprivation in adolescents is in its infancy, the data reviewed suggests that even moderate levels of sleep restriction causes impairments in various functional domains, including behaviour, mood, and cognitive function. Further research is urgently needed to extend these findings.

Finally, it appears that the trend for irregular weekday and weekend sleep schedules is almost universal, and indicates that adolescents extend sleep on the weekend to recover the cumulative sleep lost as a result of chronic weekday sleep insufficiency. Adult data demonstrates the neurobehavioural impairments resulting from sleep deprivation can be reversed by extending sleep for >8h for two or more nights. It is not clear whether these findings can be extrapolated to adolescents and the lack of research comparing the effects of weekday versus weekend sleep represents a significant gap in adolescent sleep research. An understanding of chronic sleep deprivation and recovery sleep in adolescents will facilitate the development of evidence-based recommendations for habitual sleep and recovery sleep opportunities in this unique developmental group.

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