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## Negative Affect as a Mediator of the Relationship between Vigorous-Intensity Exercise and Smoking

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### Abstract

The present cross-sectional study evaluated whether people who engage in vigorous-intensity exercise are better able to regulate negative affective states, thereby changing core maintenance factors of smoking. Participants were a community sample of adults ( $n = 270$ ) who completed self-report measures of physical activity, cigarette smoking, anxiety sensitivity, and negative affect. Consistent with hypothesis, vigorous-intensity exercise was related to lower levels of cigarette smoking, accounting for 10% of the variance in smoking. Additionally, negative affect mediated the relationship between vigorous-intensity physical activity and cigarette smoking, accounting for about 12% of this relation. Furthermore, these relationships were stronger for individuals with high anxiety sensitivity than for those with low anxiety sensitivity; including anxiety sensitivity as a moderator of the mediated relationship increased the amount of variance accounted for by negative affect to 17%. The findings are discussed in relation to developing further scientific insight into the mechanisms and pathways relevant to understanding the association among vigorous-intensity exercise, smoking, and emotional vulnerability.

### Keywords

Exercise; Physical Activity; Smoking; Negative Affect; Anxiety Sensitivity

## 1. Introduction

Growing evidence points to the role of negative affect in the maintenance of smoking and smoking cessation relapse. For example, when asked about triggers of smoking cessation relapse smokers consistently point to the experience of stress and negative affect (Brandon & Baker, 1991; Piper et al., 2004). These retrospective reports are complemented by prospective studies that indicate that negative affect is an important precipitating factor in smoking lapses and relapses. Specifically, ecological momentary assessments from 215 smokers collected during the two weeks before and four weeks after initiation of smoking cessation treatment

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indicate that abrupt increases in negative affect are associated with smoking lapses (Shiffman & Waters, 2004). Similarly, baseline negative affect and increases in negative affect during treatment have been shown to be the most reliable predictors of relapse in clinical trials of smoking cessation treatments (Covey, Glassman, & Stetner, 1990; Hitsman et al., 1999; Kahler et al., 2002; Lerman et al., 2002; Niaura et al., 2001; Zelman, Brandon, Jorenby, & Baker, 1992; Zvolensky et al., 2008). Lastly, reducing negative affect during smoking cessation treatment has been shown to improve abstinence outcomes both with psychological interventions (e.g., Fergusson, Goodwin, & Horwood, 2003; Hall, Muñoz, & Reus, 1994; Haas, Muñoz, Humfleet, Reus, & Hall, 2004) and pharmacological interventions (e.g., Hughes, Stead, & Lancaster, 2007; Prochazka, Kick, Steinbrunn, Miyoshi, & Fryer, 2004; Richmond & Zwar, 2003). Collectively, these findings suggests that addressing negative affect in smokers may be important especially for those who are more prone to experience negative affect (Brown et al., 2001; Haas et al., 2004).

