

Interaction between smoking rate and anxiety sensitivity: Relation to anticipatory anxiety and panic-relevant avoidance among daily smokers

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Received 13 October 2006; received in revised form 5 November 2006; accepted 7 November 2006

Abstract

The aim of the present investigation was to evaluate the moderating role of anxiety sensitivity (AS) in the relation between smoking rate and panic vulnerability variables, both concurrently and prospectively (3 months), among a community-based sample of 125 daily smokers (60 females; $M_{\text{age}} = 26.02$ years, $S.D. = 10.98$). Consistent with prediction, the interaction between AS and smoking rate significantly predicted concurrent agoraphobic avoidance (3.2% of unique variance) and change in levels of anticipatory anxiety during the 3-month follow-up period (4.7% unique variance). Smokers high in AS who also smoked at greater rates reported the highest levels of avoidance and greatest increase in anticipatory anxiety. Overall, these data suggest that AS is an important individual difference factor that, when coupled with higher rates of smoking, is associated with greater levels of avoidance and anticipatory anxiety among daily smokers.

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Keywords: Panic vulnerability; Agoraphobic avoidance; Anticipatory anxiety; Smoking; Anxiety sensitivity

A variety of studies has indicated that smoking is related to the future risk of developing panic-spectrum problems (panic attacks, panic disorder, and agoraphobia) as well as more severe concurrent expression of such problems (see Zvolensky, Feldner, Leen-Feldner, & McLeish, 2005, for a review). The vast majority of work in this domain has thus far focused on

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documenting whether a relation exists between smoking and certain panic-spectrum problems (i.e., main effect-oriented tests; Zvolensky et al., 2005). Yet, less scholarly attention has been directed at identifying factors that may qualify such smoking-panic effects.

One factor that may play an important role in the smoking-panic association is anxiety sensitivity (AS), a trait-like cognitive characteristic defined as the fear of arousal-related physical and psychological sensations (McNally, 2002; Reiss & McNally, 1985). AS encompasses fears of physical, mental, and publicly observable anxiety experiences (Zinbarg, Barlow, & Brown, 1997) and is theorized to predispose individuals to the development of panic problems (Reiss & Haverkamp, 1996). For example, if a person perceives bodily sensations that are associated with autonomic arousal as a sign of imminent personal harm, this “high AS” individual is theorized to experience elevated levels of anxiety and be at an increased risk for a panic attack. At least three lines of research have strongly supported this line of theorizing. First, prospective studies with adolescents and adults indicate AS predicts the future occurrence of panic attacks and worry about the future occurrence of such attacks (Schmidt, Lerew, & Jackson, 1997; Schmidt, Zvolensky, & Maner, 2006; Weems, Hayward, Killen, & Taylor, 2002). Second, AS is a significant predictor of responses to panic provocation procedures in the laboratory even after controlling for negative affectivity (Zinbarg, Brown, Barlow, & Rapee, 2001). Finally, AS is elevated among persons with a history of PD compared to those without the disorder (Taylor, Koch, & McNally, 1992). Because AS also decreases with remission of panic psychopathology through intervention (Telch et al., 1993), unlike many other panic risk factors (e.g., family history of PD, personal history of panic attacks), it can easily be targeted for therapeutic change in future prevention work.

Recent studies have indicated that AS may influence the documented smoking-panic association by fostering greater anxiety-driven reactions to smoking-related cues (e.g., nicotine withdrawal symptoms, health impairment; Zvolensky, Schmidt, & Stewart, 2003). Specifically, smokers higher in AS should be more apt to react to these cues with greater degrees of anxiety, and by extension, learn that such bodily cues may be personally dangerous (Zvolensky & Bernstein, 2005). Accordingly, daily smokers with high compared to low AS may be more likely to fear aversive interoceptive cues (anticipatory anxiety) and take steps to avoid panic-related situations in the future (panic-relevant avoidance). Broadly consistent with these models, adolescent smokers compared to nonsmokers who were high in AS were more likely to report anxiety focused on bodily cues induced by voluntarily hyperventilation (Leen-Feldner et al., *in press*). Similarly, among a representative sample of adult, daily smokers from Russia, AS interacted with smoking rate in terms of its concurrent relation to agoraphobic avoidance (Zvolensky, Kotov, Antipova, & Schmidt, 2003). A study focused on between-group smoking and AS effects yielded conceptually similar results (Zvolensky, Kotov, Bonn-Miller, Schmidt, & Antipova, *in press*). In these previous studies, no interactive effect was evident for panic attacks, suggesting the interplay between smoking rate and AS may be most applicable to anticipatory anxiety and panic-relevant avoidance rather than panic attacks per se (Leen-Feldner et al., *in press*; Zvolensky, Kotov et al., 2003). Overall, these findings suggest smokers may not be a homogeneous group in regard to key facets of panic vulnerability and that individual differences in AS may be an important factor in accounting for such differences.

