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A Multimethod Examination of the Effect of Insomnia Symptoms on Anxious Responding to a Social Stressor

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Abstract

A growing number of studies identify insomnia symptoms as a potential risk factor for the development of anxiety disorders. However, little research has examined potential mechanisms through which insomnia could confer increased risk for anxiety. A separate line of literature suggests sleep is necessary for adaptive emotional and behavioral responding to stressors, a potential mechanism linking insomnia symptoms to anxiety risk. To test whether insomnia symptoms affect emotional and behavioral responding to an anxiety-relevant stressor, the current study recruited a sample of 99 undergraduates with varying levels of insomnia symptoms. Participants completed self-report and psychophysiological measures before, during, and after an impromptu speech task. Results indicated that, after covarying for negative affectivity, increased insomnia symptoms were significantly associated with elevated anticipatory anxiety and skin conductance response prior to the speech, and increased skin conductance response, emotion regulation difficulties, and safety aid use during the speech. Taken together, results provide evidence for the notion that insomnia symptoms are associated with maladaptive emotional and behavioral responding to an anxiety-relevant stressor.

Keywords

insomnia; anxiety; sleep

Insomnia symptoms, defined as difficulty initiating and maintaining sleep, are prevalent in the general population and associated with a variety of negative outcomes. Specifically, about 30% of individuals experience insomnia symptoms on an occasional basis (Mai & Buysse, 2008). For 10% of the population or more, insomnia symptoms are paired with daytime dysfunction, such as fatigue, irritability, decreased positive emotions, and difficulty concentrating (Ancoli-Israel & Roth, 1999; Buysse et al., 2007; Mai & Buysse, 2008). A smaller number of about 5–6% of individuals meet full criteria for insomnia disorder (Ancoli-Israel & Roth, 1999; Mai & Buysse, 2008), with insomnia symptoms and daytime dysfunction occurring on a regular basis, as well as clinically significant distress and impairment. In the long term, insomnia symptoms result in a variety of negative

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consequences, which range from poorer quality of life, physical health problems, pain, social and occupational dysfunction, and increased risk for accidents (Roth, 2007).

One consequence of insomnia symptoms is increased risk for anxiety disorders. Indeed, several studies indicate that insomnia symptoms prospectively predict the development of new anxiety disorders or worsened anxiety symptoms (Batterham, Glozier, & Christensen, 2012; Ford & Kamerow, 1989; Jackson, Sztendur, Diamond, Byles & Bruck, 2014; D.; Jansson-Fröjmark, Lundqvist, Lundqvist, & Linton, 2007; Morphy, Dunn, Lewis, Boardman, & Croft, 2007; Weissman, Greenwald, Niño-Murcia, & Dement, 1997). This literature suggests that insomnia symptoms predict the occurrence of new anxiety disorders for up to 10 years (e.g., Neckelmann, Mykletun, & Dahl, 2007). It is possible that some of the risk for anxiety symptoms conferred by insomnia is explained by shared risk factors. However, more recent studies have covaried for shared risk factors for both insomnia and anxiety (e.g., neuroticism, ruminative response style) and found that these shared risk factors alone do not account for associations between insomnia symptoms and future anxiety symptoms (Batterham et al., 2012; Jackson et al., 2014; Morphy et al., 2007; Neckelmann et al., 2007). This suggests insomnia itself may increase risk for the development or exacerbation of anxiety symptoms. However, the mechanism through which insomnia confers risk for anxiety is mostly unknown.

One hypothesis for how insomnia may increase for psychopathology is that insomnia symptoms affect how individuals emotionally and behaviorally respond to stressors in the environment (Goldstein & Walker, 2014; Harvey, 2008, 2011). In particular, insomnia symptoms may interfere with adaptive responding to anxiety-related stressors. Evidence suggests that sleep loss causes heightened anticipatory anxiety to stressors (Goldstein et al., 2013) and disturbed sleep increases our negative emotional reactivity to these stressors. Specifically, sleep deprivation may lower our threshold for what we perceive to be stressful, and cause us to evaluate even minor problems as stressful (Minkel et al., 2012). Furthermore, sleep loss and poor sleep quality are associated with increases in the negative emotions (e.g., distress, frustration, irritation) associated with stressors (Dinges et al., 1997; Short, Allan, & Schmidt, 2017; Short et al., 2016; Zohar, Tzischinsky, Epstein, & Lavie, 2005).