The relatively poor outcomes of standard smoking cessation treatments (Fiore, 2000; Hughes, Keeley, & Naud, 2004; Piasecki, 2006) combined with the observation that targeting negative affect during treatment may be critical to cessation success for many smokers (i.e., those who are prone to experience negative affect) provide justification for investigating the utility of exercise as an intervention for smoking cessation. Indeed, exercise is associated with reduced negative affect (Focht, Knapp, Gavin, Raedeke, & Hickner, 2007; Hassmen, Koivula, & Uutela, 2000; Penedo & Dahn, 2005; Reed & Ones, 2006; Schlicht, 1994) and more importantly, exercise interventions have shown efficacy for the treatment of mood and anxiety problems (Smits et al., 2008; Stathopoulou, Powers, Berry, Smits, & Otto, 2006; Broocks et al., 1998; Martinsen, Hoffart, & Solberg, 1989a, 1989b). Furthermore, cross-sectional surveys have consistently shown a negative relationship between physical activity levels and smoking (e.g. Boutelle, Murray, Jeffery, Hennrikus, & Lando, 2000; Boyle, O'Connor, Pronk, & Tan, 2000; Hu et al., 2002). Likewise, there is initial evidence from randomized controlled trials indicating that exercise interventions can decrease withdrawal symptoms and negative affect in smokers (Bock, Marcus, King, Borrelli, & Roberts, 1999; Schneider, Spring, & Pagoto, 2007; Taylor, Ussher, & Faulkner, 2007) as well as improve smoking cessation outcomes among adults receiving standard cessation treatments (cf. Ussher, Taylor, & Faulkner, 2008; Marcus, Albrecht, Niaura, Abrams, & Thompson 1991; Marcus et al., 1995; Marcus et al., 1999; Martin, Kalfas, & Patten, 1997). For example, Marcus and colleagues (1999) randomized 281 sedentary female smokers to either a 12-week cognitive-behavioral smoking cessation program with vigorous-intensity exercise (three sessions a week of 30 to 40 minutes at 60–85% of heart rate reserve), or a 12-week cognitive-behavioral smoking cessation program with contact control (three 45–60 minute health education sessions a week). All participants initiated the intervention three weeks prior to the quit date of the smoking cessation program. Results revealed that participants receiving the exercise intervention were more likely than participants in the control intervention to be continuously abstinent during the 8, 20, and 60 weeks following the quit date. Unfortunately, neither this study nor other studies in this area have investigated whether the association between exercise and reduced smoking is accounted for by reductions in negative affect. Evidence for this mediational hypothesis would help determine whether exercise is a viable option for smokers for whom negative affect operates prominently in the maintenance of smoking and smoking cessation relapse.

This study aimed to provide a preliminary test of the hypothesis that the association between exercise and smoking is, at least in part, accounted for by reduced negative affect. Using cross-sectional data, we examined self-reported negative affect as a mediator of the relationship between self-reported vigorous-intensity exercise levels and smoking. We chose to evaluate the relationship between *vigorous-intensity* exercise and smoking because there is evidence to suggest that the association between exercise and cigarette smoking is stronger for vigorous-intensity exercise relative to moderate- or low-intensity exercise (cf. Kaczynski, Manske,

Mannell, & Grewal, 2008). We also investigated the possibility that the strength of these meditational effects would vary as a function of anxiety sensitivity. Anxiety sensitivity, conceptualized as an emotional vulnerability variable, is a relatively stable trait (Peterson & Plehn, 1999; Weems, Hayward, Killen, & Taylor, 2002) characterized by the fear of both anxiety and related autonomic arousal sensations (e.g., racing heart, sweating, nausea; Reiss, Peterson, Gursky, & McNally, 1986). We selected anxiety sensitivity as a possible moderator of the hypothesized mechanism because of the increasing evidence that individuals with elevated levels of anxiety sensitivity, relative to persons with low levels of anxiety sensitivity, are more likely to smoke in response to negative affect (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Brown, Lejuez, Kahler, & Strong, 2002; Novak, Burgess, Clark, Zvolensky, & Brown, 2003; Zvolensky, Bonn-Miller, Bernstein, & Marshall, 2006). Furthermore, smokers with higher levels of anxiety sensitivity are more likely to report negative affect reduction as a smoking outcome expectancy than smokers with lower levels of anxiety sensitivity (Brown et al., 2001; Zvolensky et al., 2007). Accordingly, negative affect reduction as a mechanism underlying the relationship between exercise and smoking may be more salient for individuals with high versus low anxiety sensitivity. We tested the following specific hypotheses: (1) vigorous-intensity exercise engagement would be associated with decreased smoking; (2) the relationship between vigorous-intensity exercise engagement and smoking would be partially mediated by negative affect; and (3) anxiety sensitivity would moderate these mediated relationships such that the mediational role of negative affect would be stronger for individuals with high levels of anxiety sensitivity relative to those with low levels of anxiety sensitivity. Based on the available evidence, we predicted that anxiety sensitivity would moderate the relationship between negative affect and smoking (i.e. the “b” path, see Figure 3) as opposed to the relationship between exercise and negative affect (i.e. the “a” path, see Figure 3).

