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Manifestations of Overarousal Account for the Association between Cognitive Anxiety Sensitivity and Suicidal Ideation

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Abstract

**Background:** Recent evidence suggests an association between cognitive anxiety sensitivity and suicidal ideation. Cognitive anxiety sensitivity has also been implicated as a precursor to various forms of overarousal. These manifestations of overarousal (i.e., agitation, insomnia, nightmares, and anger) may account for the association between cognitive anxiety sensitivity and suicidal ideation. **Methods:** In Study 1, undergraduate students selectively sampled for recent suicidal ideation completed all measures online. In Study 2, clinical outpatients completed all measures prior to their initial intake appointments at a psychology clinic. **Results:** Study 1 demonstrated that agitation and insomnia individually and jointly accounted for the association between cognitive anxiety sensitivity and suicidal ideation, controlling for general anxiety and demographic variables. Study 2 replicated and extended these findings, such that, controlling for demographics, general anxiety, and physical and social anxiety sensitivity, agitation and anger each independently and together accounted for the association between cognitive anxiety sensitivity and suicidal ideation, whereas insomnia and nightmares did not. **Limitations:** This study utilized a cross-sectional design and self-report measures in both samples as well as a sample of undergraduate students in Study 1. **Conclusions:** Together, these findings suggest that agitation and anger may explain the previously established relationship between cognitive anxiety sensitivity and suicidal ideation. Targeting cognitive anxiety sensitivity in treatment may in turn reduce these forms of overarousal and thereby suicide risk.

Keywords: suicide, cognitive anxiety sensitivity, agitation, insomnia, nightmares, anger
Manifestations of Overarousal Account for the Associations between Cognitive Anxiety Sensitivity and Suicidal Ideation

Extant research has examined the role of cognitive vulnerabilities in the etiology and maintenance of suicidal ideation. Notably, anxiety sensitivity cognitive concerns (ASCC), a component of anxiety sensitivity originally derived from the panic disorder literature (Dixon et al., 2013; Taylor et al., 2007), has recently garnered empirical attention in relation to suicidality. ASCC, defined as fear of losing control of one’s cognitions or emotions, is the facet of anxiety sensitivity most closely linked to suicidal thinking (Capron et al., 2012a) and has been found to be related to suicidal ideation in a variety of samples (Capron et al., 2012a; Capron et al., 2012b; Capron et al., 2012c; Capron et al., 2013; Lamis and Jahn, 2013; Schmidt et al., 2001).

ASCC may also influence suicidal ideation indirectly through the development and maintenance of overarousal, an important predictor of suicide (Ribeiro et al., 2013). Anxiety sensitivity more generally has been associated with exaggerated perceived arousal in a number of studies (e.g., Asmundson et al., 1994; Carter et al., 2009; Conrod, 2006; Thibodeau et al., 2012). Paired with an increased fearlessness about death, this overarousal may provide individuals with the energy needed to make a suicide attempt (Ribeiro et al., 2014; Ribeiro et al., 2015). Links between specific manifestations of overarousal and anxiety sensitivity have also been demonstrated. For instance, anxiety sensitivity, particularly ASCC (Calkins et al., 2013), has been associated with sleep disturbances (Hoge et al., 2011), which have been implicated as a warning sign for suicide (Bernert and Joiner, 2007). Specifically, associations between insomnia and nightmare frequency and suicidal ideation and attempts have been demonstrated in a wide range of samples (e.g., Bernert and Nadorff, 2015; Bernert et al., 2014; Goldstein et al., 2008; Goodwin and Marusic, 2008; Pigeon et al., 2012).
Agitation, another manifestation of overarousal, has also been implicated as a warning sign for suicide (Bryan et al., 2014; Rudd et al., 2006; Sani et al., 2011). Severe agitation is often experienced in suicide decedents in the week prior to their deaths (Busch et al., 1993; Busch et al., 2003). Anxiety sensitivity is closely linked to symptoms of panic disorder and anxiety (Dixon et al., 2013), both of which entail elements of motor restlessness and agitation (American Psychiatric Association, 2013). Thus, it is plausible that agitation may underlie the association between ASCC and suicidality.

Although a number of studies have determined the independent contributions of ASCC and overarousal to suicide risk, no research to date has examined overarousal as a mechanism through which cognitive anxiety sensitivity confers risk for suicide. As such, the present studies test the hypothesis that overarousal accounts for the relationship between ASCC and suicidal ideation. Study 1 examines the indirect effects of ASCC and suicidal ideation through agitation and insomnia in a selectively sampled pool of young adults. Study 2 replicates and extends these findings to other manifestations of overarousal (i.e., nightmares, anger) that have also been associated with increased risk for suicide (e.g., Hawkins et al., 2014; Horesh et al., 1997) in a sample of psychiatric outpatients.

