



# 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.

## 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 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, Wohlgenuth, 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

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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, Reid, 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, Bédanger, & 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

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