## 2. Material and methods

### 2.1. Participants

The sample consisted of 270 young adult smokers and non-smokers (see Table 1). Interested persons responded to advertisements for a study on emotional vulnerability within the greater Burlington, Vermont community. Exclusion criteria for the current study included: (1) limited mental competency or the inability to provide informed, written consent; (2) current suicidal or homicidal ideation; (3) current or past history of psychosis; (4) current (past 6-month) Axis I psychopathology (except for substance use disorders); (5) current major medical problems (e.g., heart disease, cancer); (6) current substance dependence (other than nicotine); and (7) self-reported pregnancy.

The racial distribution of the sample generally reflected that of the State of Vermont (State of Vermont Department of Health, 2007; see Table 1). Those that identified themselves as smokers (approximately 50%) averaged 12.99 cigarettes per day ( $SD = 7.61$ ) with a mean age of onset for daily cigarette use of 16.20 ( $SD = 3.15$ ) years of age. Mean expired air CO levels among smokers in this sample was 15.3 ppm (2.8%), which is consistent with that of a regular daily smoker (10 ppm cutoff; Coccores, 1993). The mean score on the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991) among smokers was 3.53 ( $SD = 2.05$ ), indicating a relatively low-level of nicotine dependence.

### 2.2. Measures

**2.2.1. Diagnostic screen**—The Structured Clinical Interview-Non-Patient Version for DSM-IV (SCID-N/P; First & Gibbon, 2004) screening questions were administered to rule out psychopathology and assess for current suicidal ideation (see exclusionary criteria).

**2.2.2. Vigorous-intensity exercise**—The Exercise Habits Questionnaire Revised (EHQ-R; Zvolensky, 2008) is a self-report measure used to obtain information about participants' engagement in physical activity. The EHQ-R asks respondents to indicate for 29 different physical activities (e.g., running, stair stepping, walking/hiking, swimming, hockey, golf, martial arts, rock climbing, yoga) the number of sessions they have completed in the past two weeks as well as the time spent per session (e.g., less than 20 minutes; 20–29 minutes; 30–39 minutes; 40–49 minutes; 50 minutes or more). This information was used in combination with the compendium of physical activities (Ainsworth et al., 2000) to calculate total minutes of weekly vigorous-intensity exercise.<sup>1</sup>

**2.2.3. Smoking**—The Smoking History Questionnaire (SHQ; Brown et al., 2002) was used to assess current daily smoking. Our dependent variable was number of cigarettes smoked per day. The SHQ has been successfully used in previous studies as a descriptive measure of smoking history (Brown et al., 2002; Zvolensky, Lejuez, Kahler, & Brown, 2004; Zvolensky et al., 2005).

**2.2.4. Nicotine dependence**—The Fagerström Test for Nicotine Dependence (FTND; Heatherton et al., 1991) was administered to measure tobacco dependence. The FTND has shown high internal consistency, positive relations with key smoking variables (e.g., saliva nicotine; Heatherton et al., 1991), and adequate retest reliability (Pomerleau, Carton, Lutzke, & Flessland, 1994).

**2.2.5. Carbon monoxide analysis**—Biochemical verification of smoking history was completed by carbon monoxide (CO) analysis of breath samples (10 ppm cutoff; Cocomares, 1993). Expired air CO levels were assessed using a CMD/CO Carbon Monoxide Monitor (Model 3110; Spirometrics, Inc.). CO analysis of breath samples were used to verify smoking status (abstinence/smoking).

