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Anxiety sensitivity interacts with marijuana use in the prediction of anxiety symptoms and panic-related catastrophic thinking among daily tobacco users

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Abstract

The present investigation evaluated whether anxiety sensitivity interacted with marijuana use in relation to the prediction of panic-relevant variables among young adult tobacco smokers ($n = 265$). Approximately 73% of the sample was composed of current marijuana smokers, with 78.5% of this sub-sample using marijuana more than once per week. As expected, after covarying cigarettes per day, alcohol use, and negative affectivity, the interaction between marijuana use and anxiety sensitivity predicted anxiety symptoms and agoraphobic cognitions. Partially consistent with prediction, the interaction between frequency of marijuana use and anxiety sensitivity predicted only anxiety symptoms. These results are discussed in relation to better understanding the potential role of regular marijuana use and anxiety sensitivity for panic-relevant emotional vulnerability among regular tobacco smokers.

Keywords: Marijuana; Anxiety; Catastrophic thinking; Depression; Tobacco; Polysubstance use; Panic attacks

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Introduction

Individuals who smoke cigarettes on a daily basis compared to those that do not are at greater risk for panic psychopathology. For example, descriptive (Zvolensky, Kotov, Antipova, & Schmidt, 2003; Zvolensky, Schmidt, & McCreary, 2003), prospective (Isensee, Wittchen, Stein, Hofler, & Lieb, 2003; Johnson et al., 2000), and laboratory (Zvolensky, Leen-Feldner et al., 2004) studies suggest daily smokers relative to non-smokers are more prone to panic disorder, panic attacks, and anxious responding to bodily sensations. Although these investigations suggest cigarette smokers are an "at risk" segment of the population in regard to panic problems, little is known about the nature of other types of drug use potentially related to panic vulnerability among this population (Zvolensky & Bernstein, 2005). This limitation in knowledge is unfortunate, as cigarette smoking frequently co-occurs with other types of substance use and abuse (Amos, Wiltshire, Bostock, Haw, & McNeill, 2004; Gfroerer, 1995; Hays, Farabee, & Miller, 1998). Marijuana is the most frequently used illicit drug among cigarette smokers (Smart & Ogborne, 2000). Indeed, 43% of young adult current tobacco smokers are "repeat marijuana users" (Report from The National Center on Addiction and Substance Abuse at Columbia University and The American Legacy Foundation, 2003). Other empirical reports suggest that marijuana use is highest among young adults (Fergusson, Horwood, & Swain-Campbell, 2002; NIDA, 1997) and may be on the rise among this particular segment of the population (Ashton, 2001; Ogborne & Smart, 2000; Webb, Ashton, Kelly, & Kamali, 1996).

Aside from occurring at high rates among young adult cigarette smokers, marijuana use may be related to increased risk of emotional disturbances. Although the vast majority of this work has focused on depressive problems (e.g., Bovasso, 2001; Brook, Brook, Zhang, Cohen, & Whiteman, 2002; Grant, 1995; Weller & Halikas, 1985), recent work has linked marijuana use to anxiety vulnerability (Dannon, Lowengrub, Amiaz, Grunhaus, & Kotler, 2004; Tournier, Sorbara, Gindre, Swendsen, & Verdoux, 2003). This work has been stimulated by: (1) the recognition that marijuana use can produce acute episodes of dissociation, anxiety, and panic attacks (Graham, Schultz, & Wilford, 1998; Moran, 1986; Office of Applied Studies [SAMHSA], 2001; Roy-Byrne & Uhde, 1988); and (2) findings that regular use of this drug is associated with increases in risk of physical health problems (e.g., respiratory disease), bodily sensations, and poorer perceptions of health (Bonn-Miller, Zvolensky, Leen-Feldner, Feldner, & Yartz, 2005; Cohen, 1981; Farrow, Rees, & Worthington-Roberts, 1987). For example, regular marijuana-using adults report greater anxiety symptoms than both occasional and non-users (Bonn-Miller et al., 2005; Fergusson & Horwood, 1997; Troisi, Pasini, Saracco, & Spalletta, 1998). Similar results have been reported among adolescents (Rey, Sawyer, Raphael, Patton, & Lynskey, 2002). Moreover, there is an association between marijuana use and increased risk of panic-related problems (Chowdhury & Bera, 1994; Earleywine, 2001; Tournier et al., 2003).

Although existing work on marijuana and anxiety vulnerability generally and panic problems specifically is promising, it has thus far been limited in at least four key respects. First, research has exclusively focused main effects for marijuana use in relation to an anxiety outcome. This approach does not take into consideration individual differences that may qualify such effects. To build more comprehensive models of marijuana-panic processes, it will be important to understand marijuana effects in relation to established individual difference factors associated with panic outcomes. Second, despite marijuana being frequently comorbid with tobacco use

