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Self-Medication for Sleep in College Students: Concurrent and Prospective Associations with Sleep and Alcohol Behavior

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Abstract

Objective/Background—College students are at an increased risk for poor sleep and associated sleep problems. Emerging evidence suggests that a substantial subset of college students self-medicate with alcohol, marijuana, and/or over-the-counter medications to help sleep. The current study identified demographic, psychosocial, and sleep- and alcohol-related correlates of self-medication for sleep, and assessed its concurrent and prospective associations with insomnia symptoms, alcohol drinking and negative drinking consequences.

Participants—Undergraduate students (N = 171; mean age = 19 years [SD = 1.35], 32% male, 74% White) enrolled in a four-year university in the northeastern United States.

Methods—Data were drawn from a short-term two-wave longitudinal study. Participants completed two online surveys, separated by an average interval of 68 days (SD = 10.22).

Results—At Time 1, 25% of students reported using at least one substance (alcohol, marijuana, and/or over-the-counter medications) for sleep aid in the past two weeks. Male and older students were more likely to report using substances for sleep. Sleep aid use at Time 1 was concurrently associated with greater levels of alcohol frequency, negative drinking consequences, and insomnia symptoms. Further, sleep aid use at Time 1 was associated with an increase in negative drinking consequences from Time 1 to Time 2, but not with changes in alcohol frequency or insomnia symptoms.

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Conclusions—These findings indicate that substances are widely used among college students for sleep aid. Sleep aid use is associated with greater concurrent drinking and insomnia symptoms, and increases in negative drinking consequences over a short time period.

Keywords

insomnia; alcohol; marijuana; over-the-counter medication; college

Self-Medication for Sleep in College Students: Concurrent and Prospective Associations with Sleep and Alcohol Behavior

College students are at an increased risk for poor sleep and associated sleep problems. Approximately two thirds of college students do not get the recommended amount of sleep for their age group (i.e., 7 - 9 hours; Hirshkowitz et al., 2015; Lund et al., 2010). Further, 12 – 23% of college students have endorsed clinically significant insomnia according to validated screening cut-offs (Gellis et al., 2014; Gress-Smith et al., 2015). Poor sleep among college students has been concurrently associated with a plethora of problem behaviors, including increased alcohol consumption and physical illness (DeMartini & Fucito, 2014; Lund et al., 2010), fighting and risky sexual behaviors (Vail-Smith, Felts, & Becker, 2009), and motor vehicle accidents (Taylor & Bramoweth, 2010).

Emerging evidence suggests that a substantial subgroup of college students opt to self-medicate their sleep with substances such as alcohol, marijuana, and over-the-counter medications. In a large college student sample (N = 1,125), 10% of poor-quality sleepers endorsed using alcohol, and 33% endorsed using over-the-counter or prescription medications to help sleep (Lund et al., 2010). In another large study of college students (N = 1,039), 11% of all students endorsed the use of alcohol as a sleep aid and 2% endorsed the use of over-the-counter medications, regardless of sleep quality (Taylor & Bramoweth, 2010). Despite the drastic increase in marijuana use among college students in recent years (Johnston et al., 2015), however, no research has assessed marijuana use for sleep aid among college students. Given the limited number of extant studies, further research is needed to reconcile discrepancies in the prevalence of alcohol and over-the-counter medications for sleep aid and assess the likely prevalent trend of marijuana use for sleep aid among college students.

According to Brower's model of the reciprocal associations between insomnia and alcoholism (2003), self-medication for sleep is an integral part of a negative feedback loop of exacerbating insomnia and alcohol problems over time. This model maintains that insomnia increases alcohol use in an effort to self-medicate sleep; at the same time, however, alcohol use causes or worsens insomnia due to ethanol's toxicity on sleep-related brain systems. The troubling implication of this model is that the use of alcohol for sleep aid increases in response to worsening insomnia, thereby increasing the risk of worsening or relapsing alcohol problems over time. However, this model was developed for alcoholics and has not been applied to a college student sample. Thus, it remains unknown how insomnia and diverse alcohol behaviors synergistically influence each other over time among this group. Further, this feedback model has focused exclusively on alcohol and has not

encompassed other substances commonly utilized for sleep aid such as over-the-counter medications or marijuana. The current study sought to apply this promising model to self-medication for sleep (alcohol, marijuana, and over-the-counter medications) among college students and examine its correlates and short-term insomnia- and alcohol-related outcomes.

