HHS Public Access

Author manuscript

J Adolesc Health. Author manuscript; available in PMC 2018 February 01.

Published in final edited form as:

J Adolesc Health. 2017 February ; 60(2): 154–160. doi:10.1016/j.jadohealth.2016.08.019.

The Prospective Association between Sleep and Initiation of Substance Use in Young Adolescents

Mary Beth Miller, Ph.D.[a], **Tim Janssen, Ph.D.**[a], and **Kristina M. Jackson, Ph.D.**[a]

[a]Center for Alcohol and Addiction Studies, Department of Behavioral and Social Sciences, Brown University School of Public Health, Box G-S121-5, Providence, RI 02912

Abstract

Objective—This study aimed to determine the unique utility of poor sleep health in predicting the onset of substance use in adolescents.

Method—Middle school students (N=829, M_{age}=12.6y, 52% female, 73% White) who participated in an ongoing prospective study of alcohol onset and progression completed a set of web-based health behavior surveys over a four-year period, with recruitment beginning in 2009. Surveys included assessments of alcohol, cigarette, and marijuana use as well as sleep duration, bedtime delay (from weekdays to weekends), and daytime sleepiness. Data were analyzed using discrete-time Cox Proportional Hazards regression.

Results—Controlling for participant age at sleep assessment, gender, and internalizing and externalizing behaviors, shorter sleep duration and greater daytime sleepiness in Year 1 were associated with increased odds of having had a full drink of alcohol, having engaged in heavy episodic drinking, and having experienced alcohol-related consequences by Year 4. Shorter sleep duration was also associated with increased odds of marijuana use by Year 4. No sleep parameter was uniquely associated with increased odds of cigarette use (first puff or first full cigarette). Effects were evident for both males and females. Age at sleep assessment did not moderate effects.

Conclusion—Sleep health is a prospective predictor of the onset of alcohol and marijuana use among adolescents. Increased efforts to prevent and intervene in sleep problems among adolescents are warranted.

Keywords

alcohol; tobacco; marijuana; sleep health; bedtime

Poor sleep health and heavy substance use are public health concerns that tend to co-occur among adolescents and young adults.[1,2] Both of these behaviors have been associated with a host of negative consequences, including impaired immune functioning, motor vehicle

Correspondence concerning this article should be addressed to Mary Beth Miller, Center for Alcohol and Addiction Studies, Box G-S121-5, Providence, RI, 02912. Phone: 573.619.1538. Fax: 401.863.6647. millerme04@gmail.com.

Implications and Contribution

This study suggests that poor sleep health is a prospective predictor of alcohol and marijuana use in adolescents, independent of internalizing and externalizing behaviors. The independent effect of sleep on substance use suggests that interventions targeting the types of sleep problems that are common in adolescence are needed.

accidents, depression, and suicidal ideation.[3,4] Yet little is known about the direction and degree of prospective associations between these health behaviors. Research delineating the temporal precedence and significance of the association between poor sleep and substance use is needed to inform prevention and intervention efforts.

Research has documented longitudinal, bidirectional relationships between sleep and substance use in adolescents. Alcohol and illicit drug use has been associated prospectively with delayed bedtimes[1,5] and disturbed sleep physiology.[6–8] Conversely, poor sleep health (defined as <9 hours per night for adolescents, inconsistent timing of sleep, trouble falling/staying asleep, daytime sleepiness, or poor subjective sleep quality[9]) prospectively predicts the likelihood and frequency of cigarette, marijuana, and alcohol use in adolescents and young adults.[5,10–12] Poor sleep health has also been associated with increased likelihood of binge drinking[11] and may compound risk for alcohol-related consequences.[11,13] However, few studies have examined the impact of sleep on the initiation of substance use in youth who are substance-naïve. Those that have examined this association have been limited by parent report of substance use,[14,15] recruitment of high-risk participants,[12,14,15] and failure to control for psychiatric symptoms that may impact sleep and substance use.[12,13]

Without examining sleep in individuals who are substance-naïve, the directionality of the association between poor sleep health and substance use cannot be determined. Evidence suggests that substance use creates disturbances in sleep health,[6,8] which may then perpetuate substance use and related risk. However, it could also be that poor sleep health confers unique and independent risk for the onset of substance use and problems. In the case of the latter, increased efforts to prevent and treat sleep problems in childhood and early adolescence may be warranted.

The limited focus on sleep relative to substance use research in adolescents may stem in part from the field's tendency to view difficulty falling/staying asleep as a symptom of an underlying psychiatric disorder.[16] Psychiatric disorders have been linked to adolescent substance use in a number of studies,[17] and individuals with psychiatric disorders tend to demonstrate signs of disturbed sleep.[18] Thus, the association between sleep and substance use may be attributable to their respective associations with psychopathology. Few studies have examined the association between adolescent sleep and substance use in the context of psychiatric symptoms, and the results of these studies have been mixed. Specifically, one study found that the cross-sectional association between sleep and substance use is attributable in part to internalizing/externalizing behaviors,[19] while others found that controlling for these behaviors did not weaken the prospective impact of sleep on substance use.[2,14,15]

This study extends previous research[11–15] by determining the unique utility of poor sleep health in predicting the onset of self-reported substance use in adolescents, after accounting for internalizing and externalizing behaviors. We hypothesized that shorter sleep duration, greater discrepancy between weekday and weekend bedtimes (bedtime delay), and greater daytime sleepiness would be associated with greater probability of alcohol, cigarette, and marijuana use. We also investigated the moderating effect of gender, as gender differences in these associations have been found inconsistently in previous studies.[2,11,15]