**2.2.6. Negative affect**—The Negative Affect subscale of the Positive Affect Negative Affect Schedule (PANAS-NA; Watson, Clark, & Tellegen, 1988) is a 10-item self-report instrument that measures the tendency to experience negative affective symptoms. The PANAS-NA asks respondents to indicate on a 5-point Likert scale (1 = “very slightly” to 5 = “extremely”) the degree to which they typically feel a list of negative affective states (e.g., “irritable,” “upset,” “afraid”) in the past week. Scores can range from 10 (least amount of reported negative affect) to 50 (greatest amount of reported negative affect). The PANAS-NA has demonstrated good internal consistency in both clinical and non-clinical samples (as ranging from .85 to .93), retest reliability (*rs* ranging from .71 for 2 months to .43 for 72 months), as well as convergent and discriminant validity (Watson, 2000). Internal consistency in the current sample was  $\alpha = .85$ .

**2.2.7. Anxiety sensitivity**—The Anxiety Sensitivity Index (ASI; Reiss et al., 1986) is a 16-item measure, which asks respondents indicate on a five-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., “When I notice that my heart is beating rapidly, I worry that I might have a heart attack,” “It scares me when I am nervous”). The ASI is unique from, and demonstrates incremental validity to, trait anxiety (Rapee and Medoro, 1994) and trait-level negative affectivity/neuroticism (Zvolensky et al., 2005). The ASI has sound psychometric properties in both clinical and nonclinical samples including high internal consistency (Peterson & Reiss, 1992; Taylor, Koch, McNally, 1992; Telch, Shermis, & Lucas,

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<sup>1</sup>For minutes spent per session, we used the midpoint of the range (e.g. 1 = < 20 minutes equaled 10 minutes; 2 = 20–29 minutes equaled 24.5 minutes) and for “50 minutes or more,” we used 50 minutes. We classified activities associated with metabolic equivalent (METS) values greater than 6 as vigorous (Ainsworth et al., 2000).

1989), good retest reliability (Peterson & Reiss, 1992; Maller & Reiss, 1992), and good construct validity (McNally & Lorenz, 1987). Internal consistency for this sample was  $\alpha = .86$ .

### 2.3. Procedure

Interested smokers and non-smokers responding to community advertisements contacted the research team via telephone to participate. Potential participants were given a brief detailed description of the study. After providing verbal consent, trained research assistants further screened potential participants by administering the SCID-NP-screener. Those meeting inclusion criteria were scheduled to participate in the study. Upon arrival to the lab, participants (1) provided verbal and written informed consent, (2) provided carbon monoxide analysis of breath samples to biochemically verify their smoking status; and (3) completed the self-report assessments.

## 3. Results

### 3.1. Preliminary Analyses

Means, standard deviations, and correlations among the study variables are presented in Table 2. All correlations were significant, except for the relationship between anxiety sensitivity and cigarettes smoked per day ( $r = .11, p = .08$ ). Additionally, we screened our data for outliers and, due to the large differences in scales standardized all variables, aiding the interpretation of results.

### 3.2. The Relationship between Vigorous-intensity Exercise and Smoking

We used linear regression to test the hypothesis that vigorous-intensity exercise would be associated with reduced smoking (i.e., the  $c$  path; see Figure 1). Results indicated that higher levels of vigorous-intensity exercise were associated with decreased cigarette smoking ( $b = -.32, p < .01$ ) with vigorous-intensity exercise accounting for 10% of the variance in smoking (see Figure 1).

### 3.3. Negative Affect as a Mediator of the Relationship between Vigorous-intensity Exercise and Smoking

The mediation model is presented in Figure 2. We employed the distribution of products test (MacKinnon, Fairchild, & Fritz, 2007; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002), which has been shown to have greater power and more accurate Type I error rates (MacKinnon et al., 2007; MacKinnon et al., 2002) than the causal steps approach (e.g., Baron & Kenny, 1986). This test also has virtually the same power as bias-corrected bootstrapping (.72 vs. .73) but with lower Type I error rates (.03 vs. .06; MacKinnon, Lockwood, & Williams, 2004). It also has the advantage of being exactly replicable, which is not the case for bootstrapping (MacKinnon et al., 2004). The distribution of products test calculates the magnitude of the joint mediated pathway (the  $a-b$  pathway; see Figure 2) by multiplying the regression coefficients of the two segments of the mediated pathway (i.e.,  $a * b$ ). We then calculated the 95% confidence interval (CI) for this product; CI's that do not include 0 indicate a significant mediated pathway (MacKinnon et al., 2004). To estimate the effect size of the mediated pathway, we calculated the proportion mediated ( $P_M$ ; Shrout & Bolger, 2002).  $P_M$  is the proportion of the total effect of the independent variable on the dependent variable (i.e., the  $c$  path; see Figure 1) mediated by the mediator and is calculated by the formula  $(a * b)/c$ .

