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Changes in Sleep as a Function of Adolescent Development

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Abstract

Adolescence is marked by dramatic changes in sleep. Older adolescents go to bed later, have an increased preference for evening activities, and sleep less than younger adolescents. This behavior change is driven by external factors, notably increased pressures from academic, social, and extracurricular activities and by biological circadian factors. There are also substantial changes in sleep architecture across adolescence, with dramatic declines in slow wave sleep, and slow wave activity (delta, ~ 0.5–4.5 Hz). These changes are associated with underlying changes in brain structure and organization, with a decrease in synaptic density likely underlying the reduction in high amplitude slow waveforms. While changes in sleep across adolescence are a normal part of development, many adolescents are getting insufficient sleep and are consequently, less likely to perform well at school, more likely to develop mood-related disturbances, be obese, and are at greater risk for traffic accidents, alcohol and drug abuse.

Keywords

Sleep; Adolescence; Sleepiness; Circadian

Introduction

The human lifespan is characterized by alternating periods of growth, stability and regression. These different patterns can be mapped onto domains of somatic, neural, social, behavioral and cognitive development. Arguably the postnatal time that shows the greatest confluence of differing domain development is adolescence, defined as the period between the onset of puberty and the acquisition of sexual maturity and adulthood. Of the many changes that occur during this time, sleep changes are among the more dramatic.

Sleep is a fundamental behavior ubiquitous in the animal kingdom (Siegel 2009). In classes of animals with highly developed nervous systems (mammals and birds), sleep can be

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characterized by the periodic appearance of two very different yet highly organized patterns of electroencephalographic (EEG) activity that occur in the absence of conscious wakefulness, rapid desynchronized small amplitude waveforms of rapid eye movement (REM) sleep and slow, high amplitude synchronized waveforms of deep “slow wave” non-REM (NREM) sleep. EEG activity at the scalp reflects patterns of brain activity and is thus influenced by changes in brain structure and organization. Sleep states are, however, active processes that function to support reorganization of brain circuitry (Tononi and Cirelli 2006). It should, therefore, not be surprising that adolescence, a time of dramatic change in brain structure and organization, is also a period in which sleep EEG also undergoes substantial change.

In addition to changes in EEG, many aspects of sleep behavior change in response to altered biological control mechanisms, increased societal demands for wakefulness and an incompatibility in many developed countries at least, among physiological, societal and educational demands. This tension leads to the presentation of sleep disorders not typically seen in younger children, compromised physical health and safety, increased potential for psychoactive substance use and abuse, and suboptimal cognitive function. The goal of this review is to outline the changes that occur in sleep over adolescence, placing them in the context of the hormonal, somatic and neurological development occurring at this time, and then to describe some of the psychological and other health consequences of poor or inadequate sleep in the population, a problem that is reaching epidemic proportions.

Pubertal Development

Adolescence is the transitional period spanning the second decade between childhood and adulthood, during which time social and cognitive behaviors mature (Sisk and Foster 2004). Puberty occurs within the broader scope of adolescence and refers to the activation of the previously dormant hypothalamic-pituitary-gonadal axis that culminates in gonadal maturation (Sisk and Foster 2004). This process usually begins between ages 8 and 14 years in girls and between ages 9 and 15 years in boys (Blakemore et al. 2010); the mechanisms underlying this sex difference in pubertal timing are unknown (Kauffman 2010). The onset of puberty is reflected by increased gondadotropin releasing hormone (GnRH) secretion from the hypothalamus, which activates the rest of the reproductive axis, with a consequent increase in gonadal steroid hormone production. During puberty, there is a 26-fold increase in testosterone levels in boys and a 10-fold increase in estradiol levels in girls (Ducharme and Forest 1993). There is also a dramatic increase in growth hormone (Gabriel et al. 1992). In association with these hormonal changes, there is rapid physical growth and increased height and physical changes of sexual maturation. The process of puberty is most commonly tracked by assessing the development of secondary sexual characteristics with Tanner staging (Tanner 1962), either by physical exam or self-report. (It should be noted that age and puberty development are confounded in studies of adolescence leading to difficulty in determining which variables are changed by age and which by pubertal status. See Table 1).

In addition to the physical and neurobiological changes associated with maturation, adolescence is marked by developments in psychological and social domains. During the transition from adolescence to adulthood, there are changes in emotional regulation, identity,

independence, and affiliations with peers and parents (Schulenberg et al. 2004). As they get older, adolescents also face increasing academic demands, increased exposure to extracurricular and social activities, and are more likely to have part-time jobs. Adolescence is, therefore, a period marked by dramatic biological and social changes that can affect health and behavior, including sleep.

Changes in Sleep Architecture and Homeostatic Regulation of Sleep

Sleep architecture changes across adolescence. Most notable is the marked reduction in slow wave sleep (SWS), of approximately 40% during the second decade of life (Carskadon 1982). REM sleep decreases in absolute terms, but not as a percentage of total sleep time (Ohayon et al. 2004). Quantitative analysis of the sleep EEG has provided a more detailed description of the changes in sleep across adolescence with an emphasis on changes in the characteristics of slow waves. Cross-sectional studies have reported a dramatic decrease in slow wave (delta) activity (Feinberg et al. 2006; Gaudreau et al. 2001; Jenni and Carskadon 2004), slow wave amplitude and slope of slow waves (Kurth et al. 2010a), and total slow wave counts (Coble et al. 1987) across adolescence. A recent longitudinal study of adolescents across the age range of 9 to 17 years has confirmed the decline in delta power during NREM sleep and provided further insight of the timing of maturational development in adolescence (Campbell and Feinberg 2009). Delta power is maintained between age 9 and 11 years, followed by a steep decline of about 66% beginning between age 11 and 12 years (Campbell and Feinberg 2009), which begins earlier in girls (Campbell et al. 2005). The onset of the theta decline occurs earlier, before age 6 years (Campbell and Feinberg 2009; Feinberg and Campbell 2010) (See Fig. 1). Spectral activity within higher frequency bands, including sigma and beta is also lower in older, more mature adolescents compared to prepubertal adolescents (Jenni and Carskadon 2004).

As recently reviewed by Segalowitz et al. (2010), changes in spontaneous EEG occur during wakefulness in adolescence. Total EEG power decreases reflecting a decrease in absolute power in all frequency bands but most marked in slow frequency delta activity. Similar changes occur in REM sleep (Jenni and Carskadon 2004; Tarokh and Carskadon 2010), suggesting that, in general, neural EEG-generating mechanisms change across adolescence, rather than, or in addition to changes that are specific to NREM sleep. As reviewed below, these EEG changes appear to relate to underlying changes in adolescent brain structure.

Most investigations of whether homeostatic regulation of sleep changes across adolescence have focused on measuring the rate of decline in EEG SWA across a normal night and following sleep deprivation. Gaudreau et al. (2001) conducted a cross-sectional study of small numbers of children, adolescents, young and middle-aged adults. They reported a flattening in the curve describing the early to late night decline in SWA with increasing age. Young adults (aged 19–29 years) had a relatively flat profile across the night compared to children (aged 6–10 years); adolescents (aged 14–16 years) were intermediate between the groups. The authors interpreted the data as being supportive of a decline in homeostatic sleep pressure across adolescence.

Jenni et al. (2005b) compared baseline and recovery sleep following 36 h of sleep deprivation in more mature adolescents (Tanner stage=5, mean age 14.2 years) compared with pre- or early pubertal adolescents (Tanner stage 1 or 2, mean age 11.9 years). They reported similar increases in daytime sleepiness and the same decline in sleep pressure (measured as SWA), implying no difference in sleep homeostasis between the two groups. Of interest, older adolescents had proportionally greater increase in SWA over baseline at the beginning of the night (39% increase) relative to Tanner stages 1 and 2 children (18% increase). Mathematical modeling of the build-up of sleep pressure over the sleep deprivation period indicated a slower build-up of sleep pressure in more mature adolescents (Jenni et al. 2005a). However, the modeling needs to be validated in future studies with EEG data collection during wakefulness. A second study using the same methods (and possibly the same groups of subjects) reported data from sleep latency tests conducted over the 36 h sleep deprivation period (Taylor et al. 2005). Sleep onset latencies were similar between the two groups until the normal scheduled bedtime. At 22:30, after 14.5 h of wakefulness, the younger children displayed a sudden decrease in sleep onset latency, whereas the older adolescence displayed a gradual decrease over the next 6 h, not reaching the same levels of sleepiness as the younger children until 04:30. These results support the idea of a slower buildup of sleep pressure in older adolescents.