Method

Participants

Data were collected between 2009 and 2015 from a sample of adolescents in an ongoing prospective study examining the extent to which individual and contextual risk factors explain initiation of and progression through drinking milestones.[20] Five cohorts of adolescents (N=1023) from six schools in urban, suburban, and rural Rhode Island were enrolled every six months from 2009 to 2011. Of the 1023 respondents, 992 (97%) contributed follow-up data within the four-year assessment interval, 923 (90%) provided data on sleep parameters, and 829 (81%) were accompanied by parent report data on internalizing/externalizing behaviors. Thus, the current sample consisted of 829 adolescents aged 12 to 16 years (52% female, 15% non-White, 12% Hispanic; see Table 1). Participants who were excluded from and included in analyses did not differ in terms of gender [$t(1021)=0.20$, $p=.84$] or ethnicity [$t(1021)=-0.22$, $p=.82$]. Those excluded were significantly older than those included [$t(1021)=2.87$, $p=.004$] and more likely to report non-White race [$t(260)= -2.68$, $p=.01$].

Procedure

Interested youth whose parents provided written informed consent attended a two-hour orientation session, during which they provided assent, and completed the 45-minute baseline survey (Time 1) in a private location. Follow-up assessments, which were administered in waves based on school cohort, were conducted using web-based surveys. These assessments took place every six months for a period of two years (Times 2–5); at three-year follow-up (Time 6); and at quarterly intervals thereafter, from which we coded a four-year follow-up (Time 7). In the first two years of the study, participants also completed brief monthly surveys assessing substance use; if participants denied substance use in the past 30 days, they completed filler items regarding a variety of health behaviors, one of which was sleep. Sleep parameters were assessed a maximum of two times, depending on whether or not the participant had already engaged in substance use. The first sleep assessment was conducted in Month 1 or Month 7 (depending on school cohort); for those who did not complete the first sleep assessment (n=34, 4%), we used data from their second sleep assessment, which was conducted in Month 13 or 19. Because the discrepancy in timing of these assessments led to age differences between participants, all analyses controlled for age at first sleep assessment. Participants were compensated with a $25 gift card for the orientation session, a $20 gift card for each follow-up survey, and $10 cash for each monthly survey. Response rates ranged from 92% (Time 2) to 79% (Time 7).

One self-selected parent (86% biological mother) provided data on participants' internalizing and externalizing behaviors. These assessments were collected via mailed, 30-minute, paper-and-pencil surveys at baseline and one-year post-baseline. Parents received a $30 gift card for survey completion. The university's Institutional Review Board approved all procedures.

Measures

Demographic information—Participants provided information regarding gender, age, race, and ethnicity.

Sleep parameters—Items from the School Sleep Habits Survey[21] were used to calculate sleep duration, bedtime delay, and daytime sleepiness. Participants indicated what times they had usually gone to bed and woken up on school days and weekends in the last two weeks. Responses were transformed and averaged to calculate *sleep duration*. We subtracted average bedtime on school days from average bedtime on weekends to calculate *bedtime delay*. Participants also indicated to what extent they feel sleepy or struggle to stay awake during the day (*daytime sleepiness*) on a scale from 0 (*no problem at all*) to 4 (*a very big problem*). These select items were chosen because they are believed to reflect the sleep duration, sleep timing, and daytime sleepiness components of sleep health.[9]

Onset of alcohol use—Drinking and drug use milestones were assessed at each follow-up of the study, using measures designed based on NIAAA recommendations.[22] Participants indicated if they had ever had a full drink of alcohol (*onset of full drink*) and if they had ever had 3+ drinks of alcohol in one sitting (*onset of heavy episodic drinking*). After the third year of the study, participants indicated the greatest number of drinks they had ever had in one sitting; responses ≥ 3 were coded as indicating onset of heavy episodic drinking.

Onset of alcohol-related consequences—*Onset of alcohol-related consequences* was assessed using an 18-item version of the Rutgers Alcohol Problems Index (RAPI).[23] Participants indicated how often in the past year they had experienced outcomes such as not being "able to do your homework or study for a test" or having "a bad time" due to drinking. Response options ranged from 0 (*none*) to 3 (*more than 5 times*). The RAPI has been validated among youth age 13 to 19 years old.[24] At each follow-up, participants were coded as having experienced onset of alcohol consequences if they endorsed any RAPI item.

Onset of smoking—At each follow-up, participants indicated if they had ever tried or experimented with cigarette smoking, "even a few puffs/drags" (*onset of cigarette puffed*) or if they had ever smoked a whole cigarette (*onset of full cigarette use*).[25]

Onset of marijuana use—Participants also indicated at each follow-up if they had ever used marijuana (pot, hash, hash oil, etc.).

Internalizing/externalizing behaviors—Parents completed 110 items from the Child Behavior Checklist,[26] which assessed their child's behavior in the past six months on a three- point scale: 0 (*not true, as far as you know*), 1 (*somewhat or sometimes true*), or 2 (*very true or often true*). Internalizing problem scores (range 0–93; α=.87) were calculated as the sum of the withdrawn/depressed (8 items), somatic complaints (11 items), and anxious/depressed scales (12 items). Externalizing problem scores (range 0–105; α=.91) were calculated as the sum of the aggressive behavior (18 items) and rule-breaking scales (17 items).

Social anxiety—Participants completed the Social Anxiety Subscale of the Multidimensional Anxiety Scale for Children,[27] which assesses humiliation/rejection (*M*=4 items) and performance fears (*M*=3 items). Response options ranged from 0 (*never true about me*) to 3 (*often true about me*). Social anxiety was calculated as the mean of the two sub-scales (α=.85).

