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A randomized clinical trial examining the effects of an anxiety sensitivity intervention on insomnia symptoms: Replication and extension

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Abstract

Insomnia disorder is impairing and prevalent, particularly among individuals with comorbid anxiety disorders. Despite the availability of effective computerized treatments for insomnia, there are few that target both insomnia as well as co-occurring anxiety symptoms. The current study tests the efficacy of a computerized treatment for anxiety sensitivity cognitive concerns, a transdiagnostic risk factor for anxiety, mood, and insomnia symptoms, against a repeated contact control, on reducing insomnia symptoms. Hypotheses were tested in a mixed clinical sample of community individuals presenting for a treatment study ($n = 151$) who were followed up 1-, 3- and 6-months after treatment. Results indicated that the anxiety sensitivity intervention resulted in reductions in insomnia symptoms and clinically significant insomnia scores at 3- and 6-month follow-ups. These reductions remained significant when covarying for concurrent reductions in depression and anxiety. Models accounted for 15–54% of the variance in follow-up insomnia symptoms. Current findings add to a growing body of literature suggesting anxiety sensitivity may play a causal role in insomnia symptoms. Results also suggest that targeting anxiety sensitivity may be an effective way to reduce insomnia symptoms in a brief and portable intervention that also reduces symptoms commonly comorbid with insomnia disorder.

Keywords

Insomnia; Anxiety sensitivity; Sleep; Computerized treatment

1. Introduction

Nearly one third of the general population reports occasional difficulties initiating or maintaining sleep (Breslau, Roth, Rosenthal, & Andreski, 1996; Ohayon, 2002), symptoms which are often associated with insomnia disorder. A smaller but still substantial minority of the population (9–15%) experience daytime dysfunction caused by their insomnia

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symptoms, such as irritability, fatigue, and dysphoric mood (Mai & Buysse, 2008; Morin & Jarrin, 2013; Ohayon, 2002). Significant distress and/or impairment related to difficulties initiating or maintaining sleep, paired with daytime dysfunction, may meet criteria for a formal diagnosis of insomnia disorder. It is estimated that 6% of the population meet criteria for insomnia disorder diagnoses, which is associated with several negative consequences, including difficulty functioning, absence from work, problems with concentration and memory, irritability, and poorer quality of life (Ancoli-Israel & Roth, 1999; Mai & Buysse, 2008). Considering the prevalence of insomnia symptoms among the general population, there is a significant need for researchers to continue to understand the most efficacious and effective ways to deliver treatment for insomnia.

Fortunately, psychological treatments, such as cognitive behavioral therapy for insomnia (CBT-I) effectively reduce insomnia symptoms (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001). However, there are many barriers to disseminating these services, including the amount of time and cost associated with treatment. This calls into question whether there are less expensive, briefer interventions that are effective for reducing symptoms of insomnia disorder. Indeed, brief and portable interventions for insomnia disorder have been tested in two primary formats. A shortened six-week, online format of CBT-I demonstrated improved insomnia symptoms compared to a waitlist control in a sample of adults with primary chronic insomnia (Tan et al., 2012). Additionally, a 6-week CBT-I protocol combined with mindfulness techniques produced significant decreases in both insomnia symptoms and secondary trait measures of arousal (Ong, Shapiro, & Manber, 2008). Recent studies suggest even briefer treatments such as one-session administrations of CBT-I can reduce insomnia (Ellis, Cushing, & Germain, 2015; Swift et al., 2012). Together, these studies suggest that brief psychological treatments effectively reduce insomnia disorder symptoms, and that transdiagnostic interventions can address insomnia and comorbid symptoms such as anxiety-related arousal. However, only a small body of literature has explored targeting insomnia through brief, transdiagnostic interventions.

One such transdiagnostic risk factor associated with insomnia symptoms is anxiety sensitivity (AS). Broadly defined as a fear of negative consequences associated with anxious arousal (Reiss, Peterson, Gursky, & McNally, 1986), individuals with elevated AS tend to hold exaggerated negative beliefs and concerns about the adverse cognitive (e.g., difficulty concentrating means one is “losing their mind”), physical (e.g., a racing heart is a sign of an impending heart attack), and social (blushing or shaking when nervous is embarrassing) consequences of anxiety (Taylor et al., 2007; Zinbarg, Barlow, & Brown, 1997). AS is known as a transdiagnostic risk factor given its robust relationship to various psychiatric conditions, including depression (Cox, Enns, & Taylor, 2001), eating pathology (Anestis, Holm-Denoma, Gordon, Schmidt, & Joiner, 2008), posttraumatic stress (Short et al., 2017a), suicidal ideation (Capron et al., 2012), substance use (Assayag, Bernstein, Zvolensky, Steeves, & Stewart, 2012; Zvolensky et al., 2009), and various anxiety disorders (Naragon-Gainey, 2010; Short, Fuller, Norr, & Schmidt, 2017b; Taylor, Koch, & McNally, 1992). Relevant to the present study, several investigations have shown AS is positively related to insomnia symptoms (Babson, Trainor, Bunaciu, & Feldner, 2008; Hoge et al., 2011; Short, Allan, Raines, & Schmidt, 2015; Vincent & Walker, 2001).

There are several theoretical pathways by which AS may contribute to insomnia symptoms. Because AS is posited to amplify anxious arousal, it may contribute to overarousal and sleep difficulties during sleep onset. To this end, elevated AS is associated with increased sleep onset latency among anxious youth (McNally & Eke, 1996) and individuals with panic disorder (Hoge et al., 2011). Consistent with the idea that AS amplifies anxious arousal at bedtime, Babson et al. (2008) examined the moderating role of AS on the association between increased sleep anticipatory anxiety and longer sleep onset latency. Results indicated that the association between elevated sleep anticipatory anxiety and longer sleep onset latency was stronger as AS physical concerns increased.

In addition to physical concerns, AS cognitive concerns may result in a tendency for individuals with insomnia to attend to and catastrophize daytime insomnia symptoms, such as fatigue, decreased alertness, and problems with concentration and memory. In fact, AS cognitive concerns were uniquely related to daytime sleep-related impairment in adults with chronic insomnia (Vincent & Walker, 2001). In other studies, AS cognitive concerns were a significant predictor of global insomnia symptoms, leading the authors to suggest that distress related to cognitive functioning may be a mechanism by which sleep dysfunction is maintained (Calkins, Hearon, Capozzoli, & Otto, 2012; Capron et al., 2016). As further evidence of this, Taylor, Lichstein, Durrence, Reidel, and Bush (2005) reported that AS cognitive concerns mediated the relationship between the ‘unacceptable thoughts’ domain of obsessive compulsive symptoms and symptoms of insomnia in adults. Taken together, these studies support the role of AS in maintaining insomnia symptoms, suggesting it is a potential therapeutic target in the treatment of insomnia.