A more recent 36 h sleep deprivation experiment compared Tanner stage 1/2 with Tanner stage 4/5 adolescents and used period amplitude analysis to evaluate the dynamics of the amplitude, incidence and slope of delta waves in recovery versus baseline sleep (Kurth et al. 2010a). Recovery sleep showed enhanced SWS, faster sleep onset latency and reduced stage1 sleep relative to baseline in both groups. Both groups demonstrated a similar increase in slow wave slope and in slow wave amplitude in the first NREM period on the recovery night and similar increases in slow wave incidence across the entire recovery night, implying no change in sleep homeostasis.

In summary, many aspects of sleep homeostasis appear unaffected in adolescence, with the possible exception of the rate of build-up of sleep pressure. Future experiments will need to be carefully designed to disentangle the roles of sleep homeostasis and circadian changes on sleep behavior.

Relations between EEG and Brain Changes during Adolescence

While much has been learned from animal studies and evaluation of human tissue postmortem, rapid expansion in the study of brain development in adolescence has followed the widespread use of in vivo magnetic resonance imaging (MRI) methodologies. Most work has been done using structural imaging, which permits the segmentation of the brain into three broad classes of tissue, gray matter, white matter and cerebrospinal fluid, based on the differing water content and thus hydrogen proton signal strength. White matter is thought to comprise the myelinated axons of neurons throughout the brain. Gray matter has a more heterogeneous composition and comprises cell bodies, dendrites, associated neuropil and vasculature and unmeylineated axons. Recent studies have focused on diffusion tensor imaging, a method that permits evaluation of the microstructural integrity of white matter tracts. Several recent reviews cover structural MRI and diffusion tensor imaging (DTI)

changes in adolescence (Bava and Tapert 2010; Giedd et al. 2010; Paus 2010; Schmithorst and Yuan 2010) and more generally from gestation to adulthood (Stiles and Jernigan 2010).

The adolescent brain undergoes dramatic maturational changes, which may partly be influenced by underlying pubertal hormone changes (De Bellis et al. 2001; Giedd 2004). For example, studies in animal models, such as the Syrian hamster, have indicated that pubertal steroid hormones influence development of the adolescent brain by organizing and activating neural circuits as they do during the perinatal period (Schulz et al. 2009). Sex steroids influence neurogenesis, synaptic organization, receptor expression, and neurite outgrowth (Peper et al. 2009; Romeo et al. 2004; Schulz and Sisk 2006). Gray matter development in girls across puberty has been shown to be directly associated with increased levels of estradiol (Peper et al. 2009).

Cross-sectional studies have indicated that cortical gray matter volume shows a curvinelar developmental trajectory with growth in childhood, followed by a rapid decline in adolescence (Giedd 2004; Jernigan et al. 1991; Sowell et al. 2002; Steen et al. 1997) that stabilizes into a more gradual decline across the remainder of the adult lifespan (Pfefferbaum et al. 1994), although there is substantial inter-subject variability (Giedd et al. 2010). Longitudinal studies have found that there are regionally specific nonlinear prepubertal increases, followed by post-pubertal decreases in cortical gray matter (Giedd et al. 1999a; Gogtay et al. 2004). The decreases in cortical gray matter volume begin in the more dorsal parietal cortices, and then spread rostrally over the frontal cortex, until the dorsolateral prefrontal cortex, which loses gray matter volume only at the end of adolescence (Gogtay et al. 2004). The decrease in gray matter across adolescence likely reflects, in part, the process of synaptic pruning (Feinberg and Campbell 2010; Giedd 2004), the term used to describe the reduction in synaptic density during childhood and adolescence (Huttenlocher 1979; Seeman 1999).

Adolescence is also associated with ongoing myelination of neuronal axons (Yakovlev and Lecours 1967), with a consequent increase in white matter (Courchesne et al. 2000; Giedd et al. 1999a; Pfefferbaum, et al. 1994; Reiss et al. 1996; Sowell et al. 2004). Myelination, which continues into adulthood, progresses from inferior to superior brain regions, and from posterior to anterior (Yakovlev and Lecours 1967). The corpus callosum, the great band of white matter tracts connecting the cerebral hemispheres, undergoes significant changes, with the anterior sections reaching adult size sooner than posterior sections (Giedd et al. 1999b; Thompson et al. 2000).

DTI studies indicate increases in myelination and axonal thickness in areas such as the prefrontal regions, internal capsule, and corpus callosum with increasing age across childhood and adolescence (Barnea-Goraly et al. 2005). Nagy et al. (2004) found that fractional anisotropy (FA), an index of orientation and coherence of fiber tracts (Le Bihan 2003; Pfefferbaum and Sullivan 2010; Sullivan and Pfefferbaum 2010) of both the frontal and temporal regions correlated with age in both boys and girls, and that FA in these regions correlated positively with the development of working memory capacity and reading ability. Recent, two-point longitudinal data from older adolescents (Bava & Tapert 2010) showed increased FA in a number of right hemisphere tracts and decreased mean diffusivity in both

hemispheres. Mean diffusivity provides an index of the magnitude of freely diffusing water molecules, without respect to orientation, and reflects the presence of fluid in tissue (for review see (Chanraud et al. 2010)). The general pattern of DTI data in adolescents indicates that mean diffusivity changes are largely due to decreases in radial diffusivity (Schmithorst & Yuan) that is thought to reflect myelin integrity (Song et al. 2002; Sun et al. 2006). There is limited evidence for some interesting sex differences, with anisotropy changes in girls seeming to relate to increased thickness of the myelin sheath (Perrin et al. 2009) and those in boys to increased axonal diameter (Perrin et al. 2008).

Aspects of brain development, such as neuronal connectivity and receptor density are not captured by gross structural MRI measures (Giedd, et al. 2010). EEG provides a potential functional correlate of changes in synaptic density, because the EEG signal is produced largely by summed inhibitory and excitatory post-synaptic potentials (Lopes Da Silva and van Rotterdam 2005). In this context, the decline in SWS during adolescence is hypothesized to be a normal, programmed maturational event that represents underlying dendritic pruning essential for normal brain development (Feinberg et al. 1990). The sleep EEG, therefore, reflects changes in the brain and is a useful tool for the study of brain development.

Feinberg and Campbell (2010) provide a detailed review of the evidence supporting relations between changes in synaptic density, cerebral metabolic rate and delta EEG during adolescence, relations first proposed by Feinberg in the 1970's (Feinberg 1977) and elegantly supported in the most extensive longitudinal study of adolescent sleep ever undertaken (Campbell et al. 2005; Campbell and Feinberg 2009; Campbell et al. 2007; Feinberg et al. 2010 ; Feinberg, et al. 2006). To summarize the results thus far, delta activity begins declining at age 11 or 12 and shows a 66% decline by age 17 years, with preliminary evidence indicating some flattening of the slope of decline at 17. Theta activity declines earlier than age 12, and their ongoing study of a cohort started at age 6 will determine when its decline commences.

In their review, Feinberg and Campbell point to two hypotheses for the decline in theta activity occurring earlier than the decline in delta activity. The first hypothesis proposes that the later decline in delta is associated with later pruning in frontal cortex (Giedd, et al. 2010), the putative generator focus for delta activity (Dang-Vu et al. 2005; Kurth et al. 2010b). The second hypothesis is that the earlier theta decline is consistent with allocortical cortical (phylogenetically older tissue with fewer than 6 layers) generation, with all cortical areas showing earlier development than those of necrotic (Shaw et al. 2008). They also highlight that the reduction in power has to be due to changes in excess of those in gray matter volume or cortical thickness given that the > 60% change in EEG is occurring at the same time as an approximate 10% decline in cortical thickness (Shaw et al. 2008).

