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Insomnia Treatment Effects among Young Adult Drinkers: Secondary Outcomes of a Randomized Pilot Trial

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Abstract

Background: Cognitive Behavioral Therapy for Insomnia (CBT-I) has moderate to large effects on insomnia among young adult drinkers, with preliminary data indicating that improvements in insomnia may have downstream effects on alcohol-related consequences. However, the mechanism(s) by which insomnia treatment may facilitate reductions in alcohol-related problems is unclear. Secondary outcome data from a randomized pilot trial were used to examine CBT-I effects on four proposed mediators of the insomnia/alcohol link: alcohol craving, delay discounting, negative affect, and difficulties with emotion regulation.

Methods: Young adults (ages 18-30y) with insomnia who reported 1+ binge drinking episode (4/5+ drinks for women/men) in the past month were randomized to CBT-I (n=28) or sleep hygiene control (n=28). Outcomes were assessed at baseline, post-treatment, and one month.

Results: Relative to those in sleep hygiene, CBT-I participants reported greater post-treatment decreases in alcohol craving (d=0.33) and greater one-month decreases in delay discounting of large rewards (d=0.42). CBT-I did not have a significant effect on delay discounting of smaller rewards or momentary negative affect. There was also no significant treatment effect on difficulties with emotion regulation, although findings were confounded by a significant group difference in difficulties with emotion regulation at baseline.

Conclusions: Treatment of insomnia may lead to improvements in alcohol craving and delay discounting of large rewards among young adult drinkers with insomnia. Additional research examining improvement in insomnia as a mechanism for improvement in addiction domains is warranted.

Keywords

alcohol; insomnia; sleep; treatment; mechanism

Introduction

Alcohol use is prevalent among young adults in the United States, with 68% of 19- to 30-year-olds reporting alcohol use in the past 30 days (Schulenberg et al., 2020). Among young

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adults, binge drinking (defined as 5+ drinks in a row in the past 2 weeks) has remained at a relatively stable 32% over the past five years, while rates of 10 or more drinks in a row increased to 12% in 2019 (Schulenberg et al., 2020). These heavier rates of drinking are especially concerning, as they have been linked to both negative consequences (e.g., behavior you later regretted, poor physical health, damaged relationships) and increased odds of concurrent alcohol use disorder (Patrick et al., 2020, Linden-Carmichael et al., 2017). However, given data indicating that drinking only explains 15-25% of the variance in alcohol-related consequences (Prince et al., 2018), research examining other ways to reduce the burden of alcohol use among young adults is warranted.

A number of theoretical models attempt to explain the development of addiction (Bechara et al., 2019, Bickel et al., 2018). One of these, derived from the field of neuroeconomics, proposes competing neurobehavioral decision-making systems (Bechara et al., 2019). Specifically, the dual systems model posits that addiction occurs as the result of imbalance between two decision-making systems in the brain: an impulsive system in the limbic/paralimbic regions and an executive system in the prefrontal cortex (Bechara et al., 2019). In a healthy adult, these two systems are thought to work in parallel. However, in the case of addiction, the impulsive decision-making system exerts greater control than the executive decision-making system (Bechara et al., 2019), akin to a car with a sensitive accelerator and worn-out brakes. This model is appealing in the context of substance use treatment because it implies that any intervention that restores balance between these two systems may facilitate recovery from addiction (Bechara et al., 2019). Theoretically, if applied in the context of prevention, then maintaining balance within these systems might also be expected to prevent the onset of addiction. One health behavior that has been associated with both impulsivity and executive functioning – but has not been targeted as a mechanism of improvement in substance use – is sleep.

Sleep has been posited as a neurobiological contributor to each stage of the addiction cycle: binge/intoxication, withdrawal/negative affect, and preoccupation/anticipation (Koob and Colrain, 2019). According to the Addictions Neuroclinical Assessment (Kwako et al., 2017), different neuroscience domains are especially salient within each of these stages: incentive salience (associated with craving) is particularly salient in the binge/intoxication phase, negative emotionality is particularly salient in the withdrawal/negative affect phase, and executive function is salient in the preoccupation/anticipation phase. Sleep disturbance has been associated with each of these neurocognitive domains. Although understudied in young adults, sleep disturbance has been associated with alcohol craving among individuals in recovery from alcohol use disorder (Kolla et al., 2020, Brooks et al., 2019). It has also been associated with lower positive affect, higher negative affect, and more difficulties with emotion regulation in a variety of studies (Galambos et al., 2009, Konjarski et al., 2018, Saksvik-Lehouillier et al., 2020, Baum et al., 2014, Cox et al., 2019). Finally, sleep disturbance has been associated with deficits in executive functions, or the cognitive abilities involved in pre-potent response inhibition, working memory updating, and task-set shifting (Miyake and Friedman, 2012). For instance, a longitudinal study demonstrated that greater parent-reported sleep disturbances in childhood predicted worse working memory performance and greater risk-taking behavior in adolescence (Thomas et al., 2015); and sleep deprivation has been associated with more impulsive decision making (Killgore et al.,

2006, Schnyer et al., 2009, Venkatraman et al., 2007). Insomnia has also been linked to delay discounting, or the tendency to choose smaller immediate rewards over larger delayed rewards (Oshri et al., 2017), which is proposed as an indicator of impaired executive function within the Addictions Neuroclinical Assessment (Kwako et al., 2016). If treatment of sleep problems leads to improvements in these brain systems, then it may also help delay onset of heavy substance use and/or prevent progression to problematic use.