The purpose of the present investigation was to replicate and extend past work in a novel manner by addressing two key issues. First, we sought to replicate the Zvolensky, Kotov et al. (2003) AS-smoking rate interaction effect (concurrent) observed in Russia for panic-related avoidance among a sample of young adult daily smokers from North America. Second, we sought to use prospective methodology (3 months) to explore whether AS interacts with smoking

rate in regard to short-term change in anticipatory anxiety among daily smokers. Individuals were excluded from the investigation for a history of panic disorder or agoraphobia, as the investigation was theoretically oriented on the etiology of these problems.¹ For both aims, it was hypothesized that AS would interact with smoking rate to predict greater levels of panic-related avoidance (concurrently) and change in anticipatory anxiety (prospectively). Specifically, higher levels of AS among smokers who smoke at greater rates should theoretically lead to more worry about, and avoidance of, internal cues; however, this same process does not necessarily mean that these same individuals will experience panic attacks, particularly among a sample where pre-existing panic psychopathology was excluded. Additionally, as tests of specificity, no interaction between AS and smoking rate was expected for panic attacks in either the cross-sectional or prospective test, as the conceptual model guiding this work (Zvolensky & Bernstein, 2005) and previous empirical findings (Leen-Feldner et al., *in press*; Zvolensky, Kotov et al., 2003) suggest that the effects should be related to worry about panic symptoms and avoidance rather than the occurrence of panic attacks per se.

1. Method

1.1. Participants

The sample consisted of 125 daily smokers (60 females; $M_{\text{age}} = 26.02$ years, $S.D. = 10.98$). The racial composition of the studied sample generally reflected that of the local population (State of Vermont Department of Health, 2000): approximately 95% of the sample was Caucasian, 4% African-American, and 1% other. Approximately 10% of the sample had at least a 4-year college education, 74% had some college education, 10% had a high school degree or the equivalent, and the remaining 6% did not have a high school education.

Participants smoked, on average, 17.6 cigarettes per day ($S.D. = 8.43$), had smoked cigarettes daily for 7.61 ($S.D. = 8.49$) years, began cigarette smoking at a mean age of 13.48 ($S.D. = 2.91$) years, and considered themselves daily smokers by a mean age of 15.79 ($S.D. = 2.8$) years. The average level of nicotine dependence, as indexed by the Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991), was 3.37 ($S.D. = 2.02$); this reflects a low level of overall nicotine dependence (Heatherton et al., 1991). The average expired carbon monoxide (CO) level for the current sample was 16.2 ppm ($S.D. = 11.23$); scores above 8 ppm are considered indicative of daily smoking (Jarvis, Tunstall-Pedoe, Feyerabend, Vesey, & Saloojee, 1987). 69.6% ($n = 87$) of the participants were regular alcohol users, drinking five to six alcoholic beverages approximately two to three times per week.

Participants were administered the Structured Clinical Interview for DSM-IV Axis I Disorders-Non-Patient Edition by trained raters (First, Spitzer, Gibbon, & Williams, 1995). The participants reported the following history of (current or past) psychiatric problems: 28% had major depressive disorder, 25.6% had experienced non-clinical panic attacks, 11.2% had post-traumatic stress disorder, 4.8% had generalized anxiety disorder, 4.8% had social phobia, and 2.4% had obsessive-compulsive disorder. Reliability ratings by an independent rater were completed on a random selection of 20% of the protocols, with no cases of disagreement being noted.

¹ If smokers with panic disorder or agoraphobia were included in the present investigation, it would not be possible to rule out whether any observed effects are attributable to such conditions rather than the smoking rate by AS interaction.