(Amos et al., 2004; Gfroerer, 1995; Hays et al., 1998), few studies have evaluated whether marijuana use relates to anxiety factors over and above the effects of cigarette smoking. Given the established associations between cigarette smoking and panic vulnerability processes (see Zvolensky, Feldner, Leen-Feldner, & McLeish, 2005, for a review), in particular, it is important to isolate marijuana effects from those attributable to tobacco. Third, past work has focused on anxiety symptoms but not addressed other factors important to the study of panic problems. For example, biopsychosocial models of panic disorder suggest cognitive processes, such as catastrophic thinking about anxiety and bodily sensations, are a key characteristic of such problems (Barlow, 2002). Thus, it would be useful to evaluate whether marijuana use relates to panic-relevant cognitive processes in addition to anxiety symptoms. Finally, it is noteworthy that it is presently unclear whether associations between panic problems and marijuana may be attributable to a common personality-based diathesis. In a recent review, for example, Degenhardt, Hall, and Lynskey (2001) highlighted the need to evaluate whether observed associations between marijuana use and emotional problems is accounted for by other common factors such as negative affectivity (i.e., tendency to experience negative affect states). Thus, it is important and timely for research to evaluate the incremental validity of marijuana use over relative to negative affectivity.

One promising approach to address the limitations noted from existing work would be to evaluate the role of anxiety sensitivity in regard to marijuana use among regular cigarette smokers. Anxiety sensitivity, defined as the fear of anxiety and anxiety-related sensations (McNally, 2002), is a traitlike cognitive characteristic that can theoretically predispose individuals to the development of panic problems. Empirical work has supported the basic predictions derived from anxiety sensitivity theory; namely, this variable is related to increased risk of anxiety symptoms (Zinbarg, Brown, Barlow, & Rapee, 2001; Zvolensky, Kotov, Antipova, & Schmidt, 2005) and panic attacks (Hayward, Killen, Kraemer, & Taylor, 2000; Schmidt, Lerew, & Jackson, 1997, 1999). Other work has found that anxiety sensitivity is greatly elevated among individuals who fear interoceptive cues, such as individuals with panic disorder, but only moderately elevated in other anxiety disordered populations (Taylor, Koch, & McNally, 1992). These findings are relevant to the study of marijuana and anxiety vulnerability in that they may suggest that high, but not low, anxiety-sensitive individuals would be more emotionally reactive to internal cues elicited by a history of marijuana use. In fact, past work has shown that marijuana use can produce a variety of internal cues, including anxiety symptoms (Bonn-Miller et al., 2005), bodily sensations (Adams & Martin, 1996), episodes of cognitive dyscontrol (Block & Ghoneim, 1993), and physical disease (Farrow, Rees, & Worthington-Roberts, 1987). Regardless of the exact source of interoceptive cues, high anxiety-sensitive individuals may be apt to perceive such internal stimuli as personally threatening (e.g., "I'm dying," "I'm losing control") or anxiety evoking (e.g., escalating anxiety symptoms). These experiences presumably would represent panic-relevant emotional learning opportunities, wherein internal cues are associated with anxiety (Barlow, 2002; Bouton, Mineka, & Barlow, 2001). From this perspective, associations between marijuana use and panic vulnerability would be qualified by individual differences in anxiety sensitivity. Moreover, anxiety sensitivity should be relatively specific to this emotional state and not covary with affective distress in general (cf. depressive vulnerability).

The overarching aim of the present study was to evaluate a marijuana-anxiety sensitivity model of anxiety and panic-relevant cognitions among regular tobacco users. In this investigation, young

adult tobacco smokers, as opposed to other age groups, were the target population because (1) marijuana use is most prevalent among this segment of the community (Johnston, O'Malley, & Bachman, 2002) and (2) better understanding of emotion vulnerability processes among polysubstance using populations is needed (Newcomb, Vargas-Carmona, & Galaif, 1999). First, it was hypothesized that regular marijuana use compared to non-use would interact with anxiety sensitivity to predict anxiety symptoms and catastrophic thinking about bodily events; these between-subject effects were expected to be over and above variance accounted for by cigarettes per day, alcohol consumption, and the tendency to experience negative affect (i.e., negative affectivity) as well as the main effects of marijuana use and anxiety sensitivity. This prediction is based upon our conceptual analysis that a high anxiety-sensitive marijuana user would be exposed to numerous anxiety-relevant learning experiences involving interoceptive cues. As a test of explanatory specificity, no marijuana by anxiety sensitivity interaction was expected for depressive symptoms because such interoceptive conditioning should be less relevant to depressive symptomatology. Second, among regular marijuana smokers, it was hypothesized that a greater frequency of use would interact with anxiety sensitivity to confer greater risk for anxiety symptoms and catastrophic thinking related to bodily cues, relative to the other theoretically-relevant factors. These within-subjects hypotheses were driven by the idea that more frequent use would provide for a greater amount of anxiety-relevant learning opportunities and increased exposure to bodily sensations. As an index of explanatory specificity, again, no such effect was expected for depressive symptoms.