Brower (2003) proposed several demographics (e.g., sex), depression and anxiety symptoms, sleep-related characteristics (e.g., sleep duration), and alcohol use patterns as correlates of co-occurring insomnia and alcoholism. However, it has yet to be examined whether the same set of factors along with other potentially relevant individual characteristics are associated with self-medication for sleep among college students. Such findings regarding demographic, sleep and alcohol use characteristics associated with sleep aid use would be valuable in identifying at-risk students, potentially allowing for earlier intervention. Regarding demographics, a finding from the limited research of college students supports that male sex is indeed associated with alcohol sleep aid use (Taylor & Bramoweth, 2010). Given that full-time college students are homogeneous in terms of age (typically 18 to 25 years old; National Center for Education Statistics, 2016), and increases in alcohol frequency are not observed over time among college students (Nealis et al., 2017), age or legal drinking status may not be associated with substance use for sleep; however, these associations have yet to be tested empirically. Empirical evidence from insomnia and alcohol studies of college student suggests that negative mood, sleep- and alcohol-related characteristics may be associated with self-medication for sleep. Depression and anxiety symptoms have been associated with both insomnia (Gress-Smith et al., 2015), and alcohol/substance use and negative drinking consequences (Geisner, Mallett, & Kilmer, 2012; Shannon R. Kenney et al., 2013) among college students. Also, because sleep aid use is associated with poor sleep (Lund et al., 2010), it would be useful to characterize specific sleep-related characteristics that are associated with college students to self-medicate with substances. Pre-sleep arousal (Nicassio et al., 1985), inconsistent sleep scheduling (Gellis et al., 2014), and evening circadian preference (Fernández-Mendoza et al., 2010) have been associated with poor sleep among college students and therefore worthy to explore their association with sleep aid use. Further, risky drinking patterns might predispose students to sleep aid use, as alcohol sleep aid has been concurrently associated with greater quantity of alcohol use (Lund et al., 2010).

Self-medication for sleep among college students is especially concerning considering its potential role in the development of exacerbated alcohol/substance use and associated consequences over time. While alcohol, marijuana, and/or over-the-counter medications possess sedative qualities and are thus temporarily effective in initiating sleep-onset (Chait & Perry, 1994; Rickels et al., 1983; Roehrs et al., 1999), the use of sleep aids is shown to negatively impact objective sleep measures. For example, alcohol use before bed typically results in more awakenings and restlessness during the second half of the night (Roehrs, Yoon, & Roth, 1991). Likewise, over-the-counter sleep aid use is associated with residual sedation during the following day and can also result in rebound insomnia, making it difficult to fall asleep naturally without sleep aid use (Katayose et al., 2012). Thus, self-medication for sleep may contribute to a dangerous feedback loop by which students hurt their sleep and continue to compensate with alcohol/substances, thereby increasing their risk of developing risky alcohol/substance use habits (Brower, 2003). This effect might be

exacerbated by the fact that the desired effects of sleep aids reduce or extinguish over time (Bedi et al., 2010; Rundell et al., 1972; Schweitzer, Muehlbach, & Walsh, 1994), likely leading sleep aid users to increase consumption to combat tolerance. For example, the desired effects of marijuana on sleep efficiency and satisfaction were found to last for only the first eight days of intake, after which a higher dose would be required achieve similar effects (Bedi et al., 2010). However, research regarding sleep aid use among college students remains exclusively cross-sectional in nature, and therefore its prospective effects on subsequent insomnia symptoms, alcohol use and alcohol problems over time are unknown.

Study Aims and Hypotheses

This study aimed to characterize prevalence of self-medication (alcohol, marijuana, and/or over-the-counter medication) for sleep among college students, identify its demographic, psychosocial, sleep and alcohol related correlates, and assess its short-term prospective associations with alcohol and sleep behaviors. Specifically, the current study examined differences between sleep aid users and non-users in concurrent demographic, psychosocial, sleep-related, and alcohol-related characteristics. It was hypothesized that, compared to non-sleep aid users, sleep aid (i.e., alcohol, marijuana, and/or over-the-counter medication use for sleep) users would be more likely to be male, and report greater concurrent levels of depression/anxiety symptoms, poor sleep (i.e., shorter sleep duration, greater insomnia severity), sleep characteristics associated with poor sleep (i.e., greater sleep scheduling inconsistency, greater pre-sleep arousal, and more evening circadian preference), alcohol frequency and negative drinking consequences. Additionally, short-term prospective associations of sleep aid use with changes in insomnia severity, alcohol frequency, and negative drinking consequences were examined. It was hypothesized that sleep aid use would be associated with greater increases in insomnia severity, alcohol frequency, and negative drinking consequences over a short period of time.

Method

Participants and Procedure

Data were drawn from a two-wave longitudinal study of 171 undergraduate students (mean age = 19 years [*SD* = 1.35, range = 18 to 28], 32% male) at a four-year university in the northeastern United States (Gellis et al., 2014; Park et al., 2014). Students were eligible to participate if they were 18 years of age or older and endorsed having had at least one alcoholic drink in the past 30 days. The sample consisted of 74% White, 12% Asian, 6% Black or African American, 6% multiracial, 1% American Indian or Alaska Native, and 1% missing on race. Participants were recruited from psychology classes and offered course credit incentive. Participants completed two online surveys, with an average interval of 68 days (*SD* = 10.22). Of the 171 participants at Time 1 (T1), 157 (92%) also participated at Time 2 (T2). A logistic regression including all T1 variables showed that attrition was predicted only by insomnia severity (OR = 1.24; 95% CI [1.04, 1.48]), indicating that students reporting greater insomnia severity at T1 were more likely to drop out from study.