1.2. Measures

1.2.1. Structured Clinical Interview for DSM-IV Axis I Disorders-Non-Patient Edition (SCID-NP)

The SCID-NP (First et al., 1995) is a well-established diagnostic interview for psychiatric problems. The interview was administered in order to determine participants' history of psychiatric problems, including panic attacks.

1.2.2. Anxiety Sensitivity Index (ASI)

The ASI (Reiss, Peterson, Gursky, & McNally, 1986) measures the degree to which participants fear negative consequences stemming from anxiety symptoms. The ASI shows adequate test–retest reliability ($r = .75$ for 2 weeks), criterion validity (e.g., individuals with agoraphobia score higher than those with other anxiety disorders and those with no disorder), and is distinct from trait anxiety (Reiss et al., 1986).

1.2.3. Panic Disorder Severity Scale (PDSS)

The PDSS is a semi-structured interview rating scale for various facets of panic disorder (Shear et al., 1997), including anticipatory anxiety. The PDSS has good psychometric properties (Shear et al., 1997) and has been used successfully in the past across a range of populations to index various facets of panic-related problems (Shear et al., 2001; Zvolensky, Leen-Feldner et al., 2004). Consistent with past work, we utilized a composite of avoidance of situations and avoidance of bodily sensations (items 4 and 5) to measure panic-related avoidance (Shear et al., 2001). We also used the anticipatory anxiety question (item 3) to measure worry about interoceptive cues. This approach to indexing the key constructs under study was in line with the nature of the sampled population (i.e., individuals without panic disorder or agoraphobia).

1.2.4. Positive Affect Negative Affect Schedule (PANAS)

The PANAS is a mood measure commonly used in psychopathology research (Watson, Clark, & Tellegen, 1988). It assesses two global dimensions of affect: negative and positive. Only the negative affectivity scale (PANAS-NA) was used in this study. A large body of literature supports validity of the PANAS (Watson, 2000; Watson et al., 1988).

1.2.5. Smoking History Questionnaire (SHQ)

Smoking history and pattern was assessed with the SHQ (Brown, Lejuez, Kahler, & Strong, 2002), a measure that includes items pertaining to smoking rate, age of onset of initiation, years of being a daily smoker, etc. The SHQ has successfully been used in previous studies as a measure of smoking history (Brown et al., 2002; Zvolensky, Lejuez, Kahler, & Brown, 2004).

1.2.6. Fagerstrom Test for Nicotine Dependence (FTND)

The FTND is designed to assess gradations in tobacco dependence (Heatherton et al., 1991). The FTND has shown good internal consistency, positive relations with key smoking variables (e.g., saliva cotinine; Heatherton et al., 1991; Payne, Smith, McCracken, McSherry, & Antony, 1994), and high degrees of test–retest reliability (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994).

1.2.7. Expired carbon monoxide

Biochemical verification of smoking status was completed by carbon monoxide (CO) analysis of breath samples assessed using a Bedfont Micro III Smokerlyzer CO Monitor (Model EC50;

Bedfont Scientific USA, Medford, NJ). Research indicates that 8 ppm is an optimal cutoff score for reliably discriminating smoking status (Jarvis et al., 1987).

1.2.8. Alcohol Use Disorders Identification Test (AUDIT)

The AUDIT is a 10-item screening measure developed by the World Health Organization to identify individuals with alcohol problems (Babor, de la Fuente, Saunders, & Grant, 1992). There is a large body of literature attesting to the validity of the AUDIT (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). As in past work (Stewart, Zvolensky, & Eifert, 2001), we used the frequency and quantity items of the AUDIT to index weekly alcohol consumption (an average weekly-based frequency by quantity per occasion composite).

1.3. Procedure

Smoking status was biochemically verified via CO analysis, participants were administered the SCID-NP and PDSS by a trained rater, and then they completed the self-report measures. For ease of presentation, we refer to this first assessment from hereafter as Time 1. Participants were then given instructions regarding procedures for the follow-up period and given US\$ 20 compensation. Participants returned to the laboratory after 3 months and were administered the SCID-NP panic attack module (to index panic attack frequency during the past 3 months) and the PDSS (to index change in anticipatory anxiety ratings over the 3-month assessment).² Participants were then debriefed and given US\$ 30 compensation for their efforts. We refer to this second assessment from hereafter as Time 2.