Given the theoretical link between sleep and the brain systems that contribute to addiction (Koob and Colrain, 2019, Bechara et al., 2019) and the empirical link between insomnia symptoms and alcohol-related consequences (Kenney et al., 2012, Miller et al., 2016), we conducted a randomized pilot trial examining the impact of Cognitive Behavioral Therapy for Insomnia (CBT-I) on alcohol use outcomes among young adult drinkers with insomnia. CBT-I had a large effect on insomnia symptoms immediately following treatment, with moderate improvements maintained at one-month follow-up (Miller et al., 2020). While the treatment did not have a direct effect on alcohol use outcomes, post-treatment improvements in insomnia were associated with decreases in alcohol-related consequences at one-month follow-up (Miller et al., 2020). This study reports the extent to which CBT-I impacted the theorized mediators of the association between insomnia and alcohol-related problems. Specifically, we hypothesized that CBT-I would be more effective than single-session sleep hygiene control in reducing alcohol craving, delay discounting, negative affect, and difficulties with emotion regulation. These outcomes were chosen because they map onto the incentive salience, executive function, and negative emotionality domains of the Addictions Neuroclinical Assessment (Kwako et al., 2017).

Materials and Methods

Participants and Procedure

Recruitment.—From August 2018 to June 2019, we recruited young adults between 18 and 30 years via flyers, university email, and Facebook advertising to participate in an insomnia treatment study for young adults who drink alcohol. Of the 342 individuals who filled out the online screening survey, 134 did not meet screening criteria, 21 did not provide contact information, 44 were never reached, and 56 declined to participate. Eighty-seven individuals completed the baseline assessment.

Participants.—Eligibility criteria for research participation included (1) age 18 to 30 years, (2) at least one binge drinking episode (4/5+ drinks for women/men) in the past 30 days, and (3) diagnostic criteria for Insomnia Disorder. Participants with insomnia reported >30 minutes sleep onset latency, wake after sleep onset, or early morning awakening at least 3 nights per week for 3 or more months. They also scored ≥10 on the Insomnia Severity Index (Bastien et al., 2001). Exclusion criteria included (1) initiation of a new sleep medication in the past 6 weeks at baseline, (2) history of mania or seizure disorder, (3) symptoms requiring immediate clinical attention (e.g., severe sleep apnea, suicidal intent), and (4) current treatment for insomnia or alcohol use.

Of the 87 individuals who completed baseline, 56 met all eligibility criteria and were randomized to treatment condition. There were no significant differences between those

included and excluded on demographic variables (e.g., age, sex, race, ethnicity), insomnia severity, drinking quantity, or alcohol-related consequences (see Table 1). Groups did differ significantly on one outcome variable at baseline; specifically, CBT-I participants reported significantly greater difficulties with emotion regulation at baseline than sleep hygiene participants, $t(54) = -2.28$, $p = .03$ (see Table 2).

Procedure.—At baseline, a trained assessor conducted an in-person clinical interview using the MINI International Neuropsychiatric Interview for DSM-5 (©1992-2016 Sheehan DV) and administered other baseline measures (see below). Participants also wore an actiwatch and completed online sleep diaries each morning during the 7-day baseline assessment period. We randomly assigned eligible participants to either CBT-I (n=28) or the sleep hygiene (n=28) control condition. Participants were told that they were receiving either "brief" (sleep hygiene) or "more intense" (CBT-I) treatment for insomnia, in an effort to blind them to condition. Participants in both conditions were instructed to complete daily sleep diaries throughout the treatment phase of the study. Follow-up assessments occurred at the end of the 5-week treatment period (post-treatment) and one-month post-treatment. Participants completed follow-up assessments via computer to minimize detection bias, and the principal investigator and therapists were blinded to follow-up results.

Interventions

Cognitive behavioral therapy for insomnia (CBT-I).—CBT-I was delivered individually in a five-session protocol derived from manuals with demonstrated efficacy (Manber et al., 2014, McCrae et al., 2019). Interventionists reviewed and provided feedback on participants' daily sleep diaries at the beginning of each treatment session. Individual sessions covered treatment rationale and sleep hygiene (Session 1), sleep restriction and stimulus control (Session 2), relaxation techniques (Session 3), and cognitive therapy (Session 4). In the final session (Session 5), participants reviewed the treatment rationale and their current progress and discussed ways to prevent recurrent insomnia episodes.

Sleep hygiene (SH).—Sleep hygiene was delivered individually in a single session in an effort to emulate "usual care" (i.e., the sleep information likely provided in a routine primary care visit). Participants reviewed a handout of sleep hygiene recommendations from the National Sleep Foundation (2018) website (e.g., limiting naps, avoiding caffeine and nicotine before bed) with the study interventionist. Because sleep hygiene was discussed in both the control and treatment conditions, all participants were encouraged to avoid alcohol use before bedtime. Alcohol use was not directly targeted in any other aspect of either intervention.

Treatment integrity.—Treatments were delivered by predoctoral psychology students in clinical or counseling programs. Graduate students were supervised by two licensed clinical psychologists (MBM and CSM), one of whom is also board certified in behavioral sleep medicine (CSM). Consistent with recommendations (Lichstein et al., 1994), treatment integrity was assured by (1) providing interventionists with initial training via mock therapy sessions, as well as ongoing training and supervision utilizing audiotapes and discussion of

treatment sessions; (2) providing participants with a workbook of treatment materials; and (3) reviewing homework completion and barriers to participant adherence each week.