To establish AS as a meaningful risk factor for insomnia disorder, AS must be malleable, and affect subsequent change on insomnia symptoms (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). To this first point, brief cognitive-behavioral interventions are capable of reducing AS (Broman-Fulks & Storey, 2008; Feldner, Zvolensky, Babson, Leen-Feldner, & Schmidt, 2008; Gardenswartz & Craske, 2001). Moreover, randomized controlled trials have examined fully computerized AS interventions that comprise (1) a psychoeducation component, designed to normalize the nature and effects of anxious arousal, and (2) interoceptive exposure (IE) exercises for the purpose of habituating to feared physical sensations (Harvey, 2001; Keough & Schmidt, 2012; Schmidt et al., 2007). These interventions are associated with 30–60% reductions in AS (Capron & Schmidt, 2016; Keough & Schmidt, 2012), lower incidence of Axis I diagnoses (Schmidt et al., 2007), and reductions in symptoms of anxiety, depression, and suicide (Harvey, 2001; Schmidt, Capron, Raines, & Allan, 2014). As such, these brief, portable interventions significantly impact AS, with subsequent benefits that extend to symptoms of psychological disorders associated with elevated AS.

In sum, despite the literature indicating that AS may be a malleable factor in the development of maintenance of insomnia symptoms, only one prior study has tested the effects of AS interventions on insomnia symptoms. Short et al. (2015) evaluated participants with elevated AS who were randomly assigned to either an AS cognitive concerns treatment or a physical health control condition (both were fully computerized, one-session interventions). Results indicated the AS intervention reduced symptoms of insomnia through

reductions in AS. However, this prior study suffered from several limitations. First, an abbreviated Insomnia Severity Index (ISI; Morin, Belleville, Blanger, & Ivers, 2011) was used making it difficult to determine whether scores were clinically significant, as well as to compare to other trials in the insomnia literature. Second, there were no other sleep measures to characterize the sample in terms of sleep quality. Third, temporal mediation could not be tested and finally, participants were only followed for 1-month, precluding tests of longer term outcomes.

The current study design improved upon each of the prior limitations by including the full ISI and longer term follow-ups. We also tested temporal mediation and tested whether effects held when covarying for concurrent reductions in depression and anxiety. Consistent with the previous study, we hypothesized there would not be direct effects on insomnia symptoms or clinically significant scores at Month-3 or Month-6. Second, we hypothesized there would be an indirect effect such that the active condition would lead to greater reductions in insomnia symptoms and clinically significant scores at Month-3 and Month-6 through post-treatment reductions in AS. Third, we hypothesized that these indirect effects would hold after covarying for concurrent changes in depression and anxiety. Fourth, we hypothesized that results would be specific to AS and not another transdiagnostic risk factor for insomnia (i.e., negative affect).

2. Method

2.1. Participants

Participants consisted of 151 individuals drawn from a larger sample of participants recruited from the community to participate in a larger randomized clinical trial examining the efficacy of a computerized intervention for AS (NCT01941862). The current results are secondary analyses unrelated to the primary objectives of the original trial. Sample size for the larger trial was determined by power analysis. Participants were recruited between November 2013, and March 2016. Eligible participants were 18 years of age or older, English speakers, and demonstrated elevated levels of at least one suicide risk factor (i.e., AS cognitive concerns, perceived burdensomeness, or thwarted belongingness; Van Orden et al., 2010). Elevated suicide risk factors (e.g., AS cognitive concerns) were required for participation in the larger study; however, participants were not required to have elevated suicidality (i.e., ideation, intent, prior attempts, etc.). Participants were excluded if they showed evidence of a current psychotic and/or bipolar spectrum disorder, or unstable psychiatric medication usage (i.e., participants were required to maintain the same prescription for at least 6 weeks before starting the trial).

For the current study, participants were selected if they participated in one of the two conditions of interest: the active AS condition or the repeated contact control. The other two conditions in the larger study included a mood condition, focusing on reducing perceived burdensomeness and thwarted belongingness (Van Orden et al., 2010), and a combined condition, which received both the mood and AS interventions. We chose to focus only on the AS condition and the control to simplify interpretation of results and because the mood condition theoretically would not influence AS, which results supported.

Participant age ranged from 18 to 74 ($M = 36.35$, $SD = 16.47$). Gender was evenly distributed (54.9% female). Fifty-five percent of the sample identified as White, 34.0% African American, 2.7% Asian/Pacific Islander, 0.7% American Indian/Native American, and 7.8% Other (e.g., bi-racial). Regarding psychiatric diagnoses, 41.8% met criteria for a mood disorder (both depressive and bipolar disorders), 41.8% for at least one anxiety disorder, 48.4% exceeded the clinical cut off on the Insomnia Severity Index, indicating a potential insomnia diagnosis, and 10.5% had no primary diagnosis during the baseline interview. Sleep quality scores on the Pittsburgh Sleep Quality Index (PSQI) at baseline ranged from 6 to 20 ($M = 10.85$, $SD = 2.74$). On average, participants far surpassed the clinical cut-off, with scores above 5 indicative of a 'poor sleeper' (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Specifically, participants reported average sleep efficiencies ranging from 65 to 74%, and using medicine to help them sleep less than once a week according to the Use of Sleep Medication component of the PSQI ($M = 1.06$, $SD = 1.27$).

2.2. Procedure

Participants were recruited from the local Tallahassee community via newspaper advertisements, flyers, and other media outlets from 2013 to 2016. Interested individuals completed a telephone screen before scheduling a baseline appointment to determine eligibility. At the baseline appointment, informed consent was obtained. All procedures took place within the outpatient clinic at Florida State University and our research lab. Participants then completed a battery of self-report questionnaires and a semi-structured clinical interview for the DSM-5 (SCID-5-RV; First, Williams, Karg, & Spitzer, 2015). Next, participants were randomized by research assistants using a random number generator to either an AS treatment condition, a mood-focused condition, a combined condition, or a repeated contact control. For the purposes of the current study, we focused only on participants in the AS treatment condition and repeated contact control.

Participants came to the laboratory once per week over three weeks (i.e., intervention sessions 1–3), then returned to the laboratory one month, three months, and 6 months post-intervention. All self-report measures were administered at each time point (with the exception of during Session 2 of the intervention period, in which a briefer battery of measures was given). All participants provided written informed consent and study procedures were approved by the Florida State University's institutional review board.

2.3. Experimental conditions

Cognitive Anxiety Sensitivity Treatment (CAST) + BIAS.—The CAST + BIAS protocol comprised two components, each targeting AS cognitive concerns. The first, completed during participant's first intervention session, was described in detail by Schmidt et al. (2014). This intervention combines computerized psychoeducation regarding the physiological effects of anxious arousal and a hyperventilation interoceptive exposure exercise.

The second, completed during all three intervention appointments, was a computerized cognitive bias modification paradigm for interpretation bias (CBM-I) known as Bias Interpretation for Anxiety Sensitivity (BIAS), described in detail by previous studies

(Capron & Schmidt, 2016; Capron, Norr, Allan, & Schmidt, 2017). During the CBM-I component, participants were shown an ambiguous one or two word phrase (e.g., “tingly”) for 1 s, followed by a sentence that resolved the word-sentence pairing to have either an interpretation that is either benign (e.g., “You lightly bang your elbow and it feels funny”) or threatening (e.g., “Something is terribly wrong with you”). Benign and threat resolutions were randomized according to a predetermined schedule, such that half of the total trials had a benign resolution, and half a threatening resolution. Participants were then asked to indicate whether the word-sentence pair was related, for which they were provided corrective feedback to encourage benign interpretations. Participants in the CAST + BIAS condition only received suicide risk assessments and preventative strategies if necessary to ensure their safety. Specifically, they completed risk assessments if they began the study at moderate or higher risk for suicide, or endorsed questionnaires at any time point in a manner that suggested suicide risk (i.e., Beck Depression Inventory – 2nd Edition, item regarding suicide was rated at least 1 [*I have thoughts of suicide but would not carry them out*]).