2. Results

2.1. Analytic approach

The main and interactive effects of smoking rate and AS for the primary dependent variables were evaluated using a hierarchical multiple regression (or logistic regression for binary dependent variables) procedure (Cohen & Cohen, 1983). For the first set of analyses, separate models were constructed for predicting agoraphobic avoidance and a lifetime history of panic attacks. Negative affectivity and weekly alcohol consumption were entered as covariates at step one in the model. At the second step in the model, the main effects for smoking rate and AS were simultaneously entered into the model in order to estimate the amount of variance accounted for by these variables individually. At the third step, the interaction term (mean centered) between smoking rate and AS was entered into the model (Baron & Kenny, 1986).

Hierarchical regression also was employed for the prospective test to determine if the interaction between AS and cigarettes per day at Time 1 accounted for unique variance in predicting the presence of panic attacks during the follow-up period and change in anticipatory anxiety at Time 2. Separate models were, again, constructed for each of the dependent variables.

² Although the PDSS measures other facets of panic problems, we focused on the anticipatory anxiety facet for the prospective analyses for two key reasons. First, anticipatory anxiety indexes a specific worry about interoceptive cues, and hence, is applicable to the model being tested (Zvolensky & Bernstein, 2005). Second, as we ruled out panic disorder and agoraphobia, it did not make empirical or theoretical sense to use a global composite of panic disorder severity (i.e., because they did not have the disorder). Overall, by focusing on anticipatory anxiety and avoidance, we could make the present analytic approach most applicable to the study aims and sampled population.

History of panic attacks was entered as a covariate at step 1 for the panic attack analysis, and level of anticipatory anxiety at Time 1 was entered as a covariate for the anticipatory anxiety analysis. AS and cigarettes smoked per day at Time 1 were entered together at step 2 to evaluate the main effects of these variables. Lastly, the mean centered interaction term (AS $\times$ cigarettes per day at Time 1) was entered at step 3 to test whether the interaction between AS at Time 1 and smoking history at Time 1 predicted panic attacks and anticipatory anxiety at Time 2.

2.2. Zero-order associations among the predictor variables

Please see Table 1 for associations among covariates, predictor variables, and criterion variables for both Time 1 and Time 2. 83.2% of the participants ($n = 104$) returned for the Time 2 assessment and were thus included in the Time 2 analyses. The correlation between smoking rate and AS at Time 1 was minimal ($r = .02, p = .82$). Smoking rate was significantly associated with lifetime history of panic attacks ($r = .19, p < .05$), but was not significantly associated with the presence of panic attacks during the follow-up period ($r = .06, p = .56$). At Time 1, AS was significantly correlated with both agoraphobic avoidance ($r = .41, p < .01$), and lifetime history of panic attacks ($r = .28, p < .01$). AS was not significantly associated with panic attacks ($r = .11, p = .29$) or anticipatory anxiety ($r = .17, p = .09$) at Time 2.

2.3. Time 1 regression equations

In regard to the Time 1 linear regression analysis predicting agoraphobic avoidance, the first step accounted for 18.3% of variance. Negative affectivity was a significant predictor at step 1 of the model ($t = 5.01, \beta = .42, p < .01$), but weekly alcohol consumption was not a significant predictor ($t = -1.04, \beta = -.09, p = .30$). There were no main effects for either AS or smoking rate at step 2 of the model ($t = 1.52, \beta = .19, p = .13$ and $t = .92, \beta = .08, p = .36$, respectively). As hypothesized, the interaction between AS and smoking rate significantly predicted agoraphobic avoidance; the interaction accounted for 3.2% of unique variance ($t = 2.19, \beta = .19, p < .05$).