**Study 1**

The aim of the first study was to examine the nature of the relationship between ASCC, agitation and insomnia, and suicidal ideation. Specifically, we predicted that ASCC would be positively associated with agitation, insomnia, and suicidal ideation, and that agitation and insomnia would relate positively to suicidal ideation. Further, we hypothesized that, when
controlling for general anxiety and demographic predictors of suicide, agitation and insomnia would account for the association between ASCC and suicidal ideation.

**Study 1 Methods**

**Participants and Procedure**

Participants were 186 undergraduate students (79.6% female) from a large southern state university, aged 18 to 54 ($M = 19.81; SD = 4.16$). Participants primarily self-identified as White/Caucasian (83.3%), Native American (10.8%), Black/African American (6.5%), Hispanic or Latino/Latina (5.4%), and Asian/Pacific Islander (5.4%). Percentages do not sum to 100% as participants were allowed to self-identify with multiple ethnicities.

Oversampling procedures were utilized to ensure that suicidal ideation was present in the sample. All research pool participants (approximately 300-360 students) over the three-semester collection period who indicated recent suicidal ideation (within the past year) on the Suicidal Behavior Questionnaire – Revised (SBQ-R; Osman et al., 2001) were recruited to participate. A smaller portion of students not experiencing recent suicidal ideation (10% of the remaining prescreen pool of 1,500-2,000 students) were also recruited. Participants provided informed consent, completed all questionnaires online, and were provided with mental health resources (i.e., local/national counseling services) upon completion. All procedures were approved by the university’s Institutional Review Board.

** Measures**

**Anxiety Sensitivity Index 3 - Cognitive Concerns Subscale (ASI-3; Taylor et al., 2007).** The 6-item cognitive concerns subscale of the ASI-3 was administered to assess anxiety regarding the fear of losing cognitive and emotional control (e.g., "When my thoughts seem to speed up, I worry that I might be going crazy"). Responses are on a 5-point scale ranging from 1
(Very Little) to 5 (Very Much). The ASI-3 and its subscales have demonstrated high reliability and validity in previous studies of undergraduate students and psychiatric patients (Kemper et al., 2012; Taylor et al., 2007). Internal consistency in this sample was high ($\alpha = .91$).

**Brief Agitation Measure (BAM; Ribeiro et al., 2011).** The BAM is a 3-item self-report measure of agitated feelings experienced in the last week (e.g., "I feel so stirred up inside I want to scream"). Participants responded on a 7-point Likert-type scale ranging from 1 (Not at all true) to 7 (Very true). Previous studies have provided evidence for the reliability and validity of the BAM in undergraduate students and psychiatric outpatients (Ribeiro et al., 2011). Internal consistency was good in Study 1 ($\alpha = .88$).

**Insomnia Severity Index (ISI; Bastien et al., 2001).** The ISI is a 7-item self-report measure of insomnia symptoms (i.e., type of sleep disturbance, severity, and impact) over the previous two weeks (e.g., "How worried/distressed are you about your current sleep problem(s)?"). Responses are on a 5-point Likert-type scale ranging from 0 to 4, with response options varying across items. Previous research has provided evidence for the scale’s reliability, validity, and sensitivity in detecting changes in sleep difficulties in a variety of samples (Bastien et al., 2001; Morin et al., 2011). The internal consistency of the ISI was good in Study 1 ($\alpha = .88$).

**Beck Scale for Suicide Ideation (BSS; Beck and Steer, 1991).** The BSS is a 21-item self-report measure of suicidal ideation over the past week (e.g., "I have no wish to die/a weak wish to die/a moderate to strong wish to die"). Participants are asked to respond on a 3-point Likert-type scale ranging from 0 to 2, with response options varying between items. The BSS demonstrated strong psychometric properties in previous research (e.g., Beck et al., 2006), as well as excellent internal consistency in Study 1 ($\alpha = .91$).
Beck Anxiety Inventory (BAI; Beck et al., 1988a). The BAI is a 21-item self-report measure that assesses general symptoms of anxiety (e.g., shaky, faint, nervous). Participants indicate the degree to which they experienced each symptom over the past two weeks on a 4-point scale, ranging from Not at all to Severely. The BAI has demonstrated strong psychometric properties in previous research (e.g., Beck et al., 1988a; Osman et al., 1997), and internal consistency was high in the present sample (α = .95).

Data Analytic Strategy

Descriptive statistics and bivariate correlations were computed to determine the prevalence and interrelatedness of ASCC, agitation, insomnia, and suicidal ideation. Indirect effect analyses were run to test the hypothesis that ASCC would be related to suicidal ideation through agitation and insomnia symptoms. The PROCESS macro was used to test indirect effects with 5,000 bootstrapped samples as outlined by Hayes (2013). ASCC served as the predictor variable in all analyses. Three indirect effect analyses were executed: one in which agitation served as the mediator, one in which insomnia symptoms served as the mediator, and one in which agitation and insomnia symptoms served as parallel mediators. Age, gender, and general anxiety were included as covariates. Two measures of effect sizes are reported: kappa squared ($\kappa^2$) and ratio of indirect to total effect ($P_M$); however, it is important to note that due to restrictions of the PROCESS macro, kappa squared effect sizes are unable to be reported with the inclusion of covariates or in models with multiple mediators (Preacher and Kelley, 2011). Thus, reported kappa squared coefficients were obtained from models excluding covariates.

