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Daily timing of the adolescent sleep phase: Insights from a cross-species comparison

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ABSTRACT

Adolescence is a time of tremendous adjustment and includes changes in cognition, emotion, independence, social environment, and physiology. One of the most consistent changes exhibited by human adolescents is a dramatic delay in the daily timing of the sleep-wake cycle. This delay is strongly correlated with pubertal maturation and is believed to be influenced by gonadal hormone-induced changes in the neural mechanisms regulating sleep and/or circadian timing. Data from both human and non-human animals indicate that developmental changes in the intrinsic period of the circadian mechanism or its sensitivity to light are not adequate to explain adolescent changes in the daily timing of sleep and wakefulness. Rather, current evidence suggests that pubertal changes in the homeostatic drive to sleep and/or behaviorally induced changes in the amount and/or timing of light exposure permit adolescents to stay up later in the evening and cause them to wake up later in the morning.

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1. Adolescent changes in the amount and timing of sleep in humans

One of the most noticeable and reliable behavioral changes during adolescence is the development of an "evening chronotype," in which teenagers stay up late into the evening and sleep late into the morning. Adolescents from a variety of cultures consistently exhibit a pronounced delay in the daily timing of the sleep-wake cycle in comparison to both children and adults (Andrade et al., 1993; Andrade and Menna-Barreto, 2002; Carskadon, 1990; Gau and Soong, 2003, 1995; Gianotti and Cortesi, 2002; Laberge et al., 2001; Reid et al., 2002; Russo et al., 2007; Strauch and Meier, 1988; Thorleifsdottir et al., 2002; Wolfson and Carskadon, 1998; Yang et al., 2005). A delayed sleep-wake cycle is just one component of a pervasive "phase delay" in circadian timing during adolescence, in which a variety of daily rhythms (e.g., alertness, phase preference,

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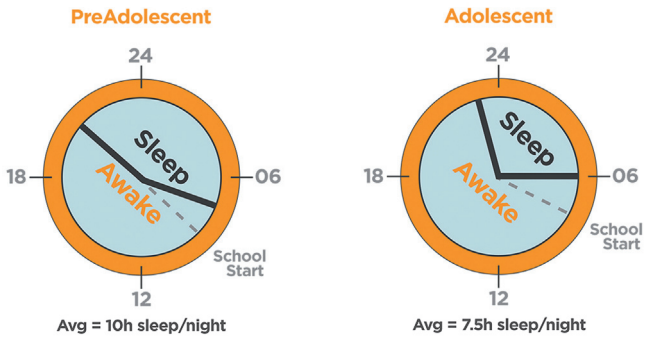

Fig. 1. The impact of delayed sleep and earlier school start times on sleep duration in adolescents. This figure models the rapid decrease in nightly sleep duration endured by children in many American schools as they begin pubertal development, experience a delayed sleep phase, and often start school earlier than before. A typical example is a girl entering middle school on the sleep/school schedule of a preadolescent in grade 6. The next year as she enters puberty and begins phase-delaying, she is required to start school earlier, resulting in dramatically decreased sleep duration. Schools around the country are now discussing the problems associated with adolescent sleep deprivation, and working on a later school start-time for middle and high school students. While there are improvements, few have adapted sufficiently so that adolescents get the needed 9 h of sleep per night throughout the week.

sleepiness, melatonin secretion) occur at a later phase with respect to the light-dark cycle (Carskadon et al., 2004, 1997, 1993; Crowley et al., 2006; Taylor et al., 2005). The adolescent delay in the daily timing of the sleep phase coincides with the age at which work and school schedules in some cultures demand that teenagers wake up earlier than before (Crowley et al., 2007; Roenneberg et al., 2004; Russo et al., 2007; Thorleifsdottir et al., 2002; Yang et al., 2005). As a result, teenagers adopt later bedtimes even during the school week when they must wake up early, and therefore get less sleep at night than young children (Fig. 1; Carskadon et al., 1998, 1980; National Sleep Foundation, 2006; Russo et al., 2007; Thorleifsdottir et al., 2002; Yang et al., 2005). There is no corresponding decrease in the amount of sleep that adolescents require (Carskadon, 1982), which means that the reduction in nighttime sleep contributes to a dramatic rise in daytime sleepiness (Carskadon et al., 1980; Russo et al., 2007; Thorleifsdottir et al., 2002; Yang et al., 2005).

