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Anxiety sensitivity risk reduction in smokers: A randomized control trial examining effects on panic

Norman B. Schmidt [a, *], Amanda M. Raines [a], Nicholas P. Allan [a], Michael J. Zvolensky [b, c]

[a] Florida State University, Tallahassee, FL, USA
[b] University of Houston, Houston, TX, USA
[c] University of Texas MD Anderson Cancer Center, Houston, TX, USA

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ABSTRACT

Empirical evidence has identified several risk factors for panic psychopathology, including smoking and anxiety sensitivity (AS; the fear of anxiety-related sensations). Smokers with elevated AS are therefore a particularly vulnerable population for panic. Yet, there is little knowledge about how to reduce risk of panic among high AS smokers. The present study prospectively evaluated panic outcomes within the context of a controlled randomized risk reduction program for smokers. Participants ($N = 526$) included current smokers who all received a state-of-the-art smoking cessation intervention with approximately half randomized to the AS reduction intervention termed Panic-smoking Program (PSP). The primary hypotheses focus on examining the effects of a PSP on panic symptoms in the context of this vulnerable population. Consistent with prediction, there was a significant effect of treatment condition on AS, such that individuals in the PSP condition, compared to those in the control condition, demonstrated greater decreases in AS throughout treatment and the follow-up period. In addition, PSP treatment resulted in lower rates of panic-related symptomatology. Moreover, mediation analyses indicated that reductions in AS resulted in lower panic symptoms. The present study provides the first empirical evidence that brief, targeted psychoeducational interventions can mitigate panic risk among smokers.

Empirical evidence suggests that panic attacks and anxiety problems co-occur with smoking at rates that exceed those found in the general non-psychiatric population. For instance, Lasser et al. (2000) found that in an analysis of over 4000 respondents from the National Comorbidity Survey (NCS), current smoking rates for respondents with panic disorder (PD) in the past month or lifetime were significantly greater than smoking rates among respondents with no mental illness. Moreover, reported rates of smoking were highest among individuals with panic-related problems (i.e., history of panic attacks) and other anxiety disorders where panic attacks are common (i.e., posttraumatic stress disorder and generalized anxiety disorder). In regard to smoking contributing to panic specifically, data suggest that smoking initiation typically precedes the onset of panic-related problems (Breslau, Johnson, Hiripi, & Kessler, 2001). For example, Breslau and Klein (1999) tested the association between daily smoking and risk for panic attacks and PD. Results indicated that there was a significant lifetime association between daily smoking and onset of panic attacks and PD; daily smokers were almost 4 times more apt to experience panic attacks and 13 times more likely to develop PD after controlling for major depression and gender.

An integrated theoretical model has been developed to specify how smoking and panic factors are hypothesized to relate to one another (Zvolensky & Schmidt, 2003). In particular, research suggests that among certain daily smokers, smoking serves important affect regulatory functions. This is particularly true for smokers who fear anxiety such as those high in AS. AS, otherwise known as a fear of fear, is a trait like characteristic reflecting a propensity to fear anxiety-related sensations due to the belief that these symptoms have harmful physical, cognitive, and/or social consequences (Reiss & McNally, 1985). At this point in time, AS is perhaps the best-established cognitive causal risk factor for anxiety and panic-spectrum psychopathology (McNally, 2002). In fact, AS is now recognized in the DSM-V as a risk factor for panic (American Psychiatric Association, 2013). One notable aspect of the AS scientific literature is that it comprises both cross-sectional and laboratory tests that have utilized a diverse array of methodological

* Corresponding author. Department of Psychology, Florida State University, 1107 W. Call St., Tallahassee, FL 32306-4301, USA.
E-mail address: schmidt@psy.fsu.edu (N.B. Schmidt).

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approaches and assessment modalities. In regard to cross-sectional tests, for example, there is consistent evidence that AS, as measured by preexposure to biological challenge (panic provocation), is a significant predictor of postchallenge anxiety symptoms and panic attacks among nonclinical individuals (McNally & Eke, 1996). These effects are apparent from adolescence through adulthood (Leen-Feldner, Feldner, Bernstein, McCormick, & Zvolensky, 2005; Rabian, Embry, & MacIntyre, 1999; Schmidt, Lerew, & Jackson, 1997; Zvolensky, Feldner, Eifert, & Stewart, 2001).

Prospective investigations similarly suggest AS predicts the future onset of unexpected panic attacks for adolescents (Hayward, Killen, Kraemer, & Taylor, 2000; Weems, Hayward, Killen, & Taylor, 2002) and adults (Schmidt et al., 1997; Schmidt, Lerew, & Jackson, 1999). The prospective studies also suggest that these effects are apparent for other anxiety symptoms (Schmidt et al., 1997, 1999). Though less well-studied, there are two reports that suggest that AS is related to the future development of anxiety psychopathology, with some degree of specificity for panic disorder relative to other anxiety conditions (Maller & Reiss, 1992; Schmidt, Zvolensky, & Maner, 2006). Collectively, extant findings indicate AS is associated concurrently and prospectively with anxiety symptoms, panic attack symptoms, and full-blown panic attacks.