Study 1 Results and Discussion

Descriptive Statistics and Bivariate Correlations
Means, standard deviations, and bivariate correlations of all study variables are presented in Table 1. General anxiety, agitation, ASCC, and suicidal ideation were leptokurtic and positively skewed despite sampling procedures. These variables were log-transformed to correct for non-normality, resulting in diminished skewness and kurtosis (< 1 for all variables other than suicidal ideation, which remained slightly positively skewed [1.59]). For ease of interpretation, we refer to non-transformed descriptive statistics in tables and text. When presenting results of multivariate analyses (e.g., correlations, indirect effects analyses), we refer to analyses conducted after the transformation of the original variables. Listwise deletion was used to handle missing data (1.1%). Sixty six participants (35.5%) endorsed suicidal ideation in the week previous to participation.

**Indirect Effects of ASCC, Agitation, and Insomnia on Suicidal Ideation**

ASCC had an indirect relationship with suicidal ideation through agitation, point estimate = .2765, \(SE = .0913\), 95% bias-corrected CI [.1247, .4862], \(\kappa^2 = .2840\), \(P_M = .5669\) (see Table 2). The indirect effect model accounted for 39.51% of the total variance of suicidal ideation (\(F[5, 178] = 23.25, p < .001\)). The direct effect was not significant (point estimate = .2113, \(SE = .1558\), 95% bias-corrected CI [-.0963, .5188]).

ASCC also had an indirect relationship with suicidal ideation through insomnia, point estimate = .1600, \(SE = .0664\), 95% bias-corrected CI [.0500, .3167], \(\kappa^2 = .1708\), \(P_M = .3280\) (Table 2). The indirect effect model accounted for 30.83% of the total variance of suicidal ideation (\(F[5, 178] = 15.87, p < .001\)). The direct effect of ASCC on suicidal ideation was not significant (point estimate = .3278, \(SE = .1664\), 95% bias-corrected CI [-.0005, .6561]).

When agitation and symptoms of insomnia were tested as parallel mediators (see Table 2), agitation (point estimate = .2506, \(SE = .0868\), 95% bias-corrected CI [.1084, .4537], \(P_M =
.5138) and insomnia (point estimate = .0941, SE = .0561, 95% bias-corrected CI [.0057, .2326], $P_M = 1930$) each significantly accounted for the association between ASCC and suicidal ideation. Pairwise comparisons did not reveal significant differences between indirect effects (95% bias-corrected CI [-.0467, .3823]), indicating that agitation and insomnia each explained unique variance in the link between ASCC and suicidal ideation. Further, the direct effect was non-significant (point estimate = .1430, $SE = .1577$, 95% bias-corrected CI [-.1681, .4541]). The parallel indirect effects model accounted for 41.01% of the total variance in suicidal ideation ($F[6, 177] = 20.51, p < .001$).

**Study 2**

Study 1 demonstrated that agitation and insomnia independently and jointly accounted for the association between cognitive anxiety sensitivity and suicidal ideation in a sample of undergraduate students. Undergraduate samples, however, tend to be relatively healthy, even when oversampling for suicidality, and it is unclear if these findings generalize to clinical samples. This particular sample was also small and relatively homogeneous with regard to age and ethnicity. Moreover, other, less-studied manifestations of overarousal (i.e., nightmares, anger; Bernert and Joiner, 2007; Hawkins et al., 2014) may be relevant in explaining the association between ASCC and suicidal ideation. Though not yet studied in relation to ASCC, nightmares and anger may similarly and incrementally account for the relation between ASCC and suicidal ideation. Study 1 also presents analyses using only demographic variables and general anxiety as covariates. As anxiety sensitivity includes cognitive, physical, and social fears, the first study cannot attribute results to ASCC specifically, as information regarding these other forms of anxiety sensitivity were not collected.
Thus, Study 2 examined these relationships in a larger clinical sample in an attempt to replicate and extend the findings of Study 1. Specifically, we hypothesized that all forms of overarousal (agitation, insomnia, nightmares, and anger) would independently explain the association between ASCC and suicidal ideation. Moreover, we tested an indirect effects model with all four forms of overarousal simultaneously serving as mediators; we expected that agitation and insomnia, again, would remain significant, but we made no a priori hypotheses regarding nightmares and anger. In all analyses, demographic variables, general anxiety, and physical and social anxiety sensitivity were included as covariates to establish that these associations are specific to ASCC, rather than demographics, other facets of anxiety sensitivity, or anxiety more generally.