In addition to increasing the anxiety and distress prior to and during stressful experiences, insomnia symptoms may be paired with an inability to recruit higher-level cognitive functions to successfully down regulate emotional responding. For example, poor sleep quality has been cross-sectionally associated with difficulties using cognitive reappraisal to down regulate negative emotions assessed in experimental paradigms and via self-report (Mauss, Troy, & LeBourgeois, 2013; Pickett, Barbaro, & Mello, 2016). This could be due to an impairment of the top-down control the prefrontal cortex is typically able to exert on amygdala function when sleep is disturbed (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). A combination of increased fear response, along with a lack of access to adaptive emotion regulation strategies may lead to increased propensity to avoid possible threats, increasing anxiety in the long term.

Avoidance is known to be a central factor leading to and exacerbating symptoms of anxiety (Foa & Kozak, 1986), and can take many forms, ranging from overt behavioral avoidance (e.g., completely avoiding a threatening situation) to more subtle forms of avoidance such as safety aids. Safety aids vary, often dependent upon the anxiety symptoms at hand, but are typically perceived by individuals as a way to “protect” themselves from feared outcomes (Schmidt et al., 2012). For example, an individual with social anxiety may avoid eye contact or mentally rehearse what to say to evade experiencing negative evaluation. Safety aids are thought to be a form of avoidance because they are often a method used for engaging in a situation but avoiding the most threatening aspects of it, and because, like avoidance, they prevent the disconfirmation of threat-related beliefs (Clark & Wells, 1995) and thus maintain anxiety symptoms.

In sum, insomnia symptoms prospectively predict anxiety symptoms but how these symptoms confer risk for anxiety is unclear. A parallel line of research suggests that sleep is important for emotional and behavioral responding to stressors, ultimately leading to increased distress and avoidance regarding threatening situations, which provides a potential link accounting for associations between insomnia and risk for anxiety. However, little research has examined the impact of *insomnia symptoms* (vs. sleep deprivation) on anxiety-relevant emotional and behavioral responses. Furthermore, many studies examining insomnia symptoms and anxiety have failed to account for shared risk factors that could explain their co-occurrence (e.g., negative affectivity; Mai & Buysse, 2008; Roth, 2007; Watson, Clark, & Carey, 1988). Considering this, research is needed to determine the effect of insomnia symptoms on emotional and behavioral responding to an anxiety-relevant stressor, accounting for shared vulnerability factors (e.g., negative affectivity). Thus, we examined the role of insomnia symptoms in predicting response to an impromptu speech task. We chose to use an impromptu speech task because it has been shown to elicit distress among individuals without a specific anxiety diagnosis (Hofmann, Heering, Sawyer, & Asnaani, 2009). We hypothesized that, after covarying for negative affect, insomnia symptoms would predict (a) increased anticipatory anxiety prior to the speech, assessed by self-report and skin conductance response (SCR); (b) a perceived lack of effective emotion regulation strategies during the speech; (c) increased distress during the speech assessed by self-report and SCR; and (d) increased self-reported use of safety aids during the speech.

Method

PARTICIPANTS

Participants were 105 undergraduates recruited from the undergraduate research participation pool of a large university. Participants were invited to participate in the current study if they were at least 18 years of age, participated in the university’s research screening survey, and were either good sleepers (i.e., scoring 8 or less on the Insomnia Severity Index [ISI]) or poor sleepers (i.e., scoring 10 or more on the ISI; Morin, Belleville, Blanger, & Ivers, 2011). This was done to ensure adequate range in insomnia symptoms. After the initial screening, no participants were excluded from the study based on their ISI scores at their appointment. Of the total N , six participants did not complete the entire study appointment (i.e., had to leave early), and were thus excluded from analyses ($n = 99$).

Participants' ages ranged from 18 to 35 ($M = 19.36$, $SD = 2.08$), and most participants were women (78.1%). The majority of the students identified as White (66.9%), followed by Black/African American (9.1%), other (e.g., more than one race; 7.0%), and Asian (5.0%). Regarding ethnicity, 22.3% reported being of Hispanic or Latino descent.

