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## Prospective Evaluation of the Effect of an Anxiety Sensitivity Intervention on Suicidality among Smokers

Daniel W. Capron<sup>1</sup>, Aaron M. Norr<sup>1</sup>, Michael J. Zvolensky<sup>2</sup>, and Norman B. Schmidt<sup>1</sup>

<sup>1</sup>Department of Psychology, Florida State University

<sup>2</sup>Department of Psychology, University of Houston, Houston, TX

### Abstract

Recent empirical work has revealed a significant relationship between anxiety sensitivity (AS), particularly the AS cognitive concerns subfactor, and suicidality among cigarette smokers. The current study prospectively tested whether an intervention targeting AS (i.e. an AS augmented smoking cessation program) would predict lower suicidality in a population known to be at increased risk for death by suicide (i.e. cigarette smokers). Participants ( $N = 169$ ) were randomly assigned to a standard cognitive-behavioral smoking intervention or a cognitive-behavioral smoking intervention with an AS reduction component. Findings indicate the participants who received the AS augmented intervention had lower suicidality compared to those in the standard intervention, even accounting for baseline suicidality, substance use disorder diagnosis, current depression, current anxiety and current smoking status. As the first examination of the effect of an AS reduction program on suicidality, this study provides initial support for the hypothesis that reducing AS may lead to lower suicidality. Future work should include testing an AS intervention in a sample with elevated suicidality, as well as specifically targeting AS cognitive concerns, which has a stronger and more consistent relationship with suicidality than global AS.

### Keywords

nicotine; cigarettes; suicide

### Introduction

Death by suicide in the US is currently at its highest rate in 15 years (AFSP, 2011). In 2008, over 36,000 suicide deaths were reported and yearly medical costs for suicide are estimated at nearly \$100,000,000 (AFSP, 2011). Despite these alarming statistics, the vast majority (over 90%) of individuals who die by suicide suffer from mental health disorders and treatment of these disorders can lead to reductions in deaths by suicide (Cavanagh, Carson, Sharpe, & Lawrie, 2003).

Reducing anxiety sensitivity (AS), a well-known risk factor for a variety of mental health disorders, also may be important to reducing suicidality. AS is known as a “fear of fear” and refers to a fear of anxiety related sensations (Reiss, Peterson, Gursky, & McNally, 1986). There is a substantial literature linking AS to the development of psychopathology, including anxiety disorders (Schmidt, Zvolensky, & Maner, 2006), PTSD (Marshall, Miles, & Stewart, 2010), and substance use disorders (Schmidt, Buckner, & Keough, 2007). AS is constituted of three lower order subfactors related to physical, cognitive, and social

concerns. Although extant findings indicate an inconsistent association between overall AS and suicidality (Capron, Fitch, et al., 2012; Schmidt, Woolaway-Bickel, & Bates, 2001), an emerging literature suggests that the AS cognitive concerns subfactor is uniquely and consistently associated with elevated suicidality (Capron, Cogle, Ribeiro, Joiner, & Schmidt, 2012; Capron, Gonzalez, Parent, Zvolensky, & Schmidt, 2012).

AS cognitive concerns refer to fears of mental incapacitation or of losing control of mental process in the context of stress. From a theoretical perspective, the association between AS cognitive concerns and suicide is consistent with positive feedback models of suicide that suggest those vulnerable to catastrophic cognitions (“I might lose control of my mind”) such as individuals with high AS cognitive concerns are at increased risk of attempting suicide (Katz, Yaseen, Mojtabai, Cohen, & Galynker, 2011). The positive feedback model posits that the emergence of catastrophic cognitions represent a specific susceptibility to the activation of suicidal ideation. In the model, limbic-autonomic arousal and catastrophic cognitions perpetuate and heighten each other. Within this positive feedback loop, catastrophic cognitions are amplified, eventually activating suicidal ideation. Eventually, the combination of limbic-autonomic arousal and suicidal ideation drives an individual to act to stop the escalating emotional distress in the form of a suicide attempt (Katz et al., 2011). Escalating emotional distress leading to suicidal ideation is consistent with the escape theory of suicide (Baumeister, 1990). Alternatively, recent work suggests that suicidal ideation may actualize as the result of depression that is amplified via catastrophic cognitions/AS cognitive concerns (Capron, Norr, Macatee, & Schmidt, in press).