Measures

Sleep aid use—Three investigator-developed items assessed past two-week frequencies of using alcohol, marijuana, and/or over-the-counter medications to help sleep at T1 and T2, such as “How many days per week in the past 2 weeks have you taken alcohol to help you sleep at night?” In the absence of a standardized measure for sleep aid use, we utilized items from the Consensus Sleep Diary (Carney et al., 2012), modified wording appropriately for each substance, and adapted the response timeline for the past 2 weeks for consistency with the Insomnia Severity Index (Bastien, Vallieres, & Morin, 2001). Participants responded based on a 5-point scale (0 = *0 days*, 1 = *1-2 days*, 2 = *3-4 days*, 3 = *5-6 days*, 4 = *7 days*). For main analyses, scores from these three items were combined to create a dichotomized variable representing any use of substances for sleep aid (0 = no sleep aid use, 1 = any sleep aid use). Test-retest reliability coefficient of this dichotomized sleep aid use measure from T1 to T2 was $r = .40$, $p < .001$, suggesting a moderate level of stability of sleep aid use over a 2-month period. The dichotomized score of each of these substance use items was utilized for ancillary analyses (see Data Analytic Strategies).

Demographics—Age, sex (0 = female, 1 = male), and legal drinking status (0 = under 21, 1 = 21 or older) were assessed at T1.

Depression/anxiety—The 4-item Patient Health Questionnaire was used at T1 and T2 to assess self-reported depression/anxiety symptoms in the past two weeks (Kroenke et al., 2009). Participants indicated how often during the past two weeks they experienced each symptom according to a 4-point scale (0 = *not at all* to 3 = *nearly every day*). A sum score (Cronbach's $\alpha = .79$) was used for analyses. This scale is a reliable and valid measure of depression and anxiety symptoms among college students (Khubchandani et al., 2016).

Sleep schedule—Four investigator-developed items assessed average sleep duration and the difference in sleep schedule between weekdays and weekends (i.e., social jetlag; Wittmann et al., 2006) in the past two weeks at T1 and T2. Participants identified times that they typically go to bed and wake up on weekdays and weekends (e.g., “During the past two weeks, what time have you usually gone to bed at night during the weekdays?”). Typical weekday and weekend sleep durations were calculated as time elapsed between bed times and wake times. Participants' weighted average sleep duration was calculated by averaging typical weekday sleep duration (multiplied by five days) and weekend sleep duration (multiplied by two days) over a seven-day period. Difference in weekday versus weekend bedtime was calculated as the absolute value after subtracting weekday bedtime from weekend bedtime. The use of comparable items and variable calculations are standard among college student samples (e.g., Singleton & Wolfson, 2009).

Morning/evening preference—The 19-item Morningness-Eveningness Questionnaire was used at T1 to identify participants' circadian preference (Horne & Ostberg, 1976), by assessing tiredness during the day, and preferred times to do diverse activities and to go to sleep. A sum score was utilized for the current analyses (range = 16 – 86; Cronbach's $\alpha = .72$), with a higher score indicating a morning (versus evening) circadian preference.

Insomnia severity—The 7-item Insomnia Severity Index was administered at T1 and T2 to assess a number of insomnia-related behaviors and their severity (Bastien et al., 2001). Participants indicated the extent to which insomnia problems (e.g., difficulty falling asleep, waking up too early) impacted their following day based on a 5-point scale (0 = *None* to 4 = *Very severe*). The scale is a widely-used assessment of insomnia severity (scores 0 – 7 = no clinically significant insomnia, 8 – 14 = sub-threshold insomnia, 15 – 21 = moderate severity clinical insomnia, 22 – 28 = severe clinical insomnia; Buysse et al., 2006; Smith & Wegener, 2003). A sum score (Cronbach's α = .86) was used for analyses.

Pre-sleep arousal—The 16-item Pre-Sleep Arousal Scale was administered at T1 and T2 to assess participants' typical state of arousal before sleep (Nicassio et al., 1985). Participants rated the severity of certain behaviors that may elicit pre-sleep arousal based on a 6-point scale (1 = *not at all* to 5 = *extremely*). A sum score (range = 16 – 18; Cronbach's α = .91) was used for main analyses, with higher scores indicating a larger degree of difficulty in initiating sleep onset.