Method

Participants

The sample consisted of 265 (137 female) regular cigarette smokers from the greater Burlington, Vermont community, recruited through newspaper and other local advertisements. Specifically, we used community-based flyers posted in a local well-traveled market place and newspaper advertisements that provided information about a smoking study for a modest monetary award (e.g., "Are you a daily smoker and interested in earning some extra money?"). See the Procedure section for details. The mean age of the sample was 22.06 (SD = 7.19) years. The racial distribution of the study sample reflected that of the local population (State of Vermont Department of Health, 2000): 94% of the total sample was Caucasian, 3% Asian, 1% African-American, 1% Hispanic, and 1% other. Of those recruited, approximately 76% were considered college students. Participants averaged 12.31 (SD = 6.83) cigarettes per day, had smoked cigarettes regularly for 5.80 (SD = 6.61) years, began cigarette smoking at a mean age of 13.52 (SD = 2.62) years, and considered themselves regular smokers by a mean age of 16.10 (SD = 2.18) years. When smoking tobacco the heaviest, participants averaged 18.67 (SD = 10.01) cigarettes per day. Seventy-three percent ($n = 195$) of the participants were current marijuana smokers, with 78.5% of such participants using this drug more than once per week. Forty-two percent ($n = 110$) of the participants were regular alcohol users, drinking an average of 5 or 6 alcoholic beverages approximately 2–3 times per week.

There were three exclusionary criteria employed in the present study. First, participants were excluded if they had a current or lifetime history of an alcohol use disorder. This exclusionary criterion helped to decrease risk of interpretative complications related to any observed effects being attributed to alcohol use problems (Baker-Morissette, Gulliver, Wiegel, & Barlow, 2004). Second, individuals were excluded if they had a current or lifetime history of panic disorder. This exclusionary criterion was necessary to help ensure that any observed effect for anxiety sensitivity was not simply due to the presence of panic psychopathology, a phenotype associated with this cognitive vulnerability (Taylor et al., 1992). All diagnostic ratings were made based upon responses to the Anxiety Disorders Interview Schedule-IV (ADIS-IV; DiNardo, Brown, & Barlow, 1994); interrater reliability for the ADIS-IV in our laboratory has been very high for Axis I diagnoses across multiple investigations (e.g., Zvolensky, Leen-Feldner et al., 2004; Zvolensky, Schmidt, Antony et al., 2005). Finally, participants were excluded from the study if they evidenced limited mental competency or the inability to give informed, written consent.

Measures

Smoking history and pattern were assessed with the well-established *Smoking History Questionnaire (SHQ)* that includes items pertaining to smoking rate, age of onset at initiation, and years of being a regular smoker. The SHQ has been successfully used in previous studies as a descriptive measure of smoking history (Brown, Lejuez, Kahler, & Strong, 2002; Zvolensky, Lejuez, Kahler, & Brown, 2003; Zvolensky, Schmidt, Antony et al., 2005). We used the smoking rate item for the SHQ as a primary index of "smoking exposure". Smoking rate, compared to other potential indices of smoking history (e.g., nicotine dependence), was investigated as this represents the most well-established descriptive characteristic in past research dealing with smoking and marijuana associations with emotional vulnerability processes (e.g., Block, Gjerde, & Block, 1991; Degenhardt et al., 2001).

The *Marijuana and Alcohol Assessment (MAA)* was used to assess marijuana and alcohol use. The MAA is a five-item measure that includes items examining the (1) presence/absence of current alcohol and marijuana use and (2) frequency (weekly, monthly, and yearly) of such use. As in past work, frequency of marijuana use was standardized by computing endorsement levels on a common metric (weekly; Bonn-Miller et al., 2005; Zvolensky, Baker et al., 2004). In this calculation, amount of marijuana use per occasion is not employed, as there are numerous problems associated with gauging the amount of this drug being used (e.g., differences in potency of drug, ability to accurately recall type of marijuana used on each occasion; Stephens, 1999). Frequency and quantity of alcohol consumption were assessed on this same questionnaire in a manner used in previous research (Stewart, Peterson, & Pihl, 1995). In regard to frequency, participants reported the number of occasions per week on which they normally consumed alcohol; those who consumed alcohol on less than one occasion weekly estimated monthly or yearly frequency. In regard to quantity, participants indicated the average number of alcoholic beverages (using standardized conversions) they normally consumed per drinking occasion. As recommended, an average alcohol volume index was computed via the product of the frequency by quantity assessments (Wechsler, Davenport, Dowdall, Moeykens, & Castillo, 1994).

The *Anxiety Sensitivity Index* (ASI; Reiss, Peterson, Gursky, & McNally, 1986) is a 16-item measure in which respondents indicate on a 5-point Likert-type scale (0 = "very little" to

4 = "very much") the degree to which they are concerned about possible negative consequences of anxiety symptoms (e.g., "It scares me when I feel shaky"). Factor analysis of the scale indicates that it has a hierarchical structure, with three first-order factors entitled AS-Physical Concerns, AS-Mental Incapacitation Concerns, and AS-Social Concerns and a single, higher order general factor (Zinbarg, Barlow, & Brown, 1997). The ASI has high levels of internal consistency for the global score (range of alpha coefficients: 0.79–0.90) and good test-retest reliability ($r = .70$ for 3 years; Peterson & Reiss, 1992). The ASI is unique from, and demonstrates incremental validity relative to, trait anxiety (Rapee & Medoro, 1994); thus, this construct is distinguishable from the frequency of anxiety symptoms (McNally, 1996). In the present investigation, we utilized the total ASI score, as it represents the global-order anxiety sensitivity factor and therefore takes into consideration different types of fears, including fears of panic-related somatic, cognitive, and social cues.