Data Screening and Analysis

To identify potential sources of multicollinearity, we examined bivariate correlations between sleep parameters and potential covariates (see Table 2). We used Spearman correlations because sleep variables were highly kurtotic. We then used discrete-time Cox Proportional Hazards regression[28] to investigate whether each sleep parameter (sleep duration, bedtime delay, or daytime sleepiness) prospectively predicted each substance use outcome (first full drink, heavy drinking episode, alcohol-related consequence, puff of a cigarette, full cigarette smoked, or use of marijuana). In this analysis, the proportional hazard of event occurrence, given that the event did not occur at the previous time point, is estimated based on the observed outcomes and the values of predictors. In this study, the time interval between each assessment of the outcome was half a year, with assessments ranging from 12.0 to 19.5 years, yielding 15 discrete time points at which either the onset or final censoring event was allowed to occur.

We first examined unadjusted models, in which only the sleep parameter and age at sleep assessment were modeled as predictors of substance use outcomes. Because the significance of unadjusted and adjusted models was highly consistent, we present analyses that controlled for confounding variables. In each model, covariates (age at sleep assessment, gender, race, ethnicity, internalizing and externalizing problems, and social anxiety) were entered in Step 1, and sleep parameter was entered in Step 2. Deviance chi-square difference tests were used to examine the significance of model change from Step 1 to Step 2. We then ran exploratory models, entering all three sleep parameters simultaneously in Step 2, to determine the relative importance of each sleep parameter. Finally, we tested whether there were interaction effects between sleep parameters and either gender or age at sleep assessment, using standardized values to examine interaction effects.

Results

Descriptive Statistics

Descriptive data are depicted in Table 1. On average, participants reported sleeping 8.8 hours per night (*SD*=1.5; $M_{weekday}$=8.4, $M_{weekend}$=9.6). Approximately half of participants reported having ‘a little problem’ with feeling sleepy or struggling to stay awake during the day (49%), while fewer reported ‘no problem at all’ (38%), ‘more than a little problem’ (10%), ‘a big problem’ (2%), or ‘a very big problem’ (1%). Changes in use of alcohol, cigarettes, and marijuana are depicted in Table 1.

Main Effects

Table 3 depicts the main effects of sleep parameters on onset of alcohol use, controlling for age at sleep assessment, gender, and internalizing and externalizing behaviors. Race and

social anxiety were not significant predictors of any alcohol use outcome and, therefore, were removed from these models. In the prediction of *first full drink* of alcohol, shorter sleep duration and greater daytime sleepiness were associated with increased odds of having had a full drink. Specifically, for every hour of additional sleep, the probability of having had a full drink decreased by approximately 15%; for every one unit increase in subjective daytime sleepiness, the probability increased by approximately 26%. This pattern of outcomes was consistent in models predicting *heavy episodic drinking* and *alcohol-related consequences* (see Table 3).[1]

Table 4 depicts the main effects of sleep parameters on onset of smoking and marijuana use, controlling for the same covariates in alcohol models in addition to race. None of the sleep parameters were significant predictors of *first puff of a cigarette* or *first full cigarette smoked*. However, the association between daytime sleepiness and onset of full cigarette was not consistent over time, violating the assumption of proportional hazards. In the time-varying model, the direction of the time-varying effect suggested that the effect of daytime sleepiness on the onset of cigarette use became protective over time, while non-significant overall. Shorter sleep duration was associated with increased odds of *onset of marijuana use*, with odds increasing by approximately 14% with every additional hour of sleep lost.

To determine if results would change when multiple sleep parameters were included in the same model, we entered all sleep parameters simultaneously as predictors of substance use outcomes in Step 2 (see Table 5). Results indicated that shorter sleep duration and greater daytime sleepiness remained independently predictive of alcohol use outcomes when both were included in the model; however, sleep duration was no longer a significant predictor of marijuana use.

Moderated Effects

Gender and age at sleep assessment were examined as moderators of the associations between sleep parameters and substance use outcomes. One significant interaction emerged; specifically, longer sleep duration was more strongly protective against the onset of marijuana use for women than men (interaction effect=0.39, p=.02). There were no other significant interactions between sleep parameters and either gender (effects=0.73–1.41, ps=.20–.99) or age at sleep assessment (effects=0.80–1.24, ps=.09–.99). Because we found no evidence of a pattern in interaction outcomes, we urge cautious interpretation of this finding.

Discussion

This study adds to a growing literature indicating that sleep health in early adolescence prospectively predicts the onset of adolescent substance use, and extends this literature by documenting that it does so above and beyond internalizing and externalizing behaviors. Shorter sleep duration and greater daytime sleepiness predicted the onset of alcohol use, heavy drinking, and alcohol-related consequences; and shorter sleep duration predicted the onset of marijuana use. These findings were evident in male and female participants and did not vary based on the participant's age at sleep assessment. Thus, sleep health may play a unique role in the onset of alcohol and marijuana use in male and female adolescents, nearly half of whom report some problem staying awake and alert throughout the day.