Measures

Alcohol craving.—Participants completed the 5-item Penn Alcohol Craving Scale (Flannery et al., 1999) to indicate the intensity, frequency, and duration of their alcohol cravings, as well as their perceived ability to resist acting on those cravings and overall "average alcohol craving" for the past week. Response ranged from 0-6, with total scores ranging from 0-30. Previous studies indicate good internal consistency and construct validity for this measure (Flannery et al., 1999).

Delay discounting.—Participants completed the Monetary Choice Questionnaire (Kirby, Petry, & Bickel, 1999) as a self-report measure of delay discounting. In 27 trials, participants chose between two hypothetical rewards, namely a smaller/immediate reward or a larger/delayed reward (e.g., "Would you prefer $54 today or $55 in 117 days?"). Rate of discounting (k) was calculated using Gray and colleagues' (2016) syntax. Higher discounting rates indicate greater discounting of future rewards, such that the participant tended to choose smaller immediate rewards over the larger delayed rewards. We calculated four k values: an overall k as well as the k value for relatively small ($11-35), medium ($20-60), and large rewards ($31-85) (Gray et al., 2016). Although this study utilized hypothetical rewards, hypothetical rewards have been closely associated with actual reward choices in previous research (Lagorio and Madden, 2005). Previous research indicates acceptable test-retest reliability (correlations ~.72) for these measures at short-term (5-week) intervals (Kirby, 2009).

Negative affect.—Participants completed the Positive and Negative Affect Schedule (Watson et al., 1988) to measure current negative affect. Participants indicated the extent to which they felt 10 negative (e.g., scared, ashamed, and irritable) and positive (e.g., interested, attentive, proud) emotions in the present moment. Response options ranged from 1 (*not at all*) to 5 (*extremely*). The PANAS has demonstrated high construct validity in community samples (Crawford and Henry, 2004), and the negative affect subscale has been found to maintain acceptable psychometric properties independent of the PANAS as a whole (Tuccitto et al., 2010).

Emotion regulation.—Participants completed the 36-item Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) to indicate how often in the past month they would endorse items such as, "*I have difficulty making sense out of my feelings*," and, "*When I am upset, I become out of control*." Response options ranged from 1 (*almost never*) to 5 (*almost always*). In additional to an overall sum score, the scale captures six dimensions of emotion regulation, all of which demonstrated good internal consistency in this sample: non-acceptance of emotional responses (6 items, α=.87), difficulties engaging in goal-directed activities (5 items, α=.88), impulse control difficulties (6 items, α=.90), lack of emotional awareness (6 items, α=.86), limited access to emotion regulation strategies (8 items, α=.88), and lack of emotional clarity (5 items, α=.78). The DERS has demonstrated

good convergent and discriminant validity and good test-retest reliability in clinical and community samples (Gratz and Roemer, 2004).

Data Screening and Analysis

Missing data.—Thirteen participants were missing data at post-treatment; however, five of those completed follow-up, resulting in missing data for 8 participants at follow-up. We utilized linear multiple regression with 20 imputations (Graham et al., 2007, Hallgren and Witkiewitz, 2013) to estimate missing outcome values.

Primary data analysis.—We conducted analyses in SPSS Statistics 26. Because multilevel models account for nesting within data (i.e., time points within persons) and require fewer assumptions (Heck et al., 2014), we used these in place of general linear models. We calculated intraclass correlation coefficients (ICCs) to confirm the presence of both between- and within-person effects in each outcome. Based on ICCs, 62% of variance in alcohol craving, 39% of variance in delay discounting, 64% of variance in negative affect, 45% of variance in difficulties with emotion regulation occurred between individuals (Level 2). Conversely, 38% of variance in alcohol craving, 61% in delay discounting, 36% in negative affect, and 55% in difficulties with emotion regulation occurred within individuals over time (Level 1).

We conducted separate models for each outcome, all using an intent-to-treat approach. We included treatment group (sleep hygiene=0, CBT-I=1) as a fixed effect at Level 2. We did not include demographic covariates because participants were randomized to condition. We specified intercepts as random and all other effects as fixed. We used Bonferroni adjustment to control for inflation in Type I error when examining subscale scores for the delay discounting (α=.05/3=.02) and emotion regulation outcomes (α=.05/5=.01) and Cohen's *d* to estimate effect size (0.20 small, 0.50 medium, 0.80 large).

Supplementary analyses.—The limited number of assessments in this study precludes testing of the serial mediation model that is truly hypothesized; specifically, that treatment (time 1) will lead to reductions in insomnia (time 2), which will lead to reductions in secondary outcomes (time 3), which will lead to reductions in alcohol use outcomes (time 4). However, as a preliminary test of these associations, we conducted a series of exploratory secondary analyses.

First, we conducted a series of mediation analyses to determine if changes in insomnia accounted for treatment effects on theoretical correlates of drinking outcomes (alcohol craving, overall delay discounting, emotion regulation, or negative affect) at post or follow-up. We tested mediation using bootstrapped 95% confidence intervals with 5,000 sampling estimates (SPSS PROCESS macro, version 3.5). Specifically, we modeled post-treatment insomnia severity as a mediator of the association between treatment group and each outcome, controlling for baseline levels of the outcome, drinking quantity, and insomnia severity.