The *Positive Affect Negative Affect Scale (PANAS)* is a 20-item measure in which respondents indicate on a 5-point Likert-type scale (1 = "Very slightly or not at all" to 5 = "Extremely") the extent to which they generally feel different feelings and emotions (e.g., "Hostile"). The PANAS is a well-established mood measure commonly used in psychopathology research (Watson, Clark, & Tellegen, 1988). Factor analysis indicates that it assesses two global dimensions of affect: negative and positive. Both subscales of the PANAS have demonstrated good convergent and discriminant validity. Additionally, both the negative affect as well as the positive affect scales of the PANAS have demonstrated high levels of internal consistency (range of alpha coefficients: .83–.90 and .85–.93, respectively). A large body of literature supports the validity of the PANAS (see Watson, 2000). Only the negative affect scale (PANAS-NA) was used in the present study.

The *Mood and Anxiety Symptom Questionnaire* (MASQ) is a comprehensive measure of affective symptoms (Watson et al., 1995). Participants indicate how much they have experienced each symptom from 1 ("not at all") to 5 ("extremely") during the past week. Factor analysis indicates that this scale taps key anxiety–depression symptom domains. The Anxious Arousal scale (MASQ-AA) measures symptoms of somatic tension and arousal (e.g., "felt dizzy"). The Anhedonic Depression scale (MASQ-AD) measures a loss of interest in life (e.g., "felt nothing was enjoyable") and reverse-keyed items measure positive affect. The General Distress: Depressive Symptoms scale (MASQ: GDD) measures depressed mood expected to be non-differentiating relative to anxiety (e.g., "felt discouraged"). The General Distress: Anxious Symptoms scale (MASQ: GDA) indexes anxious mood expected to be non-differentiating relative to depression (e.g., "felt nervous"). The MASQ shows excellent convergence with other measures of anxiety and depression and good discriminative validity for anxious versus depressive symptoms via the MASQ-AA and MASQ-AD scales, respectively (Watson et al., 1995). The MASQ-AA as well as the MASQ-AD subscales have shown high levels of internal consistency (range of alpha coefficients: .86–.90 and .90–.93, respectively). In the present study, the MASQ-AA and MASQ-AD subscales were used to index anxiety and depressive symptoms, respectively.

The *Agoraphobic Cognitions Questionnaire* (ACQ) asks participants to rate the frequency of 14 maladaptive thoughts about anxiety and panic attack symptoms (Chambless, Caputo, Bright, & Gallagher, 1984). The ratings are made on a 5-point Likert scale (1 = "thought never occurs" to 5 = "thought always occurs"). There are items concerning both social/behavioral concerns and physiological concerns. Scores on the ACQ can be computed by using a total score or two separate scores for the social/behavior and physiological components. The ACQ has good internal

consistency (total score $\alpha = .80$ in populations that match the present non-clinical sample; Kotov, Schmidt, Zvolensky, Vinogradov, & Antipova, 2005), test-retest reliability, and convergent validity with other established measures (Arrindell, 1993; Chambless et al., 1984; Warren, Zgourides, & Englert, 1990; Warren, Zgourides, & Jones, 1989; Yartz, Zvolensky, Gregor, Feldner, & Leen-Feldner, 2005). As in past research (Zvolensky, Kotov et al., 2005), we utilized the ACQ total score as a global index of maladaptive thoughts about anxiety and panic attack symptoms.

Procedure

Interested participants who contacted the research team about the study were given a detailed description of the investigation and scheduled for a laboratory visit. Upon arrival, participants provided written informed consent. Thereafter, a trained research assistant administered the ADIS-IV and, if eligible, participants completed the self-report measures. After the study, participants were debriefed and compensated $30.

Results

Zero-order correlations for theoretically-relevant variables

Associations among variables are displayed in Table 1. The first set of bivariate correlations examined relations between marijuana smokers and non-marijuana smokers (i.e., between-subject tests). Marijuana use (coded as 2 = yes or 1 = no) was significantly positively associated with

Table 1
Descriptive data and intercorrelations among predictor and criterion variables

Variable	1	2	3	4	5	6	7	8	9	*M*	SD
1. MU	—	—	.24*	−.01	.20	.20*	.11	−.11	.26*	1.7	.4
2. FMU	—	—	.07	.07	.09	.06	.00	−.13	.18*	1.5	.8
3. MASQ-AA	—	—	—	−.01	.73*	.67*	.65*	.06	.12	25.4	7.9
4. MASQ-AD	—	—	—	—	.38*	.19*	.05	−.09	.03	47.9	12.9
5. ACQ	—	—	—	—	—	.73*	.77*	.10	−.02	1.6	.5
6. PANAS-NA	—	—	—	—	—	—	.59*	.04	.11	18.2	6.5
7. ASI	—	—	—	—	—	—	—	.05	.03	17.6	11.6
8. Cig/Day	—	—	—	—	—	—	—	—	−.18*	12.3	6.8
9. VAC	—	—	—	—	—	—	—	—	—	7.7	4.7

Note: An asterisk indicates correlation is significant at .05 level; MU: marijuana use (i.e., between subject associations; coded as 2 = yes or 1 = no); FMU: frequency of marijuana use (i.e., within subject associations; coded as less than once per week (0), once per week (1), or more than once per week (2)); MASQ-AA: Mood and Anxiety Symptom Questionnaire—Anxious Arousal (Watson et al., 1995); M; ASQ-AD: Mood and Anxiety Symptom Questionnaire—Anhedonic Depression (Watson et al., 1995); ACQ: Agoraphobic Cognitions Questionnaire (Chambless, Caputo, Bright, & Gallegher, 1984); PANAS-NA: Positive Affect/Negative Affect Schedule—Negative Affect Scale (Watson et al., 1988); ASI: Anxiety Sensitivity Index (Reiss et al., 1986); Cig/Day: daily cigarettes; VAC: volume of alcohol consumed.