Consistent with prediction, regression analyses revealed that vigorous-intensity exercise was inversely associated with negative affect ( $a$  path coefficient;  $b = -.18, p < .01$ ) and negative affect was positively associated with smoking ( $b$  path coefficient;  $b = .21, p < .01$ ). As indicated

by the 95% CI of the  $a * b$  product ( $-.08$  to  $-.01$ ), the mediated pathway was significant. The proportion of the relationship between vigorous-intensity exercise and cigarette smoking mediated by negative affect ( $P_M$ ) was 11.9%.

### 3.4 Anxiety Sensitivity as a Moderator of the Mediated Effect

In order to test the hypothesis that anxiety sensitivity would moderate the mediated pathway (i.e., the negative affect to smoking path [ $b$ ]), we repeated the regression analyses described above, but entered anxiety sensitivity and the anxiety sensitivity by negative affect interaction term as additional predictor variables in the analysis calculating the  $b$  path (see Figure 3). To interpret the meaning of the interaction, we followed the procedures suggested by Aiken and West (1991). Specifically, we first centered the moderator (anxiety sensitivity) at 1 SD above and below the mean and then reran the mediation model separately for those with anxiety sensitivity centered at high anxiety sensitivity and those centered at low anxiety sensitivity (Tein, Sandler, MacKinnon, & Wolchik, 2004; Edwards & Lambert, 2007). These procedures yielded  $b$  path coefficients for those high in anxiety sensitivity and for those low in anxiety sensitivity. We used these path coefficients to ascertain the relations among the variables and to determine the significance and effect size ( $P_M$ ) of the mediation model separately for those high and low in anxiety sensitivity (as per Tien et al., 2004; Edwards & Lambert, 2007).

The regression analyses revealed that the interaction between anxiety sensitivity and negative affect was associated with cigarette smoking ( $b = .15, p < .01$ ). Examining the relation between negative affect and smoking for those high and low in anxiety sensitivity, we found that negative affect was associated with increased smoking for those with high anxiety sensitivity ( $b = .29, p < .01$ ; see Figure 3), but not for individuals with low levels of anxiety sensitivity ( $b = .00, n.s.$ ). The joint mediated pathway among individuals with elevated anxiety sensitivity was significant (i.e., the 95% CI of the  $a * b$  product,  $-.11$  to  $-.02$ ). Further, the proportion of the relationship between vigorous-intensity exercise and smoking mediated by negative affect was 16.8%. These results suggest that, consistent with hypothesis, negative affect as a mechanism underlying the relationship between vigorous-intensity exercise and smoking is more salient among persons with high anxiety sensitivity relative to those with low anxiety sensitivity.