Daily cigarette smokers are one population associated with elevated suicide rates. Previously, the literature on smoking and suicide was mixed in regard to whether this relationship existed when controlling for comorbid mental health disorders (Breslau, Schultz, Johnson, Peterson, & Davis, 2005; Kessler, Borges, Sampson, Miller, & Nock, 2009). However, two recent epidemiological studies have found a direct positive relationship between smoking/nicotine dependence and suicide after controlling for a number of possible confounding mental health disorders (Bolton & Robinson, 2010; Yaworski, Robinson, Sareen, & Bolton, 2011). The first of these studies (Bolton & Robinson, 2010) found that nicotine dependence was among only four diagnoses that independently accounted for a high proportion of suicide attempts (the other three disorders were major depressive disorder, borderline personality disorder, and post-traumatic stress disorder). The second study (Yaworski et al., 2011) found that nicotine dependence was associated with suicide attempts, independent of all Axis I and Axis II conditions as well as physical disease. Additionally, Yaworski and colleagues (2011) recently found that previously nicotine dependent individuals who have abstained for at least 1 year were significantly less likely to attempt suicide than people still dependent on nicotine, which suggests a direct role of nicotine dependence on suicidality.

Extant research indicates that smokers with elevated AS may use cigarette smoking to regulate negative affect. A number of studies have found that AS is positively related to smoking to reduce negative affect, but often not other smoking motives [e.g., pleasure, handling, taste (Battista et al., 2008; Comeau, Stewart, & Loba, 2001; Leyro, Zvolensky, Vujanovic, & Bernstein, 2008; Novak, Burgess, Clark, Zvolensky, & Brown, 2003; Stewart, Karp, Pihl, & Peterson, 1997; Zvolensky et al., 2006)]. Other studies have found that AS is related to smoking negative affect reduction expectancies [i.e., beliefs smoking will reduce negative affect (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Gregor, Zvolensky, McLeish, Bernstein, & Morissette, 2008)]. Given the use of cigarettes to regulate negative affect and positive feedback models of suicidality, cigarette smokers with elevated AS appear to be a population who are at elevated risk of suicide. In fact, preliminary empirical work indicates a role of AS cognitive concerns in elevated suicidality among cigarette

smokers. Capron and colleagues (2012) found that AS cognitive concerns predicted elevated suicidality, covarying for negative affect, in a sample of community smokers participating in a smoking cessation program. In addition, Capron and colleagues (2012) found the same association, covarying for negative affect and substance use variables, among a sample of pack a day smokers in outpatient substance abuse treatment.

Despite these interesting findings, there is a notable gap in the extant literature. This empirical work has linked elevated AS cognitive concerns to elevated suicidality among smokers. However, there has never been an investigation of whether an AS augmented smoking cessation program would predict lower suicidality in this population. The aim of the current study was to address this prominent gap in the smoking–suicide literature by: (1) examining the effect of an AS augmented smoking cessation treatment on suicidality, controlling for baseline suicidality, current anxiety and current depression; and (2) testing the effect of the AS augmented smoking cessation program on depression. We also investigated (3) whether AS is a mediator of the relationship between treatment group and current suicidality and (4) examining whether baseline AS is a moderator of the relationship between treatment group and current suicidality.