Only one study has reported relations between differences in slow wave EEG power during wakefulness and differences in gray matter volume from MRI in a cross sectional sample of 10–30 year-olds (Whitford et al. 2007). Interestingly, the slow wave power was a better predictor of age than gray matter volume. A related paper from the same group (Boord et al. 2007) reported strong correlations between slow wave EEG power and estimated cerebral

metabolic rate in a sample covering a wide age range (6 to 85 years). These authors present compelling data showing similar patterns of rapid change in delta and theta EEG power and basal metabolic rate normalized for body mass, across the adolescent age range. Most recently, Buchmann et al. (2010) evaluated 3 T structural MRI analyzed with a combination of voxel based morphometry, cortical surface analysis, and sleep EEG in 36 children and adolescents, aged 8–19 years. They reported the expected decrease in slow frequency power with age and the expected decrease in gray-to-white matter ratio from whole brain analysis. There was a significant correlation between SWA at C4 (a centrally located electrode. See Fig. 2) and the whole brain gray matter volume:white matter volume ratio and with gray matter volume in a number of cortical lobes and regions, but not with subcortical structures. There were also positive correlations found between SWA and cortical thickness.

As indicated above, maturational brain changes are regionally-specific, with maturation generally progressing in a posterior-to-anterior manner, with primary sensory and motor areas developing earlier than association areas (Shaw et al. 2008). There may, therefore, be associated regional differences in the decline in EEG power across adolescence. Most studies of the sleep EEG in adolescents have analyzed EEG taken from a single central electrode site (typically C3 or C4. See Fig. 2). As far as we are aware only four studies have investigated regional changes in the sleep EEG during adolescence. Jenni and colleagues (Jenni et al. 2005b) reported that the decrease in delta power in NREM sleep in mature compared with pre-pubertal adolescents was equally evident at both frontal (Fz/Cz) and posterior (Pz/Oz) regions suggesting that maturational changes in the sleep EEG are widespread and, occur independent of derivation. Recently, another study reported that the decline in delta power over a 2-year period of adolescence was most pronounced in the left central and right occipital region, suggesting an asymmetrical decline (Tarokh and Carskadon 2010). Feinberg et al. (2010) reported longitudinal data recorded every 6 months over a periods of 6 years in children aged 6–18 years. They found that delta power declined earliest at an occipital site (O1) and latest at a frontal site (Fz), a pattern consistent with dendritic pruning patterns evidence in brain development (Shaw et al. 2008). In the most extensive study of EEG topographic changes, Kurth et al. (2010a) used 128 channel EEG recordings subjected to power spectral analysis to evaluate sleep EEG in 53 subjects aged 2.4–19.4 years. In the youngest two age groups (2–5 years and 5–8 years); SWA was seen predominantly over occipital regions, with anterior movement of the topography with increasing age. The 17–20 year-olds showed a frontal maximum for SWA similar to that previously reported for adults (Finelli et al. 2001; Massimini et al. 2004) (See Fig. 3). Source localization showed movement of the SWA source from the lingual gyrus in the younger children to the medial frontal gyrus in the oldest group (Kurth Ringli et al. 2010).

In summary, the decline in delta power appears to follow the same time course and the same regionally-dependent pattern of change as the decline in gray matter volume across adolescence strongly suggesting that these two events are related. Longitudinal studies of adolescents with EEG and MRI data collected at coincident time points should provide direct support for this relationship.

Changes in Sleep Behavior

Sleep habits changing with adolescence is not a new phenomenon, nor is it only recently that psychologists have become interested in it and its relationship to life functioning. Stanford Psychologist Lewis Terman, author of the Stanford-Binet intelligence test, wrote about the phenomenon in 1913, in volume four of the *Journal of Educational Psychology.* He and his co-author Adeline Hocking, described a change from “vesperal” to “matinal” (i.e., evening to morning) sleep patterns in 2692 children and adolescents aged 6 to 20 years, (Terman and Hocking 1913). They also showed an age-related decline in time in bed and an increase in the need to be awakened by a parent in the morning (21% of 6 year-olds rising to 58% of 18 year-olds) (Terman and Hocking 1913).

Much of the modern literature has focused on the issue of school start times as a contribution to sleep restriction in adolescence; however, the shift to later bedtimes with increasing age is evident even in countries with later school start times and even when adolescents are on vacation, unconstrained by school start-times (Crowley et al. 2007; Olds et al. 2010b; Russo et al. 2007; Szymczak et al. 1993). Older adolescents are more likely to wake-up later when unconstrained by school start-times such as on weekends or vacations (Andrade et al. 1993; Crowley et al. 2007; Russo et al. 2007). A longitudinal questionnaire-based study of over 1000 boys and girls, followed from age 10–13 years old confirmed these findings, showing that school-day and weekend bed times and weekend wake time were significantly delayed every year in both boys and girls (Laberge et al. 2001). Recently, Sadeh and colleagues (2009) used actigraphy (estimating periods of wakefulness and sleep using body movement patterns) to monitor changes in sleep in a cohort of adolescents (aged 9.9 to 11.2 years at first assessment) every year over a 3-year period. They confirmed subjective data, showing a significant delay in sleep onset time with age (Sadeh et al. 2009).

In association with a shift in timing of sleep, adolescents show a shift in chronotype towards eveningness, with a preference for evening activities (‘owls’). This shift occurs around the age of 13 years and is evident in different cultures (Carskadon et al. 1993; Diaz-Morales et al. 2007; Randler 2008; Yang et al. 2005) and appears to be independent of environmental factors (Roenneberg et al. 2004). Adolescents reach the maximum in their eveningness tendency at around the age of 20 years, with young women reaching their maximum significantly earlier (19.5 years) than young men (20.9 years) (Roenneberg, et al. 2004). After the age of 20 years, the tendency for morningness gradually increases leading to the proposal that this shift in chronotype serves as a marker for the end of adolescence (Roenneberg et al. 2004). It should be noted however, that most of the data on chronotype is based on self-reported preference without biological validation.

In addition to going to bed later, older adolescents sleep less than younger adolescents. A recent meta-analysis of data from 20 countries showed that sleep duration on a school night declined by, on average, 14 min per year of age across adolescence, with the rate of decline being slightly greater for girls than boys (Olds et al. 2010a). The decline in sleep duration across adolescence was less steep on non-school nights, declining at a rate of 6.6 (boys) and 8.0 (girls) minutes per year (Olds et al. 2010a). Overall, girls slept more than boys (11 min more per night on school days and 29 min more per night on weekends). Fewer studies have

investigated changes in sleep duration across adolescence during vacations when wake up times are not dictated by school start times and when sleep duration across days of the week is presumably more stable. However, the trend for declining sleep appears to be the same. A longitudinal analysis of Swiss adolescents during vacations over a period of 6 years showed that time in bed gradually declined from about 11 h at age 11 years to about 9 ½ hours at age 21 years (Strauch and Meier 1988).

Although the decline in sleep duration over time is apparent in adolescents from many different parts of the world, there are massive differences in sleep duration between different geographic regions as highlighted in a recent meta-analysis (Olds et al. 2010a) (see Fig. 4). Adolescents in Asian countries report getting less sleep than those from the U.S. who, in turn, report getting less sleep than those from Australia and Europe (Olds et al. 2010a). Most studies of adolescent sleep behavior have been conducted in the United States which has early school start times for high schools, with consequent limitation of school week-night sleep times. U.S. data from the CDC Youth Risk behavior Survey conducted in 2007 in 14,000 9–12th grade students in U.S. public and private schools have recently been published (Eaton et al. 2010). Sixty nine per cent of surveyed students averaged less than 7 h on school nights, with only 8% sleeping 9 h or more. The prevalence of<7 h sleep was higher in girls (71.3%) than boys (66.6%) and higher with increasing grade level (9th grade=58%, 12th grade=78%). These data are consistent with those of Noland et al. (Noland et al. 2009) who reported that 92% of the 9th to 12th graders slept less than 9 h on school nights. Data from Canada show similar results, with 70% of high school students obtaining fewer than 8.5 h of sleep on school nights (Gibson et al. 2006). Problems start before high school. A study of 6th to 8th graders (Drake et al. 2003) showed sleepiness (measured using a pediatric daytime sleepiness questionnaire) increasing across the three grades with only 24% of 8th grade students having more than 8 h sleep on school nights.