MASQ-AA ($r = .24$, $p < .05$), such that those reporting using this drug reported greater levels of anxiety symptoms (Table 2). Marijuana use was not correlated with MASQ-AD ($r = -.01$, n.s.) or agoraphobic cognitions, $r = .20$, $p > .1$. Marijuana use was significantly associated with average volume of alcohol consumed ($r = .26$, $p < .05$) and negative affectivity ($r = .20$, $p < .05$), but not amount of cigarettes smoked per day ($r = -.11$, n.s.) or anxiety sensitivity ($r = .11$, n.s.).

Table 2
Anxiety sensitivity by marijuana use (yes/no) predicting anxiety symptoms, depressive symptoms, and agoraphobic cognitions

	ΔR^2	t (each predictor)	β	sr^2	p
Criterion variable: anxiety symptoms					
Step 1	.45				<.05
Daily cigarettes		.12	.00	.00	ns
Volume of alcohol consumed		.83	.05	.00	ns
Negative affectivity		11.78	.66	.44	<.05
Step 2	.08				<.05
Marijuana use[a]		1.82	.09	.02	ns
ASI		5.06	.38	.13	<.01
Step 3	.01				<.05
ASI X marijuana use[a]		2.01	.53	.02	<.05
Criterion variable: depressive symptoms					
Step 1	.05				<.05
Daily cigarettes		−1.42	−.11	.01	ns
Volume of alcohol consumed		−.20	−.01	.00	ns
Negative affectivity		2.63	.20	.04	<.05
Step 2	.01				ns
Marijuana use[a]		−.81	−.06	.00	ns
ASI		−1.31	−.14	.00	ns
Step 3	.00				ns
ASI X marijuana use[a]		.88	.33	.00	ns
Criterion variable: agoraphobic cognitions					
Step 1	.54				<.05
Daily cigarettes		−1.03	−.10	.02	ns
Volume of alcohol consumed		−.57	−.05	.01	ns
Negative affectivity		8.24	.75	.53	<.05
Step 2	.10				<.05
Marijuana use[a]		.56	.05	.00	ns
ASI		3.95	.52	.21	<.05
Step 3	.05				<.05
ASI X marijuana use[a]		2.90	1.23	.13	<.05

Note: β = standardized beta weight; sr^2 = squared partial correlation.
[a]Yes dummy coded as "2" and No as "1"; ASI: Anxiety Sensitivity Index (Reiss et al., 1986).

The next set of descriptive zero-order correlations focused on within-subjects associations among frequency of marijuana use (i.e., restricted to only those that endorsed using the drug; $n = 126$) and the theoretically-relevant outcome variables. Frequency of marijuana use was significantly associated with average volume of alcohol consumed ($r = .18$, $p < .05$). Frequency of marijuana use was not associated with MASQ-AA scores ($r = .07$, $p > .1$), MASQ-AD scores ($r = .07$, $p > .1$), agoraphobic cognitions ($r = .09$, $p > .1$), negative affectivity ($r = .06$, $p > .1$), anxiety sensitivity ($r = .00$, $p > .1$), or amount of cigarettes smoked per day ($r = -.13$, $p > .1$).

Regression equations

The main and interactive relations between (1) anxiety sensitivity and marijuana use (yes versus no) and (2) anxiety sensitivity and frequency of marijuana use (less than once/week, once/week, or more than once/week) were evaluated in relation to the primary dependent variables using a hierarchical multiple regression procedure (Cohen & Cohen, 1983). Negative affectivity was included, specifically, as a covariate to evaluate the explanatory specificity of anxiety sensitivity relative to this "higher-order" and theoretically-relevant personality factor. Separate models were constructed for predicting anxious arousal, anhedonic depression, and agoraphobic cognitions. Negative affectivity, volume of alcohol consumed, and cigarettes smoked per day were entered as covariates at level one in the model; this analytic approach ensures that observed anxiety sensitivity-marijuana associations are above and beyond the variance accounted for by these other factors. At the second level, the main effects for anxiety sensitivity and frequency of marijuana use or anxiety sensitivity and marijuana use (depending on which model was being tested) were simultaneously entered into the model as a set in order to estimate the amount of variance accounted for by these variables individually. At the third level, the interaction term between anxiety sensitivity and the appropriate marijuana variable was entered into the model (Baron & Kenny, 1986).

Between-group analyses for marijuana users and non-users.