Based on the extant literature (Capron, Fitch, et al., 2012; Schmidt et al., 2001) we predicted that those participating in the AS augmented smoking cessation program would have significantly lower suicidality after the 4-week treatment compared with a control group and that AS would be the mechanism of change (mediation). Based on the positive-feedback model of suicidality (Katz et al., 2011), we also predicted that this association would be specific to suicidality (versus reducing global depression). Finally, because we believed that those with higher AS would benefit more from the AS intervention, we predicted an interaction between baseline AS and treatment group such that participants with higher baseline AS in the active condition would experience the greatest reductions in suicidality.

## Methods

### Participants

The sample consisted of 169 adult smokers ages 18-68 years ( $M = 42.22$ ,  $SD = 12.81$ ; 58% female) who completed a 4 week smoking cessation program. Participants were recruited from the general community through various media outlets including newspaper ads, flyers, and radio announcements. To be eligible for inclusion, participants had to be 18 years of age or older, a daily smoker for at least 1 year, currently smoke a minimum of 8 cigarettes per day, and report motivation to quit smoking within the next month. Participants were excluded if they were currently psychotic, using any other smoking cessation pharmacotherapy or other tobacco products, or if they had a history of a significant medical condition. The ethnic and racial composition of the current sample was as follows: 85% White/Caucasian, 7% Black Non-Hispanic, 1% Black Hispanic, 5% Hispanic, 1% Asian, and 2% Other.

### Procedure

Interested participants, who also met the initial requirements during a telephone screen, were scheduled to come in for a structured clinical interview to assess for the presence or absence of any Axis I condition (SCID-I/P; First et al., 1995). Individuals who were deemed eligible after the screening/interview process were then scheduled to come in for a baseline appointment to complete various demographic, smoking, anxiety, and substance use assessments. After the baseline assessment, participants were randomly assigned to either a standard cognitive-behavioral smoking cessation program (control) or a cognitive behavioral smoking cessation program with an added anxiety sensitivity component (active). Treatment

consisted of four 90-minute sessions with a trained therapist (For more details on the AS smoking intervention see Funk, Zvolensky, & Schmidt, 2011). The current report utilizes data collected during the baseline assessment which takes place prior to randomization and the onset of treatment. Prospective data (e.g. current suicidality) was collected after the 4<sup>th</sup> (final) treatment session via self-report questionnaires administered on a computer. The program was structured so that participants would quit smoking on the day of this final session. Informed consent was obtained from all participants and the study was approved by the university's institutional review board.

## Measures

**Anxiety Sensitivity Inventory 3 (ASI-3)**—The ASI-3 is an 18-item self-report questionnaire derived from a widely used multi-dimensional measure of anxiety sensitivity (Taylor et al., 2007). The ASI-3 was designed to provide a more stable assessment of the three most commonly replicated anxiety sensitivity subfactors (cognitive, social, and physical concerns). Respondents were asked to read a series of statements (e.g. “It is important to me not to appear nervous”, “It scares me when my heart beats rapidly”, “When I cannot keep my mind on a task I worry I might be going crazy”) and rate the degree to which they agreed with each statement using a 5-point likert scale ranging from 0 (*Very little*) to 4 (*Very much*). Research has demonstrated that the ASI-3 is both a reliable and valid measure of anxiety sensitivity (Taylor et al., 2007). Internal consistency in the present sample was good for ASI-3 total (cronbach  $\alpha = .85$ ) and ranged from adequate to good for the social (cronbach  $\alpha = .71$ ), physical (cronbach  $\alpha = .82$ ), and cognitive (cronbach  $\alpha = .88$ ) subscales.

