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Sleep and circadian risk factors for alcohol problems: A brief overview and proposed mechanisms

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Abstract

Disturbances in sleep and circadian rhythms may be important, albeit underappreciated, risk factors for the initiation of alcohol use and the escalation of alcohol problems. In this review, we first describe sleep and circadian changes during adolescence and young adulthood. Second, we explain how these sleep/circadian changes intersect with onset and escalation of alcohol use. Third, we briefly note how alcohol use (whether acute or chronic) affects sleep and circadian rhythms. Finally, we articulate a conceptual model containing two mechanistic pathways—broadly positive and negative reinforcement—linking sleep/circadian factors to alcohol involvement before listing key areas we believe are ripe for further inquiry.

Keywords

sleep; circadian rhythms; alcohol; positive reinforcement; negative reinforcement

INTRODUCTION

Accumulating evidence indicates that disturbances in sleep and circadian rhythms may be important, albeit underappreciated, risk factors for the initiation of alcohol use and the escalation of alcohol problems. In this review, we (1) briefly review relevant changes in sleep and circadian rhythms during adolescence into young adulthood, when alcohol use is onsetting and peaking. (2) describe burgeoning evidence that sleep/circadian disturbances often precede and/or predict alcohol involvement, (3) briefly note alcohol's effects on subsequent sleep/circadian rhythms, and (4) articulate some putative mechanisms linking sleep/circadian factors to alcohol involvement that we identify as areas deserving further inquiry.

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SLEEP AND CIRCADIAN CHANGES DURING ADOLESCENCE AND YOUNG ADULTHOOD

Marked changes in sleep and circadian rhythms begin with the onset of puberty and continue into the early 20's, corresponding with increased heavy alcohol use [1]. Sleep timing shifts later over this timespan, as evident via both studies of actual sleep timing, as well as studies of morningness/eveningness preference [2, 3]. Along with sociocultural influences, these timing shifts appear to be driven by two biological processes: (1) increasingly later circadian timing (e.g.,, per salivary melatonin) [2]; and (2) a slower accumulation of homeostatic sleep pressure (i.e., per electroencephalographic (EEG) measures of slow-wave activity) [4]. Based on studies of chronotype, the shift towards later timing appears to peak around age 20, then reverses course towards earlier timing over the rest of the lifespan [3]. The tendency towards later timing is mismatched with early school start times, resulting in difficulty falling asleep (insomnia), insufficient sleep, and daytime sleepiness on weekdays, as well as large swings in sleep timing and duration on weekends, when adolescents tend to stay up later and sleep in in order to make up for lost sleep (i.e., 'social jet lag' [5]). Current recommendations are for 8-10 hours of sleep per night on average [6], but large survey studies suggest that ~ 1/3 of adolescents achieve the recommended sleep duration [7]. The prevalence of insomnia is also elevated during adolescence and young adulthood [8].

SLEEP AND CIRCADIAN CHARACTERISTICS AS CORRELATES OF AND RISK FACTORS FOR ALCOHOL INVOLVEMENT

Concurrent associations between sleep disturbances and heavier alcohol use and/or more problems during adolescence and young adulthood are well-recognized [9]. Furthermore, worse or insufficient sleep appears to be related to engagement in alcohol use [10] and more severe consequences of alcohol use [11], particularly when coupled with higher alcohol coping motives (e.g., drinking to reduce anxiety) [12]. Perhaps the most consistent finding is that greater eveningness (a preference for relatively later activity and sleep) is associated with greater alcohol involvement [13–16]. Eveningness is also associated with greater endorsement of a range of reasons for drinking that are associated with elevated alcohol use [17]. Evening-types are more likely to experience larger weekday-weekend changes in sleep timing (i.e., social jet lag) [5], which have also been linked to higher alcohol use in adolescents and young adults [18*, 19].

Studies employing objective circadian measures (e.g., salivary melatonin) are much scarcer, but generally align with the subjective sleep literature. In a sample of high-school aged adolescents with a history of substance use disorders (SUD), later circadian timing and greater circadian misalignment were both associated with higher current SUD symptoms [20]. Circadian misalignment was operationalized as a shorter interval between the onset of melatonin secretion (typically 2-3 hours before bedtime) and the midpoint of the sleep period, consistent with sleeping at an earlier time relative to one's circadian clock. This finding was extended recently to 18-22 year-olds using the same operationalization of circadian misalignment [18*]. Greater circadian misalignment prior to the weekend was associated with greater alcohol use over the weekend. These findings suggest that it's not

simply later timing that matters, but the relative timing between one's sleep schedule and one's internal circadian clock.