## 4. Discussion

The present study provides preliminary evidence for the hypothesis that negative affect partially mediates the positive effects of exercise on smoking behavior. First, we observed a medium effect size for the relationship between vigorous-intensity exercise and cigarette smoking (i.e.,  $r = -.32$ ). This adds to the growing literature indicating a meaningful relationship between exercise and smoking (Kaczynski et al., 2008). Second, our findings suggest that negative affect accounts for significant variance ( $P_M = 11.9%$ ) in the relationship between vigorous-intensity exercise and smoking. Lastly, consistent with our prediction, these mediated effects varied as a function of anxiety sensitivity. Here, the hypothesized mechanism was evident and clinically meaningful (i.e.,  $P_M = 16.8%$ ; translating into a 40% increase in effect size) among persons with high levels of anxiety sensitivity, but not significant among persons with low levels of anxiety sensitivity. These results are consistent with extant work suggesting that smokers who are high in anxiety sensitivity are less tolerant of negative affect and more likely to regulate negative affect by smoking (Brown et al., 2001; Novak et al., 2003; Zvolensky et al., 2006). Collectively, the present results offer an emerging, albeit initial cross-sectional perspective on the relationship among vigorous-intensity exercise, smoking, and emotional vulnerability. Theoretically, the present results are important as they begin to elucidate the pathways and subgroups related to the interconnection between vigorous exercise and smoking. As in much past work (Ziedonis et al., 2008), the current data are consistent with the perspective

that negative mood vulnerability plays a key role in determining for whom vigorous-intensity exercise influences smoking behavior. Clinically, the current data may point to the possible therapeutic option of using exercise among emotionally vulnerable subgroups of daily smokers in the context of cessation. Although the current sample was not treatment-seeking, nor selected on the basis of anxiety/mood disorders, and in that sense should be considered preliminary, the data nonetheless provide support for considering vigorous-intensity exercise in the treatment of smoking cessation.

A number of limitations of the present investigation and points for future direction should be considered. First, the present study included daily, but not necessarily heavy, smokers as indexed by the rate of smoking per day and level of nicotine dependence. One next step for future work would be to study participants who are heavier smokers and manifest greater levels of nicotine dependence to aid in understanding the generalizability of the observed effect. Second, the present sample is limited in that it is comprised of a relatively homogenous (e.g., primarily young, and Caucasian) and active group. Of particular note, participants' mean time spent in weekly vigorous-intensity exercise was 117 minutes, suggesting that participants, on average, met the recommended public health dose for physical activity (Department of Health and Human Services, 2008). Accordingly, our work can be extended by examining the observed relationships in samples that are less active and more representative of the U.S. adult population. Third, given that self-report measures were employed as the assessment methodology, shared method variance may have contributed to the observed results. Hence, future work in this area should include objective assessment of physical activity (e.g., motion sensors, physiological monitoring). The inclusion of physiological measures may also offer insight into potential alternative or complementary biological mechanisms underlying the vigorous-intensity exercise-negative affect-smoking relationship. Finally, the present study utilized a cross-sectional design. This methodological design cannot elucidate processes over time or isolate causal relations between variables. Thus, the study results are best construed as a "snapshot" of mediational/moderational relations. Experimental work is needed to evaluate the temporal relations between smoking, exercise, and emotional vulnerability over time.

Overall, the present study offers novel empirical insight into the nature of the associations among smoking, exercise, and emotional vulnerability. Results indicate that negative affect accounts for significant variance in the relationship between vigorous-intensity exercise and smoking. Moreover, such mediated effects are particularly evident among persons with high anxiety sensitivity. The potential importance of these findings is amplified when considering that approximately one out five adults who is a daily smoker has a mood or anxiety disorder (Grant, Hassin, Chou, Stinson, & Dawson, 2004) and that quit rates with traditional treatments are significantly lower for these individuals relative to those without mood or anxiety disorders (Lasser et al., 2000).