**Inventory of Depression and Anxiety Symptoms (IDAS)**—The IDAS is a 64-item self-report questionnaire in which respondents were asked to read a list of feelings, sensations, problems, and experiences, and rate the degree to which each statement describes their feelings and experiences during the past two weeks (Watson et al., 2007). Ratings are made using a 5-point likert scale ranging from 1 (*Not at all*) to 5 (*extremely*). The IDAS yields 10 specific symptom scales, including: Suicidality, Lassitude, Insomnia, Appetite loss, Appetite gain, Ill temper, Well-being, Panic, Social anxiety, and Traumatic intrusions. In the current study suicidality was measured using the IDAS-suicidality scale, which is comprised of 6 items (e.g. “I had thoughts of suicide,” “I hurt myself purposely”). Depression was measured using the general depression subscale, which contains 20 items (e.g. “I felt depressed”). Anxiety was measured by combining the three anxiety relevant subscales of the IDAS (social anxiety, panic, trauma) into a total anxiety score. Research has demonstrated that the IDAS subscales have good internal consistency, short-term stability, and convergent and discriminant validity (Watson et al., 2007). Internal consistency in the current sample was excellent for the IDAS general depression subscale (cronbach  $\alpha = .92$ ) and borderline for the IDAS suicidality subscale (cronbach  $\alpha = .68$ ).

**CO Assessment**—Current smoking status was biochemically verified through breath samples. Expired air carbon monoxide levels were assessed with a Bedfont Scientific carbon monoxide monitor (Jarvis, Tunstallpedoe, Feyerabend, Vesey, & Saloojee, 1987). Detected values above the stated cutoff scores (3 parts per million) were considered indicative of smoking. The value of 3 parts per million has been found to have the best combination of sensitivity and specificity in previous work on assessing smoking abstinence with a high degree of certainty (Javors, Hatch, & Lamb, 2005).

## Data Analytic Procedure

To test the study hypothesis that the active treatment group would have lower current suicidality, an analysis of covariance (ANCOVA) was conducted, with dependent variable

grouped by treatment condition (active vs. control). Baseline suicidality, current depression, current anxiety, current smoking status, and substance use disorder diagnosis were included as covariates. Covarying for baseline suicidality allowed us to look prospectively at development of suicidality. Covarying for current depression, anxiety, and substance use disorders provided a stringent test of the effect of the intervention on current suicidality independent of any current symptoms anxiety, depression or substance use disorders. Smoking status was added as a covariate to the possibility of a direct relationship between nicotine dependence and suicide (Yaworski et al., 2011). For variables showing non-normal distributions (Baseline IDAS suicidality, current IDAS suicidality), log-transformations were conducted that improved the normality of these variables data distributions. However, the pattern of results was the same as using the non-transformed variables. Due to this finding and previous literature suggesting that transformations are not necessary in samples with  $n > 30$  (Pallant, 2007), the non-transformed variables were used in all analyses. In addition, Levene's test of equality of error variances was not significant indicating there was no violation of the assumption of homogeneity of variances.

## Results

### Group comparison on demographic, smoking, and psychopathology variables

Active and control group participants were compared on demographics and relevant baseline variables related to smoking and psychopathology. Descriptive data are shown in Table 1. Chi-square analyses did not reveal any significant group differences on gender, race, marital status, or substance use disorder diagnoses. T-test analyses did not reveal any significant group differences on age, baseline daily cigarettes smoked, baseline ASI-3 total, baseline IDAS depression or suicidality. Baseline ASI-3 scores ( $M = 4.50$ ) were well below the non-clinical means reported in the original normative data ( $M = 12.8$ ; Taylor et al., 2007).

### Effect of Anxiety Sensitivity Reduction Augmented Smoking Treatment on Suicidality

As expected, covariate results indicated that baseline suicidality ( $F(1, 159) = 24.67, p < .001$ , partial eta squared = .14) and current depression ( $F(1, 159) = 11.57, p = .001$ , partial eta squared = .07), were significantly related to current suicidality. Current anxiety ( $F(1, 159) = 3.45, p = .07$ , partial eta squared = .02) was marginally significantly related to current suicidality while smoking status ( $F(1, 159) = 2.31, p = .13$ , partial eta squared = .01) and substance use disorder diagnosis ( $F(1, 159) = .16, p = .69$ , partial eta squared < .01) were not. Moreover, after controlling for effects of current depression, current anxiety, smoking status, substance use disorder diagnosis and baseline suicidality, ANCOVA results revealed the active treatment group ( $M = 6.20, SE = .09$ ) had significantly lower current suicidality compared to the control group ( $M = 6.52, SE = .09; F(1, 159) = 6.07, p = .01$ , partial eta squared = .04).