Alcohol frequency—One of the alcohol consumption items recommended by the National Institute on Alcohol Abuse and Alcoholism (2003) was used to measure alcohol frequency at T1 and T2. This item is widely used in college drinking studies and validated in college samples (e.g., Cranford, McCabe, & Boyd, 2006; Whiteman et al., 2013; Zaso et al., 2016). The timeframe was adapted from the past 12 months to the past 2 months to accommodate the two-month time lapse between T1 and T2 assessments, and response scales were adjusted accordingly (0 = *I did not drink any alcohol in the past 2 months*, 1 = *Once in the past 2 months*, 2 = *Once a month (twice in the past 2 months)*, 3 = *2 to 3 times a month (less than once a week)*, 4 = *Once or twice a week*, 5 = *3 – 4 times a week*, 6 = *5 – 6 times a week*, 7 = *Nearly every day*, 8 = *Every day*).

Negative drinking consequences—The 23-item Rutgers Alcohol Problem Index was administered at T1 and T2 to assess experiences of diverse negative consequences of alcohol consumption during the past two months (White & Labouvie, 1989). The timeframe of this measure was modified from the past 12 months to accommodate the 2-moth timeframe of the current study. Participants indicated the frequency of experiencing alcohol-related problems (e.g., Had a bad time; Had a fight, argument, or bad feelings with a friend; Noticed a change in your personality; Neglected your responsibilities) based on a 7-point scale (0 = *Never in the past 2 months* to 6 = *Six times or more in the past 2 months*; Park et al., 2014). A sum score (Cronbach's α = .87) was used for analyses. The sum score of the 23 items reflects the degree of experience of a range of diverse consequences (White & Labouvie, 1989), which has been demonstrated to map onto a single latent construct of negative drinking consequences (see Arterberry et al., 2016; Cohn et al., 2011).

Data Analytic Strategies

Data analyses were computed using SPSS Version 23. Descriptive statistics and bivariate Pearson correlations were computed (see Table 1). Independent-sample *t*-tests and chi-square difference tests were conducted to compare sleep aid users to non-users regarding all study variables at T1 and T2 (see Table 2). These group comparisons were for descriptive

purposes to characterize the subgroup of college students who self-medicate for sleep versus those who do not without consideration of potential covariates (rather than identifying unique effects of sleep aid use over and above covariates, which were examined in the prospective regression analyses described below).

Using complete data of T1 and T2 (n = 157), regression analyses were utilized to examine short-term prospective relationships between T1 sleep aid use and T2 insomnia symptoms, alcohol frequency, and negative drinking consequences (see Table 3). The respective outcome variable at T1 was included as a covariate in each model (e.g., predicting Time 2 alcohol frequency after controlling for Time 1 alcohol frequency). Sex, legal drinking status, and depression/anxiety symptoms were also included as covariates to control for their associations with insomnia and alcohol behaviors (Galambos et al., 2013; S. R. Kenney, Jones, & Barnett, 2015; Lund et al., 2010). Linear regressions were used for normally distributed outcome variables of insomnia severity (skewness = 0.80; kurtosis = -0.16; range = 0 – 21) and alcohol frequency (skewness = -0.57; kurtosis = 1.35; range = 0 – 7; 0.6% did not drink any alcohol in the past 2 months at T2). Standardized regression coefficients were used for an effect-size measure of predictor variables. Negative binomial regressions were used for the over-dispersed outcome variable of negative drinking consequences (mean = 12.10; variance = 243.54; dispersion parameter = 0.96; skewness = 2.43; kurtosis = 6.35; range = 0 – 77; 9% reported no negative drinking consequences in the past 2 months at T2) as recommended by (Hilbe, 2011), and incidence rate ratios (IRR) were used for an effect-size measure of predictor variables.

Four sets of ancillary analyses were conducted. First, path analyses were conducted with all available data (N = 171) using missing data procedure of full information maximum likelihood estimation with robust standard errors (Graham, Cumsille, & Elek-Fisk, 2003) to assess the degree to which attrition at T2 (n = 14) affected results from regression analyses of complete data (n = 157). Second, to test for distinct patterns of specific substance used for sleep aid, separate regression analyses were conducted for each of alcohol, marijuana, and over-the-counter medications for sleep aid. Third, in order to rule out polysubstance sleep aid use as a unique predictor of prospective sleep and alcohol use outcomes, regression analyses were repeated using number of sleep aids used (i.e., none, one, two, or all three) rather than dichotomized sleep aid use as the independent variable. Fourth, to test for the effect of sleep aid use initiation at T2 on changes in insomnia symptoms, alcohol frequency, and negative consequences over time, regression analyses were repeated using initiation of sleep aid use at T2 (instead of sleep aid use at T1) as the independent variable.