**Study 2: Methods**

**Participants and Procedure**

Participants were 392 outpatients (66.8% female) between the ages of 18 and 65 ($M = 26.97$, $SD = 10.49$) at a university-based psychological clinic in the southeastern United States. Participants were primarily European American (75.3%), with 11.2% Hispanic, 9.2% African American, 1.5% Asian/Pacific Islander, and 0.5% American Indian; 2.3% did not indicate an ethnicity. The sample was relatively well-educated: 18.1% graduated from high school, 2.0% completed vocational or technical training, 53.6% finished some college, 17.3% graduated with a bachelor’s degree, and 7.1% completed postgraduate studies.

The clinic serves the community in addition to university students and staff, and due to the clinic’s inexpensive sliding scale fees, patients tend to be of lower socioeconomic status. Exclusionary criteria are minimal; individuals are only referred elsewhere if they are suffering
from psychotic or bipolar spectrum disorders that are not stabilized on medication or if they are an immediate danger to themselves or others. Overall, patients present to the clinic with a variety of conditions that range in severity. In this sample, the most common primary diagnoses were major depressive disorder (15.9%), generalized anxiety disorder (6.0%), and social anxiety disorder (4.6%). Diagnoses were made using the Structured Clinical Interview for DSM Disorders – Patient Edition (SCID-I-P; First et al., 2002) by a trained therapist. Approximately half (50.3%) of the sample reported current suicidal ideation, as evidenced by a BSS score greater than zero.

All outpatients served by the clinic complete a large battery of screening questionnaires prior to their intake appointments. The data presented in this study were drawn from this battery of screening questionnaires. All patients consented to participate in the Institutional Review Board-approved research conducted at the clinic.

**Measures**

**Anxiety Sensitivity Index (ASI; Reiss et al., 1986).** The ASI is a 16-item self-report measure comprised of three subscales (physical, social, and cognitive concerns) that assess the tendency to fear the consequences of bodily sensations associated with anxiety. The 4-item cognitive concerns subscale (e.g., “When I cannot keep my mind on a task, I worry that I might be going crazy”) was included as the predictor variable in analyses, whereas the 8-item physical concerns subscale (e.g., “It scares me when my heart beats rapidly”) and 3-item social concerns subscale (e.g., “It is important to me not to appear nervous”) were included as covariates. Responses on each item range from 0 (Very Little) to 4 (Very Much). The scale has demonstrated good reliability and predictive validity in past research (e.g., Zvolensky et al., 2001). Internal
consistency for the cognitive and physical subscales in this sample were good (αs = .83, .87, respectively), but was poor for the social subscale (α = .43).

**BAI (Beck et al., 1988a).** See Study 1 for a description of the BAI. Internal consistency was high in the present sample (α = .92).

**BAM (Ribeiro et al., 2011).** See Study 1 for a description of the BAM. The scale’s reliability in this sample was good (α = .85).

**ISI (Bastien et al., 2001).** See Study 1 for a description of the ISI. In this sample, the ISI demonstrated high reliability (α = .87).

**Disturbing Dreams and Nightmares Severity Index (DDNSI; Krakow et al., 2002).** The DDNSI was used as a measure of nightmare frequency and severity over the past year. Participants indicated how often they experience nightmares (weekly, monthly, or annually) and then reported the number of nights with nightmares in that time period (0-7 nights per week) and number of total nightmares in that time period (up to 14 per week). Responses were scaled to nightmares per week. Nightmare severity was assessed through three items of the DDNSI. Participants answered, “How would you rate the intensity of your disturbing dreams and/or nightmares?” on a 7-point scale ranging from Not intense (0) to Extremely severe intensity (6). Responses to, “How would you rate the severity of your disturbing dreams and/or nightmare problem?” were on a 7-point scale ranging from No problem (0) to Extremely severe problem (6). Responses to, “On average, do your nightmares wake you up?” were on a 5-point scale ranging from Never/Rarely (0) to Always (4). A total score greater than 10 on the DDNSI may indicate the presence of a nightmare disorder (Krakow et al., 2002); in the current sample, 145 (39.4%) participants had scores reflective of a nightmare disorder. Internal consistency of the DDNSI severity score was good (α = .81).
Clinical Anger Scale (CAS; Snell et al., 1995). The CAS is a 21-item self-report measure designed to assess the presence and severity of clinical anger symptoms. Respondents rate different dimensions of anger symptoms on a scale from 0 to 3 by selecting the statement that best represents their feelings (e.g., 0: “Things are not more irritating to me now than usual,” 1: “I feel slightly more irritated now than usual,” 2: “I feel irritated a good deal of the time,” 3: “I’m irritated all the time now”). Scores range from 0 to 63, with higher scores reflecting higher levels of clinical anger. The CAS has demonstrated sound psychometric properties in previous studies (e.g., Reyes and Hicklin, 2005) and high internal consistency in the present sample (α = .93).

BSS (Beck and Steer, 1991). See Study 1 for a description of the BSS. Internal consistency was good in the current sample (α = .88).