### Anxiety Sensitivity Reduction Augmented Smoking Treatment on Depression

We conducted a second ANCOVA to test if the treatment also affected current depression. After controlling for effects of baseline depression ( $F(1, 166) = 51.15, p < .001$ ), ANCOVA results indicated there was no significant effect of treatment group on current depression ( $F(1, 166) = 1.86, p = .17$ ).

### Mediation test for Anxiety Sensitivity-3 global and subfactors

Mediation analyses were used to examine whether the relationship between treatment group and current suicidality was mediated by global anxiety sensitivity or anxiety sensitivity subfactors. A mediation model was created to compare the indirect effect of treatment group on suicidality (the effect via anxiety sensitivity) to the null hypothesis that this effect is zero.

The analyses were conducted with the SPSS macro provided by Preacher and Hayes (2004) in order to follow the bootstrapping technique recommended for smaller sizes (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Preacher & Hayes, 2004), and used 5,000 bootstrap resamples of the data. The indirect effect was calculated as the product of the path from treatment group to anxiety sensitivity and the path from anxiety sensitivity to suicidality. In order to determine if there was a significant effect of mediation, 95% confidence intervals of the indirect effect were created. The results revealed no effect of mediation for either ASI-3 global or subfactor scores as indicated by all of the 95% confidence intervals of the indirect effect crossing zero. Additionally, no significant association was found between treatment group and ASI-3 global or treatment group and ASI-3 subfactors.

### Moderator Test

Multiple regression was performed to investigate if the relationship between treatment group and suicidality is moderated by baseline ASI-3 scores. In other words, we wanted to test the hypothesis that there was no significant association between treatment group and current ASI-3 global because many participants had such low AS that reductions in AS were not statistically detectable. However, for those participants with higher baseline AS the treatment should have an observable effect on suicidality. The model contained eight independent variables (baseline IDAS suicidality, current IDAS depression, current IDAS anxiety, smoking status, substance use disorder diagnoses, treatment group, baseline ASI-3 global, and the interaction of baseline ASI-3 global and treatment group). All variables were mean centered prior to inclusion in the model. Current IDAS depression, IDAS anxiety, and substance use disorder diagnoses were included to control for current mood, anxiety, and substance use disorder pathology. Baseline suicidality was included to control for preexisting suicidality. Smoking status was included because of recent findings that smoking may have a direct effect on suicidality (Yaworski et al., 2011). The full model containing all predictors was statistically significant ( $F(8,157) = 12.62, p < .001, R^2 = .39$ ). Among covariates, baseline suicidality ( $\beta = .33, t = 4.87, p < .001, sr^2 = .13$ ), current depression ( $\beta = .27, t = 3.25, p = .001, sr^2 = .06$ ), and current anxiety ( $\beta = .20, t = 2.39, p = .02, sr^2 = .03$ ) were all significant predictors of current IDAS suicidality. Smoking status ( $\beta = .11, t = 1.68, p = .10, sr^2 = .02$ ) was only marginally significantly associated with current suicidality and substance use disorder diagnosis was not significantly related ( $\beta = -.02, t = -.24, p = .81, sr^2 < .01$ ). After accounting for the covariates, treatment group ( $\beta = .16, t = 2.49, p = .01, sr^2 = .04$ ) and the treatment group X baseline ASI-3 global interaction ( $\beta = .14, t = 2.12, p = .03, sr^2 = .03$ ) were significant predictors of current IDAS suicidality. Baseline ASI-3 global was not a significant predictor of suicidality ( $p = .18$ ).