AS is also thought to influence smoking behavior among daily cigarette smokers. Specifically, these individuals expect tobacco use to help alleviate aversive anxiety states and are often motivated to smoke for affect regulation purposes (Zvolensky et al., 2005). As the mood-altering qualities of smoking are complex (Parrott, 1999), it may well be useful to conceptualize these processes at the cognitive level of analysis. Thus, in the absence of other more adaptive coping strategies, panic-vulnerable smokers may learn to rely on smoking to manage anxiety states and fears of bodily sensations in the short-term. Over longer periods of time, however, smoking itself will lead to increased risk of bodily sensations and aversive internal states via a number of routes, including nicotine-based withdrawal symptoms, health impairment, and physical illness. Exposure to these types of aversive stimuli may facilitate learning that internal cues can be personally harmful, dangerous, and anxiety-evoking. Although smokers with pre-morbid vulnerability factors like high AS may be particularly motivated to quit smoking, they are at high risk for problems in quitting (Zvolensky, Stewart, Vujanovic, Gavric, & Steeves, 2009). Specifically, these persons are apt to be particularly fearful of, and emotionally reactive to, internal states that occur during smoking discontinuation; they may therefore experience more distressing emotional experiences in cessation attempts (Farris, Langdon, DiBello, & Zvolensky, 2015). Thus, a forward feedback loop may develop, whereby smoking is used as a coping strategy for managing aversive states among high AS individuals in the short term yet paradoxically confers longer-term risk for panic attacks and other anxiety problems. This perspective suggests daily smokers are an "at risk" population for panic and other anxiety-related problems and it is important to target them for preventative intervention, as it could lead to improvement in both anxiety status and smoking behavior.

Given findings suggesting AS is a risk for panic and related problems (e.g., smoking), researchers have begun to evaluate the relevance of AS to preventative interventions by determining to what extent this cognitive factor can be changed (malleability). For example, a number of clinical trials with anxiety patients indicate that AS can be reduced through cognitive behavioral interventions. Several investigations focused on panic disorder treatment have reported significant reductions in AS following treatment (Barlow, Craske, Cerny, & Klosko, 1989; Schmidt et al., 2000; Telch et al., 1993; Westling & Öst, 1999). This work is complemented by investigations specifically focused on the reduction of AS as a preventative intervention in nonclinical, at risk samples. Successful preventative work on AS has included two-hour psychoeducation groups (Feldner, Zvolensky, Babson, Leen-Feldner, & Schmidt, 2008), through single day workshops (Gardenswartz & Craske, 2001), and six week exercise programs (Broman-Fulks & Storey, 2008).

To date, the largest AS focused preventative intervention was conducted by Schmidt et al. (2007). Participants ($N = 404$) with ASI scores 1.5 SDs above the nonclinical mean (Schmidt & Joiner, 2002) were randomly assigned to either the Anxiety Sensitivity Amelioration Training (ASAT) condition or a health and nutrition based control condition. The ASAT condition consisted of a 30-min computer PowerPoint presentation followed by ten minutes with an experimenter. The presentation explored the following concepts: the nature of stress, AS, myths about the harmfulness of physiological arousal, and interoceptive exercises (IE). Results indicate that both conditions produced a reduction in AS; however, the ASAT condition produced a significantly larger reduction in AS than the control condition (30% vs. 17%, respectively). In terms of the development of psychopathology, those in the ASAT condition showed a lower incidence of Axis I diagnoses during the two-year follow-up period.

Recently, an augmented version of ASAT was developed in an attempt to increase its potency (Keough & Schmidt, 2012). The revised protocol, Anxiety Sensitivity Education and Reduction Training (ASERT), included more interaction with a therapist, more intensive IE exercises, and more rigorous homework requirements. The level of overall AS reduction was substantial in the active ASERT group (close to 60% at one-month follow-up). A six-month follow-up assessment indicated that the treatment group retained the majority of their AS reduction, whereas the control group retained their elevated AS scores. Finally, Schmidt, Capron, Raines, and Allan (2014) showed that a one session, computer-administered version of ASAT with no therapist involvement was also successful in producing substantial reductions in AS that persisted during a one-month follow-up. In summary, we now have emerging evidence that AS can be effectively mitigated, even with very brief one-session treatments that require minimal therapist or experimenter involvement.

From an intervention standpoint, empirical and theoretical work on smoking and panic problems suggests it may be fruitful to simultaneously and concurrently target these risk factors (i.e., AS and smoking) in one overarching model to reduce panic problems while also stimulating cessation-oriented behavior (e.g., enhance motivation to quit). Because panic factors and smoking interact in clinically meaningful ways, addressing one of these factors without addressing the other in this same context may not result in optimal efficacy regarding intervention goals. For example, simply targeting the cognitive-based fear of anxiety (AS) without a recognition of smoking among those who often manage affect by smoking neglects clinically-relevant self-regulation processes (e.g., escape and avoidant coping for emotionally salient events). Alternatively, because AS is related to poorer success in quitting smoking (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001), a failure to target this cognitive-based affective vulnerability may yield lower rates of success in cessation. Thus, smoking should theoretically be directly targeted within the context of clinical intervention for preventing panic attacks and PD. In terms of addressing smoking and psychological factors, integrative programs are predominant (Brown, Kahler, Niaura, et al., 2001; Cinciripini et al., 1995; Hall, Muñoz, & Reus, 1994). This integrative focus for treatment planning is consistent with the larger literature on systems of integrated, concurrent care for individuals with co-occurring addictive and mental disorders (Mueser & Kavanagh, 2004; Osher, 1996; Pechter & Miller, 1997). Moreover, it is consonant with therapeutic models for anxiety disorders and comorbid addictions that have targeted