Regarding insomnia symptoms, 54.6% scored in the good sleeper range, while 36.3% met or exceeded the clinical cutoff of 10 on the ISI. A remaining 9.0% scored in between "good" and "poor" sleeper status ($ISI = 9$). Of those reporting sleep problems, the majority reported these symptoms were present for at least 1 month (94.0%). Specifically, the largest proportion of participants (28.4%) reported their sleep problems were present for 1–3 months, followed by over a year (25.4%), as long as they can remember (15.2%), 3–6 months (10.4%), and 6–12 months (7.5%). Participants reported sleeping an average of 6.6 hours ($SD = 1.44$) the prior night, with mostly "good" sleep quality (30.6%). In terms of potential psychiatric comorbidities, 4.0% had a probable diagnosis of depression as assessed by the clinical cutoff of 30 on the Beck Depression Inventory, Second Edition (Beck, Steer, & Carbin, 1988). Similarly, approximately 5.0% of the sample met criteria for a probable anxiety disorder diagnosis as measured by the clinic cutoff of 30 on the Beck Anxiety Inventory (Beck & Steer, 1993).

PROCEDURE

Research assistants e-mailed eligible participants to invite them to participate in a study examining sleep and stress, and participants were told they would receive course credits for their participation. Upon arrival to the lab, participants provided informed consent. Then, participants completed a picture-viewing task unrelated to the current study while outfitted with the SCR recording device. During this time, baseline SCR was assessed while viewing neutral images (e.g., chairs, doors, buildings). Participants then completed a battery of self-report measures.

After completion of these measures, participants were told it was time for them to give a 5-minute speech on a controversial topic. Consistent with other impromptu speech studies (e.g., Hofmann et al., 2009), participants were told the research assistant would be evaluating their performance, and that a video recording of their speech would be evaluated by a panel of experts. Participants were also told they would be allowed some time to prepare for the speech, and then completed prespeech self-report measures. Next, the research assistant gave each participant a list of speech topics to choose from (i.e., abortion, death penalty, healthcare reform), and told participants that they could pick to talk about one, two, or all three of these topics. Participants were given 2 minutes to prepare for the speech, and SCR was recorded during this time. After 2 minutes had elapsed, research assistants prompted the participant to begin his or her speech, started video and SCR recording, and began timing the participant. Participants were informed they could discontinue the speech at any time by raising their hand, but they were encouraged to speak for the full 5 minutes. If participants raised their hand, the research assistant encouraged them once to continue, and if they declined, the speech was terminated. After the 5 minutes had passed (or early termination), SCR recording was stopped and participants completed

postspeech self-report measures. Participants were then dismissed. All procedures were approved by the university's Institutional Review Board.

MEASURES

Insomnia Severity Index (ISI)—The ISI is a five-item self-report questionnaire measuring insomnia symptoms and severity (e.g., difficulties falling or staying asleep, or waking up too early), satisfaction with sleep patterns, and/or interference with daily functioning (Morin et al., 2011). Participants rate each item on a 5-point Likert scale ranging from 0 to 4. Higher scores indicate more severe insomnia symptoms. The ISI has demonstrated good psychometric properties in previous research, including reliability, validity, sensitivity to change, and the ability to discriminate those with insomnia disorder based on clinical cutoffs (Morin et al., 2011). In the current study, internal consistency was good ($\alpha = .88$).

Positive and Negative Affect Schedule—Negative Affect (PANAS-NA)—The PANAS-NA is composed of 10 words describing negative emotions (Watson & Clark, 1994; Watson, Clark, & Tellegen, 1988). Participants indicated the extent to which they experience these emotions, on a general basis, on a 5-point Likert scale. The PANAS has strong psychometric properties (Watson & Clark, 1994; Watson, Clark, & Tellegen, 1988). Reliability in the current sample was good ($\alpha = .86$).

Brief State Anxiety Measure (BSAM)—The BSAM is a six-item self-report measure assessing current levels of anxiety (Berg, Shapiro, Chambless, & Ahrens, 1998) originally drawn from the well-validated State-Trait Anxiety Inventory (Spielberger, 2010). Participants rate each item (e.g., I feel relaxed, I feel worried) on a 4-point scale. Items are summed to create a total score. The BSAM evidenced good internal consistency at pre- and postspeech (both α s = .86).

State-Difficulties in Emotion Regulation Scale (S-DERS)—The S-DERS is adapted from the DERS, a 36-item measure assessing various difficulties in emotion regulation (Gratz & Roemer, 2004; Lavender, Tull, DiLillo, Messman-Moore, & Gratz, 2017). The S-DERS modified the items of the DERS to assess current emotion dysregulation (e.g., I feel out of control. I am confused about how I feel). The S-DERS includes 21 items that are rated on a 5-point scale assessing four factors of emotion regulation difficulties: nonacceptance of current emotions, difficulties modulating emotional and behavioral responses, awareness of emotions, and limited clarity about emotions. Only the difficulties modulating emotional and behavioral responses (modulate) subscale was used in the current investigation. The S-DERS demonstrated good psychometric properties in prior research (Lavender et al., 2017). In the current study, the S-DERS modulate subscale had good internal consistency ($\alpha = .86$).