In the next step, we probed the hypothesized 2-way interaction between treatment group and baseline ASI-3 global (See Figure 1). As predicted, at high levels of ASI-3 global (.5 *SD* above the mean) the effect of treatment group was significant ( $\beta = .30, t = 3.24, p = .001, sr^2 = .06$ ), such that those participants in the active treatment who had high baseline AS saw a decline in suicidality. The effect of treatment group at low levels of ASI-3 global was not significant as predicted ( $p = .84$ ).

### Discussion

Recent work has consistently indicated a relationship between AS cognitive concerns and suicidality, across multiple clinical populations, including cigarette smokers (Capron, Blumenthal, et al., 2012; Capron, Cogle, et al., 2012; Capron, Fitch, et al., 2012). The findings from this investigation suggest that an AS augmented smoking cessation treatment may reduce suicidality among cigarette smokers, even after accounting for baseline

suicidality, current depression, current anxiety, and smoking status. This result is consistent with the previous empirical findings that AS cognitive concerns are significantly associated with elevated suicidality in two samples of regular cigarette smokers (Capron, Blumenthal, et al., 2012), and indicates that AS has a unique relationship with suicidality above and beyond the effects of depression and anxiety. Although mediation analyses revealed no effect between treatment group and reductions in AS we believe this is due to the large percentage of patients with minimal baseline AS. Moderation analyses supported this hypothesis. These moderation findings revealed that at high levels of baseline AS there was a significant interaction between treatment group and baseline AS, such that being in the active condition and having high levels of baseline AS was significantly associated with lower current suicidality.

Additionally, the lower current suicidality in the active condition, independent of depression and anxiety, suggests the ability of an AS intervention to reduce suicide risk across different domains of psychopathology. This finding is especially relevant because recent epidemiological studies have found that suicide related outcomes are independently associated with such varied psychopathology as: PTSD (Nepon, Belik, Bolton, & Sareen, 2010; Nock, Hwang, Sampson, & Kessler, 2010), substance use disorders (Nock et al., 2010), major depressive disorder (Bolton & Robinson, 2010), borderline personality disorder (Bolton & Robinson, 2010) and anxiety disorders (Cogle, Keough, Riccardi, & Sachs-Ericsson, 2009; Nepon et al., 2010; Sareen et al., 2005). Encouragingly, the utilization of brief interventions featuring psychoeducation and interoceptive exposure exercises have consistently reduced total AS and AS subfactors across multiple populations (Feldner, Zvolensky, Babson, Leen-Feldner, & Schmidt, 2008; Keough & Schmidt, in press; Schmidt, Eggleston, et al., 2007) Thus, clinicians may benefit from implementing these transdiagnostic AS reduction strategies with clients who endorse elevated suicide risk along with elevated AS.

Despite finding a potential relationship between AS and suicidality, results of mediation analyses indicated that neither anxiety sensitivity or any of its subfactors were mediators of the association between treatment condition and suicidality. There are a number of possible explanations for our failure to find support for this hypothesis. Primarily, the results from the moderation analysis that suggest only participants with high baseline AS had observable declines in suicidality, leading to a lack of power to detect a mediation effect. Another potential reason could be that the AS treatment utilized in this protocol did not, specifically, focus on cognitive AS. The majority of the AS amelioration strategy focused on overall AS, which is made up primarily of AS physical concerns.

Reduction of a related sensitivity/affect tolerance variable is another possibility why we did not see a mediation effect of AS. Factor analysis has revealed that AS and distress tolerance (DT; i.e. perceived ability to experience and endure negative emotional states) appear to be related to one another as distinct lower-order facets of a common higher-order affect tolerance and sensitivity factor (Bernstein, Zvolensky, Vujanovic, & Moos, 2009). There is preliminary evidence of a connection between DT and suicidality (Anestis, Bagge, Tull, & Joiner, 2011; Nock & Mendes, 2008), and treatments that include skills that teach patients how to better tolerate distress have been shown to reduce suicidality (Linehan, Armstrong, Suarez, Allmon, & Heard, 1991; Miller, Rathus, & Linehan, 2007; Rudd, Joiner, & Rajab, 2001). Therefore, it is possible that the AS augmented smoking treatment improved DT, and DT was the mechanism of suicidality reduction. Unfortunately, the smoking cessation protocol did not include a measure of DT as part of the assessment package, and therefore we were unable to test this hypothesis.