Second, we conducted a series of multilevel models to determine if theoretical correlates of drinking were associated with actual changes in drinking outcomes over time. Continuous

Level 1 (L1) variables were person-mean centered to reflect within-person change, and continuous Level 2 (L2) variables were grand-mean centered to reflect between-person differences (Enders and Tofighi, 2007). We conducted separate models for each outcome (drinking quantity and alcohol-related consequences), with all covariates as well as grand-mean (L2) and person-mean (L1) centered versions of each theoretical correlate (alcohol craving, overall delay discounting, negative affect, and emotion regulation) included in each model. We included treatment group (0=sleep hygiene, 1=CBT-I), drinking quantity, and insomnia severity as between-person (L2) covariates and specified intercept and time effects as random.

Results

Primary Analyses: Treatment Outcomes

Descriptive and inferential statistics for all outcomes are depicted in Tables 2–4. There was a significant group by time interaction in the prediction of alcohol craving. SH participants reported non-significant decreases in craving from baseline to post (−0.31, *SE*=.74, *p*=.68) and post to 1-month follow-up (−1.03, *SE*=0.57, *p*=.09). CBT-I participants reported significant decreases in craving from baseline to post (−1.73, *SE*=0.74, *p*=.02) and non-significant increases at 1-month follow-up (+0.98, *SE*=0.57, *p*=.09).

In the prediction of delay discounting (overall *k*), there was a significant effect of time: both groups decreased non-significantly from baseline to post-treatment and then returned to or exceeded baseline levels at 1 month (CBT-I +.005, *SE*=.004, *p*=.16; SH +.015, *SE*=.004, *p*<.001), but there was no between-group difference at any time point (see Table 2). Similar patterns were observed for small-*k* and medium-*k* delay discounting values (see Table 3). However, there was a significant group by time interaction in the prediction of large-*k* values, which corresponded to a small to medium treatment effect at 1 month (see Table 3). Participants in both groups reported non-significant decreases in large-*k* from baseline to post, after which SH participants increased significantly (+.01, *SE*=.003, *p*=.002) and CBT-I participants decreased significantly (−.01, *SE*=.003, *p*=.001) from post to follow-up.

There were no significant group, time, or interaction effects for negative affect. Both groups reported relatively low negative affect at all time points (see Table 2).

There were significant group and time effects, but not a significant group by time interaction, in the prediction of difficulties with emotion regulation (see Table 2). SH participants reported non-significant decreases from baseline to post (−5.79, *SE*=3.01, *p*=.06) and post to 1 month (−3.68, *SE*=2.87, *p*=.20). CBT-I participants reported significant decreases from baseline to post (−12.87, *SE*=3.01, *p*<.001) and non-significant increases from post to follow-up (+2.04, *SE*=2.87, *p*=.48). However, there was also a significant between-group difference in difficulties with emotion regulation at baseline, with CBT-I participants reporting significantly greater difficulties with emotion regulation than SH participants (−12.39, *SE*=5.14, *p*=.02; see Table 2). After correcting for inflation in Type I error, there were no significant group by time interactions in the prediction of any emotion regulation subscale (see Table 4).

Supplementary Analyses

Insomnia as a mediator of treatment outcomes.—Post-treatment change in insomnia severity was a significant mediator of treatment effects on alcohol craving at post-treatment (indirect effect = −1.32, 95% CI = −2.58, −0.30), but not one month (indirect effect = −0.25, 95% CI = −1.24, 0.55). Post-treatment change in insomnia did not significantly mediate treatment effects on overall delay discounting at post (indirect effect = 0.00, 95% CI = 0.00, 0.01) or follow-up (indirect effect = 0.01, 95% CI = 0.00, 0.02); on negative affect at post (indirect effect = −0.88, 95% CI = −2.62, 0.72) or follow-up (indirect effect = −0.20, 95% CI = −1.67, 1.19); or on difficulties with emotion regulation at post (indirect effect = −2.00, 95% CI = −7.29, 4.24) or follow-up (indirect effect = 2.68, 95% CI = −4.10, 9.44).

Associations between theoretical correlates and drinking outcomes.—Participants reported significant decreases in drinking quantity over time (B= −1.43, SE=0.36, p<.001). At the between-person level, heavier drinkers reported greater drinking quantities than lighter drinkers (B=0.90, SE=0.05, p<.001) and individuals with stronger alcohol cravings reported greater drinking quantities than those with weaker alcohol cravings (B=0.45, SE=0.12, p<.001). At the within-person level, individuals also reported drinking more at times that they were experiencing greater negative affect than they personally tend to experience (B=0.29, SE=0.13, p=.03). Drinking quantity did not differ between CBT-I and sleep hygiene participants or between individuals reporting higher versus lower levels of insomnia severity, delay discounting, negative affect, or emotion regulation difficulties (all p>.12). Similarly, participants did not report significantly different drinking quantity at times that they reported experiencing above-person-average alcohol craving, delay discounting, or emotion regulation difficulties (all p>.27).