Repeated Contact Control (RCC).—Participants randomized to the RCC reported to the laboratory for weekly appointments over three consecutive weeks. During these appointments, participants completed the same battery of self-report measures. In addition, participants participated in a suicide risk assessment, and appropriate preventative strategies were taken for individuals with elevated risk (Chu et al., 2015).

2.4. Measures

Structured Clinical Interview for DSM-5 Disorders- Research Version (SCID-5-RV).—Psychiatric diagnoses were determined using the SCID-5-RV (First et al., 2015). SCIDs were administered by doctoral level students who completed SCID training, including reviewing SCID training tapes, observing live SCID administrations, and practice interviews with other trained individuals. Agreement between clinical interviewers for a random sample of SCID interviews from our laboratory resulted in high inter-rater agreement (i.e., over 80% with a kappa value of 0.86; $n = 20$; Harvey, 2001).

Anxiety Sensitivity Index-3 (ASI-3).—The ASI-3 is an 18-item self-report measure designed to index the feared consequences of symptoms associated with anxious arousal (Reiss et al., 1986; Taylor et al., 2007). Participants report on a 5-point Likert-style scale how much they agree with each statement. The ASI-3 comprises 3 lower-order subscales, including physical (e.g., “It scares me when my heart beats rapidly”), cognitive (e.g., “It scares me when I am unable to keep my mind on a task”), and social concerns (e.g., “It scares me when I blush in front of people”). The ASI-3 is psychometrically sound (Taylor et al., 2007). The ASI-3 total scores demonstrated good to excellent reliability at the baseline and Month-1 (α s both = 0.94).

Insomnia Severity Index (ISI).—The ISI is a brief 7-item self-report measure assessing insomnia symptoms and severity (e.g., problems with falling asleep, staying asleep, waking too early), satisfaction/dissatisfaction with sleep patterns, and/or interference with daily functioning (Morin et al., 2011). Participants were asked to rate each item using a 5-point Likert-type scale ranging from 0 to 4, with higher scores indicating more severe sleep

difficulties. Previous research has demonstrated high internal consistency for the ISI (Morin et al., 2011). Additionally, the ISI demonstrated good psychometric properties, such as good internal consistency at both baseline, one-month follow-up, month-three follow-up, and month-six follow-up (α s = 0.90, 0.92, 0.94, and 0.93 respectively).

Pittsburgh Sleep Quality Index (PSQI).—The PSQI is a 19-item self-report measure of sleep quality (Buysse et al., 1989). Specifically, the PSQI measures 7 components of sleep quality: duration of sleep, sleep disturbances, sleep onset latency, daytime dysfunction, sleep efficiency, subjective sleep quality, and use of sleep medications. The PSQI has demonstrated excellent psychometric properties in previous research, such as good reliability and validity, and the ability to differentiate between good and poor sleepers (Buysse et al., 1989). In the current study, the PSQI demonstrated good internal consistency at baseline (α = 0.81).

Positive and Negative Affect Schedule – Trait – NA Subscale (PANAS-NA).—The NA subscale of the PANAS comprises ten words describing negative emotions (Watson, Clark, & Tellegen, 1988). Respondents indicate to what extent they feel these individual emotions, in general, on a 5-point Likert-style scale. The PANAS has demonstrated strong psychometric properties (Watson & Clark, 1994). In the present investigation, the PANAS-NA demonstrated excellent reliability at baseline, month one, and month three (α s = 0.90, 0.92, and 0.92 respectively).

Beck Depression Inventory – II (BDI-II).—The BDI-II is a 21-item self-report inventory of depressive symptoms (Beck, Steer, & Carbin, 1988). Participants read a group of statements and select which one best represents how they have felt over the past 2 weeks. These statements are scored using a 4-point Likert scale ranging from 0 to 3, with higher scores reflecting greater severity. In previous research, the BDI-II has demonstrated good psychometric properties, including strong internal consistency and good test-retest reliability (Beck et al., 1988). Internal consistency in the present sample was excellent at baseline, month three, and month six (α s = 0.94, 0.95, and 0.95 respectively).

Beck Anxiety Inventory (BAI).—The BAI is a 21-item questionnaire assessing the experience of anxiety symptoms (Beck & Steer, 1993). Participants rate how often they experience these symptoms (e.g., feeling nervous, shaky, scared, faint, etc.) on a 4-point Likert scale ranging from 0 (*not at all*) to 3 (*severely*). Previous research has established the BAI to be a psychometrically sound measure of anxiety, with good internal consistency, test-retest reliability, and construct validity. In the current study, internal consistency was excellent at baseline, month three, and month six (α s = 0.93, 0.93, and 0.94 respectively).

Data Analytic Plan.—Mediation analyses were conducted in Mplus version 7 (Muthén & Muthén, 1998–2012) to examine the impact of the intervention on ISI scores through ASI-3 total scores. Separate analyses were conducted to examine the impact of the intervention on Month-3 and Month-6 ISI scores through Month-1 ASI scores, covarying for Baseline ISI and ASI-3 scores. Similar analyses were conducted with Month-3 and Month-6 insomnia diagnoses (as assessed by the ISI) as outcome variables. We then replicated these analyses controlling for residualized BDI and BAI change scores. Specifically, Month-3 or Month-6

scores were regressed on baseline scores, and standardized residuals were included as a covariate in the main analyses. We elected to use change scores as a covariate to provide a more stringent test versus relying on Baseline or Month-3/6 measures alone. Finally, we conducted similar analyses using negative affect as an alternative mediator to test specificity of anxiety sensitivity as a mediator.

Model fit was assessed using the likelihood ratio test, based on the χ^2 value with a nonsignificant χ^2 value indicating good model fit. In addition, the comparative fit index (CFI) and root mean square error of approximation model fit indices were provided. In general, CFI values greater than 0.95 and RMSEA values less than 0.05 indicate good fit (Hu & Bentler, 1999; MacCallum, Browne, & Sugawara, 1996). Historically, significant direct effects were considered necessary for testing mediation models (Baron & Kenny, 1986). However, current conceptualizations accept that direct effects are not necessary, or even likely, especially when the outcome is not expected to be influenced directly (Hayes, 2009; Shrout & Bolger, 2002). Mediation models were conducted using maximum-likelihood estimation and asymmetric bias-corrected bootstrapped confidence intervals (CIs) with 5000 bootstrapped CIs to provide results that are both consistent and replicable (Preacher & Hayes, 2008).

3. Results

3.1. Descriptive statistics and correlations

As depicted in Fig. 1, 9 individuals in the control condition did not complete their Month-3 follow-up, and 16 did not complete their Month-6 follow-up. In the active condition, 13 did not complete their Month-3 follow-up and 19 did not complete their Month-6 follow-up. We used Maximum Likelihood estimation in MPlus, which includes all participants in analyses with complete data for predictors. We examined whether those who completed their Month-6 assessment differed from those who did not in terms of gender, age, anxiety sensitivity, insomnia symptoms, negative affectivity, depression, anxiety, and treatment condition, and there were no significant differences (all $ps > 0.328$). Baseline levels of all variables were also compared across condition using t-tests, and no significant differences were found (all $ps > 0.536$), suggesting equivalence of random assignment (see Table 1). Examination of skew and kurtosis indicated that there was no significant skew or kurtosis. In terms of means at baseline, our sample presented with clinical levels of insomnia as suggested by ISI scores averaging around 15 (Morin et al., 2011). At baseline, 47% of the active condition and 53% of the control condition exceeded the clinical cut-off of the ISI, whereas at Month-3, this decreased to 25% in the active condition and 31% of the control. Finally, at Month-6, 20% in the active condition and 29% in the control condition exceeded the clinical cut-off of the ISI. For anxiety and depression, sample means were also in the moderate range (Beck & Steer, 1993; Beck et al., 1988). All correlations were in the expected directions, with significant positive associations found between all variables of interest.