Safety Aid Scale—State (SAS—State): The SAS is a 79-item measure of safety aids individuals use to manage anxiety (Korte, 2015). Participants view a list of safety aids (e.g., speaking very little in social situations, avoiding driving during “rush hour”) and rate the extent to which they use them on a 5-point scale. It has demonstrated excellent internal consistency in prior work (Buckner et al., 2017). In the current study, the SAS was modified

to instruct participants to rate these strategies based on how often they used them *during their speech*. In addition, safety aids impossible to use in the speech setting were removed (e.g., avoiding driving during rush hour), resulting in a list of 26 possible state safety aids (e.g., rehearsing sentences in your head, avoiding eye contact, using mental distractions, fiddling with an object, speaking quickly, seeking reassurance). The modified SAS demonstrated good internal consistency in the current study ($\alpha = .89$).

Subjective Units of Distress (SUDs): SUDs are a commonly used measure of distress that quickly and simply assess level of distress and fear in the current moment (Barlow, 2002). In the current study, participants were asked to rate their SUDs ranging from 0 to 100 prior to preparing for their speech, at the beginning of the speech, and in 1-minute intervals throughout their speech. SUDs were explained by the research assistant at the beginning of the speech preparation period, and participants verbally rated their SUDs. These were recorded by the research assistant. We used the maximum SUD rating throughout the speech to assess peak distress.

Skin Conductance Response (SCR): SCR was measured using the eSense Skin Conductance System (Mindfield Biosystems, Inc., Berlin, Germany) on an iPad. eSense offers a mobile application, which was downloaded on the iPad. Two finger electrodes were attached to the middle and index finger of each participant with Velcro straps. Electrodes were then plugged into the iPad via an audio jack. Data were acquired at a sampling rate of 10 Hz and data were exported via CSV files over e-mail following each SCR assessment. The use of eSense technology has been compared to traditional SCR assessments in prior studies and data were highly correlated (Hinrichs et al., 2017) and have been used in one other study to our knowledge (Post et al., 2017). SCR data were collected during the neutral picture viewing task and was averaged to create an average baseline SCR score. Then, SCR was collected before (i.e., during speech preparation) and during the speech. Consistent with Hinrichs and colleagues (2017), we created a residual change score from average baseline SCR to peak pre- and during-speech SCR to assess SCR reactivity.

SCR data were available for 71% of participants. Of those missing SCR data, nine did not complete the speech task at all and thus had no speech-related data. An additional 16 participants completed the speech task, but research assistants reported problems with SCR data collection (e.g., sensors did not stick to participants' fingers, participant was "fiddling" with sensor during speech, data did not transmit properly, program malfunctioned and cut off recording early), and thus these participants were excluded from SCR analyses. Whether or not participants had adequate SCR data was not significantly correlated with any of the study's constructs of interest ($ps > 1.78$).

Results

DESCRIPTIVE STATISTICS AND CORRELATIONS

First, we examined descriptive statistics and zero-order correlations for all variables (Table 1). Examination of descriptive statistics, including skewness and kurtosis values, and visual inspection of the data revealed that data were entered correctly and that variables followed a normal distribution. In terms of correlations, most variables were significantly positively

correlated, as expected. However, peak SUDs were not significantly correlated with insomnia symptoms. Furthermore, SCR recordings were not associated with negative affectivity. Considering this, the PANAS-NA was not included as a covariate in SCR analyses.

PRIMARY ANALYSES

Anticipatory Anxiety—To test our first hypothesis that insomnia symptoms would predict increased anticipatory anxiety prior to the speech, we conducted two separate multiple regressions. In the first regression, insomnia symptoms and negative affect were entered as predictors of prespeech BSAM total scores. Both elevated negative affect as a covariate ($\beta = .29$, $t = 2.71$, $p = .008$, $sr^2 = .06$), and insomnia symptoms ($\beta = .27$, $t = 2.49$, $p = .015$, $sr^2 = .05$) significantly predicted more self-reported prespeech anxiety. Second, insomnia symptoms were entered as a predictor of prespeech SCR reactivity. Results revealed more severe insomnia symptoms significantly predicted increased prespeech SCR reactivity ($\beta = .26$, $t = 2.19$, $p = .032$, $sr^2 = .07$).