In contrast to adolescents in the U.S., Australian adolescents aged 16–17 years old have an average school night sleep duration of around 9 h (Olds et al. 2010b). It should be highlighted that school start times of around 9:00 AM in Australia are later than in the USA, allowing for additional sleep time during the school week. Data from Italian 17 year-olds (n=869) show nearly two thirds obtaining at least 7 h sleep per night and only 8% obtaining fewer than 6 h. In a useful cultural adaptation, 35% also usually had afternoon naps (Manni et al. 1997). Greek high school students show an even higher prevalence of napping (60%,) and obtain an average of nearly 8 h sleep over a 24 h period (Lazaratou et al. 2005).

As indicated by Olds et al. (2010a), the situation for sleep may be worst for Asian adolescents. Yang and colleagues (2005) surveyed nearly 1500 Korean students in grades 5 through 12 (mean age 13.7 +/− 2.4 years). 10th grade students slept 6.02 h on average, 11th grade students 5.62 h and 12th grade students only 4.86 h on average on school nights. These results were partly due to earlier school start times in the later grades (7:00–7:40 versus 8:30–8:40), but were also related to the high percentage of students attending additional night classes. By 11th and 12th grades 55% of the sample was attending night classes that ended after midnight. Not surprisingly, one quarter of the older students reported falling asleep in regular classes at least four times per week. As pointed out by the authors

(Yang et al. 2005), Korean adolescents are severely sleep deprived, which has consequences for mood, school performance, and health.

As is evident from the above studies, school start times are a major restricting factor on wake-up times. With adolescents going to bed later but still having to get up at a set time for school, their time in bed is reduced compared with younger children. As a result, adolescents accumulate a sleep debt during the school week (Dinges et al. 1997) which they attempt to recover on weekend nights when not restricted by set wake-up times: Adolescents have upwards of 2 h more sleep on a weekend night than on a weekday night (Andrade et al. 1993; Gau and Soong 2003; Giannotti et al. 2002; Loessl et al. 2008; Reid et al. 2002; Saarenpaa-Heikkila et al. 1995; Urner et al. 2009; Wolfson and Carskadon 1998).

It is interesting to provide an historical perspective on the influence of school start times on sleep. In 1913, Terman and Hocking (1913) reported that sleep in adolescents in the western U.S. was longer than that previously reported in studies of English (n=6180) (Ravenhill 1910) or German (Bernhard 1908) children and adolescents. One of the factors that they felt explained this difference was that school start times were an hour later (9:00 AM) in the U.S. than those in Germany and England (7:00–8:00 AM). They go so far as to state, “The American practice of beginning at 9 o’clock is far wiser, and should never be changed unless for very special reasons” pp271. (See Fig. 4)

Social and environmental factors have a major influence on sleep behavior in adolescents. Older adolescents face increasing pressures to their sleep time from academic, social, entertainment, and extracurricular activities (Carskadon 1989-1990; Crowley and Carskadon 2010; Gau and Soong 2003; Matsumoto et al. 1975; Yang et al. 2005), and there is less parental monitoring of bedtimes than in younger children (Russo et al. 2007; Wolfson and Carskadon 1998). These factors may partly drive a shift in sleep timing. Also, as described above, school schedules as well as cultural behaviors such as napping are major determinants of sleep duration and timing in adolescents. Changes in biological factors during adolescence also play a role: there are underlying changes in both circadian and homeostatic components of sleep that may contribute to the delayed sleep onset across adolescence (See Crowley et al. (2007), and Hagenauer et al. (2009) for detailed reviews).

Changes in Circadian Regulation of Sleep

To identify factors that underlie the tendency for adolescents to shift to a later bedtime and more eveningness as they get older, Carskadon and colleagues have conducted a number of carefully designed laboratory studies to evaluate ‘unmasked’ circadian rhythms in adolescents. They found that more mature, older adolescents had a later phase of melatonin-secretion offset than less mature, younger adolescents. The study involved assessment under highly controlled conditions of a constant routine protocol in which subjects remained in bed under dim lighting conditions (<20 lx), received small meals and fluid every 2 h for a 34 h period of wakefulness (Carskadon et al. 1997). Thus circadian rhythms were assessed without the normal confounding effects of sleep, daylight exposure, food metabolism and physical activity. The conclusion from the study is that the later bedtime of older adolescents, is accompanied by a later intrinsic circadian phase.

To investigate the hypothesis that the shift in circadian phase during adolescence may be due to a longer intrinsic period, Carskadon and colleagues conducted a challenging forced-desynchrony protocol—the only such study in adolescents to date (Carskadon et al. 1999). Subjects live in a controlled environment where lights are switched on and off to provide a 28 h day, with 16.33 h in the light period and 11.67 h in the dark period. As with the constant routine methodology described above, the 28 h day schedule permits assessment of circadian rhythmicity independent of the normal 24 h external cues. Carskadon and colleagues successfully completed the protocol in 10 adolescents (mean age of 13.7 years). Circadian period, based on melatonin onset and offset was 24.33 ±0.21 h and 24.35±0.21 h, respectively, which is within the range reported for young and older adults recorded in a similar 28-h dim-light forced desynchrony protocol (Range: 23.924.5, with 90% of the sample between 24.00 and 24.35 h (Czeisler et al. 1999), although three adolescents had periods outside the range reported for adults (Carskadon et al. 1999). There was no correlation of period with pubertal status in this small sample. From this study it cannot be confirmed or refuted whether intrinsic circadian period becomes longer across development, which may drive the phase shift in sleep timing; only a longitudinal study could answer that question.

Another potential reason for delayed sleep timing in adolescents is altered sensitivity of the circadian pacemaker to light (Crowley et al. 2007). Increased sensitivity to light exposure in the evening, for example, could cause a circadian phase delay. However, preliminary findings from the Carskadon laboratory, published in abstract form, do not support this hypothesis. Evening (23:00–24:00) and early morning (03:00–04:00) light of 150 and 500 lx suppressed melatonin responses equally, indicating similar sensitivity to light, in pre/early pubertal and mid/post pubertal adolescents (Carskadon et al. 2002). Given the preliminary nature of these findings, further research is needed to investigate responses to light in adolescents.

Changes in homeostatic factors across adolescence may contribute to a delay in sleep timing: Jenni and colleagues (Jenni et al. 2005a) found that the build-up of homeostatic sleep pressure is slower in more mature adolescents (Tanner stage=5, mean age 14.2 years) compared with pre- or early pubertal adolescents (Tanner stage 1 or 2, mean age 11.9 years). With a slower build up of sleep pressure, there would be a slower build up of sleep need during wake periods, such that more mature adolescents could stay awake later. A recent model developed to examine how homeostatic and circadian systems affect chronotype (eveningness/morningness) found that the interaction between both processes is critical to understanding chronotype (Philips et al. 2010). It is, therefore, feasible that changes in circadian and homeostatic sleep regulatory processes underlie the developmental shift in sleep timing seen across adolescence.

In summary, older, more mature adolescents have a phase delayed circadian rhythm compared with younger, less mature adolescents in association with their later bedtimes. However, whether this phase delay is a consequence of changes in the period of the biological clock, or of sensitivity of the clock to light, or of homeostatic sleep regulatory processes, or an interaction between these factors remains to be confirmed.

Consequences of Poor or Inadequate Sleep in Adolescence

While changes in sleep across adolescence may be considered a normal part of development, many adolescents accumulate a significant sleep debt particularly during the school week, and are consequently sleepy during the day. Sleepiness increases linearly as total sleep time declines and greater sleepiness is associated with poorer school achievement, less sense of "feeling good," frequent illness, increased anger, and lower levels of school enjoyment (Drake et al. 2003).

Mental Health, Stress, and Worry

Of major concern is the impact of the accumulated sleep debt and associated daytime sleepiness on mental health and well-being. An experimental study of sleep restriction in young adolescents (girls 10–12, boys 11–13), midadolescents (girls 13–15, boys 14–16), and adults (30–60) compared mild sleep restriction at home (6.5 h), extreme sleep restriction in a laboratory (2 h), and two recovery nights (8.5 h) at home (Talbot et al. 2010). All groups had less positive affect when sleep deprived but no significant increase in negative effect. However, younger adolescents had a larger impact of sleep deprivation on worry, with their most worrisome item being viewed as significantly more threatening when sleep deprived. The authors interpreted this finding as being indicative of an increased vulnerability to elevated anxiety in the younger adolescent group when sleep deprived. Also, high school students with lower sleep times (7.5 h) have higher levels of perceived stress than students with greater sleep times (9.7 h) (Noland et al. 2009). The data from another study (Roberts et al. 2001) highlight the correlates of insomnia in an adolescent population, with insomnia showing significant predictive odds ratios for mood disturbance, suicidal ideation, unhappiness, interpersonal problems, poor perceived health, school absence due to illness, and fatigue. Of those with insomnia, 42% reported at least three of the above problems with life functioning, with an odds ratio of 6.3 for reporting more than six problems. Also concerning, are recent findings showing that depression and suicidal ideation are associated with short sleep duration and later parent-set bedtimes in American adolescents (Gangwisch et al. 2010). In this study of over 15,000 adolescents in grades 7 to 12, those with bedtimes set by parents of midnight or later were 24% more likely to suffer from depression (OR=1.24, 95% CI 1.04–1.49) and 20% more likely to have suicidal ideation (1.20, 1.01–1.41) than adolescents with parental set bedtimes of 10:00 PM or earlier.