It is important to note a few additional limitations in the current study. First, the association between an AS augmented smoking cessation treatment and suicidality may not generalize to actual death by suicide. However, suicidal ideation and suicidal self-injury are strong predictors of death by suicide (Suominen et al., 2004), and both are included on the IDAS suicidality subscale. Use of suicide related outcomes in place of death by suicide is comparable to other research on the connection between anxiety and suicide (Cogle et al., 2009; Sareen et al., 2007; Schmidt et al., 2001). Second, overall suicidality was relatively low in the current sample. It is possible that these findings would not translate to a sample with higher suicidality. However, the level of suicidality is consistent with other work evaluating suicidality and anxiety sensitivity (Schmidt et al., 2001). Third, suicidality was not measured every week. Therefore, we were unable to assess changes over time from baseline to quit week with HLM or linear growth modeling. These statistical methods would have been a more powerful statistical test of the effect of the treatment on suicidality.

The current investigation also had a number of notable characteristics. Primarily, this was the first study to evaluate whether an intervention designed to impact AS would predict lower post-treatment suicidality. This is an important preliminary step forward in this literature as previous work in this area has not come from an AS amelioration project. Second, this sample represents a population (i.e. cigarette smokers) at elevated suicide risk. Recent findings indicate smoking may have a direct role in elevating suicidality, so determining malleable risk factors such as AS, that are related to both smoking and suicide is an important public health consideration. Third, the current report utilized the most comprehensive measures of AS and suicidality in the AS-suicide literature to date. The ASI-3 was designed to accurately measure the AS subfactors and the IDAS suicidality measures several suicide related outcomes such as ideation and self-harm.

The potential for brief anxiety sensitivity interventions to have an effect on mental health outcomes as severe as suicide, clearly warrants further work in this area. One potential future direction is to refine existing AS protocols to include a greater emphasis on AS cognitive concerns. This AS cognitive focused intervention could be used to investigate whether AS cognitive concerns can be targeted more specifically, and if reductions in AS cognitive concerns lead to reductions in suicidality. In addition, this area would also benefit from elucidating the associations and hierarchies between AS and non-AS affect tolerance and sensitivity factors both in general and in association with suicidality.