Table 1
Descriptive data and intercorrelations among predictor and criterion variables

	1	2	3	4	5	6	7	8	9	<i>M</i>	<i>S.D.</i>
1. PANAS-NA	—	.03	.14	.72**	.36**	.39**	.25**	.27**	.17	21.3	8.46
2. Alcohol	—	—	-.30**	.01	-.07	.01	-.17	-.05	-.11	6.68	5.36
3. Cig/Day	—	—	—	.02	.10	.19*	.09	.06	.06	17.6	8.43
4. ASI	—	—	—	—	.41**	.28**	.30**	.11	.17	23.7	15.3
5. T1 Avoidance	—	—	—	—	—	.41**	.81**	.08	-.06	0.21	0.73
6. T1 Panic Attacks	—	—	—	—	—	—	.36**	.36	.22*	—	—
7. T1 AA	—	—	—	—	—	—	—	.03	.03	0.12	0.52
8. T2 Panic Attacks	—	—	—	—	—	—	—	—	.42**	—	—
9. T2 AA	—	—	—	—	—	—	—	—	—	0.06	0.27

Note: A single asterisk indicates correlation is significant at .05 level; a double asterisk indicates correlation is significant at .01 level; PANAS: Positive and Negative Affect Scale-Negative Affectivity subscale (Watson et al., 1988); Alcohol: weekly alcohol consumption; Cig/Day: daily cigarettes; ASI: Anxiety Sensitivity Index-Total Score (Reiss et al., 1986); T1 Avoidance: agoraphobic avoidance at Time 1 assessed via PDSS (Shear et al., 1997); T1 Panic Attacks: history of panic attacks (yes/no) assessed via SCID-NP (First et al., 1995); T1 AA: anticipatory anxiety at Time 1 assessed via PDSS (Shear et al., 1997); T2 Panic Attacks: panic attacks during 3-month follow-up period (yes/no) assessed via SCID-NP (First et al., 1995); T2 AA: anticipatory anxiety at Time 2 assessed via PDSS (Shear et al., 1997).

Based on recommendations of Cohen and Cohen (1983; p. 323, 419), the form of significant interaction was examined by inserting specific values one half of a standard deviation above and below the mean for AS and smoking rate, respectively into the regression equation and plotted (see Fig. 1). Among individuals with higher AS, smoking a greater number of cigarettes was associated with higher levels of agoraphobic avoidance at Time 1, whereas smoking rate had a relatively weaker association with agoraphobic avoidance across the other variable combinations.

In terms of the Time 1 logistic regression predicting lifetime history of panic attacks, negative affectivity ($OR = 1.15, p < .01, 95\% CI = 1.08–1.22$), but not alcohol consumption ($OR = 1.03, p = .53, 95\% CI = .94–1.13$), was associated with a unique change in the odds of having a lifetime history of panic attacks. Neither AS ($OR = .98, p = .47, 95\% CI = .94–1.03$) nor smoking rate ($OR = 1.05, p = .09, 95\% CI = .99–1.11$) were associated with a unique change in the odds of having a lifetime history of panic attacks above and beyond the covariates. The interaction between AS and smoking rate was not related to lifetime history of panic attacks ($OR = 1.00, p = .51, 95\% CI = .99–1.00$).

2.4. Time 2 regression equations

In regard to Time 2 anticipatory anxiety, Time 1 anticipatory anxiety was not a significant predictor at step 1 ($t = .28, \beta = .03, p = .78$). There were no main effects for either AS or smoking rate at step 2 of the model ($t = 1.72, \beta = .18, p = .09$ and $t = .68, \beta = .07, p = .49$, respectively). As hypothesized, the interaction between AS and smoking rate significantly predicted anticipatory anxiety at Time 2 ($t = 2.22, \beta = .22, p < .05$), accounting for 4.7% unique variance. Based on recommendations of Cohen and Cohen (1983; p. 323, 419), the form of significant interaction was examined by inserting specific values one half of a standard deviation above and below the mean for AS and smoking rate, respectively into the regression equation and plotted (see Fig. 2). Higher levels of AS and higher rates of daily smoking were associated with the greatest elevations in anticipatory anxiety at Time 2 compared to other variable combinations.