Data Analytic Strategy

A similar analytic strategy was utilized in Study 2 to assess and interpret descriptive statistics, bivariate correlations, and indirect effects, with 5,000 bootstrap resamples utilized in indirect effects analyses using the PROCESS macro (Hayes, 2013). As in Study 1, ASCC was the predictor variable, suicidal ideation the outcome variable, and manifestations of overarousal the mediator variables. Mediators were first assessed in independent models and then jointly in a single model as parallel mediators. Additionally, age, gender, general anxiety symptoms, and physical and social anxiety sensitivity were included as covariates. Finally, to test the specificity of these associations to ASCC, indirect effects models with physical and social anxiety sensitivity as predictor variables were examined. As in Study 1, two measures of effect sizes are reported: kappa squared ($\kappa^2$) and ratio of indirect to total effect ($P_M$). Again, reported kappa
squared coefficients were obtained from models excluding covariates (Preacher and Kelley, 2011).

**Study 2: Results and Discussion**

All variables were examined for univariate and multivariate normality. Nightmares were slightly positively skewed, and anger and suicidal ideation were both leptokurtic and positively skewed. These variables underwent a log transformation that corrected for non-normality, resulting in diminished skew and kurtosis (< 1 in all cases). We refer to non-transformed descriptive statistics in tables and text. When presenting results of multivariate analyses (e.g., correlations, indirect effects analyses), we refer to analyses conducted after the transformation of the original variables. Missing data were minimal (0.5%) and handled via listwise deletion. See Table 3 for means, standard deviations, ranges, and bivariate correlations of all variables utilized in analyses.

**Indirect Effects of ASCC and Overarousal on Suicidal Ideation**

ASCC had a significant indirect relationship with suicidal ideation through agitation, point estimate = .0164, SE = .0050, 95% bias-corrected CI [.0080, .0279], $\kappa^2 = .14$, $P_M = .54$ (see Table 4). The indirect effect model accounted for 18.18% of the total variance in suicidal ideation ($F[7, 384] = 12.19, p < .001$). The direct effect between ASCC and suicidal ideation was not significant (point estimate = .0137, SE = .0148, 95% bias-corrected CI [-.0155, .0428]).

The indirect relationship of ASCC with suicidal ideation through insomnia was not significant, point estimate = .0021, SE = .0024, 95% bias-corrected CI [-.0013, .0083], $\kappa^2 = .05$, $P_M = .07$ (Table 4). The indirect effect model accounted for 14.92% of the total variance in suicidal ideation ($F[7, 384] = 9.62, p < .001$). The direct effect between ASCC and suicidal
ideation was also non-significant (point estimate = .0279, \( SE = .0147 \), 95% bias-corrected CI [-.0010, .0569]).

The indirect relationship of ASCC with suicidal ideation through nightmares was also non-significant, point estimate = .0027, \( SE = .0030 \), 95% bias-corrected CI [-.0025, .0098], \( \kappa^2 = .06 \), \( P_M = .09 \) (Table 4). The indirect effect model accounted for 16.99% of the total variance in suicidal ideation \((F[7, 384] = 11.22, p < .001)\). The direct effect between ASCC and suicidal ideation was non-significant (point estimate = .0274, \( SE = .0145 \), 95% bias-corrected CI [-.0012, .0560]).

ASCC had a significant relationship with suicidal ideation through anger, point estimate = .0105, \( SE = .0042 \), 95% bias-corrected CI [.0042, .0208], \( \kappa^2 = .08 \), \( P_M = .34 \) (Table 4). The indirect effect model accounted for 17.30% of the total variance in suicidal ideation \((F[7, 382] = 11.41, p < .001)\). The direct effect of ASCC on suicidal ideation was not significant (point estimate = .0203, \( SE = .0148 \), 95% bias-corrected CI [-.0087, .0494]).

When entering agitation, insomnia, nightmares, and anger as parallel mediators of the association between ASCC and suicidal ideation, the indirect effect model accounted for 21.45% of the total variance in suicidal ideation \((F[10, 379] = 10.35, p < .001)\). Agitation (point estimate = .0195, \( SE = .0061 \), 95% bias-corrected CI [.0091, .0333], \( P_M = .35 \)) and anger (point estimate = .0061, \( SE = .0034 \), 95% bias-corrected CI [.0011, .0146], \( P_M = .20 \)) significantly accounted for the association between ASCC and suicidal ideation, whereas insomnia (point estimate = .0009, \( SE = .0016 \), 95% bias-corrected CI [-.0008, .0062], \( P_M = .03 \)) and nightmares (point estimate = .0019, \( SE = .0020 \), 95% bias-corrected CI [-.0008, .0078], \( P_M = .06 \)) did not. Further, pairwise comparisons indicated that agitation explained significantly more variance in the relation between ASCC and suicidal ideation than did insomnia and nightmares. The direct effect was not
significant in this model (point estimate = .0113, \(SE = .0147\), 95% bias-corrected CI [-.0176, .0403]).