School Performance

Restricted sleep and elevated levels of sleepiness should logically lead to problems with school performance. Research in this area is plagued, however, with a number of difficulties in determining the proportion of variance of grades explained by sleepiness as opposed to differences in student ability, parental interaction, socio-economic status, teacher ability, and the other multifactorial contributors to educational performance. Larger scale surveys often rely on self-report data for both sleep behavior and school performance. Even when data are available from schools or teachers, differences between schools in how grades are allocated and grade point averages calculated make comparisons challenging. A final issue is that if the majority of students are sleep deprived, then the grading distribution is largely based on sleepy students. Nonetheless, there is substantial indirect evidence for relationships between

sleep and academic functioning at school. Students (from a sample of 3000) with higher grades reported more total sleep, earlier school night bed-times, and a lower difference between school night and weekend bedtimes (Wolfson and Carskadon 1998). Poor sleepers were nearly twice as likely to fail a year of school than good sleepers (21% vs. 11%) (Kahn et al. 1989). College students with more regular sleep habits (Gray and Watson 2002; Trockel et al. 2000) and longer sleep (Kelly et al. 2001) have also been shown to have better grade point averages. In an attempt to quantify sleep quality/school performance relationships a meta-analysis was conducted of 17 studies based largely on self-report measures (Dewald et al. 2010). Significant relationships were found for sleep quality and sleep duration although the effect sizes were small (r=0.096 for sleep quality and r=0.069 for sleep duration). As indicated by Curcio and colleagues (2006) in their review, there is sufficient evidence to conclude that both cognitive function and school behavior deteriorate in association with poor sleep, and that this may well relate to a disturbance in the optimal relationship between sleep and memory consolidation (Walker 2008).

Weight Gain and Obesity

Several recent studies in younger children and adults have highlighted a link between short sleep and increased body mass index (BMI), with the underlying mechanisms of the relationship thought to relate to decreased glucose tolerance, decreased insulin sensitivity, decreased levels of leptin (a hormone underlying satiety), increased nocturnal cortisol levels and increased secretion of ghrelin (a hormone responsible for hunger) and subsequent increased hunger and appetite (Knutson et al. 2007; Knutson and Van Cauter 2008; Leproult and Van Cauter 2010; Spiegel et al. 2005, 2009; 2010; Van Cauter and Knutson 2008; Van Cauter et al. 2008). The data on the relationship between short sleep and obesity in adolescents are sparse. Some studies show adolescents to have a similar relationship to that reported in younger children and in adults. For example, a survey of 384 9th-12th graders found that those with a BMI in the healthy range slept on average 30 min more per school night (7:34±1.2 h) than those with a BMI in the overweight range (7:03±1.23 h) (Noland et al. 2009).

In a sample of over 4000 adolescents, Knutson (Knutson 2005) used logistic regression to show a relationship between mean self-reported sleep duration and being overweight in boys (OR 0.9; 95% CI: 0.82–1.00) with a BMI Z-score increase of 0.8 units per hour of sleep reduction, however, no significant relationship was reported in girls. This sex difference was reported in a sample of over 6000 Australian children and adolescents (Eisenmann et al. 2006), with reduced sleep predicting elevated BMI in boys but not girls. Again using logistic regression 14 to 16.5 year-old boys with less than 8 h of sleep had an odds ratio of 4.85 relative to those who slept more than 10 h. Although another recent large scale Australian survey of 5 to 15 year-olds also found that the odds ratio for sleep hours predicting obesity was significant in boys but not girls in the whole sample, the effect seemed to be driven more by the younger children with no differences in BMI levels in longer (> 10 h) versus shorter (< 9 h) sleepers for the 13–15 year olds surveyed (Olds et al. 2010b). It is likely, however, that the >10 h of sleep per night comparison point while sensible for the younger children was too long for the older adolescents, and no differentiation was made in the survey between weekday and weekend sleep.

Experimental studies have smaller samples sizes than the larger scale epidemiology surveys listed above, but have the advantage of more accurate sleep assessment with diaries, actigraphy (measurement of daily activity) or even polysomnography. A recent study (Weiss et al. 2010) investigated 240 older adolescents (mean age 17.7 years) using 3 days of actigraphy and a 3-day, 24-hour food diary to evaluate relationships between sleep, activity levels, BMI and food intake. The prevalence of short sleep (< 8 h) was significantly higher for obese (80%) than non-obese (63%) adolescents, with short sleep being more prevalent in boys than girls (77% vs. 57%). Short sleep was associated with higher proportion of daily calories from fat (+2.2%) and lower proportion of carbohydrates (−3.0%), but contrary to what would be expected from the survey data (Eisenmann et al. 2006; Knutson 2005; Noland et al. 2009), this association was significant only in girls not boys. Gupta and colleagues (Gupta et al. 2002) evaluated actigraphy data from 383, 11–16 year-olds relative to measured percentage body fat and BMI levels. The data indicated an 80% increase in the odds of obesity occurring with each hour of reduced sleep, with total sleep time predicting a higher prevalence of obesity in both boys (r=−0.36) and girls (r=−0.29). Increased BMI puts adolescents at risk for the early development of health problems. While many factors contribute to obesity, ensuring adequate sleep duration may be one factor that if addressed, could help manage body weight problems in adolescents.

Alcohol and Substance Use/Abuse

Insufficient or poor quality sleep is thought to be a risk factor for alcohol, tobacco and other substance use in adolescents. A survey of mid west U.S. high school students found 3% using alcohol, 6% smoking in the evenings and 6% using sleeping pills specifically to aid poor sleep (Noland et al. 2009). Data from 4175 Texan middle and high school students showed 20% of students sleeping fewer than 6 h on school nights, with this short sleep significantly predicting drug use (OR 1.71, 95%CI: 1.20–2.44) (Roberts et al. 2009). Short sleep on both weeknights and weekends had a similar prediction (OR 1.66, 95%CI: 1.02–2.7). Poor sleeping Italian adolescents were found to be more likely to smoke, drink coffee and drink alcohol than good sleepers (Manni et al. 1997). Structural equation modeling of data from over 4000 Finnish adolescents (Tynjala et al. 1997) revealed that in 15 year old boys, perceived tiredness and poor sleep habits accounted for 26% of the variance in psychoactive substance use, with less variance (12%) being explained by the factors in girls.

Drowsy Driving

Most drivers acquire an independent license during adolescence, and, thus, for many during a period of chronic sleep restriction and elevated levels of sleepiness. A recent national survey indicated that greater than 62% of 11th grade students and 68% of 12th grade students admitting to driving while sleepy in the previous year with 15% of those surveyed admitting to it as a regular behavior (Carskadon et al. 2006.). The Department of Transportation estimates around 100,000 motor vehicle accidents per year in the U.S. have drowsy driving as the primary cause, and there are data to suggest that over half of these involved drivers under the age of 25 (Pack et al. 1995). A similar pattern was reported in Italian teenagers, with 40% of those surveyed reporting sleepiness while driving, and 15% of those who have had a crash considering sleepiness to be the main cause. A growing body of evidence from simulator studies confirms the original findings of Dawson and Reid (1997)

that sleep restriction leads to levels of driving impairment analogous to those seen in illegal blood alcohol levels with 19 h of wakefulness equivalent to a blood alcohol level of 0.05 and 22 h equivalent to a level of 0.08 (Arnedt et al. 2001). As reviewed by Orzel-Gryglewska (2010), chronic sleep restriction also leads to driving impairment commensurate with that produced by alcohol.