Results

Descriptive Analyses

Descriptive statistics and bivariate Pearson correlation coefficients of study variables are reported in Table 1. Participants reported 8 hours of sleep on average, sub-threshold insomnia severity, and drinking alcohol 1-2 times per week at T1. At T1 and T2 assessments, 23 – 25% reported using any alcohol, marijuana, and/or over-the-counter medication for sleep aid in the past two weeks. Sleep aid use was significantly associated with alcohol frequency at Time 1 (r = .21, p = .01) but not at Time 2 (r = .12, p = .15),

demonstrating a small to medium sized association at both time points. Regarding T1 alcohol variables, T1 sleep aid users reported drinking more frequently and experiencing more negative drinking consequences than non-users.

Regarding specific substance used as sleep aid (data not shown in tables), 7-10% reported using alcohol, 14-15% reported using marijuana, and 14-16% reported using over-the-counter medications for sleep aid at T1 and T2 assessments. The average frequencies of all the three substances among users were 1-2 nights per week at T1 and T2. Of all participants, 19-21% reported using only one substance for sleep aid in the past two weeks, while 3-6% used two substances and 3-4% used all three. Among sleep aid users at T1 (n = 43), 63% also endorsed sleep aid use at T2. Out of non-sleep aid users at T1 (n = 128), 19% endorsed initiating sleep aid use at T2. Initiation of sleep aid use at T2 was not associated with insomnia symptoms at T1 (r = -.06, p = .43) or at T2 (r = -.04, p = .63) or any sleep-related characteristics at T1 (including morning/evening preference, r = -.04, p = .58; sleep duration, r = .07, p = .42).

Group Comparisons between Sleep Aid Users versus Non-Users

Table 2 presents results from independent-sample t-tests and chi-square tests to compare T1 sleep aid users and non-users. Regarding demographics, T1 sleep aid users were more likely to be male, older, and at legal drinking age. Regarding T1 sleep variables, sleep aid users also reported higher insomnia severity and pre-sleep arousal compared to non-users, but no group differences were found in sleep duration, difference in weekday/weekend bedtime, or morning/evening preference. Regarding T1 alcohol variables, T1 sleep aid users reported drinking more frequently and experiencing more negative drinking consequences than non-users.

Regarding T2 sleep and alcohol variables, T1 sleep aid users reported more negative drinking consequences and higher insomnia severity at T2 than non-users. Groups did not differ in insomnia diurnal impact, pre-sleep arousal, alcohol frequency, or heavy-drinking days at T2.

Prospective Analyses

Results from regression analyses predicting insomnia symptoms, alcohol frequency and negative drinking consequences at T2 from sleep aid use at T1 are presented in Table 3. Sleep aid use at T1 was significantly and positively associated with T2 negative drinking consequences, even after accounting for T1 negative drinking consequences (i.e., changes in negative consequences over time) as well as sex, legal drinking age, and depression/anxiety symptoms. However, sleep aid use at T1 was not significantly associated with T2 insomnia severity or alcohol frequency after controlling for covariates.

Ancillary Analyses

Path analyses of all available data using missing data procedure (N = 171) yielded the same patterns of significance as aforementioned linear and negative binomial regression analyses of complete data only (n = 157), suggesting that attrition at T2 (n = 14) was not likely to have affected overall findings.

Analyses of specific substance use for sleep aid showed that both alcohol (IRR = 2.80, $p = .006$) and marijuana (IRR = 2.01, $p = .004$) sleep aid use at T1 were significantly and positively associated with changes in negative drinking consequences, but not in insomnia symptoms or alcohol frequency. Time 1 over-the-counter medication use was not associated with any T2 outcomes at $p < .05$.

Analyses using number of sleep aids used (0 to 3) yielded results consistent with results from analyses using a dichotomized sleep aid use variable reported herein, indicating that the number of sleep aid use at T1 was positively associated with changes in negative drinking consequences, IRR = 1.35, $p = .01$, but not in insomnia symptoms and alcohol frequency at $p < .05$.

Analyses using the initiation of self-medication for sleep at T2 also yielded results consistent with results from analyses using T1 sleep aid use, indicating that the initiation of self-medication at Time 1 was significantly and positively associated with changes in negative drinking consequences (IRR = 2.00, $p = .003$), but not in insomnia symptoms or alcohol frequency at $p < .05$.

Discussion

This is the first prospective evaluation of sleep aid use and associated outcomes, broadening the knowledge of co-occurring sleep problems and substance use among college students. Of the current sample, 25% endorsed sleep aid use (i.e., alcohol, marijuana, and/or over-the-counter medication), suggesting that a substantial group of college students self-medicate to help sleep. Compared to non-users, sleep aid users were more likely to be male, older, at legal drinking age, and reported higher levels of pre-sleep arousal, insomnia severity, alcohol frequency, and negative drinking consequences. Sleep aid use was associated with an increase in negative drinking consequences, but not, in alcohol frequency or negative drinking consequences over a short period of time. Further, initiation into self-medication for sleep was also associated with an increase in negative drinking consequences (but not in improvement in insomnia symptoms) over a two month period. Collectively, these findings suggest that students self-medicating for sleep experience more adverse drinking consequences over time without the intended benefit of improved sleep.