To determine the specificity of these indirect effects to ASCC, rather than other facets of anxiety sensitivity, models were tested in which physical and social anxiety sensitivity were the predictor variables, with ASCC included as a covariate. With physical anxiety sensitivity as the predictor variable and suicidal ideation as the outcome variable, neither agitation (point estimate = -.0019, \(SE = .0016\), 95% bias-corrected CI [-.0061, .0005], \(P_M = .09\)), insomnia (point estimate = .0003, \(SE = .0007\), 95% bias-corrected CI [.0005, .0030], \(P_M = -.01\)), nightmares (point estimate = .0002, \(SE = .0012\), 95% bias-corrected CI [.0000, .0031], \(P_M = -.01\)), nor anger (point estimate = -.0008, \(SE = .0011\), 95% bias-corrected CI [-.0038, .0008], \(P_M = .04\)) accounted for the association between physical anxiety sensitivity and suicidal ideation. A similar pattern emerged with social anxiety sensitivity as the predictor variable: neither agitation (point estimate = .0005, \(SE = .0040\), 95% bias-corrected CI [.0077, .0089], \(P_M = .03\)), insomnia (point estimate = -.0002, \(SE = .0021\), 95% bias-corrected CI [-.0067, .0028], \(P_M = -.02\)), nightmares (point estimate = .0046, \(SE = .0037\), 95% bias-corrected CI [.0004, .0151], \(P_M = .33\)), nor anger (point estimate = -.0006, \(SE = .0034\), 95% bias-corrected CI [-.0086, .0057], \(P_M = -.04\)) explained the relationship between social anxiety sensitivity and suicidal ideation. This largely demonstrates specificity of our model to ASCC.

**General Discussion**

Although the number of deaths by suicide is increasing in United States and worldwide (World Health Organization, 2014), the prediction of suicidal ideation and behavior is imprecise. Manifestations of overarousal, such as agitation, sleep disturbances, and anger, have been implicated as acute risk factors for suicide (Ribeiro et al., 2013). The link between ASCC and
suicidal ideation (Capron et al., 2012a), on the other hand, is believed to be more distal, yet mechanisms have not yet been identified. To fill this gap in the literature, the present study examined forms of overarousal as mechanisms of the association between ASCC and suicidal ideation.

Study 1 tested both agitation and insomnia as explanatory links in the relationship between ASCC and suicidal ideation. As hypothesized, both agitation and insomnia independently and in parallel accounted for the association between ASCC and suicidal ideation. The findings from Study 2 replicated and extended those from Study 1, using a different measure of ASCC. Agitation and anger each independently and jointly explained the association between ASCC and suicidal ideation in a psychiatric outpatient sample, whereas the indirect effects through insomnia and nightmares were not significant. This suggests that the experience of agitation and anger may explain the previously found association between ASCC and suicidal ideation (Capron et al., 2012a). On the other hand, sleep disturbances are not a mechanism through which ASCC confers risk for suicidal ideation. Notably, these findings were specific to ASCC; no forms of overarousal accounted for a link between physical or social anxiety sensitivity and suicidal ideation (these associations were non-significant directly as well). This suggests that anxiety about the fear of losing control of one’s thoughts and emotions may relate to suicidal ideation through overarousal that occurs in one’s waking state (i.e., agitation, anger), not through overarousal related to sleep and sleep disturbances (i.e., insomnia, nightmares). Future studies should examine these relations further.

Of note, although insomnia accounted for the association between ASCC and suicidal ideation in Study 1, this indirect effect was non-significant in Study 2. This may be due to differences in the study samples; for instance, participants in Study 2 were psychiatric
outpatients, many of whom were diagnosed with an affective disorder commonly associated with insomnia and related sleep disturbances (e.g., major depressive disorder, generalized anxiety disorder). These patients, in turn, exhibited greater insomnia severity scores ($M = 11.77$) than did the undergraduate student sample of Study 1 ($M = 8.18$). Thus, insomnia symptoms in Study 2 may be better accounted for by general psychopathology, rather than as an explanatory link between ASCC and suicidal ideation, as evidenced in Study 1. This possibility should be examined in future research with samples of varying levels of insomnia severity.

Though interpretations of these findings are limited due to this study’s cross-sectional design, the finding that ASCC may be a precursor to manifestations of overarousal that are associated with acute suicide risk is encouraging, clinically. Anxiety sensitivity is malleable, with even single session interventions of psychoeducation and interoceptive exposure exercises demonstrating an amelioration of symptoms that endures even two years later (Schmidt et al., 2007). These interventions may in turn indirectly reduce suicide risk through a reduction of overarousal symptoms.