Direct Effects of Anxiety Sensitivity and Treatment Condition on Insomnia

Symptoms.—First, we used regression to identify the direct effects of anxiety sensitivity

and treatment condition on insomnia symptoms. Results indicated that after covarying for baseline insomnia symptoms, condition did not have a significant direct effect on Month-3 insomnia symptoms ($B = -0.30$, $SE = 1.07$, $p = 0.779$, $sr^2 < 0.01$, 95% CI $[-2.41, 1.79]$) or clinical cut-offs ($B = -0.02$, $SE = 0.07$, $p = 0.84$, $sr^2 < 0.01$, 95% CI $[-0.16, 0.13]$). In addition, there was not a direct effect of the intervention on Month-6 insomnia symptoms ($B = 1.41$, $SE = 1.21$, $p = 0.246$, $sr^2 = 0.01$, 95% CI $[-3.97, 4.65]$) or clinical cut-offs ($B = -0.03$, $SE = 0.08$, $p = 0.733$, $sr^2 < 0.01$, 95% CI $[-0.02, 0.20]$). However, insomnia symptoms were concurrently associated with anxiety sensitivity at Month-3 ($B = 0.12$, $SE = 0.03$, $p = 0.001$, $sr^2 = 0.06$), and Month-6 ($B = 0.13$, $SE = 0.04$, $p = 0.001$, $sr^2 = 0.06$). These models accounted for 37.8% of the variance in insomnia symptoms and 14.9% of the variance in clinical cut-offs at Month-3, and 41.3% and 20.4% of the variance in insomnia symptoms and clinical cut-offs at Month-6, respectively.

Indirect Effects on Insomnia Symptoms and Clinical Cut-Offs.—A total of four separate analyses were conducted to examine the indirect effect of condition on insomnia symptoms (at Months 3 and 6) and clinical cut-off (at Months 3 and 6) through anxiety sensitivity (at Month 1), covarying for baseline insomnia symptoms and anxiety sensitivity. Model fit was acceptable to good for most indices for models examining Month-3 insomnia symptoms ($\chi^2 = 9.91$, $p = 0.007$, CFI = 0.94, RMSEA = 0.18) and clinical cut-off ($\chi^2 = 1.51$, $p = 0.50$, CFI = 1.00, RMSEA = 0.00), as well as Month-6 insomnia symptoms ($\chi^2 = 2.84$, $p = 0.24$, CFI = 0.99, RMSEA = 0.06) and clinical cut-off ($\chi^2 = 0.65$, $p = 0.72$, CFI = 1.00, RMSEA = 0.00). For all models, baseline ISI or clinical cut-off and post-treatment anxiety sensitivity significantly predicted outcomes (see Table 2). Furthermore, an indirect effect was found in all models such that there was a significant indirect effect of condition on Month-3 and 6 insomnia symptoms and clinical cut-off through anxiety sensitivity. These models accounted for 39.6% and 22.2% of the variance in insomnia symptoms and cut-offs at Month-3, respectively, and 43.5% and 25.7% of the variance in insomnia symptoms and cut-offs Month-6, respectively.

Indirect Effects on Insomnia Symptoms and Clinical Cut-Offs Covarying for Concurrent Changes in Depression and Anxiety.—Next, we conducted an additional four separate analyses identical to the prior four, but this time covarying for concurrent changes in depression and anxiety (i.e., for Month-3 insomnia symptoms, we covaried for changes in depression and anxiety from Baseline to Month-3). Again, model fit was acceptable to good for most indices for models examining Month-3 insomnia symptoms ($\chi^2 = 7.83$, $p = 0.02$, CFI = 0.96, RMSEA = 0.15) and clinical cut-off ($\chi^2 = 0.96$, $p = 0.62$, CFI = 1.00, RMSEA = 0.00), as well as Month-6 insomnia symptoms ($\chi^2 = 3.69$, $p = 0.16$, CFI = 0.98, RMSEA = 0.08) and clinical cut-off ($\chi^2 = 0.25$, $p = 0.88$, CFI = 1.00, RMSEA = 0.00). In general, model fit was similar to analyses with or without covariates. Changes in depression and anxiety significantly predicted Month-3 insomnia symptoms, but did not consistently predict the other insomnia outcomes. Overall, these analyses were similar to those without covarying for changes in depression and anxiety. Specifically, there was an indirect effect of condition on all insomnia outcomes through anxiety sensitivity. These models accounted for 51.2% and 41.0% of the variance in insomnia symptoms and cut-offs

at Month-3, respectively, and 54.2% and 26.6% of the variance in insomnia symptoms and cut-offs Month-6, respectively (see Table 3).

Specificity Tests Using Negative Affect as an Alternative Mediator.—Finally, we conducted four separate analyses similar to those above, but using negative affect as a rival mediator. In these analyses, we did not covary for concurrent changes in depression and anxiety. Model fit was acceptable to good for most indices for models examining Month-3 insomnia symptoms ($\chi^2 = 5.45$, $p = 0.06$, CFI = 0.98, RMSEA = 0.12) and clinical cut-off ($\chi^2 = 0.77$, $p = 0.68$, CFI = 1.00, RMSEA = 0.00), as well as Month-6 insomnia symptoms ($\chi^2 = 3.98$, $p = 0.14$, CFI = 0.98, RMSEA = 0.09) and clinical cut-off ($\chi^2 = 0.44$, $p = 0.80$, CFI = 1.00, RMSEA = 0.00). Model fits were similar to analyses with anxiety sensitivity as a mediator. Negative affect was not a significant mediator of condition on Month-3 insomnia symptoms ($B = -0.44$, 95% CI [-1.27, 0.23]) or clinical cut-off ($B = -0.03$, 95% CI [-0.09, 0.02]), or Month-6 insomnia symptoms ($B = -0.02$, 95% CI [-1.23, 0.08]) or clinical cut-off ($B = -0.02$, 95% CI [-0.08, 0.01]).

4. Discussion

The current findings expand upon previous research suggesting targeting AS may ameliorate symptoms of insomnia. Specifically, results indicate that an AS-focused intervention, vs. a repeated contact control, reduces insomnia symptoms and diagnoses through reductions in AS. Results are consistent with prior research indicating that a computerized, AS focused interventions result in reduced insomnia symptoms via changes in AS at one month post-treatment (Short et al., 2015). However, the current study provides new information by suggesting these reductions were durable through a six month follow-up. Furthermore, unlike in prior studies, we were able to examine whether individuals met or exceeded the clinical cut-off on the ISI due to administering the full ISI. Findings indicated that CAST + BIAS resulted in decreased clinically significant insomnia scores through reductions in AS. Indeed, mean ISI scores went from exceeding the clinical cut-off at baseline to below it at Months 3 and 6 for the active group. Overall, this suggests that AS interventions may have fairly durable and clinically relevant effects on insomnia symptoms.