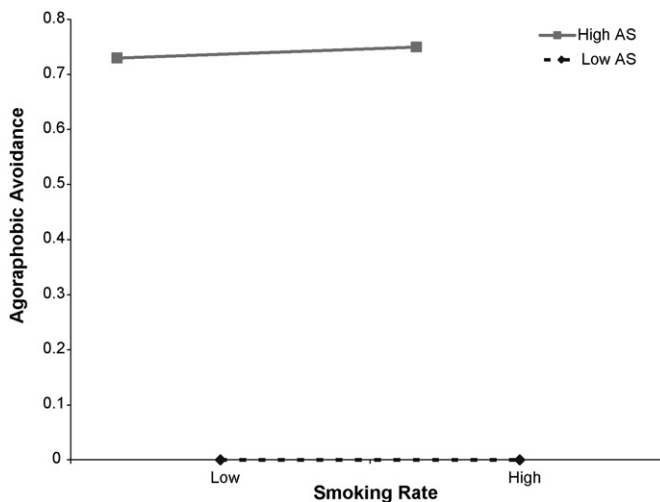


Fig. 1. Time 1 agoraphobic avoidance, as indexed by the Panic Disorder Severity Scale (Shear et al., 1997), as a function of AS and number of cigarettes smoked per day among participants one-half of a standard deviation above and/or below the mean for each predictor.

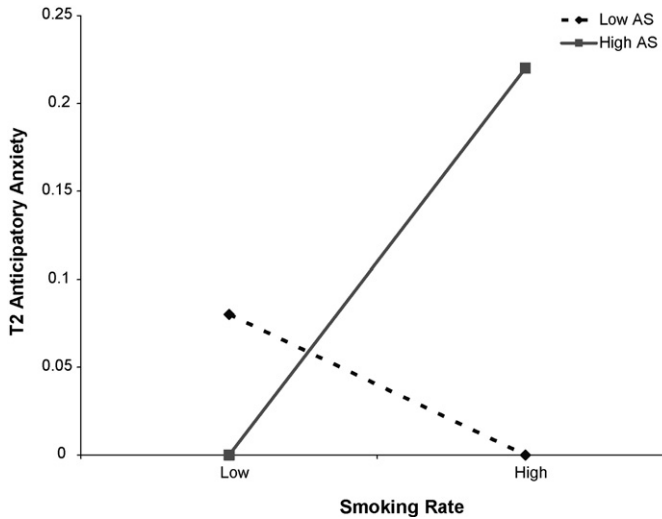


Fig. 2. Time 2 anticipatory anxiety, as indexed by the Panic Disorder Severity Scale (Shear et al., 1997), as a function of AS physical concerns and number of cigarettes smoked per day among participants one-half of a standard deviation above and/or below the mean for each predictor.

Lifetime history of panic attacks ($OR = 8.42, p < .01, 95\% CI = 2.56\text{--}27.68$) was associated with a unique change in the odds of experiencing a panic attack during the follow-up period. Neither AS ($OR = .99, p = .79, 95\% CI = .96\text{--}1.03$) nor smoking rate ($OR = 1.00, p = .98, 95\% CI = .93\text{--}1.07$) were associated with a unique change in the odds of having panic attacks during the follow-up period above and beyond the covariate. As predicted, the interaction between AS and smoking rate was not related to increased likelihood of experiencing panic attacks during the follow-up period ($OR = 1.00, p = .99, 95\% CI = .996\text{--}1.00$).³

3. Discussion

Consistent with prediction, the interaction between AS and smoking rate significantly predicted agoraphobic avoidance in the cross-sectional test. This significant effect, accounting for 3.2% of unique variance, was over and above the main effects as well as the theoretically-relevant covariates of negative affectivity and alcohol consumption. The form of this interaction indicated that smokers high in AS who also smoked at greater rates reported the highest levels of avoidance. Also as expected, there was no such interaction for panic attacks, suggesting some explanatory specificity between AS and smoking rate for avoidance processes. These data replicate and extend past work in Russia (Zvolensky, Kotov et al., 2003) to a young adult sample

³ As an alternative analytic strategy, we also ran these analyses excluding participants with a history of panic attacks rather than using history of panic attacks as a covariate. The pattern of results was similar when these participants were excluded. Neither AS ($OR = .99, p = .77, 95\% CI = .93\text{--}1.05$) nor smoking rate ($OR = .98, p = .79, 95\% CI = .88\text{--}1.10$) were associated with a unique change in the odds of having panic attacks during the follow-up period. The interaction between AS and smoking rate was also not related to increased likelihood of experiencing panic attacks during the follow-up period ($OR = 1.00, p = .60, 95\% CI = .99\text{--}1.01$), although the exclusion of 26% of our sample likely left us underpowered to detect small effects.