Importantly, an increasing number of longitudinal studies of sleep and alcohol involvement have been published in the last decade. Almost without exception, these studies have consistently reported that poor sleep, short sleep, and later and/or more variable sleep timing predict the onset and/or escalation of alcohol use and alcohol-related problems [21–25]. One limitation of some of these studies has been the necessary reliance on convenience measures of sleep, often based on single items from larger questionnaires (e.g., Child Behavior Check List). However, more recent publications have begun to address this limitation. For example, a recent paper from the National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA) study assessed a wider range of sleep/circadian characteristics using full questionnaires and/or abbreviated versions of validated questionnaires [26*]. In that study, greater eveningness, later bedtimes (weekday and weekend), and shorter weekday sleep duration at baseline were all predictive of an increase in heavy drinking a year later, accounting for baseline drinking.

EFFECTS OF ALCOHOL USE ON SLEEP AND CIRCADIAN RHYTHMS

While a detailed review of alcohol effects on sleep and circadian rhythms is beyond the scope of the present paper (see [27, 28] for more thorough discussion), we offer a concise review of the most pertinent findings here. Acute use of alcohol in healthy adults tends to speed the onset of sleep and improve sleep consolidation in the first half of the night, including increasing slow wave sleep, while disrupting sleep in the second half of the night, including reducing rapid eye movement (REM) sleep [27]. These acute effects may depend on both time-of-day and developmental stage. Evening use of alcohol may be relatively more stimulating for adolescents and emerging adults than at other times of day [29], and alcohol is generally less sedating during adolescence [30], which together may explain findings suggesting that alcohol use does not reduce the time to fall asleep in adolescents [31]. In laboratory studies of chronic alcohol use (repeated alcohol administration), any short-term benefits for sleep in the first half of the night disappear [32]. Sleep disorders are common (36-91%) among people with an alcohol use disorder (AUD) [33], consistent with long-term heavy alcohol use disrupting sleep. Although relatively less studied in adolescents, one longitudinal study suggests that adolescents with an AUD at baseline have persistent sleep disturbances at least 5 years later [22]. Alcohol use is also associated with altered circadian rhythms in humans [28] and animal models, with the latter suggesting that alcohol disrupts light's phase-shifting effects on circadian timing [34], although a study in humans failed to replicate this effect [35].

The effects of alcohol on sleep may be moderated by sleep/circadian factors. A few laboratory-based alcohol administration studies suggest that adults with insomnia differentially benefit from alcohol use in the short-term, with relatively larger increases in slow wave sleep and reductions in anxiety compared to adults without insomnia [32]. However, these benefits diminish quickly with consecutive nights of alcohol use [36]. Although similar studies have not been conducted in adolescents with insomnia, data

suggests that adolescents with insomnia are more likely to self-medicate with alcohol, particularly among males [37].

POSSIBLE MECHANISMS LINKING SLEEP/CIRCADIAN FACTORS TO RISK FOR ALCOHOL INVOLVEMENT

We put forth a conceptual model (see Figure 1) that integrates existing literature with future directions to further our understanding of how sleep and circadian disturbances increase risk for alcohol problems. Based on the extant evidence, we contend that putative mechanisms linking sleep/circadian factors to increased risk for alcohol involvement fall into two categories that can be broadly labeled positive and negative reinforcement, respectively.

As reviewed in more detail elsewhere [38, 39], a burgeoning literature implicates reward-related processes (i.e., positive reinforcement) in the link between sleep/circadian disturbance and adolescent alcohol involvement. Robust evidence supports sleep/circadian modulation of reward-related behavior (e.g., alcohol use and drug-seeking behavior) and its underlying physiology [40–45]. In adolescent and young adult samples, increased eveningness is associated with greater self-reported sensation- and novelty-seeking, reward dependence, and impulsivity [46–49]. Furthermore, we have shown in late adolescence that greater eveningness is associated with altered neural reactivity to reward and in turn correlated with greater alcohol use and AUD symptoms [50], including prospectively [51]. Although these studies have generally employed monetary reward tasks, preliminary evidence from a alcohol administration study we conducted suggests that later sleep timing may be associated with increased sensitivity to alcohol-related reward [52*]. Specifically, we found that later sleep timing in an adult, moderate-drinking sample was associated with greater self-reported stimulation following alcohol consumption (relative to drinking a non-alcohol beverage). Notably, this appeared to be only true in White, not Black participants (see below for further discussion).