Participants also reported significant decreases in alcohol-related consequences over time (B= −0.95, SE=0.36, p=.01). Across time points, heavier drinkers reported more consequences than lighter drinkers (B=0.13, SE=0.05, p=.01), and individuals with more severe insomnia reported more consequences than those with less severe insomnia (B=0.28, SE=0.12, p=.02). Participants also reported fewer consequences at times that they reported above-person-average levels of craving (B= −0.45, SE=0.16, p=.01). Consequences did not differ significantly between individuals reporting higher versus lower levels of delay discounting, negative affect, or difficulties with emotion regulation (all p>.11); and participants did not report significant changes in alcohol-related consequences at times they were experiencing above-person-average levels of delay discounting, negative affect, or emotion regulation difficulties (all p>.43).

Discussion

This research examined the efficacy of CBT-I in reducing proposed mediators of the association between insomnia and alcohol-related problems among young adult drinkers with insomnia. Relative to a single-session sleep hygiene control, CBT-I was associated with modest post-treatment reductions in alcohol craving; and post-treatment changes in insomnia mediated this effect. This is consistent with data linking sleep disturbance to craving (Kolla

et al., 2020, Brooks et al., 2019), as well as research linking pharmacological treatment of insomnia (via gabapentin) to reductions in both subjective and affectively-evoked craving among individuals with alcohol use disorder (Mason et al., 2014). The potential for insomnia treatment to reduce craving has significant implications for prevention and treatment of alcohol use disorders, as alcohol craving (and incentive salience) is viewed as central to the binge/intoxication stage of addiction (Kwako et al., 2017). Notably, however, the confidence interval surrounding the effect size for this outcome included zero, and this effect would not have been significant if we had controlled for inflation in Type I error in a priori hypotheses. Treatment effects on alcohol craving were also no longer evident by one-month follow-up; thus, the reliability and generalizability of this effect is uncertain. It is possible that the relatively low levels of craving endorsed at baseline (5-6 on a 30-point scale) negatively impacted our ability to detect change in this outcome. Overall, the combination of these findings and other evidence (Mason et al., 2014) indicate tentative promise for insomnia as a mechanism for improvement in alcohol craving.

In contrast to theoretical models and previous studies, alcohol craving was associated with drinking at the between-person – but not within-person – level, and participants reported fewer (not more) consequences on days that they reported higher levels of craving. Given the timeframes of these measures, we speculate that the directionality of the craving/consequences association is actually reversed, such that participants reported stronger current craving because they had not experienced as many consequences as a result of drinking in the past month. It is also possible that positive associations between craving and drinking quantity are more evident among individuals with stronger or more variable levels of alcohol craving (Wemm et al., 2019, Serre et al., 2018, Szeto et al., 2019) or that they are more evident at certain times throughout the day (Hisler et al., 2021). Additional research examining these within-person associations in samples of primarily social drinkers is needed.

Although it was not associated with significant reductions in delay discounting of all amounts, CBT-I was associated with reductions in delay discounting of relatively large rewards ($31-85). This finding is exciting because delay discounting is posited as an indicator of impaired executive function, which is viewed as central to the preoccupation/anticipation stage of addiction (Kwako et al., 2017). It is also consistent with research indicating that CBT-I reduces other measures of executive function among individuals with insomnia and fibromyalgia (Miro et al., 2011). Interestingly, however, treatment effects on delay discounting were not evident until the follow-up period, after the active treatment phase had ended. This raises questions about which treatment elements are influencing these outcomes and when. In this sample, 2 CBT-I participants and 8 sleep hygiene participants still met study criteria for insomnia post-treatment, and these numbers dropped to 1 CBT-I participant and 6 sleep hygiene participants at follow-up (Miller et al., 2020). One could infer from these data that participants in both groups continued implementing the skills they learned in the post-treatment phase, as they demonstrated continued improvements with no evidence of relapse among those who improved. We are not able to test this hypothesis empirically because we did not assess participant compliance with treatment recommendations beyond the treatment phase; however, we recommend exploration of this possibility in future research.

Notably, the confidence interval surrounding the effect size for the delay discounting of large awards also included zero, indicating considerable variability in this treatment outcome. Although short sleepers (i.e., those reporting ≤6 hours) tend to exhibit more delay discounting than longer sleepers (7-9 hours) (Curtis et al., 2018), a number of experimental studies have failed to demonstrate that acute sleep deprivation impacts overall delay discounting in otherwise healthy adults (Libedinsky et al., 2013, Demos et al., 2016, Acheson et al., 2007). It seems plausible that individuals with insomnia would have more room for improvement in sleep disturbance than healthy individuals, in which case the association between sleep and delay discounting may be more evident in this population. However, participants also reported considerable within-person variability in delay discounting over time. Although this measure has demonstrated adequate test-retest reliability in previous research, particularly at the short-term intervals assessed here (Kirby, 2009), it is possible (as with all measures) that measurement error contributed to outcomes. Additional research is needed to confirm or refute the potential for insomnia treatment to impact delay discounting of rewards.

In contrast to hypotheses, CBT-I did not have a significant impact on negative affect. This contradicts a wealth of literature linking better sleep quality to higher positive affect and lower negative affect the following day (Galambos et al., 2009, Konjarski et al., 2018), as well as experimental research linking acute sleep deprivation to decreased positive affect (Saksvik-Lehouillier et al., 2020). It is possible that we failed to document this association because our measure assessed negative affect "in this moment," rather than negative affect over a longer period of time. Indeed, a number of daily studies asked participants to rate negative/positive affect over the last hour or the course of the entire day (Galambos et al., 2009, Doane and Thurston, 2014, Fortier et al., 2015), rather than in the current moment. Since this study was examining past-month changes in sleep, it may have been preferable to assess negative affect over the same timeframe. However, it is also possible that there are daily associations between insomnia and negative affect that do not persist over longer periods of time. Given the within-person association between negative affect and drinking quantity, additional strategies to target and potentially mitigate this theoretical and empirical correlate of drinking are needed.