Anxiety and Emotion Regulation Difficulties During Speech—Second, two additional multiple regressions were conducted with the same predictors and during speech SUDs and SCR as dependent variables. Negative affect ($\beta = .40$, $t = 3.59$, $p = .001$, $sr^2 = .12$), but not insomnia symptoms ($\beta = -.08$, $t = -.75$, $p = .458$, $sr^2 = .01$), significantly predicted peak SUDs during the speech. Higher insomnia symptoms did significantly predict increased SCR reactivity during the speech ($\beta = .29$, $t = 2.50$, $p = .015$, $sr^2 = .08$).

Third, we examined insomnia symptoms and negative affect as predictors of difficulties modulating emotional and behavioral responses (S-DERS-Modulate). Higher levels of both negative affect ($\beta = .43$, $t = 4.47$, $p < .001$, $sr^2 = .15$) and insomnia symptoms ($\beta = .26$, $t = 2.64$, $p = .010$, $sr^2 = .05$) significantly predicted increases in these emotion regulation difficulties.

Fourth, we investigated insomnia symptoms and negative affect as predictors of self-reported safety aid usage during the speech. Both negative affect ($\beta = .45$, $t = 4.61$, $p < .001$, $sr^2 = .16$) and insomnia symptoms ($\beta = .23$, $t = 2.32$, $p = .022$, $sr^2 = .04$) significantly predicted increased safety aid use.

Discussion

The current study expands upon prior literature suggesting that insomnia symptoms may be a risk factor for anxiety by testing whether insomnia symptoms cross-sectionally predict emotional and behavioral responding to an anxiety-relevant stressor (i.e., an impromptu speech). First, results revealed that elevated insomnia symptoms were associated with increased anticipatory anxiety to the speech task, after accounting for negative affectivity. This is consistent with prior research suggesting that sleep deprivation leads to increased anticipatory brain responding to cues for negative pictures. However, our findings are the first, to our knowledge, to suggest that naturally occurring insomnia symptoms are associated with increased self-reported anticipatory anxiety. Furthermore, these results were complemented by psychophysiological measures: insomnia symptoms were associated with

increased SCR reactivity during the anticipation period, providing further confidence for the association between insomnia symptoms and anticipatory anxiety. Enhanced anticipation to threat could be one pathway through which insomnia symptoms increase risk for anxiety disorders, as elevated anticipatory reactions to potential threat are present in individuals with anxiety disorders and are associated with worry, rumination, and elevated and increased motivation to engage in avoidance (Etkin & Wager, 2007; Nitschke et al., 2009).

Second, findings indicated that insomnia symptoms were associated with increased self-reported difficulties modulating emotional and behavioral responses. This is in line with prior experimental research noting that sleep deprivation may impair higher-level cognitive control to regulate emotional responding (Yoo et al., 2007). Additionally, this finding is consistent with research indicating that poor sleep quality is associated with difficulties effectively utilizing cognitive reappraisal to reduce one's sadness in response to a sad film clip (Mauss et al., 2013) and self-reported emotion regulation difficulties (Pickett et al., 2016). The current study advanced these findings by covarying for trait negative affect, providing further confidence for the specificity of the association between insomnia symptoms and emotion dysregulation. In terms of anxiety risk, prior research has noted that the perception that one has limited access to effective emotion regulation strategies is associated with elevated anxiety symptoms (Tull, Barrett, McMillan, & Roemer, 2007).

Third, we found mixed results as to whether insomnia symptoms were associated with increased distress *during* the speech. Consistent with our hypothesis, insomnia symptoms were significantly associated with increased SCR reactivity during the speech. This reflects prior research findings that disturbed sleep is associated with increased reactivity to stressors, assessed via self-reported negative affect, cortisol responding, and neuroimaging (Minkel et al., 2014; Short et al., 2016; Yoo et al., 2007; Zohar et al., 2005). However, inconsistent with our hypothesis, insomnia symptoms were not significantly associated with self-reported distress. Future research is needed to clarify this finding, as much more research has examined sleep deprivation as a predictor of increased reactivity to stressors, while less has examined insomnia symptoms. It is possible that total sleep deprivation may increase reactivity to stressors, but insomnia symptoms do not.