Sleep duration and daytime sleepiness predicted each alcohol use outcome assessed. This is consistent with previous research indicating that various aspects of poor sleep health predict subsequent alcohol use among adolescents.[5,10,12,13,15] The fact that sleep parameters predicted substance use onset after controlling for psychiatric symptoms suggests that this association is not due entirely to psychopathology; thus, the mechanisms underlying these associations remain to be determined. There is some evidence that deficits in working memory and emotion regulation may be involved. Parent- and child-reported difficulty falling/staying asleep and overtiredness have been associated with deficits in working memory, which in turn predict substance use among adolescents.[29] Five days of 'mild' sleep deprivation has also been associated with immediate impairments in adolescents' abilities to regulate emotions,[30] which has been associated with problematic substance use.[31,32] Future research may examine these attributes as mediators of poor sleep health's effect on alcohol use in adolescents.

The finding that shorter sleep duration predicts the onset of marijuana use is also consistent with previous research.[12,15] Although the manner by which sleep health influences marijuana use is also unclear, similar mechanisms may underlie sleep's impact on use of multiple substances. For example, poor response inhibition in adolescence has been found to mediate the effect of overtiredness in childhood on illicit drug use in young adulthood.[11,15] Thus, poor sleep health may be associated with deficits in executive functioning (e.g., working memory, impulse control) that result in increased risk of alcohol and other drug use in adolescents.

Our findings also suggest that the association between shorter sleep duration and onset of marijuana use may be stronger for females than males. The number of interactions we examined (n=36) inflates the probability of Type I error in these analyses; therefore, this result should be replicated before a systematic interaction by gender is concluded. That being said, there is evidence of puberty-dependent changes in sleep that may impact substance use outcomes; specifically, male and female adolescents who are more mature report delayed circadian phase preferences and more sleep problems, which are associated with increased alcohol use.[2] Given gender differences in the onset of puberty, future studies may examine the extent to which male and female adolescents' sleep patterns differ as a function of sexual maturity and the impact of these differences on subsequent substance use.

In contrast to findings for alcohol and marijuana use outcomes, none of the sleep health parameters assessed in this study were associated with greater odds of first tobacco use. However, findings also indicated that the association between some sleep parameters (i.e., daytime sleepiness) and cigarette use is not linear over time. Rather, daytime sleepiness was associated with sharper increases in the hazard of cigarette use at younger as opposed to older ages, indicating that sleep may have a stronger influence on cigarette use in younger samples. It is also possible that non-significant findings are the result of a floor effect, as only 8% of participants had smoked a full cigarette at four-year follow-up.

Given the prevalence and significance of poor sleep health among adolescents, increased efforts to prevent and intervene in sleep problems for adolescents are warranted. The majority of adolescents do not receive the 9 to 9.3 hours of sleep they need per night.[33]

Moreover, changes in puberty and shifts in melatonin secretion tend to result in delayed circadian sleep phase, in which adolescents experience sleepiness later at night and wake up naturally later in the morning. Not surprisingly, then, circadian rhythm disorders (delayed sleep phase) and insomnia are among the most common sleep disorders diagnosed in adolescents.[33] Efforts have been made to design and evaluate behavioral interventions for these disorders among adolescents,[34] and preliminary evidence suggests that insomnia treatment for adolescents improves both sleep outcomes and executive function.[35,36] However, few studies have examined the efficacy of sleep interventions for adolescents who use or are at high risk for using alcohol and other drugs.[37] Given the significance of sleep health in predicting the onset of substance use in adolescents, research is needed to determine the efficacy of sleep interventions in delaying or preventing the onset of substance use in this population.

Data for this study were derived from a large, diverse sample of adolescents who had not yet initiated substance use. While this sample is ideal for studying predictors of substance use onset, the results of this study should be interpreted with limitations in mind. First, because we excluded participants who had already used substances at the time of the first sleep assessment, findings from our study do not represent these particularly young users. Similarly, while a number of participants were non-White by ethnicity or race, our exclusion criteria also resulted in differential inclusion of White over non-White participants. Second, as data were collected as part of a larger study, only brief measures of sleep health were available for secondary analysis. Moreover, sleep parameters were not assessed at the time participants initiated substance use. While we corrected for this variance in analyses, replication of these findings using more proximal sleep assessments are needed. Outcome data were also collected via self-report, which can be subject to recall bias and social desirability. However, adolescents have been found to provide valid estimates of their substance use when privacy and confidentiality are assured,[38,39] and self-reported sleep patterns in adolescents correlate with measures taken via actigraphy.[40] Thus, the likelihood that objective measures of these outcomes would produce meaningfully different results is small. Finally, future research may examine gender by sleep interactions in the context of puberty, which is known to impact substance use.[2]

Conclusion

Sleep health is a prospective predictor of the onset of alcohol and marijuana use among adolescents and accounts for variance in outcomes that is unexplained by internalizing and externalizing problem behaviors. Short sleep duration and daytime sleepiness are unique predictors of the onset of adolescent alcohol use and related problems, and short sleep duration is associated with increased odds of marijuana use. The independent effect of sleep on substance use within this population suggests that interventions targeting the types of sleep problems that are common in adolescence (e.g., delayed sleep phase) may be needed.

Acknowledgments

This research was supported by grant numbers R01 AA16838 (PI: Kristina Jackson), K02 AA021761 (PI: Kristina Jackson), and T32-AA007459 (PI: Peter Monti) from the National Institute on Alcohol Abuse and Alcoholism at the National Institutes of Health. NIH had no role in the study design, collection, analysis, or interpretation of the data, writing the manuscript, or the decision to submit the paper for publication.