It is thus logical to assume that permitting adolescents more time for sleep and reducing their levels of sleepiness should lead to a reduction in teen motor vehicle accidents. This hypothesis was supported in a landmark study by Danner and Phillips (2008), who evaluated data from a single school district county in Kentucky with over 10,000 students that moved high school start times from 7:30 to 8:30 AM. The percentage of students reporting at least 8 h of sleep per school night increased from 35% to 50%, with high school seniors obtaining 30 more minutes sleep on average. The pre-post comparison of Department of Motor Vehicles data for 2 years on either side of the change showed a decrease in teen motor vehicle crashes of 16.5% for the county with delayed start times as compared to a 7.8% increase for the rest of the state. Later start times were associated with more sleep, less sleepiness, and fewer accidents.

Sleep Disorders in Adolescence

Associated with the changes in sleep across adolescence, there are changes in the prevalence of sleep disorders, with sleep disorders common in childhood, such as parasomnias decreasing, and adulthood sleep disorders like insomnia increasing in prevalence during adolescence.

Parasomnias are disorders characterized by abnormal behavioral or physiological events such as sleepwalking, sleep terrors, enuresis, and bruxism (tooth grinding) that occur in association with sleep, specific sleep stages, or sleep-wake transitions. The prevalence of parasomnias peaks in childhood and declines steadily into adolescence (Bloomfield and Shatkin 2009; Laberge et al. 2000). Sleep walking is reported in about 15% of children, with a peak prevalence between ages 4 and 8 (Bloomfield and Shatkin 2009; Laberge et al. 2000). In most cases, sleepwalking has ceased before the age of 10 years; however, longitudinal data indicated that 24% of children who were sleepwalking at age 11 were still sleepwalking at age 13 (Laberge et al. 2000). Similarly, the prevalence of sleep terrors and bruxism declines across childhood and early adolescence (Bloomfield and Shatkin 2009). Recent data show that reports of sleep bruxism increase again later in adolescence, from the age of 15 to 23 years old (Strausz et al. 2010). Prevalence of nocturnal enuresis (bedwetting) declines by approximately 15% for each year after age 5 (Bloomfield and Shatkin 2009) such that 88% of children with enuresis have outgrown the condition by age 13 years (Laberge et al. 2000).

The loss of SWS and its associated sleep protective delta activity leads to increased levels of sleep disturbance and the appearance of insomnia symptoms. For example, a sample of over 700 French high school students revealed that 40% reported at least one of the following symptoms: trouble falling asleep, early awakenings, need for more sleep, bad sleep quality, or sleeping pill use. There was a higher prevalence of all symptoms in girls relative to boys, other than pill use (Vignau et al. 1997). A study of Italian 17 year-olds found 19% of girls

and 12% of boys classified as poor sleepers (Manni et al. 1997). This sex difference is also present in Greek high school students, with 61.5% of girls and 50% of boys endorsing at least one of five items on an insomnia scale, with the most common problems being delayed sleep onset (29%) and reduced total sleep duration (33%) relative to what was perceived as needed (Lazaratou et al. 2005).

It is possible that chronic levels of sleep restriction in U.S. adolescents provide some paradoxical “protection” from insomnia, by keeping sleep pressure at a high level. Roberts et al. (2001) indicated a 6.4% point prevalence for insomnia and a 12.8% point prevalence for hypersomnia in a school based survey of over 5000 students from Houston Texas. Despite the European data and the known sex differences in adult insomnia prevalence (1.4:1, women: men; (Phillips et al. 2008)), there were no sex differences in the Houston sample.

Summary and Conclusions

There are profound and relatively sudden changes in sleep behavior and sleep architecture associated with the onset of adolescence. These relate both to the timing of sleep, and sleepiness, and the composition of the sleep and wake EEG with dramatic reductions in slow frequency activity and consequent reductions in measured slow wave sleep. The brain completes its transition from its highly plastic, high energy use, highly interconnected child form to the lower energy use, more efficient and faster processing adult form, with substantial synaptic pruning and myelination of white matter tracts occurring during adolescence. As a result of these combined changes in brain and sleep behavior and timing, insomnia-like problems emerge in adolescence in individuals who had good quality refreshing sleep as children. Changes in sleep quality, quantity and composition interact with the socially determined demands of the adolescent period, with dramatically increased academic demands and workload, and the normal developmental processes designed to promote independence from the family such as increased extent and amount of extra-familial social interactions. Unfortunately, they can also interact with the developmental tendency for increased risk taking, with the subsequent increased likelihood of alcohol or substance abuse, and driving while drowsy.

Many questions regarding the causal relationships between different observed changes in sleep across adolescence remain unanswered. For example, the extent to which synaptic loss versus increased integrity of white mater tracts influence the observed EEG changes; whether the changes in brain structure and organization relate to the alterations in circadian phase preference; the extent to which sleep homeostasis is altered during adolescence and whether it too has an influence on the observed circadian changes. The lack of a complete set of answers to these questions should not prevent action being taken to address what is clearly an epidemic of insufficient sleep in the adolescent populations in North America and developed Asian nations, and to a lesser extent in Europe and Australia. Growing recognition of the role of early school start times and the benefits of delaying them for high school students has not yet translated into substantial change in educational practice. Science and education are several steps behind real world behavior in terms of the increased sleep pressures being placed on adolescents by omnipresent communications technology and its associated ubiquitous social networking.

Sleep is one of the few fundamental human behaviors needed to support life along with breathing, drinking and eating. Psychologists, sleep scientists, and health professionals need to be vigilant in ensuring that adolescents, their parents and teachers view sleep as being of fundamental importance in supporting growth, health, cognitive development and learning, rather than something that is largely optional that can be sacrificed to meet the other demands of a 24×7 lifestyle.

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Acronyms

NREM	non-rapid eye movement sleep
REM	rapid eye movement sleep
EEG	electroencephalograph
SWS	slow wave sleep
SWA	slow wave activity (~0.5–4.5 Hz). Also referred to as delta power
MRI	magnetic resonance imaging
DTI	diffusion tensor imaging
FA	fractional anisotropy

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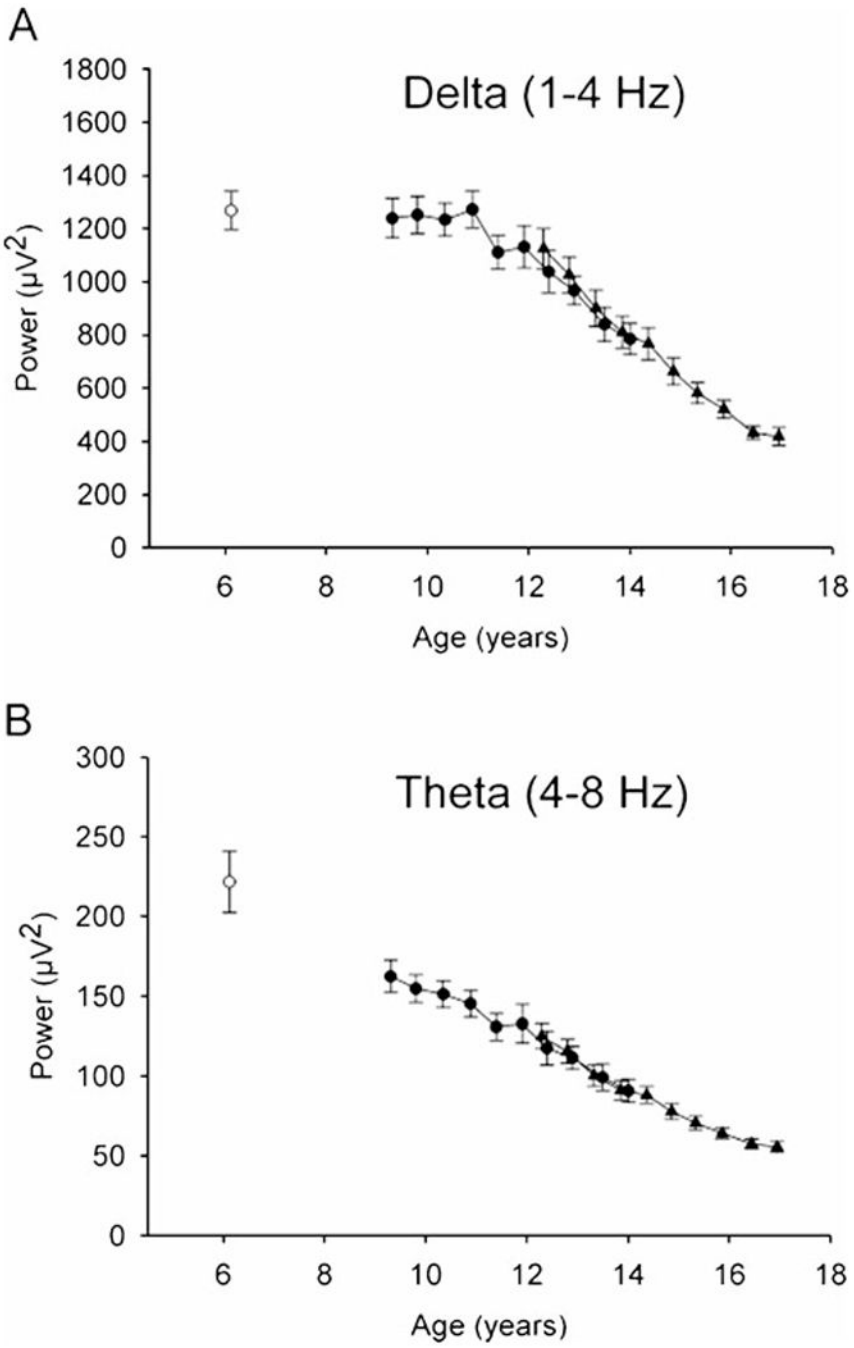