We also felt it was important to examine the specificity of AS as a mediating factor in reducing insomnia symptoms because insomnia is commonly comorbid with depression and anxiety (Ohayon, Caulet, & Lemoine, 1998), and AS interventions decrease symptoms of depression and anxiety (Schmidt, Norr, Allan, Raines, & Capron, 2017). As such, it is possible that reductions in insomnia could be due simply to reductions in depression and anxiety. However, after covarying for reductions in depression and anxiety, the indirect effect of CAST + BIAS on insomnia symptoms remained significant. This suggests that, above and beyond shared associations with depression and anxiety, AS may be a causal factor in insomnia symptoms (Babson et al., 2008; Calkins et al., 2012; Vincent & Walker, 2001). We also examined negative affect as an alternative mediator. Results suggested that NA was not a significant mediator of the relationship between CAST + BIAS and reductions in insomnia symptoms. These results provide further confidence for the specificity of AS as a treatment target for insomnia symptoms.

Consistent with prior research, there were no direct effects of CAST + BIAS on insomnia symptoms (Short et al., 2015). Although direct effects were traditionally considered necessary to evaluate indirect effects, more recent conceptualizations of mediation suggest that direct effects are not necessary, or even likely, when examining more distal intervention outcomes (Hayes, 2009; Shrout & Bolger, 2002). Indeed, many studies find that the effects of AS interventions on distal outcomes, such as depression, anxiety, and suicidality, are indirect effects mediated by reductions in AS (Schmidt et al., 2014, 2017). In sum, when the intervention reduces AS, its primary target, it also reduces insomnia symptoms.

Taken together, results are consistent with the idea that AS may fit into existing theoretical models of insomnia (Harvey, 2002). Specifically, it is likely that AS amplifies pre-sleep arousal by magnifying the effects of conditioned arousal and pre-sleep anxiety. For example, many individuals with insomnia report anxiety and tension when trying to fall asleep (Harvey, 2002). This existing arousal would be increased for individuals with fears of sensations associated with anxiety, making the individual even less likely to fall asleep easily. It is also likely that AS plays a role in daytime symptoms of insomnia. For example, many individuals may feel their daytime performance and concentration is impaired by their difficulty sleeping (Harvey, 2002). These perceptions may become more anxiety-inducing for individuals who believe being unable to concentrate is a sign of significant cognitive dysfunction. In these ways, AS may serve to amplify the spiral of distress and arousal associated with the maintenance of insomnia. However, future research is needed to test these specific hypotheses.

The current study also has clinical implications. In extension of prior research (Short et al., 2015), the present study further suggests that CAST + BIAS may have utility for clients suffering from both subclinical and clinically-significant insomnia. While many effective treatments for improving insomnia symptoms exist (e.g., CBT-I), these interventions often target the client's sleep directly and require them to make potentially challenging and temporarily uncomfortable changes to their sleep pattern. This approach is typically tolerable for the majority of clients. However, for some clients, this direct approach may be difficult to tolerate, and might contribute to dropout from CBT-I (Ong, Kuo, & Manber, 2008). In contrast, participants receiving CAST were not required to make significant sleep behavior changes in order to ameliorate difficulties sleeping despite the relative severity of their insomnia. Moreover, CAST + BIAS also reduces symptoms of anxiety and depression, which are commonly comorbid with insomnia (Ohayon & Roth, 2003). Thus, CAST + BIAS may be an ideal intervention for those with low treatment motivation, comorbid anxiety/depression symptoms, or severe insomnia who are not willing to engage in treatments directly targeting their sleep. Importantly, CAST has also demonstrated high patient acceptability (Norr, Gibby, Fuller, Portero, & Schmidt, 2017; Short et al., 2017b) and potential for dissemination (Norr et al., 2017), further underscoring CAST's potential clinical utility.

These findings should be considered in light of several limitations. First, all data relied on self-reported symptomology, which is subject to biased reporting and limited by participants' insight. Future research should assess each construct using multiple levels of analysis, including clinical interviews and objective measures (e.g., polysomnography/

actigraphy) to better assess sleep (Chesson et al., 1997; Westermeyer et al., 2007). Specifically, it is important to note that our self-reported, retrospective measure of insomnia has several limitations in its assessment of insomnia symptoms. Although the ISI is a well-validated measure (Morin et al., 2011), there are problems with the accuracy of retrospective measures of insomnia in comparison to prospective measures (Hartmann, Carney, Lachowski, & Edinger, 2015), which may be magnified in samples with psychiatric disorders. The use of prospective measures of sleep (e.g., the Consensus Sleep Diary; Carney et al., 2012) could overcome this limitation in future research. Furthermore, although the ISI can determine whether individuals have clinically significant insomnia symptoms utilizing cut scores, it cannot determine whether other sleep disorders are present, which could potentially confound the ISI. Clinical interviews of sleep would be beneficial for future research as these can also assist in ruling out other sleep disorders potentially confounded with insomnia (e.g., the Duke Structured Interview for Sleep Disorders; Edinger et al., 2004). Second, the sample included individuals with a range of insomnia severities and comorbid psychopathologies. While future research should evaluate if these findings generalize to patients with only insomnia, it is important to note the significant comorbidity rates of insomnia with anxiety and/or depression (Taylor et al., 2005), which suggests that these comorbidities are a norm rather than exception. Third, the PSQI was only administered at the pre-treatment appointment, preventing the evaluation of the influence of CAST + BIAS on specific facets of sleep quality (e.g., sleep efficiency, use of sleep medications, etc.), leaving a gap in our understanding of these effects which future research should seek to address. Fourth, future research should utilize more advanced statistical techniques such as latent difference score models and growth curve techniques to have the ability to examine the bidirectional associations between reductions in AS and insomnia symptoms. Similarly, we did not specify insomnia as a latent variable due to mixed results about its factor structure from prior studies (e.g., Bastien, Vallieres, & Morin, 2001; Chung, Kan, & Yeung, 2011), but future research should examine this to improve upon the current techniques by accounting for measurement error.

Despite these limitations, the current study adds to the literature on the role of AS in the development and maintenance of insomnia symptoms, as well as the utility of targeting AS to treat insomnia. The current results replicate findings that targeting AS may be a brief and effective strategy for reducing insomnia symptoms and diagnoses among individuals with mood and anxiety disorders. Considering the transdiagnostic, brief, and portable nature of the CAST + BIAS intervention, future research into this area would be a valuable avenue for targeting a broad array of psychological symptoms, and for reaching more individuals who could benefit from insomnia treatments.