References

1. Sivertsen B, Skogen JC, Jakobsen R, Hysin M. Sleep and use of alcohol and drug in adolescence. A large population-based study of Norwegian adolescents aged 16 to 19 years. Drug and Alcohol Dependence. 2015; 149:180–186. [PubMed: 25707706]
2. Pieters S, Van Der Vorst H, Burk WJ, Wiers RW, Engels RCME. Puberty-dependent sleep regulation and alcohol use in early adolescents. Alcoholism: Clinical and Experimental Research. 2010; 34(9): 1512–1518.
3. CDC. Alcohol and Public Health. Retrieved March 13, 2016 from http://www.cdc.gov/alcohol/2016
4. Watson NF, Badr MS, Belenky G, et al. Recommended Amount of Sleep for a Healthy Adult: A Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society. Sleep. 2015; 38(6):843–844. [PubMed: 26039963]
5. Pasch KE, Latimer LA, Cance JD, Moe SG, Lytle LA. Longitudinal bi-directional relationships between sleep and youth substance use. Journal of Youth and Adolescence. 2012; 41:1184–1196. [PubMed: 22752878]
6. Chan JKM, Trinder J, Colrain IM, Nicholas CL. The acute effects of alcohol on sleep electroencephalogram power spectra in late adolescence. Alcoholism: Clinical and Experimental Research. 2015; 39(2):291–299.
7. Van Reen E, Jenni OG, Carskadon MA. Effects of alcohol on sleep and the sleep electroencephalogram in healthy young women. Alcoholism: Clinical and Experimental Research. 2006; 30(6):974–981.
8. Schierenbeck T, Riemann D, Berger M, Hornyak M. Effect of illicit recreational drugs upon sleep: cocaine, ecstasy and marijuana. Sleep Med Rev. 2008; 12(5):381–389. [PubMed: 18313952]
9. Buysse DJ. Sleep health: Can we define it? Does it matter? SLEEP. 2014; 37(1):9–17. [PubMed: 24470692]
10. Pieters S, Burk WJ, Van der Vorst H, Dahl RE, Wiers RW, Engels RCME. Prospective relationships between sleep problems and substance use, internalizing and externalizing problems. Journal of Youth and Adolescence. 2015; 44:379–388. [PubMed: 25385390]
11. Wong MM, Brower KJ, Nigg JT, Zucker RA. Childhood sleep problems, response inhibition, and alcohol and drug outcomes in adolescence and young adulthood. Alcoholism: Clinical and Experimental Research. 2010; 34(6):1033–1044.
12. Hasler BP, Kirisci L, Clark DB. Restless sleep and variable sleep timing during late childhood accelerate the onset of alcohol and other drug involvement. Journal of Studies on Alcohol and Drugs. 2016; 77:649–655. [PubMed: 27340970]
13. Wong MM, Robertson GC, Dyson RB. Prospective relationship between poor sleep and substance-related problems in a national sample of adolescents. Alcoholism: Clinical & Experimental Research. 2015; 39(2):355–362.
14. Wong MM, Brower KJ, Fitzgerald HE, Zucker RA. Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. Alcoholism: Clinical and Experimental Research. 2004; 28(4):578–587.
15. Wong MM, Brower KJ, Zucker RA. Childhood sleep problems, early onset of substance use, and behavioral problems in adolescence. Sleep Medicine. 2009; 10(7):787–796. [PubMed: 19138880]
16. Krystal AD. Psychiatric disorders and sleep. Neurologic Clinics. 2012; 30(4):1389–1413. [PubMed: 23099143]
17. Couwenbergh C, van den Brink W, Zwart K, Vreugdenhil C, van Wijngaarden-Cremers P, van der Gaag RJ. Comorbid psychopathology in adolescents and young adults treated for substance use disorders: A review. European Child & Adolescent Psychiatry. 2006; 15(6):319–328. [PubMed: 16648966]
18. Tarokh L, Hamann C, Schimmelmann BG. Sleep in child and adolescent psychiatry: Overlooked and underappreciated. European Child & Adolescent Psychiatry. 2014; 23(6):396–372.
19. Johnson EO, Breslau N. Sleep problems and substance use in adolescence. Drug and Alcohol Dependence. 2001; 64(1):1–7. [PubMed: 11470335]