Over-the-counter medications (14 – 16%) and marijuana (14 – 15%) were more popular choices of sleep aid than alcohol (7 – 10%), all of which were used 1-2 nights per week. A large study of college students (Taylor & Bramoweth, 2010) found prevalence and frequency of alcohol use for sleep aid relatively consistent with the current study, though over-the-counter medication use for sleep aid was notably less prevalent (2%) but more frequent (3 – 4 nights per week) than the current sample. Little is known about the frequency of over-the-counter medication use for sleep among college students, which makes it difficult to gauge potential reasons of this drastic difference in over-the-counter medication use for sleep (while a rate of alcohol use for sleep is comparable). Future multi-campus studies of larger sample sizes should assess differences in diverse sleep aid preferences as a function of student demographics and campus characteristics.

Importantly, 7 – 9% of students endorsed utilizing multiple substances for sleep aid during the past two weeks. When consumed in combination, substances such as alcohol and marijuana have an additive effect on behavioral impairment (Chait et al., 1994). Further, polysubstance-using young adults are more likely to experience alcohol-related harm such as blackouts and acute memory loss (Hingson et al., 2016). Therefore, these students potentially represent a group at increased risk of dangerous substance combinations. However, given the current study design, it cannot be determined whether students used multiple substances for sleep aid on a single evening. In order to investigate the prevalence and potential risk associated poly-substance sleep aid use, future studies should assess daily patterns of substance use, sleep aid selection, and negative consequences of substance use.

Several individual characteristics associated with this pattern of substance use (i.e., correlates) were identified. Sleep aid use was more common among men, consistent with extant findings (Taylor & Bramoweth, 2010). Sleep aid use was also more prevalent among older students and at legal drinking age. These unexpected results, which are not in line with findings in college drinking, may be due to the fact that students at legal drinking age have better access to alcohol than underage students, and/or that male and older students are more likely to be experienced alcohol/substance users, and therefore more readily opt to use alcohol/substance for sleep. This is consistent with our finding of a positive association between sleep aid use and concurrent alcohol frequency, suggesting that sleep aid users may use more substances for sleep mainly because they use more substances for a wider array of reasons in general. Extant literature has demonstrated that risky drinking college students endorse higher levels of diverse drinking motives in general (e.g., coping and enhancement) compared to low-moderate-risky drinking peers (Littlefield et al., 2013; Merrill et al., 2016). However, substance use for sleep motives has historically not been included in widely used alcohol/substance use motives assessments, which make it difficult to assess unique effects of substance use for sleep over and above substance use for other reasons. Also, sleep aid users concurrently endorsed significantly more insomnia symptoms and pre-sleep arousal compared to non-users, but did not differ in average sleep duration, inconsistent sleep scheduling, or circadian preference. Students may use sleep aids to manage pre-sleep arousal and insomnia symptoms (which may be improved by the sedative effects of sleep aids in a short term), but not sleep duration and inconsistent sleep scheduling (which are largely controlled by external factors such as class schedules and social events). Interestingly, sleep aid use was not concurrently associated with depression/anxiety symptoms, suggesting that sleep aid use is more driven by pre-sleep factors and heavy and problematic substance use than mood-related problems. Overall, these findings suggest that some correlates of sleep aid use may differ from correlates of poor sleep (e.g., depression/anxiety, sleep duration, inconsistent sleep scheduling, or circadian preference) and of drinking behaviors (e.g., age or legal drinking age) among college students, albeit with some overlaps (e.g., male sex for drinking, and pre-sleep arousal for insomnia). Further research is necessary to replicate these novel findings and investigate a more comprehensive set of correlates and proximal antecedents to sleep aid use.

The current findings also have significant implications for potential adverse consequences of sleep aid use over time. Sleep aid use was associated with increases in subsequent negative drinking consequences, but not with frequent drinking patterns. Despite the hypnotic effects

of alcohol, alcohol use before bed negatively impacts sleep architecture, and tolerance to the hypnotic effects of alcohol develops rapidly (Roehrs et al., 1999), at which point associated functional impairments likely become exacerbated. Therefore, although students do not appear to increase the frequency of alcohol consumption over time, they may become more susceptible to the accompanying negative consequences (such as impaired school or work performance) due to repeated sleep aid use. Future research should examine the nuanced, day-to-day changes in the experiences of sleep, substance use, and associated consequences.