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References

- Ancoli-Israel S, & Roth T (1999). Characteristics of insomnia in the United States: Results of the 1991 National sleep Foundation Survey. *Sleep*, 22, S347–S353. [PubMed: 10394606]
- Anestis MD, Holm-Denoma JM, Gordon KH, Schmidt NB, & Joiner TE (2008). The role of anxiety sensitivity in eating pathology. *Cognitive Therapy and Research*, 32(3), 370–385.
- Assayag Y, Bernstein A, Zvolensky MJ, Steeves D, & Stewart SS (2012). Nature and role of change in anxiety sensitivity during NRT-aided cognitive-behavioral smoking cessation treatment. *Cognitive Behaviour Therapy*, 41(1), 51–62. [PubMed: 22375732]
- Babson KA, Trainor CD, Bunaciu BA, & Feldner MT (2008). An examination of anxiety sensitivity as a moderator of the relation between sleep anticipatory anxiety and sleep onset latency. *Journal of Cognitive Psychotherapy*, 22(3), 258–270.
- Baron RM, & Kenny DA (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51(6), 1173–1182. [PubMed: 3806354]
- Bastien CH, Vallieres A, & Morin CM (2001). Validation of the insomnia severity index as a clinical outcome measure for insomnia research. *Sleep Medicine*, 2, 297–307. [PubMed: 11438246]
- Beck AT, & Steer RA (1993). *Beck anxiety inventory manual*. San Antonio, TX: Harcourt Brace and Company.
- Beck AT, Steer RA, & Carbin MG (1988). Psychometric properties of the Beck depression inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, 8(1), 77–100.
- Breslau N, Roth T, Rosenthal L, & Andreski P (1996). Sleep disturbance and psychiatric disorders: A longitudinal epidemiological study of young adults. *Biological Psychiatry*, 39(6), 411–418. [PubMed: 8679786]
- Broman-Fulks JJ, & Storey KM (2008). Evaluation of a brief aerobic exercise intervention for high anxiety sensitivity. *Anxiety, Stress, & Coping*, 21(2), 117–118.
- Buysse DJ, Reynolds CF, Monk TH, Berman SR, & Kupfer DJ (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193–213. [PubMed: 2748771]
- Calkins AW, Hearon BA, Capozzoli MC, & Otto MW (2012). Psychosocial predictors of sleep dysfunction: The role of anxiety sensitivity, dysfunctional beliefs, and neuroticism. *Behavioral Sleep Medicine*, 11(2), 133–143. [PubMed: 23136825]
- Capron DW, Bujarski SJ, Gratz KL, Anestis MD, Fairholme CP, & Tull MT (2016). Suicide risk among male substance users in residential treatment: Evaluation of the depression–distress amplification model. *Psychiatry Research*, 237, 22–26. [PubMed: 26921047]
- Capron DW, Fitch K, Medley A, Blagg C, Mallott M, & Joiner TE (2012). Role of anxiety sensitivity subfactors in suicidal ideation and suicide attempt history. *Depression and Anxiety*, 29(3), 195–201. [PubMed: 21818826]
- Capron DW, Norr AM, Allan NP, & Schmidt NB (2017). Combined “top-down” and “bottom-up” intervention for anxiety sensitivity: Pilot randomized trial testing the additive effect of interpretation bias modification. *Journal of Psychiatric Research*, 85, 75–82. [PubMed: 27837660]
- Capron DW, & Schmidt NB (2016). Development and randomized trial evaluation of a novel computer-delivered anxiety sensitivity intervention. *Behavior Research and Therapy*, 81, 47–55.
- Carney CE, Buysse DJ, Ancoli-Israel S, Edinger JD, Krystal AD, Lichstein KL, et al. (2012). The consensus sleep diary: Standardizing prospective sleep self-monitoring. *Sleep*, 35(2), 287–302. [PubMed: 22294820]
- Chesson AL, Ferber RA, Fry JM, Grigg-Damberger M, Hartse KM, Hurwitz TD, et al. (1997). The indications for polysomnography and related procedures. *Sleep*, 20(6), 423–487. [PubMed: 9302726]
- Chu C, Klein KM, Buchman-Schmitt JM, Hom MA, Hagan CR, & Joiner TE (2015). Routinized assessment of suicide risk in clinical Practice: An empirically informed update. *Journal of Clinical Psychology*, 71(12), 1186–1200. [PubMed: 26287362]

- Chung KF, Kan KK, & Yeung WF (2011). Assessing insomnia in adolescents: Comparison of insomnia severity index, Athens insomnia scale and sleep quality index. *Sleep Medicine*, 12(5), 463–470. [PubMed: 21493134]
- Cox BJ, Enns MW, & Taylor S (2001). The effect of rumination as a mediator of elevated anxiety sensitivity in major depression. *Cognitive Therapy and Research*, 25(5), 525–534.
- Edinger J, Kirby A, Lineberger M, Loiselle M, Wohlgemuth W, & Means M (2004). The duke structured interview for sleep disorders. Duke University Medical Center.
- Edinger JD, Wohlgemuth WK, Radtke RA, Marsh GR, & Quillian RE (2001). Cognitive behavioral therapy for treatment of chronic primary insomnia: A randomized controlled trial. *JAMA*, 285(14), 1856–1864. [PubMed: 11308399]
- Ellis JG, Cushing T, & Germain A (2015). Treating acute insomnia: A randomized controlled trial of a “single-shot” of cognitive behavioral therapy for insomnia. *Sleep*, 38(6), 971–978. [PubMed: 25515106]
- Feldner MT, Zvolensky MJ, Babson K, Leen-Feldner EW, & Schmidt NB (2008). An integrated approach to panic prevention targeting the empirically supported risk factors of smoking and anxiety sensitivity: Theoretical basis and evidence from a pilot project evaluating feasibility and short-term efficacy. *Journal of Anxiety Disorders*, 22(7), 1227–1243. [PubMed: 18281190]
- First MB, Williams JBW, Karg RS, & Spitzer RL (2015). Structured clinical interview for DSM-5—research version (SCID-5 for DSM-5, research version; SCID-5-RV). Arlington, VA: American Psychiatric Association.
- Gardenswartz CA, & Craske MG (2001). Prevention of panic disorder. *Behavior Therapy*, 32(4), 725–737.
- Hartmann JA, Carney CE, Lachowski A, & Edinger JD (2015). Exploring the construct of subjective sleep quality in patients with insomnia. *Journal of Clinical Psychiatry*, 76(6), e768–e773. [PubMed: 26132684]
- Harvey AG (2001). Insomnia: Symptom or diagnosis? *Clinical Psychology Review*, 21(7), 1037–1059. [PubMed: 11584515]
- Harvey AG (2002). A cognitive model of insomnia. *Behavior Research and Therapy*, 40(8), 869–893.
- Hayes AF (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs*, 76(4), 408–420.
- Hoge EA, Marques L, Wechsler RS, Lasky AK, Delong HR, Jacoby RJ, et al. (2011). The role of anxiety sensitivity in sleep disturbance in panic disorder. *Journal of Anxiety Disorders*, 25, 536–538. [PubMed: 21277737]
- Hu LT, & Bentler PM (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, 6(1), 1–55.
- Keough ME, & Schmidt NB (2012). Refinement of a brief anxiety sensitivity reduction intervention. *Journal of Consulting and Clinical Psychology*, 80(5), 766–772. [PubMed: 22468909]
- Kraemer HC, Stice E, Kazdin A, Offord D, & Kupfer D (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry*, 158(6), 848–856. [PubMed: 11384888]
- MacCallum RC, Browne MW, & Sugawara HM (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods*, 1(2), 130–149.
- Mai E, & Buysse DJ (2008). Insomnia: Prevalence, impact, pathogenesis, differential diagnosis, and evaluation. *Sleep Medicine Clinics*, 3(2), 167–174. [PubMed: 19122760]
- McNally RJ, & Eke M (1996). Anxiety sensitivity, suffocation fear, and breath-holding duration as predictors of response to carbon dioxide challenge. *Journal of Abnormal Psychology*, 105(1), 146–153. [PubMed: 8666705]
- Morin CM, Belleville G, Bélanger L, & Ivers H (2011). The insomnia severity index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, 34(5), 601–608. [PubMed: 21532953]
- Morin CM, & Jarrin DC (2013). Epidemiology of insomnia. *Sleep Medicine Clinics*, 8(3), 281–297.
- Muthén LK, & Muthén BO (1998–2012). *Mplus User’s guide*. Seventh edition. Los Angeles, CA: Muthén & Muthén.