20. Jackson KM, Roberts ME, Colby SM, Barnett NP, Abar CC, Merrill JE. Willingness to drink as a function of peer offers and peer norms in early adolescence. Journal of Studies on Alcohol and Drugs. 2014; 75:404–414. [PubMed: 24766752]
21. Wolfson AR, Carskadon MA. Sleep schedules and daytime functioning in adolescents. Child Development. 1998; 69(4):875–887. [PubMed: 9768476]
22. Sobell, LC., Sobell, MB. Alcohol consumption measures. In: JAaMC. , editor. Assessing Alcohol Problems: A Guide for Clinicians and Researchers (NIAAA Treatment Handbook Series 4, NIH Publication No 95-3745). Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism; 2003. p. 75-101.
23. White HR, Labouvie EW. Longitudinal trends in problem drinking as measured by the Rutgers Alcohol Problem Index. Alcoholism: Clinical and Experimental Research. 2000; 24(76A)
24. Ginzler JA, Garrett SB, Baer JS, Peterson PL. Measurement of negative consequences of substance use in street youth: An expanded use of the Rutgers Alcohol Problem Index. Addictive Behaviors. 2007; 32(7):1519–1525. [PubMed: 17236724]
25. Choi WS, Gilpin EA, Farkas AJ, Pierce JP. Determining the probability of future smoking among adolescents. Addiction. 2001; 96(2):313–323. [PubMed: 11182877]
26. Achenbach, TM. Manual for the Child Behavior Checklist/4-18 and 1991 profile. Burlington: University of Vermont, Department of Psychiatry; 1991b.
27. March JS, Parker JDA, Sullivan K, Stallings P, Conners CK. The Multidimensional Anxiety Scale for Children (MASC): Factor structure, reliability, and validity. Journal of the American Academy of Child and Adolescent Psychiatry. 1997; 36(4):554–565. [PubMed: 9100431]
28. Cox DR. Regression models and life-tables. Journal of the Royal Statistical Society, Series B (methodological). 1972; 34(2):187–220.
29. Thomas AG, Monahan CJ, Lukowski AF, Cauffman E. Sleep problems across development: A pathway to adolescent risk taking through working memory. Journal of Youth and Adolescence. 2015; 44:447–464. [PubMed: 25213135]
30. Baum KT, Desai A, Field J, Miller LE, Rausch J, Beebe DW. Sleep restriction worsens mood and emotion regulation in adolescents. Journal of Child Psychology and Psychiatry. 2014; 55(2):180–190. [PubMed: 24889207]
31. Coskunpinar A, Dir AL, Cyders MA. Multidimensionality in impulsivity and alcohol use: A meta-analysis using the UPPS model of impulsivity. Alcoholism: Clinical and Experimental Research. 2013; 37(9):1441–1450.
32. Poon JA, Turpyn CC, Hansen A, Jacangelo J, Chaplin TM. Adolescent substance use and psychopathology: Interactive effects of cortisol reactivity and emotion regulation. Cognitive Therapy and Research. 2015
33. Meltzer LJ, Mindell JA. Sleep and sleep disorders in children and adolescents. Psychiatric Clinics of North America. 2006; 29:1059–1076. [PubMed: 17118282]
34. Harvey AG. A transdiagnostic intervention for youth sleep and circadian problems. Cognitive and Behavioral Practice. 2015
35. de Bruin EJ, Dewald-Kaufmann JF, Ooort FJ, Bogels SM, Meijer AM. Differential effects of online insomnia treatment on executive functions in adolescents. Sleep Medicine. 2015; 16(4): 510–520. [PubMed: 25796966]
36. de Bruin EJ, Bogels SM, Oort FJ, Meijer AM. Efficacy of a cognitive behavioral therapy for insomnia in adolescents: A randomized controlled trial with internet therapy, group therapy, and a waiting list condition. Sleep: Journal of Sleep and Sleep Disorders Research. 2015; 38(12):1913–1926.
37. Bootzin RR, Stevens SJ. Adolescents, substance abuse, and the treatment of insomnia and daytime sleepiness. Clin Psychol Rev. 2005; 25(5):629–644. [PubMed: 15953666]
38. Stone AL, Latimer WW. Adolescent substance use assessment: Concordance between tools using self-administered and interview formats. Substance Use and Misuse. 2005; 40:1865–1874. [PubMed: 16419561]
39. Winters, KC., Stinchfield, R., Bukstein, OG. Assessing adolescent substance use and abuse. In: YKOGB. , editor. Adolescent substance abuse: Psychiatric comorbidity and high-risk behaviors. New York, NY: Routledge/Taylor & Francis Group; 2008. p. 53-85.

40. Wolfson AR, Carskadon MA, Acebo C, et al. Evidence for the validity of a sleep habits survey for adolescents. Sleep: Journal of Sleep and Sleep Disorders Research. 2003; 26(2):213–216.

Table 1

Demographic characteristics, sleep parameters, and substance use for participants who provided sleep and parent report data (N=829).

	N	*M* or %	*SD*
Age at T1	829	12.6	1.02
Female (%)	434	52.4	–
Non-White (%)	127	15.3	–
Hispanic (%)	97	11.7	–
Sleep duration (hours)	815	8.79	1.47
Bedtime delay (hours)	805	1.64	1.91
Daytime sleepiness	827	1.80	0.79
Internalizing behavior	829	5.02	5.66
Externalizing behavior	827	4.81	6.47
Social anxiety	829	7.28	5.26
	N substance-naïve at first sleep assessment	% reporting substance use by T7[a]	
Full drink of alcohol	779	37.7	
Heavy episodic drinking	779	22.2	
Alcohol problems	779	13.7	
Puffing cigarette	771	15.7	
Full cigarette smoking	771	7.9	
Marijuana use	777	25.1	

Note.

[a]Participants who were substance-naïve at initial assessment of sleep parameters and endorsed substance use at any subsequent assessment. T1=Time 1. T7=Time 7.

Table 2

Spearman correlations between sleep parameters and demographics (N=829).

	1.	2.	3.	4.	5.	6.	7.	8	9.	10.
1. Sleep duration	–	−16**	−11**	−.20**	−.004	.06	−03	−.07*	−13**	−.01
2. Bedtime delay		–	08*	−.04	.01	−19**	.20**	10**	16**	.02
3. Daytime sleepiness			–	10**	−.05	−.03	.02	15**	.06	23**
4. Age at T1				–	.03	.05	−.05	−.04	.02	−.03
5. Gender					–	.06	−.004	−.02	.03	−15**
6. Race (White)						–	_41**	−09*	−10**	.002
7. Ethnicity (Hispanic)							–	13**	14**	−.01
8. Internalizing								–	.57**	17**
9. Externalizing									–	.02
10. Social anxiety										–

Note.

*p < .05.

**p < .01.

T1=Time 1.

Table 3

Step 2 results of discrete-time Cox regressions for alcohol use outcomes.