In terms of the interaction between anxiety sensitivity and marijuana use predicting anxiety symptoms, level one of the model accounted for 45% of the variance. Negative affectivity ($t = 11.78$, $\beta = .66$, $p < .05$) was the only significant predictor. After controlling for variance accounted for by level one of the model, there was a significant main effect for anxiety sensitivity in predicting anxiety symptoms at level two ($t = 5.06$, $\beta = .38$, $p < .05$). As hypothesized, the interaction term was a significant predictor at level 3 ($t = 2.01$, $\beta = .53$, $p < .05$).

In regard to depression, level one of the model accounted for 5% of the variance in this criterion variable, with negative affectivity being the only significant predictor, $t = 2.63$, $\beta = .20$, $p < .05$. After controlling for the non-significant variance accounted for by levels one and two of the model, the interaction term was not a significant predictor of depressive symptoms at level three, as expected.

In terms of agoraphobic cognitions, the first level accounted for 54% of the variance. Negative affectivity ($t = 8.24$, $\beta = .75$, $p < .05$) was the only significant predictor. A main effect for anxiety sensitivity was found at level two of the model ($t = 3.95$, $\beta = .52$, $p < .05$). As hypothesized, the interaction between anxiety sensitivity and marijuana use significantly predicted agoraphobic

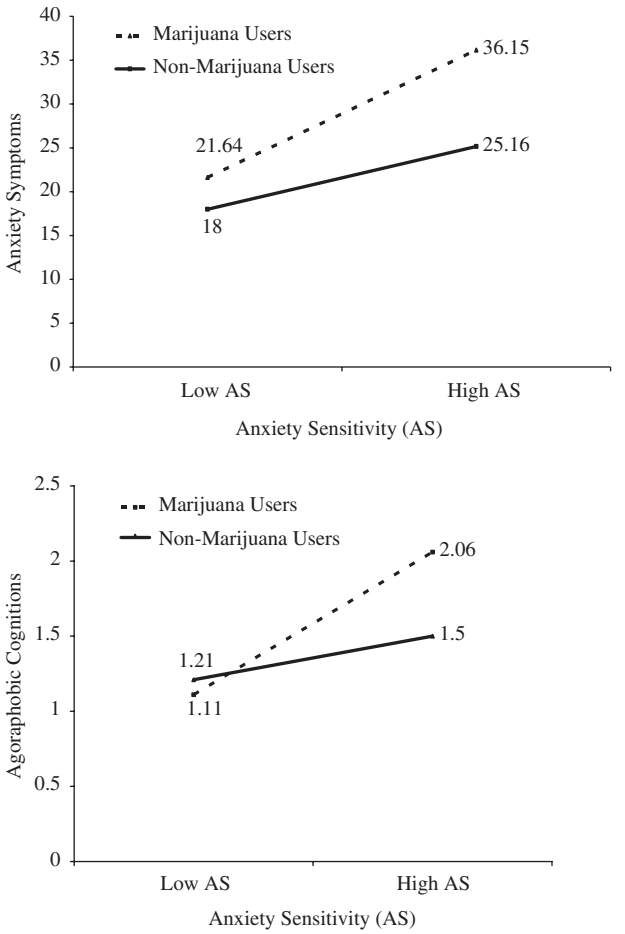

Fig. 1. Interactions between marijuana use (yes versus no) and anxiety sensitivity predicting anxiety symptoms and agoraphobic cognitions.

cognitions at level three of the model; it accounted for approximately 5% of unique variance ($t = 2.90$, $\beta = 1.23$, $p < .05$).

Based on recommendations of Cohen and Cohen (1983, pp. 323, 419), the form of these interactions were examined by inserting specific values for each predictor variable into the regression equations associated with the described analysis. As can be seen in Fig. 1, the form of the interactions supported hypotheses. Specifically, high levels of AS and marijuana use are associated with increased anxiety symptoms and agoraphobic cognitions compared to being high on only one or neither of these factors.

Within-group analyses for marijuana users

In terms of the interaction between frequency of marijuana use and anxiety sensitivity predicting anxiety symptoms, level one of the model accounted for 54% of the variance, with negative affectivity ($t = 11.48$, $\beta = .72$, $p < .05$) being the only significant predictor. A main effect

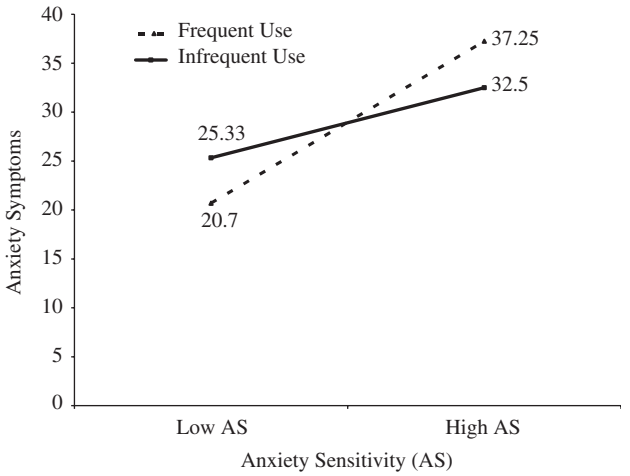

Fig. 2. Interactions between frequency of marijuana use and anxiety sensitivity predicting anxiety symptoms.

for anxiety sensitivity was found at level two ($t = 4.03$, $\beta = .37$, $p < .05$). As hypothesized, after controlling for the variance accounted for by levels one and two of the model, the interaction term was a significant predictor of anxiety symptoms at level three, accounting for 1% of unique variance ($t = 2.03$, $\beta = .28$, $p < .05$). Once again, evaluation of the form of the interaction (see Fig. 2) was consistent with prediction.