Insufficient sleep amongst adolescents is a public health concern that threatens the academic success, health, and safety of young people and warrants greater attention from parents, educators, clinicians, researchers, and policy makers alike. Reduced sleep negatively impacts cognitive function in adolescents, including attention, memory, psychomotor speed, abstract thinking, creativity, and executive function (de Bruin et al., 2016; Goel et al., 2009; Kopasz et al., 2010; Randazzo et al., 1998; Tarokh et al., 2016). Reduced sleep duration is associated with attention problems, decreased academic motivation, truancy, and poor academic performance (Dewald-Kaufmann et al., 2013; Wahlstrom, 2002; Wolfson and Carskadon, 2003). Short sleep duration is also associated with higher levels of depression, risky behavior (e.g., alcohol use and sexual behavior), and suicidality (Carskadon, 2011; Dewald-Kaufmann et al., 2013; Fitzgerald et al., 2011; Gangwisch et al., 2010; Liu, 2004; O'Brien and Mindell, 2005; Wahlstrom, 2002; Wolfson and Carskadon, 1998; Tarokh et al., 2016). Furthermore, delays in the timing of the daily sleep phase and later chronotypes are associated with higher levels of depression in adolescents (Alvaro et al., 2014; de Souza and Hidalgo, 2014; Katharina et al., 2016; Pabst et al., 2009; Tzischinsky and Shochat, 2011; Tarokh et al., 2016). Gangwisch et al. (2010) demonstrated that adolescents with parent-set bedtimes of midnight or later exhibit higher rates of depression and suicidal ideation than adolescents with parent-set bedtimes of 10 p.m. or earlier. These increased risks were explained to a large degree by the shorter sleep duration concomitant with later bedtimes. These data suggest that the timing and duration of sleep are causally linked to depression in adolescents, and that earlier parent-set bedtimes (and thus longer sleep duration) may protect against teenage depression and suicide.

Sleep loss also negatively impacts immune function (Krueger et al., 1999) and has been linked to increased illness in adolescents (Orzech et al., 2014). Short sleep duration is a strong and consistent risk factor for obesity in adolescents (Fatima et al., 2015). There is evidence in adolescents to suggest that sleep loss negatively impacts energy balance, in part by disrupting glucose metabolism. Short sleep duration is associated with decreased insulin sensitivity in adolescents (De Bernardi Rodrigues et al., 2016; Matthews et al., 2012); and experimentally-induced sleep restriction has been shown to increase insulin resistance in male adolescents (Klingenberg et al., 2013). Short sleep duration in adolescents is also associated with dietary choices which promote weight gain and obesity, such as decreased consumption of healthy foods (e.g., fruits, vegetables, fish, etc.) and greater consumption of junk food (Garaulet et al., 2011). Beebe et al. (2013) demonstrated that experimentally-induced sleep restriction increases the consumption of high carbohydrate foods by adolescent participants. Whether or not these eating patterns related to sleep loss result from endocrine changes which promote hunger and eating is unclear. Taheri et al. (2004) reported that shorter sleep duration in an adult population was associated with elevated levels of ghrelin and reduced levels of leptin, hormones which increase and decrease appetite, respectively. Results from adolescent populations are less consistent. Al-Disi et al. (2010) found that shorter sleep duration was associated with higher circulating levels of ghrelin in a population of adolescent females. Positive relationships (Boeke et al., 2014), negative relationships (Hitze et al., 2009), and a lack of relationship (Al-Disi et al., 2010; Martinez-Gomez et al., 2011) between sleep duration and leptin levels have all been reported in adolescent populations. Furthermore, Klingenberg et al. (2012) failed to find an effect of experimental sleep restriction (4 h/night for 3 nights) on circulating levels of ghrelin or leptin in adolescent boys. The effects of sleep loss, however, are almost certainly cumulative. Additional research is needed to clarify the effect of chronic sleep loss on adolescent endocrine function.

Insufficient sleep also puts adolescents at risk for having a car accident. Pack et al. (1995) demonstrated that motor vehicle crashes, in which the driver had fallen asleep but was not intoxicated, peak at times of day which coincide with increased sleepiness (e.g., midnight to 7 a.m. and 3 to 5 p.m.). The majority of these car accidents occur in individuals under the age of 25 and peak at age 20.

2. Challenges with defining adolescence

Adolescence is the transitional period between childhood and adulthood characterized by extraordinary physical and psychological change. Researchers do not agree on precisely which physiological and/or psychological changes mark the beginning and end of adolescence or which changes differentiate stages of adolescent development (e.g., pre, early, mid, late). This issue becomes even more challenging when describing adolescence in non-human animals. In this review, we discuss adolescent changes in the sleep-wake cycle using puberty as a reference point. When possible, we explain how stages of pubertal development within a particular species have been defined by investigators (pre, peri, post, etc.). Many studies which report on adolescent changes in the sleep-wake cycle do not assess or report the development of secondary sex characteristics or other indices of pubertal maturation. In these instances, we report the age of the subject, noting that pubertal