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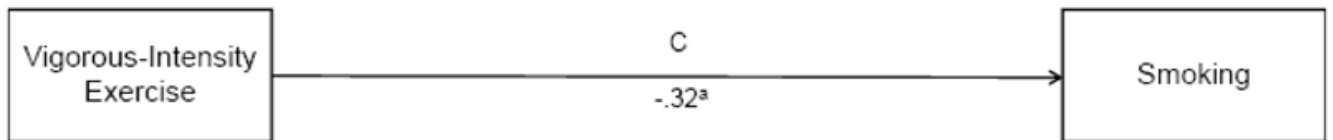
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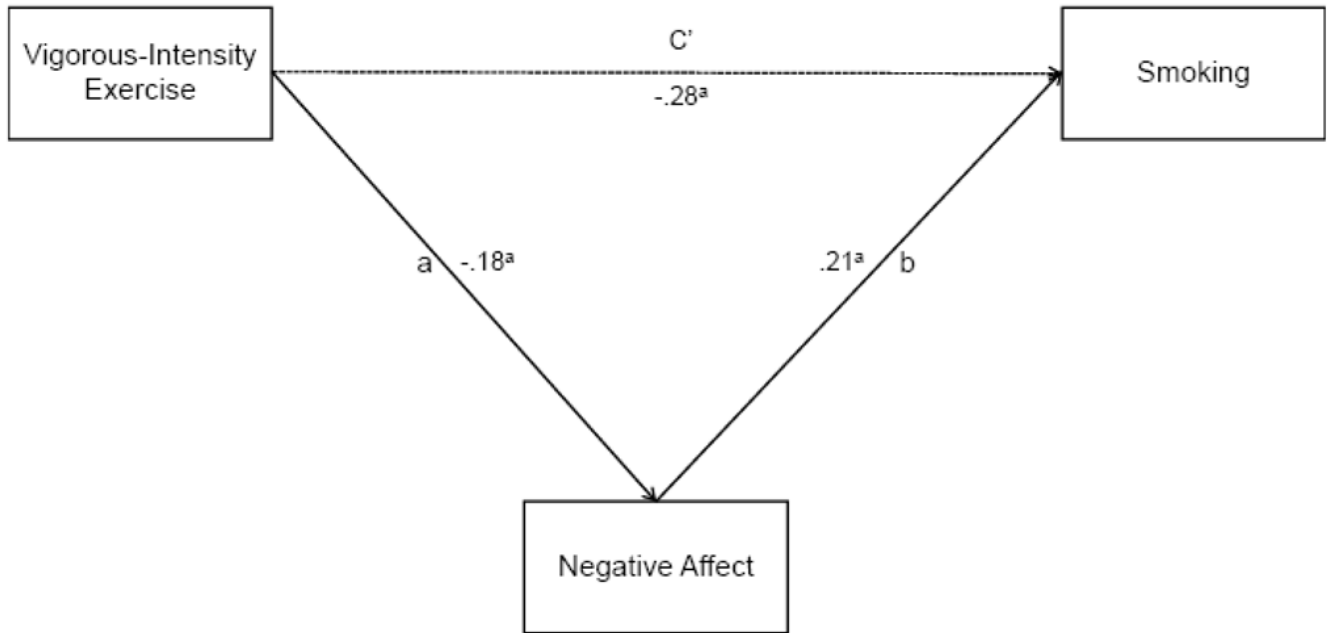
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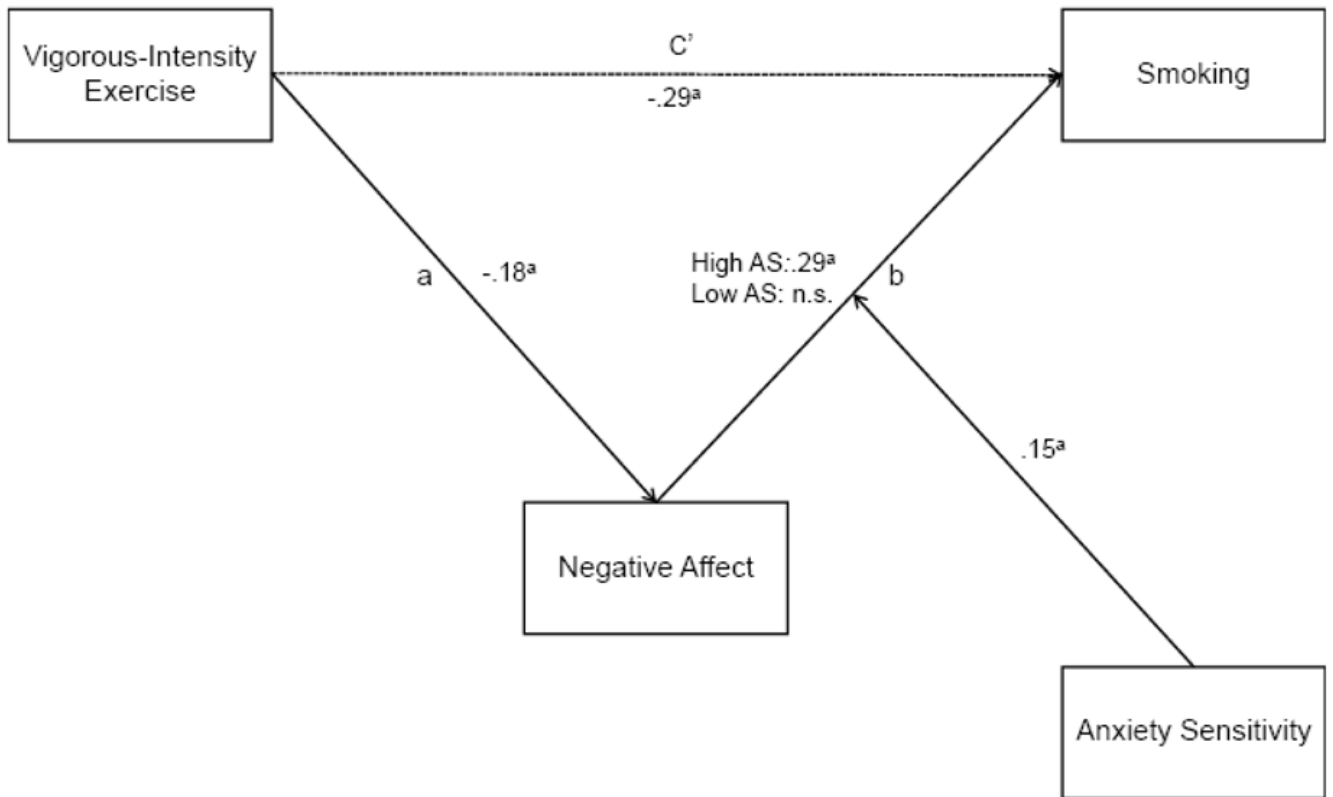
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**Figure 1.**  
The relationship between vigorous-intensity exercise and smoking. *Note:*  $^a p < .01$



