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Sleep and circadian contributions to adolescent alcohol use disorder

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ABSTRACT

Adolescence is a time of marked changes across sleep, circadian rhythms, brain function, and alcohol use. Starting at puberty, adolescents' endogenous circadian rhythms and preferred sleep times shift later, often leading to a mismatch with the schedules imposed by secondary education. This mismatch induces circadian misalignment and sleep loss, which have been associated with affect dysregulation, increased drug and alcohol use, and other risk-taking behaviors in adolescents and adults. In parallel to developmental changes in sleep, adolescent brains are undergoing structural and functional changes in the circuits subserving the pursuit and processing of rewards. These developmental changes in reward processing likely contribute to the initiation of alcohol use during adolescence. Abundant evidence indicates that sleep and circadian rhythms modulate reward function, suggesting that adolescent sleep and circadian disturbance may contribute to altered reward function, and in turn, alcohol involvement. In this review, we summarize the relevant evidence and propose that these parallel developmental changes in sleep, circadian rhythms, and neural processing of reward interact to increase risk for alcohol use disorder (AUD).

Introduction

Adolescence is a time of marked changes across sleep, circadian rhythms, brain function, and alcohol use. Starting at puberty, adolescents' endogenous circadian rhythms and preferred sleep times shift later. This can lead to a mismatch with the schedules imposed by secondary education. Consequently, adolescents often suffer from circadian misalignment, sleep disturbance, and sleep loss. In parallel, adolescent brains are undergoing structural and functional changes in the circuits subserving the pursuit and processing of rewards. Alcohol use initiation typically occurs during adolescence, resulting in risk for alcohol use disorder (AUD). We propose that these parallel developmental changes in sleep, circadian rhythms, and neural processing of reward interact to increase risk for AUD.

Circadian rhythms primer

We live on a rotating planet, with light and dark periods that alternate over the 24-h day. Accordingly, most organisms have evolved to experience internal rhythms with approximately 24-h periods, known as circadian rhythms. Circadian rhythms exist in many physiological, behavioral, and psychological processes, including the sleep-wake cycle, and serve to organize these processes for optimal interaction with the environment. In humans and other mammals, these rhythms, which exist in tissues throughout the brain and body, are kept in time by a central clock located in the suprachiasmatic nucleus (SCN) of the hypothalamus. The SCN coordinates rhythms of various peptides and hormones, as well as temperature, which serve as internal messengers and synchronize rhythms in other brain areas and the periphery (Hastings, Reddy, & Maywood, 2003; Reppert & Weaver, 2002). The clock is primarily *entrained* – that is, synchronized to the environment – by light. Light is the most powerful entraining cue, or *zeitgeber* ("time giver" in German). Other cues, such as food, social interaction, activity, and drugs, also can entrain rhythms (Mistlberger & Skene, 2005).

Disruptions in regular exposure to light and, to a lesser degree, disruptions in other zeitgebers, can lead to internal desynchrony among various rhythms. Sustained desynchrony can have adverse effects on health and well-being. In shift work and jet lag, the sleep-wake cycle and exposure to the external light/dark cycle are misaligned from internal timing, requiring re-entrainment to the new schedule. In the case of night shift work, some individuals never entrain to the reversed schedule, and chronically exhibit circadian misalignment (Sack et al., 2007a). Jet lag is another example. After plane travel to other time zones, re-entrainment can require multiple days and proceeds at different rates in various internal processes. Circadian misalignment compromises function across physiological systems, and is likely responsible for the health ills ranging from gastrointestinal distress and affective disturbance

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associated with jet lag, to diabetes and cancer associated with long-term shift work (Davis & Mirick, 2006; Drake, Roehrs, Richardson, Walsh, & Roth, 2004; Monk & Buysse, 2013; Rogers & Reilly, 2002).

An important individual difference in circadian timing is termed chronotype. Early chronotypes have relatively advanced circadian timing, defined as being predisposed to earlier sleep-wake schedules. Late chronotypes have relatively delayed circadian timing, defined as being predisposed to later sleep-wake schedules (Roenneberg, Wirz-Justice, & Merrow, 2003). Definitive determination of chronotype requires the measurement of endogenous circadian phase via a physiological circadian marker such as melatonin or core body temperature. However, this is not always practical for larger studies or clinical settings. As a result, self-report measures of chronotype, also termed morningness-eveningness or diurnal preference, have achieved wide usage (e.g., Adan et al., 2012).

Early and late chronotypes (or morning- and evening-types) not only differ on sleep and circadian variables, but also exhibit marked differences in other areas of physical and mental health. As described in more detail below, late chronotypes (evening-types) tend to report more disturbed sleep and more irregular sleep timing, more depression, and increased rates of drug and alcohol use (Adan, 1994; Broms et al., 2011; Drennan, Klauber, Kripke, & Goyette, 1991; Gau et al., 2007; Hasler, Allen, Sbarra, Bootzin, & Bernert, 2010; Negriff, Dorn, Pabst, & Susman, 2011; Pieters, Van Der Vorst, Burk, Wiers, & Engels, 2010; Wittmann, Dinich, Merrow, & Roenneberg, 2006). These differences have been attributed to a phenomenon called *social jet lag* (Wittmann et al., 2006), which is operationalized as the difference between weekday and weekend sleep timing (typically the midpoint of sleep). Based on the social jet lag hypothesis, the later sleep-wake schedules preferred by evening-types are poorly matched with schedules imposed by school or work. As a result, evening-types suffer sleep onset insomnia and sleep loss on school or work days (typically weekdays). They have trouble falling asleep when attempting to sleep at a point in the circadian cycle incompatible with sleep onset, and their sleep bouts are curtailed by early rise times. In contrast, on free days (typically weekends) they tend toward later sleep-wake schedules and longer sleep durations, which result, in turn, in delays in their circadian timing. The "jet lag" occurs as the evening-types try to shift back to an earlier schedule at the conclusion of the weekend.