In terms of depressed mood, level one of the model accounted for 9% of the variance in depressive symptoms, with negative affectivity being the only significant predictor ($t = 2.90$, $\beta = .26$, $p < .05$). As hypothesized, the interaction between frequency of marijuana use and anxiety sensitivity was not a significant predictor at level three (see Table 3).

In regard to the interaction between frequency of marijuana use and anxiety sensitivity predicting agoraphobic cognitions, the first level accounted for 55% of the variance. Negative affectivity ($t = 7.19$, $\beta = .76$, $p < .05$) was the only significant predictor. At level two, a significant main effect was only found for anxiety sensitivity ($t = 3.97$, $\beta = .64$, $p < .05$). In contrast to prediction, the interaction did not predict agoraphobic cognitions (see Table 3).

Discussion

Consistent with prediction, marijuana users compared to non-users were at increased risk for anxiety symptoms and catastrophic thinking about bodily events among cigarette smokers high but not low in anxiety sensitivity; a difference between users and non-users was only seen for those high in anxiety sensitivity, not for those low in anxiety sensitivity (see Fig. 1). These significant effects, ranging in size from 2% to 13% of unique variance, were above and beyond variance accounted for by theoretically-relevant covariates (i.e., negative affectivity, alcohol use, and cigarettes per day) as well as the respective main effects. Also as expected, there was explanatory specificity to the marijuana by anxiety sensitivity interaction, as no significant interaction was evident for depressive symptoms. Thus, there was overarching consistency in this cross-sectional

Table 3
Anxiety sensitivity × frequency of marijuana use predicting anxiety symptoms, depressive symptoms, and agoraphobic cognitions

	ΔR^2	t (each predictor)	β	sr^2	p
Criterion variable: anxiety symptoms					
Step 1	.54				<.05
Daily cigarettes		1.24	.08	.01	ns
Volume of alcohol consumed		.75	.05	.00	ns
Negative affectivity		11.48	.72	.52	<.05
Step 2	.06				<.05
Frequency of marijuana use		.76	.04	.00	ns
ASI		4.03	.37	.12	<.05
Step 3	.01				<.05
ASI X frequency of marijuana use		2.03	.28	.03	<.05
Criterion variable: depressive symptoms					
Step 1	.09				<.05
Daily cigarettes		−2.25	−.20	.04	<.05
Volume of alcohol consumed		.52	.05	.00	ns
Negative affectivity		2.90	.26	.06	<.05
Step 2	.01				ns
Frequency of marijuana use		−.69	−.06	.00	ns
ASI		−.50	−.07	.00	ns
Step 3	.01				ns
ASI X frequency of marijuana use		−.94	−.20	.01	ns
Criterion variable: agoraphobic cognitions					
Step 1	.55				<.05
Daily cigarettes		−.64	−.07	.01	ns
Volume of alcohol consumed		−.30	−.03	.00	ns
Negative affectivity		7.19	.76	.54	<.05
Step 2	.13				<.05
Frequency of marijuana use		.70	.06	.01	ns
ASI		3.97	.64	.27	<.05
Step 3	.02				ns
ASI X frequency of marijuana use		1.70	.37	.07	ns

Note: β = standardized beta weight; sr^2 = squared partial correlation; ASI: Anxiety Sensitivity Index (Reiss et al., 1986).

study that anxiety sensitivity is an important cognitive factor in terms of better understanding the relation between marijuana use and panic-relevant processes among young adult tobacco users. The potential clinical significance of the observed effects should be understood within the context in which they were examined, namely, after controlling for the variance associated with a number of theoretically-relevant factors (53% and 64% of variance, respectively, for anxiety symptoms and catastrophic thinking). Moreover, by virtue of the screening criteria employed, none of the

participants had a pre-existing axis I psychiatric history. Thus, the observed findings cannot be attributed to psychiatric conditions associated with marijuana use or anxiety sensitivity. Indeed, these data suggest that the combination of marijuana use and anxiety sensitivity offers unique explanatory value in regard to anxiety symptoms and panic-related catastrophic thinking among young adult tobacco smokers.