Contrary to *a priori* hypotheses, self-medication for sleep was not associated with changes in insomnia symptoms over this short period of time. Despite well-documented acute sleep disturbance following substance use for sleep aid (Katayose et al., 2012; Roehrs et al., 1991; Williams, MacLean, & Cairns, 1983), there appears to be no subjective detriment to sleep over a two-month period in college students. However, current findings do not rule out the possibility of long-term consequences of occasional or chronic sleep aid use among college students. Prospective longitudinal studies of a longer time lapse between assessments are needed to elucidate long-term sleep-related outcomes. Further, sleep aid users at T1 may have developed problematic sleep aid use habits prior to T1, and therefore the negative feedback loop outlined by Brower (2003) may have already begun, where self-medication for sleep leads to exacerbated insomnia symptoms and therefore increased self-medication over time. However, ancillary analysis results also provide preliminary evidence for the exacerbation of negative drinking consequences (but not of insomnia symptoms or alcohol frequency) from T1 to T2 as a result of initiation into sleep aid use at T2. Multi-wave longitudinal studies with younger or inexperienced samples (e.g., adolescents) are needed to track sleep aid users from the point of initiation would help to resolve whether sleep aid use plays a unique role in the trajectory toward developing risky substance use habits or dependence.

Limitations of the current study must be considered. Although this prospective design allows for a better assessment of temporal relationships than cross-sectional designs, the current study remains correlational in nature and thus causal inferences are speculative. Also, findings of the current study are based on subjective reports of sleep. Future investigators might consider the incorporation of objective sleep measures, such as actigraphy and polysomnography, which may be affected by sleep aid use. Also, the current analyses of self-medication for sleep did not account for potential confounding effects of using prescription medications for sleep (e.g., zolpidem, antidepressants, and benzodiazepines) that may be associated with sleep and alcohol/substance use behaviors in complex ways. Another limitation is that items used to assess sleep aid use cannot isolate timing of use (i.e., initial sleep onset versus sleep re-initiation) or primary motivation for use (i.e., sleep aid versus other substance use reasons such as social, coping, and mood enhancement). These items also cannot determine a specific sleep problem for which individuals self-medicate (insomnia symptoms, other sleep-related disorders such as Delayed Sleep Phase Syndrome or obstructive sleep apnea, nightmares etc.). Future studies should consider revising the phrasing of these items to explicitly assess timing of sleep aid use, as well as including additional items to investigate the primary motives of substance use before bed. Event-level investigations of motivation for substance use are necessary to isolate self-medication for sleep from alcohol/substance use for other purposes and to characterize the nuanced and

dynamic associations among sleep, substance use, and associated consequences. Also, data were drawn from a predominantly white, first-year sample of students enrolled in a northeastern private university. Because substance use and sleep patterns vary by diverse individual and school characteristics (Johnston et al., 2015; Lichstein et al., 2004), these sample characteristics should be considered in interpreting the findings of this study. Replication with larger and more representative samples is necessary for generalization of our findings, and further multi-campus research may help to characterize how different school-level characteristics may be associated with distinct patterns of sleep aid use and its consequences over time. Additionally, students were eligible for participation only if they had consumed alcohol in the last 30 days; thus, the current findings might not be applicable to students who abstain from alcohol use (such as students utilizing exclusively marijuana and/or over-the-counter medications for sleep aid). In general college samples, approximately 80% of student endorse having used alcohol (Johnston et al., 2015). Further, mean levels of insomnia severity and alcohol use for sleep aid found in the current study were remarkably similar to prior studies of college students (Gress-Smith et al., 2015; Taylor & Bramoweth, 2010), suggesting that this sampling bias may not be substantial. Nonetheless, these sample characteristics should be considered when interpreting the current findings, and replication with more representative samples is necessary for generalization.

Despite the aforementioned limitations, the current findings present a significant step toward characterizing sleep aid use and its association with exacerbated negative drinking consequences over time. The current findings highlight the potential role of sleep aid use in the maintenance of co-occurring sleep problems and exacerbated substance problems among college students.

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Table 1

Descriptive Statistics and Bivariate Correlations among Study Variables at Time 1 (T1) and Time 2 (T2)