These results also interface with Acute Suicidal Affective Disturbance (ASAD), a recently proposed disorder of acute suicide risk (Joiner et al., under review; Rogers et al., in prep; Tucker et al., in press). Specifically, the forms of overarousal examined in this study (agitation, insomnia, nightmares, and anger) match those that are included in the proposed ASAD diagnostic criterion of overarousal (with anger being a proxy for irritability). Given that ASCC was significantly positively associated with each form of overarousal in this study, it is possible that individuals who are high in ASCC may be more likely to develop ASAD symptoms as well. Future studies should test these associations as mediated by ASAD symptoms. Furthermore, when tested in parallel, agitation was a stronger indirect predictor of suicidal
ideation than insomnia and nightmares. This suggests that certain constellations of overarousal symptoms may be more strongly related to suicide-related outcomes than others, much like certain symptoms of depression are associated with greater suicide risk than others (Beck et al., 1988b). Future research should examine this possibility further in an effort to better understand which facet of overarousal confers greatest risk for suicide.

Though this study was the first to our knowledge to examine potential mechanisms of the association between ASCC and suicidal ideation, the implications of these findings should be considered in the context of several limitations. First, the generalizability of these results is limited, given that samples with lower severity of psychopathology were utilized (selectively sampled undergraduate students and psychiatric outpatients). These results should be replicated in other samples, such as inpatient and military samples, as well as samples of individuals with more severe psychopathology (i.e., psychotic and bipolar spectrum disorders) and in those with high levels of anxiety sensitivity (e.g., those with panic disorder) and other specific diagnoses that relate to overarousal and suicide risk (e.g., mood disorders, generalized anxiety disorder), in order to ensure the generalizability of our findings. Moreover, this study is limited by its cross-sectional design – thus precluding inferences of causality – as well as exclusive use of self-report measures. Future studies should use a longitudinal approach to examine causal relationships between these constructs. Further, the use of physiological or behavioral measures for overarousal constructs could mitigate the challenges associated with self-report measurement.

Caution is also warranted in the interpretation of these findings in relation to ASAD. Namely, we assessed the presence and severity of suicidal ideation over a two week period, rather than assessing for a rapid onset of suicidality suggested by ASAD criteria. Additionally, the Study 2 sample consisted of treatment-seeking outpatients whose symptoms may be better accounted for
by other psychiatric disorders (including mood pathology). Nevertheless, despite these limitations, our findings reflect the overarousal component of ASAD and warrant replication in other samples.

This study also used suicidal ideation, rather than suicide attempts, as the outcome variable. Generally, very few people who experience suicidal thoughts go on to make a suicide attempt (Kessler et al., 1999; Klonsky and May, 2014). Despite the need for future research in this vein to examine suicide attempts and death by suicide as primary outcomes, this study was an important first step in establishing relationships between ASCC, overarousal, and suicidality more generally. Future research should examine these relationships with suicide attempts rather than ideation. Klonsky and May's (2014) ideation-to-action framework places the emphasis on identifying those with suicidal ideation who are prone to act on their ideation and is an ideal theoretical framework for future research in this domain.

Overall, this study represents a novel contribution to the literature on cognitive anxiety sensitivity, overarousal, and suicide. The results of this study replicate previous findings that ASCC outperforms the other two facets of anxiety sensitivity in predicting suicidal ideation (Capron et al., 2012a; Capron et al., 2012b) and that ASCC, as expected, was more strongly related to overarousal than were physical and social anxiety sensitivity. Additionally, by studying explanatory links between the newly established relationship between cognitive anxiety sensitivity and suicidal ideation, we have offered an explanation for how cognitive anxiety sensitivity leads to overarousal, which in turn exacerbates suicidal ideation. Ultimately, these findings are a first step in better understanding these relationships and in developing interventions that effectively treat and reduce suicide risk.
Acknowledgements

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References


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doi:http://dx.doi.org/10.1001/archpsyc.56.7.617


doi:http://dx.doi.org.proxy.lib.fsu.edu/10.1111/sltb.12068


Rogers, M.L., Stanley, I.H., Hom, M.A., Chiurliza, B., Podlogar, M.C., Joiner, T.E., under review. What is left over when you covary depression out of suicidal ideation? Not much except error.


Footnotes

1 We opted not to control for depressive symptoms in the present studies, due to emerging evidence that this practice results in a prediction of largely error, rather than conceptual, variance, in the prediction of suicidal ideation (Rogers et al., under review). Namely, suicidal ideation, in many ways, is a manifestation of depressive symptomatology, and by covarying out depression, suicidal ideation loses much of its conceptual (and empirical) significance.

2 Reversed direction models were tested with ASCC as the mediator and agitation/insomnia as the independent variables. The indirect effect of agitation on suicidal ideation through ASCC was not significant (point estimate = .0481, $SE = .0456$, 95% bias-corrected CI [-.0259, .1580], $\chi^2 = .07$, $PM = .06$), nor was the indirect effect of insomnia on suicidal ideation through ASCC (point estimate = .0063, $SE = .0040$, 95% bias-corrected CI [.0000, .0161], $\chi^2 = .11$, $PM = .14$), indicating the specificity of our effects.