Fourth, we found insomnia symptoms were significantly associated with increased use of safety aids during the speech, after accounting for negative affectivity. Although it has been suggested that the problems with affective responding associated with poor sleep could lead to increased reliance on avoidance (e.g., Short, Raines, Oglesby, Zvolensky, & Schmidt, 2014), this study is the first, to our knowledge, to actually test this association. This is important because safety aid use, or avoidance in general, could represent a mechanism accounting for how insomnia symptoms lead to increased risk for anxiety disorder development. Future research should continue to examine whether insomnia symptoms prospectively predict increased use of safety aids and other forms of avoidance.

The current study also has clinical implications. Results, when considered along with prior research, suggest that risk for anxiety is elevated among individuals with insomnia symptoms. Clinicians should assess anxiety among individuals presenting for insomnia treatment, and consider treatment options. Specifically, insomnia can be treated with various

psychosocial interventions, including cognitive-behavioral therapy for insomnia (CBT-I; Edinger, Wohlgenuth, Radtke, Marsh, & Quillian, 2001). These treatments also tend to have small to moderate effects in reducing comorbid symptoms of anxiety (Belleville, Cousineau, Levrier, & St-Pierre-Delorme, 2011). Further research could inform clinicians as to whether CBT-I may also reduce the risk for the development of anxiety disorders.

Findings from the current study should be interpreted in light of the study's limitations, which also provide direction for future research. First, the current study utilizes cross-sectional data, and thus causal inferences cannot be made. Future studies should prospectively examine whether insomnia symptoms predict these emotional and behavioral responses to stressors over time. Second, we did not include clinical interviews to formally assess insomnia disorder or other psychiatric diagnoses. Although the ISI is a well-regarded measure of insomnia symptoms that provides a suggested clinical cutoff for insomnia disorder (Morin et al., 2011), a clinical interview such as the Duke Structured Interview for Sleep Disorders would complement self-report by providing diagnostic data (Edinger et al., 2009). Third and relatedly, we did not utilize a polysomnographic assessment of sleep, which could help to confirm insomnia disorder and rule out other sleep disorders (Chesson et al., 1997). Inclusion of polysomnography in future studies could provide further information as to the specific disruptions in sleep (i.e., changes in sleep architecture, objective sleep efficiency) that are associated with impaired emotional and behavioral responding to stressors. Fourth, although we included a psychophysiological measure of distress (i.e., SCR), we relied on self-report to assess emotion regulation strategies. Considering there is no consensus as to the proper methods to assess emotion regulation objectively, use of the S-DERS seems to be appropriate, but future research could use experimental paradigms to ascertain whether individuals with insomnia actually have difficulty using emotion regulation strategies thought to be effective (e.g., Mauss et al., 2013).

Despite these limitations, the current study adds valuable information to the emerging literature identifying insomnia symptoms as a potential risk factor for anxiety by evaluating potential mechanisms of how insomnia may confer risk for anxiety. The current results suggest insomnia symptoms may negatively affect how individuals respond emotionally and behaviorally to anxiety-relevant stressors, potentially putting them at increased risk for the development of anxiety disorders over time.

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Table 1

Descriptive Statistics and Zero-Order Correlations for All Variables of Interest

	1	2	3	4	5	6	7	8
1. Insomnia Symptoms	-	-	-	-	-	-	-	-
2. Negative Affect	.48***	-	-	-	-	-	-	-
3. Prespeech Anxiety	.39***	.41***	-	-	-	-	-	-
4. Prespeech SCR	.26*	.15	.14	-	-	-	-	-
5. Speech Peak SUDs	.07	.36***	.48***	.01	-	-	-	-
6. Speech SCR	.29*	.14	.09	.94***	.01	-	-	-
7. Postspeech S-DERS	.35***	.59***	.36***	.14	.28**	.18	-	-
8. Postspeech Safety Aids	.42***	.55***	.33**	.02	.37***	.06	.50***	-
<i>M (SD)</i>	8.63 (5.95)	18.60 (6.23)	12.82 (3.89)	2.72 (2.64)	50.77 (24.36)	3.38 (3.108)	11.00 (4.12)	24.56 (14.73)

Note. SCR = skin conductance response; SCR values are change from neutral average SCR to peak pre- or during-speech SCR; SUDs = subjective units of distress; S-DERS = State Difficulties in Emotion Regulation Scale.

* = $p < .05$,

** = $p < .01$,

*** = $p < .001$.