of daily smokers from North America. Confidence in this pattern of results was strengthened by the second set of complimentary analyses focused on the prospective assessment. Here, the interaction between AS and smoking rate significantly predicted anticipatory anxiety at the 3-month follow-up period; the size of the effect was clinically meaningful at 4.7% unique variance. Again, higher levels of AS and higher rates of smoking were associated with greater change in anticipatory anxiety. As for the cross-sectional test, no interactive effects were evident for panic attacks. Overall, these data suggest that AS is an important individual difference factor that, when coupled with higher rates of smoking, is associated with greater levels of avoidance and anticipatory anxiety among daily smokers. Importantly, due to the screening process, persons with pre-existing panic psychopathology (panic disorder and agoraphobia) were excluded from the present study and therefore this clinical condition does not account for the observed results.

The present findings add to a growing body of empirical work that suggests AS is useful for identifying subpopulations of smokers who are at greatest risk for panic-relevant vulnerability (Leen-Feldner et al., *in press*; Zvolensky, Kotov et al., 2003). One implication of such work is that specialized prevention approaches for panic psychopathology prevention may usefully target high AS smokers for intervention (Zvolensky, Schmidt, Bernstein, & Keough, 2006). A second implication is that with such an improved understanding of the interactive effects of AS and smoking rate, future scientific attention can be applied to explicating the mechanisms linking these factors, and by extension, refine theoretical models of smoking-panic comorbidity. For example, future research may benefit by using daily monitoring tactics (e.g., ecological momentary devices) for tracking nicotine withdrawal symptoms and evaluating whether such smoking-related cues mediate the association between smoking status and subsequent risk for panic problems. This type of work would enhance our understanding of the processes related to panic vulnerability specifically and serve to illustrate how drug-anxiety processes may work more generally.

Though promising, a number of interpretative caveats warrant consideration. First, although we used community-based advertisements in the recruitment of participants for the present investigation, it is noteworthy that the sample was comprised of relatively young adult daily smokers. The sample may have been younger, on average, than would be expected from typical community-based recruitment due to the fact that advertisements for the study were largely posted in areas of the community frequently visited by young adults (e.g., shopping centers, restaurants, bars) and therefore may have attracted younger adults to a greater extent than older adult smokers. Second, the sample was comprised of regular (daily), but not heavy, smokers. As previous research indicates that the panic-smoking association often is most apparent among heavy smokers, it may prove fruitful for future research to examine panic-vulnerability associations in light, moderate, and heavy smokers. Along these same lines, it may be useful to compare panic-vulnerability associations with smoking rate versus nicotine dependence. A comparison of smoking rate and dependence in the prediction of panic problems would serve to increase specificity of the current biopsychosocial model of panic disorder etiology by specifying which factor(s) play more formative roles in promoting risk.

Third, although the sample was representative of the ethnic composition of the state of Vermont, it was comprised of predominately Caucasian young adults. To improve generalizability of the observed effects, future research could sample from locations with more diverse demographic characteristics. Fourth, a 3-month follow-up period is a fairly short period of time. Although we opted to design the present investigation for 3-month follow-up to establish precedent for examining these matters and assess retention rates among this at-risk population, large changes in symptomatology during this specific window of time were not evident. Future

studies employing longer follow-up periods would better document symptom progression. Finally, self-report measures were utilized as the primary assessment methodology for many of the key constructs. The utilization of self-report methods does not fully protect against reporting errors and may be influenced by shared method variance. Thus, future studies could build on the present work by utilizing laboratory based assessments to provide information about smoking behavior in “real time.” For example, evaluating the predictive power of AS by smoking rate in the prediction of emotional responsivity to biological challenge would help document response patterns across systems.

Together, the present findings suggest daily smokers who have higher smoking rates and higher levels of AS report greater agoraphobic avoidance and increases in anticipatory anxiety over a short period of time. The primary implication of the present findings is that there may be segments of the cigarette smoking population who are at relatively greater risk for panic symptoms by virtue of individual differences in AS. The identification of such moderating effects is clinically important, as it helps to refine our understanding of complex associations between drug behavior and panic vulnerability.

Acknowledgements

This paper was supported by a National Institute on Drug Abuse research grants (1 R21 DA016227-01, 1 R01 DA018734-01A1, and 1 R03 DA016566-01A2) awarded to Dr. Zvolensky.

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