Fig. 1.

Reproduced from Feinberg and Campbell (2010). The data from their ongoing longitudinal study of sleep EEG show the change in (A) delta and (B) theta EEG activity across childhood and adolescence

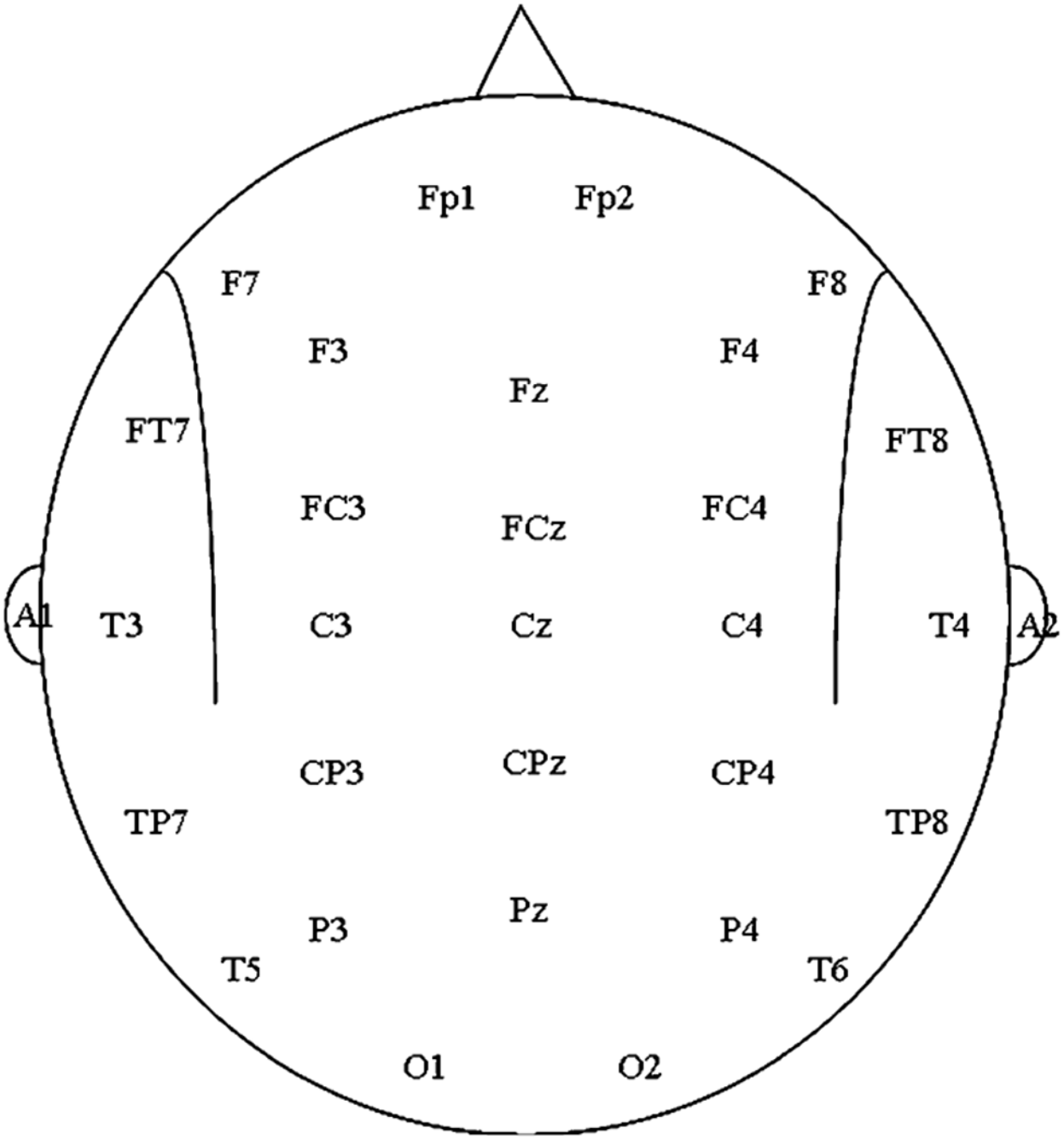

Fig. 2.

Representation of electrodes placed on the scalp according to the 10–20 convention. Most clinical sleep studies used C3 and C4. The new American Academy of Sleep Medicine guidelines (Iber et al. 2007) specify the addition of a frontal lead (F3 or F4) and an occipital lead (O1 or O2). High density EEG recordings (e.g., (Kurth, Ringli, et al. 2010)) can use more electrodes than those represented by adding additional sites in between the 10–20 positions