Treatment effects on difficulties with emotion regulation are difficult to interpret, as CBT-I participants reported more difficulty with emotion regulation than sleep hygiene participants at baseline. This was an unfortunate failure of randomization that precludes conclusions based on these pilot data. However, it seems notable that the only two treatment effect sizes with 95% confidence intervals that did not include zero were for emotion regulation subscales; specifically, the impulse control (e.g., "*I experience my emotions as overwhelming and out of control*" and "*When I'm upset, I feel out of control*") and emotion regulation strategies subscales (e.g., "*When I'm upset, I believe that I will remain that way for a long time*" and "*When I'm upset, I believe that there is nothing I can do to make myself feel better*"). Both longitudinal and experimental research have linked poor sleep or insomnia to difficulties with emotion regulation (Cox et al., 2019, O'Leary et al., 2017, Baum et al., 2014, Mauss et al., 2013), and better sleep quality has been associated with more effective emotion regulation over time (Tavernier and Willoughby, 2015). Based on

these data, continued examination of insomnia treatment effects on difficulties with emotion regulation is warranted.

Clinical Implications

The potential for insomnia treatment to impact domains of addiction has significant implications for the prevention and treatment of alcohol use disorders. Previous research has found that the majority of variability in alcohol-related consequences is not explained by existing indicators of alcohol use (e.g., drinking quantity/frequency, binge drinking, getting drunk) (Prince et al., 2018). This suggests that factors beyond drinking contribute to alcohol-related problems. Sleep complaints are common in both heavy-drinking samples (39-54%) (Canham et al., 2015) and those with alcohol use disorders (63-76%) (Chakravorty et al., 2016). As such, treatment for sleep disturbance is likely relevant for a large number of individuals at risk for alcohol-related harm. Moreover, unlike the myriad other risk factors for alcohol problems (e.g., sex, genetics, environment, impulsivity), highly effective behavioral treatments for insomnia exist. CBT-I was chosen as the insomnia treatment in this study because it is a first-line treatment for insomnia that has demonstrated efficacy across a range of populations (Siebern and Manber, 2011, Chand, 2015), including those with alcohol use disorders (Miller et al., 2017). Data from this study expand on this research by demonstrating that CBT-I may also impact theoretical and empirical correlates of alcohol use disorder in at-risk young adults.

Limitations and Future Directions

A number of study limitations should be considered when interpreting results. First, the control condition in this study (single-session sleep hygiene) was not matched with CBT-I for time and content. This study design was intentional, as the success of mechanistic trials often depends on the production of the largest possible between-group difference in the outcome (Freedland et al., 2011). However, this also means that observed results may not be unique to CBT-I and may be attributable instead to non-specific therapy effects. Moreover, a large number of participants who screened eligible for the study (27%) declined to participate, and only 2 out of 3 CBT-I participants completed all five treatment sessions (Miller et al., 2020). Thus, remote delivery options and shorter protocols may be needed in clinical settings. The duration of the study was also quite short, with our longest follow-up occurring only one month after treatment, and effects on alcohol craving were not sustained. Future research examining the extent to which CBT-I participants continue implementing treatment strategies – and may continue benefiting from various cognitive and behavioral techniques – may help inform efforts to sustain treatment effects.

It is also worth noting that, while our measures map theoretically onto each of the neuroclinical addiction domains, some of these measures may not be the most useful or valid indicators of each domain. For example, delay discounting was included as a proposed measure of executive function in the original Addictions Neuroclinical Assessment battery (Kwako et al., 2016); however, this was the only hypothesized indicator of executive function that did not load on the executive function factor when tested empirically (Kwako et al., 2019). Since research in this area is ongoing, future studies may need to modify the proposed measures for each domain. This research also presumes that the ‘mechanism’ from

insomnia to alcohol use and related problems is consistent across individuals; however, it is possible that the extent to which insomnia impacts alcohol-related outcomes depends on other variables, such as motives for drinking. For example, pathways linking insomnia to drinking through negative affect or emotion regulation may be more salient for individuals who report drinking to cope than for individuals who drink primarily for social or enhancement reasons.

Finally, 75% of participants identified as female and 82% identified as non-Hispanic White. Insomnia is more prevalent among women than men, but the ratio is typically closer to 3:1 (Theorell-Haglow et al., 2018, Ford et al., 2015). Rates of insomnia also tend to be similar across racial groups in the United States, and rates of poor sleep quality tend to higher among Blacks than Whites (Chen et al., 2015, Carnethon et al., 2016). Thus, men and racial/ethnic minority groups were underrepresented in this sample. College students were also overrepresented (73% of this sample), which may limit our confidence in generalizing findings to non-college populations. While college students are indeed a high-risk population for heavy drinking and alcohol-related problems, non-college young adults also engage in high-risk drinking (Linden-Carmichael and Lanza, 2018); therefore, intervention efforts focusing specifically on this subgroup are needed.