		r													
Study Variables	***M (SD) or %***	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**
1. Sleep Aid Use at T1	25%	—													
2. Male Sex (vs. Female)	32%	**.16**	—												
3. Age (years) at T1	18.95 (1.35)	**.23**	**.19**	—											
4. Legal Drinking Status at T1	12%	**.19**	.09	**.78**	—										
5. Depression/anxiety at T1	3.65 (2.71)	.10	-.06	-.02	-.04	—									
6. Average Sleep Duration (hours) at T1	7.97 (1.19)	-.10	.01	-.15	-.11	**.16**	—								
7. Difference WE/WD bedtime (hours) at T1	2.14 (1.12)	-.03	.03	.05	.11	-.13	.10	—							
8. Morning/Evening Preference at T1	46.81 (7.48)	-.09	-.01	**.17**	**.23**	-.08	-.05	.13	—						
9. Insomnia Severity at T1	8.98 (5.48)	**.20**	-.13	.05	.11	**.44**	**-.29**	-.09	-.06	—					
10. Pre-Sleep Arousal at T1	33.36 (10.90)	**.18**	**-.16**	-.12	-.07	**.60**	**-.17**	-.05	-.12	**.66**	—				
11. Alcohol Frequency at T1	3.88 (0.98)	**.21**	**.18**	.04	.05	-.05	.05	.12	-.07	.03	.00	—			
12. Negative Drinking Consequences at T1	11.42 (11.61)	**.30**	.06	-.05	.00	**.35**	-.13	.06	-.03	**.34**	**.39**	**.34**	—		
13. Insomnia Severity at T2	7.39 (5.30)	**.20**	-.03	.00	.03	**.34**	-.13	.03	-.12	**.55**	**.49**	**.20**	**.28**	—	
14. Alcohol Frequency at T2	3.71 (1.06)	.12	**.06**	-.02	-.02	-.11	.10	.12	-.12	-.03	-.04	**.55**	**.16**	.02	—
15. Negative Drinking Consequences at T2	12.10 (15.61)	**.30**	.13	-.10	-.12	**.24**	-.10	-.01	**-.20**	**.18**	**.30**	**.21**	**.40**	**.42**	**.18**

Note. N = 171. WE/WD = weekend versus weekday. Significant group differences at $p < .05$ are highlighted in bold font.

Table 2

Means or Percentages of Study Variables as a Function of Self-Medication for Sleep at Time 1

Variable	Non-Sleep Aid Users at Time 1 (75%; *n* = 128)	Sleep Aid Users at Time 1 (25%; *n* = 43)	Group Comparison Test Statistics
Time 1 Variables (*N* = 171)			
Male Sex (vs. Female)	**27%**	**44%**	**$\chi^2(1) = 4.23^{*}$**
Age (years)	**18.77 (1.03)**	**19.49 (1.96)**	**$t(169) = 2.29^{*}$**
Legal Drinking Status	**9%**	**23%**	**$\chi^2(1) = 6.42^{*}$**
Depression/anxiety	3.50 (2.74)	4.09 (2.65)	$t(169) = 1.24$
Average Sleep Duration (hours)	8.05 (0.98)	7.76 (1.68)	$t(163) = -1.04$
Difference WE/WD bedtime (hours)	2.17 (1.08)	2.08 (1.22)	$t(163) = -0.43$
Morning/Evening Preference	47.18 (7.53)	45.70 (7.31)	$t(169) = -1.13$
Insomnia Severity	**8.35 (5.04)**	**10.84 (6.31)**	**$t(169) = 2.62^{**}$**
Pre-Sleep Arousal	**32.23 (9.99)**	**36.72 (12.79)**	**$t(169) = 2.37^{*}$**
Alcohol Frequency	**3.76 (0.95)**	**4.23 (1.00)**	**$t(169) = 2.79^{**}$**
Negative Drinking Consequences	**9.39 (9.74)**	**17.49 (14.45)**	**$t(169) = 3.42^{**}$**
Time 2 Variables (*N* = 157)			
Sleep Aid Users	78% (*n* = 122)	22% (*n* = 35)	—
Insomnia Severity	**6.82 (4.75)**	**9.40 (6.58)**	**$t(155) = 2.16^{*}$**
Alcohol Frequency	3.65 (1.05)	3.94 (1.08)	$t(155) = 1.46$
Negative Drinking Consequences	**9.63 (12.92)**	**20.7 (20.62)**	**$t(155) = 3.02^{**}$**

Note. WE/WD = weekend versus weekday. Significant group differences at $p < .05$ are highlighted in bold font.

$^{*}p < .05$.

$^{**}p < .01$.

Table 3

Regression Analyses Examining Effect of Sleep Aid Use at Time 1 on Sleep and Alcohol Outcomes at Time 2

	Time 2 Outcomes		
	Insomnia Severity	**Alcohol Frequency**	**Negative Drinking Consequences**
Time 1 Sleep Aid Use	.11	-.01	**1.63** *
Time 1 Outcome	**.48** ***	**.54** ***	**1.04** ***
Male Sex	.03	.08	**1.57** *
Time 1 Depression/anxiety	.13	-.09	**1.07** *
Time 1 Legal Drinking Status	-.04	-.04	**0.56** *

Note. N = 157. Sleep Aid Use = the use of any alcohol, marijuana, and/or over-the-counter medication for sleep aid in the past two weeks. Results of negative drinking consequences are based on a negative binomial regression, and incidence rate ratios are reported; results from all other variables are based on linear regressions, standardized coefficients are reported. Significant coefficients at $p < .05$ are highlighted in bold font.

* $p < .05$.

*** $p < .001$.