Evidence for the negative reinforcement pathway linking sleep/circadian factors to alcohol involvement comes from evidence that alcohol is often used to improve sleep and reduce anxiety (which may, in turn, ease the transition to sleep). Alcohol is a commonly reported “sleep aid” in individuals with poor sleep [53], with prevalence estimates up to ~30% in the general US population [54]. Although, as described above, the evidence clearly indicates that alcohol use is detrimental to sleep overall, at least one national survey reported that ~67% of individuals with insomnia view alcohol as effective for improving sleep [54]. Notably, anxiety is commonly comorbid with insomnia [55] and alcohol problems [56]. Higher anxiety predicts drinking to cope with negative mood [57] and greater reduction in tension/anxiety following alcohol consumption [58]. This may explain in part the aforementioned finding that individuals with insomnia exhibit relatively greater sleep benefits after alcohol use [59*]. The insomnia group (n=11) in that study also reported a greater post-alcohol decrease in anxiety relative to the control group (n=9), suggesting that alcohol may be more reinforcing to individuals with insomnia due to a larger reduction in anxiety. Insomnia and related sleep disturbances appear to increase the risk of relapse in

alcohol-dependent individuals during abstinence [60], suggesting that insomnia may engage negative reinforcement processes at multiple stages of the AUD course.

Impulsivity could play an exacerbating role in both positive and negative reinforcement pathways, given evidence of its sensitivity to sleep/circadian factors (e.g., [61*]) and relevance to alcohol problems [62]. Specifically, impulse control appears to be diminished in the context of late sleep timing, sleep disturbance and/or sleep loss, making affected individuals more likely to engage in heavy alcohol use during periods of worsened mood (i.e., negative urgency) or when feeling more stimulated from alcohol.

FUTURE DIRECTIONS

Although we believe the existing evidence makes a compelling case for the value of understanding the role of sleep and circadian characteristics in risk for later alcohol involvement and alcohol use disorder, a number of important questions remain.

In particular, research is needed to articulate which specific sleep (e.g., timing, duration, continuity) and circadian (e.g., chronotype, time of alcohol use) factors are mechanistically related to responses to alcohol, mood (positive and/or negatively-valenced), impulsivity, and their interaction (e.g., negative urgency: acting rashly while in a strong negative mood). Identification of biobehavioral mechanisms by which sleep/circadian factors influence alcohol involvement will directly contribute to the optimization of prevention and intervention approaches. Examination of the dynamic transaction between sleep, impulsivity, mood and alcohol response is needed to further understanding of for whom sleep/circadian factors are likely to increase problematic alcohol use.

Such work will require moving beyond the cross-sectional study designs that have dominated the extant literature to prospective and experimental designs. Although longitudinal papers in this area are accumulating, many are limited by use of single items rather than well-validated full self-report measures or objective alternatives (e.g, wrist actigraphy). We assert the need for more intensive, prospective studies using ecological momentary assessment (EMA) methods and robust sleep measures to investigate more proximal relationships between sleep/circadian factors, putative mechanisms, and alcohol use. A few such studies are emerging, including a recent one in college students that reported complex, bidirectional day-to-day relationships between sleep and drinking. Specifically, Fucito and colleagues [63*] reported that heavy drinking predicted delayed timing and shorter sleep that night, while short sleep, early wake times, and greater sleep quality predicted heavier drinking the following day. And, of course, to properly assess causality and directionality, we need more experimental studies, including both sleep/circadian manipulation protocols and alcohol administration protocols, both of which can be combined with EMA.

As in other areas of the literature, there has been insufficient consideration to date of the role of sex and/or race in sleep/circadian-alcohol associations. Sleep/circadian characteristics vary by sex and race [64–66], as does alcohol use behavior and risk for alcohol problems [67, 68]. For example, male individuals tend to exhibit later sleep timing [65], while female

individuals report more insomnia [66]. Black individuals tend to experience both shorter and worse sleep than White individuals [69] but may be less subject to apparent effects of sleep timing on alcohol response [52]. Given that all of these sleep/circadian characteristics have been linked in turn to alcohol involvement, it is plausible that they may contribute to relative risk differences across sex and race. Indeed, a handful of studies suggest that sex moderates the respective relationships between sleep/circadian characteristics and sensation-seeking, reward dependence, depression, and nicotine use [70*–73], and two recent studies suggest that men may be particularly vulnerable to sleep-related risk for alcohol problems [37, 74*].