Also consistent with prediction, among marijuana users, a significant anxiety sensitivity by marijuana frequency (weekly amount) interaction was observed; it explained 1% of unique variance. Inspection of the form of the interaction (see Fig. 2) indicated that higher levels of anxiety sensitivity and higher levels of amount of marijuana used per week, but not other combinations of these factors, were associated with increased risk of anxiety symptoms. As before, these effects were over and above the variance accounted for by a variety of theoretically-relevant emotional (e.g., negative affectivity) and drug (e.g., cigarettes per day) variables as well as the main effects of anxiety sensitivity and frequency of marijuana use. Additionally, there was no evidence of a significant frequency of marijuana use by anxiety sensitivity interaction for depressive symptoms, again providing evidence of explanatory specificity. In contrast to prediction, however, there also was no significant interaction for catastrophic thinking about interoceptive cues. These data suggest that amount of marijuana used per week does not interact with anxiety sensitivity to increase the risk of catastrophic thinking. On the one hand, these data highlight the apparently robust nature of simply being a regular marijuana user, regardless of frequency of usage. On the other hand, the self-reported measurement of frequency of marijuana use (e.g., once/week) may be limited as an index due to the potential influence of marijuana-related recall biases and memory distortions. This assessment issue has historically been an overarching challenge to marijuana research (see Stephens, 1999, for a discussion). Future work might attempt to improve the assessment of frequency of marijuana use through ecological momentary recording devices that prompt individuals to record amount used in real time. It also may prove useful to extend marijuana assessment beyond frequency of use and incorporate other measurement parameters such as potency of drug and amount inhaled. This type of work, of course, will require utilization of laboratory paradigms and biochemical technologies.

Overall, there was broad-based consistency of the hypothesized anxiety sensitivity by marijuana interactions in regard to panic-relevant outcomes. 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 anxiety symptoms and catastrophic thinking by virtue of comorbid marijuana use and individual differences in anxiety sensitivity. The identification of such effects is clinically important, as it helps to refine our understanding of complex associations between drug behavior and panic vulnerability. Although generally in accord with theory, the mechanisms by which these effects are achieved cannot be explicated in the present investigation. One important avenue for future research might be to evaluate biobehavioral processes using emotion elicitation paradigms. For example, it would be useful to document whether anxiety sensitivity and marijuana use predict panic attack symptoms using biological challenge procedures (Zvolensky & Eifert, 2000). This work, aside from removing concern about recall biases or memory distortions, would permit an evaluation of physiological processes as well as self-report data in one overarching model. This work, if consistent with the current observed effects, would set the stage for theory-driven work aimed at uncovering the mechanisms of action.

There are a number of interpretative caveats and directions for future study that warrant comment. First, the present cross-sectional design does not permit causal-oriented hypothesis testing. Although we attempted to strengthen confidence in the observed findings by controlling for alcohol dependence, cigarettes per day, and negative affectivity, the direction of the observed relations cannot be unambiguously determined. Thus, the present data will need to be extended to vulnerability processes across settings and larger time periods. Prospective high-risk designs, which involve monitoring psychologically healthy people who have specific combinations of elevated theoretically-relevant risk factors over time, are one way to examine such issues. Second, given that the present sample, by virtue of selection criteria, was comprised of young adult cigarette smokers, the findings are not generalizable to all cigarette smokers. To further enhance the generalizability of the study results, future work may benefit by sampling from a more diverse smoking population. Third, an ongoing challenge to marijuana research is attaining reliable and valid assessments of the parameters of this drug behavior (Stephens, 1999). In the present investigation, we followed previous work and examined marijuana use and frequency of such use. However, it was not possible to attain an assessment of quantity of use due to the wide degree of variability in marijuana types (i.e., potency) and problems related to reliance on participants' recall of amount used (i.e., memory distortions). Although there is no consensus regarding how to unambiguously address this issue, utilization of biochemical methods would be helpful in further corroborating a distinction of at least "use versus non-use" of marijuana. Here, it also would be advisable for researchers to track motivation for marijuana use rather than just amount of degree or level of use. That is, there may be important function-based patterns of use among marijuana users with differing levels of anxiety sensitivity or other psychological characteristics.

Fourth, the present investigation was focused on regular users of marijuana who also were regular tobacco users. This is a clinically-relevant, albeit understudied population, in terms of panic vulnerability (Zvolensky, Feldner et al., 2005). Nonetheless, future work could begin to meaningfully extend such work in new directions by evaluating marijuana in relation to panic vulnerability among different developmental phases of drug use. For example, it may be that attempts to discontinue marijuana use provide a fertile context for individuals high in anxiety sensitivity to react with fear and panic attacks. Such effects may be particularly relevant if the individual was attempting to discontinue both marijuana use and quit cigarette smoking. Identifying answers to these types of questions should yield important information both in terms of better understanding panic vulnerability and perhaps facilitating success in discontinuing marijuana use. Finally, it is noteworthy that recent work on anxiety sensitivity, across diverse populations of youth and adults, has indicated that it is taxonic (e.g., Bernstein, Zvolensky, Kotov, Arrindell et al., in press; Bernstein, Zvolensky, Weems, Stickle, & Leen-Feldner, 2005; Schmidt, Kotov, Lerew, Joiner, & Ialongo, in press); that is, there are two discrete forms of anxiety sensitivity that differ between individuals qualitatively (see Meehl, 2004, for a discussion of taxa). Although we did not use a taxonic model of anxiety sensitivity in the present investigation due to an insufficient number of individuals to conduct such analyses, future work could further extend the present findings by employing such analytic approaches. In this same context, it may be fruitful for future research to evaluate the explanatory specificity of anxiety sensitivity effects. For example, future research could test the competing roles of anxiety sensitivity and negative affectivity by entering them in regression equations in a sequenced,

alternative format. This test would clarify the degree to which these variables offer unique explanatory value relative to one another.

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