3 Reversed direction models were tested with ASCC as the mediator and overarousal variables as the independent variables. ASCC did not account for the associations between agitation (point estimate = .0033, $SE = .0041$, 95% bias-corrected CI [-.0037, .0123], $\chi^2 = .02$, $PM = .04$), insomnia (point estimate = .0009, $SE = .0011$, 95% bias-corrected CI [-.0004, .0045], $\chi^2 = .04$, $PM = .04$), nightmares (point estimate = .0063, $SE = .0088$, 95% bias-corrected CI [-.0035, .0358], $\chi^2 = .04$, $PM = .02$), or anger (point estimate = .0142, $SE = .0131$, 95% bias-corrected CI [-.0048, .0489], $\chi^2 = .04$, $PM = .06$) and suicidal ideation. This provides some evidence for the directionality of our effects.
Table 1

Measures, Standard Deviations, Ranges, and Bivariate Correlations of Study 1 Variables

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<tr>
<th>Variable</th>
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<td>.41***</td>
<td>.61***</td>
<td>.47***</td>
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</tr>
</tbody>
</table>

Mean

| 19.81 | 1.81 | 10.93 | 9.59 | 6.38 | 8.18 | 1.68 |

Standard Deviation

| 4.15  | .40  | 11.73 | 4.78 | 4.81 | 5.89 | 4.00 |

Minimum

| 18    | 1    | 0    | 6    | 3    | 0    | 0    |

Maximum

| 54    | 2    | 53   | 29   | 21   | 25   | 26   |

Note: † p < .10, * p < .05, ** p < .01, ***p < .001. Gender: 1 = Male, 2 = Female; Anxiety = Beck Anxiety Inventory (BAI) total score; ASCC = Anxiety Sensitivity Index - 3 (ASI-3) Cognitive Concerns subscale; Insomnia = Insomnia Severity Index (ISI) total score; Agitation = Brief Agitation Measure (BAM) total score; Suicidal Ideation = Beck Scale for Suicide Ideation (BSS) total score.
Table 2.

*Indirect Effects of Anxiety Sensitivity Cognitive Concerns (ASCC), Insomnia, and Agitation on Suicidal Ideation in Study 1.*

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Mediating Variable</th>
<th>Outcome Variable</th>
<th>Effect Estimate</th>
<th>SE</th>
<th>95% BC CI</th>
<th>$\kappa^2$</th>
<th>$\kappa^2$ CI</th>
<th>$P_M$</th>
<th>$P_M$ CI</th>
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</table>

*Note:* Anxiety Sensitivity Index - 3 (ASI-3) Cognitive Concerns subscale; Insomnia = Insomnia Severity Index (ISI) total score; Agitation = Brief Agitation Measure (BAM) total score; Suicidal Ideation = Beck Scale for Suicide Ideation (BSS) total score; BC CI = bias corrected confidence interval; 5,000 bootstrap samples; L = Lower estimate of confidence interval; U = Upper level of confidence interval; * = Parallel multiple mediation model.
Table 3.

Means, Standard Deviations, Ranges, and Bivariate correlations of all Study 2 Variables.

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<td>12</td>
<td>28</td>
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Note: † p < .10, * p < .05, ** p < .01, *** p < .001. Gender: 1 = Female, 2 = Male; Anxiety = Beck Anxiety Inventory (BAI) total score; ASCC = Anxiety Sensitivity Index Cognitive Concerns subscale; ASPC = Anxiety Sensitivity Index Physical Concerns subscale; ASSC = Anxiety Sensitivity Index Social Concerns subscale; Agitation = Brief Agitation Measure (BAM) total score; Insomnia = Insomnia Severity Index (ISI) total score; Nightmares = Disturbing Dreams and Nightmares Severity Index (DDNSI) total score; Anger = Clinical Anger Scale (CAS) total score; Ideation = Beck Scale for Suicide Ideation (BSS) total score.
### Table 4.

*Indirect Effects of Anxiety Sensitivity Cognitive Concerns (ASCC), Agitation, Insomnia, Nightmares, and Anger on Suicidal Ideation in Study 2.*

<table>
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<tr>
<th>Predictor Variable</th>
<th>Mediating Variable</th>
<th>Outcome Variable</th>
<th>Effect Estimate</th>
<th>SE</th>
<th>95% BC CI</th>
<th>( R^2 )</th>
<th>( \kappa^2 )</th>
<th>( \kappa^2 ) CI</th>
<th>( P_M )</th>
<th>( P_M ) CI</th>
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*Note: BC = bias corrected; CI = confidence interval; 5,000 bootstrap samples; L = Lower estimate of confidence interval; U = Upper level of confidence interval; * = Parallel multiple mediation model. ASCC = Anxiety Sensitivity Index Cognitive Concerns subscale; ASPC = Anxiety Sensitivity Index Physical Concerns subscale; ASSC = Anxiety Sensitivity Index Social Concerns subscale; Agitation = Brief Agitation Measure (BAM) total score; Insomnia = Insomnia Severity Index (ISI) total score; Nightmares = Disturbing Dreams and Nightmares Severity Index (DDNSI) total score; Anger = Clinical Anger Scale (CAS) total score; Ideation = Beck Scale for Suicide Ideation (BSS) total score.