**Figure 2.** Negative affect as a mediator of the relationship between vigorous-intensity exercise and smoking. *Note:* <sup>a</sup> $p < .01$ .



**Figure 3.** The meditational effects of negative affect as a function of anxiety sensitivity (AS). Note:  $^a p < .01$ .

**Table 1**

## Demographics of Sample

<b>Variables</b>	
Age	
<i>M</i>	22.4
<i>SD</i>	9.0
Gender	
% <i>Female</i>	52.6
Race	
% <i>White</i>	90.4
Education	
% <i>H.S. Diploma/GED or less</i>	77.8
% <i>Some college or more</i>	21.2

Table 2

## Correlations and Means of Study Variables

Variable	1	2	3	M (SD)	Range
1. Vigorous-intensity exercise				116.68 (150.64)	0–933
2. Negative affect	-.18 <sup>b</sup>			17.85 (5.67)	10–44
3. Anxiety sensitivity	-.15 <sup>a</sup>	.44 <sup>b</sup>		16.49 (8.52)	0–45
4. Smoking	-.32 <sup>b</sup>	.27 <sup>b</sup>	.11	6.74 (8.46)	0–40

<sup>a</sup>  $p < .05$ ,

<sup>b</sup>  $p < .01$

Note: Vigorous-intensity exercise reflects minutes per week spent in physical activities (>6 METS; Ainsworth et al., 2000); Negative affect was measured using the PANAS-NA (Watson et al., 1988). Anxiety sensitivity was measured using the ASI (Peterson & Reiss, 1986); Smoking was measured using the SHQ (Zvolensky, 2008).