Conclusion

Results of this study suggest that improvements in insomnia may lead to improvements in some theoretical and empirical correlates of alcohol-related harm; specifically, alcohol craving and delay discounting of large rewards. The potential for insomnia treatment to positively influence these constructs is promising, as they map onto two neurobiological domains that are salient in the binge/intoxication and preoccupation/anticipation stages of addiction, respectively (Kwako et al., 2017). Given the limitations of the data presented here, additional research on the potential for insomnia treatment to improve difficulties with emotion regulation is warranted. More accurately and effectively targeting these proposed mechanisms of behavior change is expected to alleviate some of the health burden associated with problematic alcohol use among young adults.

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Table 1

Group demographics at baseline (N=56).

	Total sample (*N* = 56)	CBT-I (*N* = 28)	Sleep Hygiene (*n* = 28)		
	***n* (%)**	***n* (%)**	***n* (%)**	**χ^2 (df)**	***p***
Female (vs. male)	42 (75%)	22 (79%)	20 (71%)	0.38 (1)	.54
Race	---	---	---	---	---
European American	46 (82%)	24 (86%)	22 (79%)	0.49 (1)	.49
African American	3 (5%)	1 (4%)	3 (11%)	1.08 (1)	.30
Asian American	0 (0%)	0 (0%)	0 (0%)	---	---
Nat. Am. or Nat. Al.	1 (2%)	1 (4%)	0 (0%)	1.02 (1)	.31
Nat. Haw. or Pac. Isl.	0 (0%)	0 (0%)	0 (0%)	---	---
Two or more races	5 (9%)	2 (7%)	3 (11%)	0.22 (1)	.64
Hispanic or Latino/a/x	2 (4%)	2 (7%)	0 (0%)	2.07 (1)	.15
Highest level of education	---	---	---	---	---
Grade 12 or GED	3 (5%)	2 (7%)	1 (4%)	0.35 (1)	.55
Some college/tech. school	36 (64%)	19 (68%)	17 (61%)	0.31 (1)	.58
College graduate	17 (30%)	7 (25%)	10 (36%)	0.76 (1)	.38
Current college enrollment	41 (73%)	21 (75%)	20 (71%)	0.09 (1)	.76
Fraternity member/pledge	15 (27%)	6 (29%)	9 (45%)	1.19 (1)	.28
Use of sleep medication[1]	18 (32%)	11 (39%)	7 (25%)	1.31 (1)	.25
Aleve PM	1 (2%)	0 (0%)	1 (4%)	1.02 (1)	.31
Diphenhydramine	7 (13%)	3 (11%)	4 (14%)	0.16 (1)	.69
Doxylamine	2 (4%)	0 (0%)	2 (7%)	2.07 (1)	.15
Melatonin	12 (21%)	9 (32%)	3 (11%)	3.82 (1)	.05
Trazodone	1 (2%)	0 (0%)	1 (4%)	1.02 (1)	.31
	***M* (*SD*)**	***M* (*SD*)**	***M* (*SD*)**	***t* or *F* (df)**	***p***
Age	22.4 (2.7)	22.3 (2.7)	22.5 (2.8)	0.34 (54)	.74
Insomnia severity	16.2 (3.5)	16.4 (4.1)	15.9 (2.9)	−0.53 (54)	.60
Drinks per week	12.6 (7.3)	12.1 (6.0)	13.1 (8.4)	0.49 (54)	.62
Alcohol consequences	7.1 (5.3)	8.3 (6.5)	5.9 (3.6)	−1.68 (42[1])	.10

Note.

[1]Two sleep hygiene participants reported use of 2 sleep medications on baseline diaries.

Nat Al = Native Alaskan. Nat Am = Native American. Nat Haw = Native Hawaiian. Pac Isl = Pacific Islander. Tech. = technical.

Table 2

Descriptive and inferential statistics for secondary trial outcomes (N=56).

	Baseline		Post-Treatment		1 Month		Group		Time		G x T		Cohen's *d* (95% CI)	
	M	SD	M	SD	M	SD	F	p	F	p	F	*p*	BL to post	BL to 1mo
Alcohol craving	---	---	---	---	---	---	0.52	.48	3.71	.03	3.58	.03	0.33	−0.19
CBT-I	5.75	4.27	3.61 *W*	5.14	5.00	3.74							(−0.20, 0.85)	(0.00, 0.70)
Sleep Hygiene	6.00	3.29	5.69	4.06	4.69 *W*	3.20								
Delay discounting	---	---	---	---	---	---	1.80	.18	9.01	<.001	2.19	.12	0.10	0.37
CBT-I	0.04	0.05	0.03	0.02	0.04	0.03							(−0.42, 0.62)	(−0.16, 0.90)
Sleep Hygiene	0.03	0.04	0.02	0.02	0.04	0.03								
Negative affect	---	---	---	---	---	---	0.26	.61	1.61	.21	0.03	.97	−0.05	−0.06
CBT-I	15.86	5.71	16.26	7.24	15.33	5.44							(0.00, 0.36)	(0.00, 0.39)
Sleep Hygiene	15.39	4.43	15.65	5.87	14.61	4.11								
DERS	---	---	---	---	---	---	5.77	.02	10.75	<.001	1.92	.16	0.45	0.07
CBT-I	86.71 *B*	23.22	73.84 *W*	13.80	75.89 *WB*	20.28							(−0.08, 1.00)	(−0.45, 0.60)
Sleep Hygiene	74.32 *B*	16.95	68.53	16.90	64.85 *WB*	17.02								

Note. Cohen's *d* interpreted as 0.20 small, 0.50 medium, and 0.80 large (negative effect size indicates change in favor of the control group). 1mo = 1-month follow-up. BL = baseline. CI = confidence interval. DERS = Difficulties with Emotion Regulation scale. G X T = group by time interaction. Post = post-treatment. SD = standard deviation.