- Naragon-Gainey K (2010). Meta-analysis of the relations of anxiety sensitivity to the depressive and anxiety disorders. *Psychological Bulletin*, 136(1), 128–150. [PubMed: 20063929]
- Norr AM, Gibby BA, Fuller KL, Portero AK, & Schmidt NB (2017). Online dissemination of the cognitive anxiety sensitivity treatment (CAST) using craigslist: A pilot study. *Cognitive therapy and research*.
- Ohayon MM (2002). Epidemiology of insomnia: What we know and what we still need to learn. *Sleep Medicine Reviews*, 6(2), 97–111. [PubMed: 12531146]
- Ohayon MM, Caulet M, & Lemoine P (1998). Comorbidity of mental and insomnia disorders in the general population. *Comprehensive Psychiatry*, 39(4), 185–197. [PubMed: 9675502]
- Ohayon MM, & Roth T (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research*, 37(1), 9–15. [PubMed: 12482465]
- Ong JC, Kuo TF, & Manber R (2008). Who is at risk for dropout from group cognitive-behavior therapy for insomnia? *Journal of Psychosomatic Research*, 64(4), 419–425. [PubMed: 18374742]
- Ong JC, Shapiro SL, & Manber R (2008). Combining mindfulness meditation with cognitive-behavior therapy for insomnia: A treatment-development study. *Behavior Therapy*, 39(2), 171–182. [PubMed: 18502250]
- Van Orden KA, Witte TK, Cukrowicz KC, Braithwaite SR, Selby EA, et al. (2010). The interpersonal theory of suicide. *Psychological Review*, 117(2), 575–600. [PubMed: 20438238]
- Preacher KJ, & Hayes AF (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, 40(3), 879–891. [PubMed: 18697684]
- Reiss S, Peterson RA, Gursky DM, & McNally RJ (1986). Anxiety sensitivity, anxiety frequency and the prediction of fearfulness. *Behavior Research and Therapy*, 24(1), 1–8.
- Schmidt NB, Capron DW, Raines AM, & Allan NP (2014). Randomized clinical trial evaluating the efficacy of a brief intervention targeting anxiety sensitivity cognitive concerns. *Journal of Consulting and Clinical Psychology*, 82(6), 1023–1033. [PubMed: 24821096]
- Schmidt NB, Eggleston AM, Woolaway-Bickel K, Fitzpatrick KK, Vasey MW, & Richey JA (2007). Anxiety sensitivity amelioration training (ASAT): A longitudinal primary prevention program targeting cognitive vulnerability. *Journal of Anxiety Disorders*, 21(3), 302–319. [PubMed: 16889931]
- Schmidt NB, Norr AM, Allan NP, Raines AM, & Capron DW (2017). Randomized clinical trial evaluating the efficacy of an automated intervention targeting anxiety sensitivity cognitive concerns for patients with suicidal ideation. *Journal of Consulting and Clinical Psychology*, 85(6), 596–610. [PubMed: 28287798]
- Short NA, Allan NP, Raines AM, & Schmidt NB (2015). The effects of an anxiety sensitivity intervention on insomnia symptoms. *Sleep Medicine*, 16(1), 152–159. [PubMed: 25547037]
- Short NA, Boffa JW, Norr AM, Albanese BJ, Allan NP, & Schmidt NB (2017a). Randomized clinical trial investigating the effects of an anxiety sensitivity intervention on posttraumatic stress symptoms: A replication and extension. *Journal of Traumatic Stress*, 30(3), 296–303. [PubMed: 28585746]
- Short NA, Fuller K, Norr AM, & Schmidt NB (2017b). Acceptability of a brief computerized intervention targeting anxiety sensitivity. *Cognitive Behaviour Therapy*, 46(3), 250–264. [PubMed: 27712458]
- Shrout PE, & Bolger N (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, 7(4), 422–445. [PubMed: 12530702]
- Swift N, Stewart R, Andiappan M, Smith A, Espie CA, & Brown JS (2012). The effectiveness of community day-long CBT-I workshops for participants with insomnia symptoms: A randomised controlled trial. *Journal of Sleep Research*, 21(3), 270–280. [PubMed: 21848573]
- Tan PZ, Forbes EE, Dahl RE, Ryan ND, Siegle GJ, Ladouceur CD, et al. (2012). Emotional reactivity and regulation in anxious and nonanxious youth: A cellphone ecological momentary assessment study. *Journal of Child Psychology and Psychiatry*, 53(2), 197–206. [PubMed: 22176136]
- Taylor S, Koch WJ, & McNally RJ (1992). How does anxiety sensitivity vary across the anxiety disorders? *Journal of Anxiety Disorders*, 6(3), 249–259.

- Taylor DJ, Lichstein KL, Durrence HH, Reidel BW, & Bush AJ (2005). Epidemiology of insomnia, depression, and anxiety. *Sleep*, 28(11), 1457–1464. [PubMed: 16335332]
- Taylor S, Zvolensky MJ, Cox BJ, Deacon B, Heimberg RG, Ledley DR, et al. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the anxiety sensitivity Index-3. *Psychological Assessment*, 19(2), 176–188. [PubMed: 17563199]
- Vincent N, & Walker J (2001). Anxiety sensitivity: Predictor of sleep-related impairment and medication use in chronic insomnia. *Depression and Anxiety*, 14, 238–243. [PubMed: 11754132]
- Watson D, & Clark LA (1994). The PANAS-X: Manual for the positive and negative affect schedule-expanded form. Ames: The University of Iowa.
- Watson D, Clark LA, & Tellegen A (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54(6), 1063–1070. [PubMed: 3397865]
- Westermeyer J, Sutherland RJ, Freerks M, Martin K, Thuras P, Johnson D, et al. (2007). Reliability of sleep log data versus actigraphy in veterans with sleep disturbance and PTSD. *Journal of Anxiety Disorders*, 21(7), 966–975. [PubMed: 17291714]
- Zinbarg RE, Barlow DH, & Brown TA (1997). Hierarchical structure and general factor saturation of the anxiety sensitivity index: Evidence and implications. *Psychological Assessment*, 9(3), 277–284.
- Zvolensky MJ, Marshall EC, Johnson K, Hogan J, Bernstein A, & Bonn-Miller MO (2009). Relations between anxiety sensitivity, distress tolerance, and fear reactivity to bodily sensations to coping and conformity marijuana use motives among young adult marijuana users. *Experimental and Clinical Psychopharmacology*, 17(1), 31–42. [PubMed: 19186932]

Fig. 1.
CONSORT diagram.