Finally, while the literature to-date suggests that sleep-focused treatments benefit sleep in heavy-drinking individuals or those with AUD, no studies have yet shown a differential benefit for alcohol-related outcomes [75, 76]. We are excited about the prospect that as our understanding of mechanisms improves, the possibility for enhancing current treatment approaches will likely increase, perhaps by incorporating chronotherapeutic approaches (e.g., bright light) that have not yet been employed in AUD patients. This holds great promise considering sleep-focused treatment carries less stigma than alcohol-focused interventions [77*, 78], which could motivate relevant individuals to seek help for sleep while paving the way for eventual focus on their alcohol involvement.

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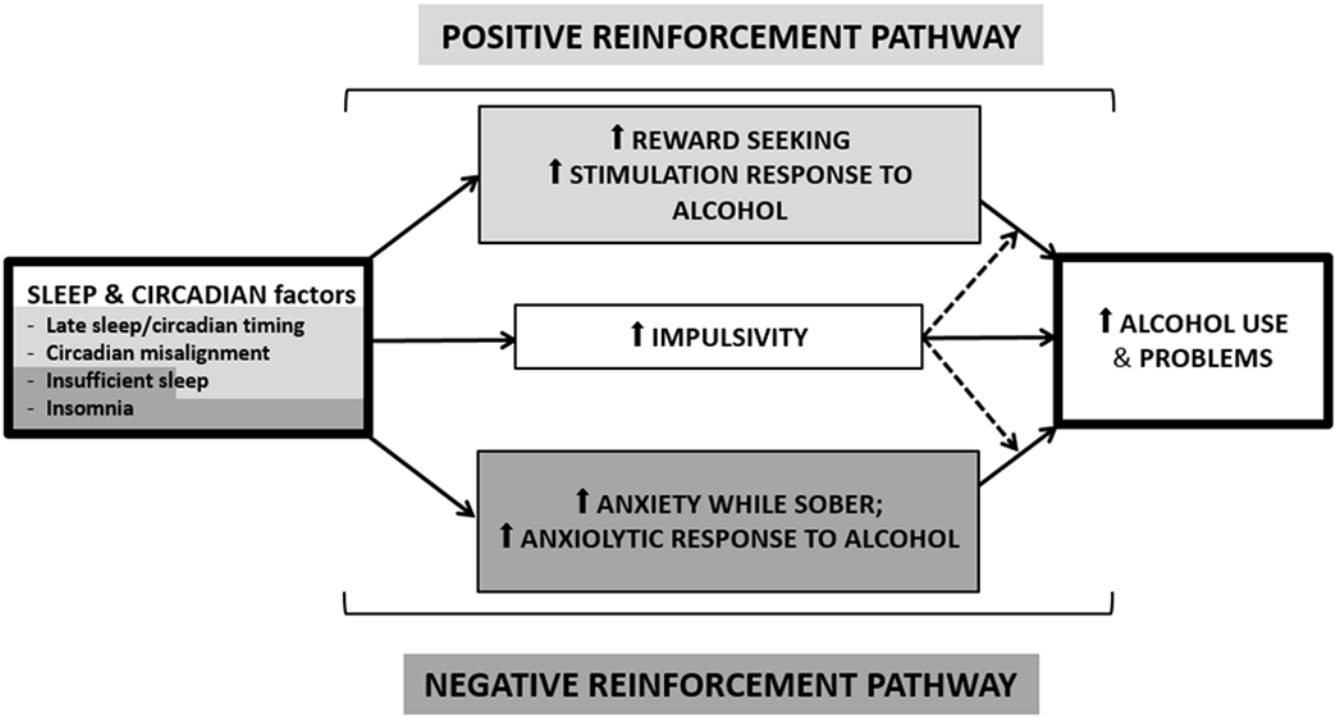

Figure 1.

Conceptual model of how sleep and circadian disturbances may increase risk for alcohol problems via positive and negative reinforcement pathways. Furthermore, impulsivity could play an exacerbating role in both positive and negative reinforcement pathways. Shading is used to distinguish the two separate pathways, including indicating potentially distinct pathways from specific sleep/circadian characteristics, such as circadian misalignment influencing alcohol use via positive reinforcement processes, while insomnia influencing alcohol use via negative reinforcement processes. The bi-color shading of insufficient sleep is intended to indicate that it may influence risk via both positive and negative reinforcement pathways.