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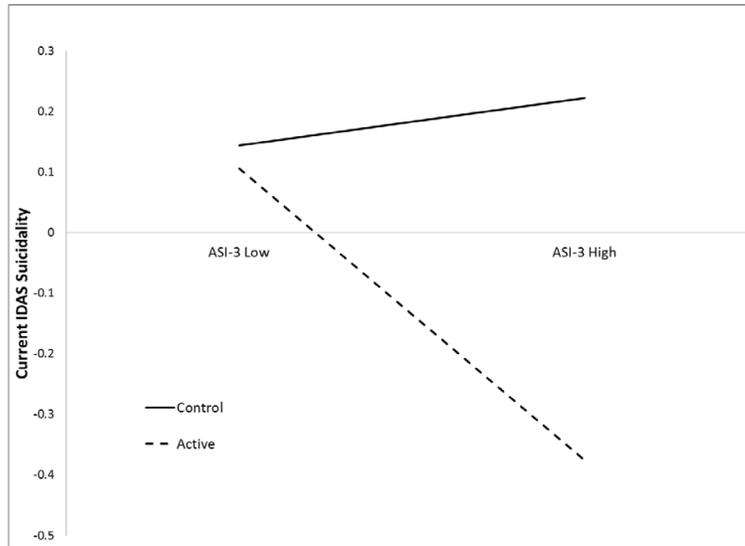
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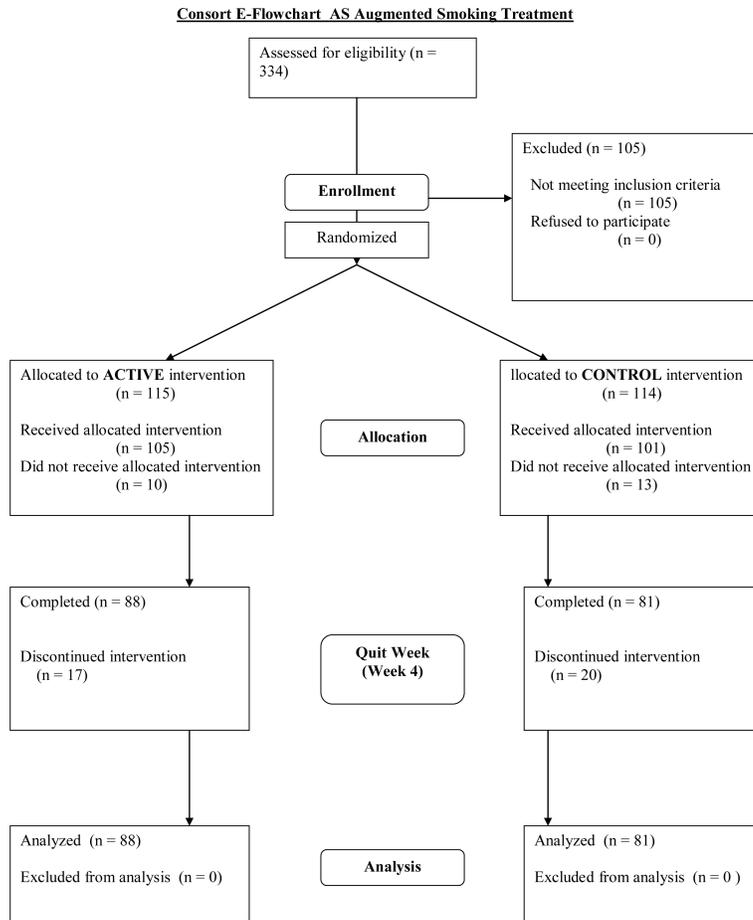
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**Figure 1.** Effect of baseline ASI-3 global score on treatment group in predicting current suicidality among a community sample cigarette smokers. This analysis controlled for baseline suicidality, substance use disorder diagnosis, current depression, current smoking status, and current anxiety. ASI-3 = Anxiety Sensitivity Index-3. IDAS = Inventory of Depression and Anxiety Symptoms.



**Figure 2.**  
CONSORT flow chart.

**Table 1**

Differences between Active and Control Groups on Demographic, Smoking, and Baseline Psychopathology Variables

Variable	Active Group (n = 88)	Control Group (n = 81)
Demographics		
Female	53 (60.2%)	46 (56.8%)
Married	41 (46.6%)	39 (48.1%)
White	76 (86.4%)	68 (84.0%)
Cigarettes Smoked Per Day		
0-10	20 (22.7%)	28 (34.6%)
11-20	46 (52.3%)	37 (45.7%)
21-30	6 (6.8%)	8 (9.9%)
31+	7 (8.0%)	5 (6.2%)
Baseline Psychopathology		
ASI-3	4.88 (5.84)	4.09 (5.34)
IDAS Anxiety	23.45 (7.08)	21.57 (5.28)
IDAS Depression	39.06 (11.37)	37.36 (12.74)
IDAS Suicidality	6.72 (1.33)	6.67 (1.47)
SUD Diagnosis	3 (3.4%)	2 (2.5%)

*Note.* No significant between group differences ( $p > .05$ ). ASI = Anxiety Sensitivity Index. IDAS = Inventory of Depression and Anxiety Symptoms.

SUD = Substance Use Disorder.