W =significant (p<.05) within-group change from baseline.

B =significant (p<.05) between-group difference at that time point.

Table 3

Descriptive and inferential statistics for delay discounting subscales (N=56).

	Baseline		Post-Treatment		1 Month		Group		Time		G x T		Cohen's *d* (95% CI)	
	M	**SD**	**M**	**SD**	**M**	**SD**	**F**	**p**	**F**	**p**	**F**	***p***	**BL to post**	**BL to 1mo**
Small K	---	---	---	---	---	---	3.26	.08	14.39	<.001	0.36	.70	0.11	0.24
CBT-I	.054	.061	.040 *B*	.034	.066	0.43							(−0.41, 0.64)	(−0.28, 0.77)
Sleep Hygiene	.032	.037	.024 *B*	.020	.058 *W*	.058								
Medium K	---	---	---	---	---	---	1.36	.25	4.53	.01	0.86	.43	−0.12	0.01
CBT-I	.041	.051	.035	.031	.041	.036							(0.00, 0.61)	(−0.51, 0.54)
Sleep Hygiene	.034	.060	.020	.028	.035	.035								
Large K	---	---	---	---	---	---	3.20	.08	1.23	.30	12.36	<.001	−0.01	0.42
CBT-I	.036	.050	.028 *B*	.020	.018 *W*	.016							(0.00, 0.06)	(−0.12, 0.94)
Sleep Hygiene	.020	.036	.013 *B*	.011	.022	.022								

Note. Significant effects depicted in bold. Cohen's *d* interpreted as 0.20 small, 0.50 medium, and 0.80 large (negative effect size indicates change in favor of the control group). 1mo = 1-month follow-up. BL = baseline. CI = confidence interval. G X T = group by time interaction. Post = post-treatment. SD = standard deviation.

W =significant (p<.05) within-group change from baseline.

B =significant (p<.05) between-group difference at that time point.

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Table 4

Descriptive and inferential statistics for difficulties with emotion regulation subscales (N=56).

	Baseline		Post-Treatment		1 Month		Group		Time		G x T		Cohen's *d* (95% CI)	
	M	SD	M	SD	M	SD	F	p	F	p	F	*p*	BL to post	BL to 1mo
Non-accept	---	---	---	---	---	---	1.69	.20	3.34	.04	2.03	.14	0.49	0.18
CBT-I	13.54	5.34	11.03 *W*	4.20	11.80	5.42							(−0.04, 1.02)	(−0.34, 0.71)
Sleep Hygiene	11.07	4.41	10.69	4.84	10.25	4.71								
Goals	---	---	---	---	---	---	1.57	.22	5.45	.01	0.22	.81	0.11	−0.03
CBT-I	14.46	5.03	12.67 *W*	3.37	12.51 *W*	3.58							(−0.41, 0.63)	(0.00, 0.20)
Sleep Hygiene	13.32	4.61	12.01	4.28	11.22 *W*	3.42								
Impulse control	---	---	---	---	---	---	5.16	.03	2.20	.12	3.37	.04	0.54	0.16
CBT-I	11.36 *B*	6.12	9.19 *W*	2.24	9.61 *B*	3.76							(0.001, 1.07)	(−0.37, 0.68)
Sleep Hygiene	8.89 *B*	3.06	9.14	2.41	7.88 *B*	1.65								
Awareness	---	---	---	---	---	---	1.98	.17	6.06	.004	0.51	.60	0.25	0.23
CBT-I	16.82	5.42	15.70 *W*	4.45	15.22 *W*	5.23							(0.00, 0.76)	(0.00, 0.74)
Sleep Hygiene	15.93	5.51	13.66	4.83	13.23	5.38								
Strategies	---	---	---	---	---	---	4.98	.03	6.92	.002	2.82	.07	0.60	0.15
CBT-I	18.68 *B*	7.15	14.92 *W*	4.00	15.69 *W*	5.57							(0.05, 1.13)	(−0.27, 0.78)
Sleep Hygiene	14.68 *B*	5.14	13.71	4.19	13.25	4.64								
Clarity	---	---	---	---	---	---	4.63	.04	5.05	.01	0.89	.42	0.13	−0.20
CBT-I	11.86	3.71	10.33 *W*	2.81	11.06 *B*	2.75							(−0.39, 0.66)	(0.00, 0.71)
Sleep Hygiene	10.43	3.55	9.32	3.52	9.02 *WB*	3.05								

Note. Significant effects depicted in bold. Cohen's *d* interpreted as 0.20 small, 0.50 medium, and 0.80 large (negative effect size indicates change in favor of the control group). 1mo = 1-month follow-up. BL = baseline. CI = confidence interval. G X T = group by time interaction. Post = post-treatment. SD = standard deviation.

W =significant (p<.05) within-group change from baseline.

B =significant (p<.05) between-group difference at that time point.