Table 1

Descriptive statistics and zero-order correlations for variables of interest.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. BL ASI-3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
2. BL ISI	0.37 [*]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
3. BL ISI C/O	0.29 [*]	0.84 [*]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
4. BL NA	0.58 [*]	0.48 [*]	0.45 [*]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
5. BL BDI-2	0.51 [*]	0.49 [*]	0.42 [*]	0.71 [*]	–	–	–	–	–	–	–	–	–	–	–	–	–	–
6. BL BAI	0.61 [*]	0.44 [*]	0.36 [*]	0.72 [*]	0.59 [*]	–	–	–	–	–	–	–	–	–	–	–	–	–
7. M1 ASI-3	0.61 [*]	0.41 [*]	0.28 [*]	0.48 [*]	0.50 [*]	0.49 [*]	–	–	–	–	–	–	–	–	–	–	–	–
8. M1 ISI	0.35 [*]	0.68 [*]	0.41 [*]	0.36 [*]	0.48 [*]	0.48 [*]	0.46 [*]	–	–	–	–	–	–	–	–	–	–	–
9. M1 NA	0.47 [*]	0.43 [*]	0.33 [*]	0.63 [*]	0.60 [*]	0.52 [*]	0.62 [*]	0.59 [*]	–	–	–	–	–	–	–	–	–	–
10. M3 ASI-3	0.55 [*]	0.37 [*]	0.26 [*]	0.47 [*]	0.51 [*]	0.50 [*]	0.87 [*]	0.49 [*]	0.55 [*]	–	–	–	–	–	–	–	–	–
11. M3 ISI	0.24 [*]	0.61 [*]	0.49 [*]	0.37 [*]	0.47 [*]	0.40 [*]	0.45 [*]	0.81 [*]	0.54 [*]	0.48 [*]	–	–	–	–	–	–	–	–
12. M3 ISI C/O	0.23 [*]	0.59 [*]	0.39 [*]	0.33 [*]	0.40 [*]	0.36 [*]	0.40 [*]	0.68 [*]	0.51 [*]	0.33 [*]	0.82 [*]	–	–	–	–	–	–	–
13. M3 BDI-2	0.29 [*]	0.47 [*]	0.31 [*]	0.47 [*]	0.72 [*]	0.40 [*]	0.52 [*]	0.60 [*]	0.70 [*]	0.54 [*]	0.69 [*]	0.63 [*]	–	–	–	–	–	–
14. M3-BAI	0.43 [*]	0.42 [*]	0.27 [*]	0.54 [*]	0.56 [*]	0.62 [*]	0.64 [*]	0.58 [*]	0.69 [*]	0.68 [*]	0.57 [*]	0.51 [*]	0.70 [*]	–	–	–	–	–
15. M6 ISI	0.34 [*]	0.64 [*]	0.52 [*]	0.36 [*]	0.44 [*]	0.39 [*]	0.43 [*]	0.71 [*]	0.45 [*]	0.35 [*]	0.76 [*]	0.62 [*]	0.57 [*]	0.51 [*]	–	–	–	–
16. M6 ISI C/O	0.29 [*]	0.56 [*]	0.45 [*]	0.34 [*]	0.38 [*]	0.45 [*]	0.41 [*]	0.55 [*]	0.43 [*]	0.33 [*]	0.70 [*]	0.58 [*]	0.50 [*]	0.53 [*]	0.79 [*]	–	–	–
17. M6 BDI-2	0.39 [*]	0.42 [*]	0.30 [*]	0.41 [*]	0.62 [*]	0.39 [*]	0.53 [*]	0.40 [*]	0.59 [*]	0.52 [*]	0.46 [*]	0.44 [*]	0.78 [*]	0.67 [*]	0.52 [*]	0.40 [*]	–	–
18. M6 BAI	0.51 [*]	0.37 [*]	0.24 [*]	0.49 [*]	0.52 [*]	0.60 [*]	0.55 [*]	0.49 [*]	0.52 [*]	0.55 [*]	0.43 [*]	0.32 [*]	0.54 [*]	0.70 [*]	0.57 [*]	0.54 [*]	0.63 [*]	–
Active Mean	32.99	14.10	0.47	27.14	23.12	19.85	16.23	10.62	20.95	15.82	9.69	0.25	14.69	11.43	8.72	0.20	12.48	11.96
Active SD	17.65	7.08	0.50	9.28	13.10	12.61	13.69	7.19	9.27	14.77	7.34	0.43	13.38	9.64	7.39	0.40	10.83	11.89
Control Mean	32.59	15.07	0.53	26.01	21.57	18.20	24.69	12.86	21.57	24.70	11.16	0.31	16.11	13.49	11.57	0.29	11.67	11.15
Control SD	17.88	7.31	0.50	9.41	12.99	13.76	17.93	7.04	9.68	16.30	7.39	0.47	13.99	13.05	7.49	0.46	13.03	10.96

Note. $N = 95$ across variables, BL = Baseline, M1 = Month 1, ASI-3 = Anxiety Sensitivity Index-3, BAI = Beck Anxiety Inventory, BDI-2 = Beck Depression Inventory-2, C/O = Cut-off.

* $p < 0.05$.

Table 2

Indirect effects of condition on insomnia symptoms through anxiety sensitivity.

	Month 3 ISI			Month 3 Clinical Cut-Off			Month 6 ISI			Month 3 Clinical Cut-Off		
	<i>B</i>	95% CI	<i>U</i>	<i>B</i>	95% CI	<i>U</i>	<i>B</i>	95% CI	<i>U</i>	<i>B</i>	95% CI	<i>U</i>
BL ISI or Clinical Cut-Off	.55	.36	.69	.27	.13	.42	.61	.43	.79	.34	.18	.50
Condition	.40	-1.75	2.44	.05	-.09	.17	-.88	-3.19	.16	.02	-.13	.18
Month-1 ASI	.10	.04	.17	.01	.004	.01	.08	.00	.16	.01	.002	.01
Indirect Effect	-.94	-1.91	-.31	-.08	-.15	-.04	-.71	-1.83	-.02	-.06	-.14	-.02

Note. For Intervention Condition 0 = control, 1 = active. CI = Confidence interval, L = Lower, U = Upper. BL = Baseline. ISI = Insomnia Severity Index. ASI = Anxiety Sensitivity Index. Significant effects are in bold.

Indirect effects of condition on insomnia symptoms through anxiety sensitivity, covarying for concurrent changes in depression and anxiety.

Table 3

	Month 3 ISI			Month 3 Clinical Cut-Off			Month 6 ISI			Month 6 Clinical Cut-Off		
	B	L	U	B	L	U	B	L	U	B	L	U
BL ISI or Clinical Cut-Off	.55	.31	.76	.29	.13	.43	.63	.42	.83	.33	.17	.50
Condition	.85	-1.33	.18	.06	-.08	.21	-.60	-2.89	1.65	.03	-.12	.18
Month-1 ASI	.12	.05	.18	.01	.004	.02	.11	.01	.21	.01	.003	.01
Depression Changes	4.51	-14.54	21.97	.59	-1.03	2.06	.18	-2.89	1.65	.28	-1.52	1.97
Anxiety Changes	15.07	-9.46	36.60	.05	-1.45	1.51	43.24	1.46	79.28	.05	-3.34	3.05
Indirect Effect	-1.08	-2.13	.38	-.09	-.17	-.03	-.98	-1.74	-.03	-.08	-.17	-.03

Note. For Intervention Condition 0 = control, 1 = active. CI = Confidence interval, L = Lower, U = Upper. BL = Baseline. ISI = Insomnia Severity Index. ASI = Anxiety Sensitivity Index. Changes in depression and anxiety were calculated as Month-3 minus Month 1 total scores for Month-3 analyses, and using Month 6 scores for Month-6 analyses. Significant effects are in bold.