		Full Drink				Heavy Episodic Drinking				Alcohol-Related Consequences			
		HR	**P**	**95% CI**		**HR**	**P**	**95% CI**		**HR**	**P**	**95% CI**	
Step 1	Age at sleep assmt	0 60	< .001	0.52	0.70	0.61	< .001	0.50	0.75	0.58	< .001	0.44	0.77
	Male gender	0 60	< .001	0.47	0.76	0.62	.002	0.45	0.84	0.46	< .001	0.30	0.70
	Internalizing	0 98	.10	0.95	1.00	0.96	.01	0.93	0.99	0.98	.30	0.94	1.02
	Externalizing	104	< .001	1.02	1.06	1.07	.01	0.93	0.99	1.05	.001	1.02	1.09
Step 2	Age at sleep assmt	0.58	< .001	0.49	0.67	.57	< .001	.46	.70	.55	< .001	.41	.73
	Male gender	0.59	< .001	0.47	0.75	.60	.002	.44	.83	.45	< .001	.30	.69
	Internalizing	0.98	.07	0.95	1.00	.95	.007	.92	.99	.98	.25	.93	1.02
	Externalizing	1.04	< .001	1.02	1.06	1.07	< .001	1.05	1.10	1.05	.001	1.02	1.09
	Sleep duration§	**0.87**	**.004**	**0.79**	**0.96**	**.80**	**.001**	**.70**	**.91**	**.82**	**.01**	**.70**	**.96**
		$\Delta\chi^2$ (1, N=766)= 8.40, *p*= .004				χ^2(1, N=763)=10.89, *p*=.001				χ^2(1, N=766)=5.99, *p*=.01			
Step 2	Age at sleep assmt	0.60	< .001	0.51	0.70	.60	< .001	.48	.74	.57	< .001	.43	.76
	Male gender	0.61	< .001	0.48	0.78	.63	.004	.46	.86	.48	.001	.32	.73
	Internalizing	0.98	.09	0.95	1.00	.96	.01	.92	.99	.98	.27	.94	1.02
	Externalizing	1.04	< .001	1.02	1.06	1.07	< .001	1.04	1.09	1.05	.001	1.02	1.09
	Bedtime Delay§	1.03	.24	0.98	1.08	1.05	.12	.99	1.12	1.04	.38	.95	1.13
		χ^2(1, N=755)= 1.23, *p*=.27				χ^2(1, N=752)=2.03, *p*=.15				χ^2(1, N=755)=0.68, *p*=.41			
Step 2	Age at sleep assmt	0.61	< .001	0.52	0.71	.60	< .001	.49	.74	.56	< .001	.43	.74
	Male gender	0.61	< .001	0.48	0.77	.62	.003	.46	.85	.47	< .001	.31	.71
	Internalizing	0.97	.03	0.94	1.00	.95	.003	.92	.98	.97	.13	.92	1.01
	Externalizing	1.04	< .001	1.02	1.07	1.07	< .001	1.05	1.10	1.06	< .001	1.02	1.09
	Daytime sleepiness§	**1.26**	**.002**	**1.09**	**1.46**	**1.31**	**.006**	**1.08**	**1.60**	**1.42**	**.004**	**1.12**	**1.80**
		χ^2(1, N=776)=8.85, *p*=.003				χ^2(1, N=773)=7.01, *p*=.008				χ^2(1, N=776)=7.55, *p*=.006			

Note.

§Variables added to the model in Step 2. Assmt=assessment. HR=Hazard Ratio. Bold items indicate variables that added significantly to the model in Step 2 (based on pseudo- χ^2 values).

Table 4

Step 2 results of discrete-time Cox regressions for smoking and marijuana use outcomes.

		Cigarette Puffed				Full Cigarette Smoked				Marijuana Used			
		HR	***p***	**95% CI**		**HR**	***p***	**95% CI**		**HR**	***p***	**95% CI**	
Step 1	Age at sleep assmt	0.83	.08	0.67	1.02	0.94	.68	0.69	1.28	0.66	< .001	0.55	0.79
	Male gender	0.64	.02	0.44	0.93	0.89	.66	0.53	1.50	0.68	.01	0.51	0.92
	White race	0.74	.21	0.46	1.19	1.52	.34	0.64	3.60	0.61	.01	0.43	0.87
	Internalizing	0.97	.16	0.93	1.01	0.99	.69	0.94	1.04	0.95	.002	0.92	0.98
	Externalizing	1.08	< .001	1.05	1.11	1.06	.01	1.02	1.11	1.07	< .001	1.04	1.09
Step 2	Age at sleep assmt	0.82	.07	0.66	1.02	0.93	.65	0.68	1.28	0.64	< .001	0.53	0.76
	Male gender	0.63	.02	0.44	0.93	0.89	.66	0.53	1.50	0.67	.008	0.50	0.90
	White race	0.74	.22	0.46	1.19	1.52	.34	0.65	3.60	0.63	.01	0.45	0.90
	Internalizing	0.97	.16	0.93	1.01	0.99	.68	0.94	1.04	0.95	.001	0.91	0.98
	Externalizing	1.08	< .001	1.05	1.11	1.06	.01	1.02	1.11	1.07	< .001	1.04	1.09
	Sleep duration[§]	0.98	.80	0.85	1.13	0.98	.81	0.80	1.19	**0.88**	**.046**	**0.78**	**0.99**
		χ^2 (1, N=760)=0.07, *p*=.80				χ^2(1, N=760)=0.06, *p*=.81				χ^2(1, N=766)=3.97, *p*=.046			
Step 2	Age at sleep assmt	0.82	.07	0.67	1.02	0.94	.68	0.69	1.28	0.67	< .001	0.56	0.80
	Male gender	0.62	.01	0.42	0.91	0.80	.41	0.48	1.36	0.67	.01	0.50	0.90
	White race	0.79	.35	0.49	1.29	1.75	.21	0.73	4.23	0.64	.02	0.44	0.92
	Internalizing	0.97	.12	0.93	1.01	0.98	.49	0.93	1.04	0.94	.001	0.91	0.98
	Externalizing	1.08	< .001	1.05	1.12	1.06	.007	1.02	1.11	1.07	< .001	1.04	1.10
	Bedtime Delay[§]	1.05	.25	0.97	1.14	1.08	.22	0.96	1.23	1.03	.40	0.97	1.09
		χ^2(1, N=749)=1.15, *p*=.28				χ^2(1, N=749)=1.23, *p*=.27				χ^2(1, N=754)=0.65, *p*=.42			
Step 2	Age at sleep assmt	0.82	.07	0.67	1.01	0.93	.65	0.69	1.26	0.66	< .001	0.56	0.79
	Male gender	0.64	.02	0.44	0.93	0.89	.65	0.54	1.48	0.69	.01	0.52	0.93
	White race	0.79	.31	0.49	1.26	1.67	.24	0.71	3.94	0.65	.01	0.46	0.92
	Internalizing	0.97	.08	0.93	1.01	0.97	.33	0.92	1.03	0.94	.001	0.91	0.98
	Externalizing	1.08	< .001	1.05	1.11	1.06	.005	1.02	1.11	1.07	< .001	1.04	1.09