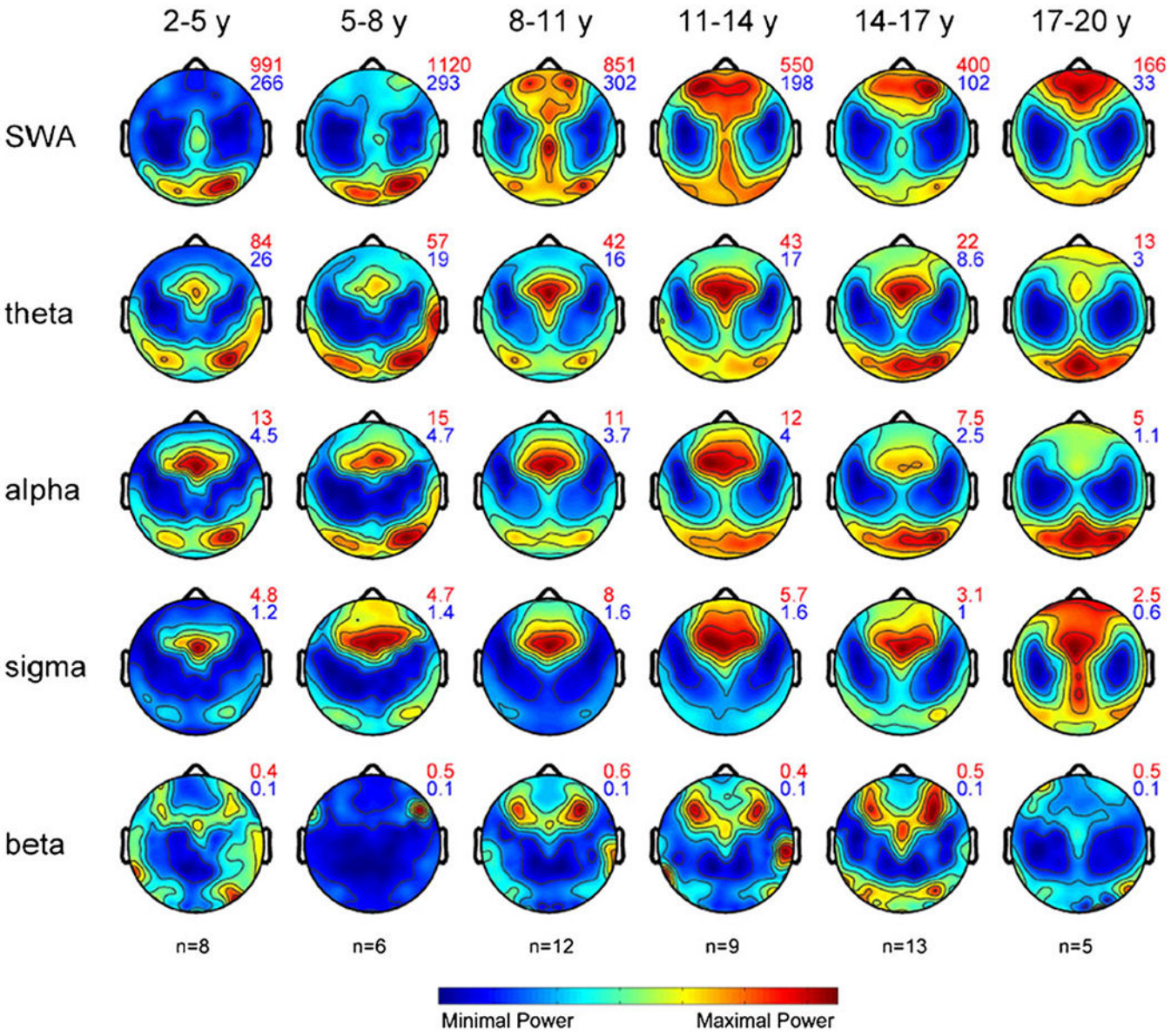

Fig. 3.

Reproduced from Kurth, Ringli, et al. (2010). Maps of EEG power during NREM sleep. Topographical distribution of NREM sleep EEG power for the defined age groups and frequency ranges (n=53). Maps are based on 109 derivations from the first 60 min of NREM sleep stages 2 and 3. Maps were normalized for each individual and then averaged for each age group. Values are color coded (maxima in red, minima in blue) and plotted on the planar projection of the hemispheric scalp model. To optimize contrast, each map was proportionally scaled, and values between the electrodes were interpolated. At the top right of the maps, numbers indicate maxima and minima (in square microvolts) for each plot

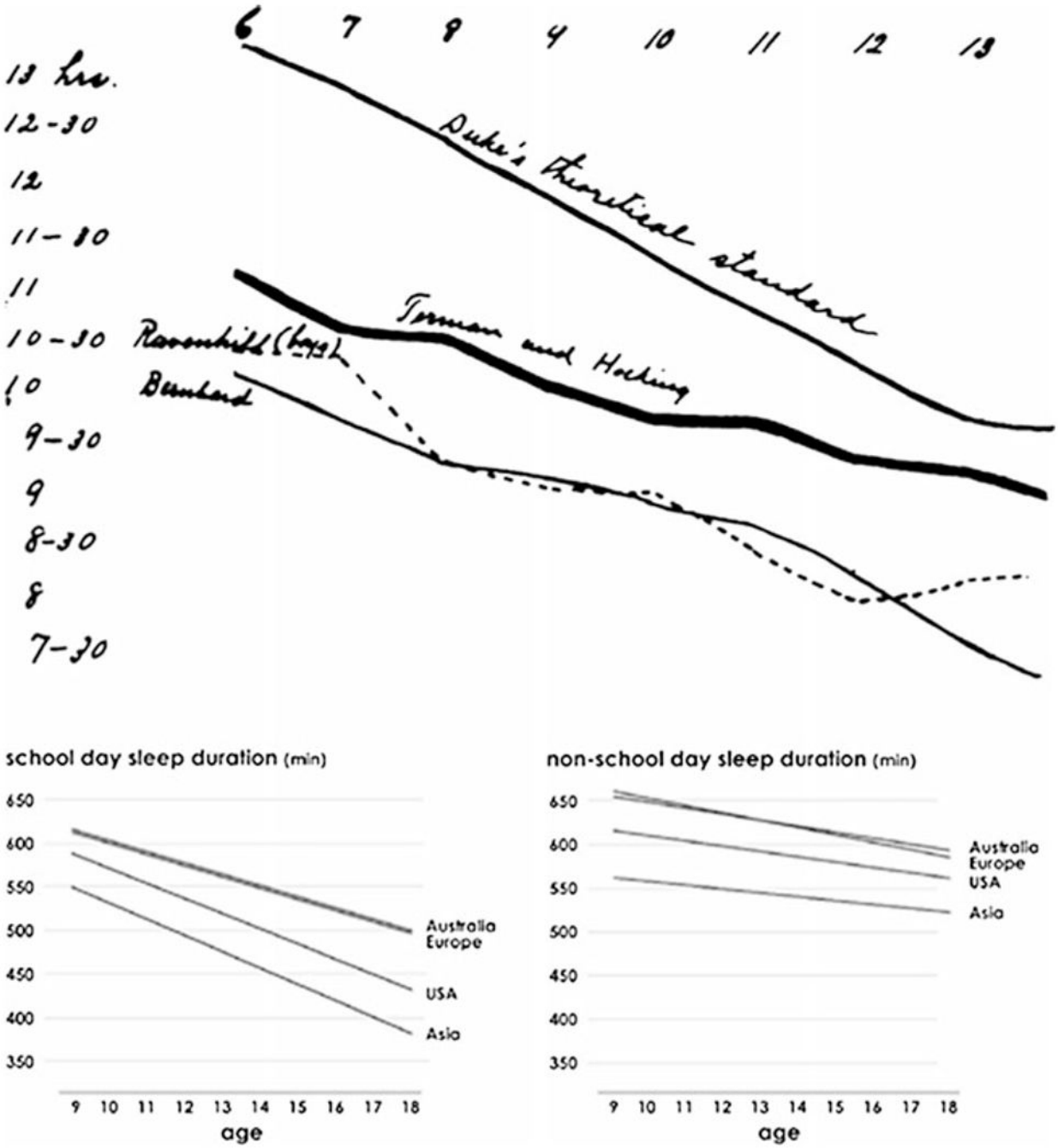

Fig. 4.

Upper panel reproduced from Terman and Hocking (1913). The data reflect hours of sleep in children aged 6 to 13 years. The thick line represents the data collected by Terman and Hocking in the western U.S. The dotted thin line represent data from Ravenhill (1910) collected in England and the thin solid line represent data from Bernhard (1908) collected in Germany. The comparison line at the top of the figure are the theoretical ideal values postulated by Dukes (1899). Lower panel reproduced from Olds et al. (2010a) showing the results of a meta analysis of data from different geographic regions. The data represent the

output of linear regressions conducted within each regions predicting total sleep time in minutes plotted by age

Table 1

Are changes in sleep due to pubertal development or aging?

A major issue in research in adolescents is differentiating age from pubertal status effects to determine if outcomes occurring in adolescence are part of “getting older,” are linked specifically to pubertal development, or are due to an interaction effect (Angold et al. 1998). Some of the changes in sleep across adolescence are associated with increased maturation and may be due to the activational effects of gonadal hormones on sleep homeostatic and circadian systems (Hagenauer et al. 2009). For example, Carskadon found that the increase in eveningness during adolescence was related to an increase in sexual maturity as reflected by Tanner stage in 5th and 6th grade students aged 11–12 years (Carskadon and Acebo 1993). A longitudinal study of sleep assessed with actigraphy across adolescence found significant correlations between sleep onset time, sleep efficiency, sleep time, and number of night awakenings and the increase in pubertal rating from Time 1 to Time 2 data collection points after statistically controlling for the effect of age (Sadeh et al. 2009). Subsequent analysis revealed that delayed sleep onset as well as other changes in sleep-wake patterns predicted more pubertal developed across time, whereas there was no significant prediction in the opposite direction (Sadeh et al. 2009). These findings suggest that adolescent changes in sleep may be evident before bodily changes associated with puberty (Sadeh et al. 2009). Other changes in sleep, notably the decline in SWA, are strongly correlated with age and not with pubertal status (Feinberg et al. 2006). More longitudinal studies that track pubertal hormonal changes (as well as changes in secondary sexual characteristics) in association with sleep measures may be better able to identify contributions of age versus pubertal development to the changes in sleep across adolescence.