Adolescent sleep and circadian rhythms – brief overview

Important sleep and circadian changes occur during adolescence. Bedtimes shift later during middle school and high school, reflecting an increasing preference for later sleep times, a.k.a. eveningness (Carskadon, Acebo, & Jenni, 2004; Crowley, Acebo, & Carskadon, 2007; Randler, 2008; Roenneberg et al., 2004). The increase in eveningness continues until approximately age 18–20, when sleep preference undergoes a long slow shift toward earlier sleep times, a.k.a. morningness (Frey, Balu, Greusing, Rothen, & Cajochen, 2009; Roenneberg et al., 2004). These changes reflect, in part, alterations in the endogenous circadian clock, which shift to a later (more delayed) phase during puberty (Carskadon, Vieira, & Acebo, 1993). Reduced sensitivity to the homeostatic sleep drive that builds during extended wakefulness may also contribute to later bedtimes in adolescents (Hagenauer, Perryman, Lee, & Carskadon, 2009).

In the absence of social-cultural constraints, these normative developmental changes in sleep and circadian rhythms might be adaptive. Indeed, some have suggested that these changes serve as an evolutionary adaption aimed at increasing autonomy and independence by driving adolescents to be engaging the world at times when their parents have already retired to bed (Ellis et al., 2012). Instead, adolescents are faced with school schedules that sharply conflict with their pre-disposed timing (Hansen, Janssen, Schiff, Zee, & Dubocovich, 2005). Consequently, adolescents are forced to adopt schedules that are too early during the school week, resulting in difficulty falling asleep at night, curtailed sleep duration, and daytime fatigue. Then, on the weekend, adolescents return to their preferred timing, staying up later, and sleeping into the late morning or afternoon to make up for sleep loss during the school week. Sleeping later has consequences for the circadian system, which responds to the later rise times and later exposure to light by delaying internal timing (Crowley & Carskadon, 2010). This maintains adolescents' later circadian timing, resulting in insomnia and sleep loss as the school week begins. In other words, many adolescents must do the equivalent of traveling multiple time zones each Sunday night and Monday morning. This social jet lag, when operationalized via differences in weekday-weekend sleep timing, is associated with mood disturbance and drug and alcohol use in both adolescents (O'Brien & Mindell, 2005; Pasch, Laska, Lytle, & Moe, 2010) and adults (Levandovski et al., 2011; Wittmann et al., 2006). Increasingly prevalent use of social media and late-night exposure to the light from electronic devices may compound delays in sleep timing (Munezawa et al., 2011; Pieters et al., 2012; Van den Bulck, 2007; Wood, Rea, Plitnick, & Figueiro, 2013).

Cross-sectional associations between sleep, circadian rhythms, and alcohol involvement

Sleep problems/disorders

Sleep problems include disturbed sleep continuity (difficulty falling or staying asleep), insufficient sleep duration (sleep deprivation or restriction), and hypersomnia (excessive sleep is desired and/or required). Although there are important distinctions among these categories, all have been associated with alcohol problems, suggesting that any sleep disturbance increases risk for AUDs.

Multiple studies have demonstrated cross-sectional associations between sleep problems and alcohol use/AUDs in adolescents and adults. Past-year insomnia symptoms were associated with increased use of alcohol, cannabis, and other drugs, after controlling for sex effects, in a sample of 4494 12–18-year-olds (Roane & Taylor, 2008). In 703 South African adolescents, overall sleep problems (trouble falling or staying sleep, tiredness in the morning, and/or daytime sleepiness) were associated with greater lifetime alcohol and drug use, and this association was independent of the effect of learning difficulties (Fakier & Wild, 2011). Comparing adolescents with AUDs and a reference group, Clark and colleagues (Clark, Lynch, Donovan, & Block, 2001) found self-reported sleep problems (5 items) to be significantly associated with AUD, negative emotionality, and tobacco involvement. The association between AUD and sleep problems remained statistically significant after controlling for negative emotionality and tobacco involvement. Other studies have linked sleep problems to consequences of alcohol use. Self-reported sleep quality over the past month was associated with greater binge drinking and more alcohol-related consequences (e.g., "Had a fight, argument, or bad feelings with a friend") in a sample of 261 college students (Kenney, LaBrie, Hummer, & Pham, 2012). Furthermore, in that study and a larger follow-up study, the authors reported compounding effects of sleep problems and alcohol involvement. Poorer sleep quality was associated with greater alcohol-related consequences in heavy drinkers (Kenney et al., 2012). The combination of poorer sleep quality and higher coping motives (e.g., to forget your worries) was associated with worse alcohol-related consequences (Kenney, Lac, Labrie, Hummer, & Pham, 2013). Findings such as these suggest a complex, synergistic, and likely bidirectional relationship between sleep and alcohol use.