	Cigarette Puffed				Full Cigarette Smoked				Marijuana Used			
	HR	***p***	**95% CI**		**HR**	***p***	**95% CI**		**HR**	***p***	**95% CI**	
Daytime sleepiness[§]	1.16	.19	0.93	1.45	1.29	.11	0.95	1.76	1.13	.17	0.95	1.35
	$\chi^2(1, N=768)=1.63, p=.20$				$\chi^2(1, N=768)=2.45, p=.12$				$\chi^2(1, N=774)=1.85, p=.17$			

Note.

[§]Variables added to the model in Step 2. Assmt=assessment. HR=Hazard Ratio. Sleepiness refers to daytime sleepiness. Bold items indicate variables that added significantly to the model in Step 2 (based on pseudo- χ^2 values).

Table 5

Step 2 results of discrete-time Cox regressions using simultaneous sleep predictors of substance use outcomes.

		Full Drink				**Heavy Episodic Drinking**				**Alcohol-Related Consequences**			
		HR	***p***	**95% CI**		**HR**	***p***	**95% CI**		**HR**	***p***	**95% CI**	
Step 2	Age at sleep assmt	0.55	< .001	0.47	0.65	0.55	< .001	0.44	0.69	0.53	< .001	0.40	0.71
	Male gender	0.60	< .001	0.47	0.76	0.61	.003	0.45	0.84	0.46	< .001	0.30	0.71
	Internalizing	0.97	.02	0.94	1.00	0.95	.002	0.91	0.98	0.97	0.13	0.92	1.01
	Externalizing	1.04	< .001	1.02	1.07	1.07	< .001	1.04	1.10	1.05	.001	1.02	1.09
	Sleep duration§	**0.88**	**.01**	**0.80**	**0.97**	**0.82**	**.004**	**0.72**	**0.94**	**0.84**	**0.04**	**0.72**	**0.99**
	Bedtime delay§	1.02	.54	0.96	1.07	1.03	.34	0.97	1.11	1.02	0.75	0.92	1.12
	Daytime sleepiness§	**1.21**	**.01**	**1.04**	**1.41**	**1.24**	**.04**	**1.02**	**1.51**	**1.31**	**0.04**	**1.02**	**1.68**
		χ^2 (3, N=747)=15.60, *p*=.001				χ^2 (3, N=744)=15.87, *p*=.001				χ^2 (3, N=747)=10.29, *p*=.02			
		Cigarette Puffed				**Full Cigarette Smoked**				**Marijuana Used**			
		HR	*p*	95% CI		HR	*p*	95% CI		HR	*p*	95% CI	
Step 2	Age at sleep assmt	0.81	.07	0.65	1.02	0.93	.66	0.68	1.28	0.63	< .001	0.52	0.76
	Male gender	0.63	.02	0.43	.93	0.84	.52	0.49	1.43	0.66	.006	0.49	0.89
	White race	0.77	.29	0.47	1.25	1.68	.25	0.70	4.03	0.63	.01	0.44	0.90
	Internalizing	0.97	.13	0.93	1.01	0.98	.47	0.93	1.04	0.94	.001	0.91	0.98
	Externalizing	1.08	< .001	1.05	1.11	1.06	.007	1.02	1.11	1.07	< .001	1.04	1.10
	Sleep duration§	0.99	.90	0.86	1.15	1.01	.96	0.83	1.22	0.89	.06	0.78	1.00
	Bedtime delay§	1.04	.38	0.95	1.13	1.05	.51	0.90	1.22	1.01	.65	0.95	1.08
	Daytime sleepiness§	1.13	.31	0.89	1.43	1.29	.13	0.93	1.79	1.09	.37	0.91	1.30
		χ^2 (3, N=742)=1.83, *p*=.61				χ^2 (3, N=742)=2.90, *p*=.41				χ^2 (3, N=747)=5.17, *p*=.16			

Note. HR: Hazard Ratio.

§Variables added to the model in Step 2. Assmt=assessment. Sleepiness refers to daytime sleepiness. Bold items indicate variables that contributed significantly to the model in Step 2.